THE

JOURNAL

OF

NERVOUS AND MENTAL DISEASE

A MONTHLY PERIODICAL

EDITED BY

B. SACHS, M.D., NEW YORK

WITH THE ACTIVE CO-OPERATION OF

W. R. BIRDSALL, M.D., C. L. DANA, M.D., WM. A. HAMMOND, M.D., G. W. JACOBY, M.D., GRACE PECKHAM, M.D., E. C. SEGUIN, M.D., E. C. SPITZKA, M.D., M. ALLEN STARR, M.D., New York; MORTON PRINCE, M.D., Boston; L. C. GRAY, M.D., Brooklyn; J. G. KIERNAN, M.D., Chicago; ISAAC OTT, M.D., Easton, Pa.; C. K. MILLS, M.D., Philadelphia; Prof. BURT G. WILDER, M.D., Ithaca, and others.

VOLUME XIII.

AMS PRESS, INC.

NEW YORK

Reprinted with permission of
The Jelliffe Trust

Abrahams Magazine Service, Inc.
A division of
AMS Press, Inc.
New York, N.Y. 10003
1968

Manufactured in the U. S. A.

CONTENTS OF VOL. XIII. (Whole Series).

Original Articles.

PAGE

A Contribution to the Pathology of Hemianopsia of Central Origin (Cortex-Hemianopsia). By E. C. SEGUIN, M.D. 1

A Case of Paralysis of the Trigeminus Followed by Alternate Hemiplegia—Its Relations to the Nerve of Taste. By C. L. DANA, M.D. . 65

Race and Insanity. By JAS. G. KIERNAN, M.D. 74

Notes on Electricity. By M. R. CRAIN, M.D. 77

Affections of the Eye Dependent upon Hysteria. By M. LANDESBERG, A.M., M.D. 85

Clinical Observations on Reflex Genital Neuroses in the Female. By PAUL F. MUNDÉ, M.D. 129

Massage in Nervous Diseases. By GEORGE W. JACOBY, M.D. . . 140

Contribution to Neurological Therapeutics. By S. V. CLEVENGER, M.D. 160

A Contribution to the Localization of Focal Lesions in the Pons-Oblongata Transition. By E. C. SPITZKA, M.D. 193

Notes on the Foramina of Magendie in Man and the Cat. By BURT G. WILDER, M.D. 206

Massage in Nervous Diseases. By GEORGE W. JACOBY, M.D. . . 208

A Case of Abscess of the Occipital Lobe with Hemianopsia. By E. G. JANEWAY, M.D. 224

Race and Insanity. By JAMES G. KIERNAN, M.D. 229

The Paroccipital, a Newly Recognized Fissural Integer. By BURT G. WILDER, M.D. (With five figures.) 301

Massage in Nervous Diseases. By GEORGE W. JACOBY, M.D. . . 316

Ophthalmoscopic Studies of Acute Mania, with Notes of Cases. By LOUIS J. LAUTENBACH, M.D., and Dr. ALICE BENNETT . . 337

On the Cause of Electrotonus and of the Normal Formula of Polar Reactions. By G. BETTON MASSEY, M.D. (With four wood-cuts) . 379

Race and Insanity. By JAS. G. KIERNAN, M.D., Chicago . . . 389

A Contribution to the Localization of Focal Lesions in the Pons Oblongata Transition. By E. C. SPITZKA, M.D. (Continued from p. 205) 393

On Convulsive Tic with Explosive Disturbances of Speech (So-Called Gilles de la Tourette's Disease). By C. L. DANA, M.D., and W. P. WILKIN, M.D. 407

A Clinical Study of Lateral Hemianopsia. By E. C. SEGUIN, M.D. . 445

On the Psycho-Neurotic Affections Which Accompany and often Mask Phthisical Disease. By LEONARD WEBER, M.D. 455
The Collocation of a Suture and Fissure in the Human Fœtus. By BURT G. WILDER, M.D. 463
Notes on the Brain. By BURT G. WILDER, M.D. 469
The Evolution of the Special Senses. By W. C. CAHALL, M.D. . . 473
Remarks on Epilepsy. By E. D. FISHER, M.D. 481
PRESIDENTIAL ADDRESS :
Part I.—On the Work of the American Neurological Association. By CHARLES K. MILLS, M.D. 517
Part II.—Arrested and Aberrant Development of Fissures and Gyres in the Brains of Paranoiacs, Criminals, Idiots, and Negroes. By CHARLES K. MILLS, M.D. 523
Part III. Preliminary Study of a Chinese Brain. By A. J. PARKER, M.D., and CHARLES K. MILLS, M.D. 550
Lesion of Both Temporal Lobes, without Word-Deafness or Deafness. By LANDON CARTER GRAY, M.D. 554
Trance Cases in Inebriety. By T. D. CROTHERS, M.D. 565
Is There a Primary Cord Lesion in Pseudo-Hypertrophic Paralysis? By V. P. GIBNEY, M.D., New York 572
On a New Portable Galvanic Battery. By J. RUDISCH, M.D. . . 575
A Contribution to the Study of Tumors of the Spinal Cord. By B. SACHS, M.D. 647
Moral Insanity: A Plea for a More Exact Cerebral Pathology. By JAS. HENDRIE LLOYD, A.M., M.D. 669
Facts and Deductions Bearing on the Action of the Nervous System. By FRANCIS X. DERCUM, M.D. 686
Katatonia. By W. P. VERITY, M.D. 695
Preliminary Communication concerning the Decussation of the Pyramids. By E. C. SPITZKA, M.D. 727
Athetosis, its Treatment and Pathology. By GRÆME M. HAMMOND, M.D. 730

Clinical Cases.

A Case of Trephining for Traumatic Epilepsy. By CHARLES K. MILLS, M.D., and J. WILLIAM WHITE, M.D. 39
Vaso-Motor and Trophic Symptoms of Central Nervous Origin. By M. ALLEN STARR, M.D., Ph.D. 93
A Moral Imbecile. By JAMES G. KIERNAN, M.D. 168
Report of Two Successful Cases of Trephining for Traumatic Epilepsy. By CARLOS F. MCDONALD, M.D. 488
Reports of Cases of Insanity from the Insane Department of the Philadelphia Hospital. By P. LEIDY and C. K. MILLS 707
Vesico-Genito-Post-Femoral Neuralgia and Neuritis. By LANDON CARTER GRAY, M.D. 743
Reports of Cases of Insanity from the Insane Department of the Philadelphia Hospital. By PHILIP LEIDY, M.D., and CHARLES K. MILLS, M.D. 746
Further Note on a Case of Trephining for Traumatic Epilepsy . . 748

Periscope.

a.—Anatomy (including Path. Anat.) of the Nervous System . 44, 245, 714

SPITZKA, The Comparative Anatomy of the Pyramid Tract.—BEEVOR, On Prof. Hamilton's Theory Concerning the Corpus Callosum.—DARKSCHEWITSCH, The Structure of the Pineal Gland.—DARKSCHEWITSCH, The Posterior Commissure of the Brain.—DARKSCHEWITSCH and FREUD. The Connections of the Inferior Peduncle of the Cerebellum with the Posterior Columns of the Cord.—On the Effect of Hardening Methods upon the Microscopic Appearances in the Spinal Cord.—LISSAUER, The Course of Fibres in the Posterior Horn of the Human Spinal Cord, and the Relation of the Lesion of Tabes to It.—OSBORN, The Origin of the Corpus Callosum: A Contribution upon the Cerebral Commissures of the Vertebrata.—BIANCHI, Descending Degeneration in the Brain and Cord Experimentally Produced, and its Relation to the Doctrine of Localization of Functions.—MARCHI, The Degeneration Following the Partial and Total Extirpation of the Cerebellum.

b.—Physiology of the Nervous System (including Experimental Physiology), 47, 96, 170, 247, 353, 580, 720

ARONSOHN and SACHS, The Heat-Centre in the Brain.—CATTELL, The Time it Takes to See and Name Objects.—LUYS, Nouvelles Expériences à Propos de la Locomobilité Intracranienne du Cerveau.—ADAMI, On the Nature of Glomerular Activity in the Kidney.—ELLIS, Plethysmographic and Vaso-Motor Experiments with Frogs.—WATERS, Spinal Vaso-Motor Nerves.—SILVA, On the Mechanical and Electrical Excitability of the Cerebral Cortex of Man through the Cranial Walls.—BECHTEREW, Compulsory Movements Following Destruction of Cortex.—GASTERNATZVY, Recherches Expérimentales sur le Tremblement dépendant de l' Encorce grise des Hémisphéres du Cerveau.—DARKSCHEWITSCH, Division of the Posterior Commissure of the Brain.—SCHIPILOFF and SCHIFF, The Nerves of the Pupil.—JERGERON, Effect of the Long Ciliary Nerves upon the Pupil.—MOSSO, Periodical Respiration and Superfluous Respiration.—GASKELL, Structure and Function of Vascular and Visceral Nerves.—The Relation between the Temperature Changes in the External Auditory Canal and the Circulation of the Brain, *Pflüger's Arekiv.*—CARLE and MUSSO, On the Modification of the Circulation of the Blood in the Brain during Chloroform Narcosis, Accompanied with Painful Excitation.—MITCHELL and LEWIS, Physiological Studies of the Knee-Jerk, and of the Reactions of Muscles under Mechanical and Other Excitants.—HORSLEY and SHAEFER, Upon the Character of the Muscular Contractions Which are Evoked by Excitation of the Various Parts of the Motor Tract.—SCHÄFER, CANNEY, and TUNSTALL, The Rhythm of Muscular Responses to Volitional Impulses in Man.—ARONSOHN, Olfaction.—GOTCH, The Electrical Discharge of the Malapterurus Electricus.—NORI and BALDI, The Cerebro-Spinal Paths of Centrifugal Conduction.—FASOLD, The Physiology of the Hippocampus Major.—FOX and GOULD, On Heat, Considered as the Retinal Intermediate of Light- and Color-Sensation.—VON GUDDEN, On Localization of Function in the Brain.

c.—General Pathology of the Nervous System 50, 99, 174, 250, 356, 413, 496, 582

BERNHARDT, Ueber die Spastiche Cerebral Paralyse im Kindesalter (Hemiplegia Spastica Infantilis), nebst einen Excurse über "Aphasie bei Kindern."—BINSWANGER, Ueber einen Fall von Porencephalie.—MARIE, Note sur l'Existence de l'Ovarie dans la Chorée de Sydenham.—OPPENHEIM, Zur Pathologischen Anatomie der Bleilähmung (Lead Paralysis); SCHULTZE, Ueber Bleilähmung.—BEEVOR, A Case of Amyotrophic Lateral Sclerosis with Clonus of the Lower Jaw; DE WATTEVILLE, with a Note on the Jaw-Jerk or Masseteric-Tendon Reaction in Health and Disease.—WESTPHAL, On a Peculiar Group of Symptoms Associated with Disease of the Posterior Columns of the Spinal Cord.—BENNETT, A Case of Locomotor Ataxia without Disease of the Posterior Columns of the Spinal Cord.—MOLLIÈRE, Fracture du Rachis.—JOFFROY, Pied-Bot Taoetique.—DU SAULLE, Eight Cases of Epilepsy Caused by the Sight of a Dead Body.—MUSSO, On General Pseudo-Paralysis by Slow Intoxication from the Oxide of Carbon.—Reflex Neurosis from Nasal Disease, *Med. Record.*—DU SAULLE, Le Rhumatisme Cérébral, la Folie Rhumatismale, et la Goutte Cérébrale. DRESCHFELD, Alcoholic Paralysis.—UHTHOFF, Loss of Pupil Reflex to Light.—OPPENHEIM, A New Method of Testing Tactile Sensibility in Unilateral Brain Lesion.—WESTPHAL, On Two Cases of Tabes Dorsalis with Presence of Knee-Jerks.—BOMPARD, Sclerose Primitive des Cordons Latéreux de la Moelle, ou Tabes Spasmodique.—MARIE, Paramyoclonus Multiplex.—BENNETT, Case of Brachial Monoplegia Due to Lesion of the Internal Capsule.—WIGGLESWORTH, A Case in Which an Old Amputation of the Left Upper Arm was Associated with an Atrophied Right Ascending Parietal Convolution.—WILKINS, Cases of Cerebellar Disease.—

Köppen, Histological Changes in Multiple Sclerosis.—Babinski. The Histological Changes in Spinal Sclerosis.—Rossolymo, Descending Degeneration in the Crus Cerebri.—Hughlings-Jackson, A Contribution to the Comparative Study of Convulsions.—Bennett, Case of Cerebral Tumor.—Edinger, Aphasia and Paresis of both Hypoglossal Nerves Due to a Small Lesion in the Centrum Semiovale.—Greidenberg, Post-Hemiplegic Disturbances of Motion.—Weil, Paralysis of the "Glottis-Dilators" as the Initial Symptom of Tabes Dorsalis.—Ross, On a Case of Locomotor Ataxia with Laryngeal Crises and One of Primary Sclerosis of the Columns of Goll, Complicated with Ophthalmoplegia Externa.—Ozeretskovski, Hysteria in Soldiers.—Kostjurin, Senile Changes in the Brain.—Sharkey, The Gulstonian Lectures on Spasm in Chronic Nerve-Disease.—Rossini, On Hysterical Anuria with Secretion of the Urine by the Stomach, and Experimental Researches Made in Hysterical Anuria in Relation to Uræmia.—Rosenbach, A Contribution to the Doctrines of the Innervation of the Movements of Expression.—Tabes with the Presence of Knee-Jerks, *Mendel's Neurol. Centralbl.*—Oppenheim and Siemerling, Contributions to the Pathology of Tabes.—Schmalfuss, The Effect of Extirpation of the Ovaries upon Nervous Symptoms.—Erb, Histological Changes in the Muscles in Cases of the "Juvenile Form" of Dystrophia Muscularis Progressiva.—Babinsky, De l' Atrophie Musculaire dans les Paralysies Hystériques.—Gombault, Note Relative à l' Existence de la Nérvite Segmentaire Périaxile, à Propos d' un Cas de Paralysie Diphtheritique.—Buzzard, Some Forms of Paralysis Depending upon Peripheral Neuritis.—Déjérine, Absence of Patellar Reflex as the only Symptom of Locomotor Ataxia.—Dupin, Ablation of the Motor Centres.—Déjérine, Radial Paralysis due to Compression.—Quinquaud, Physiological Action of Hypnone.—Pitres and Vallard, Alteration of Peripheral Nerves in Chronic Articular Rheumatism.—Langley Recent Observations on Degeneration, and on Nerve Tracts in the Spinal Cord.—Swasey, Embolism of the Medulla.—Symmetrical Vaso-Motor Paralysis of Both Hands, *Gazette des Hôpitaux*.—Schachmann, Poisoning by Carbonic Oxide.—Oppenheim, Beiträge zur Pathologie der "Multiplen Neuritis" und Alkohol-Lähmung.—Bernhardt, Ueber die Multiple Neuritis der Alkoholisten: Beiträge zur differentiellen Diagnostik dieses Leidens von der Tabes, der Poliomyelitis subacut. u. der sogenannten, Landry'schen Paralyse.—Vierorot, Degeneration of the Columns of Goll in an Alcoholic Subject.—Eulenburg, On a Family Form of Congenital Paramyotonia Occurring in Six Successive Generations.

d.—Mental Pathology 55, 106, 182, 292, 366, 418, 586

Hughes, Post Neuralgic Insanity.—Holmes, Puerperal Insanity.—Wood, Alcoholic Insanity.—Landesberg, Pyschoses after Cataract-Operations.—Brower, Insanity from Cocaine.—Tuke, Moral Imbecility.—Savage, Hæmaturia and Purpuric Extravasations in Typhomania.—Wood, Imperative Conceptions.—Southerland, Early Stages of Dementia.—Kiernan, Race and Paretic Dementia.—Hughes, Transitory Insanity.—Wood, Definition of Insanity.—Conrad, Suicide.—Stedman, Consciousness in Melancholic Frenzy.—Andrews, Schools in Hospitals for the Insane.—Cloiston, Somatic Disease and Insanity.—Wilmarth, Idiot Brains.—Wood, Classification of Insanity.—Savage, Drunkenness in Relation to Criminal Responsibility.—Wilson, Alcoholism.—Channing, The Connection between Insanity and Crime.—McDonald, Report of a Case of Insanity following Gunshot Injury to the Head; Cerebral Cyst; Aspiration; Recovery.—Benner, Phthisis and Insanity.—Insanity among Convicts.—Hughes, Transitory Insanity.—Wiggleworth, Puerperal Insanity.—Norman, On Insanity Alternating with Spasmodic Asthma.—Bivona, Cataleptic State in Acute Poisoning by Corrosive Sublimate.—Laulor, The Insane in Associated Dormitories.—Hughes, Melancholia.—Morselli, Anatomy of the Pupillary Reflex in Dementia Paralytica.—Parvin, Nymphomania.—Wright, Insanity in the United States.—Verga, Insanity in Italy.—Régis, A Seventeen-Year-Old Paretic Dement.—Mendel, Paretic Dementia and Syphilis.—Fisher, Insane Self-Mutilation to Secure Pecuniary Damages.—Shuttleworth, Kalmuc Idiocy.—Brower and Catlett, Cocaine in Insanity.—Shuttleworth, Moral Imbecility.—Magnan, Psychical Character of Hereditary Paranoiacs.—Catlett, Insanity and Aural Disease.—Kiernan, Melancholia from Paranoia.—Engel, Paretic Dementia and Cerebral Lues.—Hagenbach, Surgery among the Insane.—Rodriguez, Epileptic Insanity.—Fisher, Mental Excitement after a Cataract-Operation.—Stearns, Increase of Insanity in the Aged.—Adams, Visceral Lesion or Disorder and Mental Disease.—County Care of the Insane.

—Therapeutics of the Nervous System . . 110, 268, 373, 513, 723

Frazer, The Action and Uses of Digitalis and its Substitutes, with Special Reference to Strophanthus.—Musser, On the Influence of Age on the Dosage of Nux Vomica, with Some Remarks on its Therapeutics.—Wetherell, Hyoscine Hydrobromate.—Hypnone, a New Hypnotic.—Millard, The Treatment of Sciatica and Neuralgic Affections by Methyl Chloride.—Hypnone.—Hopeine.—Huchard, Urethane or Carbamate of

Ethyl.—KIERNAN, Quebracho in Melancholia.—STURGIS, The Treatment of Recurrent Headache in Children.—MAYS, The Nutritive Value of Some Beef Extracts —DE RENZI, Production of Sweat in Various Diseases and the Effects of Pilocarpine.—DIGNAT, A Case of Epilepsy, with Peripheral Aura, Cured by Blistering at the Seat of Aura.—CHEADLE, The Influence of Treatment on Chorea, with Special Relation to the Full Use of Arsenic and its Results.—HARE, The Physiological and Therapeutic Effect of Adonidin. —FOURNIER, A Few Words about Hypnotism from a Therapeutic Point of View.—DE WATTEVILLE, The Electrical Resistance of the Body —SOUTHAM, The Result of Nerve-Stretching for Facial Spasms Five Years after the Operation.—WHITE, Antipyrin in Headache.—JACKMAN, Urethane in the Treatment of Traumatic Tetanus.—OTTO and KOENIG, Urethane in Insanity.—FAYRES, Sciatica Cured by Puncture of the Sheath.—DOWNER, Nerve-Stretching for Anæsthesia of Leprosy.—MAYS, Another Local Anæsthetic.—SUMBROSO, Hysterical Mania Cured with Hypnotism.—CANGER, Cure of Mental Maladies by Hypodermic Injections of Hydrochlorate of Morphine in Large Doses.—HACK, Basedow's Disease Cured by Operation. KRÖNLEIN, Trephining for Meningeal Hemorrhage.

Reviews and Bibliographical Notes.

ROBERTS. Operative Surgery of Brain 113
MEYNERT. Psychiatry: A Clinical Treatise on Diseases of the Fore-Brain, Based upon a Study of its Structure, Functions, and Nutrition. 115
TUKE. The Insane in the United States and Canada 187
MOEBIUS. Allgemeine Diagnostik der Nervenkrankheiten . . . 272
ERLENMEYER. Die Principien der Epilepsie-Behandlung . . . 273
HEIBERG. Schema der Wirkungsweise der Hirnnerven. Ein Lehrmittel für Aerzte und Studirende 274
LOMBROSO. L'Uomo Delinquente (Criminal Man) 274
ERB. Die Thomsen'sche Krankheit (Myotonia Congenita) . . . 375
LANNOIS. Nosographie des Chorées 421
WEILL. Des Vertiges 422
BUZZARD. On Some Porms of Paralysis from Peripheral Neuritis of Gouty, Alcoholic, Diphtheritic, and Other Origin 423
LUCIANI and SEPPILLI. Die Functions-Localisation auf der Grosshirnrinde 589

Society Reports.

New York Neurological Society 118, 277, 425, 749
Philadelphia Neurological Society 58, 123, 191, 285, 435, 761
American Neurological Association 593

Editorial Notes and Miscellany . . 63, 128, 299, 378, 442, 644, 778

In Memoriam:

Dr. T. A. McBRIDE 642

LIST OF CONTRIBUTORS.

Bennett, Alice, M.D.
Cahall, W. C., M.D.
Clevenger, S. V., M.D.
Crain, M. R., M.D.
Crothers, T. D., M.D.
Dana, C. L., M.D.
Dercum, F. X., M.D.
Fisher, E. D., M.D.
Fuller, F. C., M.D.
Gibney, V. P., M.D.
Hammond, G. M., M.D.
Jacoby, G. W., M.D.
Janeway, E. G., M.D.
Jastrow, J.
Kiernan, J. G., M.D.
Landesberg, M., A.M., M.D.
Lautenbach, Louis J., M.D.
Leidy, Phillips, M.D.
Lloyd, J. Hendrie, A.M., M.D.
MacDonald, C. F., M.D.
Massey, G. Betton, M.D.
Mills, C. K., M.D.
Mundé, Paul F., M.D.
Ott, Isaac, M.D.
Parker, A. J., M.D.
Peckham, Grace, M.D.
Prince, Morton, M.D.
Rudisch, J., M.D.
Sachs, B., M.D.
Seguin, E. C., M.D.
Spitzka, E. C., M.D.
Starr, M. Allen, M.D., Ph.D.
Verity, W. P., M.D.
Weber, Leonard, M.D.
White, J. William, M.D.
Wilder, Burt G., M.D.
Wilkin, W. P., M.D.

VOL. XIII. JANUARY, 1886. No. 1

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

A CONTRIBUTION TO THE PATHOLOGY OF HEMIANOPSIA OF CENTRAL ORIGIN (CORTEX-HEMIANOPSIA).

By E. C. SEGUIN, M.D.

THE importance of hemianopsia with reference to the recently developed doctrine of the localization of functions in the cerebral cortex is so great as to merit the closest study on the part of the physiologist and the practical neurologist. Few subjects of such apparently small intrinsic importance have attained to such a magnitude as this one, rendering quite impossible to treat of it fully in a paper for an ordinary Society meeting. Only one or two of its phases can be considered within the time allotted me, and I shall therefore limit my remarks to the relation of the symptom hemianopsia to certain central or cerebral lesions, and endeavor to show its value for purposes of diagnosis in actual practice, rather than develop its significance for the solution of physio-psychical problems.

My apology for presenting the subject is that during the past winter it was my fortune to observe a typical case of lateral hemianopsia, stationary till the patient's death many months afterward, and almost unaccompanied by other cerebral symptoms. The topographical diagnosis made during life was verified by the post-mortem examination, so that,

apart from its scientific interest, I may venture to submit the case as an encouragement to the making of positive diagnoses *intra vitam*, in the light of the rapidly growing laws of so-called cerebral localization.

Before relating the case and presenting the specimen I should make a few remarks upon the scope of the paper, and briefly state certain data relative to parts of the subject which I cannot treat in full.

First, then, as to the scope and plan of the paper. I shall consider only the recorded cases of hemianopsia in which the autopsy revealed a lesion in some part of the brain inclusive of the optic thalami. Since the publication of Dr. Starr's valuable *résumé* of cases of hemianopsia in January, 1884, their number has somewhat increased, and I am able to tabulate forty. I should add that I have endeavored to obtain the original essay in each case, and have carefully prepared the abstracts myself: only one, No. 0, by Prévost of Geneva, has been impossible to obtain, and I quote it upon Westphal's authority, but exclude it from my tables. This labor I was induced to perform in order to avoid errors which otherwise easily occur in quoting cases, and also to be able to group these cases and more fully appreciate and present their pathological and diagnostic value. It is far from me to claim that my collection is absolutely perfect, but it is, I believe, almost complete and reliable in its critical arrangement. Let me repeat that I am anxious to present this relatively very rich and singularly harmonious collection of cases in such a way that it shall prove of most use to the practising physician for diagnostic purposes.

Second, as to the subject of hemianopsia in general.

The fact that a person might temporarily or permanently see only one half of objects placed directly in front of him has been known to physicians more than one hundred years. In 1723 Vater and Heinecke described three cases under the name of *visus dimidiatus*.

The same phenomenon was designated as hemiopia at the close of the last century, probably first by A. G. Richter, a term which prevailed and is still employed though in a dif-

ferent sense since the introduction of hemianopia by F. Monoyer in 1865, and of hemianopsia by J. Hirschberg in 1877. The latter is the preferable and preferred form.

As the terms are now accepted, hemiopia signifies loss of perceptive power in one lateral (or vertical) half of the retina, while hemianopsia means obscuration of one lateral (or vertical) half of the visual field. As rays of light cross within the eye before reaching the retina, it follows that, for example, right hemiopia is equivalent to left hemianopsia; or, in other words, that nasal hemiopia corresponds to and causes temporal hemianopsia.

In describing cases at the present time, it is customary and preferable to omit all mention of the retinal condition, or hemiopia, and to describe the hemianopsia, or the state of the visual field as determined by the perimeter or by ruder though sufficient tests.

Several varieties of hemianopsia are recognized.

1. *Horizontal*, superior, or inferior hemianopsia, almost always due to defects within the eye, and of relatively small interest to the neurologist.

2. *Vertical* hemianopsia, almost always due to lesions of the retro-ocular nervous visual tract, and hence of great importance in neuro-pathology. Quite a number of terms have been employed to designate the varieties of vertical hemianopsia. Of these we recognize and adopt:

(*a*) Temporal hemianopsia.

(*b*) Nasal hemianopsia.

(*c*) Lateral hemianopsia, often designated as homonymous hemianopsia.

The first two varieties are exclusively caused, as far as our present knowledge goes, by lesion of the optic chiasm, of its lateral, or of its frontal or caudal borders.

The last variety, lateral hemianopsia, is always produced, as far as our present knowledge goes, by lesions of one optic tract, or of the more central parts of the optic apparatus as far caudad as the cortical centre for vision in one hemisphere.

The object of this paper is to study the recorded cases of lateral hemianopsia, with autopsies, due to lesions situated

in the more caudal parts of the optic apparatus, its central portions, from the primary optic centres (lobi optici, corpora geniculata lateralia) to the cortical visual centres, of areas.

With reference to all three forms of hemianopsia, I shall assume the following propositions as established :

1. The fact of a semi-decussation in the optic chiasm or man has been proven chiefly by the researches of von Gudden. According to these recent views (which are in part a return to the ancient theoretical statements of Newton, Wollaston, Müller, Hannover, and von Graefe) the optic fasciculi are disposed as follows : The fibres of each tractus opticus at the chiasm divide into two parts : A larger one which decussates with its homologue and enters into the composition of the opposite optic nerve, supplying the nasal half of the retina. This is the fasciculus cruciatus. The other, smaller set of optic-tract fibres does not decussate but passes on directly to form a part of the optic nerve of the same side, supplying the temporal half of the retina. This is the fasciculus lateralis. Thus each retina receives nerve fibres from both optic tracts, or, in other words, each optic tract contains fibres destined for both retinæ.

The inter-retinal fasciculus of Hannover is purely imaginary ; there are no such fibres. The posterior loop of Hannover is now known, since the experiments of von Gudden, to be composed of non-optic fibres ; it is the inferior cerebral commissure.

2. The connection of the optic tracts with the corpora geniculata lateralia and the lobi optici (anterior corpora quadrigemina) is an intimate one, but probably (in man) more for trophic and reflex purposes than for vision. Whether mere perception of light (as an excitant) may take place in these bodies after removal of the hemispheres, is still an open question. Certainly sight, in the ordinary meaning of the term, is impossible under such conditions.

3. A total lesion of one tractus opticus fatally produces lateral hemianopsia of the fields opposite the lesion.

4. A lesion acting upon one side of a tractus opticus so as

to compress only some of its fibres will produce one-sided nasal hemianopsia.

5. A lesion acting simultaneously on the sides of the optic chiasm will, by injuring both fasciculi laterales, produce nasal hemianopsia in both eyes.

6. A lesion compressing the optic chiasm in its frontal or caudal borders will produce bilateral temporal hemianopsia by injury to both fasciculi cruciati.

7. All such lesions are apt to be accompanied by pupillary irregularity or immobility, by optic neuritis or atrophy; and their diagnosis is further facilitated by finding signs of paralysis of other basal nerves, or of crossed hemiplegia.

8. It must be borne in mind that a lesion of the hemisphere may be so situated as to press downward upon one tractus opticus, and thus produce hemianopsia of the peripheral type (see case of Hirschberg, No. 5).

9. Lesions of the lobi optici in man have been rarely observed, and when observed have been bilateral in their effects, so that nothing can be said at present of hemianopsia due to disease of these parts.

With this brief introduction I now pass on to the consideration of the clinical and pathological aspects of the subject of my paper as exemplified in forty cases with autopsies, and five traumatic cases without autopsies, which I have been able to collect.

After a careful analysis I have grouped these forty-five cases into six categories.

1. Cases which are indefinite or useless for the study of localization, four in number.

2. Cases of lesions of parts which we have good reason to believe unconnected with the central optic apparatus, and which produced hemianopsia by pressing upon the optic tracts or the chiasm, three in number.

3. Cases in which the hemianopsia was due to a lesion of the corpus geniculatum laterale or the thalamus opticus, or both, six in number.

4. Cases of hemianopsia due to lesion of the white substance of the occipital lobe, eleven in number.

5. Cases of traumatic hemianopsia, due to injuries of the

occipital region of the skull and lesion of the subjacent brain, five in number.

6. Cases of hemianopsia due to lesions of the cortex of the brain, cortex only, or also of the subjacent white substance, sixteen in number. In this class I have included my own case. Among these sixteen cases there are four (Nos. 28, 29, 41, 45) in which the lesion was circumscribed, and where it occupied so nearly the same spot in the cortex cerebri as to afford us, in my opinion, a solution of the problem of the location of the cortical visual centre in man.

In order to shorten this essay for publication I have tabulated the cases according to the above grouping of the cases.

The four conclusive cases I shall, however, offer in full abstract illustrated with diagrams in order to enable the reader to more fully appreciate their value. But first I shall give the details of a traumatic case which is of extreme interest, from the facts that hemianopsia has existed as the sole symptom for twenty-three years, and that the cicatrix in the head is so distinct as to allow of study at present.

CASE 3.—Keen and Thomson. P. H., a soldier, aged twenty-three, was wounded in the head by a minié ball, during the battle of Antietam, in September, 1862. The missile entered the skull in the median line, 1¼ inches above the external occipital protuberance, and made its exit at a point 2 inches distant from the median line, and 3 inches distant from the point of entrance. There was no immediate loss of consciousness. In the next few days the patient complained of impaired vision. Ten days after the injury, loss of consciousness, with right-sided hemiplegia, occurred. Paralysis and imperfect memory lasted for two or three months. Apparently no aphasia.

When seen by the authors, in 1870, there was no paralysis, and the mental functions were unimpaired. The patient complained that the vision of his right eye was deficient. The pupils, ocular muscles, and fundus were normal. The left cornea bore an old opacity. Central vision on right side, = 1; on left side, ⅔. The fault complained of by the patient was found to be a complete right lateral hemianopsia, with a vertical division line.

Recently I traced this soldier, through the Pension Bureau at Washington and the office at Philadelphia, to his home in that city. He has consented to come here this evening, in order to make the report more exact. By the courtesy of Drs. Keen

TABLE I.

Cases of Hemianopsia in which the Relation between the Lesion and the Optic Defect was not Evident; Indefinite and Insufficiently Reported Cases.—Four in Number.

No.	Observer.	Sex.	Age.	Fundus and Pupils.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
12	Charcot and Pitres, 1877.	F.	52	(?)	Left lateral H. (De Wecker and Landolt.)	(?)	Localized epileptiform attacks in l. face, neck, and arm. Left hemiplegia with flaccid muscles.	Various old patches of softening in different gyri. Recent red softening of cerebellum.	State of occipital lobes, optic tracts, and chiasm not mentioned.
21	Linnell, 1881.	M.	63	At first exam. fundus normal; at second, right side of left disk pale. V. $\frac{20}{30}$ or $\frac{20}{40}$.	Left lateral H.	Sclerosis (?) of right optic tract. Softening of corp. quad. et genic. (?)	Attacks of neuralgia in head and limbs for 8-9 years. For 4 years "paralysis agitans" right side, with numbness and tingling. Sudden blindness after severe headache: improvement in V. Blindness again, general tremor, halluc. of V. Death.	Large recent clot in right hemisphere ant. to its centre, and wholly in white substance. Left hemisphere normal. Tubercula quad. in state of white softening, as also the corp. genic. and ventral part of thalamus, more on left side. Right optic tract firmer than left, chiasm and nerves normal.	
25	Petrina, 1881.	M.	53	Normal fundus and papillæ. V. not measured: no mention of refraction or accommodation, or pupils.	No hemianopsia. Amblyopia of left eye: after using this eye alone a few minutes every thing becomes confused and gray.	Fissure in right lambdoid suture. Pachymeningitis, meningeal hemorrhage, and softening or degeneration of cortex of occipital gyri, especially the 2d and 3d, from sulcus temporalis sup. anteriorly to fissura calcarina behind. (?)	Fall backward on occiput with loss of C.; vomiting, headache, and vertigo. Failing sight. No motor or sensory symptoms. All special senses normal except sight. Died of pneumonia.		Apparently no oculist saw the case. If report is correct there was probably paralysis of accommodation in l. eye. The lesion may not have involved the cuneus.
40	Wiethe, 1884.	M.	54	Fundus normal. Pupils and V. not mentioned. Binocular V. preserved.	Superior lateral hemianopsia.	Lesion of left thalamus opticus. (?)	Fall upon occiput; unconscious, and bleeding from nose, mouth, and ears. Violent headaches, impaired mental action; no paralysis. In a few weeks apoplectic attack with left hemiplegia; recovery. Later, complete (?) blindness developed in 1½ hours: partial recovery. Death from hernia.	Atheromatous cerebral arteries. Old hemorrhagic foci in r temporal lobe, lentiform nucleus, medullary subst. of frontal lobe and gyrus olfact. Also in left parietal lobe and left thalamus. Chronic pachymeningitis.	Extent of lesions not well given. No special mention of occip. lobe and optic tracts.

TABLE II.

Cases of Hemianopsia from Lesions Unrelated to the Cortical Centre for Sight; Cases from Pressure Transmitted to Optic Tracts, etc.—Three Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
5	Hirschberg, 1875.	M.	40	Fundus normal. Central vision normal.	Right lateral H. with sharply defined vertical line passing close to point of fixation.	Left tractus opticus smaller than right.	For four years severe left-sided, intermittent headache. Imperfect V. to right. Right hemiplegia. Aphasia.	Glio-sarcoma, size of an apple, in left frontal lobe.	Tumor may have pressed on tractus. State of occipital lobe not mentioned.
8	Huguenin, 1876.	F.	46	(?)	Right lateral H. with a not well defined vertical line.	(?)	Attack of unconsciousness; right hemiplegia with partial anæsthesia, aphasia, alexia, and word-deafness.	Embolism of l. middle cerebral artery; softening of Broca's gyrus, gyrus precentralis, et postcentralis, in their lower parts; insula, external capsule, claustrum, and external division of N. lentiformis.	State of occipital lobe not mentioned.
16	Pflüger, 1878.	M.	62	(?)	Left lateral H.	Injury of inferior part of thalamus by clot, with pressure on tractus. (?)	Attack of cerebral hemorrhage.	In right lateral ventrical much semi-fluid blood. Hemorrhage in corpus striatum, and inferior part of thalamus.	State of occipital lobe not mentioned.

TABLE III.

Cases of Hemianopsia from Lesions of the Thalamus Opticus and Corpora Geniculata.—Six Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
6	H. Jackson and W. R. Gowers, 1875.	M.	65	(?)	Left lateral H.	Softening of caudal half of right thalamus; pulvinar disintegrated.	Left hemiplegia and hemianæsthesia. Death from a non-cerebral disease.		
9	Pooley, 1877.	M.	55	V. normal when first seen. Later, choked disk in left eye, normal fundus in right eye.	Fields normal when first seen. Sudden right lateral H., passing away, but returning in a few days.	Complete softening of left thalamus and surrounding white substance.	For 6 years epileptiform seizures, hallucinations of sight, maniacal attacks. Right hemiparesis, reduced sensibility, aphasic symptoms, weak memory.	In left occipital lobe there was a "gummy tumor" 12×30 mm., adherent to pia. Right lateral ventricle much dilated.	Syphilitic arteritis and tumors.
19	Dreschfeld, Case I., 1880	F.	41	(?)	Left lateral H.	A tuberculous tumor almost replaced the right thalamus, and extended laterad through int. capsule to nucleus lentiformis. Right tractus opticus reduced to a thin band.	Paroxysms of headache and weakness of left leg for 7 years. Apoplectic attack and left hemiplegia, convulsions, coma.	The tumor extended in a ventral direction almost to the surface of the brain.	
30	Dreschfeld, Case II., 1882.	M.	40	Fundus normal. Central V. good.	Left lateral H.	Cysto-sarcoma in place of lateral part of right thalamus; lobus opticus, internal capsule and part of nucleus lentiformis involved. Right optic tract flattened.	Headache, giddiness, diplopia, left-sided paresis and tremor for 18 mos. Three convulsions. Left hemianæsthesia, and hemiplegia.		No syphilis.
31	Dreschfeld, Case III., 1882.	F.	52	Pupils equal and react well. Central V. fairly good.	Left lateral H., with a vertical line not quite reaching point of fixation.	A clot, ovoid and 6×8 mm., in the upper and post. extremity of right thalamus (pulvinar). Optic tracts, lobus opticus, and corp. genic., normal.	Bright's disease and mitral stenosis. Apoplectic attack, followed by left hemiplegia and transient aphasia. Also diminution of sensibility on left side of body. Special senses normal except V.	Several clots within the r. brain: two corresponding to fasciculi from precentral gyrus, and one under the parietal gyri.	
36	Rosenbach, 1883.	F.	34	(?)	Right lateral H.	Softening of external part of left thalamus; tumors in post. segments of both lobi optici; atrophy of left tractus opticus and of right optic nerve.	Right hemiplegia, right amblyopia. Epileptoid convulsions. Amnesic aphasia. Impairment of mental activity. Symptoms of pressure.	Softening of corpus striatum and internal capsule.	

TABLE IV.

Cases of Hemianopsia from Lesions Situated Chiefly in the White Substance of the Occipital Lobe.—Eleven Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
2	Levick, 1866.	M.	40	(?)	H., of which side not stated, when first seen, Feb'y 9, 1866.	Abscess in posterior lobe of right hemisphere, 1½ inches in diameter (Pepper).	In May and June, 1865, slight (?) injuries to head, by falling objects. In July and August, headaches and extreme drowsiness; vertigo, sense of feeling drunk. No convulsions or paralysis. Death in coma eight months after injury.	Opacities in arachnoid. Various structures at base of brain softened and discolored. Small abscess in anterior lobe of left hemisphere. Lateral ventricles lined by consistent whitish-yellow inflam. lymph.	
14	Hosch, 1878.	M.	54	V. R. $\frac{10}{20}$, V. L. $\frac{10}{30}$. Slight redness and veiling of papillæ, and a few streaks of hemorrhage in retinæ.	Left lateral H. with vertical line a little to l. of point of fixation.	Large hemorrhagic cyst, almost destroying right occipital lobe to cortex.	Slight apoplectic attack and weakness of left side of body, with darkening of l. fields of V. Complete left hemiplegia after a third apoplectic attack. In fourth attack, right side paralyzed; death.	Large pigmented cicatrix in right corpus striatum, extending into thalamus. N. caud. et lentif. atrophied. Last attack due to a large fresh hemorrhage in 3d vent., causing extensive laceration.	
13	Baumgarten, 1878.	M.	(?)	Central V. normal.	Left lateral H. suddenly developed and persistent.	In the substance of the right occipital lobe was an old hemorrhagic cyst as large as a walnut, and the various convol. of the occipital lobe were softened, though recognizable.	None (?). Death in several months, from paralysis of the heart.	In centre of right thalamus was a so-called apoplectic cicatrix, half size of a lentil. Tractus optici and optic nerves normal. Fatty heart and contracted kidneys.	
15	Dmitrowsky and Lebeden, 1879.	F.	22	Papillæ congested, obscure limits, veins enlarged.	Right lateral H.	In the left cerebral hemisphere, a clot which involved the greater part of the corona radiata, and penetrating the temporal lobe almost to the cortex.	Headache, drowsiness, and difficulty in speaking. Aphasia.	?	

TABLE IV.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesions.	Other Symptoms.	Other Lesions.	Remarks.
22	Westphal, Case I., 1881.	M.	42	Slight optic neuritis in l. eye.	Left lateral H. Vertical line passing through point of fixation.	Old focus of softening in white subst. of parietal and occip. lobes, as low as 2d temp. gyrus. Gyri of parietal and occip. lobes smaller and softer than those of left side. Volume of caudal end of right hem. much less than that of left hem.	Left-sided convulsions, followed by l. hemiplegia. Clonic spasms in paroxysms, lasting days or hours, often without loss of C. At end, some contracture of l. arm. Very slight anæsthesia; transient.	Basal optic apparatus and thalami normal.	
24	Senator, 1881.	M.	69	(?)	Left lateral H.	Clot occupying greater part of white subst of right temporal lobe outside inf. horn of ventricle. Dorsally lesion destroys a large part of white subst. of inf. parietal lobule; mesad it extends to the lateral part of thalamus; complete interruption of post. part of internal capsule; occip. and temporal radiations cut through.	Left hemiparesis; contracture; conjugate deviation to right; speech normal. Sensibility to pain preserved. Later, movements of right arm ataxic; r. leg paralyzed.	Thalamus normal; right corp. genic. lat. flattened and yellowish-red. The clot also extended frontad through ext. capsule and outer segment of n. lent., as far as insula. Rest of brain normal.	
38	Stenger, Case VII., 1882.	M.	32	At close of life, for two months, blindness and hallucinations of V.	Left lateral H. Verified by repeated tests.	In white substance bordering right post. horn, is a distinct, rather broad furrow, extending from post. border of thalamus caudolateral.	Symptoms of demeotia paralytica; excitement, tremors, thick speech. Attack of l. hemispasm and hemiplegia. Later, attacks of left-sided spasm.	Decortication over left parietal lobe and whole of both occip. lobes, which are shortened and shrunken. Post. horns much dilated; the right more. Both thalami, especially in post. thirds, are collapsed and softer. Optic tracts equal and normal.	

TABLE IV.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
34	Wernicke and Hahn, 1882.	M.	45	No lesion in fundus.	Right lateral H. Vertical line passing a little to right of point of fixation.	Abscess in white subst. of l. hemisphere, latero-dorsad of post. horn of V. At its frontal end the vent was opened. The caudal end of abscess almost reached the apex of occip. lobe; mesad the abscess was limited by the ependyma. Destructioo of white subst. of sup. and inf parietal lobules.	Chronic phthisis. Pain in l. frontal and occipital regions; a cloud before right eye. Mind dull; right arm and leg awkward. Paresis of r arm and leg; loss of muscular sense in r. arm. Later, complete paralysis. Trephining in upper post. quadrant of l. parietal bone; abscess in brain evacuated by deep incision. Relief to motor symptoms, etc. On 6th day, stupor, paralysis, and death.	Ventricle perforated and containing pus. No meningitis.	The diagnosis of abscess of occip. lobe was made *intra vitam* and confirmed. Surgically the case is most encouraging.
35	Jany, 1883.	F.	21	V. much reduced. R. + $\frac{1}{60}$, S. — $\frac{6}{36}$. L. fingers counted at 8'. Neuro-retinitis passing into atrophy, more in r. eye.	Right lateral H. In r. eye vertical line a little outside point of fixation. In l. eye outer field contracted.	Cysto-sarcoma occupying nearly whole of l. occip. lobe. Solid tumor lies at apex of occip. and mesad, as far as occipito-parietal sulcus.	Severe headache, most in l. occip region Vertigo. Paresthesia and analgesia of r. hand and face. Stammering. Later, no objective symptoms, except in eyes; occip. pain, vomiting, convulsions; death.	Cystic part of tumor, of orange size, involves white subst. about post. horn of V., and under inferior parietal gyri.	
38	Richter, 1883, Case I.	M.	54	Pupils equal, normal in size and action. Optic nerves pale; vessels smaller.	Left lateral H. Vertical division line.	Clot of a certain age in r. occip. lobe in white subst., just laterad of post. horn of V., separated from it by ependyma. Cortex uninjured.	Hallucinatory paranoia Left eye weak, and left hand numb. Left hemiplegia. Death in apoplectic attack.	Most ancient clot in r temporal lobe. Third and fresh clot had disorganized the crura, filled third and fourth ventricles.	
39	Schmaltz. Cited by Vetter, 1883.	F.	69	Pupils and ocular muscles normal.	Right lateral H.	Yellow softening in left hemisphere, most destructive in occipital lobe. Caudal part of thalamus softened.	Apoplectic attack, followed by r. hemiplegia. Complete r. hemianæsthesia. Muscular sense lost. Left arm with choreiform movements. State of smell and taste uncertain.	Yellowish softening of various gyri in l. brain; pre- and post central, parietal, and occip. gyri.	

ADDITIONAL CASE

46	Prevost, in Bulletin de la société méd de la Suisse Romande, 1878.	M.	75	Fundus normal (Haltenhuff).	L. lateral H.	Yellow patch in right occipital lobe, from its mesal gyri inward into white substance along the post. horn of ventricle as far frontad as corp. genic. lat. and thalamus. Lobus opticus normal.	Attack sudden but without loss of c. Slight left hemiplegia. Partial left hemianæsthesia (hearing, taste, and smell uncertain because of stupidity).	Old thrombus of right posterior cerebral artery. Recent thrombosis of basilar. External gyri of brain and insula normal.	

TABLE V.

Cases of Hemianopsia due to Injuries to the Cranium and Brain; Lesion mostly Cortical.—Five Cases.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
3	W. W. Keen and W. H. Thomson, 1871.	M.	32	V. R. = 1. V. L. $\frac{2}{3}$. Opacities ant. cornea Pupils, ocular muscles, and fundus normal.	Right lateral H. Vertical division.	In Sept., 1862, received a gunshot wound of post. end of cranium on left side. Entrance in median line $1\frac{1}{4}$ inches (31 mm.) above the external occipital protuberance Exit 2 inches (50 mm.) from median line, and 3 in. (75 mm.) from entrance.	No immediate loss of consciousness. Impaired V. soon noticed. Ten days later loss of consciousness followed by r. hemiplegia. No aphasia. Paralysis and imperfect memory lasted for 2-3 months. No paralysis in 1870.		
Idem.	Observed by E. C Seguin, in 1885.	M.	46	Pupils normal. Eye muscles normal. Fundus: blood-vessels normal, outer temporal quadrant of both disks whiter than normal; left a little whiter than right. V. R = $\frac{20}{40}$ with — $\frac{1}{72}$; ax. 90. V. L. $\frac{18}{70}$ with — $\frac{1}{72}$; ax 90 R. reads No. 14 J. at 12" with + $\frac{1}{2}$ S. No 1 at 8". L. reads No. 14 J. at 12" with + $\frac{1}{2}$ S.; No. 6 at 8". (Dr. Hale, House Surgeon, Manhattan Eye and Ear Hospital.)	Right lateral H. Division line not quite reaching point of fixation.	Entrance cicatrix barely noticeable. Exit is a large cup-shaped depression 5 × 0.5 cent. and 1.5 cent. deep. Bridge of bone between entrance and exit only 3 cent. Bottom of depression is firm but not osseous. Injury imitated on the cadaver shows an injury of parietal lobe, dorsad of angular gyrus. Occipital gyri uninjured. White substance deeply lacerated.	One epileptiform attack 6 years ago (1879). Right side a little weaker; more easily affected by alcohol. Uses left hand habitually. Mental action good.		Hemianopsia caused by injury to optic fasciculus Occipital gyri uninjured.

TABLE V.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
4	Hughes, 1873.	M.	38	Eyes normal except hemianopsia. (Prof. Wilson, Drs. Jacob and Swansy, of Dublin.)	Right lateral H. unchanged at time of discharge from hospital.	Injured by fall of a large iron vessel on back part of head. Compound depressed fracture of occipital and parietal bones. Several pieces of bone removed and coma relieved at once. Complete recovery in 3 mon. except impairment of V.	Coma relieved by operation.		
20	Schmidt-Rimpler, 1880.	M.	(?)	?	Left lateral H.	Vertical compound fracture of right occipital and parietal bones by a fall. A handful of bony splinters and some brain substance were extracted.	No paralysis. Recovered with complete deafness of r. ear, left lat. H., and occasional dizziness with loud tinnitus in r. ear.		
23	Heuse, 1881.	M.	(?)	Slight opacities of cornea, reducing V. and rendering ophthalmoscopic exam. unsatisfactory.	Right lateral H. Right temporal and left nasal half fields were not absolutely dark but dim or very obscure.	Fall upon head causing a depressed fracture of cranium. Enormous cicatricial depression of the bones on the left side of the occipital end of the skull; a strong ridge-like depression extended from left parietal into the occipital bone.			
37	Nieden, 1883.	F.	22	During second paralytic seizure before operation: Eye muscles and pupils normal. Fields normal. Slight venous hyperæmia of papillæ and retinæ. Ninth day after operation V. L. = $\frac{12}{12}$. Field normal. V. R. $\frac{9}{20}$, temporal field wanting to median line.	After operation monocular H. temporal field of right eye and in part of its superior inner (nasal) quadrant. Field of left eye limited in its upper and nasal areas.	Fall down steps, striking occiput repeatedly on succeeding steps. Unconsciousness, vomiting, pain in l. occipital region. At operation, 9 months later, no lesion found, but the dura mater (and brain) was injured by trephine to left of median line. After operation, complained of impaired vision.	Paralysis with partial anæsthesia of right side of body. Mental action normal. Great recovery in six months. Seven months after injury severe headache, vertigo, semi-unconsciousness. Right hemiplegia; face not involved. Also right hemianæsthesia. Attacks of headache with loss of C.; jerks of *left* side muscles, face, and limbs. Trephined in left upper quadrant of occip. bone. All symptoms passed away and, 13 months after original injury, she returned to her work.		This case was probably one of hysterical nature, and even the optic symptoms may have been of same sort.

TABLE VI.

Cases in which Lateral Hemianopsia was due to Lesions of the Cortex of the Brain and Subjacent White Matter (mostly Cortical Lesions).—Sixteen Cases (Arranged in the Order of their Value for Studying the Localization of the Visual Centre).

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
1	Chaillou, 1863.	F.	69	(?)	Lateral H. Side not stated; most probably on left side, corresponding with hemianæsthesia observed	Atrophy of gyri at the end of left (?) fiss. of Sylvius and of inner face of occipital lobe; white matter of occip. lobe much atrophied.	Disorder of speech followed by coma. Recovery with imperfect V. and weakness of r. arm. Previous to admission patient had had a similar "congestive" attack, leaving left hemianæsthesia. Hemianopsia older still. Death in a third attack	Small patches of softening in left (?) thalamus. Inferior part of left corpus striatum contained a cavity 2 x 5 cent. Right hem. of cerebellum in inf. aspect contained a cavity (old patch of softening) 3 x 1 cent. Oldest lesion was that in occip. lobe.	Sides of brain probably reversed in autopsy. Lesion of occip. lobe and thalamus must have been in *right* hemisphere.
26	Westphal, Case II., 1882.	M.	38	Central V. normal. R. pupil trifle wider. Eye-muscles not stated. No lesion to ophthalmoscope.	R. Lateral H. discovered June, 1880. Nearly vertical line passes a little to left (?) of point of fixation.	Softening of gyrus angularis, of occipital lobe (much adhesion over cuneus and precuneus). Optic nerves and chiasm normal.	In June, 1879, alcoholic (?) convulsions, followed by delirium and imperfect speech. In august, awoke with r. hemiplegia (arm most) and complete loss of speech; word-deafness and amnesic aphasia. Later, repeated convulsions (clonic) on r. side. Slight r. sided anæsthesia and paresis; great loss of muscular sense.	Left hemisphere showed an extensive cortical softening, involving post-central as far as supra-marg. gyrus, the whole of parietal lobe, gyrus angularis, and nearly the whole of occipital lobe. Slight softening of cortex at junction of 2d and 3d temp. gyri. No central lesion.	
32	Stenger, Case VIII., 1882.	M.	52	Special senses normal on admission.	L. lateral hemianopsia after left hemiparesis. Persistent.	Softening of white subst. of r. par. lobe interrupting fasciculus of Gratiolet.	Admitted with dementia paralytica. Shortly after, convulsions in left side of body, followed by l. hemiparesis with numbness of l. hand. Muscular sense much impaired in l. hand. Varying l. hemiparesis. Death in general convulsions.	Int. and ext. hydrocephalus. Cortical softening over right parietal and occipital lobes; subjacent white subst., yellowish, and much atrophied. Right thalamus smaller and softer than left. Int. aspect of occip. lobe not mentioned. Right post. horn much distended.	Degeneration from visual cortex along optic fasciculus to thalamus.

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
7	Förster and Wernicke, 1876.	M.	—	(?)	At first, temp. field of right eye obscured vertically almost to point of fixation; the nasal field of left was darkened also almost to point of fixation. The left temp. field was also slightly contracted. Later, a degree of left lateral hemianopsia was found, but the original limits of the right hemianopsia remained unchanged.	The area of softening caudad of the fissure of Sylvius, including the inf. parietal lobule and gyrus angularis, penetrated to the lateral ventricle, probably interrupting the optic fasciculus.	Repeated paralytic attacks on right side; aphasia.	Embolus in left Sylvian artery; softened patch in cortex, including inf. parietal lobule, gyrus angularis, and frontal part of occip. lobe; numerous small necrotic foci in l. nucleus lenticularis, nucleus caudatus, thalamus, and external capsule. Insula, chiasma, and optic nerves normal.	This case has been sometimes cited twice, as separate accounts were published by W. and F.
10	Jastrowitz, Case I., 1877.	M.	(?)	Transient paralysis of R. N. VI. Papillæ normal; V. relatively good; fixation possible.	Right lateral hemianopsia (Hirschberg).	Soft sarcoma of left occipital lobe, involving occip. gyri and precuneus. It extended in conical form toward the post. horn of lateral ventricle as far laterad as Gratiolet's fasciculus which presented a slight discoloration and punctate hemorrhages as far as the thalamus.	Vertigo; loss of memory, of energy; general weakness; amnesic and ataxic aphasia, alexia, and agraphia. Right hemiplegia appeared later; paralysis of variable intensity.	Thalami, lobi optici, tractus, chiasma, and nervi optici presented no alterations.	
18	Curschmann, 1879.	M.	50	(?)	Left lateral H., which persisted from 10th to 16th day (death).	Large focus of softening in right occipital lobe, extending to the surface, especially on its caudal and mesal parts.	Drank sulphuric acid with usual local effects. There occurred, on 10th day, embolism of right brachial artery. A few days later, patient complained that he could not see well with his left eye. No other symptom of local disease in brain. Death, of inanition, on 16th day.	Usual lesions of œsophagus, etc. An extension of inflam. to inner coat of aorta. Complete embolism of right brachial artery.	

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
11	Jastrowitz, Case II., 1877.	(?)	(?)	(?)	Limitation of visual fields to the right.	Large patch of softening in left occipital lobe.	Aphasia.	Partial embolism of left internal carotid artery.	
17	Nothnagel, 1879.	M.	51	(?)	Partial right lateral H.; shortly before death, total blindness.	In left hemisphere (besides several small lesions) there was a yellowish red softening of the entire occipital lobe. Chiasma, optic tracts, and nerves normal.	Patient awoke with paralysis of left arm and obscurity of vision. There was monoplegia of left arm; no anæsthesia. Death by inanition.	Carcinoma of pancreas, with various secondary deposits. Endocarditis aortica verrucosa. Numerous spots of embolic softening in brain. In r. hem. a patch in middle of precentral and postcentral gyri, softening of caudal extremity of sup. parietal lobule, with extension into interparietal fissure, and into white substance to ventricle. Small patches in r. occipital lobe.	
27	Marchand, 1882.	M.	72	(?)	Left lateral H. — "complete." (Dr. Pepmüller)	Patch of softening in right occipital lobe; pia adherent; apex of occip. lobe occupied by a necrotic patch as large as a hazel-nut, separated from deeper parts of brain by a softened yellowish zone about .5 cent. thick.	Sudden left hemiplegia. Death in a few months. No details as to motor and sensory symptoms.	Gyri adjacent to occipital in same condition of yellowish softening. Arteries tortuous and thickened.	
41	Richter, Case II., 1885.	M.	70	After H. there occurred conjunctivitis, keratitis, cataract; phthisis of left eye.	Complete left lateral H. Defect persisted in right eye.	Patch of softening in right occipital lobe.	Senile dementia.		
43	Richter Case III., 1885.	M.	48	L. pupil reacts well; right very slightly. Left optic nerve normal; right, atrophied.	Left Lateral H.	Patch of softening in right occipital lobe.	Syphilis in 1865. Since 1880, repeated apoplectic attacks, accompanied by temporary r. hemiplegia and aphasia. In 1882, l. hemiplegia and hemianopsia. Dementia.	Small psammoma in r. optic nerve. Patch of softening in l. island of Reil.	

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
44	Richter, Case IV., 1885.	M.	40	Pupils react well. Fundus normal. Muscles normal. Shortly before death, no ophthalmoscopic changes.	Left lateral H. suddenly developed in hospital. Limit to left of fixation point.	Firm adhesion of pia to occipital gyri, also over cuneus, and lower temporal gyri. Tip of occip. lobe least injured. Granular bodies found in white substance of occip. lobe, from cuneus and gyrus hippocampi, and from inferior temporal gyri, along outer wall of ventricle, in a tract which can be followed to atrophied pulvinar. No atrophy of tractus or optic nerves.	Syphilis in 1861. Epileptiform attack, mental failure, staggering gait, weak legs, tongue tremulous, speech thick. Hallucination of V. Death from epilepsy.	Basal nerves normal. In various places, opacity and thickening of pia.	
45	Seguin, 1885.	M.	46	Pupils and fundus normal. Central vision good with glasses for presbyopia (Dr. C. R. Agnew).	Sudden attack of left lateral H. persisting till death. Vertical line passing a little to left of point of fixation. Was able to read and write. Always complained that his left eye alone was affected.	Patch of old softening involving almost the whole of the right cuneus, not quite reaching apex of lobe, including a part of the gyrus hippocampi, the fifth temporal, and encroaching on the fourth temporal gyrus. No other lesion. (?)	Mitral regurgitation, enlargement of the heart, various peripheral embolisms, pseudo-intermittent fever. No hemiplegia or anæsthesia. Slight ataxia of left hand. Death six months after hemianopsia appeared.	Verrucose disease of mitral valve, tissue containing balls of micrococci. Large infarcts in kidneys and spleen. No other important superficial lesions on surface of brain. Chiasm, tracts, and nerves normal.	The absence of hemiplegia, distinct anæsthesia, and convulsions makes it almost certain that there were no gross lesions within the brain. Location of lesion correctly diagnosticated during life.
28	Haab, 1882.	M.	68	Central vision — 1. Optic nerves presented "a senile grayish color."	Left lateral H. Complaint of left eye only. Vertical line reaching quite (?) up to point of fixation. H. persisted till death.	Caudal end of right hemisphere .5 cent. shorter than its fellow. Patch of softening (cavity and debris) mostly on mesal aspect of occip. lobe, including apex. It involves the cuneus in its inferior half, the fifth temporal gyrus, and fissura hippocampi. White substance destroyed as far as ventricle.	Endo- and peri-cardiitis, sudden attack of left hemiparesis rapidly passing away almost wholly. No hemianæsthesia. Death in two years.	No other cerebral lesion. Optic nerves, chiasm, and tracts normal (microscopic examination).	Diagnosis during life; embolism of artery supplying hinder part of right thalamus.

TABLE VI.—*Continued.*

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
41	Fér6, 1885.	F.	52	Ophthalmoscope showed no lesions. Pupils not mentioned.	Right lateral H. Vertical line passing through point of fixation.	Yellow patch destroying the greater part of the left cuneus, and encroaching somewhat on fifth temporal gyrus. No secondary degeneration.	Nov. 2, 1883, slight and transient attack of right hemiplegia. On admission to Salpêtrière, no motor symptoms, but slight r. hemianæsthesia to pain and cold. Hearing, taste, and smell normal.	No other cerebral lesions.	Lobi optici, tractus, chiasm, and nerves normal.
29	Huguenin, 1882.	F.	8	At first visit, April 16th, vision and hearing good. Optic nerves normal. On 27th, slight optic neuritis.	Left lateral H. discovered on May 20th.	A caseous tumor. 3 x 3 x 2.5 cent. lay on the mesal aspect of the right occipital lobe; partly embedded in the brain, firmly adherent to pia. It was over the fissura hippocampi, extending above and below it, and into the cuneus.	Pertussis followed by slow mental action and ill-health; a few months later, headache in paroxysms, vomiting; recurring convulsions; increasing dementia. Death of broncho-pneumonia; never localized paralysis, or anæsthesia.	A small tumor on the apex of the right frontal lobe. Ependyma of ventricles granular; pia over chiasm, and in both fossæ Sylvii, slightly thickened.	

and Thomson, I have the additional information that a few days ago the hemianopsia was found unchanged, twenty-three years after the reception of the injury. I made an examination of the patient, Hughes, this morning, with the following results :

He presents no distinct paralysis or anæsthesia, or aphasic symptoms. His tongue deviates a trifle to the right, and the grasp of the right hand is a little less than that of the left. Dynamometer test : L, 38, 34 ; R, 35, 34. The knee-jerk is abnormally great on both sides, but equally so.

Tests reveal no anæsthesia, but the patient thinks that his tactile sensibility is very slightly dull on the right side of head and in right hand. The muscular sense, tested by knowledge of passive movements of fingers, and by different weights laid in both hands, while the patient's eyes are closed, is normal.

He habitually uses the left hand more than the right, but this is on account of his loss of vision on the right.

He adds that when under the influence of liquor his right leg and arm feel the effects first and most.

Only one epileptiform seizure is known to have occurred, viz.: an attack in the night, about six years ago. He claims that his memory is now good. It was formerly weak, but he never, apparently, had amnesia of words.

The scalp presents two cicatrices ; that of entrance very small, that of exit of the ball enormous and greatly depressed. The following are some topographic measurements with the head placed so as to have the skull resting on the alveolo-condyloid plane of Broca.

The entrance wound is in the median line, about 3.5 cent. above the external occipital protuberance. From the bregma along the median line to this cicatrix is 15.5 cent.

The exit scar is a large cup-shaped depression situated dorso-laterad of the other, near the parietal eminence. Its frontal edge is 6.5 cent. from the bregma ; its mesal edge nearly at the median line (.5 cent. distant) ; and its fronto-lateral edge is 12.75 cent. from the left tragus. Its transverse diameter is 5 cent., its longitudinal diameter 6.5 cent. Its depth is 1.5 cent. The bridge of bone between the two scars is only 3 cent. broad.

The bottom of the exit scar is very firm though not bony, and the patient is not affected by reasonable manipulation.

A rough test with a small white object at 18 inches shows right lateral hemianopsia, with line passing outside of point of fixation ; there is besides a darkened area in the left upper temporal quadrant.

Dr. G. W. Hale, House Surgeon of the Manhattan Eye and Ear Hospital, has very kindly made an examination of H.'s eyes, and made diagrams of his visual fields. He finds the following :

R = $\frac{20}{50}$: $\frac{20}{40}$ w. — $\frac{1}{12}$; ax. 90° :
L = $\frac{160}{200}$: $\frac{16}{40}$ w. — $\frac{1}{12}$; ax. 90° :
R reads No. 14 J at 12″ w. + $\frac{1}{12}$s No. 1 J at 8″ :
L reads No. 14 J at 12″ w. + $\frac{1}{12}$ No. 6 J at 8″ :

Pupillary reaction, normal.
Eye muscles : no insufficiency either at 20′ or 1′.
Fundus : blood-vessels of normal size ; outer temporal quadrant of either disk whiter than normal, left a little whiter than right. No other lesions.

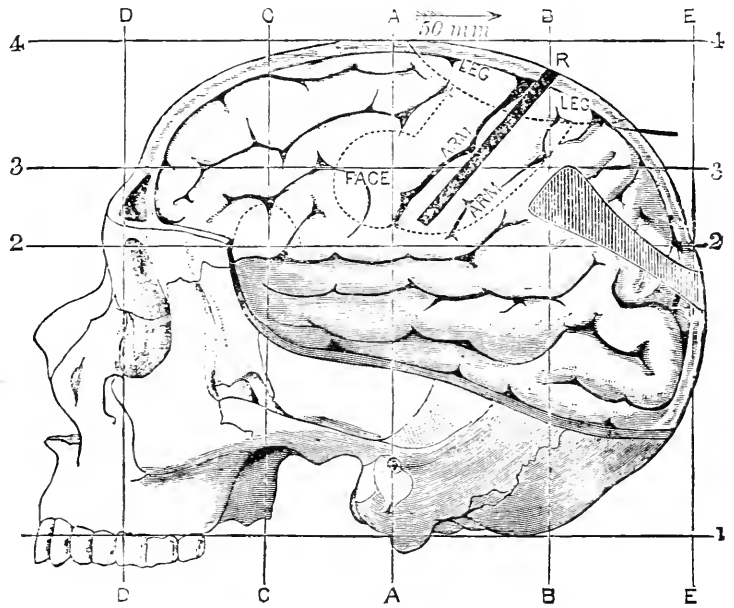


FIG. 1.—The probable course of the minié ball through the brain in case 3 (Keen and Thomson) is indicated by the club-shaped shaded figure in the occipital part of the head, extending across lines 2 and B.

By the kindness of Drs. Peabody and Ferguson I have had the opportunity of repeating the injury upon the cadaver at the New York Hospital. Trephine openings were made in the cranium of a male subject at points corresponding with the author's measurement of H.'s cicatrices, and an iron rod pushed through, followed by a large seton of jute. The hemisphere was placed in alcohol for harden-

ing. It was then found that the track of the ball was entirely dorsad of the occipital, through the parietal lobe almost to the confines of the postcentral gyrus. Its penetration was such, however, that it must have injured the optic fasciculus on its way to the cuneus. See fig. 1.

CASE 28.—Haab: male, æt sixty-eight years. In Feb., 1878, while under treatment for endo- and peri-carditis, experienced an attack of paresis of the left extremities. This rapidly passed away, leaving a certain degree of disability, for after working with the left hand patient experienced pain in the left arm, and palpitation.

When seen by Haab in July the patient complained that he

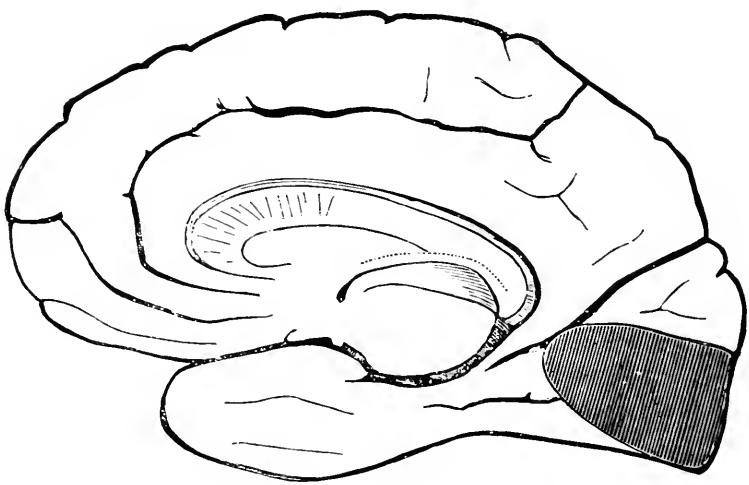


FIG. 2.—Mesal aspect of right hemisphere (Ecker) showing patch of softening in case 28 (Haab).

could not see to his left with his left eye—thought his right eye was normal. Examination showed only a trace of paresis or awkwardness in left extremities; no anæsthesia. Intelligence normal. Hearing good. Central vision = 1 (H. 2). There was left homonymous hemianopsia, the limit reaching quite (?) up to the fixation point. In right fields color-perception good.

Optic nerves present a "senile grayish color."

During the year several re-examinations gave the same results. The patient insisted that there was a veil or cloud over the *left* eye. Death in July, 1879.

Autopsy.—The caudal end of the right hemisphere was .5 mm. shorter than its fellow. There was a depression in right occipital lobe, the pia hanging loosely over a cavity containing clear fluid. The patch was mostly upon the mesal aspect of the hemisphere (including apex). It occupied the site of the fissura hippocampi, and extended beyond it above and below. The frontal end of the cyst was at six cent. from the apex of the lobe. The white substance was but slightly injured, and there was no communication between the cyst and the posterior horn of the ventricle. The vertical height of the patch was 2–3 cent.

No other cerebral lesion. The optic nerves, chiasm, and tractus were normal to a microscopic examination. Haab's diagnosis during life was embolism of an artery supplying the hinder part of right thalamus opticus.

CASE 29.—Huguenin. A girl, æt. eight years. In autumn of 1878 whooping-cough, followed by ill-health and sluggish mental action. In January, 1879, headache in paroxysms ; later, frequent vomiting, sleep broken ; no motor or ocular symptoms. At end of March severe convulsions, which have frequently recurred, constituting the principal phenomenon. Increasing dementia. Seen by Huguenin, 16th April, 1879. Child demented ; understands what is said, and, according to parents, replies well ; memory feeble ; general muscular weakness, but no localized paralysis. Vision and hearing good. Seems sensitive to pinching, etc. Optic nerves normal.

Temporary improvement under KI. and syr. ferri iodidi.

April 27th, second ophthal. exam. Slight neuritis with some swelling (no "stauung") Headaches. In middle of May it was noticed that patient held her head obliquely to the left. Exam. on 20th revealed left homonymous hemianopsia. This symptom was the only one indicating a focal lesion of the brain, and it persisted. Death in June, of broncho-pneumonia.

Autopsy.—Two tumors were found in the brain ; one at the apex of the left frontal lobe, the other near the apex of the right occipital lobe. Ependyma of ventricles granular ; slight thickening of pia over chiasma and in both fossæ Sylvii.

The second tumor lay in the mesal aspect of the right occipital lobe, projecting a few mm. above the level of the brain, firmly adherent to the pia and only slightly to the

dura. Its length was 3 cent., height 3 cent., thickness 2.5 cent.—mostly buried in brain substance. It lay directly over the sulcus hippocampi, extending to either side of it. Basis of occip. lobe not involved. Tumors caseous.

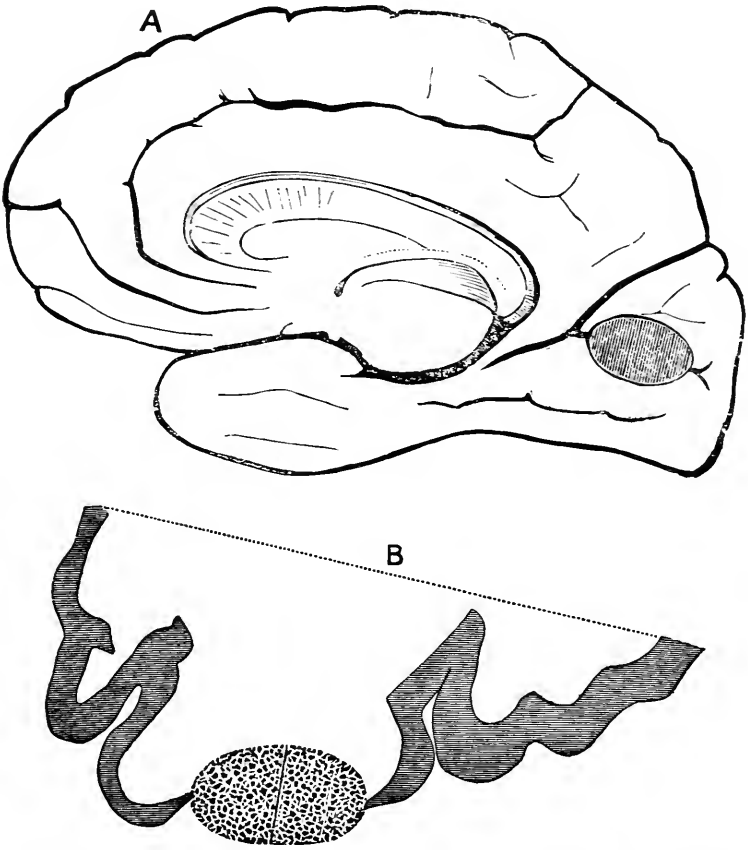


FIG. 3.—A. Tumor in mesal aspect of right occipital lobe : left lateral hemianopsia. B. Diagram of horizontal section, showing the slight penetration of the tumor. Case 29 (Huguenin).

CASE 41.—Féré. Female, æt. fifty-two. In November, 1883, sudden apoplectic attack followed by transient right hemiplegia. On admission to the Salpétrière, no motor symptoms. Partial and slight right hemianæsthesia to cold and pain. Hearing, taste, and smell normal. Typical right lateral hemianopsia, vertical line

passing through point of fixation. No ophthalmoscopic lesions; state of pupils not mentioned.

Death Dec. 24, 1884. Autopsy showed only a yellow patch destroying the greater part of the left cuneus and encroaching somewhat on the adjacent second temporal gyrus (gyr. temp. 5 of Ecker). No secondary degeneration. Corpora geniculata, lobi optici, tractus, chiasm, and optic nerves normal. Gray commissure of 3d V. absent.

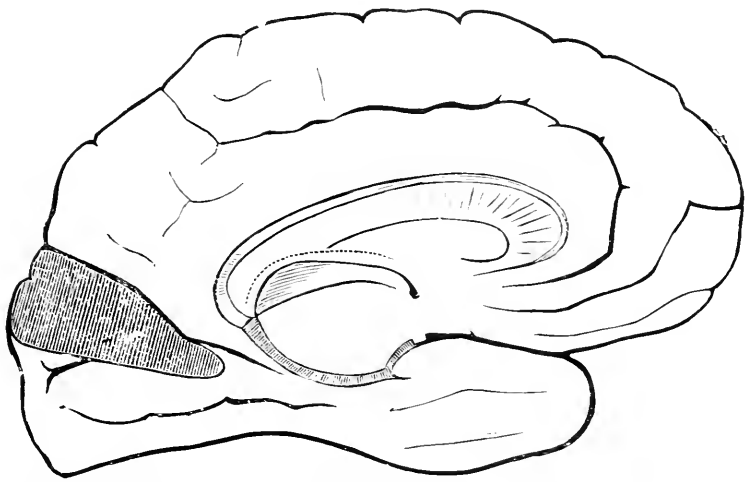


FIG. 4.—Mesal aspect of left hemisphere (Ecker). Patch of softening causing right lateral hemianopsia. Case 41 (Féré).

CASE 45.—Seguin. Mr. J. W. D., æt. 46, consulted me on January 18, 1884, for insomnia and dyspepsia. Wakefulness was most marked in early morning. Has grown paler, weaker, and thinner of late. Denies dyspnœa. Examination showed general anæmia, feeble, slow pulse (63 to 66 beats per minute). Heart feeble, with a distinct, harsh, mitral regurgitant murmur. There were pulsations in the external jugular veins. Urine normal, though of high specific gravity.

Under digitalis, cannabis indica, nux vomica, and arsenic, in various combinations, and a much more nutritious diet, with a glass of rich claret at his meals, a good recovery was obtained in about six weeks. Sleep was sound; the patient had regained weight and color.

November 26, 1884, I was sent for to visit him at his home. I learned that in the spring he had travelled south as far as Havana, and returned in excellent health, to all appearances. He was then, and for the succeeding three or four months, much overworked, and especially worried about his business, which was far from

prosperous. He had given up his claret, and, most unfortunately, purchased and used quite actively a rather strong home-gymnasium. His house was situated at the top of one of our hilliest streets, and this he climbed rapidly every day.

I found him suffering apparently from regular intermittent fever; severe chills followed by high fever and sweating. He had been severely purged, and was quite weak. His heart was larger than when first seen, and the mitral murmur was much louder and more diffused. He was given quinine and nutritious food.

A few days thereafter, about December 5th, I was sent for in haste, because of an attack of a nervous nature. I found Mr. D. considerably alarmed, but rational, and free from serious symptoms. He complained of numbness in the whole left side, cheek, arm, leg, and trunk; most marked in the hand and foot. There was no distinct hemiplegia, and no anæsthesia to ordinary tests; he thought, however, that tactile sensibility, as tested by passing his fingers over objects, was somewhat duller. He was most concerned, however, about another symptom, which he stated as a "blindness of the left eye." He could not, he said, see objects on his left without turning his head and eyes. Testing by means of a small bright object in the usual way, revealed typical left lateral hemianopsia, with a vertical division line not including the points of fixation. Central vision was as good as ever, as tested by newspaper type. Dr. C. R. Agnew was asked to see the patient the next day, and the following is a copy of his report of the condition of the patient's eyes:

"My dear Doctor: I have examined Mr. D.'s eyes. He has left hemiopia, as you say. He has opaque nerve fibres in nasal half of left optic disk, extending off a little distance into the fundus, which is physiological. He has a few punctate changes in the pigment layer of retina in *both* eyes, chiefly the left. I do not think that these things have any thing to do with the eye trouble—*that is central*, as you say. I agree with you in all you say, and have nothing to suggest in the way of topical treatment.

"Yours faithfully, C. R. Agnew."

My diagnosis at the time was embolism of a branch of the posterior central artery supplying the meso-caudal part of the right occipital lobe.

Mr. D.'s illness lasted, with most remarkable symptoms and extraordinary remissions, until May 17, 1885, when he died.

The chief features of this long sickness may be summarized as follows:

In December he had a violent attack of acute hallucinatory mania (both aural and visual hallucinations), due probably to cerebral anæmia. Under large doses of chloral, digitalis, and most persistent feeding with large quantities of milk and eggs, this subsided.

In February Mr. D. was able to go to Nassau, N. P. While there the severe chills, high fever, and sweats returned, and

proved rebellious to large doses of quinine. These chills followed no distinct type of periodicity ; they occurred twice a day, every second day or daily.

He returned to New York April 5th, and to the last, recurring febrile paroxysms usually clearly intermittent, were prominent features of the case. His general condition was better, but the heart was larger and presented an extremely loud and diffused mitral regurgitant murmur. During the month several attacks of visceral and peripheral embolism occurred, characterized by hæmaturia, splenic pain, and enlargement, a few discolored patches under the skin.

[In the preceding November, shortly after the hemianopsia, he one day complained of pain and swelling of the right palm, followed by a turgid condition of the whole hand for several days ; probably embolism of a part of the palmar arch.]

These embolisms were all recognized at the time as dependent upon the mitral disease, and it finally occurred to me that the intractable, irregular intermittent fever might also be of cardiac origin, each attack due to the detachment of microscopic particles of the diseased valves.

Dr. William H. Draper was called in consultation May 8th, and made the formal diagnosis of ulcerative or malignant endocarditis.

Previous to death, for a period of about a fortnight, the patient's speech was sometimes difficult to understand ; his articulation was defective, partly from extreme general debility, but also from some want of power in the buccal muscles. The hands both showed disorders of movements, choreiform tremors, and in the left hand slight ataxia in larger motions.

Often Mr. D. complained of numbness and coldness of the left hand.

At no time was there distinct hemiplegia or monoplegia, and repeated tests of sensibility showed it to be nearly if not quite normal, so that I was of the opinion that no emboli of any size had reached the brain since the attack in November. Several tests were made of the hemianopsia, one a few days before death. It persisted to the last unchanged, as judged by rough measurement, and vision remained good. The patient always insisted that his left eye was weak (a statement made by other hemianopsic patients). He was able to read and write easily until a few weeks before death, when increasing debility confined him to his bed.

The autopsy was made with the assistance of Dr. W. R. Birdsall, on the evening of the fatal issue, May 17th. The spleen and kidneys contained several infarcts of various ages, some very large, and looking like hemorrhagic foci.

The heart was much enlarged ; the mitral valves deformed and bearing enormous rough vegetations, one almost polypoid. Sections through some of these vegetations, stained by Gram's method, showed under the microscope globular nests of micrococci and separate colonies of bacteria. The aortic valves and aorta were normal.

The brain was generally anæmic. The basal vessels and middle cerebral arteries free from emboli and thrombi. The basal nerves, the optic tracts, and the chiasm were most carefully examined and found normal. On the right lateral aspect of the pons, caudad of the IVth nerve, a small branch of the basilar artery contained a firm thrombus of dark red color about 4 mm. in length; the vessel supplied the velum medullare anterius. The left hemisphere presented a small area of extreme congestion and ecchymosis over the folds of the second frontal gyrus; there was another patch at the foot of this gyrus extending toward the orbital gyri.

The right hemisphere had a similar superficial recent lesion (ecchymosis) at the vertex, extending over the dorsal extremity of the fissure of Rolando.

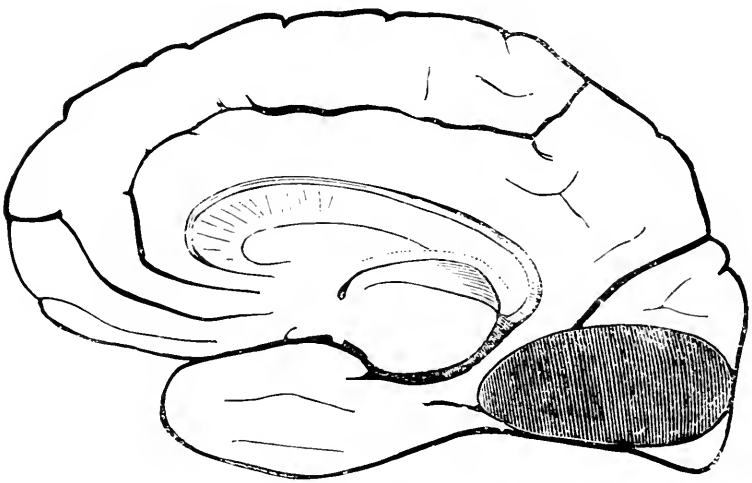


FIG. 5.—Mesal aspect of right hemisphere (Ecker). Patch of softening causing left lateral hemianopsia. Case 45 (Seguin).

Viewing the brain from above, the occipital extremity of the right hemisphere appears thinner than its homologue. This is found to be due to the destruction of the mesal surface of the right occipital lobe by a large focus of yellow softening, evidently an old patch. The lesion involves the basal part of the cuneus, the fourth and fifth temporal gyri (Ecker), and a part of the gyrus hippocampi. The destruction does not quite attain the tip of the occipital lobe.

The remaining gyri of both hemispheres were normal.

I would add that the above records of the appearance of the brain were made at the time of examination by our President, Dr. Birdsall, and me.

Most unfortunately, the brain was not cut at once. The left hemisphere was separated, leaving the "stamm" attached to the right hemisphere, and these were placed in bichromate of potassium, with the intention of making a series of sections after complete hardening.

Through mishaps the process of hardening was not very successful, and the right hemisphere particularly suffered from too prolonged pressure upon its temporal lobe, which disintegrated. The result is that I can only show you to-night the occipital half of the right hemisphere with the patch, which I consider the essential and truly causal lesion of the hemianopsia. The destruction of tissue extends only a few mm. into the subjacent white substance. The state of the internal capsule, thalami, etc., remains unknown, owing to the misfortune in preserving the specimens. From the history of the case, however, judging from the absence of hemiplegia and marked anæsthesia, it may be safely assumed, in the light of our present pathological knowledge, that there were no lesions, or at least no tangible lesions, in the central parts of the brain.

That the destruction of the right cuneus and fifth temporal gyrus was the cause of the left lateral hemianopsia during life, I have not a shadow of a doubt.

The softening was produced by embolism of the third branch of the posterior cerebral artery, the occipital artery of Duret.

The objections which may be presented against the value of my case, in consequence of its imperfect anatomical investigation, are greatly reduced in force by the consideration that the case is one in harmony with many others. Were it a contradictory or anomalous case, it would certainly possess much less value.

Now, gentlemen, what conclusion may reasonably be drawn from all these cases?

1. That lesions in the mesal aspect of the temporal lobes, or even in other basal districts of the hemispheres, may give rise to hemianopsia indirectly by pressing upon the primary optic centres or upon the tractus optici and chiasm.

2. That lesions of the corpus geniculatum laterale, pulvinar, and latero-caudal parts of the thalamus may cause hemianopsia; usually in association with hemianæsthesia and hemiplegia, or hemianæsthesia alone.

3. That a lesion of the white substance of the occipital lobe, in the caudal radiations of the internal capsule, may cause hemianopsia alone, or with hemianæsthesia.

4. That lesions of the supra-marginal gyrus, angular gyrus, and inferior parietal lobule with the subjacent white substance may cause hemianopsia—with or without other symptoms (hemiplegia, loss of muscular sense, word-deafness, etc.).

5. That a lesion of greater extent, involving the speech centre, the motor convolutions, and the parts enumerated above (4), due usually to embolism or thrombosis of the entire Sylvian artery, will, when existing on the left side, produce aphasia, alexia, hemianopsia, and hemiplegia.

6. That lesions of the occipital lobe, cortex, and subjacent white matter produce blindness when bilateral, and hemianopsia when unilateral. This conclusion is in accord with Exner's (1881).

7. That a lesion of the cuneus and adjacent 5th temporal gyrus (Ecker) on one side produces lateral hemianopsia of the opposite side.

In support of this last conclusion I would again invite your attention to the cases 28, 29, 41, and 45.

I have endeavored to fuse the diagrams of the sixteen cases with occipital lesions (exclusive of the traumatic ones) on one chart, by the successive application of layers of India ink. The larger lesions were first indicated on the outline diagram, and the most limited lesions washed last. I was somewhat hindered by the "running" of the black in the lines indicating gyri, yet I think that the maximum color due to the superposition of the greatest number of layers is over the cuneus, and next to the occipital apex, as a whole. This is a simplification of one of Exner's methods, and I think may, with some improvements, be made serviceable for clinical teaching. [The diagram was shown at the time of reading the paper.]

Let us now turn to the physiological and theoretical aspect of our subject. My time is limited, and I can only treat this highly important matter in a most summary way.

What do the most modern physiological researches teach us upon this question of the location of the cortical visual centre and its white connecting fasciculi?

The views of Munk and Ferrier are the authoritative ones.

The former physiologist has persistently taught that the visual areas, or centres for psychic vision, are in the occipital lobes, and that each visual area has connections with both retinæ. He invariably produced hemianopsia in dogs by destruction of one occipital lobe. These experimental results have been verified by Ganser, von Gudden's assistant, upon kittens.

Ferrier's theory, supported, as he believed, by experiments upon monkeys, has received an apparent verification at the hands of Prof. John C. Dalton.[1] Ferrier thought that the visual centre was in the angular gyrus. The following are his most recent conclusions, as presented to the Royal Society, and published in its Proceedings, xxxv., p. 229, and abstracted in *Brain*, April, 1884.

1. Lesions of the occipito-angular region (occipital lobes and angular gyrus) cause affections of vision without affection of the other sensory faculties or motor powers.

2. The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and angular gyri on both sides.

3. Complete extirpation of both angular gyri causes for a time total blindness, succeeded by lasting visual defect in both eyes.

4. Unilateral destruction of the cortex of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye—not of a hemiopic character.

5. Deep incisions may be made in both occipital lobes at the same time, or the greater portion of one or both occipital lobes at the same time may be removed without any appreciable impairment of vision.

6. Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopia of both eyes toward the side opposite the lesion.

7. As in none of the cases recorded, either of partial unilateral or bilateral destruction of the occipito-angular region, were the amblyopic or hemianopic symptoms permanent, it is concluded that vision is possible with both eyes if only portions of the visual centres remain intact on both sides.

[1] John C. Dalton, in *New York Medical Record*, Oct. 26, 1881.

It will be seen that the results of our pathological analysis are seemingly favorable to both the theories of Munk and of Ferrier. But, on the one hand, the most conclusive cases, *i. e.*, those with the most limited cortical lesions, are wholly opposed to Ferrier's views and in favor of Munk's; and, on the other hand, a peculiarity in the anatomy of the occipital extremity of the brain goes to explain Ferrier's results without assuming the existence of a cortical visual centre in the angular gyrus. It is this: that the optic fasciculus of Gratiolet and Wernicke, on its way from the caudo-lateral aspects of the thalamus, in the internal capsule, passing out caudad, lies latero-dorsad of the posterior horn of the lateral venticle, and close under the inferior parietal lobule and the angular gyrus, on its way to the occipital lobe (cuneus chiefly). A lesion of the angular gyrus, the supra-marginal gyrus, and even of the inferior parietal lobule, is almost certain to involve this optic fasciculus, and thus cut the communication between the visual centre and the eyes.

I pass around a specimen in which, on a horizontal longitudinal section of the human brain hardened in bichromate of potassium, the optic fasciculus is plainly visible as a homogeneous whitish band. It is evident that lesions in the angular gyrus and supra-marginal gyrus could easily penetrate deeply enough to injure this fasciculus.

It seems to me that with this anatomical knowledge the discrepancies between Ferrier and Munk's results disappear in part, and that some of the cases of my sixth category (cases 26, 32) are reconciled with the others.

Next, as to the various purely hypothetical or clinical theories of the course of the optic paths. Of these the best known is that of Prof. Charcot. His well-known diagram of the course of the optic fibres from the retina to the visual centres represents a second decussation of the fasciculi laterales through the corpora quadrigemina (lobi optici) on their way to the internal capsule, so that finally each internal capsule contains all the fibres for the opposite eye. This diagram was made to explain and support Charcot's theory of the production of amblyopia of one eye by lesion of the occipital lobe and the internal capsule of the opposite side. He

thought that he had observed that amblyopia of one eye, and not hemianopsia, was the companion of hemianæsthesia produced by lesion of the internal capsule.

I regret to say that my illustrious master's theory has not been supported by either clinical observations or by post-mortem results. I know of but one case with a post-mortem examination which is in favor of Charcot's view,[1] while the sixteen cases I have read to you speak emphatically against it. Indeed, there is reason to believe that Prof. Charcot has never attached much value to his diagram, and I understand that he has already abandoned it, yielding, as he is ever ready to do, any theoretical views of his own to opposing pathological facts.

Grasset has recently (1883) offered a modification of Charcot's diagram, which is extravagant. He would have still a third decussation (counting the chiasmic as the first) somewhere in the callosal fibres, so that after the fibres for one whole retina, according to Charcot's schema, have passed a certain distance in the internal capsule, the fibres of the fasciculus lateralis again cross the median line, so that the visual centre receives fibres from both retinæ. This far-fetched attempt to reconcile Charcot's opinion as to the effect of lesion of one internal capsule in its caudal division, with the well-established results of lesions of the occipital lobes, is hardly deserving of serious criticism ; but it may be as well to state that more recent (1884)[2] experiments by W. Bechterew show that in dogs at least section of the posterior part of the internal capsule produces lateral hemianopsia—a result in full harmony with some of our human cases.

From his latest pathological observations von Monakow[3] draws the following conclusions as to the course of the central optic fasciculi in man :

"The collective optical bundle forms a solid tractus in the sagittal white substance of the occipital part of the brain, which passes alongside of the corpus callosum fibres or tapetum, and ends in the cortex of the occipital gyri,

[1] Petrina, in *Prager Zeitsch. f. Heilk.*, II., p. 595, case viii. *Vide* Table I.

[2] W. Bechterew : Ueber die nach Durchschneidung der Sehnervenfasern im innere der Grosshirnhemisphären, etc. *Neurol. Centralbl.*, 1884, No. 1.

[3] *Westphal's Archiv f. Psychiatrie*, xvi., 352.

more especially in that of the cuneus, lobus lingualis, and gyrus descendens."

The diagram of optic paths which I offer you is, I believe, in agreement with Munk's view of the physiology of the visual centre, with what we know of the anatomy of the optic tracts by dissection and by secondary degeneration (Monakow), and lastly, best of all, with the results of now numerous post-mortem examinations.

From the above data, pathological, anatomical, and experimental, are we now in a position to induce diagnostic laws with reference to the symptom hemianopsia? I think we are, and I would propose the following as a preliminary set of rules.

1. Lateral hemianopsia always indicates an intra-cranial lesion on the opposite side from the dark fields.

2. Lateral hemianopsia with pupillary immobility, optic neuritis or atrophy, especially if joined with symptoms of basal disease, is due to lesion of one optic tract, or of the primary optic centres on one side.

This diagnosis may be further strengthened and rendered quite certain by seeking for and finding one-sided pupillary reaction, as recently suggested by Wernicke.[1] He ingeniously predicts that only one lateral half of each iris will be found to contract by the reflex effect of light when one optic tract has been interrupted. He designates this as "hemiopic pupillary reaction."

3. Lateral hemianopsia, or sector-like defects of the same geometric order, with hemianæsthesia and choreiform or ataxic movements of one half of the body without marked hemiplegia, is probably due to lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule.

4. Lateral hemianopsia, with complete hemiplegia (spastic after a few weeks) and hemianæsthesia, is probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

5. Lateral hemianopsia, with typical hemiplegia (spastic

[1] Wernicke: Ueber hemiopische Pupillenreaction. *Fortschritte der Medicin*, 1883, i., 49–53.

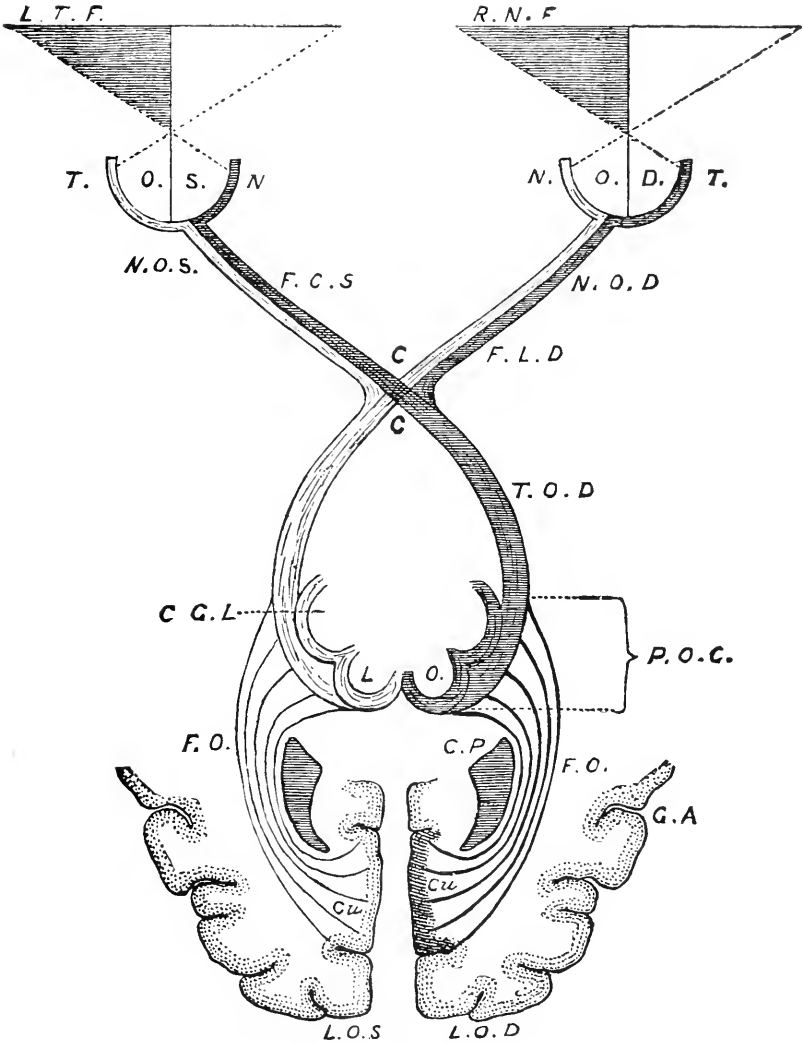


FIG. 6.—Diagram of Visual Paths ; designed to illustrate specially Left Lateral Hemianopsia from any lesion. L. T. F., left temporal half-field. R. N. F., right nasal half-field. O. S., oculus sin. O. D., oculus dexter. N. T., nasal and temporal halves of retinæ. N. O. S., nervus opticus sin. N. O. D., nervus opticus dext. F. C. S., fasciculus cruciatus sin. F. L. D., fasciculus lateralis dext. C., chiasma, or decussation of fasciculi cruciati. T. O. D., tractus opticus dext. C. G. L., corpus geniculatum laterale. L. O., lobi optici (corpus quad.). P. O. C., primary optic centres, including lobus opticus corp. genic. lat., and pulvinar of one side. F. O., fasciculus opticus (Gratiolet) in the internal capsule. C. P., cornu posterior. G. A., region of gyrus angularis. L. O. S., lobus occip. sin. L. O. D., lobus occip. dext. Cu., cuneus and subjacent gyri constituting the cortical visual centre in man. *The heavy or shaded lines represent parts connected with the right halves of both retinæ. The reader may place the lesion as he pleases.*

after a few weeks), aphasia if the right side be paralyzed, and with little or no anæsthesia, is quite certainly due to an extensive superficial lesion in the area supplied by the middle cerebral artery; we would expect to find (as in case 26, by Westphal) softening of the motor zone and of the gyri lying at the extremity of the fissure of Sylvius, viz.: the inferior parietal lobule, the supra-marginal gyrus, and the gyrus angularis. Embolism or thrombosis of the Sylvian artery would be the most likely pathological cause of the softening.

6. Lateral hemianopsia with moderate loss of power in one half of the body, especially if associated with impairment of muscular sense, would probably be due to a lesion of the inferior parietal lobule and gyrus angularis, with their subjacent white substance, penetrating deeply enough to sever or compress the optic fasciculus on its way caudad to the visual centre.

7. Lateral hemianopsia without motor, or common sensory symptoms, this symptom alone, is due, I believe, from the convincing evidence afforded by Cases 28, 29, 41, and 45, to lesion of the cuneus only, or of it and the gray matter immediately surrounding it on the mesal surface of the occipital lobe, in the hemisphere opposite to the dark half-fields. Most surgical cases come at once, or after convalescence, within this rule or in No 6 (Case 3.)

In all cases coming under rules 3 to 7 inclusive, the pupils react normally; and rarely does the ophthalmoscope show any lesion of the optic nerve, except, of course, in some tumor cases, when neuro-retinitis may be expected.

A LIST OF CASES OF HEMIANOPSIA OF CENTRAL ORIGIN, WITH AUTOPSY, AND OF TRAUMATIC HEMIANOPSIA, TO OCTOBER 30, 1885, ARRANGED IN CHRONOLOGICAL ORDER.

1, 1863. CHAILLOU. Société anatomique, Feb., 1863. *Bull. de la Soc. Anat.*, 1863, p. 71.

2, 1866. LEVICK. Case of abscess of the brain. Trans. of the College of Physicians of Phila., March 7, 1866. *Am. Journal Med. Science*, 1866, ii., p. 413.

3, 1871. W. W. KEEN and WM. THOMSON. Gunshot wound of the brain, followed by fungus cerebri, and recovery with hemiopia. *Trans. Am. Ophthal. Soc.*, 1871, p. 122. Also in "Medical and Surgical History of the War of the Rebellion," i., p. 206.

4, 1873. HUGHES, J. S. Case of compound depressed fracture of the skull. *Irish Hospital Gazette*, July 1, 1873 (abst. in *Nagel's Jahresbericht über Ophthal.*, 1874, p. 440).

5, 1875. HIRSCHBERG, J. Zur Semidecussation der Sehnervenfasern in Chiasma des Menschen. *Virchow's Archiv*, lxv., p. 116.

6, 1875. JACKSON, J. H., and GOWERS, W. R. I. A case of hemiopia, with hemianæsthesia and hemiplegia, by Dr. Jackson. *Lancet*, 1874. ii., 306. II. Autopsy on a case of hemiopia, etc., by Dr. Gowers. *Lancet*, 1875, i., 722.

7, 1876. FÖRSTER and WERNICKE. Förster in "Graefe-Saemisch Handbuch der gesammten Augenheilkunde," Bd. vii, p. 118. Also in Wernicke's "Die aphasische Symptomcomplex," p. 47. Berlin.

8, 1876. HUGUENIN. Article "Chronic Abscess of the Brain," in Am. ed. of *Ziemssen's Cyclop.*, xii., 796.

9, 1877. POOLEY, T. R. Right-sided binocular hemiopia, depending upon a gummy tumor in the left posterior lobe of the brain. *Knapp's Archives of Ophthal.*, v., p. 148; ix., p. 83.

10, 1877. JASTROWITZ, M. Tumor im linken Hinterlappen, Aphasie, rechtsseitige Hemianopsie. *Hirschberg's Centralbl. f. pract. Augenheilkunde*, 1877, p. 254.

11, 1877. JASTROWITZ. Case referred to briefly in his paper in *Hirschberg's Centralbl. f. pract. Augenheilk.*, i., p. 256.

12, 1877. CHARCOT et PITRES. Contributions à l'étude des localizations dans L'écorce des hémisphères cérébraux. *Revue mensuelle de méd. et de chir.*, i., p. 372, obs. xxxiv. (1877).

13, 1878. BAUMGARTEN, PAUL. Hemiopie nach Erkrankung der occipitalen Hirnrinde. *Centralbl. f. Med. Wissensch.*, 1878, No. 21.

14, 1878. HOSCH, FR. Zur Lehre von der Sehnervenkreuzung. *Zehender's Monatsblätter f. Augenheilk.*, 1878, p. 281.

15, 1879. DMITROWSKY and LEBEDEN. Hemianopsia dextra. Monoplegia facialis dextra. Hæmorrhagia und Erweichung in der linken Hirnhalbkugel. *Medicin. Bote*, 1879, No. 46. (Abst. in *Hirschberg's Centralbl.*, 1880, p. 84.)

16, 1879. PFLÜGER. Augenklinik in Bern. *Bericht uber das Jahr* 1878, p. 57.

17, 1879. NOTHNAGEL. Topische Diagnostik der Gehirnkrankheiten. Berlin, 1879, p. 389.

18, 1879. CURSCHMANN. In Berliner Gesellschaft f. Psych. und Nervenkrankheiten, Juni 9, 1879. *Westphal's Archiv*, xi., 822.

19, 1880. DRESCHFELD. Pathologische-anatomische Beiträge zur Lehre von der Semidecussation der Sehnervenfasern. Case ii. *Hirschberg's Centralbl. f. pract. Augenheilkunde*, 1880, p. 35.

20, 1880. SCHMIDT-RIMPLER. Sitzb. der ärztl. Verein zu Marburg, Juni 9, 1880 (abst. in *Hirschberg's Centralbl.*, 1881, p. 358).

21, 1881. LINNELL, E. H. A case of left-sided binocular hemianopsia, with report of autopsy and microscopical examination. *Knapp's Archives of Ophthal.*, x. (1881), 4, p. 446.

22, 1881. WESTPHAL, C. Zur Frage von der Localization der unilaterale Convulsionen und Hemianopsie. *Charité-Annalen*, vi., 1881, p. 342.

23, 1881. HEUSE. Hemianopsie bei Schädelverletzung. Case ii. *Hirschberg's Centralbl. f. pract. Heilkunde*, 1881, p. 205.

24, 1881. SENATOR, H., in WERNICKE'S Lehrbuch der Gehirnkrankheiten, ii., p. 70, 1881.

25, 1881. PETRINA. Ueber Sensibilitätsstörungen bei Hirnrindenlesionen. Case viii. *Zeitschr. f. Heilkunde*, Prag, 1881, ii., 375.

26, 1882. WESTPHAL, C. Zur Localization der Hemianopsie und des Muskelgefühls beim Menschen. *Charité-Annalen*, Bd. vii., 1882, p. 466.

27, 1882. MARCHAND. Beitrag zu Kenntniss der homonymen bilateralen Hemianopsie, und der Faserkreuzung in Chiasma opticum. *Archiv f. Ophthal.*, xxviii., 2, 63. Case iii.

28, 1882. HAAB, O. Ueber Cortex-Hemiopie. *Klinische Monatsblätter f. Augenheilkunde*, xx., 141, 1882.

29, 1882. HUGUENIN. Case in Haab's article.

30, 1882. DRESCHFELD, J. Pathological contributions to the course of the optic nerve fibres in the brain. *Brain*, iv., p. 543, Jan., 1882. Case i.

31, 1882. *Idem*. In *idem*. Case ii.

32, 1882. STENGER, C. Die cerebralen Sehstörungen der Paralytiker. *Arch. f. Psych.*, xiii., p. 242. Case vii.

33, 1882. *Idem*. In *idem*, p. 246. Case viii.

34, 1882. WERNICKE and HAHN. Idiopathischer Abscess des Occipitallappens, durch Trepanation entleert. *Virchow's Archiv*, lxxxvii., 335.

35, 1883. JANY, L. Ein Fall von rechtsseitiges Hemianopsie und Neuro-retinitis in Folge eines Gliosarcoms im linken occipitallappen. *Knapp's Archiv f. Ophthal.*, xii., p. 327.

36, 1883. ROSENBACH, P. Zur Casuistik der Hemianopsie. *St. Petersburg Med. Wochenschr.*, 1883, No. 12. *Neurolog. Centralbl.*, 1883, p. 442.

37, 1883. NIEDEN, A. Ein Fall von einseitger temporaler Hemianopsie des rechten Auges nach Trepanation des linken Hinterhauptbeins. *Archiv f. Ophthal.*, xxix., iii., p. 143.

38, 1883. RICHTER. Fall von dreimaliger Blutung in ein Gehirn. Gesell. f. Psych. u. Nervenkrankheiten, 11 Juni, 1883. *Neurolog. Centralbl.*, 1883, p. 307.

39, 1883. SCHMALTZ. Case given by A. Vetter; Ueber die sensorielle Function des Grosshirns, etc. *Deutsch. Archiv f. kl. Medicin*, xxxii., p. 469, 1883.

40, 1884. WIETHE, TH. A case of superior homonymous hemianopia. *Knapp's Archives of Ophthal.*, xiii., 3, 301.

41, 1885. FÉRÉ, CH. Trois autopsies pour servir à la localisation cérébrale des troubles de la vision. Case iii (from Charcot's service). *Archives de Neurologie*, Mars, 1883, p. 229.

42, 1885. RICHTER, A. Zur Frage der optischen Leitungsbahnen des menschlichen Gehirns. *Archiv f. Psych. und Nervenkrankheiten*, xvi., p. 641.

43, 1885. *Idem*. Ueber die optischen Leitungsbahnen des menschlichen Gehirns. Fall ii. *Allg. Zeitschr. f. Psych.*, xli., 1885, p 637.

44, 1885. *Idem*. In *idem*. Fall iii., p. 638.

45, 1885. SEGUIN, E. C. A contribution to the pathology of hemianopsia of central origin. Case with specimen. Read before the New York Neurological Society, October 6, 1885.

Clinical Cases.

A CASE OF TREPHINING FOR TRAUMATIC EPILEPSY.*

FRACTURE OF THE SKULL FROM A PISTOL-SHOT—CONVULSIONS DUE TO IRRITATION OF THE DURA MATER, FROM WHICH FRAGMENTS OF BONE WERE REMOVED BY OPERATION.

By CHARLES K. MILLS, M.D.,

NEUROLOGIST TO THE PHILADELPHIA HOSPITAL, ETC.,

AND

J. WILLIAM WHITE, M.D.,

SURGEON TO THE PHILADELPHIA HOSPITAL, ETC.

The patient, J. M., aged 26, white, a farmer, was brought to the service for Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College for Graduates in Medicine. After careful examination, it was determined that the case was one in which trephining was not only justifiable, but, both from a neurological and surgical point of view, imperatively demanded. He was therefore sent to this hospital. One of the resident physicians, Dr. Roberts, has carefully prepared a history of the case.

The patient knows nothing of his family history. He was in an orphan asylum until 14 years old, and was then bound out to a farmer in Delaware, and has been employed at farm labor ever since.

He smokes, chews, and drinks ; has been drinking since he was 18 years of age. He denies venereal history. He had the smallpox when a child, but has always been strong and healthy since, and never had any sickness of any kind. He never had epileptic fits before last October.

* Abstract of clinical lecture and report of operation. Lecture delivered and operation performed before the class in the amphitheatre of the Philadelphia Hospital, Jan. 27, 1886.

On the morning of July 4, 1885, while playing cards and drinking with some companions near the railroad track not far from Greenwood, Delaware, a dispute arose, and one of the party drew a revolver and shot the patient through the head. The two men were close together when the shot was fired—the patient in a stooping position over the man with the revolver. The pistol was self-cocking, and of 32 calibre. It was so close to his face that the powder burnt his cheeks, eyes, and forehead very badly. He fell at the report, but did not realize that he had been shot until he put his hand to his head and felt the blood. He was unable to walk or help himself, but did not lose consciousness. He was carried to the town, and conveyed to his home in a carriage, and a physician called. For two months he was confined to his bed on account of a left-sided hemiplegia. For three or four weeks he was unable to see on account of the swelling caused by the powder-burn. At the end of the two months he got up and could walk about, but was unable to work.

From the scar of the wound and the patient's account, it is evident that the ball entered the scalp just above the right temple. He has been told that the surgeon introduced a probe "three and a half inches and brought out some of the brain-substance on the end of it."

The patient thinks that the ball entered from the front, and, glancing off from the skull, passed out again. The facts would support this view. He wore a Derby hat, and *two* holes were found in it—in just the right positions to have been made by the ball entering and passing out.

As stated, the patient was confined to his bed, partially paralyzed, during July and August. Toward the latter part of September he returned to work again, doing light work about the farms. One morning, in the latter part of September, he was told by a man with whom he had slept the night previous, that he had had a fit during the night. He knew nothing of it himself, and did not believe that he had had a fit. He was told that "he kicked, chattered, and ground his teeth, and could not be aroused."

About three weeks after this he had a second fit, which occurred while he was at the supper-table. He was unconscious, and knew nothing until it was all over—a period, he was told, of several minutes. From the first attack until the present time—about four months—he has had seven fits in all.

Just before entering the clinic room on the day of operation the patient had a convulsion. This was the only convulsion he had had since admission to the hospital.

His gait, as he passed around the arena, showed but little of the left-sided hemiplegic condition remaining. A scarcely perceptible halting or dragging of the left foot was all that indicated any difference in power between the right and left sides; the grip of his left hand and the movements of the entire upper extremity showed nothing peculiar. Neither of the limbs was atrophied;

his face was not drawn in the slightest, and, indeed, had not at any time been so affected.

Examining his head, which had been shaved, *two* scars were found close together in the scalp, and an irregular fracture was felt spreading over a space of about one square inch at a position about two inches to the right of the median line, and one half to one inch in front of the coronal suture. There seemed to be a small depression, surrounded by a slight elevation of the skull, in this locality. The position of these scars and of the fracture beneath, in the frontal bone, corresponded to a point about the junction of the middle portion of the first frontal with the second frontal convolution. The middle of the fracture was at a point about two inches in front of an imaginary line corresponding to the fissure of Rolando. The whole area of fracture was certainly well in advance of the ascending frontal convolution and the entire so-called cortical motor zone.

In the convulsion witnessed just prior to the clinic hour he was completely unconscious ; his face and all his limbs, as well as his head and trunk, took part in the spasm ; the eyes were distorted ; his whole body was twisted somewhat to the right ; the spasms were tetanic in character ; the limbs were in conditions of tonic spasm in extension. When moved, they were moved as a whole, the rigidity remaining. The spasms were not localized ; they were not confined to one limb or to one side of the body.

For several reasons these spasmodic seizures were probably due to irritation of the dura mater, what might be termed dural or meningeal spasms. They were not due to irritation of the cortical motor zone, the form of spasm sometimes spoken of as Jacksonian epilepsy. The scalp wound and fracture, in the first place, were in a position well in front of this motor or Rolandic region. Lesions of the motor cortex cause disorders on the opposite side of the body ; irritative lesions cause monospasm or unilateral spasm of the opposite side. These spasms usually show themselves in a definite manner both as to localization and mode of action.

The researches of H. Duret on cerebral traumatisms—investigations both physiological and pathological—have thrown considerable light on the rôle which the dura mater and its nerves play in these cases. Irritation of those branches of the trigeminal or fifth pair of nerves which go to the dura mater produces, in addition to hyperæsthesia, pain, and vaso-motor disturbances, reflex spasms of peculiar character. Strong, powerful stimulation of the dura mater, continued and repeated, was shown in certain physiological experiments of Bochefontaine to determine movem nts in both sides of the face, in the neck, and in all four limbs. The movements of the limbs on the same side were more energetic than those on the opposite side. (Quoted by Duret in his work, "Sur les Traumatismes Cérbéraux," and also in *Brain*, vol. i., 1878.)

Certainly the phenomena of spasm presented by this case may

be said to correspond closely to those described as the result of dural irritation. The subject of cerebral localization and the operative surgery of the human brain are now fashionable ones with the profession. It is highly important that cases such as the one before us should be thoroughly understood; so understood that they can be differentiated from spasms due to cortical irritation.

The hemiplegia which was present and has disappeared may have been due to a meningeal hemorrhage, which has been largely absorbed.

The operation was performed by Dr. White.

The patient having been thoroughly etherized, a horse-shoe flap of large size was made by Dr. White. No adhesions or extravasations were found beneath the scalp. The skull was found to be fractured in the position already described; the entire area fractured could be covered by the trephine used by Dr. White, which was one of the largest size. The anterior portion of the trephine very quickly made its way through the skull, and subsequently it was found that at that point the inner table was partially separated from the outer. The button, irregular in shape and thickness on account of the old splintering of the inner table, was removed with great care and without wounding the dura mater, and it was then found that the portion remaining was adherent to that membrane. By careful dissection this fragment of fractured bone was finally removed. Two small spicules were picked out of the wound with forceps, and an irregular, jagged mass of bone about half an inch in its great length was found imbedded in the dura mater, one of its points passing through it into the brain substance.

The most rigid antiseptic precautions were used.

The operation was performed between 11 and 12 o'clock January 27th. At 4.30 P.M. his pulse was 108; temperature 100.4°; at 6 P.M. the pulse 90; temperature, 101°F. He was ordered powders of calomel and opium, of each grain $\frac{1}{20}$, every two hours. He had slight nausea. At 10.30, 15 grains of bromide of potassium were administered.

January 28th.—At 6.30 A.M., pulse, 82; temperature 100°. At noon his temperature had fallen to 99°. Milk was given. At 6 P.M., pulse, 80; temperature, 99.4°. Fifteen grains of bromide of potassium were given.

January 29th.—At 6 A.M., pulse, 80; temperature, 98.8°. At 6 P.M., pulse, 76; temperature, 99.2°. At 10 P.M., temperature, 99.4°. Nourishment in the form of milk and beef tea was given; the calomel and opium powders were continued; an enema was administered with good result after third trial. The wound was dressed—Lister dressing-spray.

January 30th.—At 6 A.M., pulse, 74; temperature, 99°. At 6 P.M., temperature, 99.4°.

January 31st.—At 6 A.M., pulse, 76; temperature, 99°. At 6 P.M., pulse, 56; temperature, 98.8°. Lister dressing-spray.

February 1st.—At 6 A.M., pulse, 60; temperature, 98°. At

6 P.M., pulse, 64 ; temperature, 98°. At 10 P.M. the temperature had fallen to 97.6°. Milk and beef tea were continued ; milk toast was given ; calomel and opium were continued.

February 2d.—At 6 A.M., pulse, 52 ; temperature, 97.6°. At 2 P.M. the temperature had risen to 98.8°; it fell again, and at 6 P.M., the record was pulse, 60 ; temperature, 98°.

February 3d.—At 2 A.M. and 6 A.M., pulse, 54 ; temperature, 97.4°. At 6 P.M. pulse, 60 ; temperature, 98.4°.

February 4th.—At 6 A.M., pulse, 60 ; temperature, 98°. At 6 P.M., pulse, 56 ; temperature, 98.8°.

February 5th.—At 6 A.M., pulse, 64 ; temperature, 97.8°. At 6 P.M., pulse, 64 ; temperature, 98.6°.

February 6th.—At 6 A.M., pulse, 66 ; temperature, 97.8°. At 10 A.M., pulse, 90 ; temperature, 98.2°. At 6 P.M., pulse, 64 ; temperature, 99°. At 10 P.M., temperature, 97.6°.

February 7th.—At 6 A.M., pulse, 64 ; temperature, 98.2°.

Milk, beef tea, calomel, and opium, and enemata were used. The patient has at times complained of trifling pain in the head, but ever since reacting from the operation has been entirely rational and in good general condition.

February 10th.—Two weeks after the operation the patient is in every way doing well.

February 11th.—Pulse and temperature were normal and no bad symptoms whatever were present.

In a subsequent number of the JOURNAL the further history of the case will be given.

Periscope.

a.—ANATOMY.

The Comparative Anatomy of the Pyramid Tract. By SPITZKA, *Journal of Comparative Medicine and Surgery*, Jan., 1886.

This very important article should be read by all who are interested in the anatomy of the nervous system, as it contains many new and suggestive conclusions and suffers by any attempt at condensation.

1. The presence of symmetrical columnar eminences on either side of the ventral sulcus or fissure of the oblongata is a characteristic feature of the mammalia as a group. Three different anatomical elements may, singly or in combination, produce these elevations: first, the true pyramids—man, carnivores, rodents, bats; second, the interolivary layer—elephant; third, the olives—porpoise.

2. In probably all edentates these columns are so faintly marked as to be scarcely identifiable—(sloth, armadillo).

3. In the elephant and porpoise the pyramids are demonstrably absent, as an expression probably of the physiological characters of these animals.

4. Among the remaining mammalia two different types of olivary development are found, which closely follow the zoölogical subdivisions. In the first type—primates, chiroptera, carnivora, rodentia—the pyramids are bulky, and extend as coarsely demonstrable bundles the entire length of the oblongata, and decussate as coarse bundles into the opposite half of the cord. In the second type—ungulata—they are small in proportion to the brain, do not extend as coarsely demonstrable bundles the entire length of the oblongata, and being apparently exhausted in supplying the cranial nerve nuclei, do not decussate as bundles into the opposite half of the cord.

5. With those animals having typical pyramids the cerebral portion of these tracts has the following constant characters: *a*, it is connected with the so-called motor fields of the cerebral hemispheres; *b*, runs behind the knee of the int. capsule; *c*, forms a part of the pes pedunculi; *d*, pierces the transverse fibre mass in

the caudal half of the pons ; *e*, is separated by an appreciable interval from the interolivary layer while in the pons ; *f*, runs ventrad of the trapezium ; *g*, runs ventro-mesad of the true olives ; *h*, decussates grossly into the opposite half of the spinal cord.

6. The decussation of the pyramids exhibits three different types.

A. The common type, the great mass of each pyramid passing into the opposite lateral column of the cord—primates, carnivores, some rodents.

B. The great mass of the pyramids passes into the opposite posterior column—muridæ, cavia (less pronounced).

C. The pyramid decussates on the surface of the brain, and passes into the lateral field of the oblongata—frugivorous bats.

7. Among animals presenting type A the pyramids are an index of the preponderating influence of the higher centres, and this is made manifest by two factors : first, the relative area of the trans-section of the pyramid ; second, the crowding of the olives from a deep mesal situation to a more lateral and superficial one. 1st. The relative area of the pryamid tract in the oblongata increases in the order of intelligence, but this is true only for animals of the first type. 2d. The prominence of the olive becomes more marked as we pass up in the scale of pyramid development.

8. Among the same group of animals there are instances where the pyramids are larger with bulkier species : thus they are proportionately greater in the lion than in the leopard, and in the leopard than in the cat ; larger in the bear than in the cratimundi ; in the baboon than in the cebus. This fact seems to be in relation to the greater preponderance of the isthmus as a whole, in larger species.

9. There is no parallelism in the mammalian series between the development of the transverse fibre mass of the pons and the pyramids. The elephant has the largest transverse fibre mass in the series, but it has no pyramids.

10. There is no parallelism between the development of the pyramids and the degree to which the trapezium is concealed by the pons. This concealment is complete in the elephant, and partially so in the porpoise, but neither of those animals has a pyramid.

11. There is no parallelism between the development of the olives and the pyramid. The former are fairly developed in the above species, though the pyramids are absent. But in animals of the same type (first type, class A) there is an approach to such parallelism, in that the V-shaped nucleus dentatus is larger with larger pyramids, and becomes convoluted with the highest forms.

12. Animals with a well-developed pyramid have a wider basilar raphé than those without such ; the extreme type of the latter (porpoise) having practically none.

13. It is due to the atrophy and disappearance of the pyramid

tract in the series ungulata—cetacea proboscidea—that the transverse axis of the olivary body becomes horizontal, so that the relation of the three olivary sub-nuclei which in man is that of ectal, main, and mesal, is in these animals dorsal, main, and ventral. To this general rule the elephant offers an exception owing to the vicarious development of the outer olivary layer in the place typically occupied by the pyramid. In all ungulata the type of the olive is the same.

14. The true pyramid always courses, in greater part, ventrad of the trapezium, the interolivary layer always passes through or over the trapezium, and where the trapezium is defective, as in man, in the homologous situation, which can be easily identified.

The function of the pyramid tract as deduced from comparative anatomy and physiology, is to control digital motor actions, especially of a prehensile kind. As this motion becomes more elaborate, and more subjected to higher intellectual needs, the pyramid tract is relatively enlarged. It is not present in elephant and dolphin, where it is not needed.

S. is inclined to believe that there is a closer phylogenetic relation between the pyramid tract and the posterior columns than at first sight seems plausible. The same tract may pass into the posterior columns, or into the crossed pyramidal tract, the former in the muridal, the latter in the typical forms. Even in man fibre admixtures between the interolivary tract, which goes to the post. columns, and the pyramid occur. It may be that there are transition forms between the two extremes ; and that relations between these tracts, partly of mutual support and parallel development, partly of vicarious character, are to be established.

Other conclusions reached are that the corpora geniculata externa are parallel in their development to the visual organs and tracts ; and that the corpora geniculata interna are parallel in their development to the auditory organ and tracts. Also that the column of Goll in the cord is parallel in its development with the development of the hind limbs.

The article is full of interest to the anatomist or physiologist, and merits careful study.

On Prof. Hamilton's Theory Concerning the Corpus Callosum. By C. BEEVOR, *Brain*, Oct., 1885.

The assertions of Hamilton, of Aberdeen, regarding the course of the fibres in the corpus callosum, were reported in the last number of this JOURNAL. It will be remembered that Hamilton denies that the function of this mass of white matter is to connect similar parts of the two hemispheres, and throws doubts on its commissural nature.

Spitzka has opposed this view from the study of the development of fibres in the brains of two infants, and supports the position taken by Flechsig, that the manner of development of the callosal tracts proves them to pass between symmetrical struc-

tures (*The American Journal of Neurology*, iii., 620). He also cites the well-known facts of hemiplegia in contradiction of the Scotch pathologist's position, that tracts pass from one hemisphere into the capsules of the other hemisphere.

Beevor has combated the new theory also, on the grounds of embryology. His statements are definitely opposed to those of Hamilton, and in support of the commissural view of the corpus callosum. The observations of Flechsig, Spitzka, and Beevor, are therefore in harmony, and as it is chiefly on embryological grounds that Hamilton's theory is based, the question becomes one of fact, and appears to be open to but one solution. Further careful researches will therefore succeed in deciding it definitely.

There can he no question that certain physiological acts, such as the ease of symmetrical bilateral movements, demand as an anatomical basis commissural tracts in the brain, whose function is to harmonize bilateral action. If they do not lie in the corpus callosum, they must exist elsewhere, and we know of no other structures in which they can pass. It seems as if physiology supported the older view; and as Hamilton's facts are disputed, it cannot yet be discarded. M. A. STARR.

The Structure of the Pineal Gland. By DR. L. DARKSCHEWITSCH, *Neurol. Centralbl.* (Mendel), No. 2, 1886.

A number of authors, among them Schwalbe and Edinger, have denied the ganglionic structure of this body. The author of the above paper supports the views of Meynert and Hagemann, and has succeeded in demonstrating, with the aid of Weigert's hæmatoxylin staining method, the existence of distinct sets of fibres in this "gland." These fibres can be traced to the internal capsule, the *striæ medullares*, to the fasciculus retroflexus (Meynert), the tractus opticus, or to the posterior cerebral commissure. D. is inclined to attach special importance to the last-named set of fibres, in consequence of the intimate relation existing between the posterior commissure and the oculo-motor nerves. His conclusions are based upon a study of the pineal gland in the frog, monkey, dog, ape, and in the human fœtus. B. S.

---

b.—PHYSIOLOGY.

The Heat-Centre in the Brain. By ARONSOHN and J. SACHS, *Pflüger's Arch.*, vol. xxxvii., 5 and 6, Oct. 29, 1885.

Landois has shown by sections in the internal capsule that effects on the temperature of the body can be produced analogous to those shown to take place in the cortex cerebri by Eulenburg and Landois. The vaso-motor tracts run from the cortex to the fasciculi of the corona radiata, to the internal capsule, and then into the peduncu-lus and pons where vaso-motor fibres have been long known to ex-

ist. The increase of temperature here is entirely vaso-motor—that is, due to lessened heat-dissipation. Richet has lately put forth the claim of priority of having discovered a heat-centre in the brain. He made punctures in the cortex cerebri, and claims to have produced an increase of temperature which lasted for a long time, several days. Neither Ott, nor Sachs and Aronsohn in rabbits were able to obtain any increase by puncturing the cortex, except a slight one which was fugitive. Richet also claims priority for having shown calometrically that this increase was due to increased heat-production. His claims will have to be verified before they are admitted into the dispute of priority. Ott has shown that in the neighborhood of the corpus striatum are heat centres, and that these centres are more definitely located at the anterior inner end of the optic thalami, for the increase of temperature (7° F. in an hour) was greatest at this point. He also has shown by the calorimeter that this increase of temperature or artificial fever is due to increased heat-production.

Some months after these announcements Sachs and Aronsohn show that in the nucleus caudatus and in the tissues beneath the corpus striatum—that is, about the corpus striatum, are heat-centres. They also announce at the same time that estimations of the amount of oxygen consumed and of the carbonic acid given off, as well as the nitrogen excreted before and after the puncture in the brain show that the increase of heat is due to increased heat-production. *Cf.* also Eulenburg: Ueber das waerme centrum im Grosshirn—*Centralblatt für klinische Medicin*, Verhandlung der Physiolog. Gesellschaft zu Berlin, 1884–1885, No. 16. Richet: *Pflüger's Arch.*, Band xxxvii. Heft 11 and 12. Ott: *Philadelphia Medical Times*, July 4, 1885.

The Time it takes to See and Name Objects. By J. M. CATTELL, *Mind*, January, 1866.

Mr. J. M. Cattell has made a series of experiments upon this subject. He pasted letters on a revolving drum (a physiological kymograph) and determined at what rate they could be read aloud as they passed by a slit in a screen. It was found that the time varied with the width of the slit. When the slit was one centimetre wide (the letters being one cm. apart), one letter was always in view. As the first disappeared the second took its place, etc. In this case it took nine persons experimented on from $\frac{1}{3}$ to $\frac{1}{5}$ second to read each letter. We found it took twice as long to read (aloud as fast as possible) words which have no connection as letters which make words. As the result of a large number of experiments the writer found that he had read words not making sentences at the rate of $\frac{1}{4}$ second; words making sentences at the rate of $\frac{1}{8}$ second per word. Letters not making words were read in $\frac{1}{4\frac{1}{0}}$ sec. less time than words not making sentences. The rate at which a person reads a foreign language is proportional to his familiarity with the language. The subject does not know that he is reading the foreign

language more slowly than his own ; this explains why foreigners seem to talk so fast. The time required to see and name colors and pictures of objects was determined in the same way. The time was found to be about the same (over $\frac{1}{2}$ sec.,) for colors as for pictures, and about twice as long for words and letters. Other experiments I have made show that we can recognize a single color or picture in a slightly shorter time than a word or letter, but take longer to name it. This is, because in the case of words and letters the association between the idea and the name has taken place so often that the process has become automatic, whereas in the case of colors and pictures we must, by a voluntary effort, choose the name. Such experiments would be useful in investigating aphasia. ISAAC OTT.

Nouvelles Expériences á propos de la Locomobilité Intracranienne du Cerveau. By M. G. LUYS, *Gazette des Hôpitaux*, p. 1179, 1885.

In the communication made to the French Academy of Medicine in 1884, Luys endeavored to prove, by means of experiments upon the cadaver, that the cerebral mass being smaller than the cranial cavity, and the space between brain and bony covering thus formed being filled with fluid, the brain could accomplish certain sliding movements, analogous to the movements of the fœtus in the amniotic fluid. Change therefore in the position of the head, would produce a corresponding displacement of the brain itself. In lying upon the back, the brain loses its point of contact with the cranium in front ; it is similar if the head rests upon the frontal region, then the occipital parts of the brain leave the cranium behind. Thus movement of the head will influence the position of the brain in the cranium. These movements of the brain explain the physiological action of the arachnoid. This is a serous membrane and must have a similar function to serous membranes in other parts of the body. Its function is to facilitate these movements. One objection made to Luys' views was that, as he had performed his experiments upon heads, whose bony covering had been subjected to traumatism, thus producing conditions anomalous to those normally existing, his experiments could not be utilized for the purpose of proving the correctness of his views.

L. has now endeavored to meet this objection by freezing the entire head, and then making sections of the skull and brain together, the body during the freezing process having been kept either in the horizontal or vertical positions. The sections were then made either horizontally or vertically, and tracings made through a plate of unpolished glass. The first tracing shows a horizontal section of the skull and brain of a subject placed in the horizontal position. The second, shows a vertical section, in a vertically placed subject. The results were the same as in the former experiments. The space left by the movement of the

brain, between it and the cranium, corresponded in all cases to the position of the subject, and in these spaces was found congealed fluid. Thus the proof of the movements of the brain is furnished, and at the same time, the physiological action of the arachnoid is explained. GEO. W. JACOBY.

---

c.—GENERAL PATHOLOGY.

Ueber die Spastische Cerebral Paralyse im Kindesalter (Hemiplegia Spast. Infantilis), nebst einem Excurse ueber "Aphasie bei Kindern." By PROF. M. BERNHARDT, of Berlin. *Virchow's Archiv*, p. 26, 1885.

In this article Prof. Bernhardt treats at some length of infantile spastic hemiplegia. His paper is based upon eighteen cases, of which fourteen were cases of right hemiplegia, and four of left hemiplegia. As aphasia is a prominent symptom of this disease, the author reviews the entire subject of "Aphasia in Children." Regarding aphasia from a purely clinical point of view, Bernhardt concludes (as many others have) that aphasia is of frequent occurrence in children, that its etiology is about the same as in adults, with this exception, that reflex-aphasia (so called) is more common, and that this special form may follow upon indigestion, psychical emotions (fear, fright, etc.), and may be consecutive to, or appear in the course of, many acute infectious diseases; that all acute or chronic cerebral diseases which would in the adult be accompanied by aphasia are attended by this same symptom in children; that in the majority of cases aphasia is a transient symptom which is not necessarily coupled with *right* hemiplegia, and that it is generally of the ataxic or motor type; and lastly, it is the author's opinion that the nature of the lesion in cases of congenital aphasia (without paralysis) has not been satisfactorily established. The symptoms of the disease, for which the author advocates the term "(Infantile) Spastic *Cerebral* Paralysis," are the following: At first general or unilateral convulsions, preceded occasionally by tremors in the affected part, and followed at irregular intervals by typical epileptic convulsions; aphasia; intellectual impairment, which may lead to idiocy; hemiplegia of the cerebral type, the leg not as severely paralyzed as the arm; athetoid movements most pronounced when the attempt is made to move the paretic fingers; associated movements of the healthy side similar to those attempted on the hemiplegic side, or *vice versa*; no marked sensory disturbances; no R. D.; knee-jerks present, and somewhat exaggerated on the affected side.

While the author believes that we are justified in considering this disease a *clinical* entity, he states that the pathological lesions are not always the same; the various pathological processes having but this one feature in common, that they ultimately lead, if the life of the patient be spared, to an atrophy and sclerosis

of the component elements of the entire hemisphere. For the present then the disease should be designated according to the clinical symptoms and not by the pathological terms—Porencephaly (Kundrat), or Poliencephalitis (Strümpell). For a detailed differential diagnosis between this disease and the better-known infantile spinal paralysis, we refer the reader to the original article, which is well worth reading, and also to the article of Dr. McNutt on Double Inf. Sp. Hemipl. (in the *Am. J. of Med. Sci.*, January, 1884), with which Bernhardt does not seem to be acquainted.

Ueber einen Fall von Porencephalie. By PROF. O. BINSWANGER, of Jena. *Virchow's Archiv*, vol. cii., p. 13, 1885.

In connection with the above article of Bernhardt, Prof. Binswanger's paper is of especial interest. It is a pathologico-anatomical contribution to the subject of which Bernhardt has treated from a purely clinical point of view. This case of defective cortical development which Binswanger describes is especially noteworthy, as the individual concerned attained the age of forty. The history of the patient was as follows: No hereditary predisposition to mental or nervous diseases. The patient was born with his right upper extremity atrophied and paralyzed; it could never be used. The right lower extremity paretic. Epileptic convulsions from earliest childhood on; the attacks were severe; the patient would often be in an unconscious condition for days and weeks at a time; marked dementia. In later years the epileptic attacks were followed by a maniacal condition, for which reason he was committed to the insane wards of the Charité. The patient could never learn to speak properly; his entire vocabulary consisted of a very few words and a few stereotyped phrases which he had occasion to use frequently. He was able to write only his name (with his left hand). He died after a series of very severe epileptic attacks. The autopsy, though not complete in every respect, revealed that the following cortical areas were wanting: 1. The entire third frontal convolution. 2. The lower third, and almost the whole of the middle third of both central convolutions. 3. The whole of the inferior parietal lobule (lobulus supramarginalis and gyrus angularis). 4. The whole first temporal convolution, and the region of the island of Reil, though possibly some of the latter's convolutions were preserved. In the region surrounding the porencephalic area the normal convolutions radiated outward from the centre of the destroyed area, in the manner which Kundrat supposes to be characteristic of congenital porencephaly, but on the other hand the arachnoid membrane did not simply stretch across the porencephalic area, as Kundrat says is the rule in these cases, but the thickened arachnoid adhered firmly to the central portions of this area. For various reasons Binswanger argues that this pathological process did not set in until toward the latter part of fœtal life. In this case the Sylvian artery and all its branches were poorly developed, and the author

supposes that this was due either to disturbances in nutrition, or to some traumatic local inflammatory process resulting in necrosis (anæmic) of cerebral tissues and defective development. The entire right hemisphere was quite normal ; it is certainly strange, therefore, that this hemisphere did not assume certain functions of speech ; the author is probably correct in assuming that this was due rather to the inferior mental status which did not require much speech, than to inability of the right hemisphere to assume these functions.

(From a comparison of the clinical and pathological data in cases of this sort, it is very evident that most of the symptoms will vary in each case according to the area or areas involved. The question is still an open one, therefore, whether it will be wiser to designate these affections according to the clinical symptoms or the pathological lesion.) B. S.

Note sur l'Existence de l'Ovarie dans la Chorée de Sydenham. By P. MARIE. *Progrès Médical*, p. 39, 1886.

Marie calls attention to the various points of tenderness and pain upon pressure which are met with in chorea. After mentioning the points described by Rosenbach and Seifert, the apophysal, thoracic, and lumbar, he calls attention to the occurrence of pain upon pressure over the ovaries. In the case of a small choreic girl he found, besides certain hysterical symptoms, well-marked pain over the ovaries. In 33 cases of chorea minor, of which 27 were girls and 6 boys, pain in the ovarial region was absent only 9 times. Sometimes the pain was spontaneous, but in most cases it could only be produced by pressure. Of the 9 cases in which it was not found 5 were boys, and pressure upon the spermatic cord or testicles did not cause any pain whatever. Marie says that the side upon which the ovarian point is found is always the side upon which the choreic movements commenced. The author inclines to the view that chorea is a form of hysteria, but would like more corroborative evidence before expressing himself positively. G. W. JACOBY.

Zur Pathologischen Anatomie der Bleilähmung (Lead Paralysis). By OPPENHEIM. *Arch. f. Psych.*, xvi., p. 476. And **Ueber Bleilähmung.** By SCHULTZE. *Arch. f. Psych.*, xvi., p. 791.

The question of the exact nature of lead palsy is still open. Erb and Remak argue very strongly in favor of the view that it has a central origin, and that it is due to lesion in the anterior cornua of the spinal cord. The symmetrical bilateral occurrence of paralysis with atrophy ; the peculiar localization of the paralysis in certain muscles, as if only particular groups of cells in the cord were affected, as in the case of infantile paralysis ; the resemblance in other respects, chiefly in the absence of sensory symptoms, and the presence of vaso-motor and trophic phenomena,

which lead palsy bears to anterior poliomyelitis, are cited in favor of this hypothesis. And it is considered by the authors cited as definitely proven by the discovery in a considerable number of cases (12) of lesion in the gray matter of the anterior horns of the spinal cord. To these cases Oppenheim adds another in which an extensive anterior myelitis was found in an individual who gave a clear history of lead poisoning, and who had suffered from general lead palsy on several occasions and finally succumbed to the disease. The history is fully detailed, and the lesions clearly defined and described by the author. He shows that the condition was one of primary anterior poliomyelitis, affecting especially certain definite portions of the gray matter in the enlargements, and that it could not be traced to an extension of inflammation inward from a peripheral neuritis. The lateral portions of the anterior cornua escaped, and as the extensions were chiefly implicated the conclusion is drawn that these muscles are not innervated by the lateral groups of cells.

An interesting complication hitherto unnoticed was a gangrenous stomatitis, which occurred just prior to death.

On the opposite side of this question Schultze, of Heidelberg, brings an equally strong argument. He holds that the distribution of the paralysis may be equally well explained on the theory of a peripheral neuritis—the motor symptoms in multiple neuritis and poliomyelitis anterior having been shown by Leyden to resemble one another too closely to be distinguished in many cases. In both conditions atrophy and reaction of degeneration are common ; and it is certain that neuritis does not necessarily affect nerve trunks, but may involve only motor terminal branches, so that from the escape of certain muscles in the domain of a nerve, while others are paralyzed, nothing can be concluded. As to the absence of sensory symptoms, it is well known that some poisons have a selective action on the peripheral nerves, and that the motor or the sensory filaments may be affected independently by them, *e. g.*, conium, curare, atropin, pilocarpin. Why should not lead affect chiefly the motor nerves, which, in many parts, lie wholly separate from the sensory nerves in the extremities ?

It is certainly no more likely that lead should display a selective action for certain groups of cells in the cord than for certain sets of fibres in the nerves. The analogies to infantile paralysis are subjected to a searching criticism and shown to be less strong than has been supposed. The symptoms, therefore, are subject to an interpretation which favors the theory that peripheral neuritis is to be regarded as the lesion in lead palsy.

To the large number of cases in which an autopsy has demonstrated the absence of changes in the cord and the presence of changes in the peripheral nerves, Schultze adds another. In the radial nerves, below the point where the branch to the supinator longus muscle is given off, a marked atrophy, with disappearance of nerve fibres and increase of connective-tissue nuclei, was discovered, the sensory branches of the nerves being slightly, if at

all, affected. The spinal cord was generally normal, no evidence of myelitis being present, no atrophied degenerated cells. A slight difference from normal consisted in there being a rather smaller number of cells in the anterior inner group in both enlargements on both sides, but even here no evidence of any change in their structure was to be detected.

He concludes that a lesion of the cord is certainly not a constant occurrence in lead palsy ; and then examining the case of Oppenheim, already described, concludes that in it a condition of myelitis had developed, independent of the condition of lead paralysis. In other cases cited by Oppenheim, similar objections can be made, and in many of them no examination of the peripheral nerves was made. Further, Schultze shows that according to the law of Wallerian degeneration, if the lesion destroys the motor cell, its fibre degenerates in its entire length, while in lead palsy it is only the peripheral terminations of the nerve-fibres, not their whole length, which is atrophied. In many of the cases of lead palsy, there has been no affection of the anterior nerve-roots, which must have occurred had the cells from which they come been the seat of the lesion, as in infantile paralysis. From the existence of atrophy, limited to the peripheral terminations of the motor nerves in lead palsy, the lesion must be considered a peripheral neuritis. No explanation of the distribution of the paralysis can be given on either hypothesis.

The third view taken by Freidlander, that the lesion is primarily in the muscles, Schultze combats on the ground of the presence of a change in the nerves, which, as far as known, is never secondary to muscular atrophy. M. A. STARR.

A Case of Amyotrophic Lateral Sclerosis with Clonus of the Lower Jaw. By C. E. BEEVOR, M.D., etc. **With a Note on the Jaw-Jerk or Masseteric-Tendon Reaction in Health and Disease.** By A. DE WATTEVILLE. Reprint from *Brain*, part xxxii.

The "jaw clonus" is the only symptom of especial interest in this case. Dr. Beevor states that in his patient it could be obtained by placing the finger on the teeth of the lower jaw, and then depressing it. * * * "The lower jaw would then vibrate as long as the pressure was kept up by the finger on the teeth." Clonus could also be readily produced by striking the masseters. Dr. de Watteville, in his "Note," gives his method of eliciting the "jaw-jerk." He finds it best to introduce into the experimentee's opened mouth a tongue-depressor, or a paper-knife, which is made to press with its flat surface upon the teeth of the lower jaw. * * * "The blade is held down with a firm pressure, care being taken that the jaw is not fixed by any undue muscular contraction. A sharp tap with a convenient object, such as a ruler, or best of all with a percussion hammer, is then struck upon the paper-knife, close to the teeth." Dr. de

Watteville surmises that this symptom may at times acquire a diagnostic value in cases of bulbar or other disease. De W. and Waller obtained myographic tracings of the jaw-jerk, and found that .02 second is the time for the latency of the contraction of the muscles of mastication, both in the healthy and the diseased subject, a fact which, if corroborated, might argue in favor of the view that the so-called tendon reflexes are phenomena, due not to a true reflex excitation, but to the direct stimulation of the muscles by the sudden extensile impulse of the blow. (As Dr. de Watteville states in this note that the phenomenon he describes was first mentioned in America, but that he can find no reference to it, we would say that Dr. Morris J. Lewis was the first to record what he (Lewis) termed the "*chin-reflex*," that his case was reported in the *Philadelphia Med. News*, March 11, 1882, and that mention is made of it in the proceedings of the Philadelphia Neurol. Soc., published in this JOURNAL for April, 1885. As for the two terms, de Watteville's designation seems to us the more appropriate one, though we do not approve of the unpleasant alliteration.) B. S.

---

d.—MENTAL PATHOLOGY.

Post-Neuralgic Insanity. By Dr. C. H. HUGHES. *Alienist and Neurologist*, October, 1885.

Reports of two cases of insanity which were the "symptomatic expression of cerebral exhaustion." In one case the cure of precedent neuralgia, and in the other the marked amelioration of neuralgic symptoms, furnished the exciting cause precipitating the mental sequence. The mental symptoms presented were those ordinarily found in psychoses due to physical exhaustion, and both patients had been much run down. Schüle, in his manual, had pointed out a relation between insanity and neuralgia, but this is not of the kind here indicated.

Puerperal Insanity. By Dr. T. K. HOLMES. *Detroit Lancet*, November, 1885.

Dr. Holmes is of opinion from examination of twelve cases that cervical laceration is a not infrequent cause of puerperal insanity. He is evidently not aware of the fact that puerperal insanity has been repeatedly observed to occur independently of any uterine lesion, and further that most cases have a natural tendency to recovery. Dr. Holmes uses mania as a synonym for insanity most of the cases being decidedly not cases of that psychosis.

Alcoholic Insanity. By Dr. H. C. WOOD. *Polyclinic*, November, 15, 1885.

W. reports the case of a man who killed his son. C. B., a forty-five year old man, had been an exemplary husband and citi-

izen, until the last two years. He had long used alcohol freely, but until the last year had not drunk to excess. During this period he had become an habitual sot, and was generally under the influence of liquor, and had then abused his wife, and accused her, in the grossest manner, of being unchaste. He was excessively violent toward her, on several occasions pursuing her with some weapon, with the intention of killing her. Upon the stand witnesses stated that he would have killed her had he not been restrained by force. On one occasion he had put her out of the house, publicly declaring "that she was no better than a hound bitch, and that he would stand her on the porch and the men would come to her." He had sometimes been so furiously violent that he had to be tied, hand and foot, for hours. As it was generally believed that he would kill his wife, she separated from him. About four weeks before the crime for which he was tried, he declared that he would cease drinking and endeavor to get his wife back, but before her return, he had taken to drink again, and by the advice of friends she had remained away; the children were left with him. At no time were his business faculties impaired; not rarely was he almost dead-drunk, and only at long intervals was he twenty-four hours without drinking. When he abstained several days from liquor he was kind to his family and wife as formerly, and stated to witnesses that he had no belief in the accusations he made when drunk, so perpetually and freely, with apparent sincerity, against his wife. For these last reasons Dr. H. C. Wood does not believe the case one of alcoholic insanity, but as Dr. C. K. Mills says: "Many individuals who, when sober, are in a delusional condition, have then sufficient power to restrain themselves from committing overt acts. Some of these, when under the influence of liquor, commit crimes which are the results of their delusions. The exciting cause of the overt act is alcohol taking." It has been said by Spitzka, and it is in accord with the experience of the majority of alienists, that alcoholic lunatics are not consistently regulated by their delusions. Thus one patient may live in comparative tranquillity with a wife whom he suspects of committing adultery in the boldest manner and before his face night after night. In all probability the case was one of alcoholic insanity.

Pyschoses after Cataract-Operations. By LANDESBERG, *Medical and Surgical Reporter*, October 17, 1885.

Reports of two cases of insanity after cataract-operations. The first, a man aged sixty-five, had suspicional delusions with auditory, visual, and gustatory hallucinations. Four days after the onset of the mental symptoms the patient recovered. The second case was that of a woman, aged fifty-seven, on whom two operations for cataract were performed. The second day after the second operation the patient was irritable, querulent, and had auditory hallucinations followed by marked but temporary depression,

and succeeded by a period of excitement. About four days after the onset of the mental symptoms the patient also recovered. The symptoms very markedly resemble those described by Schnabel (*Berichte des Naturwissen. Medic. Verein in Innsbruck*, Jahrg. viii.) as occurring in elderly people after cataract-operations. Approaching senility predisposed to insanity.

Insanity from Cocaine. By Dr. D. R. BROWER, *Journal of the American Medical Association*, Jan. 16, 1866.

B. claims that cocaine may produce such a mal-nutrition of the brain as to cause insanity, and in the course of his paper he cites the following case: Dr. B., aged thirty-five, a neurotic man, a hard-working physician, in very laborious practice. Three years ago he discontinued opium, which he had been using excessively. He began the use of cocaine last May in one-eighth-grain doses, believing it to be a harmless stimulant, and being much run down by overwork. It gave him a sense of well-being never experienced from any drug before, very much more agreeable than that derived from opium. He gradually increased the dose until it reached fifteen grains a day hypodermically. The large doses produced mental disturbance; he became irritable, quarrelsome, claimed to be possessed of a mission to revolutionize the medical practice, by being able to cure all diseases by cocaine. He gave it indiscriminately to all patients, to obstetric and syphilitic cases, to his wife, his three children, and his mother. Formerly a modest man of science, he became bold and unscientific in his methods, engaged in law-suits, carried a pistol, and frequently brandished it in public places, threatened vengeance upon all who dared to doubt his extravagant statements—a perfect terror in his neighborhood. He had been a very devout Roman Catholic, but his priest could not now restrain his wild impetuosity. By his manner and his neglect he very soon lost his practice. Piece by piece his horse, buggy, and furniture disappeared, until his family was reduced to poverty. Dr. Brower's efforts to persuade him to stop the drug resulted in making him the recipient of the wildest denunciations. Several physicians and druggists who made attempts to restrain him met with equally positive rebuffs. Extreme pallor and dryness of skin, great emaciation, loss of appetite, and no desire for sleep, so that for one week he did not assume the recumbent position, resulted. He went from bad to worse until his friends thought it best to restrain him. In pursuance Dr. F. L. Wadsworth and Dr. Brower advised the County Court to remove him to the Washingtonian Home. Here the cocaine was gradually withdrawn, but his extravagant delusions remained. He left this institution clandestinely, and is supposed to be in Canada. Were it not for Dr. Brower's reputation as an alienist it would be a not unnatural supposition to regard the patient as being a hypomaniac or in the early stages of paretic dementia. The influence of the cocaine in this case seems somewhat doubtful.

J. G. KIERNAN.

Society Reports.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, November 28, 1885.

The President, S. WEIR MITCHELL, M.D., in the chair.

Drs. CHARLES K. MILLS and JAMES HENDRIE LLOYD read "Notes on the Diagnosis of Spinal Tumors."

The subjects of general and differential diagnosis of spinal tumors were alone considered; that of local diagnosis—*i. e.*, of position of the growth at different levels of the spinal axis—was deferred for another paper. The conclusions were based on a study of fifty cases, three of which were personal observations, and the remainder were collected from American, English, French, and German literature. The general diagnosis—*i. e.*, the decision as to the spinal growth—can be made from a certain train of symptoms. These are of two classes: (1) those which are directly indicative of such a lesion; (2) those which are corroborative. The facts which point directly to a growth within the canal are: History of a constitutional or other cause, as of syphilis, cancer, tuberculosis, or traumatism. The onset and development are gradual and of a peculiar character. The first symptoms are those of an irritative lesion. This is probably due to the fact that tumors usually involve the membranes, as demonstrated by the cases studied. Hence, pain in the back or limbs is usually, but not always, present. The *sensory phenomena*, such as hyper-

æsthesia, anæsthesia, and paræsthesia, come on with more or less rapidity, with oscillating and irregular manifestations. *Motor symptoms*, such as paresis, spasm (tonic or clonic), contracture, tremor, ataxia, also manifest themselves in a similar oscillating manner. Exaggerated *reflexes* present themselves in most cases. *Trophic* and *vaso-motor disturbances* develop as the lesion progresses. These are such as wasting, ischæmia, œdema, and decubitus. *Visceral disorders*, such as paralysis of the bladder or rectum, or of their sphincters, and disorders of the heart and respiration, are, sooner or later, marked symptoms. Vomiting, gastric and intestinal disorders, are present in a limited number of cases. Mental disorders are present in only a very limited number of cases of tumors high in the spinal axis, but are not of diagnostic importance. Briefly stated, the phenomena which point with comparative certainty to the existence of spinal tumors, are symptoms of meningeal irritation gradually increasing, and symptoms of slow compression of the cord. The data in the cases studied were somewhat meagre as to *duration*. The usual duration is from six months to three years. It may be noted that fever was present at some stage in a considerable percentage of cases, but was so irregular as not to be of much diagnostic value.

The differential diagnosis of spinal tumors was considered in reference to the following affections: Spinal congestion, hemorrhage, meningitis, caries, traumatisms, sclerosis, aneurisms, neuritis, metallic and infectious disorders, and hysteria.

Spinal tumors are of constitutional or special origin, as syphilis, cancer, tuberculosis, etc.

Onset is gradual and irregular. Duration is comparatively long.

Progress is gradual, by irregular advances, toward a fatal termination.

Symptoms are inclined to be irregular, *i. e.*, unilateral or local; later, bilateral. Special symptoms, as paralysis, spasm, sensory and visceral disorders, are irregular as to time.

Decubitus and trophic changes are common late.

Reactions of degeneration are often present.

In *spinal congestion*, constitutional cause is not present.

Onset is usually sudden and after exposure. Duration is short, from a few days to four months.

Disease is stationary, then retrogression of symptoms toward recovery occurs.

Symptoms are more uniformly bilateral; motor and other symptoms develop about the same time.

Decubitus is rare.

Reactions of degeneration are rare.

In *spinal hemorrhages*, there is no special history, or a history of cardiac or vascular degeneration.

Onset is sudden.

Progress is more regular.

The first symptoms persist, and secondary degenerations follow, and differ according to extent and location of lesion, but are more likely to be uniformly bilateral.

In *meningitis*, the symptoms of localized compression are absent. The girdle symptom is absent. The affection is sometimes curable.

Reactions of degeneration are absent.

In *caries*, deformity is rarely absent. Rigidity of the muscles of back is a very important symptom.

In *traumatisms*, there is usually a history. The symptoms are those of caries, myelitis, meningitis, or combinations of these, according to character of case.

In *sclerosis*, the symptoms are usually of progressive systemic affections Compression symptoms are absent.

Duration is longer.

Progress is gradual, and more regular.

Aneurisms are only to be distinguished when extra-spinal, causing erosion and compression.

In *neuritis*, irregular sensory, motor, and reflex disturbances are present; compression symptoms and visceral disorders being absent. It is curable.

In *metallic and infectious disorders*, a history of definite causation is present. In metallic disorders there may be special characteristic signs, such as lead line, etc.

In *hysteria* a precedent hysterical history is usual.

Onset is often sudden. An emotional element is present. The symptoms are bilateral. Trophic changes are absent. No reactions of degeneration are present.

DR. E. N. BRUSH PRESENTED THE BRAIN AND SPINAL CORD REMOVED THAT DAY FROM A CASE OF GENERAL PARESIS.

The case was an interesting one in connection with the question of the association of the physical and mental symptoms of paresis. In this instance physical symptoms preceded the mental by some months. In the winter of 1883–84 the patient was discovered by his physician to be ataxic; symptoms of posterior spinal sclerosis increased, and in January were associated with some mental disturbance. The patient was irritable and forgetful, but in a general way complacent. He thought his business was unusually successful, and that he possessed remarkable ability to transact business affairs. He was easily confused, and in attempting to make a short journey alone over a route with which he was perfetly familiar lost his way, and found himself several miles from his home on the wrong train.

His mental disturbance became more aggravated, and at last it became necessary to place him in a hospital. He was first placed in a private institution in a neighboring State, but there—doubtless after a slight epileptiform seizure—became so much disturbed that his removal was requested. In August last he was admitted to the Insane Department of the Pennsylvania Hospital.

On admission his mind was markedly impaired. He was complacent, quietly submitted to suggestion, and was apparently demented. There were considerable disturbance of speech, marked tremor of tongue and lips and of upper extremities. Patellar tendon reflex abolished; pupils contracted to pin-points and not responsive to light; Argyll-Robertson symptom was present; gait ataxic.

During his time in the hospital the patient had three epileptiform seizures, such as are met with in cases of paresis. From these he readily recovered, but with increased impairment of mental and physical powers. After two hours of the seizures he was aphasic for some hours.

At eight o'clock on the morning of the day of his death, the patient had a slight vertiginous attack, followed by loss of power in arms and legs to a great degree, and disturbance of the power of co-ordination to such an extent that he could not, except after repeated trial, direct his hand to grasp an article held up before him. He protruded his tongue, opened and closed his eyes, and did other simple things upon direction; but to every thing responded "yes," and did not seem able to articulate any thing else.

At 3.30 P.M. the nurse's attention was attracted by the dusky appearance of his face, which had previously been pale.

The patient was seen at once by Dr. Brush, who found his face almost cyanotic, eyes injected, pupils closely contracted. In a few minutes he had a severe convulsion. At this time his temperature was 102⅖° F. The convulsion continued, and at 4.15 the temperature was 108½° F. Death occurred at 5.45. The temperature was then 107⅖°, and the body was bathed in perspiration. An hour after death the temperature of the body was 107° F. Post-mortem rigidity came on rapidly, and at eight o'clock was very pronounced.

Examination, sixteen hours after death: The conditions found were but briefly referred to, as the speaker intends to report this and some other cases more in detail to the Society.

The skull, especially in the temporal region, was quite thin; the dura was strongly adherent to the skull, and along the median fissure to the arachnoid or pia; the arachnoid was, as would be seen, thickened and opaque over the frontal and temporal convolutions, and along the Sylvian fissures; the pia in these locations was intimately connected with the cortex, and could not be separated without tearing it or the cortex cerebri. The sections made through the brain showed marked sclerosis; indeed, sections made through the anterior convolutions gave considerable resistance to the knife. The cord in the middle dorsal region was much injected.

Card from Dr. Morton.

Dr. Morton would say to his associate editors, collaborators, subscribers, and other friends, that with the present issue he will retire from the editorial management of the Journal. Accumulating literary and professional engagements render it impossible for him to do justice to the work; and he has therefore for the present transferred all the responsibilities of the Journal and its control to the gentleman who has so ably assisted him during the last year, and who now accepts the task with enthusiasm. The retiring editor would therefore bespeak for the new editor a continuation of that kindly consideration which has been his own only reward in connection with the Journal.

The present editor assumes his new duties with a full sense of the responsibilities they entail upon him. It will be his aim not only to sustain the enviable reputation the Journal has gained for itself, but to do all in his power to raise the standard of this publication, and to increase its usefulness among specialists and the medical world in general.

To this end, the following changes will be made:

1. *The Journal is to be published monthly.* This will help to lend renewed life to the Journal; it will ensure the early publication of original articles, and will enable the editor and contributors to report promptly on the most recent developments of neurological science. Each number is to consist of about sixty-four pages.

2. *Not more than one half the space of each number will be devoted to original articles.* Short papers will be quite in order; lengthy papers will be published in instalments.

3. *The department of "Clinical Cases"* will be constituted a regular feature of the Journal.

4. *Special attention will be given to the "Periscope."* The editor

recognizes and approves of the importance attached to such work by the American neurologist and general practitioner. All noteworthy publications will be promptly reviewed, and with such critical remarks as the individual reporter may see fit to make. Each review will be signed by the name or initials of the writer. It is particularly desirable that as many as possible should join hands in this work of review. The editor is deeply grateful to the five gentlemen who have done all the "Periscope" work during the past year. It is to relieve them of some of the work, and to enable them to contribute to the several subdivisions of the "Periscope," that he has asked others to co-operate with them.

5. *Book reviews* will be published as heretofore, though not necessarily in every number.

6. *The Proceedings of the New York and Philadelphia Neurological Societies* will be published as fully as the space of the JOURNAL will permit. Reports of other societies will be published occasionally, at the discretion of the editor.

7. The Editorial Department, as such, will be abolished. Under "Editorial Notes and Miscellany" information of general interest to the profession will be published from time to time.

CERTAIN minor changes in the arrangement of the JOURNAL will be detected by a close inspection of this number. The present number may be considered typical of those that are to follow in all but one respect—the space devoted to original articles. Dr. Seguin's article was in type before the changes in the editorial management had been effected. As stated above, articles of similar length will hereafter be published in instalments.

VOL. XIII. FEBRUARY, 1886. No. 2

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

A CASE OF PARALYSIS OF THE TRIGEMINUS FOLLOWED BY ALTERNATE HEMIPLEGIA—ITS RELATIONS TO THE NERVE OF TASTE.

By C. L. DANA, M.D.

Mr. H., single, æt. thirty-six, Canada, was born of healthy parents, and had never suffered from any disease except a very severe and chronic eczema of the scalp. He has drank and smoked moderately, has played racket and tennis a great deal, being, I believe, one of the champion racket-players of Montreal.

He denies absolutely ever having had syphilis, and gives no history of it.

Last August (1885) he noticed a feeling of numbness upon the left side of the face. The onset of this was not accompanied with any marked symptoms, and he did not consult a physician.

His eczema becoming worse, he came, on Nov. 29, 1885, to Dr. George L. Fox for treatment.

On Nov. 30th he had been a little dizzy, but went to bed feeling well. Next morning he awoke paralyzed on the right side in arm and leg, and in left side of face.

The loss of motion was greatest in arm, but was not complete. The facial paralysis was slight but unmistakable, and the tongue protruded to the left (affected) side. There was also anæsthesia complete in the left side of the face and left half of the mouth and tongue as far back as the pillars of the fauces. The uvula hung straight. The muscles of mastication were also slightly involved, judging from the fact that the patient could not chew on the left side because "the jaws felt too weak" for the effort. He had always chewed on that side before. There was a perceptible atrophy of the masseter on the left side. Some ataxic aphasia

was present for a few days. There was some paræsthesia on the right side, but no anæsthesia at all.

The temperature on the two sides the same evening was equal and normal. The patient grew a little worse during the first twenty-four hours, then began steadily to grow better, except as regards the paralysis of the fifth nerve.

The tongue was tested carefully and repeatedly for loss of taste, but the patient could distinguish bitters, acids, salts, and sweets equally well on each side both in front and at the back. He simply complained of a feeling of numbness on the left side of the mouth.

The sense of smell was not affected, and the nasal branches of the fifth responded to ammonia as well in the left nostril as in the right.

There was no disturbance of hearing.

On Dec. 27th, three and a half weeks after the seizure, I examined him again.

He could walk very well, and move his arm and use his hand. Grip of right hand, 28; left, 48. No sensory disturbance on right side, and no contractures.

The left facial paralysis had almost entirely disappeared, but the left half of the forehead looked a little smoother, and the tongue protruded a little to the left.

Anæsthesia was almost complete over the whole of the left side of the face, from the chin to a line running vertically from the auditory meatus, and as far back as the tragus. The lower part of the chin and cheek were a little more sensitive, owing to the psoriasis and application of irritative medicaments, which had caused some inflammatory congestion.

Tactile, temperature, and pathic sense were all abolished. The patient could just feel the hard pressure of blunt or sharp points, but could not count or locate them. The cornea was insensible also. So were the left half of the tongue as far back as its root, and the gums and palate as far back as and including the anterior pillars.

There was no apparent disturbance of the mucous or salivary secretion. The left eye showed slight myosis and myopia, and vision was less distinct. The pupillary reflexes to light and accommodation were normal. There were no trophic disturbances.

On Jan. 1st the patient was again attacked with a hemiplegia, affecting mainly the left arm. The paralysis came on slowly without much mental disturbance. The face was slightly affected. There was no change in the anæsthesia.

It is quite impossible as yet to say positively by what paths sensations of taste reach the brain. It has been generally taught that the glosso-pharyngeal is the nerve of taste for the posterior third of the tongue and the trigeminal for the anterior two thirds.

Dr. Gowers has, however (*Jour. of Physiology*, vol. iii., p, 384), published a report of a case of total trigeminal paralysis, with total loss of sense of taste over the whole of the tongue on the affected side. He affirms, in his work on "Diagnosis of Brain Diseases," that all taste-sensations reach the brain through the fifth cranial nerve, and that the idea that the glosso-pharyngeal is a nerve of taste is a curious physiological myth. Vintschgau (Hermann's "Handb. der Phys.") states just as positively, after reviewing the question, that no physiologist at the present day doubts that the glosso-pharyngeal supplies taste to the back part of the tongue. Landois, Duval, Lussana, Vulpian, and many of the earlier physiologists, assign to the glosso-pharyngeal or the intermediary nerve of Wrisberg, all taste-functions.

So far as opinions go, therefore, we really do not seem to have any definite nerve of taste. It appears to me to be something of a reproach that a special sense peculiarly mammalian, and still more peculiarly the characteristic of civilized man, should be without a known conducting organ; and that the afferent impulses excited by the modern dinner should pass up to the brain through uncertain and unknown channels.

My own case has naturally led me to doubt the dogmatic assertion of Gowers; and its teachings, so far as they go, are in harmony with the view which I have of late held, that from the glosso-pharyngeal nucleus come all the fibres that conduct taste-sensations to the brain. Although I am quite aware that there are some serious objections to this view, yet it has so much *a priori* probability that I am constrained to use my case as the basis of a plea for the glosso-pharyngeal nerve.

The evidence that the glosso-pharyngeal is, in part at least, a nerve of taste is almost overwhelming.

1. By anatomical dissections the fibres of this nerve have been traced directly to the taste-organs (Vintschgau).

2. By the Wallerian and atrophy methods, Vintschgau and Honigschmied have found that after resecting the glosso-pharyngeal nerves in young rabbits the peripheral portions and the taste-buds atrophy and disappear (Hermann's "Handb. der Phys.").

3. The physiological experiments of Magendie, Panizza, J. Reid, Broughton, Valentin, Wagner, Stannius, Lussana, and others, show that after resecting the glosso-pharyngeals the sense of taste is partly or wholly destroyed.

4. The clinical evidence that the glosso-pharyngeal has something to do with the function of taste is almost unanimous. In all cases of trigeminal paralysis reported, so far as I can find, there is either no loss of taste or loss of taste only on the anterior two thirds of the tongue, Gowers' cases being the only exceptions to this rule.

The single case which he has reported in full (*vide loc. cit.*) was that of a patient who also had paralysis of the third and sixth cranial nerves, and as there was no post mortem it would be quite impossible to exclude the glosso-pharyngeal absolutely, and more especially any connecting filaments carrying taste-fibres to the fifth, as Carl, Prevost, and Vintschgau suggest.

But, furthermore, Gowers can certainly not be aware of the positive evidence in favor of a gustatory function for the glosso-pharyngeal

Thus K. B. Lehmann reports a case of one-sided traumatic paralysis of the glosso-pharyngeal. The tongue on the affected side was entirely insensible to the taste of sugar or quinine on the border, middle, and posteriorly in the region of the circumvallate papillæ. On the other hand acids, salts, and astringents caused certain taste-sensations, though apparently perverted. There was no loss of general sensibility (*Arch. f. gesammt. Phys.*, xxxiii., p. 194).

Again, Grasset has observed loss of taste in two cases of glosso-labio-laryngeal paralysis (" Mal. des Syst. Nerv.").

Another case of isolated paralysis of the glosso-pharyngeal is reported by M. Gendrin, in a translation of Dr. Abercrombie's work, on " Diseases of the Nervous System," p. 627. Here the nerve, on one side, was atrophied by pressure of cyst. There was loss of taste on the affected side over the whole surface. The case is reported with great explicitness.

I have now under treatment a patient suffering from bulbar paralysis, some of whose taste-sensations are per-

verted. A solution of quinine applied to his tongue he repeatedly assured me tasted sweet. If, to all the positive evidence thus referred to, we add the negative evidence, that trigeminal paralysis, no matter how complete, does not, with a very few exceptions, cause loss of taste at the back of the tongue, it appears to me that Gowers' rather flippant disposal of the glosso-pharyngeal as a nerve of taste has very little foundation.

Assuming, therefore, what has heretofore been almost uniformly admitted, that the glosso-pharyngeal nerve certainly carries the taste-sensations from the posterior surface of the tongue, the real problem remains to determine what nerve supplies the remainder of this organ.

No one now denies that the chorda tympani of the facial, almost always, if not uniformly, carries taste-fibres to the lingualis branch of the fifth, and through it supplies the anterior two thirds of the tongue.

But there are at least three views as to the origin of the taste-fibres of the chorda.

1st. The one usually accepted—viz.: that these fibres, running up the chorda, pass to the geniculate ganglion, then, via the large superior petrosal nerve, to Meckel's ganglion, and so via the second branch of the fifth nerve to the brain. This is the view first placed on a firm basis by the experiments of Schiff (1867–73).

It is supported by experiments upon animals and by cases of trigeminal paralysis with loss of taste in the anterior portion of the tongue, reported by Romberg, Von Meyer, Rigler, Austin, Hirschberg, Kocher, Guttmann, Erb, Senator, Gowers, and Seeligmann. The evidence thus furnished was sufficient to convince Erb that, "in most cases," this was their source; but he appears to think that there may be individuals in whom the paths of taste-sensations take other courses; and he tells us that a specially differentiated nerve, with definite peripheral end-organs, has no definite path to the brain. This is certainly not in accordance with analogies; nor should we expect nature to be so careless or indifferent in informing man of the sapidity of his environment. A special sense, which is as old as the

mollusks, should apparently have its *own* nerve-paths, and not be ministered to by a nerve like the fifth, already having the two functions of general and tactile sensibility.

Besides, there has now accumulated a great deal of evidence against the view that the trigeminus has any taste-fibres.

The physiological experiments of Panizza, Valentin, J. Reid, Prevost, Lussana, and Duchenne all point against this view. The cases of trigeminal paralysis without loss of taste now reported are so numerous and authenticated that they cannot be disposed of in the way that Erb did when he analyzed them ten years ago. Dr. Hislop, for example, had a case in which the tregeminal root and the spheno-palatin ganglion were destroyed without loss of taste. (Wilkes' "Diseases of Nervous System.") The cases of Vulpian (cited later) and my own must be added to those first criticised by Erb—viz., of Romberg, Stamm, Berard, Renzi, Viccioli, Nixon, and Althaus.

In *Brain*, Jan., 1886, Dr. Thos. Harris reports a case in which a tumor involved the right side of the pons, entirely destroying the fifth nerve and Gasserian ganglion on that side, and causing total hemianæsthesia of the face and tongue, without loss of taste.

Vulpian (*Compt. Rend.* No. 21, 1885) has recently stated his opinion, based on experiment and a clinical case, that the nerve of Wrisberg sends gustatory and vaso-dilator fibres to the anterior part of the tongue, through the facial and chorda t., and similar fibres to the soft palate, via the great superficial petrosal and Meckel's ganglion. The intermediary nerve of Wrisberg is, he thinks, the sensory root of the fifth.

He reports a case somewhat like mine. A man suffered with hemiparesis and hemianæsthesia of the left side of the trunk and extremities, but only left hemianæsthesia of the face. There was a *right* facial paralysis and partial loss of sense of taste on the *right* side of the tongue over its anterior two thirds, and on the right side of the soft palate. There was diminution of tactile sensibility on the left side of the tongue, but no loss of taste.

Recently this patient died, and Vulpian (*Prog. Méd.*, Jan.

9, 1886) found a small tumor of the size of a hazel-nut in the upper part of the right half of the medulla.

Dr. C. A. Carl (*Arch. f. Ohrenheilk.*, x., p. 152) describes a personal experience. He has absolutely no sense of taste in the left anterior half of his tongue. All his cranial nerves are normal, but he has had a chronic otorrhœa in the left ear, which has destroyed the drum. Carl thinks from direct experiments upon it that his chorda is not injured or paralyzed, and that the affected taste-fibres come from the glosso-pharyngeal leaving the ganglion petrosum, through Jacobson's nerve, tympanic plexus, thence in greatest part through the N. super-petros. minor to the otic ganglion, and so to the lingualis branch of the fifth; others, and in his case the smallest part, passing from the tympanic plexus to the geniculate ganglion, and thence to the facial, the chorda, and so to the lingualis.

The view that the taste-fibres of the chorda come from the intermediary nerve of Wrisberg has had some supporters, and Vulpian's case, above cited, is in its favor.

Spitzka (*Med. Record*, Jan. 31, 1880) claims that the fibres of origin of the nervus intermedius of Wrisberg do not originate in the facial nucleus, but in a "diffuse" nucleus lying below the altitude of the facial nucleus and in an ideal continuation of the gelatinous column of the trigeminal region; or, in other words, in the sensory gray column of the medulla.

Horatio R. Bigelow (*Med. Record*, Jan. 17, 1880) states, as the result of numerous dissections, that the chorda tympani is a distinct nerve from the facial, and is continuous anatomically with the nervus intermedius of Wrisberg.

Prof. M. Duval claims to have traced the N. intermedius to a continuation of the glosso-pharyngeal nucleus, of which he considers it an erratic branch.

The view that the nerve of Wrisberg is a sensory branch of the facial, carrying taste-fibres, is however contradicted by almost all clinical experience. In cases of facial paralysis there is usually no loss of taste observed unless the lesion is at or distal to the geniculate ganglion.

With regard to the very positive evidence that in some

cases of trigeminal paralysis there is loss of taste, this may be said. In all the cases of which I have been able to read the account the lesion was a peripheral one, involving the roots of the fifth and generally that of other neighboring nerves (*e. g.*, cases of Gowers, Erb, Romberg, Zambaco, *Méd.-chir. Rev.*, 1863, vol. i., p. 42), Ramskill (*Lancet*, Mar. 28, 1868, p. 406), Beveridge (*Med. Times and Gaz.*, 1868, vol. i., p. 199). Now the glosso-pharyngeal root is not far from that of the trigeminus; and the Vidian nerve, through which communication might be established with the glosso-pharyngeal, runs from Meckel's ganglion up through the foramen lacerum, passes directly under the Gasserian ganglion, and might easily be affected in diseases of that ganglion.

I think I may assert with some confidence that, as a point for local diagnosis, when there is a trigeminal paralysis with loss of taste, the lesion is probably peripheral; when taste is not involved, the lesion is probably central.

It appears to me, therefore, that we are justified in saying:

1st. That the glosso-pharyngeal sends taste-fibres directly to the posterior third of the tongue.

2d. That by the communications suggested by Dr. Carl it may send taste-fibres to the anterior two thirds of the tongue, *i. e.*, either through the chorda tympani or by various channels directly to the second branch of the inferior maxillary nerve.

The glosso-pharyngeal may communicate with the fifth, through the tympanic nerve and plexus, and thence via the small petrosal nerve to the otic ganglion, or via the large petrosal nerve and Meckel's ganglion.

3d. Paralysis of the trigeminus with loss of taste is due probably to a peripheral lesion, and other cranial nerves are generally involved; paralysis of it without loss of taste, to a central lesion.

I. The view that the glosso-pharyngeal is the sole nerve of taste is supported by:	II. The view that the glosso-pharyngeal supplies part of the tongue with taste is supported in addition by:	III. The view that the chorda carries some or all the taste-fibres to the anterior two thirds of the tongue is not denied. IV. The view that these fibres come from a nucleus of the glosso-pharyngeal through the nerve of Wrisberg, or some other channel, is supported by:
Panizza, 1834, by experiments.	Magendie, 1824, by experiment.	Duchenne, 1850, by experiment and clinical observation.
Stannius, 1848, " "	J. Müller, 1837, " "	Lusanna, 1864–70, " " " " "
Hall and Broughton, 1836–39, by experiment.	Rapp, 1832.	Duval (?)
J. Reid (?), 1838, by experiment.	Elsasser, 1834.	Carl, 1875, " " " " "
Valentin, 1834–64, " "	Wagner, 1845, by experiment.	Vulpian, 1885, " " " " "
Bidder, 1846.	Alcock, 1837, " "	Indirectly supported by cases of trigeminal paralysis without loss of taste, reported by:
Duchenne, 1850, by experiment and clinical observation.	Vintschgau, " "	Romberg.
Lussana, 1864–70, by experiment and clinical observation.	Biffi and Morganti, 1846, by experiment.	Stamm.
Prevost, 1873, by experiments.	C. Bernard, 1843–58, by experiment and clinical observation.	Berard.
Carl, 1874, by clinical observation.	Longet, 1869.	Renzi.
V. Urbantschitsch, 1876, by clin. obs.	By a long list of clinical cases of trigeminal paralysis, reported by Erb, Moos, Neumann.	Viccioli.
	By Lehmann, a case of glosso-pharyngeal paralysis.	Nixon.
	By Grasset's cases of bulbar paralysis.	Althaus.
		Hislop.
		Dana.
		Harris.
		Indirectly by experiments of Prevost in extirpating Meckel's ganglion.
		Against it are:
		The experiments of Schiff.
		Numerous cases of trigeminal paralysis, with loss of taste in anterior two thirds of tongue, reported by:
		Romberg, Von Meyer, Rigler, Austin, Hirschberg, Kocher, Guttmann, Seeligmüller, Erb, Senator, Gowers.

RACE AND INSANITY.

By JAS. G. KIERNAN, M.D., Chicago,

LATE MEDICAL SUPERINTENDENT COOK COUNTY HOSPITAL FOR INSANE.

THE NEGRO RACE.[1]

THE number of negroes suffering from paranoia[2] admitted to the Cook County Asylum was comparatively large, when it is remembered that the intellectual phenomena of this psychosis are such as would enable the negro paranoiac to pass muster as a superior person to the average member of his race. At the same time the fact that Chicago is a commercial city, permitting comparative equality on the part of the negro, leads to the quick incarceration of such persons, when brought in contact with Aryans and Shemites, for the mental phenomena displayed would be such as to lead to a rapid suspicion of insanity by a sceptical, practical Aryan or Shemitic business man. For example, one of the most marked cases of this type claimed to have been persecuted by means of mesmerism, the fact of which he accepted but ascribed to witchcraft, as a sane but ignorant man might have done. The talk of witchcraft renders a practical business man suspicious of insanity, and would soon bring the mental condition of an otherwise seemingly intelligent negro under suspicion. The case is as follows:

Case I.—T. A., æt. forty-three, was born in Mississippi, of a family in which nervous disease existed. Skull decidedly tectocephalic; occiput flattened. No history of early life attainable,

[1] A series of studies in ethnological psychiatry.

[2] Synonymous with *monomanie systematisée*, monomania of Spitzka, primäre Verrücktheit of the Germans.

except that he was a bright boy, and worked for a doctor named Mansfield, at whose home he saw some experiments in "mesmerism," soon after which he had what seems to have been a dream passing into a systematized delusion that he was destined to be a prophet of great medical ability. He evidently experienced some anomalous sensations, and was auditorily hallucinated in a disagreeable way. He was about, or soon after, this time an inmate of a hospital for the insane, placed there in consequence of a complaint made before a justice of the peace that the Mansfield family had bewitched him. He evidently believed that there were laws against witchcraft still on the statute-books, as he from time to time pestered lawyers in the attempt to have these alleged laws enforced. Soon after an interview with a lawyer on the subject of witchcraft laws, he committed larceny, and was sent to a State prison. The larceny was committed under the influence of an hallucination, and his conviction was due to his own confession, made with the idea of securing protection from persecution by incarceration in the prison, where it appears he studied several trades, and made an endeavor to learn law, in order to punish his persecutors, who, he believed, were liable to the law. At about the time his term in the State prison was nearly expired, he became convinced from some newspapers that a large fortune had been left him, and that the continued persecution of the Mansfield family was due to the fact that in order to prevent him from securing the fortune and achieving the destiny already mentioned, they found it necessary to make him crazy. He was, in consequence of this, sent to the Elgin Hospital for the Insane. From there he was sent to Jefferson, in 1875, whence his perverse tendencies subsiding, he was discharged "cured," but soon after was sent to Joliet Penitentiary, for larceny, evidently of hallucinatory origin. While there he was hoaxed into believing that John H. Logan and General Grant had had the witchcraft laws repealed, and upon learning this his persecutional delusions and hallucinations began to increase in number and force. He cut off his thumb, and made an attempt to cut his throat, in consequence of his hallucinations, which commanded him so to do, lest a worse thing happen. On his second entrance into the Cook County Hospital for the Insane, he remained for about two months relatively quiet, being under the belief that after Jan. 1, 1885, Gov. Oglesby would enforce by common law an action for conspiracy against his persecutors. February 1, 1885, his persecutory delusions again became active, and he struck a patient, who had the habit of talking in response to hallucinated voices, claiming that the latter was informing the Mansfield family of his whereabouts. From this time his delusions became less dominant, and he was sent out to work on parole with the kalsominers, and worked well for a while, until one of these men (all of whom were ex-ward workers) ridiculed him as a "nigger crank," for having such ideas as witchcraft, which he at first took in a relatively calm manner, until it was repeated in an offensive way, when he struck one of his tor-

mentors, and immediately refused to work, having new material for his delusions of persecution. He cited Grant's illness as an evidence that his persecution was about to stop, since the people would see that this illness of Grant was due to the latter's action in having the witchcraft laws repealed. He, at this time, became relatively quiet, proclaimed his abilities and his claim to his fortune with much more freedom than formerly, when he was rather secretive for fear of fresh persecution. It may be said that this man scouted Voodooism as having any thing to do with his persecution. It was, he said, all nonsense, but the mesmerism was a different thing. He was transferred to Kankakee, whence he escaped, went to Washington, and called on President Cleveland, in order to have the mesmeric influence of the doctor aforesaid removed by enforcement of the common law against conspiracy.

The other four cases, two men and two women, were tecto-cephalic, with flat occiputs; had all been guilty of attempts at homicide in consequence of systematized delusions, but these delusions were to some extent really the indirect result of bad treatment. One of the women had sexual delusions, directly due to the fact that males had been allowed keys to the female wards. Paranoia among negroes is usually accompanied by a relatively high amount of intelligence and education. The reasoning of T. A. on his delusions was decidedly logical. The man at times fully recognized the insane nature of some of his imperative conceptions, but referred these to witchcraft, as he called mesmerism, and his explanation of the way these ideas were introduced into his mind closely resembled the description which Matthews[1] gave of his own case. T. A. said that the first evidence he had of the influence of mesmerism was that he could not think his own thoughts; then they introduced crazy ideas into his head, and he could only think these ideas. In a barbaric country T. A. would have founded a kingdom or a religion. In the United States it is by no means improbable that he might have played the part of a Guiteau, but for his arrest before reaching President Cleveland.

[1] Haslam: "Illustrations of Madness."

NOTES ON ELECTRICITY.

By M. R. CRAIN, M.D., Rutland, Vt.

I.—THE DYNAMO IN ELECTRO-THERAPEUTICS.

IT has been suggested by several in the past few years that the dynamo would in the near future be used for medical purposes, as it is a much cheapear method of generating electricity than by galvanic cells. Most doctors have supposed that a dynamo would have to be constructed especially for medical use. Although that could be done, as all that would be necessary in ordering one would be to give the dynamo manufacturer the E. M. F. and capacity—as example, E. M. F., 70 volts; capacity $\frac{1}{4}$ of an ampère, that would make a small dynamo, but would be large enough for medical purposes.

I do not think dynamos constructed especially for medical purposes will come into general use, as there are several obstacles in the way.

First it is necessary to have a very steady power, and that is not generally available without too much expense, and if we have a steady power it requires a great deal of skill to keep it well adjusted; if the power is not steady the current will be very wavy, which makes it so harsh and painful that it is not adapted for medical use. Secondly, it requires a degree of mechanical skill to keep a dynamo in order that is not generally possessed by medical men.

The figures that I gave above as being all that are required in ordering a dynamo adapted for medical purposes, I gave on the authority of an electrician representing the Edison company.

There is a dynamo used in the arts that *is* adapted for medical use, and it is coming into use so fast that it will soon be available in the cities and all of the towns of any considerable size, viz.: the dynamo used for incandescent lighting.

I have been experimenting with, and using in my practice since Oct. 5, 1885, the current used for Edison incandescent lights, produced by two dynamos that are running with an E. M. F. of 110 volts connected in surface.

The current at my office where I take it off has an E. M. F. of 100 volts nearly.

I extend the wires from the ceiling to about the level of the table, where they terminate in metal posts that have holes bored in them to make connections with the rheophores or conducting wires. I use for conducting wires those used for connecting the lights in the Edison system, composed of twelve strands of copper wire covered first with rubber, then linen, and last of all silk.

I find these far superior to the ordinary conducting wires furnished with batteries.

I connect one wire with the rheostat,[1] one that I have constructed for the purpose; then I connect the rheostat with an exact galvanometer graduated in milliampères, made by John A. Barrett of New York.

I then connect the galvanometer with one of the electrodes and the other conducting wire with the other electrode; then every thing is ready for work.

Although the dynamo current is an alternating one when produced, it is turned one way by means of a commutator in all dynamos used for incandescent lighting; it then has all the properties of a galvanic current; it will magnetize steel, heat wire, and produce electrolysis of water, or the solutions of the salts, and is practically a galvanic current.

---

[1] I omitted the description of my rheostat because I am going to change its form in order to make it more convenient.

The current used for incandescent lighting is not dangerous to use unless the lights are in series.

It is necessary to use a resistance of from 10,000 to 100,000 ohms for every 100 volts of E. M. F.

The E. M. F. can be ascertained, if the lights are arranged in surface, by simply looking at the top of the lamp.

This dynamo current is well adapted for medical purposes, as its E. M. F. 100 volts is not much higher than some office batteries, and although there are 160 ampères produced by the dynamos, this makes no difference, as with 100 volts E. M. F., and say 100,000 ohms resistance, we can get only one milliampère, whether the dynamos have a capacity of 160 or $\frac{1}{10}$ of an ampère.

There is a theoretical objection to the dynamo current, and that is, it is a little wavy. Although the one used for incandescent lighting gives a comparatively steady current, it is impossible to construct a dynamo that will give as constant a current strength as can be produced by chemical action.

In calculating on the constancy of an electric current, one must take into consideration not only the E. M. F. of the machine, but also the constancy of the resistance.

Now as the body offers a very inconstant resistance, frequently varying as much as one fourth or one third in three or four minutes by the soaking of the skin and the increased blood supply to the parts, it follows that with the most constant battery we have a very inconstant current strength; that can be easily proved by an exact galvanometer.

Now suppose we take a battery and pass a current through the body, which has, say 3,000 ohms resistance; in three minutes it becomes reduced to, say 2,000 ohms, which increases the current strength one third in three minutes. Now take the powerful dynamo current and interpose a constant resistance of say 100,000 ohms, then add the body with its 3,000 ohms, and we have 103,000 ohms in the circuit.

If the resistance of the body is reduced to 2,000 ohms in three minutes, we have 102,000 ohms resistance in the circuit; that is, the current has increased only $\frac{1}{100}$ in three minutes. Thus we see with the most constant battery, unless we have a large number of cells and interpose a high resistance, it is impossible to get as constant a current strength as we get with a dynamo.

The dynamo current used in this way is smoother and less painful than a battery current of the same strength.

I have proved this repeatedly by giving to several persons a current of a definite strength—say five milliampères, and, with very few exceptions, they decided that the dynamo current was smoother and less painful.

I have found that the motor nerves and muscles react the same to the dynamo current as to the galvanic, viz.: K. C. C.; A. C. C.; A. O. C.; K. O. C., in the order named.

I find that the sensory nerves react in the same way as the motor nerves, viz.: K. C. S.; A. C. S.; A. O. S.; K. O. S.

The reaction of the gustatory nerves are interesting, but does not differ from that I have observed with the galvanic.

The positive pole, when placed on the tip of the tongue, gives a stronger metallic taste at the point of contact, also when placed on the side of the tongue, as near its base as possible.

The negative pole, when placed on the tip of the tongue, produces more irritation of the tactile nerves, but the metallic taste is very faint at the point of contact, while at the base it is very strong; but when the negative pole is applied near the base of the tongue, the metallic taste is much weaker than when the positive is placed there. I think there is no doubt but that the metallic taste is stronger at the positive pole, and the foregoing observation can be explained by De Watteville's polar and peri-polar zone theory.

The effect on the optic apparatus is the same as with the galvanic current; that is, the conversion of electric stimulus into impressions of light.

The color of the flashes varies with different individuals.

The negative electrode produces more irritation of the tactile nerves, unless we use very strong currents; we get the heating effects, which are mostly at the positive pole, consequently there is more pain produced at the positive pole with very strong currents.

Electrolysis.

In producing electrolysis of blood, and also of a mixture of water, salt, and the white of an egg, I find that a light, frothy mass is produced at the negative pole, while at the positive a firm clot is produced.

The amount of gas given out at the negative pole tends to make it frothy, while the alkaline reaction probably tends to keep it in a semi-liquid state.

The heating effects at the positive pole, and probably the acid reaction, tend to make the clot firm and hard.

In producing electrolysis of beef, there is more liquefying action at the negative pole.

Electrotonus.

In carrying out my experiments on this subject, I have followed the method of De Watteville,[1] with such modifications as were necessary in using the dynamo current.

Experiment I.

I first pass the current from a secondary induction-coil through 20,000 ohms resistance. I place the negative electrode over the ulnar nerve, the positive on an indifferent point, and see at what distance of the secondary from the primary coil the induced current begins to produce contractions. I next connect the positive pole of the dynamo with the negative pole of the secondary induction-coil with the same resistance, that gives me five milliampères of dynamo electricity. I then place the combined negative electrode on the nerve; this produces very powerful contractions, and it produces moderate contractions with a much less powerful faradic current.

Experiment II.

I pass the faradic current through the same resistance with the positive pole on the nerve strong enough to produce moderate contractions. I then connect the negative pole of the faradic with the negative pole of the dynamo, with the same resistance; that gives me five milliampères of electricity.

The electrode that I place over the nerve combines the positive of the faradic and the negative of the dynamo current, and that produces no contractions.

I have not given the detail of any of the rest of my ex-

[1] De Watteville's "Medical Electricity," p. 111.

periments, because they were performed the same as if a galvanic battery with a rheostat had been used.

As the result of my experiments and observations, I find that the physiological and therapeutic effects of the dynamo currents are the same as those of the galvanic. What, then, are the advantages of the dynamo current? Economy and convenience.

In utilizing the dynamo current, all the appliances that are necessary are a rheostat and a galvanometer, and they are necessary accessories to an office battery, if one wishes to apply galvanism scientifically; so one can save the expense of the battery on the first outlay, and, besides, one saves the expense of repairs, which are considerable on a large office battery.

With the dynamo, eight or ten cents' worth of electricity is sufficient to treat patients, with average-strength currents, two hours a day for a year, and you have no battery to keep in repairs.

It is no more trouble to turn on the electricity for medical use than for purposes of lighting.

II.—HIGH OR LOW RESISTANCE BATTERIES FOR MEDICAL USES.

There seems to be a great deal of confusion on this subject which could be settled in a few minutes by considering the facts in the case.

Many writers on medical electricity claim that cells of high internal resistance give a smooth current especially adapted for medical uses; which they describe in a vague way as tension.

Bartholow says: "The best results are attained when the interior resistance of the battery is equal to the resistance in the exterior circuit."

He claims the best results are obtained from a modified Daniell cell, which has its internal resistance increased by papier-maché packing and a porcelain diaphragm, so that it equals the resistance offered by any part of the body.

Hence it is said to be smooth and unirritating when the

same number of elements of Stöhrer give rise to great irritation and burning.

In this fact we find the true explanation of the superiority in curative action of the large elements of a permanent battery as compared with the small elements of a portable battery.

He says the internal resistance should be 20 ohms or more per cell. Now the facts are these: 50 Daniell cells with 20 ohms resistance each, give when passed through—say 2,000 ohms external resistance, $\frac{52}{2000+1000} = \frac{1}{60}$ of an ampère. While 50 Stöhrer give $\frac{70}{2000+100} = \frac{1}{30}$ of an ampère, or a current twice as strong.

Although this explains the main difference in the amount of irritation produced. I think it is not the only factor; as the amount of irritation produced by an electric current passing through the human body is also dependent on its constancy.

Now as the resistance of the body varies frequently as much as $\frac{1}{4}$ or $\frac{1}{3}$ in three to five minutes by the soaking of the skin and the increased blood supply of the parts caused by the irritation of the electrodes, it follows that with the most constant battery we get a very inconstant current strength. If we use a battery composed of 50 Stöhrer cells, each 2 ohms resistance, and pass it through the body with 2,000 ohms, we have $\frac{70}{2000+100} = \frac{1}{30}$, if in three minutes the resistance of the body is reduced to 1,500 ohms, we have $\frac{70}{1500+100}$ we have $\frac{1}{23}$ nearly, or a variation of $\frac{1}{98}$ of an ampère in three minutes; while with 50 Daniell cells we have $\frac{50}{2000+1000} = \frac{1}{60}$ and $\frac{50}{1500+1000} = \frac{1}{50}$, or a variation of only $\frac{1}{300}$ of an ampère.

Hence the variation in one case is not $\frac{1}{3}$ as much as it is in the other.

Now as it makes no difference whether the resistance is internal or external, provided it is constant as far as the effects are concerned, it follows that we should reduce the internal resistance as much as possible in constructing a battery for medical uses, and then if we wish to increase the constancy of the current, use a rheostat; or if we want strong currents, dispense with the rheostat and soak

the skin with hot water, or hot brine, before applying the electrodes.

We see, therefore, that we should reduce the resistance of a battery for medical uses as low as possible, and this is especially important for portable batteries, as we want to get strong currents from a small battery.

AFFECTIONS OF THE EYE DEPENDENT UPON HYSTERIA.

By M. LANDESBERG, A.M., M.D.

AS a contribution to the knowledge of the various forms of ocular disorders which are manifested in hysteria, I may be allowed to place on record the most interesting of the many cases which I have met with during my practice:

CASE I.—*Spasm of the accommodative muscle, simulating high degree of myopia. Spasmodic contracture of the internal rectus:*

R., a delicate, pale-looking, and nervous boy of thirteen years of age, was brought to me in order to have his left eye examined, the sight of which had rapidly failed during the last twenty-four hours, after an attack of intense blepharospasm had preceded shortly before. There was nothing abnormal in the external condition of the eyeball. With the naked eye vision was $\frac{6}{200}$, which increased to $\frac{15}{20}$ with the help of concave $4\frac{1}{2}$. The smallest print of the test-tables could be read by holding the book close to the eye, the head turned a little to the left. Field of vision, perception of colors, and background of the eye, were absolutely normal. The right eye showed central leucoma with greatly reduced vision.

The statements of the parents in regard to the development of the eye-trouble were very positive. The sight has always been good, as well for near as distant objects, and when at school the boy could easily distinguish letters or figures from the blackboard at a distance of about 30′. However unique and novel the observation was to me, I had to yield to the evidence and satisfy myself that I had to deal in this instance with a case of sudden development of spasm of the accommodative muscle, which had caused artificial myopia.

Abstaining from all therapeutics, I ordered the patient to present himself the next day for renewed examination, when I was startled by the presence of a new disorder which had taken place

in the meantime. There was a spasmodic contracture of the internal rectus, with total loss of mobility of the eyeball to the left. Only rotary excursions could be effected upwards and downwards. In all other respects the eye remained unchanged. A close inquiry into the general condition of the boy revealed a true history of hysteria with convulsive attacks and spells of barking, coughing, crying, and laughing, of which I had a full exhibition in my office. He had also suffered lately from peculiar twitching movements of the limbs, which yielded to the use of electricity and tonics. An attentive examination of the body showed slight impairment of cutaneous sensibility on the left side of the trunk, with circumscribed areas of total loss of sensibility to pain but not to pressure—and to the differences in temperature. The anæsthesia was mostly marked in the left arm and leg. The sensibility, however, of the affected eye was not disturbed. Testicles were not tender on pressure. No organic lesion of any sort. There was a neurotic tendency in the families of both parents.

The application of different kinds of metals and magnets to the left temple and to the eyeball itself had not the slightest effect upon the condition of the spasm of the accommodative muscle and the contracture of the internal rectus. The latter, however, relaxed entirely after the patient had been brought under the full influence of ether. Coördination in the movements of the eyeballs was fully restored. I fixed the left eyeball in the position of the median line, and steadied it there until the effects of narcosis had fully passed off. When left to itself the eyeball maintained this forced position for over seven hours, after which it gradually returned to the old one of extreme convergence. The spasm of the ciliary muscle, however, seemed to have subsided entirely. The boy could easily read fine print at a distance of from $2\frac{1}{2}''$ to $15''$. Determined by means of the ophthalmoscope, the refraction proved to be absolutely normal. A thorough examination on the next day showed an emmetropic condition of the eye with normal vision and normal power of accommodation. Internal rectus maintained the position of extreme contracture.

CASE 2.—*Accommodative and retinal asthenopia.*

Miss R., twenty-three years of age, consulted me on account of asthenopic troubles, from which she has been suffering for the last few months and which lately aggravated to such a degree that she had to give up her position as school-teacher. She is not able to use her eyes at all. Every attempt to read a few lines only or to look intently at a distant object is punished by the appearance of the most distressing symptoms of neuralgia, by pressure in forehead and temples, and very often by nausea, and occasionally by vomiting. She directs my attention especially to the hammering pain she feels in the occiput, from which it often radiates to the vertex, causing there a feeling of intense pressure and heaviness which sometimes will produce syncope. Vacant look is the most

agreeable condition, and horizontal position with closed lids gives a feeling of intense comfort. Patient speaks with downcast eyes, the lids half-closed. The face is pale and somewhat anxious, telling of much suffering. The figure is slim and delicate, betraying neurotic constitution.

The outward appearance and the associated movements of the eyes are normal. Muscles are in perfect equilibrium. There is not the slightest trace of photophobia or lachrymation. Pupils are of normal shape and reaction. The condition of vision in either eye varies from $\frac{10}{30}$ to $\frac{10}{50}$; it is $\frac{10}{30}$ on the first test and diminishes to $\frac{10}{50}$ during the succeeding ones. Glasses are discarded as doing harm, and patient soon feels so nervous and prostrated that further examination has to be dispensed with. After an absolute rest of twenty-four hours, examination shows V $\frac{10}{10}$, but after this effort the eyes give away and only the medium large print (No. 7 and 6) of Jaeger's test-tables can be indistinctly distinguished. Every further attempt of fixation calls forth clonic spasms of the lids, flashes and streaks of light in the visual field, and a sensation of sea-sickness. When, after half an hour of rest, patient, in order to comply with my urgent request, tries again to read at a distance of 10′ Snellen's test-tables, she shows only V $\frac{10}{70}$ in either eye. The letters are blurred, and she complains of round, bluish spots, which are located just in the centre of the visual field before each eye, moving quickly from right to left, which she had never experienced before. While she was expatiating on this strange phenomenon she at once stopped talking, gasped for breath, pressed both her hands against her heart, and sunk back into the chair, the whole body shaken with convulsions. The eyes were closed, the features distorted, and there was frothing at the mouth. The exhalations of the body were very offensive to my sense of smell. After a few minutes the muscles relaxed, patient opened the eyes, made a deep, sonorous inspiration, and fell into a crying fit. But good spirits were soon recovered, and no exhaustion followed the attack. I subsequently learned that patient has been suffering for the last year from such paroxysms, and from a host of other hysterical manifestations, of which the perversion of smell has been the most predominant. She cannot bear the smell of roses, and takes delight in foul odors. The condition of accommodative and retinal asthenopia was ascertained on repeated examination. Vision was normal when the eyes had rested; but every effort of fixation exhausted at once the energy of the perceptive organ and of the ciliary muscle. There was no error of refraction. Field of vision and perception of colors were normal, and the state of the background of the eye did not allow any suspicion of morbid alteration to be entertained. Monthlies were regular. Uterine troubles were denied.

Country life, housework, and subsequently marriage brought about perfect recovery.

CASE 3.—*Neuralgia of the ciliary body.*

Miss B., twenty-seven years of age, after having suffered for many a year from the most various and distressing symptoms of hysteria, began to complain of a feeling of the most severe pain on the top of her left eye, which aggravates with every movement of the latter, or even by twinkling of the lids. For the most time patient lies motionless in her bed with closed lids, which she raises but very seldom. At first the family physician did not pay much attention to the new symptom, the more so as the outward appearance of the eye did not show any morbid change; but when patient persisted in her complaints, and when no amount of persuasion could induce her to open the eyes which she had kept closed for over a week, I was called in consultation to investigate the matter. According to the statement of the physician, the patient had remained confined to bed for over three months and was suffering for the present from daily attacks of convulsions with transitory paralysis of a leg or an arm, and from anæsthesia of the left side of the trunk. I found the patient of remarkably developed and well-preserved form, lying in bed, face downward, the head buried into the pillows. She could only with great difficulty be persuaded to turn her face, which was somewhat flushed, but no amount of reasoning could induce her to open the lids, which appeared perfectly normal. Pressure upon the right eyeball did not cause any pain, but, when in a moment the attention of the patient was diverted, I slightly touched with the tip of my finger the upper ciliary region of the left eye, patient uttered a sharp, piercing scream, the face became livid, the features distorted, and convulsive tremors ran in rapid succession through the whole body. Consciousness was lost. Examination of either eye during the trance revealed absolutely normal conditions. Pressure upon the left eyeball did not aggravate the paroxysm. The latter was arrested and consciousness restored by continued pressure upon the left ovary.

I subsequently learned from the physician that the eye-trouble passed off spontaneously and as suddenly as it had appeared.

CASE 4.—*Amblyopia, hemianopsia and impairment of color-perception.*

Miss C., thirty-five years of age, applied to me on account of sudden loss of vision in the temporal side of her right eye, which had taken place the day before. Examination showed temporal hemianopsia with irregular line of division, which ran a little to the right of the point of fixation. Central vision, $\frac{10}{100}$. Color-perception greatly impaired for all colors, with exception of red, which was correctly perceived. Shape and action of the pupil regular. Background of the eye normal. Sensibility of the cornea slightly diminished, but almost abolished in the mucous membranes. Marked loss of cutaneous sensibility in the right side of the face and the head, and, as far as I was allowed to

ascertain, in the right arm. Muscular power, however, in the latter was fully preserved. Sensibility in the drum-membrane diminished. Touching with a probe is recognized, but not felt as painful sensation. Hearing power greatly reduced. Sense of smell in the right nostril and that of taste in the right half of the tongue abolished.

Left eye and ear normal.

The nature of this trouble was at once revealed by the general condition of the patient, which plainly told the history of nervousness and hysteria. Born of neuropathic parents, patient has been suffering for the last five years from various hysterical disturbances, and had been confined to bed for about five months, on account of paralysis of the left side, which had suddenly taken place. At this period she was also troubled for a short time with a certain impediment in speech. A year ago her right breast became very tender and painful, and a lump appeared near the nipple which was pronounced by a physician as a malignant growth to be removed by operation. But it spontaneously disappeared in a few days. After a few months of comparatively good health, gastric disorders and frequent vomitings made their appearance, from which she is still suffering. The eyesight has always been good, and patient cannot remember having ever observed a disparity of vision between them.

The effects of Burquism in this instance was most striking. Having found out that copper was the metal to which patient was most sensitive I applied a few small plates of copper to the right temple, fastening them by means of a copper wire around the head. The transfer began twenty-five minutes after the application, and was perfected after fifty-five minutes. A complete exchange had taken place. The left eye and ear showed now the same disturbances which the same organs of the right side had presented before. But there was no transfer of the other morbid alterations, although a slight improvement in the cutaneous sensibility could be noticed. The removal of the copper plates from the temple induced a "retour" of the old conditions, which were re-established in about fifty-five minutes. The only improvement gained was the restitution of the normal sensibility of the eyeball.

I repeated the experiment four times with the same result in regard to restoration of vision and hearing, although the intensity of the transfer to the left diminished with each sitting and failed to take place in the last one. The improvement in the functions of the eye and ear was marked after each sitting, and full recovery was obtained after patient had applied the copper plates to the temple permanently for three days. The other disturbances remained unchanged.

CASE 5.—*Leaping amaurosis.*

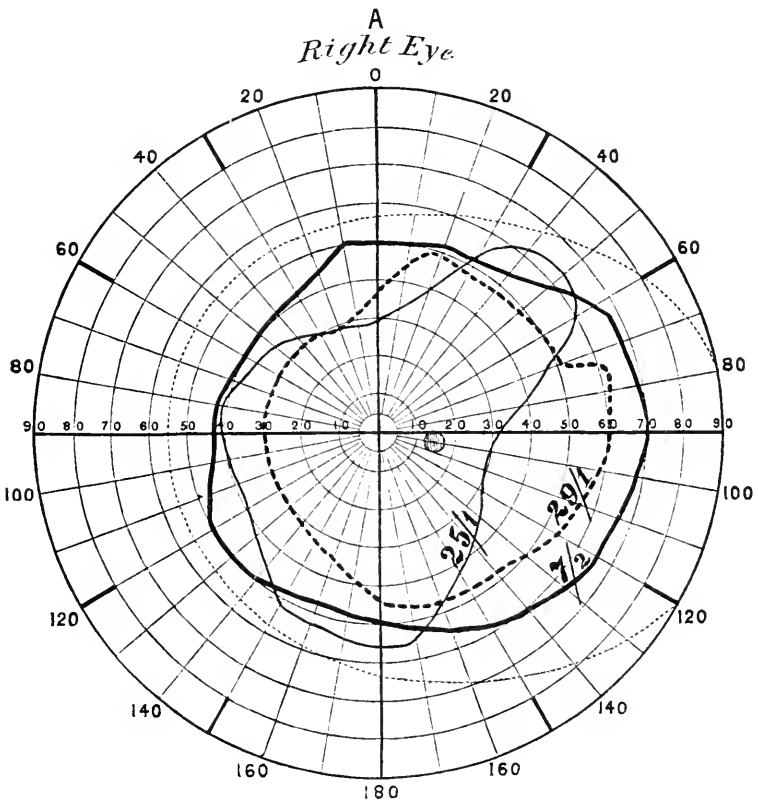
Miss G., thirty-seven years of age, had always enjoyed good sight until five weeks ago, when she was first seized by an attack of blind-

ness in her left eye, which suddenly set in and as suddenly and spontaneously disappeared after half an hour's duration. The attack was neither preceded by nor complicated with any other derangement of the system. Similar attacks of blindness have frequently recurred since, now in one eye and now in the other, but at no time in either eye simultaneously, always preserving the same brisk transition. The duration of blindness varied from about half an hour to twelve hours, with free intervals of from two to seven days. The last attack had occurred in the right eye about two hours before patient had come into my notice. The external appearance of the eye did not betray any morbid change. Movements of the eyeball were regular, and there was no difference whatever between the shape and action of either pupil, each of which fully responded to reflexes. The only abnormal condition was ascertained in the cornea, which showed complete anæsthesia. Background of the eye was absolutely normal. But vision was totally abolished and only dim perception of light remained. Phosphenes could be produced by pressure upon the eyeball. There was no anomaly in the other eye. Suspicion of simulation could not be entertained in this instance. The evidence of a most thorough examination, which excluded any possible source of error, went to show that the transmission of stimuli from the optic nerve to the central organ of vision was in fact suspended for the time. A glance at the general constitution of the patient gave the clue to this strange phenomenon : We had to deal with a person of extremely nervous temperament, who told besides a history of various manifestations of hysteria, to which she has been subjected for many years past. The hysteric troubles patient complained of at the time were : Globus hystericus, crises of vomiting and gastralgia, spells of severe hiccough, and occasionally of convulsions. While engaged to make notes of the case I was startled by the exclamation of the patient that vision had just returned to the eye, which statement was verified by immediate examination. There was complete restitution of all functions in the eye. Only the anæsthesia in the cornea remained. An attack of blindness in the left eye took place after an interval of three days, bearing the same features of sudden development and sudden disappearance. No other morbid changes of any kind were noticed. The repetition of attacks of blindness I had occasion to observe twice in either eye, with complete recovery in every instance.

CASE 6.—*Concentric limitation of the field of vision.*

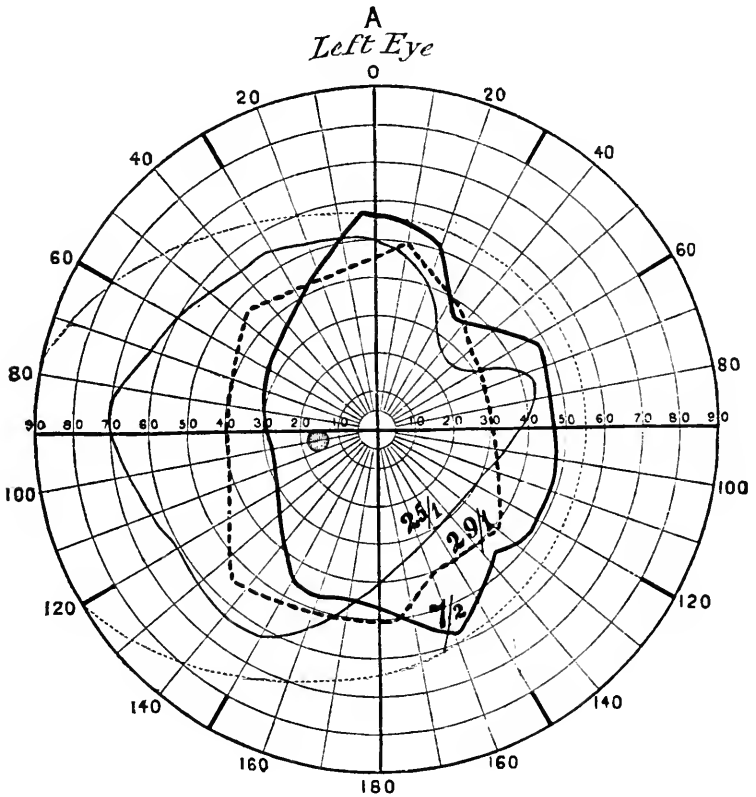
Miss A., twenty-two years of age, applied to me on account of sudden loss of voice, which had developed without any premonitory symptoms a few days previously, and for which no assignable cause could be given. She was unable to speak above a whisper. Respiratory action was normal. Laryngoscopic examination revealed no other morbid changes but paralysis of the adductors of the vocal cords, which was more pronounced in the

right one. Having satisfied myself that I had to do in this instance with a case of hysterical aphonia, to which all anamnestic data pointed, I resolved to try a procedure which, I had heard, had given brilliant results in similar instances. I pushed the laryngeal mirror somewhat harshly down the throat, when patient grasped my arm exclaiming aloud : " O doctor, how you did hurt me ! " The restoration of the voice remained. After patient had recovered somewhat from her astonishment at the speedy cure she had so



unexpectedly experienced, she requested me to have her eyes examined, the sight of which has lately become almost abolished in the peripheric region. She complained of the great difficulty she experiences in walking in the streets, and of the various troubles she has been exposed to by not being able to see what is going on to her right or left. She helps herself by turning the head constantly in either direction, to which my attention had already been attracted. She could not state exactly when the trouble had begun, but it existed already before the loss of voice had occurred. Ex-

amination showed the only anomaly to consist in *concentrical limitation of either field of vision.* All other functions and background of the eyes were absolutely normal. I renewed the test twice, on the 29th of January and 7th of February (repeatedly controlling the results), and ascertained each time the presence of



concentric limitation in the field of vision, the outlines of which varied at each sitting, as it is best illustrated by the diagram on the appended perimetric chart.

The other manifestations in the patient were frequent crying and laughing spells, pain in the occipital region, and great hyperæsthesis of the skin.

Clinical Cases.

VASO-MOTOR AND TROPHIC SYMPTOMS OF CENTRAL NERVOUS ORIGIN.

By M. ALLEN STARR, M.D., PH.D., PROF. OF NERVOUS DISEASES, NEW YORK POLYCLINIC.

Katie S., aged twelve, of healthy parents and good family history, was sent to my clinique by Dr. F. Hartley, January 15, 1885. She had general convulsions occasionally as an infant, but has had none since she was four years old. These convulsions were never followed by paralysis. At the age of four she had whooping-cough; at the age of seven, diphtheria; at the age of nine, measles, and in January, 1884, a severe attack of dysentery, from which she recovered slowly. After her recovery, in the month of March, 1884, she took somewhat violent exercise in a gymnasium to which she was in the habit of going, and on one occasion jumped from a height, jarring herself slightly. It is to this jar that the family ascribe her disease. In June, 1884, she noticed a slight ulceration about the nail of the right thumb, such as might have been caused by a "hangnail." This, instead of healing, extended around the root of the nail on one side, and under the nail, and in a few days similar ulcerations appeared about the nails of the fingers, and also on the fingers of the left hand. There was noticed a considerable degree of venous congestion of the tips of the fingers at this time, together with some swelling of the entire terminal phalanges. After a time the ulceration extended over the tip of the fingers to the palmar surface. During the continuance of the local affection in the hands the skin of the entire body became dry and rough, and her mother noticed that wherever any pressure was made on any part for a time a superficial ulceration occurred which soon scabbed over and gradually healed. For a time during September, 1884, the toes had been ulcerated in a similar manner to the fingers, but these had gradually healed, leaving the feet, however, quite tender, so that she disliked to walk. For the past six months the girl had

been losing flesh and strength, and the eyebrows, eyelashes, and, to some extent, the hair of the scalp had fallen out.

On examination she was found to be pale, anæmic, and poorly nourished, with little fat and flabby muscles. The absence of eyebrows and eyelashes gave the face a peculiar appearance, but its skin was normal. On the tips of several fingers of both hands, and on the thumbs, small ulcers were found of irregular shape, some involving the nail, others on the pulp of the finger. On one finger a small collection of pus beneath the epidermis was ready to rupture. Cicatrices on several of the fingers near the ulcers showed where similar processes had run their course. The ulceration was very superficial, involving the epidermis and upper layers of the corium only, and nowhere going down to the tendons or bone. Both hands were mottled, reddish or purplish in color, the congestion being more marked on the fingers, all phalanges being about equally involved. The fingers were swollen, hard, œdematous, irregularly congested, the mottling being deeper at certain spots. No gangrenous spots were seen, nor could it be ascertained that any had been present. The nails had not ceased to grow, and were not ridged or curved. She suffered some pain in the ulcers, and was prevented by it from using her hands. The feet were congested and mottled, but no ulcerations were found upon them. Both hands and feet were warmer than the rest of the body, and she complained of subjective sensations of heat. Sensation to touch, temperature, and pain were unaffected, the muscular sense was good, the muscles could all be put in action, their electric reactions were normal, and the grasp of the hands was as good as could be expected when the pain produced by pressure on the ulcers was considered. Over the elbows and external malleoli of the ankles dry scabs were found where ulcerations of some extent were healing. Similar crusts were present on two places over the vertebræ. The skin everywhere was dry, rough, and scaly, there being, however, no true eczema or psoriasis. Sensation and motion were nowhere impaired. She suffered slightly from headache, and her appetite was poor. The chief complaint was of the local condition. Physical examination showed heart, lungs, liver, and spleen of normal size, and furnished no evidence of disease in these organs. The urine was passed regularly in sufficient amount, sp. gr. 1021, yellow, no alb., no casts, no sugar.

The symmetrical location of the ulcerations on both hands and both feet, and the evidence from the appearance of the extremities, of vaso-motor and trophic disturbances, unaccompanied by sensory or motor symptoms, were thought to indicate a central lesion in the spinal cord. In the absence of paralysis with atrophy, and in the absence of disturbances of pain and temperature-senses, either directly or in their rate of transmission, it was impossible to consider the anterior or posterior gray horns affected. No symptoms pointed to the implication of any of the tracts surrounding the gray matter of the cord. If the disease lay in the

cord, it must have affected the central gray matter surrounding the canal. Peripheral neuritis was not possible in the absence of sensory and motor symptoms, and of tenderness along the nerves.

The occurrence of symptoms of vaso-motor and trophic character in cases of syringo-myelia, suggested the possibility of this location of the lesion, but the condition was not sufficiently serious to warrant the conclusion that an actual disintegration of tissue had occurred, and hence the case could not be called one of central myelitis.

It did not present the history of symmetrical gangrene, or Raynaud's disease.

The absence of any local cause for the symptoms, and the fact that the anæmic condition had developed in spite of good hygienic surroundings, rather supported the diagnosis.

She remained under observation for four months, during which time her general condition improved somewhat under tonic treatment with iron, arsenic, and cod-liver oil. Local applications did not seem to stimulate the ulcers to heal. Electricity was applied in several ways. Galvanic currents were applied to the spinal cord, to the peripheral nerves, and, finally, through a bath, the anode being placed on the neck, and the cathode in a basin of water, in which the hands were held. None of these methods seemed to produce any appreciable effect. Faradism was so painful that it could not be borne, and was not thoroughly applied. The local condition not improving after continued electrical treatment, she ceased to attend.

The case is reported as being a rare condition of vaso-motor and trophic disturbance. The only case which I can find at all resembling it, is one reported by C. K. Mills, in the *Amer. Jour. of Med. Science*, 1878, Oct.

Periscope.

PHYSIOLOGY (INCLUDING EXPERIMENTAL PHYSIOLOGY) OF NERVOUS SYSTEM.

On the Nature of Glomerular Activity in the Kidney. By J. G. ADAMI, *Foster's Journal*, vol. vi., No. 6.—As is well known the secretory activity of the kidney has been explained in two ways: the one, the mechanical theory; the other, the vital theory. Mr. Adami has attempted by a series of experiments to arrive nearer the truth about the matter. Nussbaum states that fishes, batrachia, ophidia, have a double renal supply: first, renal arteries running specially to the glomeruli; second, by the vena renalis advehens, or renal portal vein and the lumbar veins.

These, by their branches, aided by the vasa efferentia from the glomeruli, form the capillary network around the tubuli uriniferi. Hence, if, in the frog or newt, the renal arteries are ligatured, circulation through the glomeruli is wholly stopped. Hence, in these animals we would seem to have a means of determining the place of secretion of normal and abnormal constituents of the urine; and taking advantage of this arrangement. Nussbaum endeavored to determine the constituents of the urine which are secreted by the glomeruli and by the convoluted tubes respectively. He found that when the arteries were ligatured sugar, peptone, egg-albumin, and carmine, injected into the vascular system, did not appear in the urine, this being contrary to what is observable in the normal animal; on the other hand, urea similarly injected into the ligatured animal, still made its appearance in the urine. Hence he concluded that sugar, peptone, egg-albumen, and carmine, when present in the urine, have been passed out through the glomeruli, whereas urea is secreted through the cells of the tubules. Mr. Adami found that after closure of the renal arteries the glomeruli are not completely cut off from the circulation. Judging from his experiment with laky blood, the glomerular activity is not dependent on blood pressure, but on secretory activity; that the hypothesis of Heidenhain is correct; that the glomerular epithelium, the epithelial capsule ensheathing the Malpighian knot of vessels, has powers of a selective secre-

tory nature. Nussbaum's assumptions as to the nature of the renal secretion are based, as stated above, upon the premise that after ligature of the arteries the Malpighian vascular knots are cut off from the circulation. Adami having found that in spite of breaking off the arterial supply, blood would still seem to pass through at least some of the glomeruli, the question remains whether the conclusions that Nussbaum has drawn from the urea researches are correct? According to Heidenhain the capsule secretes water owing to its own iridescent secretory activity. This activity is excited by urea, which must pass with the blood to some of the glomeruli; hence the increased secretion of water by Nussbaum. Adami also found that diuretics, as sodium nitrate, owe their diuretic effect in part, if not wholly, to direct action on the glomerular epithelium, and not, as Nussbaum has thought to have shown and Heidenhain has considered possible, to its influence upon the cells of the tubuli.

Plethysmographic and Vaso-Motor Experiments with Frogs. By F. H. ELLIS, *Journal of Phys., Foster*, vol. vi., No. 6.—Drs. Bowditch and Warren have shown in the cat, by means of the plethysmograph, that slow, rhythmical, electrical stimulation of the peripheral end of the cut sciatic nerve of the curarized cat causes an immediate dilatation of the vessels of the leg evidenced by a rise in the plethysmographic tracing, while shocks of a certain greater degree of frequency cause an immediate contraction. Dr. Ellis has studied similar effects upon frogs. The results of Dr. Ellis' experiments point to the existence of a vaso-dilator as well as a vaso-constrictor mechanism in the frog. The vaso-motor mechanism is not so active in cold-blooded animals as in warm, and therefore he is not surprised to obtain dilatation in the vessels of the leg of a frog with greater difficulty than was experienced by Bowditch and Warren in producing the same result in the leg of the cat.

Spinal Vaso-Motor Nerves. By Dr. WATERS, *Foster's Journal*, vol. vi., No. 6.

Dr. Waters has made a series of experiments upon the vaso-motor functions of the spinal nerves of the frog. He wished to ascertain whether the distribution of vaso-motor nerves in the various regions of the frog's alimentary canal bore any distinct relation to the different spinal nerves as they are given off from the spinal cord.

From the results thus obtained, I feel justified in concluding that the blood-vessels in separate areas of the alimentary canal are governed by vaso-constrictor fibres, which travel along distinct and determinable spinal nerves, each spinal nerve conveying fibres for a certain area. ISAAC OTT.

On the Mechanical and Electrical Excitability of the Cerebral Cortex of Man through the Cranial Walls. By Dr. B. SILVA, *Rivista Clinica*, Dec., 1885.

Dr. Silva, in this and in a preceding paper, calls the attention to a new phenomenon observed by him, to which he has given the name "*fenomeno Rolandico.*" The phenomenon of Rolando is the muscular contraction of a part which is excited by percussion through the cranium of the pyscho-motor centres, and also by the use of electricity. For example, by the stroke of a small hammer upon a pleximeter of the left temporal region, corresponding to the cortical centre of the upper extremity, there will be produced a slight movement of the forearm upon the arm, and contemporaneously a slight movement of pronation of the forearm with the adduction of the thumb. If percussion is employed over the centres for the lower extremity, there will be a slight contraction of the quadriceps extensor femoris, of the tibialis anticus, and a less pronounced movement of the gastrocnemius. The excitation is proportioned to the extent of the blow, and on the opposite side of the body from the side of the cranium percussed.

The same phenomenon is observed with the faradic or galvanic current. The writer has demonstrated by means of myograms, which accompany the article, that there is always a latent period 0.15″ in length. GRACE PECKHAM.

Compulsory Movements Following Destruction of Cortex. By PROF. W. BECHTEREW, of St. Petersburg. *Virchow's Arch.*, vol. c., p. 473, 1885.

Prof. Bechterew is quite right in declaring that although Goltz, Trepier, and others, observed circus-movements and other forced movements following upon experimental destruction of certain cortical areas, but little attention had been paid to the subject hitherto. By physiological experiments Prof. Bechterew claims to have proven that circus-movements follow upon the destruction of an area corresponding to the parietal convolutions of man, and that such forced movements are similar to those which are produced by destruction of the organs of equilibrium. The author believes furthermore that these compulsory movements are irritation-symptoms, and may therefore be produced by destruction of the neighboring motor areas. Bechterew thinks (and he bases this opinion upon Flechsig's investigations) that there is good anatomical reason for assigning circus-movements to this area just caudad of the motor convolutions, for the superior peduncles joining the cerebellum and the cerebrum are seen to terminate in this parietal region of the cortex, and we know that destruction of the fibres of the superior cerebellar peduncles is followed by similar forced movements. The author cites in conclusion a number of pathological cases in which forced movements have been a prominent symptom, and refers in detail to a case which he himself had occasion to observe. The patient, a man of fifty-

four, had received some years ago a blow upon the head; there was a distinct scar on the left side of the skull on the border between the parietal and occipital bones. The patient exhibited symptoms of dementia when he came under observation. He was seized suddenly with circus-movements; he would always turn from right to left on being lifted out of bed and whenever he attempted any movement. In the sitting position the trunk and head would be turned several times in the direction which the circus-movements always took; in the recumbent position these movements would not occur. He died of pleurisy a few months after these symptoms set in. The autopsy revealed local encephalitis of the parietal region, and an otherwise normal cortex, with the exception of a slight atrophy of the frontal convolution. This area of disease corresponded closely enough to the region which for physiological and anatomical reasons had been held responsible for these forced movements. B. S.

Recherches Expérimentales sur le Trembiement dépendant de l' Ecorce grise des Hémisphéres du Cerveau. By J. GASTERNATZVY. *Progrès Mèdical*, 536, 1885.

In a former article published in 1881 G. showed that a lesion of a certain part of the antero-lateral columns of the spinal cord could produce a tremor analogous to that observed in disseminated sclerosis. In the present article he studies another kind of tremor, which cannot be produced by the above lesion,—a tremor dependent upon a lesion of certain parts of the brain. His experiments consisted in carefully applying a faradic current to the psychomotor zone of a dog. The conclusions arrived at were, that chloroform reduces the excitability of the cortex of the cerebral hemispheres and that then excitation of the psychomotor centres produces a tremor analogous to that observed in progressive paralysis of the insane. Various clinical features of this affection are then cited for the purpose of showing the similarity of the two tremors. G. W. JACOBY.

PATHOLOGY OF NERVOUS SYSTEM.

On a Peculiar Group of Symptoms Associated with Disease of the Posterior Columns of the Spinal Cord. By Prof. C. WESTPHAL. *Archiv. für Psychiatrie*, vol. xvi., pp. 498, 778, 1885.

The above case of Prof. Westphal is remarkable not only for the interest inherent in the case itself, but for the masterly analysis of all the symptoms. While some of the symptoms resembled those of multiple sclerosis, there were others which excluded the affection from any of the established forms of spinal-cord diseases. We cannot do better than to give the author's summary of the clinical and pathological aspects of the case.

The patient, a man æt. forty-seven, without any syphilitic or

alcoholic taint, was under observation from November 24, 1881, to Feb. 26, 1884, when he died of pneunomia complicating tuberculous disease of the lungs. The disease set in with paresis of one ocular muscle (the left rectus int.) followed somewhat later by ptosis and some general cerebral symptoms (vertigo). Other symptoms were: A gradually increasing weakness of the lower extremities, which terminated in complete paralysis, and a slight weakness of the upper extremities; muscular rigidity affecting certain groups of muscles in the lower extremities; exaggerated knee-jerks and paradoxical contraction; this last phenomenon being observed at first only after dorsal flexion of the foot, and later on after plantar flexion and movements of the knee and hip-joints. In the upper extremities also there was a gradual increase of motor paralysis, certain movements of the fingers were entirely impossible, and there was moderate rigidity attending some movements as well as paradoxical contractions. The muscles of the jaws were similarly affected (at a later stage),the facial muscles were not distinctly paralyzed, but the movements of the tongue which deviated to the right were seriously interfered with. There was marked anæsthesia extending by degrees over the entire body, and finally including the trigeminal region. Cutaneous reflexes were preserved; with the exception of vertigo, fear, and slight insomnia there were no cerebral symptoms of any special import. Toward the latter stages of the disease, some mental impairment was noticeable. Lancinating pains are mentioned in the earlier history of the case.

The autopsy proved a great surprise. The seat of the trouble was found to have been mainly in the posterior columns of the spinal cord. In the upper portion of the cervical cord the disease was limited to the inner portions of the columns of Goll and to the boundary between these and the columns of Burdach. Lower down in the cord the area of degeneration approaches to the posterior roots, without, however, quitting the columns of Goll. In the upper dorsal region this area is greatest; in the middle dorsal region the area of degeneration is narrowed down very much, and is limited to the immediate vicinity of the posterior fissure; a new strip of degeneration is formed in the lower dorsal region. We need not report all the minute changes; it will suffice to refer to this one additional fact that on the boundary line between the dorsal and lumbar segments there was no degeneration in the vicinity of the posterior root zones. In addition to this affection of the posterior columns, there is but a very slight diseased area in the antero-lateral columns of the cervical cord. A number of peripheral nerves (sciatic, crural, tibial, etc.) exhibited varying degrees of atrophy of the nerve-fibres without marked thickening of the interstitial connective tissue. Westphal believes also that he found distinct atrophy of a number of nerve-fibres of the posterior roots of the lumbar segment. Nothing new was gained from an examination of the muscles.—We may close this report with a reference to some points in differential diag-

nosis and to the manner in which the author now accounts for the various symptoms. The rapid spreading of anæsthesia from the extremities over the entire body, and involving even the upper extremities, is peculiar to this affection and not to tabes. The knee-jerks were not absent in this case, because the degeneration did not extend into the lower dorsal and upper lumbar segments, and did not affect the posterior root zones. Westphal finds it easy to explain the absence of ataxia, for as he has observed in many cases of dementia paralytica complicated with sclerosis of the posterior columns, a slight amount of degeneration is not sufficient to produce marked ataxia. The changes in peripheral nerves are held responsible for the sensory and motor disturbances. No attempt is made to account for the paradoxical contraction, or to answer the query whether there was any relation between the tubercular diathesis and this affection of the central and peripheral nervous system.

A Case of Locomotor Ataxy, without Disease of the Posterior Columns of the Spinal Cord. By A. HUGHES BENNETT, M.D. (Reprint from vol. xviii. of the Clinical Society's Transactions).

This case, reported by Bennett, is in curious contrast to the one of Westphal, reviewed above. The patient was a gamekeeper, æt. forty-eight, who had always been healthy, free from syphilis or other disorder. Fifteen years ago severe pains in the legs, continuing for three months, from which he completely recovered. Nine months before he came under observation he experienced gradual weakness of the legs, so that he was easily fatigued and unable to do a full day's work. Some months afterwards, severe shooting pains in lower extremities ; these were paroxysmal, and shifted from place to place. No headaches or other cerebral symptoms, except giddiness. Vision good ; no paralysis of ocular muscles, and fundi of both eyes were normal. Voluntary movements of eyeballs were accompanied with slight nystagmus ; left pupil somewhat smaller than right ; both pupils reacted well ; functions of bladder, rectum, and sexual organs were intact. The patient walked with a slow, unsteady, hesitating, and markedly ataxic gait. Romberg's symptom very pronounced. Knee-jerk on both sides absent ; plantar reflexes diminished. Sensibility to touch and pain everywhere normal. Dull aching pains in back and lower limbs, with shifting lancinating pains ; muscles spare throughout body, but not atrophied. Weakness of legs and insecurity of gait increased, so that patient could not stand. For many weeks there were attacks of uncontrollable vomiting. He was under observation for two and a half months. During the last week patient became semi-comatose, and died after a severe convulsive attack.

At the autopsy, there was found in the substance of each frontal lobe, involving the anterior cornua of the lateral ventricle, a cir-

cumscribed patch of softening on the left side, about the size of a hen's egg, on the right somewhat smaller ; a similar patch of softening in cerebellum—all of these of recent origin. On opening the spinal canal, vessels of the membranes were found deeply congested, especially in the lower dorsal region, where there were small patches of superficial hemorrhage ; the cord was enucleated without difficulty. The pia mater was studded on the dorsal surface with small *sarcomatous tumors*. In the middle dorsal region, there were perhaps six to eight of these tumors to the square inch ; in the lumbar region they became quite confluent. On microscopical examination the neoplasm was seen to embrace the posterior and, to a slight extent, the anterior roots ; in the dorsal region the posterior roots only ; in the cervical region the cell growth had greatly diminished in extent, and did not involve any of the nerve roots. Under high power it was determined that the posterior roots had suffered but little ; there was only an unusual swelling of the white substance of Schwann. (It is a matter of regret that the peripheral nerves were not more carefully examined, and that so little is said regarding the appearances of the posterior columns, though we are willing to believe, on Dr. Bennett's authority, that there were no changes whatever in these.—Rep.) The tumor had insinuated itself into the upper part of the medulla, displacing, without destroying, the healthy tissues. No bulbar symptoms during life.

We agree with the author in thinking that symptoms very similar to those of sclerosis of the posterior columns may follow interruption of the co-ordinating paths in another locality, namely, in the posterior nerve roots.

Prof. Westphal's and Bennett's cases would go to show that there are marked exceptions to the ordinary clinical symptoms which we have been in the habit of associating with disease of the posterior columns. B. S.

Fracture du Rachis. D. MOLLIÈRE. *Gazette des Hôpitaux*, p. 1058, 1885.

Patient male, æt. forty-five, mason ; fell while intoxicated from a first story, striking upon the vertex, September 20th. He was able at once, with the aid of two neighbors, to go to his dwelling. The same evening he was brought to the hospital. Could answer all questions ; temperatere increased ; pulse 108 ; respiration rapid ; no change in pupils ; no priapism ; no trouble with bladder or rectum ; paraplegia of inferior extremities ; no disorder of sensation ; pressure along the spinal column reveals a painful spot over the seventh cervical vertebra.

September 21st.—Same condition ; temperature 40.3°; evening, 40.4°.

September 22d.—Axillary temperature 40.4 ; respiration short, frequent, and superficial. Death at 11 A.M.

Autopsy.—Fracture of the fifth and sixth cervical vertebræ ; compression of the cord.

Remarks.—The interesting points are: 1. The paraplegia occurring four hours after the traumatic lesion and limited entirely to the inferior extremities. The medullary compression cannot account for this, because the compression was localized at the cervical enlargement, and the superior extremities showed neither paralysis nor anæsthesia. The last dorsal and sacral vertebræ were intact. There was no exudation into the spinal canal and no lesion of any vessel, therefore a slow compression must also be excluded. The rapid rise in temperature without any inflammatory process, makes the author think of a lesion of the thermic centres.

Pied-Bot Tabetique. JOFFROY. *Gazette des Hôpitaux*, p. 1060, 1885.

A communication relating to a deformation of the feet occurring in certain ataxics, and which is called "tabetic club-foot." The foot is in a state of more or less pronounced extension, the toes of both feet pointing towards each other. The patient is able to flex the feet without effort. J. has observed this deformity to a slight degree in a woman who had been confined to her bed for two months. In another woman, who had been in bed for two years, the deformity was well marked on the right foot, but only slightly upon the left. Symmetrical development is the rule in this affection, but upon one side it is generally more developed than upon the other. This is particularly noticeable in patients who have kept to their bed for several years. Some authors attribute its production to the weight of the bedclothes, but J. considers this to be only of secondary importance, and believes it to be a true club-foot, caused by inaction of the muscles and loss of sensation. If taken early in its course, the affection is remediable, but later nothing is to be done. G. W. JACOBY.

Eight Cases of Epilepsy Caused by the Sight of a Dead Body. By LEGRANDE DU SAULLE. *Gaz. des Hôp*, June, 1885. Quoted in *Lo Sperimentale*, December.

In examining the usual causes of epilepsy in their medico-legal bearing, eight cases were discovered of children who had epilepsy because of seeing a corpse.

1. Child ten years of age, daughter of an hysterical mother, a drinker, seeing the mother when dead, had an attack of epilepsy which continued daily.

2. A child of ten, daughter of a father addicted to alcohol, when six years of age saw the corpse of her father. She became enuretic, then epileptic.

3. A child twelve years of age, daughter of a father who was a drinker, of a mother lacking in intelligence and probably hysterical, had a brother who died of convulsions, saw a dead body and became epileptic, with distress in epigastrium.

4. A girl of fifteen, daughter of a drunken father, niece of an

insane aunt, had convulsions just after infancy. Had an epileptic attack when she saw her father after death; had an epileptic crisis; manifested thievish propensities and violence toward children.

5. A girl, seven years old, saw her father's corpse, and became epileptic, with weekly attacks.

6. A young woman, twenty-one, had a most dreadful fright. Her father fell from a roof and was killed before her eyes. She had a convulsive crisis, and gradually these increased to five or six each month.

7. A woman, twenty-three, saw, when ten years old, a dead man, after which she had attacks of nocturnal epilepsy; became strange and impulsive.

8. A woman twenty-five years old saw and embraced her father when dead. Immediately she had an epileptic attack. These occurred each month. She became enuretic.

On General Pseudo-Paralysis by Slow Intoxication from the Oxide of Carbon. By Dr. GIUSEPPE MUSSO. *Revista Clinica*, August, 1885.

The writer of this article compares the slow poisoning by the fumes of carbonic di-oxide to alcoholic, syphilitic, and lead poisoning, claiming that it presents as definite a clinical picture as either of these three in their constitutional effects upon the system. To support his opinion he reports five cases and arrives at the following conclusions: That persons who are shut up for many hours in a day during a time varying from a few weeks to two or three months, breathing an atmosphere vitiated by the fumes of carbon, develop serious alterations of the sensibility of mobility of the intellect and of the vaso-motor innervation.

There is presented a complexity of symptoms which resemble very closely those of general progressive paralysis. In those afflicted with this disease the etiological factors, the clinical aspect, the possibility of healing, show that the process is neither a pathological nor inflammatory nor degenerative one, but that it attacks directly the nerve centres and is an intoxication of these.

Of the chronic poisoning by the gases of carbon, Dr. Moreau wrote in 1876. He thus describes the symptoms with which those cited by Dr. Musso coincide.

The first period, which is compatible with a certain degree of physical and psychical health, is characterized by a heaviness of the head, intense cephalalgia, sense of pressure about the temples, singing in the ears, flashes of light in the eyes, phenomena which occur only when the patient is exposed to the carbon vapors, and which disappear after breathing a normal atmosphere.

After a variable time, according to the person and circumstances, the patient enters the second phase. To the above symptoms are added hallucinations and the delirium of persecution. The latter is vague, undecisive, and changeable. The hal-

lucinations are generally auditory or pertaining to the vision. Exceptionally, olfactory or gustatory.

The disease is curable if the patient is removed from the vitiated atmosphere, but more often it degenerates into an incurable dementia.

The question will arise with the reader if the author is not dealing with veritable cases of general paralysis caused by breathing an atmosphere of gases of carbon, rather than a distinctive disease, as Dr. Musso would have us believe. However this may be, we are indebted to him for calling the attention to these cases, and the possibility of such a condition arising in such an atmosphere.

Of the five cases reported, two recovered; the other three died. One occurred in a woman, the others in men who were bakers and shut up in small, close rooms with fires of charcoal.

GRACE PECKHAM.

Reflex Neuroses from Nasal Disease.—*Med. Record*, Jan 30, 1886.

An interesting discussion on this subject was lately introduced in the New York Academy of Medicine, by a paper by Dr. E. Gruening, on the "Ocular Symptoms in Nasal Affections." Following in the wake of Hack, Fraenkel, Sommerbrodt, and others, who have demonstrated a connection between many neuroses and disease of the mucous membrane of the inferior turbinated bone, Dr. Gruening thought that a certain group of ocular symptoms, namely, lachrymation, sensitiveness to light, and redness of the eyes, which are frequently met with and have resisted all ordinary treatment, might be dependent upon the same diseased conditions, especially as the same symptoms can be produced at will in all eyes by mechanical irritation of the nasal mucous membrane. In his paper he reports a series of such cases, a few of the large number he has met with, all cured by treatment of the nasal disease.

Dr. Thomas A. McBride reported five cases of reflex disturbance (hemicrania, headache, neuralgia) arising from nasal disease, and cured by treatment of the latter. Dr. Beverly Robinson followed, dwelling at some length on the connection between hay-fever and asthma and nasal disease. It will be remembered that Hack, of Freiburg, originally pointed out the connection between migraine, asthma, hay-fever, supra- and infra-orbital neuralgia, and other neuroses to disease of the covering of the inferior turbinated bone. Since then his results have been confirmed by a number of clinicians. Hack reported 240 cases of hemicrania and 87 of asthma cured by destroying the erectile tissue over the inferior turbinated bone with the galvano-cautery. Although experience shows that the nasal iritation may be due to various forms of nasal disease, the most common is a peculiar swollen, puffed-up (not hyperplastic) condition of the covering of the in-

ferior turbinated bone, due to distension of the underlying erectile tissue. A most important point also is the fact that the nasal symptoms may be so slight as not to call attention to the nose.

MORTON PRINCE.

MENTAL PATHOLOGY.

Moral Imbecility.—Dr. Hack Tuke (*Journal of Mental Science*, Oct., 1885) reports a case of a forty-two-year-old man who, from an early age, had been purposelessly cruel to animals and young children. He had made one successful assault on young girls and several unsuccessful attempts. He had been guilty of several minor and major crimes. "The very sight of blood had a strange effect on the man, and worked a wonderful transformation. His countenance assumed a pallid hue; he became nervous and restless, and unless he was where he could be watched, he lost control of himself and indulged in the proclivities for which he was notorious." Commenting on this case, Dr. Tuke says: "Is it not true, then, that men are born with organizations which prompt them to the commission of acts like those committed by this unfortunate, and that the lower instincts are in abnormal force, or the controlling power is weak? Such a man as this is a reversion to an old savage type, and is born by accident in the wrong century." In the discussion of this case by the Medical Psychological Association, Dr. Rayner said that moral insanity might be a persistent state persisting through the whole lifetime, but might also exist in the development of any psychosis. Dr. Chas. Cameron said that Dr. Tuke's case proved that moral attributes might be absent from a mind otherwise well endowed. Dr. Nugent was of opinion that cases of the type described were best designated as moral imbecility. Dr. Savage was of the like opinion. Dr. Tuke in closing the discussion agreed that such cases were best designated as moral imbecility.

Hæmaturia and Purpuric Extravasations in Typhomania.—Dr. Geo. H. Savage (*Jour. of Mental Science*, Jan., 1886) reports a case of, what seems from the history to have been, typhomania (Bell's disease) which was associated with purpuric extravasations resembling bruises and hæmaturia. Ten days before admission to Bethlem Hospital the patient had pain in back of head and neck; he then became emotional and depressed, and the same evening was restless, which restlessness passed rapidly into mania. He became incoherent and violent, and had delusions that his wife was insane, his nurse wanted to poison him, and that he had waded through the shining river. On autopsy hæmorrhagic pachymeningitis was found, evidently of recent origin.

Imperative Conceptions.—Dr. H. C. Wood (*Medical and Surgical Reporter*, Jan. 23, 1886) reports the following cases of imperative conceptions. One was in a man who, when a boy of eight or nine, going to school, commenced to put up the lapel of his coat, "hiking" it up as it were; he was reasoned with and punished for this action, but all to no purpose. He next commenced to rub up and down the sides of his coat with the coat sleeves; this practice lasted for years. Then came the imperative conception that he was in danger of personal contamination, and he could not be induced to touch the outside of his coat; some one else had to take it off for him. He then got the idea that he could never complete an act. He is rational, his memory is good, and he attends actively to business. He reasons about his condition, recognizes the absurdity of it, but argues his inability to resist. If he lays down a sheet of paper on the table, he must get it straight, and *it never is straight;* thus, for hours he will fool with this sheet of paper, never satisfied that he has it right. He will often consume two hours at night getting his coat off (without touching the outside) and getting it *straight* on the chair. He realizes his infirmity, and claims that he resists it, but compares this resistance to one endeavoring not to breathe; he is ultimately *forced* to yield. The most common form of this affection is the fear of contamination by filth. The second case occurred in a female who will have no money about her save crisp, clean, new bank-notes; she will not touch her outside dress, and is continually washing her hands; she knows they are not dirty, but cannot control this impulse. She would starve to death in a room rather than touch a knob to open the door. On one occasion, when giving her a prescription, he laid the paper on the arm of her chair (to test her); she would not touch it, for "you know, doctor, so many persons touch this chair." Meeting her on the street one day, he extended his hand; with profuse apologies, she declined to shake his hand, on the score of contamination. Dr. Wood told the man who could not touch the outside of his coat for fear of contamination, that if he were in his place, he would get on the wildest horse he could find, and he was quite sure that when the horse commenced to rear and kick he would grab hold of him, no matter how dirty the horse might be. The remedy was not tried. And such a remedy would not only not act longer than the man was on the horse, but would thereafter increase the disease.

Early Stages of Dementia. — Dr. Henry Sutherland (*Lancet*, Dec. 24, 1885) cites the following as symptoms precedent to paretic dementia. An inability to speak, write, walk, sing, read, or fix the attention as well as usual; a feeling when conversing with others that a blank had taken possession of the mind, rendering the patient unable for the moment to answer a simple question; a sensation of great fatigue

after a short walk, and of utter prostration after any unusual exertion; a feeling of "pins and needles" in the feet and knees without adequate cause, such as pressure upon the sciatic nerves and popliteal arteries, or exposure to cold; an inability to adjust the iris as rapidly as usual after looking at a near object and then turning suddenly to one more distant; a fixed squint, lasting several minutes, unaccompanied by loss of consciousness or epileptiform attacks, but yet causing much alarm and distress to the patient; a sensation as if hot irons were dragged along the forearms; sexual desire and erectile power much diminished, or morbid, erotic desires toward women, especially in cases where the symptoms have been caused by a blow on the head or spine; tremor of the feet in performing simple actions, such as getting out of cabs or walking down stairs; tremor in the hands in writing or any work requiring delicate manipulation; slight vertigo; profound depression without adequate cause, occasionally succeed by inappropriate outbursts of merriment; nervousness and a love of solitude, yet accompanied by a dread of being alone; a general feeling of malaise which cannot be accounted for, succeeded, if the disease be not checked, by the comfortable sensations of advanced paretic dementia.

Race and Paretic Dementia.—Dr. J. G. Kiernan (*Alienist and Neurolog.*, Jan., 1886) says that there came under his observation in the Cook County Hospital for the Insane 921 cases of insanity, of which 83, or nine per cent., were cases of paretic dementia. The races attacked by paretic dementia were as follows:

RACES.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
German	11	1	12
" American	1		1
" Austrian	1		1
" Swiss	1		1
Hollanders	4	1	5
Anglo-Saxons, American	17	1	18
" " English	3		3
" " Scotch	1		1
Scandinavian, Danish	1	1	2
" Swedish	2	2	4
" Norwegian	1		1
Celtic.			
Irish	17	7	24
Latin.			
French	1		1
French-Canadian	2		2
Sclavonic.			
Polish	1		1
SCHEMITIC.			
Hebrew	1		1
NEGRO	5		5
	70	13	83

The proportion of Irish attacked by paretic dementia is much greater in Cook County than in New York City, and this arises from the Irish in Chicago being much more addicted to speculation than those in New York. To his personal knowledge, Irish women who, in New York, would simply hoard their money and keep away from any speculation, here deal in options on wheat and pork.

Transitory Insanity. Dr. C. H. HUGHES, (*Alienist and Neurologist*, October, 1885) states that the question of transitory insanity is simply a question of how acute insanity may be, and the pathological possibilities of the brain are no more tardy than its physiological; insanity may come, as it often ceases, suddenly. A psychical spasm may be as transient as a reflex act of the spinal cord. It may come as quickly as an urticaria follows gastric irritation, and need abide no longer or not so long.

Definition of Insanity. Dr. H. C. WOOD (*Medical and Surgical Reporter*, Jan. 23, 1886) would define insanity as "a diseased condition of the mental functions, in which the disease has progressed so far as to be beyond the power or will of the patient to control the manifestations"; provided such loss of control is not due to some poison in the blood. The *raison d'être* of this definition may be apparent to Mr. Wood. It is not to any one else. If there be disease of the mental faculties, there is insanity, whether such disease manifestations be under control or not. In nearly every type of insanity, except the dementias, the patient can, at times, control the manifestations, a fact of which Dr. Wood seems to be ignorant. This definition seems intended to pander to the lawyers' ideas of insanity.

Suicide. Dr. J. S. CONRAD (*Maryland Medical Journal*, Nov., 1885) concludes respecting suicide: 1st. Suicide increases with the advance of civilization, and is but little known in the savage state of men. 2d. The act is an intelligent act (?), done with full consciousness of the act—as shown by the method of execution,—whether by the sane or insane. 3d. That suicide is done always for the purpose of escaping an evil, and for the benefit of the *felo-de-se*—whether by sane or insane. 4th. That it is a voluntary act (?),—whether by sane or insane. 5th. That it is an emotional act, whether by sane or insane—however deliberately planned and executed—since deliberation enters into the mind of both mental states. 6th. That delusions are not essential to the distinction as to the sanity or insanity of the suicide, since authorities affirm that delusions are not essential to the proof of insanity. 7th. That suicide is rare in the first class.—insanity (by Maudsley), viz.: intellectual or ideational insanity; but does occur in the vast majority of the second class—or

affective or emotional forms of insanity. 8th. Is suicide an intellectual act—notwithstanding the intelligence exercised in its execution? or is it an emotional act *per se*, since we have seen that the emotional part of mind dominates the ideational centres, and perverts the mind into becoming its humble servant? 9th. Does moral depravity satisfactorily account for it, when we have seen that moral depravity is a factor of both sane and insane? 10th. That in doubtful cases of the sanity or insanity of the *felo-de-se*, very great caution is necessary in making up a just judgment as to the one or the other. In making the second, third, and fourth conclusions Dr. Conrad ignores the cases in which suicide is committed under the influence of imperative conceptions and epileptic psychoses.

Consciousness in Melancholic Frenzy. Dr. H. R. Stedman, (*Alienist and Neurologist*, Jan., 1886) reports a case in which an epileptic melancholiac wounded, with homicidal intent, her son, and, on seeing the blood flow, lost consciousness and then continued, automatically, the act once begun, with the result of cutting his head off and seriously wounded two other children as well as himself. The case in itself is not exceptional, but in the course of its discussion before the New England Society, the question was incidentally raised whether absence of consciousness, proved the epileptic nature of an act. Dr. Fisher said that there might be complete consciousness in melancholic frenzy. That in melancholic frenzy consciousness is seemingly abolished, there can be no doubt, but, the patient is under the sway of a delusion, but this condition differs psychologically from that of the epileptic, who performs his acts automatically, although, for practical forensic purposes, seemingly the same. J. G. KIERNAN.

THERAPEUTICS.

The Action and Uses of Digitalis and its Substitutes, with Special Reference to Strophanthus. By Dr. Thomas Frazer (*Br. Med. Jour.*, Nov. 14, 1885). The author read a paper before the British Medical Association, which promises to be one of the most valuable contributions to therapeutics. This new drug, which Dr. Frazer has carefully studied, belongs to the digitalis group. It is a muscle poison in sufficient quantity and acts upon all striped muscles, increasing their contractile power. It acts on the heart more powerfully than on all other muscles, and in doses which produce no effect on other muscles. Its action on the heart is the same as that of digitalis, only more powerful-but it differs from digitalis in having very little effect in contract, ing the blood-vessels. Dr. Frazer then reported a number of extreme cases of heart disease, to which he gave strophanthus. Sphygmographic tracings are given showing plainly the effect of

the drug. In all the cases before treatment, the pulse-curves are represented simply by irregular wavy lines, and sometimes the pulse beats could not be counted. The tracings show marked and rapid improvement. The shortest time after giving the drug before improvement was noticed, was in a case where the pulse was so feeble and rapid as to be almost uncountable, $\frac{1}{50}$ gr. of strophantus was given subcutaneously. Improvement was shown in 20 minutes, and the effect of a simple dose lasted 24 hours, when the continuation of the effect of a single dose could not be noticed, as the drug then was given by the mouth.

On the Influence of Age on the Dosage of Nux Vomica, with Some Remarks on its Therapeuties. Dr. J. H. Musser (*Therap. Gaz.*, Jan. 15, 1885) states that the effect of strychnia upon the system varies with the age. From a study of his notes of 50 cases, he states that from 15 to 40 years of age, 45 drops or more of the tincture was almost invariably well borne. After 40 years it was the exception to be able to increase the dose over 35 drops without causing some disagreeable symptoms. The tincture, used was that of the pharmacopœia of 1870, which is one third stronger than that of 1880.

M. gives the usual dose, and then increases three to five drops every second day till some physiological effect, as twitching of some group of voluntary muscles, is produced. The dose is then reduced one third, and in a few days, especially if desirable to produce full effects, ascending doses are again administered. One patient, aged twenty-four, took two hundred drops three times daily. (Unfortunately M. leaves it in doubt whether or not by drops he means minims.) He claims that in the usually prescribed doses nux vomica, in most cases, has only a local action as a bitter; and even with larger doses the system readily becomes accustomed to its stimulation, requiring increasing doses.

Two disadvantageous symptoms produced by large doses were diarhœa and frequent seminal emissions. As to its therapeutic value, M. recommends it especially in atonic dyspepsia, hypochondriasis, headache, weight on the head, and other nervous phenomena, and thinks its good effects first due to its action in increasing the reflex excitability of the spinal cord and vasomotor centres. The drug is also recommended in emphysema, chronic bronchitis, mental and physical depression, etc.

Hyoscine Hydrobromate. Dr. HENRY M. WETHERELL, Jr., *Med. Times*, Dec. 26, 1885, speaks a good word for hyoscine hydrobromate as an hypnotic and sedative. His conclusions are formed from its use in the Pennsylvania Hospital for the Insane. As an hypnotic the usual dose is $\frac{1}{120}$ to $\frac{1}{90}$ grain; sometimes a less dose than $\frac{1}{120}$ grain will suffice. It is seldom necessary to repeat the dose, another decided advantage it possesses over hyoscyamine is that

small doses can be continued a long time without increase, the opposite of which is the case with hyoscyamine. In insomnia occurring in the course of acute delirious mania, it succeeded after all the usual modes of treatment proved inadequate. In the insomnia of agitated melancholia, of the morphine habit, of alcoholism, of acute mania, of neurasthenia, of chronic mental disorder with habitual wakefulness and motor activity, and in those confirmed cases of insomnia from unascertained cause, which usually prove so obnoxious to treatment, hyoscine was found to answer a very good purpose. It does not invariably succeed, but the failures have been very exceptional. Given in the daytime, it acts very well. As a general sedative Dr. W. declares it to be the "very best means at present at our disposal for calming the motor excitement of acute and chronic mental disorders in their talkative, active, noisy, destructive, or violent phases." The dose, however, sometimes has to be greater: from gr. $\frac{1}{120}$ to gr. $\frac{1}{60}$.

It would seem that any one who wants more than this must want the world, but unfortunately it is stated that hyoscine is not always well borne; occasionally the following symptoms have followed moderate doses: Nausea, vomiting, anorexia, dysuria, syncope, with small, rapid, irregular pulse, and with symptoms of partial paralysis of the pneumogastrics. The physiological effects produced by a full dose, $\frac{1}{60}$ gr., are: brief transitory bewilderment; widely dilated pupils; slow, regular, very full pulse; dryness of the throat; relaxation of the vocal cords; very slow, full respiration, sometimes becoming Cheyne-Stokes; marked suffusion of the face and of the general surface of the body; a slight rise of temperature; and free diaphoresis, which does not seem to restore the normal temperature.

Dr. W. thinks the failure of others with the drug may be due to poor preparations. In the above-mentioned hospital it is considered superior to all other hypnotics and sedatives.

Hypnone, a New Hypnotic. *Therapeutic Gaz.*, Jan 15, 1886.

Hypnone is the name of a new hypnotic, known to chemists as C_6H_5-C_0-CH_3, phenyl-methylacetone, and which was presented to the Academy of Medicine by Dujardin-Beaumetz. In doses of $\frac{3}{4}$ to 2 drops it is said to produce a deep physiological sleep. In alcoholic subjects it is said to act decidedly better than paraldehyde or chloral. In cases of insomnia due to cerebral hyperexcitation it acts promptly, but Dujardin-Beaumetz states it is wholly useless in insomnia due to pain. It is well borne by the stomach. It is best given in either a syrup or elixir, the former containing:

Hypnone	gtt. i
Alcohol (90°)	♏ xvi
Syr. aurant. flor.	♏ lxxx
Syr. lauro-cerasi	♏ xvi

Four to ten drops taken at once are, according to Dujardin-Beaumetz, sufficient to produce a refreshing and sufficiently long sleep.

MORTON PRINCE.

Reviews and Bibliographical Notes.

Operative Surgery of Brain. The Field and Limitation of the Operative Surgery of the Human Brain. By JOHN B. ROBERTS, A.M., M.D., Professor of Anatomy and Surgery in the Philadelphia Polyclinic, Surgeon to St. Mary's Hospital. Pp. 80. P. Blakiston, Son, & Co., Philadelphia.

This work is somewhat familiar to us, being the paper presented by Dr. Roberts to the American Surgical Association at its last meeting, elaborated and illustrated and arranged in a tasteful and pleasing manner. It would be interesting had we in the same binding the discussion which followed the reading of the paper. The membership of the Association comprises eminent gentlemen from various cities and those representing the different opinions on brain surgery. The discussion following the reading of any paper should equal, if not exceed, the value of the paper itself, provided each speaker refrains (which requires great self-control) from relating the minute history of his last private case allied in any possible manner to the subject under discussion, to the exclusion of his general experience and his belief in the general subject. However, the discussion on Dr. Roberts' paper can be found in the transactions of the Association if the reader is not convinced or converted by the paper itself. The book would be considered a radical one in almost any community, but in conservative Philadelphia, had its appearance not been anticipated and its author's views known, we fear that a few, at least, of the Quaker City surgeons would have succumbed immediately to attacks of apoplexy, a condition from which Dr. Roberts himself could not rescue them, apoplexy being one of the few pathological conditions not amenable to trephining.

After a short prelude, Dr. Roberts reaches his propositions, or his creed, as he asks to be permitted to call it. Brevity is one of the many commendable characteristics of the book. His propositions are as follows:

1. The complexus of symptoms called "compression of the brain" is due not so much to displacing-pressure exerted on the brain substance, as it is to some form or degree of intracranial inflammation.

2. The conversion of a closed (simple) fracture of the cranium into an open (compound) fracture by incision of the scalp, is, with the improved method of treating wounds, attended with very little increased risk of life.

3. The removal of portions of the cranium by the trephine or other cutting instruments is, if properly done, attended with but little more risk to life than amputation of a finger through the metacarpal bone.

4. In the majority of cranial fractures the inner table is more extensively shattered than the outer table.

5. Perforation of the cranium is to be adopted as an exploratory measure almost as often as it is demanded for therapeutic reasons.

6. Drainage is more essential in wounds of the brain than in wounds of other structures.

7. Many regions of the cerebral hemisphres of man may be incised and excised with comparative impunity.

8. Accidental or operative injuries to the cerebral membranes, meningeal arteries, or venous sinuses, should be treated as are similar lesions of similar structures in other localities.

9. The results of the study of cerebral localization are more necessary to the conscientious surgeon than to the neurologist.

We take pleasure in endorsing each and every one of the above propositions, and feel confident that all the New York surgeons, excepting, perhaps, the very few who are too old, or their confrères who are worse than aged and unable to grasp new ideas—they who do not believe that true antiseptic surgery is superior to sepsis and suppuration; we believe that all except these will approve of Dr. Roberts' conclusions. His experience has been our individual experience, and we think that every surgeon who can conscientiously say that he has treated every head and brain wound as antiseptically as the masters in that art direct, will endorse Dr. Roberts.

These propositions are considered individually at length by the author. They are short enough for every one to read and too succinct to review or abstract. We would dwell a moment on the first, "the complexus of symptoms called 'compression of the brain,' is due not so much to displacing-pressure exerted on the brain substance, as it is to some form of intracranial inflammation," in the hope that some one may prove or disprove the assertion—the only theoretical one in the creed. Certain cases have caused us to question whether inflammation or inflammatory irritation can come on immediately after an injury and in amount sufficient to cause complete paralysis. The paralysis follows the depression of bone almost immediately; too soon, it seems, to be due to any inflammatory process. Perhaps in cases where there is extensive depression of bone there is compression *per se;* in others it is due to inflammation.

A chapter on cerebral localization follows the argument of the propositions. It contains rules for trephining more minute than any heretofore published.

The third and last chapter is devoted to the conditions demanding operative procedure, and with a few illustrative cases is very complete.

The general appearance of the book is excellent and a credit to the publishers. F. C. F.

Psychiatry: A Clinical Treatise on Diseases of the Fore-Brain, based upon a study of its structure, functions, and nutrition. By THEODORE MEYNERT, M.D. Translated, under the authority of the author, by B. Sachs, M.D. Part I., The Anatomy, Physiology, and Chemistry of the Brain. New York, G. P. Putnam's Sons. 1885.

First Notice.

The part of the original work of which this is in part a translation was reviewed in the October, 1884, number of the JOURNAL OF NERVOUS AND MENTAL DISEASE. Meynert is perhaps the most involved of German-writing medical authors, and a translation of such an involved writer is a task of enormous magnitude. Putnam's translation of Meynert's "Brain of Mammals" was an ambitious attempt, which certainly was not a complete success by reason of its being made by an English-thinking person. The attempt, however, was worthy of all praise, since it introduced to English-speaking readers one of the great masters of cerebral anatomy. The translator of the present volume deserves great credit, apart from the merits of his translation, for a well-meant attempt to render Meynert more familiar to English-speaking medical men.

The translator, in his preface, says: "For the shortcomings of my translation I crave the indulgence of the reader. I am quite certain that those best acquainted with the original will not underrate the difficulties of the task and will be most lenient in passing judgment upon my errors." The present volume only includes that portion of the original first part which is devoted to the "Anatomy, Physiology, and Chemistry of the Brain"; the portions relating to prosencephalic diseases being reserved for a second part. That the present work of Meynert is entirely up to date cannot be said, since it bears, in many places, evidence that some of the work was written in 1875, or even earlier; some in 1880, and some between 1880 and the date of publication. A work so written can scarcely be homogeneous and this must of necessity add to the difficulties of the translator.

The preface gives a succinct digest of certain of Meynert's views, and therefore deserves quotation: "The reader will find no other definition of 'psychiatry' in this book but the one given on the title-page: 'Clinical Treatise on *Diseases of the Fore-Brain.*'" It must be obvious that this translation is not clear, and would be liable to ridicule by a captious critic, similar to the ridicule heaped upon Dr. Mann's title: "Psychological Medicine and *Allied Nervous Diseases.*" It would have been

better if Dr. Sachs had rendered this passage: "The only definition of psychiatry accepted by myself is that found on the title-page—'*Diseases of the Prosencephalon.*' [Ridicule is, by very shallow critics, too often heaped upon such ambiguities, and it is best to avoid even this ridicule.] The historical term psychiatry, *i. e.*, 'treatment of the soul,' implies more than we can accomplish. Were I to give a functional designation to the morbid affections of the fore-brain, I would choose the term, 'Diseases of the *Mind*.' And on this term I would insist, in order to avoid the common fallacy that it is permissible to regard the contents of cortical memories as paled sensory images, although we acknowledge the origin of these memories from external sensory stimuli. [The tendency to alternate the first person singular and plural here exhibited runs through the whole translation, and, it must be said, is a decided blemish.]

"We shall show in this book that the fore-brain can neither give rise to hallucinatory phenomena, nor that its functional manifestations, the so-called 'memories' are possessed of the slightest sensory qualities. It would be better, therefore, to speak of memory-symbols. In our memory of the most glaring sunlight, of the most intense roll of thunder, there is not as much as the billionth part of the light of a glow-worm, or of the sound produced by the falling of a hair upon water. But is there any other word in our language with which to designate phenomena devoid of all sensory qualities, but the word psychical? The most conspicuous fact regarding the functional activity of the fore-brain accentuates the difference between the abstract and material character of our concepts. The latter is lacking altogether, and is purely a matter of external perception. But the centres for such perception are in the basal portions of the brain, and not in the *fore*-brain. * * * Dissatisfied with the statistical method, which laid inordinate stress upon hereditary predisposition to disease, I have considered predisposition as a *form of disease*, and not as a condition antecedent to it. I have above all referred to the anatomical peculiarities constituting predisposition. I was not content, as others have been, to accept the mystical conception of heredity, but have insisted on the anatomical peculiarities in patients which constitute predisposition. And these peculiarities were inferred not only from external appearances but also from a due consideration of all abnormal proportions of the body. In an article published as early as 1878, I showed that the nutrition and excitability of the brain must be regarded as depending upon the reciprocal relations existing between the weights of the brain and of the heart.

"As regards the doctrine of predisposition, and more particularly the doctrine of heredity, which has been carried to the extreme of assuming the existence of innate ideas, and which in clinical medicine has led to the erroneous theory of moral insanity, I have deemed it necessary to criticise in its proper connections, Darwin's theory of the inheritance of acquired faculties, as has been

done before me by other German authors, among them DuBois-Reymond and Weissmann. It is taking altogether too simple a view of things, to regard morality as one of man's talents, and as a definite psychical property which is present in some persons and lacking in others. Indeed there is great truth in Weissmann's observation: 'Talents do not depend upon the possession of any special portion of the brain; there is nothing simple about them, but they are combinations of many and widely different psychical faculties.' * * * Thinking physicians * * * will distinguish between those who are possibly 'called' to disease, and that fortunately smaller number of persons who are, in the saddest sense of the term, 'chosen' for disease."

As Meynert accepts *manie raisonnante* as a clinical form, it is obvious that his objection is not to moral insanity as a psychosis, but to certain views explicatory of its existence. At the same time it seems a little like begging the question to claim that any true psychologist held that talents were aught more than the sum-total of a series of combinations; one element being deranged, the whole tumbles into confusion.

In the main, Dr. Sachs' work has been well done. He has rendered the English-speaking profession a service in presenting to it this work of Meynert's. An exhaustive review of the present section is not desirable, for the work requires reading as a whole, rather than as a segment. When completed it will be reviewed at length. The work of the publishers is in the main well done. Typographical errors, like "call" for "cull," are noticeable, but are relatively few. The perusal of the work is recommended to every one interested in psychiatry. J. G. KIERNAN.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, February 2, 1886.

LEONARD WEBER, M.D., Vice-President, in the chair.

Dr. DANA reported a case of total trigeminal paralysis. (See p. 65 of this number.)

DISCUSSION ON DR. DANA'S PAPER.

Dr. SAMUEL SEXTON said that ten years ago it was considered proper to divide the chorda tympani for tinnitus aurium, and it was supposed to have been done. This nerve has a long and circuitous course through the tympanic cavity, and is surrounded by a sheath of mucous membrane liable to inflammation in the acute purulent processes of the ear, especially in children. He had seen and studied a large number of cases in which, owing to acute purulent inflammation there was complete destruction of tympanic contents, including the chorda tympani. There was no alteration of the sense of taste, though the tests to discover such an alteration were carefully made.

An Italian writer has called attention to the chorda tympani as a nerve in intimate connection with speech; that certain word-sounds could not be found when it was absent. But Dr. Sexton's experience had not verified this. Taste was a complex function which was developed slowly by cultivation. When tympanic membrane was absent, so that a probe could touch the inner wall of the tympanum, a variety of reflexes could be evoked, such as vomiting, coughing, sneezing. Secretions, especially in the region of the tonsils and pharynx, were increased, as also the saliva.

Dr. M. A. STARR reported ten cases of total anæsthesia follow-

ing neuralgia of the trigeminus in which there was no loss of taste. In his study of the lesions of the medulla and pons there was no record of loss of taste, though all of the cranial nerves, except the ninth and tenth, were involved. All of which would support the views advanced by the author of this paper.

Dr. A. D. ROCKWELL had seen many cases, and instanced one, in which there was neuralgia of the seventh and fifth pair of nerves accompanied with loss of taste.

Dr. DANA, in closing the discussion, thought the cases cited by Dr. Sexton showed that the chorda tympani dose not carry the nerve fibres of taste at all; that they were proof that they must take another course from that described by most of the text-books. It hardly seemed probable that they had so complicated an arrangement as that claimed by Gowers, which he showed by means of a diagram.

VASO-MOTOR NEUROSES.

Dr. M. ALLEN STARR, the author of the paper, said that under this title it was intended to include a series of disturbances of circulation and nutrition whose causation could be traced to disorders of the nervous system. He selected this subject because it was one which had been somewhat neglected in the discussions of this society, and because no definite summary of the present state of knowledge of the physiology of the vaso-motor action had lately been presented, and because he had lately seen a number of cases which might be classed together under this title, and which he wished to have discussed by the members present.

Disturbances of the circulation might occur in any part of the body, and were to be regarded not as distinct diseases of the parts in which they occurred, but as symptoms of lesions of the peripheral, or sympathetic, or central nervous systems. At the present time, however, it was impossible to make a pathological classification of these symptoms, and therefore they might be discussed together. In treating of the subject of vaso-motor neuroses the discussion of the physiology and pathology of the vaso-motor system could be united to advantage.

Since changes in the force and frequency of the heart's action and variations in the total amount of blood in the body affect the body as a whole, the state of the circulation in any one organ or part depends largely upon the degree of contraction or dilatation of its vessels. This is known as the local vascular tone. It is

under the control of the system of nerve ganglia, with their subservient fibres which are found in the smaller arteries. The energy expended by these ganglia is manifested by a constant contraction of the circular muscular coat of the artery, a contraction which is constantly opposed by the dilating force exercised by the blood pressure within the vessel. In a state of health an exact equipoise between these forces never occurs. Variations, however, in the vascular tone are produced either by local influence or by influences reaching the ganglia from a distance through the vaso-constrictor or vaso-dilator fibres. The constant manifestation of energy in the maintenance of arterial tone does not have its only source in the local ganglia, but is derived partly from the central nervous system through the vaso-constrictor nerves. Here the experiments of Claude Brenard were detailed. From these experiments it was concluded that a dilatation of the vessels follows, 1, a paralysis of the local ganglia in their walls; 2, separation of these from the sympathetic ganglia; 3, a destruction of the sympathetic ganglia; 4, a separation of these from the spinal cord by a division of the anterior spinal nerve roots; 5, a disintegration of the cord; 6, a separation of these centres from the medulla; 7, the destruction of the medulla oblongata.

The action so far considered has been wholly of a vaso-constrictor kind, and the dilatation mentioned has been due to a suspension of constrictor energy normally passing outward. This is a passive dilatation. But further experiments have shown that another kind of dilatation may be produced, due to an impulse of an active kind sent to the local ganglia by the vaso-dilators. This is an inhibitory impulse arresting the constrictor action of the ganglia in the vessel walls in spite of the continued energy sent them from the central nervous system by the constrictors. Here the experiments on vaso-motor dilatation were given, and the various theories of the mechanism of dilatation in the vessel walls, it being concluded that there was no mechanism in the wall which could produce a dilatation, and that a dilatation was due wholly to the blood pressure within overcoming constrictor action.

The subject of vaso-motor reflexes was then taken up, and it was shown that the seat of these reflexes was in the dorsal region of the spinal cord, since all vaso-constrictors and vaso-dilators can be traced to that region. The particular area of the spinal cord covering these reflexes was thought to be the gray matter surrounding the central canal and including the fascicular columns

of Clarke. As bearing on this point the author referred to the observations by Jacubovitch, Schultze, and Fürstner. It was impossible to say from these cases whether vaso-motor functions are located in the column of Clarke or in the gray matter around the central canal, or in both.

Vascular tone of the thoracic and abdominal organs is regulated by centres in the pons and medulla. Experiments of Bernard were related, and cases of diabetes mellitus and diabetes insipidus produced by lesion of the medulla and pons were mentioned. The influence of the vaso-motors in the production of functional disturbance, disorders of the stomach and intestines was alluded to.

From this review of physiological experiments and pathological facts, it becomes evident that the disturbances of vascular tone, which are included under the title vaso-motor neuroses, may be produced by many different causes acting upon many different organs. The author here mentioned a number of possible causes and their location, after which he gave the histories of seven cases of vaso-motor neuroses, with remarks on the same. These cases seemed to illustrate some of the conditions which have still to be classed under the term vaso-motor neuroses, from want of a more perfect knowledge of their nature. They appear to be little noticed in the books, and rarely discussed in societies; but they certainly merit careful study, in as much as they require cautious and scientific treatment.

DISCUSSION ON DR. STARR'S PAPER.

Dr. SACHS thought that some statements made by Dr. Starr elucidated some obscure points in experiments (published in *Pflüger's Archiv für Phys.*, 1881) which he made several years ago, on animals, to discover the relation of the spinal cord to the secretion of the kidneys. Eckhardt had stated that section of the cervical spinal cord had the same effect as section of the medulla—that is, to inhibit the secretion. These experiments were difficult because of the necessity of keeping up artificial respiration, but they showed that urine was secreted as in health, though somewhat diminished in quantity. That it was actually secreted was proved by introducing chemical substances into the blood, the coloring matter of which was shown in the urine secreted. The inhibition of renal secretion, observed by Eckhardt, Dr. Sachs thought, was due to the peripheral irritation, caused by laying bare the ureters, which interfered with the secretion of the kidneys.

He asked Dr. Starr what views he advanced with reference to the course of the vaso-constrictor nerves.

Dr. STARR replied that one theory was that they passed from the lateral tracts of the cord through the fourth, fifth, and sixth cervical into the sympathetic system, and thence to the viscera. Another was that they entered the sympathetic as far down as the level of the first dorsal. There was an intimate connection with the kidney. Experiments high up in the cord would of course produce diabetes.

Dr. SACHS said that in his experiments high up in the cord there was no diabetes.

Dr. C. L. DANA considered himself indebted to Dr. Starr for so able a paper. The local vaso-motor ganglia had been assumed to exist, but had never been satisfactorily demonstrated as physiological entities. Vaso-motor disturbances were common as symptoms, and were so considered rather than as a distinct disease. In this class of cases were those vaso-motor troubles of the extremities, such as digiti mortui, and mild types of renal disease; an independent disorder, flushing of the ears; Basedow's disease and diabetes, which should also be ranked among these. It gave rise to confusion to consider the vaso-motor system as independent, in the same way as the sympathetic system, forgetful of the relation of both of these to the cerebro-spinal.

Dr. WEBER spoke of the connection of sciatica with diabetes. While sugar is found in the urine of patients suffering from protracted sciatica, there were few cases of well-developed diabetes in which there was sciatica. He related three cases of sciatica in which there had been sugar in the urine, and the attending symptoms of diabetes. He had seen cases where sugar was present *after* sciatica; but he was doubtful if cases of true diabetes were developed by sciatica.

Dr. STARR, in closing the discussion, remarked that the vaso-motor local ganglia had been very recently demonstrated by French physiologists in frogs, rabbits, and cats. He did not intend to give the impression that sciatica caused glycosuria, but referred to those cases, quite a number of which were on record, where there was a sudden appearance of sugar in the urine which afterward disappeared.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting January 26, 1886.

J. H. MUSSER, M.D., presented some notes of thirteen cases of tubercular meningitis.

All of the cases were observed in private practice, hence the lack of detail of symptoms, such as temperature, etc. They occurred in a period of six years. Nine of them were in the writer's private practice, two in institutions under his care, and two in the practice of medical friends. It may be considered a disease of frequent occurrence, for the relative proportion of its frequency to that of many other diseases, as forms of liver disease, is much in excess. The nine cases occurred in a practice ranging from 50 to 400 families yearly, during the six years. For every case of tubercular meningitis, the writer has had twenty-five of phthisis. During the same period one case each of tubercular peritonitis and interstitial tuberculosis have been under his care. In later years the disease has been less frequently seen, corresponding with the social improvement of the practice. In the first half, six cases were observed. In the latter half, the remaining ones. The mortality table of the "Homes" to which the writer has been physician, present some interesting features in this connection. In one Home of one hundred and eighty boys, aged from three to sixteen, and 30 adults, but three cases of tubercular meningitis occurred in five years. In a Home for twenty-four colored children of both sexes, mean age seven years, the disease was not observed in four years. In a third Home for children, both sexes, about eighty in number, in the records of twelve years no death occurred from this disease. During the same period of observation of these nine cases, five cases of hydrocephaloid disease, one of cerebro-spinal meningitis, and two of syphilitic meningitis were observed.

Causes: Seven of the cases were males, and six females, making sex of no causal influence. The ages ranged from seven months to fourteen years in twelve cases. One was thirty-two. Six of the patients were under five years, four between five and ten, and two between ten and fifteen. The ancestors of nine had tuberculosis in some form. In two other instances the children of the same generation had some tubercular affection. In only two cases was there no antecedent or coincident history of tuberculosis in the family. One of the patients, aged seven months, was nursed by a mother whose tubercular disease antedated her pregnancy.

The hygienic surroundings in the large number of cases were quite fair. Two alone could be pronounced as bad. One child had been weaned early in life, and improperly fed. The previous health of all save one or two was bad. Some remote local inflammatory foci, either of simple or specific nature, could be determined. In one, aged fifteen months, the bronchial and mesenteric glands, after a whooping-cough or diarrhœa, were diseased; asthma and bronchitis, with enlarged bronchial glands, no doubt, preceded in another; hip-joint disease, suppurating cervical lymphatics, chronic bronchitis, and phthisis, respectively, preceded in four cases. Very poor health in one instance, and "weak" lungs in another, also preceded. In one, the health had apparently been good; in two the state of the health was not noted. Exposure to the sun was thought to be the cause of the twelfth, but the lad was always fragile, and had diarrhœas. The nursing baby completes the list—she was always delicate.

Clinical Course: In general, it may be said to be irregular. In six cases the periods of invasion, excitation, and depression could be traced. In two instances the stage of invasion or the premonitory symptoms was absent entirely. In the other cases the stages were "mixed."

The following summary indicates the mode of development of the entire series. Case I. was a girl aged sixteen months; antecedent cause, whooping-cough and tabes mesenterica; slight general spasm, followed in a few hours by convulsions of right side of body with aphasia. In Case II., boy aged four, with asthma, etc., there were loss of flesh for four weeks, intermitting headache ten days, vertigo three days, was peevish and restless at night. Case III., girl aged ten years, had for one year headache and vertigo, extreme emaciation (cerebellar tumor). In Case IV.

there were loss of flesh, debility, restlessness, and poor appetite for several weeks. The course of the fifth was not known, while the sixth had been ill ten days definitely ; for a brief time loss of flesh with pain in the left temple, sleeplessness and constipation had been present. Sharp paroxysmal headache tormented the seventh case, a youth of nine, for three days prior to the meningitic explosion. Until twenty-four hours before very active symptoms occurred, the eighth case, an infant under two years, was considered in perfect health. Prolonged lassitude, fretfulness, and some emaciation, preceded the illness of Case IX., a boy of eight. For four weeks the tenth case, a precocious little girl five years old, suffered from symptoms not unlike malaria—languor, emaciation, fretfulness, irregular intermitting fever, restlessness at night, poor appetite, without vomiting or constipation. The eleventh case had phthisis, but observed that his customary hectic had quitted him two weeks before the development of headache and vertigo, which symptoms antedated the active brain symptoms by four days. The twelfth case did not thrive as a nursing baby should, and vomited frequently. A convulsion was the first warning of cerebral trouble. A little girl, lost appetite, was peevish, slept poorly, was feverish and became emaciated for three weeks before the cerebral symptoms developed. She had lymphatic enlargement in the neck and abdomen. Emaciation, debility, fretfulness, night-restlessness, irregular febrile movement, disorders of the gastro-intestinal tract, and headache were therefore the chief premonitory symptoms. Emaciation, it is seen, was the most frequent and most marked.

An analysis of the phenomena of the first and second stages would be tedious and without practical value. Reference in the succeeding notes will be made to some salient groups of symptoms; present more or less in both stages, and to peculiar manifestations of common symptoms. *Headache*, *motor* and *sensory irritations*, and *palsies* occurred in all the cases. In a few, the *headache* was distinctly intermitting. The *spasms* and *palsies* of groups of muscles were irregular in respective cases, as to order or frequency. Hourly change in the state of the muscles was common, and the same may be said regarding sensation. Not only does this apply to alterations of function of spinal but also of cerebral nerves. Convulsions (partial, unilateral, or general), delirium, and coma were present at the usual period, but presented no peculiar feature.

Special senses : Total blindness was observed in one case ; impairment of vision in five ; optic neuritis was recorded in three. The state of the pupils was not of special import—in one instance was there irregularity in size in the second stage. In the first stage they were not affected, save contracted in one instance, and fixed, in a dilated state, in another. Dilatation was the rule in the second stage. Nystagmus was observed in one case. Purulent conjunctivitis occurred in eight cases, in the second stage usually, and in two instances were unilateral. Ptosis was present in only one case.

In five instances local erythema and general flushings were noted. An urticaria-like rash was seen in three instances. In the latter stages pallor or a bluish hue of the face was observed. V unilateral mottling of the extremities was observed. Emaciation was marked and persistent in the prolonged cases, nine in all. Fever was present in the first stage in seven cases ; in the second, in nine ; intermitting in type, often with irregular paroxysms. Hyperpyrexia was noted in one. The *pulse* was slow seven times in the first stage ; irregular or intermitting in four. In the second stage it was usually quickened, small, rapid, and feeble ; twice, slow and irregular. It was never wiry or corded in either stage.

Vomiting occurred but three times in the first stage ; once only in the premonitory stage, and once only in the final stage. Constipation was seldom observed—three times in the first, twice in the second stage. Diarrhœa was excessive once. Offensiveness of the *breath* was very frequent, and occurred chiefly in the last stage, but was so marked at first, also, in three cases, as to be noted. In the larger (nine) number of instances, the tongue was heavily coated. The scaphoid abdomen occurred at varying periods : once in the first stage, seven times in the second stage, while three times it is recorded absent ; twice there is no record. Respiratory symptoms : first stage, irregular or Cheyne-Stokes four times, rapid twice, slow once. Second stage, Cheyne-Stokes twice, rapid six times.

Remissions of a decided character were noted twice ; after the development of the most grave symptoms, a period of improvement led to hopes of recovery.

Duration : Of premonitory symptoms, unknown twice ; prolonged, once ; one year, once, tumor in two cases, about four weeks ; in two, ten days ; in one each, three days and twenty-four hours ; in two they were absent, and in two the first stage developed at once.

First stage : Twelve hours, 2 ; four days, 2 ; five days, 2 ; ten days, 2 ; eleven days, 1 ; thirteen days, 1 ; unknown, 3.

Second stage : Twenty-four hours, 1 ; seven days, 1 ; five days, 3 ; forty-eight hours, 1 ; six days, 1 ; eight days, 1 ; four days, 2 ; nine days, 1 ; unknown 2.

Entire duration : Thirty-six hours, 1 ; four days, 1 ; seven days, 1 ; twelve days, 2 ; fifteen days, 2 ; eight days, 1 ; fourteen days, 1 ; eleven days 1 ; unknown, 3.

The difficulty of estimating the duration is evident to any one. It can only be said that a short first stage, in all the instances, determined a short second stage.

A post-mortem examination was held on four of the cases. In two the disease was largely localized, once in the cerebellar region, in another over the right frontal convolutions, the presence of the tubercle causing during life the symptoms due to localized cerebral irritation at these points. The autopsy also revealed in the case of aphasia, etc., a well-known rule, that the amount of gross lesion is not to be inferred from the extent of symptoms.

Summary.—Tubercular meningitis may be considered (1) of frequent occurrence, in the middle and lower classes especially ; (2) more common than other localizations of tuberculosis, except the pulmonary ; and (3) much more frequent than other varieties of meningitis.

Among the *causes*, most prominent are age, hereditary predisposition to tuberculosis, poor hygienic surroundings, and insufficient and poor food.

The prime factor always to be looked for, is the presence of a primary focus, inflammatory, tubercular, or "cheesy."

The important symptoms presented in this series of cases were, in addition to the usually noted ones, emaciation, irregular fever, and gastro-intestinal disorder, without vomiting ; in the succeeding stages purulent conjunctivitis, erythemas, and other vascular changes in the skin, excessive emaciation, fever, offensiveness of breath, the scaphoid abdomen, and changes in respiratory rhythm.

Editorial Notes and Miscellany.

In a short article in Mendel's *Neurol. Centralblatt*, Prof. Erb insists on the importance of a standard-size electrode to be used in all experiments on the galvanic excitability of nerves and muscles. Referring to the well-known formula for the density of a current—density $= \frac{Q}{D}$ (in which Q denotes the quantity of electricity conveyed, and D the diameter of the conducting electrode), he states very correctly that while we can define Q in absolute units, with the aid of one of the many galvanometers now in use, we have thus far neglected D altogether. Erb proposes the general adoption of a "normal electrode," and he suggests that the area of this electrode be 10 sq. cm. With a current strength of 1, 3, or 5 milliampères, the absolute density of the current could be satisfactorily expressed as Density $= \frac{1}{10}$, $\frac{3}{10}$, or $\frac{5}{10}$.

We need not enlarge upon the wisdom of this suggestion. We urge the adoption of this "normal electrode" upon all neurologists in this country who may be engaged, or interested, in careful electrical measurements.

At the instigation of de Watteville the "Neurological Society of London" has been organized. The following have been appointed officers of this society: Hughlings Jackson, President; Wilks and Crichton Browne, Vice-Presidents; Bristowe, Treasurer; de Watteville and Bennett, Secretaries. The Council is to consist of Bastian, Broadbent, Bucknill, Buzzard, Ferrier, Galton, Hutchinson, Romanes, Savage, and Schäfer. With such a formidable list of names the society should prove a success. Its papers and discussions will be watched with great interest.

VOL. XIII. MARCH, 1886. No. 3

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

CLINICAL OBSERVATIONS ON REFLEX GENITAL NEUROSES IN THE FEMALE.*

By PAUL F. MUNDÉ, M.D.,

IT is customary to ascribe the majority of nervous manifestations in the female sex to the fact that they occur in "women," and to assume that on the organs which particularly entitle them to their sex depend more or less directly the mental and nervous peculiarities so well known and so universally admired when healthfully displayed, and so feared and deprecated by the sterner sex when disordered or unduly exaggerated. But the old saying, "Propter uterum mulier est," is even less correct than to substitute "ovaria" for "uterum" since not even a combination of both "uterum" and "ovaria" fully covers the ground when we look for the organs, on the derangement of which the peculiar local and reflex neuroses which form the subject of this paper are based. Not only the uterus and ovaries, but also the bladder, rectum, vagina, clitoris, labia, perineum, and the now so fashionable Fallopian tubes, all join in producing local or reflex neuroses. To properly estimate the origin and importance of these neuroses is often as difficult as it is to avoid referring every

* Read before the Section on Neurology of the Academy of Medicine, March 12, 1886.

distinct nervous disturbance in a woman to her sexual organs. No doubt many errors are made in this latter particular, since it is but natural for every specialist to seek to explain doubtful symptoms and complications on the general principle that all the rest of the body turns about the pivotal organs in which he is specially interested, and I do not think that the gynæcologist is any worse in this respect than the neurologist, or the dermatologist, or the other "ologists" too numerous to mention.

I may be permitted to say that, personally, I have endeavored to avoid straining the relations of cause and effect between distant neuroses and insignificant genital lesions as much as possible in my practice; and I know of a number of cases where I have refused to believe a general anæmia, or a hemicrania, or a neurasthenia, to be due to a hyper-plastic uterus, or a lacerated, but healed cervix, when some of my colleagues, who differed from me, have seen fit to use local treatment or sew up the cervical tear. But I can report, on the other hand, a fair number of cases in which it seemed to the highest degree improbable that the distant neurosis had any thing whatever to do with the genital lesion, and in which treatment and cure of the latter resulted in permanent relief of the former. Before briefly relating a few of these cases, I think it will be well to give a survey of the varieties and approximate causes of the genital neuroses most commonly met with in females.

Reflex neuroses may be divided into two main groups—physical and psychical:

1. *Physical.*—These may be again divided into *local* and *peripheral.*

Local neuroses occur in any of the various organs contained in the pelvic cavity or bordering on the genital cleft. Thus we find tenesmus of the neck of the bladder, generally due to exposure to cold, or an irritable bladder, requiring frequent evacuation, commonly met with in excitable women, or under some form of mental disturbance; or pruritus vulvæ, uncomplicated by diabetes or vulvitis; or a hyperæsthetic condition of the vaginal orifice producing the condition known as vaginismus; or a throbbing, lancinating

sensation in the vagina is complained of; or the woman speaks of experiencing motions in the hypogastric region similar to those of quickening, a symptom which I have generally found to be associated with subinvolution or hyperplasia of the uterus; or a numb, dull ache is felt over the sacro-ischiatic space, when I generally expect to find a congestion or adhesion of the corresponding ovary. One of the most frequent local neuroses is sacralgia, the well-known "back-ache," to which most of our present women are victims more or less; and while it is often due to a backward displacement of the uterus, I frequently find it present without being able to detect the slightest *direct* relation between it and the rectum, the pelvic cellular tissue, or the uterus, even though the latter organ may be in a pathological condition.

Pain in the ovarian region is also not uncommon with apparently perfectly healthy ovaries and tubes, and we are obliged to include in our list of sexual diseases the vague condition called "ovarian neuralgia"; I say vague, because the minute morbid changes which *may* produce the so-called neuralgic pain, are unrecognizable by our methods of exploration.

In calling these various sensations "neuroses" I wish to emphasize the fact that ordinarily they are unassociated, so far as physical examination can reveal, with organic disease of the organ where the sensation is experienced. Thus in sacralgia, the sacrum is not diseased; in vesical tenesmus, there is no organic disease of the bladder; in pruritus vulvæ, the mucous membrane and skin of the vulva are not pathological, except in so far as is caused secondarily by scratching. The peculiar sensation resembling quickening, which I believe to be a neurosis of the intestines, is produced probably by hyperperistalsis.

These neuroses are not necessarily acutely painful, but, like the frequent micturition and hyperperistalsis, merely annoying.

At times it may be difficult to differentiate and to draw the line where a neurosis ends, and a neuralgia or actual pain begins. The true distinction is that the *pain* is usually

situated in the diseased organ; the *neurosis*, however, is reflex, no matter how near by.

Peripheral neuroses do not require the explanation just given. They are either unpleasant, annoying, or actually painful sensations experienced at greater or lesser distance, but entirely away from the genital organs.

One of the most common distinct reflex neuroses is hemicrania (migraine, sick headache), which is frequently limited to the menstrual epoch, but often comes on at irregular intervals in the intermenstrual period. It is by no means always dependent upon or associated with organic sexual disease, and hence is so often incurable. The so-called hot flushes over the face and neck are a familiar reflex neurosis. Cephalalgia, chiefly vertical and occipital pressure; also frontal headache; cardialgia, palpitation, and intermittent pulse; gastralgia and dyspepsia (Fothergill has described an "ovarian dyspepsia" dependent on chronic oöphoritis and curable only through treatment of the latter); intercostal neuralgia, are other forms of reflex peripheral neuroses.

The oculists are familiar with the optic derangements so commonly met with in women during the menstrual period: the blurred vision, myopia, strabismus, occurring before menstruation or when the flow is scanty, and which are not always referable to retinal hyperæmia or faulty muscular attachment. The laryngologist need not be told of the aphonia which may accompany menstrual suppression, or of the hacking cough so often depending on uterine disease, and the neurologist is well aware of the relation between utero-ovarian disease and so-called spinal irritation and general neurasthenia. And who has not heard of the familiar globus hystericus, of which neurosis more anon? Approaching the pelvis again, we find a very common annoying, if not painful, symptom to be the abdominal bloating of which many women complain just before their menstrual period, as well as at other times. This is not due to constipation, although such women are generally afflicted with that infirmity, but it is a reflex neurosis, the exact physical explanation of which has not always been quite clear to me; I have thought it to be a temporary paresis of

the intestine, allowing it to fill with gas, but I could not gain from the patients the information that large quantities of gas escaped before they were relieved. Asafetida generally reduced the bloating, but it must have acted as an antispasmodic and nervine, rather than as a carminative. Sciatic and crural neuroses are not uncommon as the direct result of some disturbance of or pressure on the sacral or crural nerves within the pelvis. These neuroses so closely approach the character of actual pain, as to be more properly called neuralgiæ. Pelvic exudations usually produce the sciatic, and ovarian inflammation the crural neuralgia. These may extend down to the knee and even to the ankle.

These peripheral neuroses at times change in intensity in proportion to the local pain. Thus I have a patient at present with severe ovaralgia on the right side (at least I cannot detect any organic change in the ovary), and frequent frontal cephalalgia, in whom the ovarian pain and the headache often alternate, one increasing as the other abates.

Psychical Neuroses.—The variety of these is much less numerous than the physical. The most common and familiar form is that protean monster, hysteria, which may simulate almost any and every ailment to which flesh is heir to, from pregnancy to hip-disease, and from paralysis to pulmonary phthisis. It is not necessary for me to go into detail on this familiar topic ; but I may say that, while a combination of phenomena presenting the peculiar features of hysteria but rarely occurs in the male sex, notwithstanding, hysteria by no means invariably proves the existence of or its dependence upon organic sexual disease. Hysteria is a functional disturbance of the cerebro-spinal system, occurring in highly-strung, excitable women, whose genital organs may be perfectly healthy. Hystero-epilepsy may also be considered a psychical reflex neurosis, since the epileptiform convulsions are merely a result of the reflex cerebral disturbance.

This neurosis is more frequently met with as the result of the imperfect performance or inception of the menstrual function, either from sluggishness of the circulation, de-

ficient innervation, or imperfect development of the ovaries or uterus.

The psychical neurosis which is most commonly met with toward the menopause is melancholia.

As cancer of the uterus is the physical rock on which so many women have been shipwrecked at that critical period of their lives, so is melancholia the mental danger which threatens them at the same time, and it does not necessarily follow that organic local disease must be the cause.

It may well be said that nearly all, if not all, the reflex physical neuroses enumerated above may present themselves in a woman during the longer or shorter period, extending from several months to several years, occupied by her transition from sexual activity to sexual senility. And generally they exceed in intensity the same symptoms earlier in life. Chief among these may be mentioned the abdominal distension, which often induces the firm belief that pregnancy is again present after a more or less prolonged interval; and the flushes of the face, alternating with chills. Vertigo, too, is a common neurosis, although it may depend on irregular circulation.

Rarely the cerebral functions may become impaired at the climacteric, and aphasia, dulness of intellect, and a tendency to incoherency in speech and thought may for a time alarm the friends. I have seen local lesions which at other times would be unattended by the slightest anxiety or worry, at that period produce mental depression requiring temporary seclusion in a retreat.

But I must not dilate too much on my theme, and will pass on to say a few words about the treatment. As I have already stated, it is not always easy to trace the relation between the local lesion and the neurosis; and good judgment, caution, and often, as much as any thing, honesty, are required not to extend the specialistic lines too far. But it goes almost without saying, that where a distinct local lesion exists, be it in the ovaries, tubes, uterus, perineum, bladder, pelvic peritoneum, or cellular tissue, it should be remedied to the best of our ability, even though we may not be able to trace distinctly the relation between the

local condition and the peripheral symptoms. And this indication may sometimes carry us farther than a logical sequence would lead us to go. Thus, if a woman with a moderately lacerated perineum or with one that is partly healed by cicatricial bands, shows symptoms of nervous irritability or melancholia, and we learn on careful inquiry that coition is no longer agreeable to her husband on account of the relaxed state of the vaginal orifice, or it is painful to her on account of the tender cicatrices, and she therefore shrinks from the act when her husband desires it, —then I think we are justified in proposing a restoration of the perineum to its integrity by an operation, although in itself the degree of the rent does not call for such treatment.

Again, if a patient has a distant reflex neurosis, the cause of which is obscure, which does not yield to treatment, and at the same time has a lacerated cervix or a retroverted uterus, which latter give her no inconvenience, we still should remedy these genital lesions on the principle that in the female sex it is well to remember that any functional disorder, no matter how remote its seat or how obscure the connection, *may* possibly depend on genital disease, and *may* be relieved by the cure of the latter. This is a safe rule if it is not overdone.

Besides the treatment and, as far as possible, cure of the local lesions, after which *immediate* relief should not always be expected, measures to improve the general health (tonics, phosphorus, iron, strychnia, etc., massage, "food and rest cure," general galvanization to spine and extremities), in "spinal irritation" and cerebral flushes, blisters between the shoulders, or the actual cautery along the spine, or the milder dry cups daily, should be adopted. Sexual abstinence is usually indicated. Each case should of course be treated on its own merits. I have seen much good result to the peripheral neurosis from vagino-abdominal galvanization continued for several months.

The usual routine of the antispasmodics (asafetida, valerian, Hoffmann's anodyne, etc.) is, of course, indicated; and the digestive and intestinal functions should be carefully attended to.

Improvement is usually slow to come, and relapses may occur. But where there was a distinct relation between the local lesion and the neurosis, a cure is most likely to be effected by the removal of the local cause.

I will give a few brief histories of several cases which have come under my observation, in order to illustrate the peculiar forms which the reflex genital neuroses may assume in the female sex.

CASE I.—*Hemicrania of eighteen years' standing; cured by operation on old laceration of cervix and pelveo-abdominal galvanization.*

Mrs. C. S., thirty-nine years of age; married twice; one child by first husband eighteen years ago; married seven years to second husband without conceiving. Complains of frequent pain in right groin and down right thigh, with right hemicrania, nausea, and vomiting, both of which symptoms are most marked before the menstrual flow. Examination showed a bilateral laceration of the cervix of the third degree, completely healed over; extending from the angle of the rent in the right side was a small, hard nodule, evidently an old plastic exudation, pressure on which caused pain down the right thigh. Basing on a previous good result from galvanism in acute sciatica due to pelvic exudation, but without expecting any benefit to the hemicrania, I applied galvanism by a vaginal electrode and a sponge over the right ovarian and sciatic regions, with the result of not only relieving the sciatica, but, curiously, also the hemicrania, the attacks of which diminished in frequency and severity after several weeks of galvanization, and finally ceased entirely. The cure was confirmed by excising the cicatricial tissue in the angle of the cervical tear and uniting the edges. The patient has since then (nearly five years) remained free from sciatica or hemicrania.

CASE II.—*Reflex cataleptiform syncope caused by pressure on the cicatrix of a lacerated cervix; trachelorrhaphy; cure.*

Mrs. H. B., twenty-two years old, mother of one child five years before, was brought to me in the autumn of 1881 by her husband, because since the birth of the child she had gradually lost all sexual appetite, and recently actually seemed to fall asleep during the act, of the performance and completion of which she seemed totally unconscious. On examination, I easily detected a deep laceration of the cervix, and pressing firmly into the angles of the rent, I asked the patient if I gave her pain. Receiving no reply, I looked at her face and found her fast asleep. Only deep pressure through the abdominal walls over the ovarian regions aroused her. Pressure on the cervical cicatrix at once reproduced the hypnosis.

Deep excision of the cicatrices and union of the surfaces effected a perfect cure.

Peculiar reflex neuroses from cervical lacerations have been reported by Sutton (epileptiform convulsions), Ill (loss of sexual desire and orgasm), myself (hemicrania), and I have a singular case to relate in which persistent chorea was apparently due to the same local lesion.

CASE III.—*Unilateral chorea, persisting for months after confinement, eventually cured by trachelorrhaphy.*

Mrs. F., a native of Alabama, was referred to me in 1881 by Dr. E. C. Seguin, who had in vain endeavored to relieve the choreic convulsions from which she had suffered for some time. He suspected possible uterine disease, and requested me to examine her. I found a well-marked retroversion of the uterus and a double laceration of moderate degree. I advised replacement and retention of the uterus as the first step, attaching more importance to this lesion than to the comparatively insignificant cervical laceration. This was done, but no relief to the chorea followed. The cervix was then operated on, and the first operation proving only a partial success, a second attempt was made to repair the rent, which was successful. But still the chorea continued ; the patient eventually returned home, and I put the case down as a failure, especially as I had not held out any great hopes from the operation. Imagine my surprise to receive a visit some two years later from a Southern lady, who introduced herself by saying that she was sent me by Mrs. F. whom I had "cured" so skilfully, and who was now perfectly well.

I admit the element of doubt in this case, for time may have done as much as my treatment. But in the following case, the effect of closing the cervical rent on the general neurasthenia was too rapid and too direct to admit of question.

CASE IV.—*Neurasthenia cured by trachelorrhaphy.*

Mrs. S. B., mother of several children and wife of a retired physician in a neighboring city, who had found an ample competence in commercial pursuits, called on me late in 1884, for neurasthenic symptoms as marked as I ever saw them. A lithe, active, handsome woman, she had fallen to the necessity of carrying a brandy flask with her wherever she went, to fortify herself against her uncontrollable attacks of faintness and depression. I expected to find some serious uterine lesion, but could discover merely a large laceration of the cervix, with considerable cervical catarrh. As the latter seemed to annoy her, I advised closure of

the laceration, hoping thereafter to act on her neurasthenia by other measures.

The laceration was successfully operated on in January, 1885, and early in October last the lady called on me to exhibit her perfect restoration to health. She had spent the summer on a driving trip with her husband, had grown stout and brown, the brandy bottle had been discarded, and she exultantly proclaimed herself a "healthy woman."

Certainly her appearance did not belie her words!

Now, I am constrained to admit the beneficial influence exerted on the mind in this case by the operation, but I cannot allow that this was the chief factor in the recovery of the patient, for the physical sequence was too marked.

As an illustration of the peculiar relation between an unsuspected uterine disturbance and a remote neurosis I will mention the following case:

CASE V.—*Persistent hemicrania cured permanently by the reposition and retention by a pessary of an unsuspected retroversion of the uterus.*

Miss A. S., forty years, stout, well nourished, was referred to me several years ago by the late Dr. Elsberg for persistent hemicrania, which he had in vain tried to cure. Finally, as her attacks were most severe at the menstrual epochs he suspected possible uterine disease, and sent her to me. Although she complained of absolutely no pelvic sensation, I deemed it my duty to propose a vaginal examination to which she readily consented. To my surprise I found a retroversion of the uterus of the third degree, which I replaced and permanently rectified by inserting a lever pessary. This pessary has now been worn for over four years, with occasional intermissions, and since its introduction the hemicrania has entirely disappeared. I see the lady (who lives several hundred miles from here) two or three times a year in order to watch the pessary, and am, therefore, in a position to know her condition.

In conclusion, I will report an instance of mental disturbance due to uterine displacement, which escaped the notice of several eminent neurologists.

CASE VI.—*Acute retroversion of uterus, followed by dementia, ineffectually treated in an asylum and by various neurologists. Reposition and retention of uterus by supporter speedily followed by permanent restoration to mental health.*

Miss B., nineteen years, slipped down stairs and fell heavily on her seat. Soon after she became morose, irritable, and lost

interest in her surroundings. Sent to a well-known insane asylum in a neighboring city she did not improve, and went the round of celebrated neurologists in our and other cities. In despair her friends finally took her to a notorious female quack who, of course, made a vaginal examination, found a retroversio uteri, replaced and retained the organ, and in three weeks the girl was well. She is now a healthy and happy mother.

I could multiply these instances of the intimate and only too often unsuspected and mysterious relations between the sexual organs and the brain and peripheral nervous system in the female sex almost *ad infinitum*. I could cite cases of apparent paralysis cured by removal of the ovaries (I myself have done such an operation with such a result), or by replacement of a displaced uterus; I could relate instances of mental aberration cured by treatment of the sexual organs, and could draw therefrom most logical deductions and practical suggestions as to the treatment of the female insane, and I could draw out this subject to a length far beyond that originally intended by me or agreeable to my audience. But I will forbear, and take my seat with the hope that this hastily and imperfectly written paper may be generously criticised by my hearers, and may possibly elicit an instructive discussion.

MASSAGE IN NERVOUS DISEASES.

III.

PERIPHERAL AFFECTIONS.—NEURALGIAS AND MIGRAINE.

By Dr. GEORGE W. JACOBY,

PHYSICIAN TO THE CLASS OF NERVOUS DISEASES OF THE GERMAN DISPENSARY OF THE CITY OF NEW YORK.

NEURALGIAS.

PERIPHERAL affections of the nervous system can certainly be more beneficially influenced by massage than central ones; indeed were it not for the truly remarkable results attained in many of the former by this mode of treatment, the entire question of the influence of massage upon nervous disorders would hardly have any *raison d' être*. Not but what perhaps, as has been claimed, single symptoms dependent upon certain pathological conditions can be to an extent alleviated, but to our mind the scope of massage in central affections, even as a palliative agent, is exceedingly limited. Sifting further, we find that of all nervous affections it is in the general class of neuralgias that massage has received the greatest amount of attention, and in which the greatest number and most striking successes have been attained.

The question whether we are here dealing with a disease of the nervous system itself or only with one symptom of such a disorder, cannot in any way affect the results obtained. That in very many cases the neuralgia is only a symptom of some deeper-lying affection will have to be admitted, and the fact duly recognized, but that on the other hand in many cases the neuralgia really constitutes

the entire trouble, and is dependent upon some local change in the nutritive, circulatory, or molecular status of the nerve itself, can also not be denied.

There is only one way of permanently curing the affection, and that is to remedy the condition upon which the neuralgia depends, and that this in many instances can be accomplished by massage will be shown clinically, and can be understood physiologically. In those cases, however, in which our knowledge of the causative influence is at fault, it will have to be our aim, to relieve the pain temporarily. Pain in whatever form it is met with is always pathological. That it is a morbid modification of sensation is what most physiologists suppose, therefore the study of the laws which govern the latter will also give us an insight into the conditions upon which the former is dependent.

What, however, this morbid modification consists of we do not yet know, and until the narrow confines of our knowledge upon this point are considerably extended, our treatment of neuralgias, be it as rational as possible, will nevertheless savor to a great extent of the empirical.

The enormous literature existing upon the treatment of neuralgias furnishes clear proof of our ignorance of the pathology of the affection. The remedies which have been advocated at various times, and which have subsequently been consigned to oblivion as inefficient, are innumerable. Massage will certainly take its place among these discarded remedies, if it be expected to act as an anti-neuralgic. Rubbing and kneading, but particularly nerve percussion, will cure many cases which have resisted all previous modes of treatment, but as may be easily understood, there are very many cases upon which it can exert no influence. Those neuralgias dependent upon some anatomical change in the central nervous system, or those caused by a localized new formation, will naturally not yield to massage. Therefore, if possible, the causal agent must always be determined upon before treatment is undertaken. So, it will be found that if the discrimination can be made between an anæmic and a hyperæmic neuralgia, using these terms as referring to the condition of the nerve itself, the

successes attained by the percussion treatment will be very much increased. This differentiation will also serve us as a guide as regards the length of each application, for as our aim in a hyperæmic neuralgia will be to produce contraction of the vessels, and thus cause a localized degorgement, the séance will have to be comparatively short.

On the other hand, in an-anæmic neuralgia, our object is to produce dilatation of the vessel, and an increased flow of blood to the part, and this, as we have seen, can only be accomplished after a longer period of time.

Among the frequent causes of neuralgias are localized inflammatory products in or around the nerve sheath, thus producing pain by constant or intermittent compression. Theoretical deductions from the physiological action of massage would lead us to infer that those peripheral neuralgias dependent upon such a cause, would be the ones which are most frequently relieved by massage. The fact stands, that this theoretical deduction is fully corroborated in practice. In the great majority of instances in which a neuralgia is found to be dependent upon such exudation, a cure can more or less speedily be effected by massage.

So also localized indurations of muscles, a myositis, for instance, may in the same manner produce a neuralgia which can only be relieved by removing the locus morbi. In these cases the favoring of absorption of inflammatory products, the diminution of compression, etc., are of themselves sufficient to expedite the return of the nerve to its normal condition, and therefore the search after a special antineuralgic influence of massage is superfluous. Norström particularly insists upon the importance of looking for such indurations in every case, and accents the necessity of examining carefully and repeatedly, not only locally, over the seat of pain, but also the entire surrounding territory.

Mezger has called attention to the fact that frequently indurations of the sterno-cleido will produce neuralgias of the arm, and we have in another place shown that exudations in and around joints will produce neuralgias of nerves of the respective extremities, as, for instance, it is frequent to find neuralgias of the circumflex, pro-

duced by periarthritis of the shoulder-joint, and the crural nerve is occasionally affected as a result of exudations in and around the hip-joint. In the great majority of instances, whenever muscular indurations can be found, and even in cases in which they are distant from the seat of pain, whether they are diffuse or circumscript, pressure over them will cause painful radiations along the course of the affected nerve; and in cases in which the neuralgia is an intermittent one, attacks may be produced by pressure over these indurations. This fact will be noticed in all cases in which the neuralgia is dependent upon these deposits, and in all such cases, removal of the induration by means of massage will effect a cure of the neuralgia. The modus operandi of massage in a hyperæmic or anæmic neuralgia has been shown, but besides the good results habitually obtained, when a neuralgia is dependent upon one of the above conditions, we are also able occasionally to effect a cure in cases for whose causation we are unable to find an assignable reason. Theoretically the result may be ascribed to nerve vibration, to molecular alteration, etc., but in doing so we again come back to the unanswered question of nerve action. We must content ourselves with taking the facts as they are. The prime question in all cases is when possible to make a diagnosis of the causative influence, and above all we must be certain that we are actually dealing with a neuralgia. The error of treating a neuritis in the first or second stage with massage, under the supposition that it is a neuralgia, is one that in our eyes admits of no palliation. In our mind any remedy that is potent for good is also potent for evil, and we perfectly agree with Dr. Althaus that massage is not always the harmless remedy that it is supposed to be.

These remarks apply to any active inflammation existing in the vicinity of a nerve.

Neuralgias, in very many instances, recover without any therapeutical influence; or, as experience has fortunately taught us, are easily relieved by any of the standard remedies. If in such cases massage has been used, it would evidently be a misdirection of judgment to attribute their cure to the employment of the massage. The cases which must

be submitted to massage, in order to determine any superiority for this mode of treatment, are old, inveterate cases, which have resisted numerous other therapeutic attacks. The cases observed by us, given below, are all of this class. Experience has also taught us, and others call attention to this fact, that this class of cases succumb more readily to massage than fresh ones. We have occasionally observed that fresh cases, which, for that reason, were expected to yield readily to massage, were most pertinaciously obstinate in adhering to their diseased condition; whereas inveterate cases, upon which the entire list of remedies had been exhausted, succumbed with the best possible grace to a few sittings.

The choice of the manipulation to be employed will depend very much upon each individual case, particularly upon its cause. It will be readily understood that if the neuralgia be dependent upon compression due to an old inflammatory deposit, rubbing and kneading will be of more service than percussion; but, in the majority of cases dependent upon other causes, tapotement or percussion is the form *par excellence* to be employed. In some cases tapotement executed over the affected nerve with the fingers will be found sufficient, in others it will be found advantageous to make use of the percussion hammer, and, in still others—and these form the bulk of all—nothing short of a percuteur will suffice. Our reasons for the preference of a percuteur have already been given. Granville's idea of the action of nerve-vibration in pain, as well as the mode of applying the percuteur, may best be given in his own words. He believes that the pain in a nerve is set up through abnormal vibrations which take place in that nerve. Believing, as he does, that all nerves vibrate physiologically in a certain rhythm, he concludes that when the physiological rhythm is disturbed, from whatever cause, another set of vibrations are produced which produce the pain.

He says: "The rationale of the process of relief is to overpower the tumultuous vibration of the nerve elements within their sheath, by communicating an independent and different vibratile motion to the nerve as a whole. In the

attempt to do this, the pain is for the moment frequently increased, but this exacerbation of the distressed condition, which is the continued consequence of augmented tumult, rapidly subsides—if it occurs, which is not always the case—as soon as the elements are compelled to fall into the same rhythmical vibration as that communicated to their sheath by the regular beat of the percuteur; the discord between the nerve elements and their sheath which is the cause of the pain, and the sensation, subsiding together." "If the pain does not pass away almost immediately after a momentary increase—in short, within a few seconds of the application of the percuteur, it is well to change the rate of percussion. Another point of moment is to withdraw the percuteur instantly when the pain is relieved, and wait until it returns, which it is almost sure to do in a few minutes. Then touch it again." "The application should be precise and very light, and it ought to cease the very moment the pain comes." Granville justly considers the rate of percussion an important point; at the same time his instruments can only give two sets of vibrations, and this is, in our opinion, inadequate. As little as we can accept his ingenious theory of the production of pain as the correct one, so little can we endorse the rule which he gives for the regulation of the rapidity of the strokes. He makes the pain itself serve as a guide for this choice. He says: "I percuss very rapidly if the pain be of a dull, grinding character, or at lower speed if the pain be exceedingly sharp." Our idea is that the effect to be produced should serve as a guide for the rapidity of the blows, as has been previously shown.

To the other directions of Granville we should like to add that in our experience nerve percussion alone is, as a rule, not sufficient to effect a permanent cure. It relieves the attacks, but in the interval of pain some other form of massage will have to be employed. The regularity of the treatment in all neuralgias is important, and it is well to reiterate that treatment during the attacks alone will only alleviate without curing. That we can by massage produce a direct diminution of the increased irritability of a nerve

has been shown. If, then, a nerve is regularly at certain intervals exposed to this sedative action, and this is kept up for a longer or shorter period, it is thus possible to gradually decrease this hyper-irritability, and ultimately to entirely abolish it.

Sciatica.

The opinions of the majority of writers on massage seems to be that of all neuralgias that of the sciatic is the most susceptible to the influence of this treatment. It must, however, not be forgotten that very many cases of this affection are extremely obdurate, and that perseverance and skill are the two main factors in its successful treatment. Our first experiences in the treatment of sciatica by massage were exceedingly unsatisfactory, so that we felt inclined to believe that the sanguine accounts given of cases cured were somewhat exaggerated. Since then, however, experience has taught us that it is continued and persevering treatment that is effective, and that we first became too easily discouraged. At present we can safely say that although the time required in the treatment of certain cases is frequently very long, yet in the majority of suitable cases our efforts are finally crowned with success. Naturally too much must not be expected of it, for in this affection, as in all others, failures will take place, and we have had cases which would not only not yield to the most thorough mechanical treatment, but which ultimately succumbed to the influence of some other remedy. Beuster, among other cases, also relates one which was made worse by the use of massage, and which was ultimately cured by the use of the springs of Teplitz.

Rottmann also reports an old case of sciatica in which all imaginable remedies had been employed, and in which massage also failed to effect any change. The following case also shows the failure of massage, and the subsequent success of another remedy.

CASE. Male, æt. twenty-nine, had had a right sciatica of several years' duration. The original cause was probably exposure to cold and moisture. There were no swellings discoverable along the course or in the vicinity of the sciatic.

The treatment by massage was begun with great expectations. First, tapotement was consequentially employed, Granville's percuteur and other modes, petrissage, etc., all without any result. Patient was treated regularly for nearly ten weeks without the slightest amelioration having been produced. The treatment was then abandoned.[1]

The successes attained particularly in sciatica should not surprise us, for this neuralgia is above all others the type of a purely peripheral affection of the nerve, dependent in the majority of instances upon some anatomical or vascular change of the nerve or its sheath. Other neuralgias, on the other hand, are more frequently symptomatic. Sciatica will also, less frequently than other neuralgias, be found to be dependent upon real dyscrasias. Rheumatism as an etiological factor must here be excepted. If the origin of a sciatica is rheumatic, the result of mechanical treatment is almost certain to be favorable, and in these cases tapotement will be found to be of special value. We have, however, found the percuteur to be wellnigh useless in the treatment of sciatica; the form of tapotement to be used is generally that with the closed hand, or with the stiffly extended fingers. The rubber balls with whalebone handles attached will also be found serviceable. If the sciatica is dependent upon localized swelling or deposit in or around the nerve sheath, or chronic inflammation or exudation in the surrounding tissues, it is necessary to address the treatment to the locus morbi. In these cases tapotement alone will be found of little service, massage à friction and petrissage will necessarily be our mainstays. The fact that an exudation in the pelvis may produce a sciatica, is known, and Winniwärter has shown that if this can be reached, relief is possible. He relates a case dependent upon such a cause, which was cured by pelvis massage.

Schreiber lays very great weight, in the treatment of sciatica, upon active and passive movements, and combines them with massage. His mode is to commence treatment with these movements, and only later in the course of the

[1] This case was subsequently cured by eighteen injections of osmic acid. It is Case 10 of reported cases treated by this means.

treatment to make use of massage proper. He considers these movements of particular benefit in those cases which are accompanied by atrophy of various muscles. He begins with the very simplest movements and successively goes on to the more complicated, thus endeavoring gradually to re-instruct the brain in the exertion of functions which it has from long disuse become dishabituated to execute. He believes that long disuse of certain muscles will bring about a corresponding atrophy in the central portion which presides over these muscles, and that therefore re-education of the secondarily atrophied part of the brain is absolutely necessary in order to obtain action from the atrophied muscles. Our experience and views, in the treatment of sciaticas, differ very materially from Schreiber's. We find that only in the very fewest cases are active or passive movements of any service. Except in those cases which are due to some antecedent affection of the hip-joint, the pain is not only not relieved by these movements, but generally increased. And in those cases in which atrophy of the muscles is present, we find that petrissage combined with electricity will restore their lost activity much sooner and surer than the use of either active or passive movements or both.

The successful treatment of sciatica by massage has been pursued since many years by Mezger, but as his knowledge was propagated to others only through his students, it required some time before the cognizance of its influence in this affection became to any extent general.

Faye in 1872, in speaking of some of the nervous affections curable by massage, before the Society of Medicine of Christiania, mentioned neuralgias of the sciatic as being included among them.

He also reported a case which had been subjected to various modes of treatment for a period of four weeks without any benefit, and which was then cured by massage in eighteen applications.

Johnsen has reported 20 cases of cure in from 11 to 56 sittings. Of a series of 14 cases, 4 were partly relieved by massage in from 11 to 34 sittings, 8 were cured after 14 to 56 applications, and in 2 cases no results were attained.

Winge reports the cure of a case in three months which had existed for nine years.

Gottlieb has published the following case:

M. S. Seen for first time by G. on July 4, 1874. Transient pains in the left leg, radiating from the hip to the back of the knee; pains accompanied by a distinct feeling of tension during exercise of the limb. Slight infiltration of the gluteal muscles of the affected side. The entire course of the sciatic sensitive to pressure. Urinary and digestive organs normal. Massage, July 30, 1874. Can take long walks without any feeling of tension. No sensitiveness to pressure, and no spontaneous pains. In this case a hundred applications were made before an entire cure was effected.

Norström, in his treatise on massage, publishes four cases of cure.

The first case was that of a man who had had a sacro-lumbar myositis for six years. Sixteen months prior to coming under N's care, a right sciatica supervened. The pain was so intense that the patient was obliged to remain in bed for days at a time, without being able to obtain any sleep.

Lying upon the affected side was impossible. Galvanism applied without result. Actual cautery along the course of the sciatic, with but slight relief. Extension of the nerve was suggested, but the patient refused. The commencement of January his condition was as follows: He walks with difficulty, making use of a cane and dragging his leg. In the fleshy portion of the sacro-lumbar muscles an induration of the size of a walnut, with ill-defined borders, is palpable. Pressure over this tumor produces pain. The semi-membranosus and semi-tendinosus and the triceps are contractured. Petrissage of the tumor and of the sciatic in the thigh and the gluteal region. Disappearance of the induration after six weeks. Treatment continued, and after two months the neuralgia is cured.

The second case is one of chronic bilateral sciatica in a very obese woman. The external and internal popliteal branches principally affected. Massage, one séance daily. Cure after a few days.

The third case is one of acute sciatica developed during an attack of acute articular rheumatism. Energetic massage along the course of the sciatic. The séances, which at first were of six minutes' duration, were increased to eight and ten minutes. Daily improvement noticed. After ten days, no more pain, either upon pressure or spontaneous. The patient was, however, incapacitated for five months longer on account of the rheumatism.

In the fourth case the pain reached down to the external malleolus. It is not always present, and is on some days only slight, but on others almost unendurable. Digital examination reveals a slight swelling of the gluteal region, corresponding with the course of the sciatic. After eleven days' massage, decided amelioration. At the end of twenty-three days patient is entirely well, with the exception of a slight feeling of stiffness. After the lapse of seven months, no return. Twenty-three séances in all.

Berghman reports case of a man æt. forty-four. In 1877 he first felt pain along the course of the sciatic. The pain was intermittent. The intervals decreased in length until the pain became constant, and so severe that he had no rest night or day. Electricity, combined with hydrotherapy, was used for over a year. Various modes of treatment external and internal either had no effect or only produced temporary amelioration. B. saw him for the first time at the end of March, 1880. He then walked with difficulty, dragging his leg and resting upon a cane. He occupies the horizontal position the greater part of the time. Sleep is impaired. The slightest movement of the leg causes great pain. The muscles of the gluteal region and of the posterior part of the leg are very much atrophied; also the peronei and the extensors of the toes. Sept. 8th, massage of the sciatic and of the atrophied muscles. Sept. 12th, pains considerably less; the patient walks much better. Nov. 1st, since twelve days, no more pain, except upon violent motion. Since a few days, he can, notwithstanding a certain weakness of the leg, walk quite well, without any pain. The muscles are restored to their normal calibre.

Douglas Graham, in his treatise on massage, also relates a case of old sciatica cured by massage, one séance every other day, in two weeks' time.

Schreiber relates two cases taken from many which he has treated.

1st Case.—Patient æt. nineteen. Right sciatica. Treated for a year by electricity without benefit. In 1878 she used thirty-six Moor baths, without result. During the winter of 1878–9 galvanism was again used with same result as previously.

Dec. 13, 1879, S. began the mechanical treatment, and on Feb. 2, 1880, she was completely cured.

The second case was one of bilateral sciatica. P. æt. twenty-three. After two and a half months' treatment by various means, no amelioration. The pains were so severe that patient could neither walk nor sit. The first day of treatment by massage was March 18th, and it was continued until March 27th, when the patient was very much better, but not entirely well.

Zabludowski refers to four cases of this affection treated by massage, two cases of spontaneous and two of traumatic origin. Of the traumatic cases one had lasted since one and one half years, the other since seven months. After fourteen applications there was marked improvement in both, and after three months they were cured. In the other two patients the affection had existed for five years and three years respectively. In the first case a cure was effected, and in the second a great improvement.

Lately Hümerfauth has also related the history of a patient who had suffered for very many years with sciatica, and for two years had been regularly treated with constant and induced currents, but without any amelioration. On the contrary it seemed as if electricity increased the severity of the pain. H. cured him in fifteen days by massage, consisting principally of the various forms of tapotement, the applications taking place daily and lasting about one half hour each. No return of pain after two years.

Of the various cases under our care during the last few years, the following are selected to show the mode of treatment.

CASE I.—*Right sciatica;* since six years; paresis and slight atrophy of the entire limb; relief of pain after twenty seven applications.

Patient, Louis S., æt. thirty-six, was thrown from a horse when he was thirty years of age. He sustained no injury beyond a severe bruising of the entire right leg, and was able to walk home alone. The following day he complained of a feeling of numbness along the posterior part of the thigh. This gradually developed into a severe pain, so that he consulted a physician. The pain extended from the hip down to the heel of the foot. It kept getting worse and worse, so that at times it was almost unendurable. He also noticed his leg getting weaker. The pain was not continuous. This condition lasted, with more or less variation, during the entire six years. Morphine was the only remedy that relieved the attacks. He was under treatment periodically for the greater part of this time. We saw him first in December, 1880. We found the leg somewhat atrophied, particularly the glutei and the triceps. Pain upon pressure along the entire course of the sciatic. Not able to find any enlargement in the muscles or around the nerve. Massage was recommended.

Dec. 17th.—First application; consisted of effleurage and massage à friction. Although very little force was used, the pain was so great that the séance only lasted about three minutes.

Dec. 18th.—Patient had more pain after the massage than he has had for some time; bears the treatment better; still painful.

Dec. 19th.—Applications more energetic; massage à friction and tapotement; séance lasted five minutes.

Jan. 2, 1881.—Has been massaged almost every day since last note; last séance lasted ten minutes and caused no pain.

Jan. 10th.—Daily treatment has been continued; still has attacks of pain, but they are of short duration and not severe.

Jan. 16th.—Has had no pain since yesterday; his leg also feels stronger; this was the first time that he had been free from pain for twenty-four hours since he began treatment.

Treatment was kept up until end of March, first every other day and towards the end twice a week. As he had then had no pain for over a week and his leg felt perfectly strong, he was considered cured. Has since then remained perfectly well.

CASE 2.—Male. Left sciatica since two years. Myositis. Massage. Cure.

P. æt. twenty-eight, had an attack of articular rheumatism in 1878, the ankle- and knee-joints of the left side being particularly inflamed. After the swelling went down he noticed pain along the course of the sciatic, which was not particularly severe and only lasted a few days when it became well of itself.

He was then perfectly well until 1881 when he had another attack of rheumatism, again localized principally in the left leg, but without swelling of joints. The pain gradually seemed to localize itself more and more along the sciatic, and became very severe. Did not have pain continually, but it came in paroxysms and lasted for several hours at a time. The intervals between the pain grew shorter and shorter until he was rarely free from it. When we saw him in February, 1883, his condition was about the same. Upon examination, there was no atrophy of muscles, and the leg seemed not to differ much from the other. At the lower border of the gluteus maximus, however, was found a circumscribed induration of about the size and form of a pigeon's egg. Strong pressure over this induration produced very severe pain along the entire course of the sciatic. Pressure over the sciatic itself, produced a feeling of numbness but not actual pain. The employment of massage was here recommended at once and no other remedies made use of. After ten daily séances, of petrissage and massage à friction, principally applied to the induration, this had distinctly diminished in size. But the pain was yet as severe and as continuous as ever. After still fourteen more applications, one every other day, the induration had entirely disappeared, and the pain had greatly diminished in intensity and duration. It however still required nearly two months of regular treatment before he

was entirely free from pain; so that he was under treatment for over three months. He has since then had no relapse.

Trigeminal Neuralgia and Migraine.

Neuralgic affections of the trigeminus are probably quite as frequent, and perhaps even more frequent, than those of the sciatic; but in these cases the foundation is much more frequently laid by general causes, such as neuropathic disposition, anæmia, general disorders of nutrition, various dyscrasias, frequently malaria, disease of proximate organs, etc., that we ought not to be surprised if many cases are, so to say, incurable, or if very few cases can be treated in one and the same manner. Perhaps on this account many writers have taken a very unfavorable view of the possibilities of therapeutic influence upon this affection, and Schreiber, in speaking of the treatment of trigeminal neuralgia by massage, says: "As great as is the probability with which we may count upon the favorable action of the mechanical treatment in all neuralgias which are seated in the muscles, so unreliable is the treatment in all neuralgias which are situated between skin and bone." "In particular is it against neuralgias of the trigeminus that mechanical therapeutics battles in vain."

The fact that these neuralgias are occasionally also dependent upon purely local causes, such as thickening and swelling of the neurilemna, might lead us to expect some effect from massage. As regards this cause, Schreiber is right; here we can do nothing; tapotement is of no avail in such conditions, and effleurage and petrissage do not reach the affected part on account of the many bony canals and recesses through which the branches of this nerve pass. But neuralgias of the trigeminus are very frequently dependent upon active or passive hyperæmia of the nerve itself, of anæmia of the nerve, and of circumscribed indurations either near by or distant from one of its branches. We have convinced ourselves that this latter cause is very much more frequent than is generally supposed, and that the majority of these cases can be relieved by massage, and by massage alone. So also in cases dependent upon hyperæmia

and anæmia of the nerve itself. It will be found that although Schreiber is right when he speaks of the unreliability of massage, understanding by that, as he does, the various described forms, including tapotement with the fingers or hand, yet these cases can in very many instances either be cured or the intervals between attacks essentially shortened. It is here that the manner of treatment is all-important. What Schreiber has ignored, and what will be found of supreme benefit in this class of cases, is the rational use of the percuteur. Naturally here, as in neuralgias of all nerves, our diagnosis must be a positive one; we must be careful to select our cases, and it is well to again lay stress upon the fact that a case which will not yield to percussion, may get well upon a few doses of chinin. If we do not pay careful attention to the etiological factors, our failures will, with percussion as with any other single remedy, certainly exceed our successes. We have obtained our best results with our tuning-fork percuteur, and the number of vibrations must every time be regulated to meet the exigencies of the case. The rate of vibration to be employed can only be determined upon, for each case, after repeated trials. The pain ought to cease in a very short time after application of the percuteur; if it does not, it is advisable to change the rate of percussion gradually. Thus, after beginning with the fastest or slowest set of vibrations obtainable with a given instrument, the number of vibrations is decreased or increased until the desired number is reached.

One important fact must be borne in mind, and that is, relief of pain is not cure, and for that reason either this or some other form of massage, as already stated, must be used between the attacks.

The following case illustrates the mode of treatment: P. æt. thirty-four, male. Had an attack of pain along the supra-orbital branch in March, 1884. This pain came on without any appreciable cause; it was very severe, and lasted for twenty-four hours, keeping him awake during the night. He had no return of pain for over a fortnight, when he had another attack, which implicated both the supra-orbital and infra-orbital branches. The severe pain left him

after a few hours, but a dull, sore feeling remained. This soreness became a sharp pain after any exertion—walking up stairs, stooping to pick any thing from the floor, etc. He then began treatment. First internal remedies were tried, but without avail. Then galvanism; also no result. He came to us in Feb., 1884, nine months after the first attack of neuralgia, during all which time the longest period of rest had been the fortnight between the first and second attack. We treated him with the galvanic current for four weeks, and then gave it up as useless.

March 5th.—The percussion treatment was begun. Granville's percuteur was employed. The pain occasioned was so intense that the attempt was abandoned.

March 6th.—Another unsuccessful attempt to use the same percuteur.

March 8th.—Tuning-fork percuteur, disc end, short fork, fastest set of vibrations, applied over the supraorbital foramen. Time of application, one minute.

Pain, which was very severe at first, ceased entirely. Pain returned again after a few minutes. Again applied to same spot with relief of pain. The percuteur was then applied over the infra-orbital, with the same result.

March 9th.—Patient says that he was entirely free from pain for about two hours after the treatment, but that the pain then returned the same as ever.

March 10th.—Application of percuteur over points particularly painful to pressure. After a short time entire freedom from pain.

March 20th.—The percuteur has been applied daily since last note. From night before last until to-day perfectly free from pain (thirty-six hours).

March 31st.—Application every other day since last note. Now only has occasional attacks of pain, lasting from half an hour to several hours.

April 10th.—Patient considers himself well, and is discharged.

Sept. 3d.—No return of pain.

In this case percuteur had been applied twenty-eight times, with the result of a perfect cure. We must not for-

get to state that the patient's wife, who was a very intelligent woman, applied effleurage over the affected nerves every day that he did not come to us for treatment.

Dr. W. H. Neale reports the case of a policeman, æt. thirty-five, who suffered from a trigeminal neuralgia for five and one half years, and who, after having submitted to various modes of treatment in vain, was subsequently cured by the use of Granville's percuteur. The first application was made on June 3, 1884. These were repeated daily with few exceptions until June 26th, when he was entirely free from pain. No further application.

Aug. 15th.—No return.

Oct. 24th.—Still no return.

CASE.—Personal. Neuralgia of nasal and supra-orbital branches. Induration about the size and shape of a bean in the temporal fossa. Massage. Cure.

P., female æt. thirty, had had a peripheral facial paralysis of the right side caused by draught in September, 1882. Recovered entirely in a few weeks. About six weeks after leaving off treatment she felt pain over the trigeminus, of the same side, particularly over the nasal and supra-orbital branches. The pain grew more and more severe, and lasted a longer period of time. When she came to us in October, 1884, she had pain almost continually. Could sleep well. Pain, always brought on with greater violence by mastication. She was first treated by galvanism for several weeks without any result. Then several further weeks were devoted to the treatment by the percuteur, both Granville's and the tuning-fork being employed, but also without result. A careful examination at this time revealed an indurated formation in the temporal fossa of the right side of about the shape and size of a small bean. Pressure over this produced an intense paroxysm of pain along the affected branches, which subsided when the pressure was removed. This experiment was repeated several times, always with the same result. The treatment was then directed to the induration itself. This treatment (petrissage and effleurage) was begun early in January, 1885. The middle of February, during which time she had been subjected to more or less

regular application, the induration had entirely disappeared, and with it the severe pain. There was still, however, a dull feeling of pain along the affected branches, which was, however, relieved by the internal administration of iron.

July 10, 1885.—Has been entirely well for several months.

October 17, 1885.—No pain whatever.

Berghman has reported a case of trigeminal neuralgia in a man æt. fifty-four.

Had lasted about two and a half years. The pain is exceedingly severe, and is brought on by any movement of the face; speaking, eating, or the slightest touch, is sufficient to bring on a paroxysm. The pain commences by a tearing sensation in the vicinity of the labial commissure, and extends upward to the cheek, and downward to the angle of the inferior maxilla. The temporal, infra-orbital, and parotid regions are free from pain. The pain was so intense upon the teeth of the superior maxilla that he had the incisors, canine, and all the molars of that side extracted. No spasms of the facial muscles. After treatment by electricity and morphine, Berghmann began the employment of massage. After six days, the patient asserts that the pain disappears almost completely after each séance. But it always returns. The last day he had one and three quarter hours' rest. The nights are always restless. After three further days he is easy during two hours, and is able to sleep at night. Three more days and the attacks come on only at long intervals. After two weeks of treatment the pain disappeared entirely; he is able to sleep all night, and nothing of the trouble remains except a slight stiffness and insensibility of the cheek. The manipulations consisted of percussion, each séance lasting from five to six minutes.

Norström details a case of neuralgia of the nasal and frontal branches in a female, æt. thirty-six. Suffering since four months. Electricity used for three weeks without any result. Rubbing with various preparations also had no effect. Massage. Cure after eighteen applications. No return.

Wagner cured a case of trigeminal neuralgia, which had lasted two weeks, in eight applications.

Zabludowski also reports a case of supra-orbital neuralgia which was much improved after eight séances of massage.

Douglas Graham says of a male, æt. sixty: "Mr. S. came to me with supra-orbital neuralgia, which had troubled him for a year in spite of tonics, sedatives, liniments, and electricity. I gave him nine massages in three weeks, and he was so much improved that the slight pain left soon disappeared without further treatment." Many cases of trigeminal neuralgia which cannot be cured by massage or any other means, can at any rate be alleviated or temporarily relieved by massage. Of this class Beuster treated three cases, which had persisted for six, eight, and twenty-six years respectively. Tapotement relieved these cases, but only for a time. A permanent cure was not effected, although in the case of six years' standing the condition was very materially improved.

(*To be continued.*)

CONTRIBUTION TO NEUROLOGICAL THERAPEUTICS.

By S. V. CLEVENGER, M.D.,

CHICAGO, ILL.

THE recently-published work on Pharmacology, by Lauder Brunton, foreshadows the comparative therapeutics of the coming era, when it will be realized that only through careful observation of the effects of material medica articles upon all living organisms will we be enabled to discover true rationale. But as an illustration of the utter impossibility of any one, however talented, being able to compass satisfactorily a single branch of medicine, to say nothing of all branches, the book referred to is conspicuous. Such important articles as conium maculatum and secale cornutum are not accorded, by the author of the work, as much consideration as Harley or Trousseau gave them, when to-day in the *armamentaria* of those who treat mental and nervous diseases there are no more reliable and important drugs.

The most ignorant of the laity know of the abortifacient properties of ergot, or fancy they do; while to the accoucheur it is the *vade mecum*. Surgeons and gynæcologists find it valuable in the discussion of tumors and combined with KI. The general practitioner has come to use it to lessen aneurismal swellings. I incline to regard digitalis as more serviceable in aortic aneurism. Hammond ("Nervous Diseases," p. 67) credits Dr. Charles Aldrich (West Riding Lunatic Asylum Reports, vol. i., p. 71, London, 1871) with first noticing "contraction of the arteries of the retina and loss of the capillary tint of the disc" after its use; but

I think Brown-Séquard first suggested that the intracranial blood might be lessened through its action upon the muscular tissues of the cerebral vessels. At least, Dr. J. Crichton Brown (*Practitioner*, June, 1871) accords Brown-Séquard priority, and used it successfully in lessening the excitement of maniacs.

Hammond advised its applicability in active cerebral congestion; but I believe that Spitzka originally conceived the idea of equalizing the circulation of epileptics by its use, and thus aborting the arterial spasm upon which the disease apparently depended.

In my hospital and private practice I have had good opportunities to test the efficacy of special drugs, and since introducing full use of secale cornutum, the internes find diminished use for opium.

More than twenty epileptics at my clinics were benefited by ergot, in combination with natrium bromide. Half these cases I have every reason to regard as permanently made better—if not cured. Every one was treated as individual peculiarities seemed to require, and routine dosing was found to be hurtful. In one case of cerebral gumma, and another with a cardiac lesion, ergot intensified the fits, whereupon it was displaced by specific treatment and digitalis, with good results.

Lessening of the vertigo and psychical aberrations followed its administration in one case of cerebellar tumor, proven to have been such *post mortem*.

Three cases of exophthalmic goitre began to amend with the first drachm of the fluid extract. One female, æt. forty, had been treated for rheumatism and anæmia, the pains and facial pallor misleading the prescribers. The color returned to the face and the intrascapular suffering ceased as the tumor of the neck disappeared under the constringing influence of the medicine.

It is conceivable that want of blood in the face and head may often depend upon dilatation of vessels in other parts of the body, and thus mere symptom-treatment would be absurd. Where such paleness was due to an aneurism, physiologically, the etiolation should have little consideration.

Instead of chloral I use secale with Na Br in delirium tremens, and the sequelæ of alcoholic debauches. The insomnia, tremors, and restlessness are rapidly relieved by this combination, where the chloral temporarily stupefies. In aggravated cases conium maculatum and hyoscyamus are added with prompt results.

I succeeded in restoring the reason and use of his limbs to a post-hemiplegic insane inventor by continued use of ergot with KI. The inception and persistence of the trouble were clearly due to an engorged brain, which, outside of the hospital had rapidly grown worse under large morphine doses given by a homœopath.

Ergot quells the excited stage of katatonia, and may be given during the stuporous period for the purpose of anticipating the succeeding mania. Indeed there is reason to think that all alternating phases of this psychosis are abbreviated by its use.

The full value of ergot is by no means appreciated by the profession, yet, as is the case with every other drug, it is capable of doing injury.

In hysteria, with or without digitalis, I have seldom found it amiss. According to my theory (set forth in "Comparative Physiology and Psychology," 1885), in this condition there is a disparity between the cerebral blood supply to centres and the cerebro-spinal nervous stimulation, so that when excitation occurs, over a cerebral or spinal tract, the necessary customary vaso-motor synchronous action does not take place, or it drives the blood to a contiguous or other point, distant from the centre that healthily should receive the nutrition. Ergot tends to hold the calibre of the arterial system in tone and to overcome such aberrations. Digitalis aids this tonus, through its special cardiac effect, and partially also antagonizes hysteria by the nausea it induces, when taken in pretty full doses.

A case of recurrent congestive headaches in my own family led me to notice the full value of ergot when judiciously given. When these cerebral torments were of the active or arterial type the relief afforded by a teaspoonful dose of the fluid extract was noticeable, particularly in the inception, in

the stage that could be, for the nonce, relieved by pressure upon the carotids. But when the venous engorgement was the main cause of the head-fulness, Paullinia sorbilis or a little wine, answered better, unless the condition were extreme, when catharsis was the only means of relief.

Even in the active hyperæmias, when secale had closed the avenues toward engorging the brain, I saw that pressure was simply transferred from the arterial distribution to the ventricles, and that derivation to the bowels must be conjoined with attempts to meet requirements, by consideration of the primary source of the ailment, affording one of the innumerable evidences that the physician must *think*, and must have a physiological basis for thought, to be able to cope with disease in its multiformity.

A well-marked case of spinal meningitis was brought into the Alexian Hospital, and under my direction was treated with large secale doses by Dr. Hoelscher, the interne. The immediate relief afforded and the rapid institution of convalescence, led both the interne and myself to anxiously review the history, progress, and recovery of the case, as there was no literature to justify such treatment alone. The remissions turned out to be so protracted and the recovery so decided as to leave us no doubt of the value of the medicine in its practical workings. I was induced to use it from purely theoretical considerations easily arrived at by neurologists.

Discussing the subject with a medical general practitioner, Dr. Lydoton, he tells me that he has had good results in the use of secale in the hyperæmic stage of pneumonia. Theory led me to use it in the trivial annoyance occasioned by parotitis in two children, with favorable apparent reduction of the swelling, and Dr. Hoelscher informs me that he has used it in epididymitis and phlebitis with satisfaction. He is somewhat enthusiastic, not unjustifiably so, in its use since I introduced it to his notice, and together recently we witnessed the decided relief it afforded a cerebral erysipelas case in clearing up his mind and diminishing the painful facial turgescence.

I can see why in a case of terrible *delirium grave* secale

availed nothing, when the brain I took out after her death was œdematous in the extreme. Acute drastics might have been better, though I expect only temporarily so.

Dr. Seguin's interesting addition (in the January number of this JOURNAL) to our knowledge of optic-disease association with the gyrus cuneus, so satisfactorily elaborated by Exner, is timely. There are other optic disturbances connected with injury to this region, which I resist the temptation to speculate upon in what is intended to be a purely clinical, therapeutic article, and shall here merely mention that a gentleman sustained a contused hurt to his skull thirty years ago, above and over the torcular herophili, and subsequent to business anxieties was treated for delirium tremens unjustifiably. Falling into my hands, I lessened the circumscribed hyperæmia, toned up his general system, endeavored to soothe cerebral excitement with hyoscyamus, and withheld chloral or any thing else which afforded merely temporary relief and that did not ensure equable blood distribution. Needless to say I used secale, and, after a six months' bout with optic hallucinations, during which he avers he "did not sleep a wink," to-day he is attending to his business nearly as well as a year ago.

I have not lost consideration of my old preceptor's disquisitions against the undue effects of this medicine, by any means, but have found occasion to modify my ideas derived from him and "the books" considerably. The gangrene is only likely to occur in senile cases and where used by "tradesmen doctors" unphysiologically. Hammond is right in stating that dosage is too guarded—that we do not use enough. I have found that three drachms daily of the fluid extract was effective in a case of cervico-brachial neuralgia, due to effusion in the course of the brachial distribution, with a precedent rheumatic etiology, where previously ten-minim doses had been unserviceable.

A priori we might expect interference with the catamenia by its prolonged use. I have been puzzled to note that no such thing has taken place. On the other hand, in four female cases of epilepsy there was an increase in the menstrual discharge, and, *mirabile dictu*, epistaxis accom-

panied one month's experience. I ascribed this access to the bromide, which was given at the same time, but am by no means satisfied that this was the cause of the hemorrhages.

Drachm doses of the fluid extract have, in my practice, repeatedly *completely* relieved the dull cephalalgia of typhoid fever, and stopped the nose-bleeding that depended upon the same condition.

As an evidence that the use of secale cornutum is not, in epilepsy, a transatlantic innovation, I will state that I was called in consultation by a German physician in this city to attend his brother-in-law, who had suffered a year or two from epileptic paroxysms. The doctor saw at once the physiological reasons for the use of the arterial equalizer, and stated that he had just come from Erb's clinics in Germany, and the only thing mentioned there was bromide of potassium for this disease.

Hammond's latest edition, Ross' and Webber's recent works also do not hint at it.

In addition to the instances wherein it was above mentioned as serviceable, I have used it effectively in hypochondria, paretic dementia, melancholia with cardiac weakness, facial flushings, associated with menstrual irregularity, hemicrania, opium habituation, and insomnia from cerebral hyperæmia.

It is seldom of use in coarse brain disease, or in cerebral syphilis. I have reason to think one case of transitory frenzy was caused by it, and at the Home of the Incurables in this city a female is said to be suffering from multiple cerebro-spinal sclerosis, through excessive use of ergot during an attempt to dissipate a uterine tumor. Cases of locomotor ataxia are reported from its undue use. Where it has been found serviceable in ataxia I incline to doubt the diagnosis, as aneurisms may induce conditions simulating tabes dorsalis.

Neither in chronic nor acute chorea has it proven useful in my experience, and once I noticed a tendency toward alopœcia in a patient using it, which ceased on discontinuance of the drug.

In these cases, when giving diuretics, such as acetate of potassium, spts. æth. nitr., and ergot, at the same time, strangury occurred, from which I inferred that when the kidneys required controlling, this combination, guardedly used, might prove to be of value.

Squibb's fluid extract is the only reliable preparation, in my opinion. The solid extracts, including Bonjean's ergotine, I am perfectly satisfied are not to be depended upon.

The active principles of ergot are essentially amides or amines, both of which pass to volatile compounds, which the presence of alcohol in the fluid extract would restrain. Hence solids are apt to decompose. The latter have a nitrogenous odor resembling beef extract, which indicates an unfavorable change in the drug.

The newly introduced antipyrine has been largely used at both the Alexian and Michael Reese hospitals in this city, in typhoid-fever cases, and occasionally with good results in traumatic and phthisical elevations of temperature. A Polander suffering from pulmonary consumption at the Alexian was brought in with well-marked phthisical insanity, destructive and suspicious. Pulse rapid and weak. Noticing the temperature to be 103°, I advised Dr. Hoelscher to use about seven grains of antipyrine, and observe the influence it had upon the mental condition as well as heat reduction. In an hour the insanity ended, after a duration of two weeks, and after two weeks' further sojourn in hospital, the case was discharged cured as to the psychosis, and improved slightly with reference to the lung trouble.

The case of recurrent cerebral congestion, referred to in this paper, wherein ergot had been tried, was complicated with an excessively irritable stomach, causing the frequent rejection of the fluid extract, and, as before stated, the solid extracts did no good. In conversing with my friend, Dr. Thilo Brauns, I asked him if he regarded the cotton-root bark he used in his gynæcological practice as equalling secale as a constringent of the uterus. He replied that he had come to rely upon it as better than the ergot. As the physiological action of the two drugs must be the same, and the gossypium does not nauseate, I concluded to try it in

all cases where previously I had used the ergot, with the following results: Gossypii radicis cortex acts more energetically than ergot in reducing the calibre of arterioles. Its action is swifter—the effects being manifest in two to three minutes. The pulse-rate dropped in that time from 112 to 78, in a case of cerebral hyperæmia in which it was used by me.

Up to the present writing I have prescribed gossypium twenty-two times in

3 cases active hyperæmia,
9 " epilepsy,
1 case post hemiplegic insanity,
2 cases aneurism aorta,
1 case angio-paralytic hemicrania,
1 " spinal congestion,
2 cases delirium tremens,
2 " cephalalgia from debauch,
1 case traumatic cerebral meningitis,

with favorable results.

I find it best to use less of the gossypium than of the ergot, and, approximately, might estimate the energy of the two drugs as represented by doses of $\frac{2}{3}$ or $\frac{3}{4}$ the former to one of the latter.

I noticed also that the contraction induced by the gossypium was more enduring, and, in the active recurrent cerebral hyperæmia case alluded to, the effects of a few doses prevented attacks for two weeks, which, theretofore, had occurred nearly every other day. But I was compelled to desist from its further use, when—in lieu of the frenzied headaches—cardiac and gastric spasms succeeded, due, in my opinion, to either effusion or thrombi (compression symptoms) in the pneumogastric centres. These dangerous paroxysms disappeared after a hemorrhoidal hemorrhage, to be followed in a few days by a return of the headaches, while the gossypium and all other remedies were refused, as even food could not be tolerated. Atropine and morphine hypodermatics were then resorted to instead, affording some relief, but this was a final resource, for organic disease of the brain has advanced to such a stage as to render all but palliative methods useless.

Clinical Cases.

A MORAL IMBECILE.

By JAS. G. KIERNAN, M.D., Chicago, Ill.,

late medical superintendent cook county hospital for the insane, member society of medical jurisprudence, etc.

In presenting the present case I simply wish to call attention to certain features of forensic importance. Mrs. T., Irish-Celt, was an intelligent, well-behaved, and for her station, refined wife and mother, up to an attack of typhus fever attended by a high temperature and delirium. Subsequent to recovery from this there were observable a coarseness not present before and a tendency to malicious mischief toward her husband, whose sense of propriety she took an especial delight in outraging. She indulged in sprees, not because she liked alcohol, but because the sprees by the shock they gave her husband gave her an intense pleasure. Her conduct was so demonstrably outrageous, that even the very obtuse Illinois juries, which try the insane as if they were criminals, readily adjudged her insane, but this diagnosis was based simply on her acts, which were totally out of accord with her surrounding, and of such a seemingly immoral character as to be ordinarily called simply wickedness. When she first came under my observation, my attention was attracted to her by the fact that she clearly realized the nature of every act she committed, but as she had been badly treated by the political attendants with whom she was brought in contact she was distrustful, since they treated her, as the world does such cases, as a criminal worthy of punishment.

Her own description of her case at the time I last examined her is as follows: She is insane, a thief, and a liar, and from the "nature of her mind" she believes it all to be a disease, capable of treatment by medicine, and asks to be cured. She says that sometimes she has "weak spells of mischief," which she can control if people do not look down on her and treat her as a "bad woman."

She is very witty at times. At one time she took a great delight in soiling my clothing and pulling my whiskers, but later explained that she had "a great power on her to do this." The "will part of her mind was weak," but my kindness and "doctor's talk about her insanity" made it stronger when she had only "weak spells of mischief."

Her "strong spells" come on suddenly like "fits of the falling sickness"; she "knows what she is about" but "can't help it." These "spells" are preceded by a "dull head" and then she knows the "power of the strong spells" is coming on. She "does not hear voices nor any nonsense of that kind" but this "power of the strong spells" comes on her in "thoughts," which have a power over her like the "fits do over people in the falling sickness."

While it is obvious that she acts under the influence of imperative conceptions, the acts so performed are somewhat complicated. Thus she would quietly take her window from its frame and noiselessly put it over the transom of her room door, suspending it by a sheet. By the time the attendant could reach the room she would be in bed apparently asleep. At nine o'clock one night she took a new pail noiselessly apart and placed its staves on the transom of her room, balancing them adroitly.

She has, according to her own account, "strong spells of secret love," when she smashes windows to feel "happy" from seeing the blood run from her cut fingers. It is obvious that these are erotic attacks. When these come on she uses obscene language if she can't smash glass and see the blood run. She is healthily depressed after these attacks, from a sense of shame, for her old modesty comes to the surface at times and, for the time being, dominates her.

I have designated this a case of moral imbecility, and have so likened it to the psychoses which are secondary to the essential fevers.

The points of forensic interest are:

First—The imperative and complicated nature of the acts performed.

Second—The recognition of their immoral nature by the patient, who also recognized their imperative character.

Third—The fact that these acts were of so demonstratively an insane character as to convince, despite their immoral nature, even an Illinois insane trial jury of the patient's insanity.

The psychiatrical interest of the case, does not lie so much in the fact of the rarity of such a case as it does in the patient's intellectual analysis of her mental condition, and the powers of observation she displayed in her comparison of her mental condition to that of the patients around her.

Periscope.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

Division of the Posterior Commissure of the Brain. *Pflüger's Archiv*, Band 38, Heft 3 and 4.

Dr. Darkschewitsch has made a series of experiments upon this subject.

The changes of the iris by the light reflex, which ensues after a lesion of the posterior commissure, permit the following conclusion to be drawn : a lesion of the posterior commissure causes a depression of the irritability of the oculomotor nerves. The degree of the diminished irritability is dependent on the extent of the injury to the fibres of the posterior commissure. A complete destruction of the posterior commissure causes a complete loss of irritability of the oculomotor nerves, as regards the light reflex.

The Nerves of the Pupil. *Pflüger's Archiv*, Band 38, Heft 5 and 6.

Miss Schipiloff and Prof. Schiff have made a series of experiments upon frogs in regard to this subject. She holds that nerves dilate and contract the pupil, but protests against the way they do this as stated in the text-books. The nerves of the sympathetic going to the eye contain dilator nerves, in the sense that their irritation dilates, and their paralysis lessens the dilatation. The normal physiological dilatation is not called out through the sympathetic but through the letting up of the activity of the oculomotor.

In regard to the activity of the oculomotor and its relation to the sympathetic, she has made a few experiments upon frogs. She tried to find out how the activity of the sympathetic is caused, or when the oculomotor is paralyzed. In frogs the nerve was cut within the skull, and a considerable dilatation ensued. If now the frog was frightened or irritated in the sensory sphere, the pupil became larger, and soon after the cessation of the irritation it returned to the size it was before. After a longer or shorter time the sympathetic, at the level of the uppermost vertebra, was divided. This caused a slight narrowing of the pupil, notwithstand-

ing the paralysis of the oculomotor. The pupil dilates upon irritations of the skin, of whatever nature, and this dilatation is due to the sympathetic, and disappears by complete section of the roots of the nerves going through the sympathetic to the iris.

The influence of ocular movement upon the size of the pupil in frogs is small. She does not believe in the tonic influence of the ganglia along the sympathetic upon the pupil, but that all tonic influences are in the central nervous system, and that they are not spontaneous, but called out by sensory peripheral irritation.

Effect of the Long Ciliary Nerves Upon the Pupil. *Du Bois' Archiv*, 1886, 1 and 2 Heft.

Dr. J. Jegeron has studied the influence of these nerves upon the pupil in the cat and dog. The animals were curarized, and the nerves were irritated by mechanical or electrical means. His experiments led him to draw the following conclusions :

1. All dilator nerves of the pupil pass to the ocular bulb without the mediation of the ciliary ganglion. 2. After section of all the long ciliary nerves the pupil is contracted, but preserves its regular form. Section afterwards of the sympathetic causes no increased contraction of the pupil, and irritation of the peripheral and of the sympathetic or the central end of a sensory nerve causes no dilatation of the pupil. 3. The irritation of the peripheral end of a long ciliary nerve causes a partial contraction of the iris—that is to say, a one-sided dilatation of the pupil.

If all the long ciliary nerves are not cut, the pupil is narrowed, but has an irregular form. If now the cervical sympathetic is cut, then the pupil is narrower, whilst the form of it is regular. If the peripheral end of the sympathetic is irritated, or the central end of a sensory nerve, then a dilation of the pupil ensues, although irregular in form.

Hence it is demonstrated that the dilator fibres of the Gasserian ganglion leave it through the first branch of the trigeminus and reach the iris through the long ciliary nerves.

Periodical Respiration and Superfluous Respiration. *Archives Italiennes de Biologie*, tome vii , fasc. i.

Prof. Mosso, of Turin, has made a series of experiments upon this subject. He finds that the movements of respiration are not always uniform and regular. In profound repose, and specially in sleep, there are in man, as in animals, groups of inspirations, whose amplitude successively increases and diminishes. The old view, that there is only one respiratory centre, should be abandoned, as the muscles of the face, the diaphragm, the thorax, and abdomen have special centres which act in an automatic manner. The number and amplitude of the respiratory movements are not always in direct relation with the respiration of the tissues and blood, and proportional to the need of oxygen introduced or of carbonic acid exhaled. Inhalations of oxygen and artificial respiration are not

able to modify the periods and intermittency of the respiratory acts. It is not possible to admit an absolute, continuous relation between the mechanical and chemical part of the respiration. The study of the circulation of the blood in the forearm shows that the blood-vessels do not take part in the phenomena of remittent, periodical respiratory movement. Periodical respiratory movement is not in direct relation with the vascular phenomena. The changes of the circulation in the nerve-centres are not able to produce the phenomena of periodical respiration.

The long pause and the intermittence in periodical respiration are not due to apnœ, as Lilehne believes. He explains the periods in respiratory acts as follows: That when the nerve-centres incline to rest, they forget to set up respiratory acts, and the organism does not perceive this short pause in the respiration.

ISAAC OTT.

Structure and Function of Vascular and Visceral Nerves. W. H. GASKELL. *Journal of Physiology*, vii., No. 1.

The spinal cord of the dog has connections at every level with the sympathetic system by certain visceral branches of the spinal nerves, which pass to the ganglion lying on the vertebral column (lateral ganglia). From these ganglia nerves pass to a second set of ganglia (collateral ganglia), and from these, in turn, nerves pass to the third set of ganglia (terminal ganglia) in the organ or vessel walls. The first set is the proximal set; the second and third sets are distal sets. Each spinal nerve has a visceral branch. Each thoracic visceral nerve has apparently a white and a gray portion. The white portion issues in both anterior and posterior nerve-roots from the cord, but is only found in the nerves from the second thoracic to the second lumbar segment of the cord. Above and below these levels the entire visceral part of the spinal nerve consists of the gray portion only. The white rami communicantes pass not only to the lateral ganglia opposite their exit, but also turn up and down, and thus reach both the cervical ganglia above and the lumbar and sacral ganglia below. They alone make the connection between the spinal cord and the sympathetic system; for the gray portions really spring from the lateral ganglia and pass to the spinal cord, supplying the spinal nerves, the vertebræ, and the membranes of the spinal cord, and having no function in transmitting impulses from the spinal cord outward. All impulses from the spinal cord to the sympathetic system make their exit in the nerve between the second thoracic and second lumbar nerves, inclusive. These nerves are the only ones (with two exceptions soon to be mentioned) which contain very small nerve fibres—5.4 μ to 3.6 μ in diameter. Hence the conclusion is reached that the calibre of the visceral nerve fibres is small. Such small fibres are also found in the second and third sacral nerve-roots which constitute the nervi erigentes. Gaskell proposes to call the rami communicantes from the spinal cord to the cervical

ganglia the cervical splanchnics; those to the abdominal ganglia the abdominal splanchnics; those to the nervi erigentes the pelvic splanchnics. In the spinal accessory nerve small fibres are found which collect together and pass to the ganglion trunci vagi. These are the only visceral fibres from the cervical cord. The vagus and glosso-pharyngeal contain a few such fibres in the medulla.

The distribution of the visceral nerves is summed up as follows:

They come from the central nervous system in definite sacral, thoracic, and cervico-cranial regions, whence they pass into the ganglia of the visceral system.

From the sacral region they pass out in a single stream to the ganglia of the collateral chain.

From the thoracic region they pass out in a double stream, one to the ganglia of the lateral chain, the other to the ganglia of the collateral clain.

From the upper cervical region they pass out in a single stream to the ganglia on the main stems of the vagus and glossopharyngeal nerves.

Vaso-motor (constrictor) nerves for all parts of the body can be traced as bundles of the finest medullated fibres (varying in size from 1.8 μ to 3.6 μ in the anterior roots of all the spinal nerves between the second thoracic and second lumbar inclusive, along the corresponding ramus visceralis, to the ganglia of the lateral chain (main sympathetic chain), where they become non-medullated, and are thence distributed to their destination, either directly or after communication with other ganglia. There is no satisfactory evidence for the presence of vaso-motor (constrictor) nerves in the roots of the cranial nerves. All the vaso-constrictors originate in the thoracic portion of the spinal cord.

The visceral motor nerves, upon which the peristaltic contraction of the thoracic portion of the œsophagus, stomach, and intestines depends, leave the central nervous system in the outflow of fine medullated visceral nerves which occurs in the upper part of the cervical region, and pass by way of the rami viscerales of the spinal accessory and vagus nerves to the ganglion trunci vagi, where they become non-medullated. They therefore resemble in structure and distribution the vaso-motor nerves.

The vaso-inhibitory or vaso-motor nerves are medullated all the way to their termination, which is in the distal ganglia of the sympathetic system; whether they there become non-medullated and pass on to the terminal ganglia, is undecided. The inhibitory nerves of the lumbar muscles of the alimentary canal in its appendages leave the central nervous system in the anterior roots and pass out among the fine medullated fibres of the rami viscerales into the distal ganglia, without communication with the proximal ganglia.

Fibres which are medullated from the cord to the ganglia, lose their medullary sheath at the ganglia. Each fibre passes out of the ganglion not as a single non-medullated fibre, but as a group of

non-medullated fibres. The ganglion cells not only assist in the conversion of a single nerve fibre into a group of fibres, but at the same time are centres for the members of the group, in so far as they possess a nutritive power over them ; they are not, however, centres in the sense of being capable of reflexly setting these fibres into activity. M. A. STARR.

PATHOLOGY OF NERVOUS SYSTEM.

Le Rhumatisme Cérébral, la Folie Rhumatismale, et la Goutte Cérébrale. LEGRAND DU SAULLE, *Gazette des hôpitaux*, pp. 57, 58, 59, etc.

In a series of lectures Legrand du Saulle calls attention to the various disorders of the intellect found in rheumatic and gouty patients. After first speaking against the prevailing tendency of considering insanity as a morbid entity, he says that more attention should be paid to the connection between mental affections and the various diatheses and dyscrasias. So he now wishes to call attention to the influence of rheumatism and gout in the production of these disorders. The attacks of cerebral rheumatism (rheumatic insanity) may be either overacute, acute, subacute, or chronic. The meninges in this affection comport themselves in the same manner as serous membranes in other parts of the body, those of the joints, for instance, and may be attacked in the same manner. The lesion and process are the same in both. The same as that joint which is the weakest is the one generally attacked in rheumatic patients, so the meninges are generally affected in those whose nervous system is weakened through whatever cause. Therefore heredity is an important etiological factor. Hysterical and epileptic patients seem to form an exception. Those addicted to alcohol, on the contrary, are frequently affected. The better class of patients are the ones most frequently affected for the reason that their brain is more used and that they suffer more from cerebral fatigue. Therefore intellectual pursuits and severe mental efforts act as exciting causes. The overacute and acute forms are preceded by certain prodromal symptoms. They rarely occur before the fifth day of a rheumatic attack ; generally between the fifth and twentieth. Exceptionally high temperature and intensity of cardiac manifestations are significant, but restlessness of the patient, moral anxiety, preoccupation for the future, transitory delirium, and persistent insomnia, are to be looked upon with the greatest apprehension. The overacute form is sometimes preceded by one or another of these symptoms, but generally it attacks the patient with intense suddenness. This is the rarest of all the forms, having occurred only five times in sixty-nine cases of cerebral rheumatism. The acute form occurred fifty-two times in the sixty-nine. It is preceded by the various symptoms : the temperature rises, the pulse increases, muscular twitchings occur, and the delirium appears. The delirium is generally of a low-

muttering type, which is exceptionally followed by convulsions. either eclamptic or choreic. Soon somnolence and coma set in, Death may occur in from twelve to twenty-four hours, or may not occur for five or six days. The subacute form is the one generally known as rheumatic insanity, or rheumatic mania. It is exceedingly rare, according to Simon, occurring once among four hundred insane, and once among one hundred rheumatic, but Bull considers even these figures too high. This form generally occurs when the rheumatic fever is on the decline. Sometimes, however, it sets in during the rheumatic attack itself. The first symptoms occasionally consist in a violent delirium, somewhat similar to the delirium of the acute form of cerebral rheumatism, but more frequently the cerebral affection takes on a melancholic or depressive character. The author cites various cases of this form. The most prominent symptoms are: (1) A depressive, melancholic delirium; (2) hallucinations, particularly of sight, but also of hearing, taste, smell, and touch; (3) a weakening of the intellectual faculties, always well marked and sometimes permanent; (4) choreiform movements; (5) a profound cachexia. Generally this form terminates in recovery. When death does occur it is due to the progressive cachexia. The author then enters into the question whether rheumatic insanity is really a special form of insanity, or a simple melancholia, attacking persons so predisposed. This is decided in favor of its being a special form. "Rheumatic insanity is a reality." GEO. W. JACOBY.

Alcoholic Paralysis. J. DRESCHFELD. *Brain*, xxxii., p. 432. The observations of Dreschfeld on alcoholic paralysis, published in 1884, have excited general interest, and elicited a number of able articles with numerous reports of cases, both in Europe and America, to which reference is made in this (his second) paper on the subject. He now divides alcoholic paralysis into two groups, according to the more prominent symptoms present: the alcoholic ataxia, and the alcoholic paralysis. In the ataxic form it is necessary to distinguish between cases where there is marked incoördination without much paralysis, and those where the gait resembles the ataxic gait but is in some measure due to the paralysis of the legs. In both, severe lancinating pains in the legs, and occasionally in the arms; spots of anæsthesia and of retarded sensibility; tenderness in the muscles of the calf, and absence of tendon reflexes are present. Argyll-Robertson pupil is not found, and there are no ophthalmoscopic changes. In the latter cases a moderate atrophy of the muscles is present and reaction of degeneration is to be elicited. In the paralytic form there is marked progressive paralysis with atrophy and reaction of degeneration in various groups of muscles of the legs, so that the patients are unable to walk at all. There is loss of tendon reflexes. Cutaneous anæsthesia and muscular hyperæsthesia are well marked. An occasional sudden "giving way" of the legs, and consequent falling, is observed in some cases.

In both forms of alcoholic paralysis the mental condition is markedly changed. The patients may answer questions without hesitation or incoherence. But they are subject to peculiar delusions ; fancy themselves in places far from their actual home, and give long and detailed accounts of things that they have seen, walks, and even journeys, that they have taken, or visits that they have made, when in fact they have not been out of bed. One patient said that he got up every day, went into the next ward, and conversed with other patients, though he had never left the bed at all. When further pressed he gave a description of the ward, and details of his conversation with other patients, with a minuteness and readiness which was astonishing. (The recorder can confirm this by an observation recently made upon a lady, who gave, with many particulars, a long story of having made a call upon a certain physician on the morning of the day on which she was seen, of having to wait in his office, of her conversation with the waitress, of the sudden departure of the physician without seeing her, and of a message she left for him. She had not been out of her room at all on that day.) Dreschfeld considers this mental state as characteristic of alcoholic conditions.

H. Hun, in a very complete review of this subject (*Amer. Jour. of the Med. Sci.*, 1885, April), considers mental delusions, delirium, and loss of memory constant symptoms. The delirium is never as active, however, as in an acute attack of delirium tremens.

All authorities agree that the lesion in alcoholic paralysis is a multiple peripheral neuritis. The prognosis is good when the general strength of the patient is fair. The chief danger is heart failure or some intercurrent disease. The treatment consists chiefly in cutting off the supply of alcohol, either suddenly or gradually. Warm baths, milk diet, tonic treatment, and strychnine are the remedies usually found of service.

Loss of Pupil Reflex to Light. *Neurol. Centralbl.*, 1886, No. 1.

Uhthoff has investigated a very large number of individuals, both healthy and diseased, with the view of determining the conditions under which the pupil fails to respond to light. In no healthy individual was this condition found, though several hundreds were tested. It was lost in 136 cases out of 550 nervous cases and 12,000 cases of eye-disease. Of these cases 92 were suffering from locomotor ataxia (64 % of the cases of this disease examined presenting this symptom) ; 12 were suffering from dementia paralytica ; 8 had a gross cerebral lesion ; 11 had cerebral syphilis ; 2 had a congenital defect of the iris ; 2 had multiple sclerosis ; 2 were suffering from railway spinal shock, and in one case of retinitis pigment., of head injury, of aneurism of the aorta, of abuse of tobacco, and of hystero-epilepsy, it was present. The symptom is, therefore, confined to a very few classes of disease, and is, therefore, of important diagnostic value.

A New Method of Testing Tactile Sensibility in Unilateral Brain Lesion. H. OPPENHEIM. *Neurol. Centralbl.*, 1885, No. 23.

In unilateral brain lesion a partial hemianæsthesia is often present, which may be overlooked. If the slightly anæsthetic side is touched, the patient says he feels the touch, and is sometimes unable or unwilling to admit a difference in the sensation between this and the unaffected side. Oppenheim finds that in these cases, if the patient's eyes are covered, and the symmetrical spots on the body or limbs are touched at once on both sides, he will perceive the touch on both sides if no anæsthesia is present ; but on the unaffected side only, if the other is slightly anæsthetic. The recorder can confirm this statement as tested on two cases.

M. A. STARR.

On Two Cases of Tabes Dorsalis with Presence of Knee-Jerks. By Prof. C. WESTPHAL. (Discussed at meeting of March 8, 1886, of the Berlin Society of Psychiatry and Nervous Diseases, and reported in the *Neurol. Centralblatt*, March 15, 1886.)

The first case is that of a man fifty years of age, whose disease began in summer of 1882, and who was received into the Charité in July, 1883. At that time there was distinct ataxia, a peculiar rigidity following upon abduction of the thigh, and upon rapid flexion of the knee, diminution of power in the lower extremities, marked disturbances of sensibility, incontinence of urine, etc. Knee-jerks, at first easily elicited ; they were diminished in September, and were totally abolished in October, 1883. The autopsy made at the beginning of 1884 revealed an affection of the external portion of the posterior columns which extended up to the margin of what W. terms the root-zone of the posterior columns, which zone is adjacent to the region of the substantia gelatinosa Rolandi. There was also disease of the columns of Clarke (involving both the nerve fibres and the nerve cells), and of portions of the lateral columns.

The second case was very similar. Beginning of disease in 1882, with ataxia, motor paresis, etc. Knee-jerks normal until Nov. 24, 1884 ; Jan. 17, 1885, they were very weak ; Jan 23d they had disappeared altogether ; Jan. 24th, death. A complicated affection involving the lateral columns and the columns of Clarke ; the gray degeneration of the postero-external columns extended outward as far as the "root-zone."

Westphal thinks it a mere coincidence that various portions of the cord were found diseased. He opposes the views of Déjerine and others that the affection of the lateral columns in such cases as these is due to meningitic processes ; in these cases there was no thickening of the pia over the lateral columns. In the cases just quoted Westphal attributes the motor paresis and the rigidity to the affection of the lateral columns. [The reader is also referred to a case reported by Westphal in which there was

presence of knee-jerks in spite of affection of the posterior columns, which (case) was reviewed in the preceding number of this JOURNAL.] B. S.

Sclerose primitive des cordons latéraux de la moelle, ou tabes spasmodique. Dr. E. BOMPARD. *Gazette des hôpitaux*, p. 35, 1886.

Case of a man aged thirty-three years. Entered the hospital Oct. 23, 1885; has always been well. Family history unimportant. In October, 1881, he was exposed during an entire night to a severe rain-storm. Two weeks later, severe shooting pains in the lumbar region. These pains lasted for several months. He then noticed a weakness of the genital functions, and a lassitude of the inferior extremities. In April, 1882, he observed that he dragged his feet somewhat, and that he *wore out his shoes at the toes*. Pains had disappeared. Difficulty of walking increased. In January, 1883, he was obliged to use a cane. In April his feet dragged along the ground while walking, and he could hardly lift them a centimètre from the ground. The legs now commenced to become stiff. When seated, a severe tremor would occur in the legs. In November he could only walk by aiding himself with his arms, dragging himself along by holding on to objects in the room. At this time a weakness and stiffness of the upper extremities became apparent. January, 1884, complete impotence of both inferior and superior extremities. The condition at time of writing was as follows: No affection of speech, no anæsthesia, no hyperæsthesia. Electric contractility of muscles preserved. Increased reflexes. Severe ankle clonus. Legs stiff and can only be slightly flexed by the patient. Arms contractured and in proximity with the body. The forearm flexed upon the arm at right angles, and the fingers flexed upon the palm. The patient is unable to execute any movement with the fingers. The contracture can be forcibly overcome and the arm extended, but it returns again to the acquired position. Patient was treated in the hospital, but left in December, not wishing to remain any longer; for this reason no autopsy was obtained. Owing to the rarity of these cases the author publishes this one, incomplete as it is.

Paramyoclonus Multiplex. P. MARIE. *Progrès médical*, p. 152, 1886.

The case described by Marie is the third of the kind, the first having been observed by Friederich and the second by Löwenfeld. Marie's case is as follows: Patient—male, age fifty-two years. Family history unimportant. Had a chancre at the age of twenty. No secondary symptoms, no specific treatment. Married at age of thirty-four. One child after twelve years, which has always been healthy. At age of twenty-seven patient had dull, not lancinating, pains in the legs. At this time he also had dull pains in the arms and shoulders, also pains between the shoulder blades.

In the morning upon awakening his arms were heavy and cold, so that he could hardly lift them. His legs were weak, and he grew fatigued easily. Early in August he had an attack of vertigo, which was followed by loss of consciousness, lasting for about twenty minutes. Subsequently delirium. Since about three years he has had shocks, agitative movements (sécousses) in the lower extremities. When the patient first came under observation it was noticed that from time to time a peculiar movement took place at the knee joint, which could not be clearly described either as flexion or extension. In standing these movements only occurred at more or less long intervals, but when the patient was made to lie down they increased very much in frequency and intensity. These shocks occur sometimes at intervals of several minutes, and sometimes at intervals of as many seconds; they are sometimes isolated and sometimes extend over several muscles. Their general seat is over the muscles of the thigh, producing either a slight movement of the thigh itself or a slight jumping movement, a kind of sudden extension of both legs, with almost immediate genuflexion. These shocks are also observed in the muscles of the trunk and shoulders. Voluntary movements were not impeded. Certain excitations seemed to increase the phenomenon, as, for instance, percussion over the patellar tendon. The idio muscular contractility is very much exaggerated. Percussion over the muscles themselves also produces the peculiar agitation. It seems to Marie as if the irritation of the skin itself bears a great part in its production. Tickling the soles of the feet produces these shocks with more certainty and intensity than any other means. Tickling of other parts of the body gives no result, but pricking with a pin does. A prolonged pressure exercised upon the external vastus of the left side produces distinct contraction of the right triceps, about forty-four per minute, while the compressed side contracts only infrequently. Position of the limbs also seems to influence the contractions. During sleep the contractions cease entirely. Various myographic tracings were taken, for which and for more detailed description of the symptoms we must refer to the original. Electrical examination showed no modification, either quantitatively or qualitatively, but the examination itself, and particularly electrical irritation of the skin, produced intense contractions in the various muscles.

GEO. W. JACOBY.

Case of Brachial Monoplegia Due to Lesion of the Internal Capsule. By A. H. BENNETT. *Brain*, xxix., p. 78.

An old gentleman had a stroke of apoplexy attended by paresis of the face, and tongue on the left side, and followed the next day by total paralysis of the left arm, difficulty of deglutition and of speech, and involuntary evacuation. In the course of a month all the symptoms except the paralysis of the left arm had passed off; the paralysis remained until his death two months af-

ter his first attack. The autopsy showed a small ($\frac{5}{8}$ x $\frac{3}{8}$ x $\frac{1}{9}$ inch) area of softening at the junction of the internal capsule and centrum semiovale, involving the upper margin of the lenticular nucleus at a point just beneath the middle of the posterior central convolution. It had divided a few of the strands of fibres of the motor tract as they entered the internal capsule. The fact that such a lesion should cause permanent paralysis of the left arm, only leaving the left face and leg unimpaired, shows that the fibres connecting motor impulses from the cerebral cortex run in separate ribbon-shaped bundles through the internal capsule. The motor centres for face, arm, leg, and trunk lie above one another, beginning from below on the cortex. The four bundles of conducting fibres descending from those centres in a downward and obliquely inward course may be compared when viewed from in front to four rays of a half-opened fan. These converging in this attitude toward the upper part of the internal capsule enter its knee, and as they do so change their direction. Preserving their relative stations they become twisted from their former oblique position to one completely antero-posterior ; so that what was external and in front becomes anterior, what was internal and behind becomes posterior. The half-opened fan is now shut, and its rays which before were seen obliquely in front are now seen from the side. In the last position all the series of fibres pass through the internal capsule in well-defined ribbon-shaped bundles, those for the face being the most anterior, behind which in successive order we find those for the arm, leg, and trunk. In the case reported the second ray of the fan was injured. The situation of this band in a horizontal section of the capsule is a little behind the knee, and opposite the apex of the lenticular nucleus.

The case confirms the statements made by Flechsig in 1881, regarding the course of the motor tract and the situation of its respective bundles. M. A. STARR.

A Case in which an Old Amputation of the Left Upper Arm was Associated with an Atrophied Right-Ascending Parietal Convolution. By JOSEPH WIGLESWORTH, M.D. *Journal of Mental Sciences*, vol. xxxii., p. 50.

This is the case of a female epileptic who died at the age of fifty-six, and who in the fourth year of her age met with an accident which necessitated amputation of the left upper extremity. " For a period, therefore, of fifty-two years this patient was deficient in the movements and impressions connected with the left arm and hand, and it was consequently to be expected that the cerebral centre in correspondence with this region would exhibit some amount of defective development." The post-mortem examination showed that the *right* ascending parietal convolution in its lower three-fourths was half the size of the corresponding convolution on the opposite side. The atrophied region would correspond to Ferrier's centre for movements of hand and wrist.

[According to the more prevalent view the chief atrophy should in this case have been in the ascending *frontal* convolution, in the "arm centre," but the plate published with the article exhibits a much larger ascending frontal convolution on the right than on the left side. The type of convolutions (in this case) is very irregular, and we should be very careful not to use this single case as proof of the existence of an "arm centre" or of a "hand and wrist centre" within the former.]

Cases of Cerebellar Disease. By GEORGE WILKINS, M.D., Professor of Med. Jurisprudence, etc., McGill University. *Canada Medical and Surgical Journal*, vol. xiv., p. 513, 1886.

Prof. Wilkins reports three cases of cerebellar disease. In two of these cases, the diagnosis was supported by the post-mortem examination; in the third no post-mortem was allowed, but from the history of the case we believe the author justified in making a diagnosis of cerebellar apoplexy. In all three cases extreme suddenness of death, due to effusion of blood or pus into fourth ventricle, was a marked symptom. The salient points of the two cases with autopsy are as follows:

CASE 1. A young lady of nineteen complained of intense headache and had a sudden fainting fit in the early morning; from this she did not recover, and before the physician arrived she died. The autopsy, made a fortnight after interment, showed that the under and lateral surfaces of the right temporal lobe were covered with an extensive but thin clot; so also were pons and medulla. Right lobe of cerebellum appeared considerably larger than left. On cutting into it a large clot, the size of a plum, was discovered close to the central lobe. On examining more closely a pinhole aperture was seen on the superior surface of this lobe of the cerebellum, and was here continuous with the clot covering the temporal lobe. A blood-vessel had burst in this lobe of the cerebellum, the blood escaping into the subarachnoid space covering it, and along this space forward on to the cerebrum and downward into the fourth ventricle.

CASE 2 ("3"). A boy aged seventeen fell from a cart, striking his shoulders and then his head; from that time onward headache, pains all over body, dizziness and vomiting which persisted; endeavored to walk, but was unsteady and weak. On the fifteenth day after fall became suddenly cyanotic and almost asphyxiated. Pulse 120, regular; pupils contracted; unable to open mouth; artificial respiration was kept up for a while. Patient died within two or three minutes after its cessation. Post-mortem:—membranes of brain were found normal, except a small portion overlying the cerebellum between the flocculus and medulla oblongata. On carefully removing the membranes and raising the medulla several drops of thick, creamy pus were observed between the cerebellum and the floor of fourth ventricle. An abscess cavity of the size of a large filbert, filled with pus, was

found in the right lobe of cerebellum ; in this case the pus had found its way into the fourth ventricle. These cases are interesting, but call for no further comment. B. S.

MENTAL PATHOLOGY.

Schools in Hospitals for the Insane. Dr. J. B. ANDREWS (Buffalo, New York, Insane Hospital, 1884, Report) says concerning the school recently established in his hospital :

"The school is held in one of the dining-rooms during the morning hours. The teacher is an attendant upon the ward who has had experience in one of the schools of Buffalo. The order and good conduct observed are the same as exist in outside schools." We do not speak of this effort as any thing new or to compare it to what is being done in some other institutions, but simply to note it as among the means of occupation found useful in certain cases. It assimilates the life of these younger people to that of others of the same age or to their former life outside the asylum, and they certainly derive benefit from this mental effort. This was recognized by Dr. Brigham, who states (Utica Asylum Annual Report, 1844) : " The school is beneficial especially to the convalescent, those that are melancholy, and those who appear to be losing their mental powers and sinking into a demented condition. Those who have recovered but continue with us for fear of a relapse, and to test the permanency of their recovery, derive both pleasure and profit from attending. Those that are melancholy and depressed are beguiled from their sorrows, and for a while made to forget them, and thus the way is often prepared for their restoration. Those who appear to be losing in mental power are much benefited by the daily and regular exercise of their minds ; their memories improve, and they become more active and cheerful. The want of proper mental occupation according to our observation is one of the most pressing wants of lunatic asylums." Since this was written schools have been included among the means of moral treatment in several asylums, but it remained for Dr. Lalor, to elevate it to such prominence that the Richmond District Lunatic Asylum in Dublin, Ireland, is noted for its regular systematic instruction given to a large number of patients. In his hands it has proved an invaluable agent of treatment. Dr. Dwyer, of the Mullingar District Asylum, Ireland, has met with such success that he writes : "While I am on the subject of schools I cannot refrain from expressing my astonishment that every well-organized asylum has not its school. I have seen wonderful results from schools." Pinel says : " Thirty years' experience has taught me that a striking analogy subsists between the act of educating and teaching the young and that of managing the insane, as the same principles are applicable to both." This analogy is constantly noted by those who have charge of the insane, but all do not recognize it as so far-reaching as to warrant the effort to teach all of the insane. The different

classes of patients treated in different institutions will always and very properly lead to diversity of practice as to the numbers taught in schools. Despite the beneficial results of these schools they were, as has been pointed out in this JOURNAL, abandoned by Dr. Gray because of his coarse materialistic views of insanity.

Somatic Disease and Insanity. Dr. T. S. CLOUSTON (Morningside Asylum Report, 1884,) has had one case of a very unfavorable character, which made a most complete and unexpected recovery. She came from another asylum with a bad history. To every question put to her she would only answer: "Tak awa' my life." She would not occupy herself in any work. She was constantly attempting to end her existence. She slept little, and looked the very picture of utter despair. He often pointed her out as a person who did not need to go to Hades to be made as miserable as it was possible for a human being to be, and live. She needed constant watching by night and day. Her misery was so great, and had continued so long, that her attitude and features were getting fixedly those of profound mental depression. I must say I had put her down as incurable, and thought that she was one of the cases that some day would evade the attendants' watchfulness, and succeed in her great desire. But she took a disease, with intense feverishness, and was for weeks between life and death. As she recovered from the bodily disease, she got far stronger and more healthy than she had been since her mental trouble began, and the depression with the suicidal feelings passed off, while her attitude became erect and hopeful, and her face calm. She began to employ herself, and made a perfect recovery in body and mind at last, and keeps well in America, where she went, up to this time. The cure of this case prevents one losing hope about many others. There are such examples in insanity, as in other diseases, of a new complaint arising, especially if it is sharp and febrile, and producing a constitutional change which cures the mental disease. Indeed he believes it to be one of the hopes of cure of many cases in the future, that there will be discovered some means of causing a manageable fever, and so producing such a change in the general nutrition as will tend towards recovery from the mental disease. He is never better pleased than to see a crop of boils, for instance, coming out in a case that is hanging fire as regards recovery.

Idiot Brains. Dr. A. W. WILMARTH (*Medical and Surgical Reporter*, May 9, 1885) states that it is in the arrangement of the convolutions that some of the most interesting features are met. In congenital imbeciles, and particularly in idiots, striking differences are found. One type of brain, in this class of children, is very simple in its outward configuration. The convolutions are usually coarse, but little convoluted, and comparatively free from secondary folds. The fissures tend to assume a confluent type, as in lower grades of intellect, and this tendency increases.

Another variety, found so far among the lowest grades of idiocy, might well be termed an "atypic" variety. The brain previously described as without a corpus callosum, is a marked example of this type. In the frontal lobe of the right hemisphere the first frontal convolution is quite regular. Below this, from the centre of the lobe, seven radiating fissures pass in different directions, cutting the tube into a number of radiating convolutions, entirely different from its usual appearance. The short fissure of Sylvius, about three inches in length, passes upward, turns sharply at nearly a right angle, and passes almost directly backward. Two parallel gyri curve around its posterior extremity. The arrangement of the convolutions of the temporal and parietal lobes are so exceedingly irregular and complex, that it is impossible to classify them. In the occipital lobe, on the contrary, the gyri are complete in number and regular in their arrangement. In the left hemisphere the arrangement of the frontal convolutions is more regular, but the temporal and parietal lobes present the same complicated area of surface folding, bearing but little resemblance to the normal brain. The tendency of the convolutions to arrange themselves in parallel curves around the posterior extremity of the fissure of Sylvius, is well shown in the brain of a boy of exceedingly low intellect. The frontal lobes in this brain are proportionately large, the convolutions straight, especially the third frontal, the fissure shallow. In the left temporal lobe they are nearly obliterated from pressure of fluid in the ventricles. The ascending frontal convolution on each side appears to be wanting. On the left side a large bridging convolution crosses the middle of the fissure of Rolando. Confluence of fissure is a decided feature of idiot brains. Even where confluence is not complete, the tendency of the principal fissures to cut through separating convolutions is very evident; and were the cases where confluence is *nearly* complete included, the number would be considerably augmented. In these cases confluence is complete, and the examination comprises fifteen brains from children of all grades of imbecility. The fissure of Sylvius passes into the fissure of Rolando, in one case on both sides, in another on one side only. In two other cases they are connected by deep secondary fissures. The inter-parietalis has its origin in the fissure of Sylvius, in four cases on both sides, in five cases on one side only.

The calcarine fissure passes completely across the gyrus fornicatus; on both sides in two cases, on one side in four cases. In one case the first occipital convolution sank nearly beneath the surface, the next occipital gyrus projecting over it, forming a partial operculum. There also seems to be a strong tendency to form annectant gyri in the upper part of the parieto-occipital fissure. In no less than six hemispheres of the fifteen brains were these supplementary gyri found more or less complete. In one case on both sides, in five cases on one side, the parieto-occipital fissure cut through the first occipital convolution into the interparietal fissure. A tendency of the transverse occipital fissure to

approach the parieto-occipital fissure is very apparent, though in no case do they coincide. The folds of the cerebral cortex, from a lack of the stimulus of healthy growth, sometimes revert to forms resembling those found in other groups of the kingdom.

Classification of Insanity. Dr. H. C. Wood (*Medical and Surgical Reporter*, Oct. 31, 1885) makes the following classification of insanity :

A. Insanities not dependent upon a previous neurotic condition. B. Insanities, the evidence of a continuous neurotic vice. B. Insanities caused by organic disease, toxæmia, or injury. A. (*a*) Not dependent upon evolution of life. (*b*) Dependent upon evolution of life. 1 *a*. Profound or emotional disturbance. 2 *a*. No emotional disturbance. Mania, melancholia, katatonia—terminating in dementia, and forming the great mass of cases of insanity. The word insanity represents all cases where there is no known organic lesion, and excludes paretic dementia, etc., where there are known organic lesions. *Katatonia* is a very peculiar and rare form of insanity, of which Dr. Wood has never seen a case, and there have been only two or three reported in Philadelphia in which the emotional nature is neither exalted nor depressed. There exists a peculiar pathetic condition. It will be obvious that this classification is modified from that of Spitzka, but Dr. Wood does not clearly recognize the essential basic principle of that classification.

Alcoholism. Dr. Jas. C. Wilson (*Polyclinic*, December 15, 1885) makes the following division of alcoholism :

I. Acute alcoholism.
  (A) Ordinary typical form.
  (B) Irregular forms.
    1. Maniacal.
    2. Convulsive.
    3. In persons of unsound mind.
  (C) Acute poisoning by alcohol (lethal dose).
II. Chronic alcoholism.
  (A) Visceral derangements.
    1. Local disorders.
      a. Of the digestive system.
      b. Of the liver.
      c. Of the respiratory organs.
      d. Of the circulatory system.
      e. Of the genito-urinary apparatus.
    2. Disorders of special structures.
      a. Of the locomotor apparatus.
      b. Of the skin.
  (B) Derangements of the nervous system. Cerebro - spinal disorders.
    1. Cerebral disorders. Complications.
      a. Lepto-meningitis.
      b. Pachymeningitis.
      c. Epilepsy.
      d. Cerebral congestion.
      e. Cerebral softening.
      f. Sclerosis.
    2. Spinal disorders.
    3. Disorders of the special senses.
  (C) Psychical derangements.
    1. Alcoholic delirium in general.
    2. Delirium tremens.
    3. Alcoholic psychoses.
III. Hereditary alcoholism.
IV. Dipsomania.

True dipsomania is not a manifestation of alcoholism, but a periodical insanity. The relations of alcohol to it are symptomatological, not ætiological. J. G. Kiernan.

Drunkenness in Relation to Criminal Responsibility. By GEO. H. SAVAGE, F.R.C.P. *Jour. of Ment. Sciences*, vol. ci., p. 23.

If we read Dr. Savage's remarks correctly, he would formulate no definite rule with regard to the punishment of drunkards for acts of violence perpetrated during the intoxicated state. He would have every case tried on its own merits. He is not willing to allow that while acute drunkenness shall be no sufficient excuse for acts committed, no person shall be held responsible for an act committed during an attack of delirium tremens. "Such a patient (one who has had delirium tremens) knows from past experience what danger he runs, and, though his will-power may be weakened, yet it seems to me that in some cases persons who have committed crimes while suffering from delirium tremens might fairly be punished." Dr. Savage gives the history of three interesting criminal cases, in each of which the act for which the prisoner was indicted was committed under the influence of drink. The verdicts in these three cases differed widely enough. We make a few further quotations to show how legal opinion may differ on this subject. Justice D. decided that a crime committed during drunkenness was as much a crime as if it were committed during sobriety, and that the jury had nothing to do with the fact that the man was drunk. The prisoner was supposed to know the effect of drink, and if he took away his senses by means of drink, it was no excuse at all. Justice M. says "that a state of disease brought about by a person's own act, *e. g.*, delirium tremens, caused by excessive drinking, was no excuse for committing a crime, unless the disease so produced was permanent." This another Justice D. criticises by saying "that the question was whether there was insanity or not; that it was immaterial whether it was caused by the person himself or by the vices of his ancestors; . . . that it was immaterial whether the insanity was permanent or temporary"; he would note, furthermore, "that if a man were in such a state of intoxication that he did not know the nature of his act or that his act was wrongful, his act would be excusable."

The Connection between Insanity and Crime. By WALTER CHANNING, M.D. *Reprint.*

This is termed a report on the Bibliography of Insanity read at the meeting (1885) of the Association of Medical Superintendents of American Institutions for the Insane. Instead of being a report on Bibliography, it is a carefully prepared digest (with some few critical remarks) on the chapter in Sir James Stephens' History of the Criminal Law of England, which treats of the "Relation of Madness to Crime." The article is well worth reading by those who are anxious to inform themselves on Sir James Stephens' views on this subject and who have not the time to read the original.

B. S.

Reviews and Bibliographical Notes.

The Insane in the United States and Canada. By D. H. Tuke, M.D. London, Eng. H. K. Lewis, 1886.

The present volume being sketchy and desultory in style is exempt from very rigid criticism. From the author nothing but a well-written work was to be expected. Dr. Tuke opens very fittingly with a critical, just account of the career and labors of America's pioneer and greatest alienist, Rush, who, in many respects, was far in advance not only of his own period, but of the generation of alienists just now passing away.

Dr. Tuke's labors in Canada had decidedly good results. He unearthed abuses in the Longue-Point Insane Hospital, managed, under a contract, by the Sisters of Charity, who ran it in total defiance of the directions of the government inspector, Dr. Howard. That gentleman was unable even to secure the discharge of recovered patients. Money-patients were kept in restraint and dark seclusion to an extent equalled only in the United States county institutions. This is the second investigation of the kind, under the same control, in Canada, which has been shown to be badly managed.

In his sketch of the progress of provision for the insane in the United States Dr. Tuke fails to discern what a tremendous obstacle to progress the Kirkbride-Gray Clique of the American Insane-Hospital Superintendents' Association was. This clique virulently criticized Dr. Gray, one of its members, for having published, in a spirit of fairness, which he seems to have since lost, a paper of Dr. Galt, containing in essence the doctrines of insane-hospital architecture now becoming dominant ; which shows how destitute of a scientific spirit, even in 1855, many insane-hospital superintendents were. Dr. Gray so profited by this lesson, that in 1878 he refused to publish in the *Journal of Insanity* a protest against extravagantly built insane-hospitals, by Dr. Isaac Ray. The work of the Insane-Hospital Superintendents' Association is the subject of too much eulogy. Dr. Tuke entirely ignores all defects of that body, even those which he criticised in the *Journal of Mental Science.*

He does not inform his readers that so late as 1875 paddling (more inhuman than whipping) was used in the Auburn, New York

State, Criminal Insane Hospital by the superintendent as a punishment, and that (New York Neurological Society, Answer, 1880) "at the very time when these abuses were committed at this institution, the American Association of Superintendents of Insane Asylums met at Auburn. They spent a half-day in inspecting its wards, and then passed resolutions which contained the following language: 'That their visit had been peculiarly interesting, as giving most obvious evidences of good management.'" Neither does he tell his readers that in the year 1882, nearly a century after the beginning of the labors of Pinel and Chiarruggi, chains were used in a Kentucky State insane hospital, and still are used in many county insane institutions in Illinois and elsewhere. He passes over in silence these and other facts with which he has been made acquainted through his connection with the *Journal of Mental Science*.

It is clear, therefore, that Dr. Tuke is not as free from bias as might have been expected. This is rendered still more obvious by a comparison of the present work with Bucknill's "Notes on Asylums in America," published in 1876. Bucknill was a good deal of a Boswell; like that prince of biographers he was, so far as his mental limitations allowed, true to history. In 1876, Bucknill finds the ruling majority of the Insane-Hospital Superintendents' Association arrogant, defiant of medical public opinion, and resenting as an insult the idea of governmental supervision of their labors. In 1884, Dr. Tuke finds a feeling in favor of governmental supervision so strong that the contrast requires explanation. Allowing for deceit, like that practised by Dr. J. P. Gray on Dr. Bucknill ("Notes on Asylums in America") in regard to restraint, on the one hand, and the increased and beneficial influence of Drs. Ray, Nichols, Godding, Gundry, Wallace, Hughes, Smith, Earle, Bryce, Macfarland, and others within the Association itself on the other, the change from 1876 to 1884 is not accounted for.

In 1878 the New York Neurological Society began the first organized medical movement for reform in psychiatry, and especially in insane-hospital management. To its labors are clearly due the change in the tone of the Association, the reforms in the study of psychiatry, and the improvements in insane-hospital management, now evident in American insane hospitals. How great the change so produced is, can be realized only by a comparison of the dogmatic views expressed to Dr. Bucknill in regard to restraint with the present status of American insane hospitals in this respect. In 1876 Dr. Bucknill was told that a vote of the Association had settled the question of restraint forever in the United States; in 1884 Dr. Carlos F. Macdonald was managing the Auburn Criminal Insane Hospital (the institution in which the paddle was regarded as necessary in 1875) without restraint. The movement of the New York Neurological Society, which rendered possible all reforms since Dr. Bucknill's visit, by diminishing the power medical politicians were able to wield

through their control of the Association, is passed over in complete silence by Dr. Tuke, although historical accuracy would require some notice of it in a volume like this. Dr. Tuke cannot plead ignorance, for he commended the paper which initiated this movement, in the following language (*Journal of Mental Science*, July, 1878): "Under the above title, Dr. Spitzka publishes an address to the New York Neurological Society, in the April, 1878, number of the JOURNAL OF MENTAL AND NERVOUS DISEASE, in which he criticises most severely, many people would say intemperately, the work of American asylum physicians, and the policy of the American Association of Asylum Superintendents. There is much truth, however, in what Dr. Spitzka says, and we think our American brethren will do well to take heed to this and many other indications that a more liberal and open mode of conducting their asylums and managing their Association is required. . . . For example, we have never sympathized with the exclusive and unscientific spirit which shuts out assistant medical officers of asylums from the privilege of membership. We hold it to be a mistake in policy, a misfortune in practice, and unjustifiable on any ground." It is difficult to understand why Dr. Tuke should have ignored that movement altogether; why he should not have commented on the contrasts between the conditions found by Dr. Bucknill as compared with those found by himself. The work, therefore, is lacking in information as to the history of provision for the insane in the United States.

Like Dr. Bucknill, Dr. Tuke has been led into commending the sham pathological work of Utica. The absurd results of the pathological labors of the Utica school are best judged by the following extracts from a paper by Dr. John P. Gray (*Amer. Jour. of Insanity*, Jan., 1878), based on the laboratory's microphotographs. He describes "granular bodies" (p. 10), "grumous granular matter" (p. 17), "encysted morbid products" (p. 21), "colloid bodies" (p. 23), "globules of a fatty nature" (p. 27), as being found in every form of insanity. These were shown by Spitzka (JOURNAL OF NERVOUS AND MENTAL DISEASE, 1878) to be artefacts due to the action of alcohol on nerve tissue. Dr. Savage has since corroborated Dr. Spitzka. Yet upon them was based the following conclusion: "A fact which seems of the utmost importance is the similarity of histological changes attending the different forms of insanity, as represented in the microphotographs, and indeed in all cases which have fallen under observation. If such regularity is displayed in future investigations, as I am strongly led to believe will be the case, this fact will practically confirm the principle, that in insanity we have to contend with *only one* DIATHESIS, manifesting itself under different phases in its progress and results."

Dr. Tuke should have remembered that not only Dr. Spitzka, but his own colleague, Dr. Savage, had demonstrated the uselessness of these Utica laboratory results. Nothing need be said had these been only useless, but they have been used to justify a

judicial murder. The findings in the case of Grappotte, executed for murder in obedience to the testimony of one of the Utica school, thus contrast with changes in the insane by Dr. Gray:

"THE INSANE.—Chain of granule cells with the residua of a disintegrated capillary. Cluster of granule cells, with residua of disintegrated capillaries.

"The gray cortical substance of the vascular system of the brain[1] [of Grappotte] revealed a fully normal supply of blood in all parts. Scattered through the tissues, however, especially at the base of the brain, there were many residua of degenerated and obliterated arterioles and capillaries. They represented small stems or shrubs with two, three, or more branches, lined by irregularly thickened hyaline walls of a fibrillous structure."

The present work of Dr. Tuke, so far as it relates to the insane in the United States, is decidedly faulty; it is more than just to the clique he criticised in 1878, and does not do justice to the labors of those who have nearly destroyed the autocratic power of that clique. The book is well issued.

J. G. K.

[1] So in original.

Society Reports.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated meeting, January 26, 1886.

(*Continued from page* 127.)

Dr. William Osler then read a paper on "The Structure of Certain Gliomata."

He desired to call attention to the histological character of certain brain tumors which present peculiarities of structure separating them from the ordinary small-celled gliomata.

The specimens which he showed were from three cases, the features, clinical and anatomical, of which may be thus summarized :

Case 1.—Girl aged sixteen, blind from third year ; intelligent ; head not large. Occasional convulsions and spasms of muscle of neck. Death sudden. Tumor occupied the surface of the left thalamus, and extended into the third ventricle. There was extensive dilatation of the lateral ventricles.

Case 2.—Girl aged fifteen. Jacksonian epilepsy for over fourteen years. Small, firm tumor occupied the upper part of ascending convolution.

Case 3.—Man aged forty. Head pain, mental disturbance, drowsiness, the chief symptoms. Tumor the size of a lemon occupied left anterior lobe of brain.

The physical characters of these three tumors differed considerably. In Case 1, the mass on the thalamus was firm, but the portion projecting into the third ventricle was soft, grayish in color, and looked like an actively growing neoplasm. In Case 2, the small tumor at the upper part of ascending frontal convolution resembled a patch of sclerosis, while in Case 3 the tumor had a large central area of fibro-caseous change, with a peripheral zone of actively growing grayish-red tissue.

Histologically these tumors are similar in the dense feltwork of fibres which make up the chief mass of each, the fibres varying somewhat in thickness and in closeness of arrangement. Careful observation of teased specimens shows that the fibres are, for the most part, in connection with cells, and so far the growths conform to the type of glioma. True, we do not find here the typical arrangement of small cells with delicate protoplasm and numerous fine ramifying processes which gives to many gliomas an appearance not unlike that of a small-celled sarcoma. There are gliomata, however, with larger and more irregular cells and with coarser fibres than the description in text-books would lead us to suppose, and it was more particularly certain characters of the cells in tumors of this kind that he wished to call attention to. A study of teased, fresh specimens can alone give a clear idea of the shape, size, and relations of the cell elements.

These tumors conform to the variety described as neuro-glioma by Klebs, who holds that the large ganglion-like cells found as such important constituents of these growths are derived directly from the nerve cells of the gray matter, and that in the development of this variety all the elements of the nerve tissue participate. Certainly the resemblance between many of the large cells and nerve elements is very striking, but I have not been able to satisfy myself of their relation to the preëxisting tissue parts. This is, of course, extremely difficult, but in a careful study of sections taken from the advancing regions of the growths, I have not met with appearances which would lead me to suppose that the nerve cells were in process of proliferation. Klebs states that he has demonstrated by means of osmic acid and gold chloride the nature of the cells and their processes, but this has not been confirmed, and I could not determine that the cells or fibres described above behaved in a characteristic manner with these reagents. That they are probably connective-tissue elements seems probable from an examination of a large number of the cells in teased preparations. Gradations and intermediate forms can be seen between cells closely resembling unipolar or bipolar nerve ganglia and the typical spider cells with innumerable processes. Gliomata of this form are not uncommon. Klebs described fourteen or fifteen, and of five cerebral tumors of the glioma type which I have met with, only two were of the small-celled variety.

VOL. XIII. APRIL AND MAY, 1886. NOS. 4 AND 5.

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

A CONTRIBUTION TO THE LOCALIZATION OF FOCAL LESIONS IN THE PONS-OBLONGATA TRANSITION.

BY E. C. SPITZKA.

THE following case not only illustrates the certainty with which limited lesions of the cerebral isthmus may be occasionally located, but also constitutes a contribution to the list of cases in which disturbances of associated eye-movements were observed during life, and brought into relation with special lesions after death:

Mary A., aged twenty-three, married, a factory operative, was kindly referred to me by Seneca Powell for examination on May 15, 1884, with the following history:

During the month of June, 1883, she had regularly every morning an attack of nausea or vomiting, or both, before going to her work. In July the vomiting ceased to be so regular, and occurred only in conjunction with the seizures which she experienced at this time. On three occasions, down to the month of September, she had an attack of mingled vertigo and a feeling, to use her expression, "as if going out of her mind"; this of such severity that she would keep her bed for from two to three days. It seemed, as she expressed it, "as though every thing in the room gyrated around her." In the third spell of this kind she

first noticed that her vision was blurred. She recovered from this as from the other spells entirely, returning to her occupation (that of a carpet-weaver) immediately after. Subsequently a numbness of the left side was noticed, but she continued at her work. It frequently became the subject of mirth among her fellow-operatives, that her mouth would suddenly become crooked—that is, drawn to the right —while at work. In November of the same year she had an attack of double vision, but is unable to say how long it lasted, further than that it was at most a few days. Recently she has not manifested distinct double vision, but complains of a dancing movement of objects. This subjective perverted perception is increased when she walks, and particularly when she attempts to look to the left, making her feel, as it were, "drunk." The diplopia previously referred to was of such a character, that both images were on the same horizon; the dancing movement of objects, on the other hand, seemed to be chiefly in the vertical plane, though the patient is uncertain on this head.

About two months ago, March, 1884, her jaws became "tight"; it was impossible for her to separate the dental rows further than to admit a teaspoon between them. She felt this stiffness subjectively on the right side. Mastication was in consequence difficult, and continued impaired for some time after the spasmodic condition disappeared. For some months her voice has been noticed to have become nasal in character. There is commonly a subjective feeling of drowsiness, which is aggravated whenever her stomach is full.

The first permanent and continuous symptom was difficulty in swallowing. It began about the time of the seizure in September. At its onset it was extreme, and on more than one occasion threatened a fatal result, the saliva accumulating in her mouth during the night causing her to choke and wake up. Sometimes she would experience as many as from forty to sixty distressing spells of choking and strangling in one day. The attacks have become fewer but of greater severity. At first fluid food was more difficult for her to manage, but she can drink now under certain precautions, while dry

food, which gave her no distress at the start, does so now. She occasionally had a sensation as if something in the head pulled it back, at other times her heart gives a sudden violent thump, and the patient asserts that it has "held over"—that is, stopped—for as many as two or three beats. These evidences of disturbed cardiac action were most marked in March, 1884. Then and now she has attacks of anxiety associated with shortness of breath, which are aggravated or provoked when she drinks or eats.

The hemi-numbness has continued increasing during the last six months, and a sensation of pins and needles is often noticed in the left extremities. The left leg sometimes drags, the toe has never turned under, nor have her ankle or knee ever given way. For some time, whenever she touched her left leg or arm, a "funny" sensation resulted, this later was compared to a cold sensation, and remained limited to the spot touched. She frequently has a spontaneous cold sensation on the left side of the body, in warm weather, and when the right side enjoys the normal appreciation of the outside temperature. The numb sensation in the left arm becomes aggravated into a pin-and-needle and going-to-sleep feeling, if it is allowed to rest long in one position. To some extent, she claims to be able to overcome this, using her expression, "by rubbing life into it."

She had a buzzing noise in her right ear two weeks ago, and has it now and then at intervals in varying intensity. It has not been recollected whether any such sensation accompanied her vertiginous seizures.

The functions of the stomach have been more or less disordered; at present she is able to retain her food, but is compelled to remain perfectly still, or else she would vomit it. She finds difficulty in eructation of the stomach gases, which form in considerable quantity.

The patient is a well-nourished, rather good-looking woman of robust build. She has been married three years and seven months. Shortly after marriage, when pregnant about three months, four sores appeared on her labia. These were a little larger than the conventional cent; they opened under local treatment, and, according to the account, dis-

charged matter and blood. The child was born in the seventh month of gestation and died four days after birth. Since its delivery the mother has suffered from metrorrhagia, and during the year past from what was pronounced uterine catarrh. Her hair fell out to a remarkable extent after the child's birth. She speaks of a white patch in her throat, which was treated shortly after marriage, and to which she called Doctor Powell's attention. I was unable to find any certain traces of past throat trouble. Aside from pityriasis versicolor, a cicatrix over the left breast from mammary abscess, and gastro-intestinal disturbances, the patient presents no other anomaly in her present condition or her history than those mentioned, aside from the symptoms of axial cerebral disease now to be detailed.

Locomotion.—There is no anomaly observable in walking, in turning, in standing, or walking with the eyes closed, in standing on either foot with the eyes open. She is nearly as well able to stand on the left leg, with the eyes closed, as on the right. It must be remembered that many normal persons with pronounced dextral preference show the same difference.

Muscular power.—Repeated crude trials show no perceptible difference in the muscular powers of the lower extremities. A stiff dynamometer shows for the right grasp sixty, for the left grasp fifty. The individual motions show no indications of enfeeblement. The patient is dextral.

Muscular sense.—The patient is not uncertain about the position of the segments of her arm and leg; as to degree of flexion or extension, she is not always able to place her left upper and lower extremities in position symmetrical with a passively given position of the corresponding right limb, when her eyes are closed. She is able to touch a given part of her body correctly and quickly with the left index finger.

Cutaneous space-sense.—Repeated trials fail to show any difference in the ability to appreciate the points of the æsthesiometer. Generally the patient's space sensibility is rather under the figures assigned by physiologists. To this I can attach no importance, as those figures are far too

high—at least for certain classes of the human family. The patient localizes well, but is unable to distinguish the nature of objects and various degrees of roughness, as well with the tips of the left fingers as with the right, tested with files and rasps.

Temperature-sense.—Repeated tests failed to reveal any difference between the two sides. She can tell whether the object touching her be warm or cold, but contact of both warm and cold objects produces an ice-cold after-sensation.

Cutaneous pressure-sense.—The patient does not appreciate five-fold weight differences with the left side that she is able to appreciate with the right. She can feel the addition of a penny to a silver dollar in the right palm, and the addition of a dollar to a dollar with the left, but not of a quarter nor always of a half dollar. The same difference is noted with the foot; the microtome cylinders being used, she is able on the dorsum of the left foot to detect the addition of the intermediate size, but not of the smallest and lightest, which she does notice with the right. The patient has herself noticed that she does not feel a grip of the hand nor the pressure of a shoe as well on the left as on the right side.

Pain sense is normal.

General cutaneous sensations.—Touching any part of the left leg or arm, which six months ago produced what she calls a "funny sensation," now produces a cold feeling, limited to the part touched—as previously stated—even if the subject be warm. A stroke of the hand down either limb, but most marked with the leg, produces a cold streak, as if a stream of ice-cold water were running down the limb. Her left side feels cold at times, when the other side feels properly warm in warm weather.

Reflexes.—The tendon and abdominal reflexes are symmetrical and normal; the tickling reflexes are all exaggerated, notably the solar.

Facial nerve.—There is total paralysis of every external branch of the right nerve; the right side of the face is blank, and no voluntary or reflex movement is possible; there is consequent lagophthalmus; the uvula, however,

deviates to the right; there is excessive lachrymation of the right eye, which it is impossible to close.

There was a slight increase in both faradic and galvanic excitability, but no change of the formula; the muscular contractions appeared retarded. This was found to apply to all the external branches of the nerve. There occurred no further opportunity of repeating and extending these tests, owing to the failure of the patient to report to me in person. The patient and her sister state that the left side was more drawn (antagonistic contracture) a few weeks ago than it is now.

Hypoglossal nerve.—The tongue, as a whole, deviates to the right, while its axis is curvilinear, the point tending to the left; there are no twitchings nor tremors.

Gustatory function.—The patient can detect the bitterness of quinine, the sweetness of a syrup, and the flavor of assa-fœtida on both sides, but with greater slowness and hesitation on the right side.

Vocal cords.—No paralysis of the cords; both movements of phonation and respiration normal.

Fifth pair.—The patient feels subjective numbness on the right side of the face, and limits it accurately to the right of the median line; it does not extend to the hairy scalp. There is no objective difference of the cutaneous space-temperature- or pain-sense on the face or tongue.

The palate is relaxed, and the patient, in swallowing water, evidently resorts to a manœuvre which one of my patients with glosso-labio-laryngeal paralysis calls "resting on the swallow." The stomach is distended with gas, and the patient, in trying to eructate, finds it impossible to raise the wind, as she calls it, above the level of the sternum, borborygmi can be heard to rise to that level. The heart-sounds are normal, the auscultation being continued for ten minutes; an unusually loud systolic sound was heard, accompanied by a visibly stronger apex impulse, and followed, after a pause equivalent to three quarters of a beat, by a number of rapid, indistinct pulsations which gradually passed into the normal. A second sensation, like that accompanying this episode, had been previously experienced

by the patient while in the office, the whole time being an hour and a half. The sphygmographic tracing was typical; tension moderate; no tracing was obtained of the phenomenon. I have lost it. The urine had been examined by Dr. Powell and found normal. I never obtained a specimen. No abnormality in micturition nor thirst.

The *optic nerve* is normal, the color sense perfect; there is no limitation of the visual field. There is no ascertainable diplopia while the patient looks directly forward or to the left.

Pupils equally and regularly four mm. under moderate illumination, react to light not very actively, contract under accommodation to pinhole dimensions, and remain in pinhole contraction after accommodation and convergence are suspended. They then return slowly to the previous condition, fully a half minute being thus occupied.

Binocular movements.—The patient is able to move both eyes together freely upward, downward, and to the left; the attempt to move either eye to the right of the middle of the palpebral fissure fails, both eyes being arrested as by an obstacle. At the time I had not read Hunnius' excellent monograph, but I recorded in my notes: "It is as if a wall were built up against the eyes at this point." Hunnius used the expression, "festgemauert" for his case three years previously. There is nystagmic oscillation with extreme movement to the left, and the dancing movement of objects previously referred to is then complained of. The patient does not localize the direction of this dancing movement.

Monocular movements.—The left eye separately examined shows no nystagmus, and can be moved in all directions; the right eye cannot be moved to the right beyond the middle of the palpebral fissure; at the extreme point it shows slight lateral oscillation; its other movements are unhindered.

When the patient is told to close the eyes, the right eye, which is left uncovered by the lid, is seen to roll upward in the normal way.

Lateral movement of the jaw is free in both directions;

the masseters both contract when the teeth are pressed together, less firmly it seems on the right side.

The diagnosis was made a focal affection: neoplasm, probably a syphiloma, in the level of the pons-oblongata transition, situated in the right reticular field of the tegmentum, involving either the facial nucleus or root—probably both—and the roots of the abducens nerves, as well as the nuclei of the pneumogastric. Mixed treatment, which Dr. Powell had previously given, ordered continued.

I heard from the patient by messenger on several occasions. She at times complained of severe occipital headache on the right side, but not to the same extent as some months previously. Doctor Powell informed me verbally that cauterization of the nuchal region relieved this. She developed a ravenous appetite, which nothing could satisfy, and this bulimia continued down to within eight days of her death, when it gave way to as pronounced an anorexia, as it happened, fortunately, for deglutition was scarcely possible.

On July 7th I saw her at her home, in company with the physicians whom I had invited to attend the autopsy, in the event of its being permitted. The patient had not slept the preceding night, sudden shocks of the lower part of her body startling her as she dozed. The eye-symptoms have remained stationary, except in so far as the nystagmus is concerned, which is only occasionally noticed, and with extreme innervation. The head when raised shows an oscillatory motion, and there is a tendency to sinistral rotation, but of a passive character. No conjugate deviation has ever been noted, nor is there any trace of it now; the right abducens muscle is more decidedly paretic in monocular as well as binocular action; the globe being turned towards the inner canthus, this position can be partly overcome under binocular innervation. The upward movement of the right eye in closing is retained.

The tongue, which is angry-looking and heavily coated, deviates as a whole to the left; its right half is much more voluminous in all dimensions than the left; it is not protruded readily, and shows fibrillary tremor. The cardiac impulses previously described are still noticed, and accom-

panied the shocks complained of last night; the dyspnœa is increased, though there is no pulmonary trouble. The right arm measures six and a half and the left six inches, in circumference. There is now distinct ataxia of movement, marked inability to assume symmetrical positions, to touch the nose with the tip of the finger while the eyes are closed, except after repeated error and explorations, while the cutaneous space- and pain-sense are normal. The patient has for the past few days micturated frequently, and large quantities of light-colored urine. No specimen could be obtained—and it was forgotten in the anxiety to secure the autopsy to order the preservation of any.

The patient being evidently approaching a moribund state, this condition precludes a lengthy examination. She can stand, but has not ventured to walk across the floor for a week, feeling uncertain as to her left lower extremity. She has noticed this uncertainty for some days before taking to her bed. She complains of a sense of heat on the right side of the face and head and a pulsating sensation in the right occipital region.

She menstruated last week, being two weeks over time. The blood was noted to be unusually dark. The pulse is feeble, variably irregular, and often intermittent.

The speech is indistinct; an undoubted anarthric element is added to the effects of palatine paralysis. In her position and situation the palate could not be distinctly examined.

Diagnosis previously made adhered to, with the modification: extension of neoplasm to hypoglossal nuclei and right interolivary layers.

The subsequent history was obtained after death.

July 8th she experienced excruciating pain in the right side of her head, sharpest in the occipital region, but extending over the entire half of the cranial region. She was beside herself with agony, crying out: "My brain! my brain!" Her sister was the only person who could understand her. Speech became so indistinct, however, that even she was unable to distinguish any thing beyond monosyllables for the twelve hours preceding death. The patient attempted to, but could not, enunciate. For one hour pre-

ceding death the pain ceased. The patient was thoroughly conscious to the last moment,—signing for water and other objects desired; was able to pass the glass to her mouth, even with the left hand, though with some uncertainty, but unable to say a word: shaking her head when asked to speak, and answering questions correctly by nods or shakes. She was also unable to swallow.

Ten minutes before death she had a chill-like convulsion of the entire body, without loss of consciousness. The night before her death the bodily surface, which had previously been cool, became hot, and remained so, till the last moment. She was noticed to choke and gurgle a great deal, to use the words of her attendants.

It had been repeatedly stated by the patient during the period of bulimia, that she did not taste the food on one side of her mouth, and eight days before death, when eating a piece of pie, she said: "Now that is funny. I can not taste this at all, any more than if it were paper." It was unfortunately neglected to test the sense of taste on the occasion of my last visit. The cold sensation had been felt in both lower limbs and the left arm, for several days before death.

The patient died a little after six o'clock on the 9th of July.

The autopsy was made, in the presence of Doctors Brill, Mollenhauer, F. A. McGuire, Barry, Harwood, and Adrian, at one o'clock the same day; only the cranial cavity was allowed to be opened, but the upper part of the spinal cord, down to the middle of the cenical enlargement, was removed as well.

The body appeared fairly well nourished; the difference between the arms noticed during life had not changed. There was a little rigor mortis; the pupils in median contraction. There is an irregular linear cicatrix on the under part of the left breast, and some spots of pityriasis versicolor are visible on the back and shoulders.

The posterior segment of the skull was removed, so as to show the basilar structures in their natural relations, and I had the gratification of anticipating a confirmation of the diagnosis made, before the occipital cap had been raised. The cervical vertebral arches having been removed, and the

membrana obturatoria dissected away, a distinct bulging of the right side of the anterior part of the oblongata, in the region of the clava and restiform body could be seen.

I would strongly recommend this method of removing the brain employed in all cases of suspected basilar or axial disease. By the ordinary method it would have been impossible to determine one point, of great pathological importance in this case, namely, the possible occurrence of pressure on other parts of the brain, or on the nerve roots. I was able to exclude this factor; the tumor was visible as soon as the bony shell was slightly raised, before it was even entirely removed, and nowhere crowded any part of the cerebellum or other neighboring parts. The choroid plexus, however, was greatly flattened; this appeared to be as much due to its widening out as to pressure, and the same explanation is to be offered for the thinning and dehiscence of the velum medullare, which was normal in texture.

The bony structures were healthy; the dura, which was intrinsically normal, appeared to be more firmly adherent than usual; the pia was healthy; and with the exceptions to be detailed the cerebral tissues showed nothing abnormaal.

The floor of the fourth ventricle in this district showed the following abnormalities:

The contour of the posterior angle was rounded, and the sides thrust asunder, so that the *alæ cinereæ* were crowded from their usual situation far ectad; they were unusually pronounced, the right indeed quite black, the left nearly as dark. The furrow which under normal circumstances diagonally subdivides these alæ was obliterated.

The inner two thirds of the posterior fovea, including the region of the *striæ medullares*, and extending over the eminentia faciales, in front, and encroaching on a narrow rim of the left side, was of a gelatinous gray color, marked here and there by a beautifully clean purplish shade. The only normal structures that could be identified in this mass were the stria. These ran in their usual distinctness and whiteness nearly to the middle line. There was considerable injection, the vessels appearing like blackish and purplish streaks in many places. The focus of the neo-

plasm had an appearance as if composed of an agglomerated mass of spheres, like boiled sago, an appearance which became more distinct during the first days of bichromate of potash hardening. Some epithelial granulations were scattered over the middle third of the ventricle and over the right ala alba medialis.

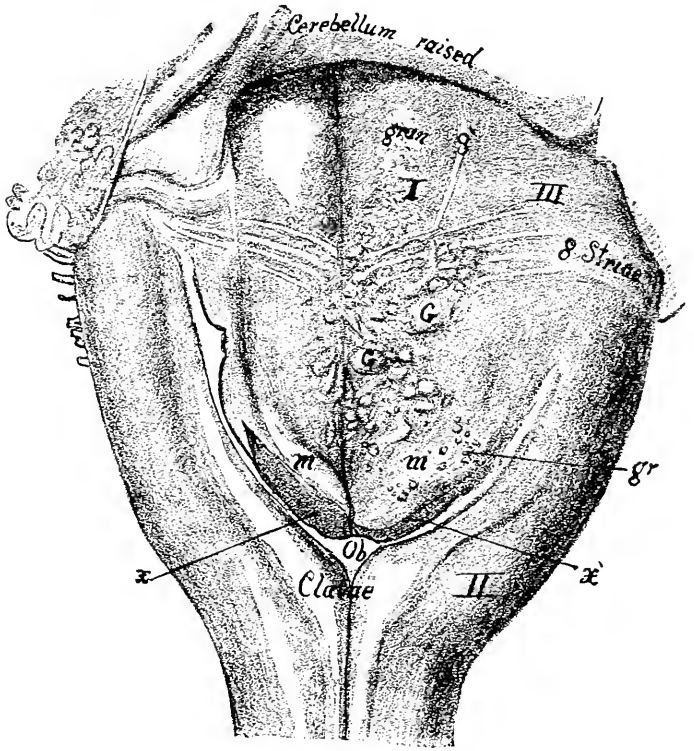


Fig. 1.

Dorsal face of pons-oblongata transition of Mary A——, magnified two diameters, and drawn from the fresh specimen. I., II., and III. indicate the inflated region, the cerebellum is raised. *Ob*, obex; *m*, *m`*, *alæ albæ mediales*, crowded asunder by *G*; *G*, *G* translucent region extending along ventricular sulcus; 8 *Striæ*, *striæ medullares albæ*; *g`*, so-called "Klangstab" (*scala rhythmica* of Bergmann); *gr* endymal granulations in posterior angle of ventricle; *gran*, same near anterior angle; x, x`, *alæ cinereæ*, crowded aside.

A section made through the upper sixth of the oblongata showed that the abnormal side was greatly hypertrophied, and that in consequence the contour lines were less sharp than in the normal condition. At the same time the more healthy side had also been distorted, chiefly because the

raphe portion had degenerated in obedience to the distending influence of the tumor.

As this was the only level where the topography of the lesion was observed while in the fresh state, its description may be here given. The section included both glossopharyngeal and subendymal vagi nuclei, the so-called nucleus of the fasciculus teres and the upper part of the hypoglossal nuclei, the restiform columns, the ascending trigeminal nerve roots, the olives, interolivary layers, pyramids, and the trans-section of the solitary bundle.

The lesion was distinctly marked by the translucent nature and gray-blue tint of its area, which while it passed gradually into the normal texture, did so within a very narrow zone, not exceeding a millimetre in width. The focus of the lesion was at the ventricular floor, at the vagus nucleus and trineural fasciculus, and extended from here deeply into the reticular formation, becoming lost before the olive was reached; the ascending root of the fifth pair and the restiform column showed their normal whiteness. The lesion extended across the median line at the ventricular floor for four millimetres, and along the left side of the raphe for the same extent; on the right side it extended down as far as the interolivary layer, including its upper half. There was a spot of softening, in the ectal half of the interolivary stratum, of the diameter of three millimetres, situated in what subsequent examination showed to have been the peripheral zone of infiltration.

The consistency of the morbid infiltration was slightly less than that of fresh nervous substance, but more elastic. It was nowhere diffluent in this level, except in so far as the spot just mentioned may be so regarded. Indeed, it maintained its consistency so well during the first stages of the hardening process, which was conducted in ice-surrounded vessels, that it was hoped nothing would interfere with the obtaining of a complete and regular set of sections. This hope was deceived; the neoplasm hardened so differently from the rest of the specimen that after-treatment with glycerin, involving all the disadvantages attending that method, became necessary.

(*To be continued.*)

NOTES ON THE FORAMINA OF MAGENDIE IN MAN AND THE CAT.

By BURT G. WILDER, M.D., Ithaca.

TWO years ago (*N. Y. Medical Journal*, April 26, 1884, p. 458) I expressed doubts as to the existence of communications between the metepicœle ("fourth ventricle") and the subarachnoid space. In the light of Hess' paper on the subject (Morphologisches Jahrbuch, x., 578–602, 1885), I have recently examined several human and cat brains, and reached the following conclusions:

1. In man and the cat the arachnoid passes from the lateral and caudal surfaces of the cerebellum to the oblongata and myel, so as to leave a "subarachnoid space."

2. In both there are "lateral recesses" of the metepicœle extending laterad just caudad of the cerebellar mediped-uncle along the course of the auditory nerve root. These recesses expand upon the sides of the oblongata more in the cat than in man, and justify Reichert's comparison with the "lateral ventricles." The endymal floor of the expanded portions rests upon the roots of the glosso-pharyngeus and vagus nerves. They are continued caudad, narrowing considerably, and open at either side into the subarachnoid space.

3. In both there are plexuses in the lateral recesses, formed by intrusions of the vascular pia as in the medicornu.

4. In the cat the metacœle (metencephalic part of the "fourth ventricle") is completely roofed in by a continuous metatele, so that there is no "foramen."

5. In man the metacœle opens into the subarachnoid

space by a mesal "foramen of Magendie" approximately rhomboidal in outline, about 5 mm. wide and 8–10 mm. long.

6. The recesses and orifices are not always described or figured in anatomical works, and never, so far as I know, fully or correctly; for example, the margins of the mesal foramen are represented as smooth by Henle ("Handbuch," Nervenlehre, fig. 232) and Schwalbe (Hoffmann u. Schwalbe, "Lehrbuch," Neurologie, fig. 256), whereas they are irregular or ragged, even when most carefully exposed.

7. The existence of any natural cœlian orifice is so significant morphologically, and so suggestive physiologically, that the parts here concerned merit very careful examination in man and comparison in animals. Among other points it is desirable to know whether the orifices permit the passage of liquid in one direction more readily than in the other.

8. When the main facts are ascertained it may be possible to determine whether the recesses constitute the lateral divisions of a tripartite metacœle, or whether they bear the same relation to the epicœle. If Gratiolet's figures of the embryonic human cerebellum (Leuret et Gratiolet, "Anat. Comp. du Syst. Nerv.," Pl. xxix, Fig. 1, 6) are correct, the cerebellar "ventricle" (epicœle) is already tripartite, as in some sharks, and the recesses may be assigned to the metacœle.

MASSAGE IN NERVOUS DISEASES.

By Dr. GEORGE W. JACOBY,

PHYSICIAN TO THE CLASS OF NERVOUS DISEASES OF THE GERMAN DISPENSARY OF THE CITY OF NEW YORK.

III.

(*Continued from page* 159.)

Migraine.

WITH all our present knowledge, and after all the classic writings of Broussais, Eulenburg, DuBois, Reymond, and Romberg as to the causation of migraine, we are still bewildered when it is required of us to assign a certain unvariable etiology to this affection. I do not believe this to be possible, and am sure that migraines in various persons are due to various causes. That there are various types of migraine is acknowledged, but that this affection may be of purely local origin is not so well known. Grasset, in 1879, went so far as to say: "Migraine is always the manifestation of a general condition, of a constitutional state" (*état*). Were this statement correct and the fact such as Grasset has so dogmatically put it, the field for massage in the modification of this affection would be a very limited one.

Certainly a migraine dependent upon a general neurasthenic condition may be relieved by modifying the general status, and that massage of the entire body is valuable in accomplishing this end is now well known, but it would be a manifest error of judgment to deduce from this that general massage is serviceable in migraine. It is the neurasthenia which is favorably influenced by it, and with an im-

provement in the general neurasthenic condition, symptoms dependent upon this will necessarily subside.

So of this, and of allied cases, I have nothing to say here, but there are two classes of cases, one of which I am inclined to believe occurs very frequently, which are in the majority of instances relieved, and in very many cured, by massage.

These are: 1st, Migraine dependent upon reflex hyperæmia, or vasomotor increase of the carotid blood-supply of the brain, which are relieved by neck-massage, or the use of the percuteur; and

2d, Migraine dependent upon peripheral irritation of one or more branches of the trigeminus.

It is this latter class which is much more frequent than is supposed. In cases occurring in anæmic neurasthenic patients, I have never seen any results from local massage, notwithstanding Reibway's assertion to the contrary. Of the first class of cases, the following two, taken from my case-book, are interesting:

CASE 1.—Female, æt. twenty-seven; married; no children; menstruates regularly. At age of fourteen had an attack of migraine, occupying principally the left side of the head, but the right side was not entirely free. This attack was attended with nausea and vomiting and great aversion to light. It lasted all day, till she went to bed at night, when she fell asleep, and woke up the next day free from pain. These attacks repeated themselves at irregular intervals during this entire time, and were always attended by the same symptoms, including flushing of the face and a feeling of throbbing in the neck. Stooping increased the pain materially. She had submitted to various modes of treatment, but in vain. For the last year she had had attacks from three to four times monthly.

Nov., 1883, she first came under my treatment. After several months of the useless employment of galvanism, I determined to attempt to cut short the next attack by the use of neck-massage. The attacks generally came on soon after getting up in the morning, and she was then obliged to spend the entire day upon the sofa. They only left her after a good night's sleep.

The next attack she had, I massaged the neck according to Gerst, for about fifteen minutes, when she said she felt much better. The pain had ceased almost entirely. She was free from pain the rest of the day. She remained under treatment until Sept., 1884, during which time I saw her only when she had an attack. The result was the same each time—almost complete relief after a comparatively short séance. The attacks however did not decrease in frequency. She then moved out of town, and I instructed her husband how to apply the massage. In a letter received in response to an inquiry of mine, dated Dec. 11, '85, she says: "I am always able to put an end to my sick-headaches by the use of the massage, and am, I think, sometimes able to prevent an attack. My husband and myself are sure that they do not occur as often now as before. Of all things that I have made use of this is the only one that has helped me in the least." So in this case where the result was only palliative, we have all reason to be satisfied with the effect.

In the following case it was the use of the percuteur and of Klemm's muscle-beater which relieved the attacks and also increased the interval between them. A cure, however, was not accomphished.

CASE 2.—J. P., male, æt sixty, an actor, has had attacks of hyperæmic (angio-paralytic) migraine ever since he was a young man. The attacks occurred at irregular intervals, but at least twice a month. He was never incapacitated from acting on account of them, but this was only due to his force of will, for during the day he did not attend to any work and spent most of his time on the lounge. When he came under my treatment, I first used massage of the neck but without any appreciable result. Then I made use of Granville's percuteur. The flat disc end was used, and the percuteur applied to the base of the mastoid process and moved forward over the temporal ridge to the forehead. Then very lightly over the scalp, moving it from before backwards, and *vice versa* in parallel lines. He was instructed to inform me as soon as he felt relief, which generally occurred in from three to five minutes. The pain frequently returned in a very short time and the percuteur

was again applied. I was invariably, from the first time on, able to relieve the attack by this means, and this for a man in his profession was invaluable. During the interval he himself made use of Klemm's muscle-beater (size 3 c.) in the manner directed by me, and he avers that the attacks became more infrequent. Of many cases of this class treated by this means I have no record of any absolute cure.

For the employment of this beater in migraine, Klemm gives the following directions: If the head is very sensitive, the beating is to be commenced very lightly and to be applied at first for about two to three minutes, later from four to six minutes. The entire application is to be divided into two periods, in such a manner that the patient beats during two to three minutes, then a pause, and then finishes in from two to three minutes more. The pause must always last until the peculiar feeling which is caused by the tapôtement has disappeared. Cases of cure of migraine by tapôtement have been reported by Laisné who uses a form called "Massage par pulpation" because he makes use of the points or pulps of his fingers. The manœuvre is very difficult of execution, and requires very great dexterity on the part of the operator. Faye has reported two cases of cure by this method. I personally have never been able to attain sufficient dexterity to employ it with satisfaction, but a description of it may be of interest.

Weiss says the procedure may be compared to the quickest tempo-pianissimo, which a pianist executes; with the exception, however, that a pianist uses the points of his fingers, whereas the masseur uses the pulps of the last phalanges. He describes the method in dealing with a migraine situated upon the one temple and spreading over the entire forehead, part of which I reproduce: "After the patient has been placed in a chair, in as comfortable a position as possible, the operator places his fingers, slightly spread upon the forehead, and now begins to beat in a quick tempo, as if he were beating a drum-reveille (Laisné), passing gradually downwards until he reaches the right ear. From this point on the beating must be discontinued, but the fingers must glide down until they have

reached the lower part of the neck, gently stroking. This is only part of the description, but is sufficient to show that the method is a combination of very rapid tapôtement and effleurage of the neck. It will certainly be found easier in practice to make use of the percuteur in conjunction with effleurage of the neck, than to attempt to execute the exceedingly wearisome and difficult massage par pulpation.

The second class of cases of migraine which are particularly amenable to massage, and which in my opinion occur much more frequently than is generally supposed, are those cases which are dependent upon some peripheral irritation of branches of the trigeminus or perhaps of the sympathetic.

In order to clearly understand the mechanism of these cases it will be necessary to take a brief glance at the anatomical distribution of these nerves.

The trigeminus branches which supply the dura mater are derived from the first branch the n. recurrens, a thin branch derived from the ophthalmic, and which spreads out between the lamellæ of the tentorium. According to Arnold this recurrent is formed from one or several delicate branches which, turning backwards, are joined by a small filament from the carotid plexus. It takes its course for a small distance in the sheath of the n. trochlearis, without, however, forming any anastomosis with it, and after leaving it, divides into several very thin filaments, which are distributed to the tentorium, to the sinus tentorii, petrosus and transversus, and terminates in their walls.

From the second branch there is also a nervus recurrens, from the supramaxillary branch to the dura mater. This branch, according to Arnold, arises by means of one or two very thin roots from the second or from the angle between the second and third branches of the trigeminal, and proceeds to the body or the anterior branch of the middle meningeal artery, and joins with the recurrent branch of the inframaxillary. From the third branch of the trigeminus there is also a recurrent nerve, arising in the foramen ovale, or immediately below it, and turning back to the interior of the cranium. This branch, which is known as the n. re-

currens inframaxillary, follows the middle meningeal artery, dividing with it into an anterior and posterior branch.

From the superior cervical ganglion of the sympathetic arise two groups of nerves which supply the heart, and which form connection with the various cerebral nerves, either directly or indirectly; the one group going upward with the internal carotid artery, and the other with the external. It is probable that the chief nerve supply of the pia mater is from this source. From these anatomical facts we can easily understand how an irritant affecting the peripheral branches of the trigeminal or the cervical sympathetic may produce a migraine of the affected side. In the first case, by direct propagation along the recurrent nerves of the trigeminus to the dura mater, and in the second case by producing a change in the calibre of the blood-vessels of the dura or pia, and thus a change in the circulation; or perhaps in both cases the pain is caused by this vasomotor change.

Certain it is that the pain in migraine must lie in the covering of the brain, and we see from this brief anatomical survey that, theoretically at any rate, the pain can be produced through peripheral irritation; practically we will see that there is a great deal to support this view of the pain production in very many cases.

In a large number of cases of migraine, points which are sensitive to pressure can be found. These points correspond to the course of the trigeminus, to the cervical sympathetic ganglia, or to the insertion or the course of the muscles of the face and neck. In most all cases in which pain upon pressure is thus found, irradiations to the habitually affected parts or frequently attacks of migraine may be produced by the pressure. At or in the vicinity of these points indurations, varying in size and consistency, will often be found. Massage of these local indurations will cause their disappearance, and with it a cure of the migraine. The probability, according to the present scientific data, is that these are inflammatory products (Lender, Mezger, Norström) and not swellings produced as a result of hyperæmia of the nervous filaments after their exit from

the bony canals, as has been affirmed by others. Lender, Henschen, Vretlind, and Norström have all cured cases of inveterate migraine by massage of these painful points. Henschen also calls attention to the fact that a careful search for these points is of importance, and he says: "It is well also to remember that in light cases of migraine, tumefactions so small are present that it is difficult to decide as to their nature." "They are the more sensitive, and the more extended, the longer the migraine has lasted. The tumefactions change in shape and volume, according to the case; generally they are very small and produce elevations of the skin scarcely perceptible to the eye."

To gain an idea of the frequency of these indurations as a cause of migraine, statistics, comprising all cases of migraine, taken indiscriminately, would be necessary. Such statistics do not exist. Norström has published a table of indurations found, together with migraine, but his cases are all selected cases, and serve at most to show the most frequent seat of these indurations.

In 32 cases he found induration at:

The superior insertion of the muscles of the posterior part of the neck—14 times.

Body and inferior insertion of same muscle—19 times.

Muscles of the anterior lateral regions and of the shoulder—9 times.

Coverings of the skull—2 times.

Temples—3 times.

Sympathetic ganglia—2 times.

Norström, as a result of large experience, lays down the following law:

"Numerous migraines result from the presence, in the neighborhood of nerves, of indurations, dependent upon acute or chronic inflammation." In these cases then the massage must be applied directly to the induration wheresoever that may be found, entirely independent of the seat of pain, during the attack of migraine, and the proper place can only be found if a careful examination of the head, face, and neck is made in each and every case.

Norström has reported twenty-nine cases of migraine

with indurations treated by massage. Of these cases twenty-one were cured, seven ameliorated, and one was still under treatment.

The following cases are taken from these of Norström :—

CASE 1.—M. L. thirty-eight years of age. Has had migraine since twelve years. During the first eight years these migraines were of a violent character. Since four years they are not so severe. The attacks have been irregular during the last three years. They are produced principally by damp cold. She has fever attacks during the summer. The pain commences at the right temple and extends rapidly to the forehead, the vertex, and the back and upper part of the neck. The pain is very violent ; the patient believes, she says, that her head is splitting ; muscular twitchings, nausea, vomiting, and then the end of the attack. During all this time she is very pale, her face covered with a cold sweat. The pulse is increased, the eyelids become heavy, the eyes suffused, and intolerance to light. She is also sensitive to the slightest noise. The attack generally lasts twenty-four hours, sometimes forty-eight, or even seventy-two. In this case it consisted principally of a continuous and severe feeling of heaviness, interrupted by true pains. Black coffee relieves her somewhat. Chloral was of some service at first but is now without effect. Since some time she takes a preparation of morphine. Electricity produced no amelioration. On the right side is found a painful tumefaction of the cranial attachments of the trapezius and splenius. In following the edge of the trapezius from above downward, at the lower part of the cervical portion, a region which is more indurated, is found. Near by, a ganglion is swollen and sensitive to pressure. On the other side there is only slight sensitiveness to pressure, over the mastoid process. Treatment by massage, commenced in September, 1883.

In a month, slight amelioration. The treatment was then interrupted until the end of November. After two months, very marked amelioration ; the attacks are less frequent, and limited to the supra-orbital region, and even here the pain is less, than before treatment. This amelioration has persisted.

CASE 2.—Male, æt. thirty-six; has had attacks of headaches since ten years. In 1878 subacute rheumatism of the leg and of the right thigh. Warm baths, douches, gymnastics, without result. Icterus in 1880; from this time on the pains disappear. Besides these rheumatic symptoms, he has a continuous headache, principally on the left side. Paroxysmal attacks once, twice, or three times a week, lasting from two to six hours. Sometimes the attack is so severe that the patient says he does not know how he stands it, and sometimes the attack produces only a feeling of fulness and vertigo. Severe pain, when pressure is exercised over a small space of two centimetres in the left parietal region. In the superior part of the trapezius of the left side several indurations are found, which are more or less resistent, and very sensitive to pressure. This pressure is sufficient to produce an attack. After raising the arm, a well-marked induration of about the size of the pulp of the small finger is easily discovered in the acromial portion of the trapezius, and pressure over this point is also painful. Treatment by massage was commenced in April; complete cure after two months. The indurations, and with them the paroxysms of headache, have entirely disappeared. Testimony to the value of massage in migraine is also given by Dr. Stoddard, of Northampton, in a communication to Dr. Douglas Graham. He says: "In the case of the same patient, a nervous headache, to which she had been long subject, was always much alleviated by the application of massage to the head."

Dr. C. K. Mills, of Philadelphia, said in 1878: "I have frequently seen the headache, of a nervous woman relieved by gentle stroking of the forehead, while energetic frictions or shampooing of the entire head are sometimes more efficacious with men."

Cervico-Occipital Neuralgia.

Personally I have had no experience in the treatment of this affection by means of massage; in fact, a pure cervico-occipital neuralgia is not a very common occurrence. In connection with migraines and particularly with neurasthenic migraine, this neuralgia occurs more frequently than

alone. In such cases general treatment perhaps with massage would be indicated. Those cases which have come under my observation, in which the n. occipitalis major was affected in conjunction with the inferior branch of the trigeminal, a concomitance which is not infrequent, the same treatment as applied to the trigeminus was also used here, and it always seemed to me that the occipital neuralgia was never as obdurate as the trigeminal.

Vretlind relates the case of a man æt. thirty. He had lancinating pains in the right half of the face and neck, shooting upwards into the occipital region, and downwards in the neighborhood of the vertebral column. These pains came and went very qnickly. In examining the nerves and muscles of the affected region, an induration is found upon the superior border of the trapezius, and also a small painful spot behind the mastoid process.

Massage. Amelioration after the fifth séance. Entire cure after ten applications.

Schreiber relates the following:

Mr. S. R., suffering from a trigeminal and occipital neuralgia. The pains in the back of the neck and the posterior part of the head, in the forehead, and on top of the head were so severe that the patient, during months, was unable to read or to write. After the patient had undergone a course of hydropathic treatment, without any result, he came under Schreiber's care. After three weeks of mechanical treatment, the occipital neuralgia disappeared entirely, but the neuralgia of the trigeminus, the pains in the forehead, etc., were not relieved, although they were always temporarily alleviated. The occipital neuralgia remained cured, but the patient went elsewhere for relief of his other pains.

Beuster also treated three cases of occipital neuralgia by massage, with the result of curing them. He considered his cases due to a periosteal enlargement, which pressed directly upon the nerve, and therefore directed his treatment entirely to this point.

Cervico-Brachial Neuralgia.

This class as commonly understood embraces neuralgias of all the nerves which are derived from the brachial plexus

or from the posterior branches of the four lower cervical nerves.

This large dissemination makes their occurrence frequent, and certain ones of them are peculiarly susceptible to the mechanical treatment. The diagnosis occasionally is a question of some difficulty, particularly frequent is the mistake of taking a cervico-brachial neuralgia for a muscular rheumatism. Should such a mistake occur, it would however only increase the chance of success by the massage treatment. If, however, an articular rheumatism or any other affection of the joint itself is the primary cause of the neuralgia, the massage can only act injuriously, except in chronic cases, where the neuralgia subsists after subsidence of the joint trouble, or in neuralgias dependent upon a periarthritis, particularly of the shoulder-joint, as is frequently the case.

Petrissage and tapôtement, together with active and passive movements, will be found of most service in the treatment of these neuralgias. Fresh cases, particularly those dependent upon a rheumatic basis, will be very easily cured by massage, and sometimes in a remarkably short time.

Besides the chronic exudation in and around the shoulder-joint, we must not neglect in these cases to examine carefully for indurations in the muscles. Mezger has called attention to the fact that certain neuralgias of the arm are caused particularly frequently by indurations in the sterno-cleido-mastoid. Should such indurations be discovered, the treatment is clearly indicated.

Westerland reports tha case of Mrs. A., æt. thirty-four, of robust constitution, not hysterical ; feels a kind of fulness in her arm in January, 1874, which soon changes to a true pain, occupying the shoulder and the right arm.

Frictions with various liniments and electricity, without result. The beginning of July the neuralgia became still more severe, in consequence of violent movements. Massage. The pain, concentrated principally around the shoulder-joint, extended as far as the elbow, following the course of the musculo-cutaneous nerve. No indurations, swelling, or redness. The motions of lifting the arm or carrying it

backward are particularly painful. Petrissage and tapôtement, amelioration after eight séances. Return of pain after six weeks. Twelve more applications. Cure. No return after fifteen months.

Norström reports the case of a Mrs. N.:

Mrs. N. complains, since March, 1877, of a sensation of *fourmillement* in the right arm. This feeling, which is more disagreeable than painful, does not trouble her. Later it increased so as to become extremely irritating. The pain is settled in the axilla, and radiates down the forearm along the course of the ulnar. Faradic currents, Russian baths, frictions with various ointments, did not bring about any result. The pain also radiated to the subscapular region and the neck. This all disappeared for a certain length of time, and she considered herself well, when, in consequence of a violent movement of the arm, the pain returned more severe than before. Sleep was difficult and restless. When Norström saw her for the first time he could find nothing abnormal in the shoulder-joint nor in the muscles of the arm. The movements had their full scope, but that of elevation and the movements backward were very painful. A pressure upon the course of the musculo-cutaneous nerve, a sudden movement of the arm or the shoulder, were sufficient to provoke a paroxysm. The same treatment as heretofore was resorted to, but this time without result. After twelve séances of massage the patient is considerably improved. Commences to use her arm. After thirty séances, radical cure.

Schreiber also relso relates a case of a male, æt. fifty-eight, who had been suffering from a cervico-brachial neuralgia for three years. He was cured after four weeks' daily mechanical treatment, each application not lasting for more than ten to fifteen minutes. S. calls attention to the great pain produced in this case by the mechanical treatment, and advises operators not to desist from thorough application on account of pain at first.

The following case, in my own practice, of radial neuralgia is still under treatment.

Mrs. P., æt. forty-eight, fell upon the ice three years ago,

striking principally upon the left shoulder. A periarthritis was developed which lasted for six months. During this time she complained of pain in various places of the upper part of the arm. Gradually the pain concentrated itself more and more, until it was located principally along the course of the radial nerve. When I saw her in September of this year I found considerable atrophy of the deltoid with its attendant symptoms, very severe pain upon pressure over the radial where it winds around the humerus, and also above the wrist-joint. The pain caused by the pressure radiated downward into the thumb and first and second fingers. The deltoid regained its consistency and power after three months of electrical treatment, combined with active and passive movements. The neuralgia was during this time also treated by means of the constant current, but without any apparent benefit. Early in December the use of massage was begun. The application took place three times a week with more or less regularity, petrissage and tapôtement being chiefly used. Over the upper and lower sensitive points Granville's percuteur was used. The effect was immediate and marked. Already, after the fifth application, the spontaneous pain, which had been exceedingly severe, had very much diminished in intensity, and the attacks of pain were more infrequent. At present she is well as far as spontaneous pain is concerned, but the nerve is still quite sensitive to pressure. I have, however, no doubt but what this also will disappear.

Berghman has also reported a case of cure of ulnar neuralgia.

Miss Sophie L. suffers since four and a half years with violent pains in the right arm. These appeared without any known cause, and soon became so severe that she had no rest night or day. They come on particularly when she wishes to work or when she performs any movement with the arm. The pain commences at the elbow and radiates downward to the ends of the fingers, particularly of the ring and small fingers. Various remedies have been tried, but always without success. Electricity had been used for an entire year. She was finally admitted into a hospital of Stock-

holm as incurable. B. began the treatment in the hospital on the 13th of May, 1873. He then noticed a constant tremor in the fingers of the left hand, particularly in the two last. Pressure upon the ulnar in the sulcus produced severe pain in the above specified territory. Pressure upon the nerve in the rest of its course also produced pain. No swelling was noticeable. Sensibility very much reduced along the ulnar side of the arm. Nothing abnormal in the muscles or joints. Motion not impaired to any greater extent than that due to pain. After eleven séances of massage patient was for the first time free from pain for a day. After the eighteenth application, a second painless day. After she had been free from pain for three weeks the treatment was experimentally interrupted, but in ten days the pain again recurred, although not nearly as severe as before. After four days of treatment the pain again disappeared.

Westerland reports a case of neuralgia of the musculocutaneous nerve, which has lasted for one month, during which time galvanism had been employed, but without effect. Massage. Cure after fifteen applications.

Intercostal Neuralgia.

Neuralgias of the trunks of the intercostal nerves are in my experience not at all susceptible to the mechanical treatment. I have always been able to obtain better results by means of some other remedy. The anatomical situation of the intercostal muscles makes it evident that they cannot be reached by means of any of the ordinary manipulations. Even the use of the percuteur, which I have tried in several cases, seems to fail in effecting any amelioration. Nevertheless, the following cases serve to show that in the hands of others it has occasionally been successful, and may therefore be tried before giving up a case as hopeless.

The pain, however, is generally so intense, as to discourage both patient and operator. When, however, the superficial branches—those which supply the skin of the abdomen and chest are alone affected—then a cure by massage may be almost positively predicted, and the manipulations

required to effect this result are of the simplest. Pressure and kneading of the affected points suffice.

Schreiber relates a case of intercostal neuralgia, which was accompanied by fever and difficulty of respiration, so that the idea of an exudation in the pleural cavity was first entertained. Upon examination a spot of great sensitiveness was found between the 6th and 7th rib in the axillary line. Pressure caused the patient, who was a physician, to cry out with pain. The pains spread from here into the hypogastrium. Massage was at once employed, and, although very painful, the patient was very much relieved by it. The following day the manipulations were repeated, with the result of effecting a complete cure.

Zabludowsky refers to a case of six years' standing in a man sixty years of age. Two séances weekly. The application consisted of intermittent pressure applied to the intercostal region. After one month's treatment the pain remained absent for several weeks. The attacks, which again recurred several times, had, however, become very slight.

Johnson has described a case of neuralgia of the right intercostal nerve between the 5th and 6th ribs, with painful radiations into the corresponding side of the abdomen, consecutive to a herpes zoster. Massage. Cure.

In this connection it is necessary to make a few remarks about muscular rheumatism, particularly rheumatic torticollis and lumbago. Although these affections do not really come under the heading of diseases of the nervous system, still they are so often referred to the specialist for treatment, and real neuralgias are so often diagnosed as "muscular rheumatism," that it may be well to speak of this affection here. Senator justly says that it is impossible to give a correct definition of muscular rheumatism at the present state of our pathological knowledge. The prominent symptom is pain, and whether this is dependent upon a disorder of the circulation, hyperæmia, or upon an exudation of serum—diffuse or circumscribed,—or upon an affection of the intramuscular sensitive nerve-terminations, cannot be determined. In the latter case it

would be a purely neuralgic affection, and although many cases described as muscular rheumatism may be due to other causes, I am inclined to the view that the majority of those unaccompanied by any swelling are really due to the latter cause. The fact that superficially situated muscles are the ones generally affected, would go to support this view. Rheumatic torticollis is really an idiopathic tonic contracture. The diagnosis must in these cases also naturally be a correct one, otherwise massage, as every thing else, will be of no avail. Muscular pains due to some central affection of the nervous system cannot be relieved by massage. There would be no disadvantage in mistaking a neuralgia of any smaller nerve for muscular rheumatism. The treatment would be about the same. Whatever the pathology of this affection, there is no fact which is now better acknowledged than that first supported by Benedict—that rest in muscular rheumatism is erroneous, and that the affected muscles can be relieved in a much shorter time by gymnastics and mechanical manipulations. Whoever has paid any attention at all to the treatment with massage cannot fail to have been surprised by the rapidity with which many cases of lumbago and torticollis may be entirely relieved. I have seen many cases cured after two to three applicatious, and all authors agree that but very few séances are necessary. Some cases require more time than others, but the final result is always an excellent one. Fresh cases are more easily benefited than old ones. As regards the kind of manipulation to be employed, there is not much to be said. Effleurage and massage à friction for superficially situated muscles, and petrissage and tapôtement for those more deeply situated. The hand will be found to be the most serviceable in these cases for executing tapôtement. Klemm's muscle-beater acts too superficially to be of any value. Very many cases of cure have been reported by Strohmeyer, Berghman, Johnson, Faye, Bruberger, Wagner, Douglas Graham, and others, so that the beneficial influence of massage upon muscular rheumatism is now generally acknowledged, and further testimony in this direction would be superfluous.

A CASE OF ABSCESS OF THE OCCIPITAL LOBE WITH HEMIANOPSIA.*

By E. G. JANEWAY, M.D.

CASES of hemianopsia are of interest, if associated with an autopsy, to show the position of the lesion inducing the symptom. The paper published by Dr. E. C. Seguin on this subject[1] is of so recent a date, and so carefully prepared, that I have only fulfilled a promise made long ago to the chairman of this section, that I would present some matter before his term of office closed, and have, in its fulfilment, thought that a practical study of some features of the particular case which I will bring to your notice would prove of interest. I do not claim for this instance any possibility of deciding the location of the visual centre, owing to the secondary effects of the lesion and the gaps in the history. It has only in this regard a cumulative influence.

The patient, J. C., a machinist, was twenty-five years of age. His history, as I learned it at the clinic, Tuesday, January 19th, and as given to the physicians at Bellevue Hospital, was that on Nov. 4th he had been struck in the head by the butt end of a revolver, the injury being inflicted on the left side at the posterior part of the vertex. He was considerably stunned, but not rendered unconscious by the blow, which caused a scalp wound. He returned to his work on the following day, but after three days had severe headache. At this time he had an attack of inflammation of the scalp, perhaps erysipelas; pus also escaped

* Read before the Neurological Section of the N. Y. Academy of Medicine.

[1] In the January number of this JOURNAL.

from the opening in the scalp. Several weeks after the accident he noticed numbness, first of the arm, and then of of the leg, on the left side.

About six weeks after the accident, and three weeks before the time when I first saw him, weakness came on in the arm and leg of the left side. This gradually increased, until at the time of observation it constituted a well-marked paresis. He had been obliged to take to his bed after it had existed two weeks (a week before admission). He had complained of headache more or less from the time of the accident until observed.

When observed, the patient was found to have a cicatrix of the scalp, on the left side, about an inch in length, situated in the supero-parietal region of left side. He had anæsthesia of the left side of face, of left arm and leg, less in the face than in the other situations, and not complete in arm and leg. He had hemiparesis of left side, but not involving the face or tongue, though some difference was carefully looked for. He lay in a sort of doze most of the time, rousing up at times, and complaining of the pain in his head. Yet, when spoken to rather sharply, he would answer questions in a perfectly rational manner, and showed no indication of word-blindness. I found that he had well-marked hemianopsia of the left field of vision. The sight seemed at first to be intact for the right field of vision, but it was somewhat blurred. An ophthalmoscopic examination showed well-marked choked disc in both eyes. For the first five days after admission he showed no elevation of temperature; after this for five days there was an evening rise to 99½. The pulse was at first slow and regular, but with the elevation of temperature became somewhat increased in frequency.

As the disease progressed he became more stupid, but still able to be aroused. Ten days after admission his pulse reached (forenoon) 106, temperature 100½; (afternoon) pulse 156, temperature 101¼. From this time on his pulse was frequent, temperature elevated, though not exceeding 102, and varying somewhat. On the thirteenth day after admission Dr. J. D. Bryant trephined

the skull on the right side, at a point nearly corresponding to the injury on the left side. Through this opening a hydopermic-syringe needle was introduced, at first directly downwards, then toward the occipital lobe. On one occasion a slight amount of slightly colored serum was withdrawn, but in subsequent exploration with an aspirator in this same direction no more fluid could be obtained. An opening was also made at the point of injury, where rarefying osteitis existed, with no result.

The pulse was over 140 at the time of the operation, which had been performed as a sort of *dernier ressort*, owing to his failing condition, in the hope of striking the abscess and relieving the symptoms. After the failure of the exploratory punctures, the question of trephining over the occipital lobe was raised, but from his increasing weakness of pulse, and the failure of the aspirator introduced on two occasions in that direction to obtain pus, it was given up. He died nine hours after the operation, though the pulse had fallen somewhat in the meantime. His temperature two hours before death was 102° F.

The autopsy showed two trephine openings on nearly opposite sides of the skull. That on the right side was situated in the postero-superior parietal region, and corresponded to the interspace between angular and superior parietal lobule. A clot of blood filled this portion, the size of half the thickness of end of little finger. There was no serous nor purulent exudation in the meshes of the pia mater. The dura was adherent to pia over the occipital lobe, both on its mesial and convex aspects. The white matter of the right hemisphere, as far forward as the fissure of Rolando, was softened and œdematous. The occipital lobe was the site of an abscess, round in shape, situated in its white matter and approaching the surface posteriorly, where but a thinned portion of gray matter and the wall of the sac intervened between it and the adherent dura and pia mater. The abscess was bounded by a capsule, and contained a thin yellowish-green sero-pus. The opposite hemisphere and the base showed nothing of note. Owing to the soft state of the affected portion, I had it immediately placed in

Müller's fluid. Unfortunately, the hardening, as you see, has not been satisfactory, and in preparing it for exhibition so as to show the relationship of the abscess, the convolutions have fallen in, in a manner to disfigure the specimen. By a comparison with the opposite side it will be seen that the abscess occupies the greater part of the occipital lobe medullary matter, so as to affect the fibres coming from the cuneus, as well as the occipital convolutions and those from the basal surface. The abscess was about two inches in longest diameter.

This case well illustrates the difficulties which at times surround a localization of a cerebral lesion. The patient comes under observation at a late period of the disease; some symptoms of importance in diagnosis are then first observed, and it is impossible to learn from the patient when they made their appearance. In the history as given, slight numbness, followed by paresis, preceded the occurrence of the hemianopsia. The time, too, of occurrence of hemianæsthesia is uncertain, though probably it antedated the paresis.

The question as to the hemisphere of the brain could only be answered in one way, viz., the right. The paresis, the anæsthesia, hemianopsia, all existing on the left, together with the constant turning of the head to the left, demonstrated a right-sided lesion. The difficulty was to define the centre of the lesion from the effects of the peripheral encephalitis. If the order of occurrence of the symptoms were taken into account as he gave them to me, it would point to a lesion not directly involving the optic fibres or centre, but producing first numbness and paresis, followed by the hemianopsia, due to involvement of these subsequently. In attempting to decide the point I felt that this was an obstacle to a positive conclusion. An inquiry of his friends, and of a physician who saw him previous to his entry into hospital, have not proved of service in establishing the periods of the development of the hemianopsia. As regards this point, I made the following statement:

The occurrence of hemianopsia and hemianæsthesia point toward the occipital lobe as the site of the lesion, but

the hemiparesis possibly antedating the hemianopsia, leads one to be somewhat uncertain in this respect, as a lesion may be so placed as to involve the central portion of the optic tract by secondary and not by primary change. Moreover, in deciding this I had to take into account the situation of the injury and the succeeding inflammation and suppuration, which were considerably anterior to the occipital lobe, and would, if the facts were of the order mentioned, point to a centre anterior.

As regards the nature of the lesion, my diagnosis was an abscess with secondary encephalitis. The reasons for this were: First, there had been an injury to the scalp with suppuration; after this, for a period of from three to six weeks, saving headache, he had been fairly well; then he began to show evidences of an advancing cerebral lesion. It was unaccompanied by the irritation phenomena of meningitis, but was accompanied by events which pointed to an involvement of the brain tissue. The occurrence of optic neuritis is of interest in connection with this case.

RACE AND INSANITY.*

By JAS. G. KIERNAN, M.D., Chicago,

LATE MEDICAL SUPERINTENDENT COOK COUNTY HOSPITAL FOR INSANE.

BEFORE proceeding to further discuss the intellectual phenomena presented by the negro insane, I have thought it best to summarize the different races in the present paper for purposes of comparison with the results

TABLE I.

Psychosis.	Male.	Female.	Total.
Mania	34	25	59
Melancholia	46	57	103
Katatonia	11	9	20
Stuporous Insanity	6	6	12
Primary Confusional Insanity	5	6	11
Acute Hysterical "	0	25	25
" Alcoholic "	15	13	28
Climacteric Insanity	0	5	5
Choreic "	1	0	1
Rheumatic "	1	1	2
Syphilitic "	6	4	10
Paranoia	92	56	148
Hebephrenia	36	15	51
Typhomania	2	3	5
Chronic Hysterical Insanity	0	31	31
" Alcoholic "	20	12	32
Phthisical Insanity	4	3	7
Traumatic "	16	5	21
Senile "	21	28	49
Periodical "	14	19	33
Circular "	9	8	17
Epileptic "	62	55	117
Paretic Dementia	79	13	92
Coarse Brain Disease Dementia	7	6	13
Terminal Dementia	15	31	46
Primary Mental Deterioration	9	8	17
Idiocy	3	3	6
Imbecility	7	6	13
Secondary Confusional Insanity	72	127	199
Total	593	550	1143

* A series of Studies in Ethnological Psychiatry.

obtained as regards race and insanity in New York city.[1] The number of insane tabulated is 1,143, and the psychoses presented are as above (Table I).

The first noticeable feature is that the psychoses which tend to increase the population of an insane hospital are not as frequent as might be expected, and this is clearly due to the enormous death rate. The Cook County Insane Hospital for the past ten years has had a death rate of more than double that of any insane hospital in the United States. The predominance of psychoses like paranoia which produce an errabund tendency is what might have been expected from the theatrical character of the wide reputation of Chicago.

TABLE II.—MANIA.

Race.	Blonde.			Brunette.					
	Male	Female.	Total.	Male.	Female.	Total.	Total Male.	Total Female.	Grand Total.
ARYAN									
Teutonic.									
Anglo-Saxon, American	4	6	10	1	2	3	5	8	13
" " English	0	1	1	1	0	1	1	1	2
German	6	3	9	0	1	1	6	4	10
Scandinavian-Danish .	1	1	2	0	0	0	1	1	2
" -Norweg.	1	1	2	0	0	0	1	1	2
Sclavonic.									
Bohemian	0	0	0	1	1	2	1	1	2
Polish	4	0	4	0	0	0	4	0	4
Celtic.									
Irish	6	4	10	1	2	3	7	6	13
" -American . .	6	2	8	0	0	0	6	2	8
" -Canadian . . .	1	0	1	0	0	0	1	0	1
Latin.									
Italian	0	0	0	1	1	2	1	1	2
Total	29	18	47	5	7	12	34	25	59

This table tends to bear out the old conclusion that mania is very frequent among blondes. That a general conclusion can be drawn of this character, free from racial elements of error, is obvious from the relative frequency of the psychosis among the blondes of races in which brunettes predominate, like the Sclavonic and Celtic. The

[1] Spitzka: JOURNAL OF NERVOUS AND MENTAL DISEASE, October, 1880.

Germans form about one half the population of Cook County, yet mania is much less frequent among them than among the Anglo-Saxons and the Celts. The English and American Anglo-Saxons seem to furnish about the same proportion of maniacs, since the American population is about six times the English. The American women seem to be more liable to attacks of mania than either the German or Celtic women. The Polish men seem most liable to mania, while the male Irish-Americans seem to be much more frequently attacked by mania than the women.

TABLE III.—MELANCHOLIA.

Race.	Blonde.			Brunette.					
	Male.	Female.	Total.	Male.	Female.	Total.	Total Male.	Total Female.	Total.
ARYAN.									
Teutonic.									
Anglo-Saxon, American	2	4	6	3	6	9	5	10	15
" " English .	0	1	1	1	0	1	1	1	2
German	3	8	11	8	6	14	11	14	25
" -American . .	3	3	6	1	0	1	4	3	7
" -Austrian . .	0	0	0	0	1	1	0	1	1
" -Swiss . . .	0	0	0	1	0	1	1	0	1
Hollander	0	0	0	1	0	1	1	0	1
Scandinavian-Danish .	1	0	1	0	1	1	1	1	2
" -Swede .	3	3	6	0	1	1	3	4	7
" -Norweg.	2	2	4	0	0	0	2	2	4
Latin.									
French-Canadian . .	0	0	0	0	1	1	0	1	1
Celtic.									
Irish	1	1	2	8	9	17	9	10	19
" -American . . .	2	1	3	4	5	9	6	6	12
Sclavonic.									
Bohemian	0	0	0	0	1	1	0	1	1
Polish	2	0	2	0	2	2	2	2	4
SHEMITIC.									
Hebrew[1]	0	0	0	0	1	1	0	1	1
Total	19	23	42	27	34	61	46	57	103

The old view as to the frequency of melancholia among brunettes seems to some extent to be supported by this table, but no very decided conclusion can be drawn from it in this respect. Melancholia seems to be more frequent

[1] Wherever Hebrew alone is used German Hebrew is to be understood.

among the Celts in Chicago than in New York, and this *a priori* was what was to have been expected. The enterprising Celt left the sea-coast, and he was much more likely to become the victim of depression than the Celt who took things coolly and showed this by remaining where he landed. The same cause which makes paretic dementia more common in Chicago among the Celts than in New York would tend to produce melancholia in the weaker Celtic brains.

TABLE IV.—KATATONIA.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	2	1	3
German	2	0	2
" -American	2	1	3
Hollander	1	0	1
Sandinavian-Norwegian	1	1	2
" -Swedish	0	2	2
Celtic.			
Irish	0	1	1
" -American	2	1	3
Sclavonic.			
Bohemian	1	0	1
Polish	0	1	1
SHEMITIC.			
Hebrew	0	1	1
Total	11	9	20

The percentage of cases of katatonia is much the same in Cook County as in New York. It seems, however, to be much less frequent among Celts than it is in New York.

TABLE V.—PRIMARY CONFUSIONAL INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	2	2	4
German	0	1	1
" -American	1	1	2
Scandinavian-Sweden	1	1	2
Celtic.			
Irish	1	0	1
" -American	0	1	1
Total	5	6	11

The causes in one half the cases were exhaustion from measles, scarlatina, varicella (one case) puerperal fever, malaria, and allied febrile conditions.

Five of the cases in the next table were due to sudden mental shock.

TABLE VI.—STUPOROUS INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, Irish	1	1	2
" " English	1	1	2
" " American	2	1	3
German-American	1	1	2
Scandinavian-Swede	1	1	2
Sclavonic.			
Bohemian	0	1	1
Total	6	6	12

TABLE VII.—ACUTE HYSTERICAL INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American		2	2
" " West Indian		1	1
German		8	8
American		1	1
Scandinavian-Dane		1	1
Celtic.			
Irish		9	9
Latin.			
Italian		1	1
HEBREW		1	1
NEGRO		1	1
Total		25	25

The Irish seem to be much more liable to acute hysterical insanity than the American Anglo-Saxons or the Germans.

This table bears out the usual conclusion as to the frequency of intemperance among Celtic women. The two Saxon American cases were the distant result of urticaria. The patients were advised to use alcohol to secure slumber, and very naturally went beyond their directions, resulting in an attack of alcoholic insanity, which was at first charged

to the urticaria, but investigation by friends, under a hint from the physician as to the real cause, soon revealed the true nature of the case.

TABLE VIII.—ACUTE ALCOHOLIC INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American		2	2
" " Scotch	1	0	1
German	4	2	6
" -American	1	1	2
Scandinavian-Norwegian	2	0	2
Slavonic.			
Polish	1	0	1
Celtic.			
Irish	4	4	8
" -American	2	4	6
Total	15	13	28

TABLE IX.—CLIMACTERIC INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American		1	1
German		2	2
Celtic.			
Irish		2	2
Total		5	5

TABLE X.

Race.	Choreic.			Rheumatic.			Syphilitic.			
	Male.	Female	Total.	Male.	Female	Total.	Male.	Female.	Total.	Grand Total.
ARYAN.										
Teutonic.										
Ang.-Sax. Am.	0	0	0	0	0	0	2	0	2	2
German	0	0	0	0	0	0	2	2	4	4
German-Amer.	1	0	1	0	0	0	1	0	1	2
Scandin.-Norwegian	0	0	0	1	0	1	0	0	0	1
Celtic.										
Irish	0	0	0	0	0	0	1	0	1	1
HEBREW	0	0	0	0	0	0	0	2	2	2
NEGRO	0	0	0	0	1	1	0	0	0	1
Total	1	0	1	1	1	2	6	4	10	13

TABLE XI.

Psychosis.	American.									Foreign.																	
	Anglo Sax.		Germ.		Celt.		Negroes.		Total.	Anglo-Sax.		Germ.		Hol.		Scan.		Hebr.		Slaves		Celts.		Latin.		Total.
	M.	F.	M.	F.	M.	F.	M.	F.		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
Mania	5	8			6	2			21	1	1	6	4	0	0	2	2	0	0	5	1	7	6	1	1	37
Melancholia	5	10	4	3	6	6			34	1	1	12	15	1	0	6	7	0	1	2	3	9	10	0	1	69
Katatonia	2	1	2	1	2	1			9	0	0	2	0	1	0	1	3	0	1	1	1	0	1	0	0	11
Stuporous Insanity	2	1	1	1	0	0			5	2	2	0	0	0	0	1	1	0	0	0	1	0	0	0	0	7
Acute Alcoholic Insanity	0	2	1	1	2	4			10	1	0	4	2	0	0	2	0	0	0	1	0	4	4	0	0	18
" Hysterical "	0	2	0	1	0	0		1	4	0	1	0	8	0	0	0	1	0	1	0	0	0	9	0	1	21
Primary Confusional Insanity	2	2	1	1	0	1			7	0	0	0	1	0	0	1	1	0	0	0	0	1	0	0	0	4
Climacteric Insanity	0	1	0	0	0	0			1	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	4
Syphilitic	2	0	1	0	0	0	0		3	0	0	2	2	0	0	0	0	0	2	0	0	1	0	0	0	7
Choreic	0	0	1	0	0	0	0		1	0	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rheumatic	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1
Total	18	27	11	8	16	14	0	2	95	5	5	26	34	2	0	14	15	0	5	9	6	22	32	1	3	179

The two Hebrew females suffering from syphilitic insanity acquired the syphilis from a criminal assault made upon them by hoodlums, and the same is true of the German females.

The chronic psychoses admitted were as follows:

TABLE XII.—PARETIC DEMENTIA.[1]

Races.	Male.	Female.	Total.
ARYAN.			
Tuetonic.			
German	12	1	13
" -American	2		2
" -Austrian	1		1
" -Swiss	1		1
Hollanders	5	1	6
Anglo-Saxons, American	19	1	20
" " English	3		3
" " Scotch	1		1
Scandinavian-Danish	1	1	2
" -Swedish	2	2	4
" -Norwegian	1		1
Celtic.			
Irish	19	7	26
Latin.			
French	1		1
French-Canadian	2		2
Sclavonic.			
Bohemian	1		1
Polish	1		1
SHEMITIC.			
Hebrew- German	1		1
" -American	1		1
NEGRO	2		2
Total	76	13	89

The psychosis in which recovery was probable formed but a small per cent. of those coming under observation—a little under five per cent. Excluding the periodical type from consideration, it is these last which swell the insane-hospital recovery rates. A large recovery rate is very fair evidence of a medical superintendent's ignorance or untrustworthiness. I have known of paranoiacs with concealed persecutory primary and exalted secondary systematized delusions being discharged as recovered from insane hospitals when a paroxysm of emotional disturbance had subsided. I have known superintendents to assure the

[1] The table concerning paranoia and a general racial summary will appear in next number, both having been mislaid.

wives of chronic alcoholic lunatics that the latter were recovering when the delusion of marital infidelity was as decided as ever. Here is one of the great sources of error which Pliny Earle has pointed out in the statistics of insane hospitals respecting recoveries. Dr. Hurd has called attention to the frequency of hallucinations in acute mania. In my opinion many of his cases belong to the primary confusional types which occur as a result of conditions of exhaustion from the specific fevers, etc.

TABLE XIII.—TYPHOMANIA.

Races.	Male.	Female.	Total.
ARYAN.			
Tuetonic.			
Anglo-Saxon, American	1	1	2
Celtic.			
Irish-American	1	2	3
Total	2	3	5

Typhomania[1] is in all probability much more frequent than here indicated, as many cases die before coming under hospital treatment, and the death is charged to "meningitis." The frequency of paretic dementia is clearly due to the speculative atmosphere of Chicago.

The percentage of hebephreniacs is much the same as in New York, the male percentage being somewhat greater. Two of the English cases had married and procreated several children before their mental condition was discovered. From certain indications I am inclined to believe that the chastity is not as distinguishing a characteristic of Celt as is generally believed. The infreqency of illigitimacy in Ireland is due to the ease with which marriage is contracted in that country as compared with the Continent. The unions formed on the Continent are in many cases just as pure and permanent, but lack the formal sanction of the law. The clan code of morality tends to prevent violations of chastity by members of the clan, but these readily occur when the individual is removed from the influence of the clan. The relative frequency of masturbation among the

[1] Délire Aigu. Bell's Disease. Grave Delirium.

relatives of the Celtic female insane, as shown in histories given, certainly is opposed to the usual view. Tribadism was found to be relatively frequent among the Celtic insane females.

TABLE XIV.—HEBEPHRENIA.

Races.	Male.	Female.	Total.
ARYAN.			
Tuetonic.			
German	3	1	4
" -American	3	1	4
" -Austrian.			
" -Swiss.			
Hollanders.			
Anglo-Saxons, American	1	1	2
" " English	4	1	5
" " Scotch	2	0	2
Scandinavian-Danish	2	1	3
" -Swedish	2	1	3
" -Norwegian	2	1	3
Celtic.			
Irish	3	1	4
" -American	6	2	8
" -Canadian	2	1	3
Sclavonic.			
Bohemian	2	1	3
Polish	2	1	3
SCHEMITIC.			
Hebrew-American	2	1	3
NEGRO	1		
Total	37	14	51

TABLE XV.—CHRONIC ALCOHOLIC INSANITY.

Races.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
German	5	2	7
" -American	1	2	3
Hollanders			
Anglo-Saxon, American	4		4
Celtic.			
Irish	6	4	10
" -American	2	3	5
Latin.			
Italian	1		1
Sclavonic.			
Bohemian	1		1
SHEMITIC.			
Hebrew		1	1
NEGRO.			
Total	20	12	32

The table seems to indicate that the Celts retain their intemperate habits in the United States, and the Germans and Latins are becoming addicted to the abuse of alcohol. While not prepared to discuss temperance issues here, it is my opinion that the abuse of the alcohol habit is rather a result of causes external to the individual, which did not act in Germany and Italy.

TABLE XVI.—CHRONIC HYSTERICAL INSANITY.

Races.	Female.
ARYAN.	
Teutonic.	
Anglo-Saxon, American	4
German	4
" -American	4
Scandinavian-Swedish	3
" -Danish	3
Latin.	
Italian	2
Celtic.	
Irish	8
SHEMITIC.	
Hebrew-German	1
" -American	1
NEGRO	1
Total	31

TABLE XVII.—TRAUMATIC INSANITY.

Races.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	1	0	1
German	6	2	8
" -American	1	1	2
Hollanders	1	0	1
Celtic.			
Irish	3	2	5
" -American	3	0	3
Latin.			
Italian	1	0	1
Total	16	5	21

TABLE XVIII.—CLIMACTERIC INSANITY.

Races.	Female.
ARYAN.	
Teutonic.	
Anglo-Saxon, American	1
German	2
Celtic.	
Irish	2
Total	5

TABLE XIX.—EPILEPTIC INSANITY.

	Equivalent.		Pre-Epil.		Post-Epil.		Mixed.		Total.		
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	T.
ARYAN.											
Teutonic.											
German	1	1	1	1	2	3	3	2	7	7	14
" -American	0	0	0	0	3	1	0	0	3	1	4
" -Austrian											
" -Swiss	0	0	0	0	0	0	0	0	0	0	0
Hollanders	1		1		1	1	2	0	5	1	6
Anglo-Saxon, American	1	1	2	3	2	3	3	3	8	10	18
" " English	1		1		1	1	0	1	3	2	5
" " Scotch	0	0	1	0	1	1	1	0	3	2	5
" " Canadian	1	0	0	0	0	0	0	0	0	1	1
Scandinavian-Danish			1	1	1	1	1	1	3	3	6
" -Swedish	1		1			1	1	2	3	2	5
" -Norwegian			1	1	1	1	1	1	3	3	6
Celtic.											
Irish	1	1	1	1	1	1	3	3	6	6	12
" -American	1		1	1	1	1	2	3	5	4	9
" -Canadian						1	1	1	1	2	3
Sclavonic.											
Bohemian			1				1		2	0	2
SHEMITIC.											
Hebrew-German			1			1	2	1	3	2	5
" -American			1		1	1	1	0	3	1	4
NEGRO	1	1	1	1	1	3		4	3	9	12
Total	9	4	15	8	16	21	22	22	61	56	117

The large percentage of epileptics suggests a ready explanation of certain mysterious murders which from time to time startle the community. Many of the female foreign-born epileptics were married and had had children. The

husband understanding fully his wife's disease but being paid to marry did so. In three instances among the Germans such a marriage was a demonstrable medical prescription for the cure of the epilepsy. The comparative frequency of epilepsy among the Scandinavians suggests an explanation of the aberrant religious tendency which shows itself in their frequent acceptances of Mormonism as a creed.

TABLE XX.—PERIODICAL INSANITY.

Races.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American,	2	2	4
" " English	0	1	1
" " Canadian	1	0	1
German	2	3	5
" -American	0	2	2
Scandinavian-Sweden	1	1	2
" Norway	0	2	2
Latin			
French-Canadian	1	0	1
Celtic			
Irish	2	2	4
American	1	3	4
Canadian	0	2	2
Sclavonic			
Bohemian	1	1	2
SHEMITIC			
Hebrew-Polish	2	0	2
NEGRO	1	0	1
Total	14	19	33

TABLE XXI.—CIRCULAR INSANITY.

Races.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	2	1	3
German	3	2	5
Scandinavian-Norwegian	1	2	3
Celtic.			
Irish	3	3	6
Total	9	8	17

TABLE XXII.—COARSE-BRAIN DISEASE DEMENTIA.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	1	2	3
German- "	2	1	3
Swedish- "	2	2	4
Celtic.			
Irish-American	1	2	3
Total	6	7	13

TABLE XXIII.—PRIMARY MENTAL DETERIORATION.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American		2	2
German		1	1
Celtic.			
Irish	3	1	4
Total	3	4	7

TABLE XXIV.—IMBECILITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	1	3	4
" " Canadian	1	0	1
German	1	1	2
Scandinavian-Swede	1		1
Celtic			
Irish	1	1	2
" -American	1	1	2
Latin.			
Italian	1	0	1
Total	7	6	13

TABLE XXV.—IDIOCY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
German	0	2	2
American	1	0	1
Scandinavian-American	2	0	2
Celtic.			
Irish	0	1	1
Total	3	3	6

TABLE XXVI.—PHTHISICAL INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	1	0	1
German	2	2	4
Celtic.			
Irish-American	1	0	1
Sclavonic.			
Bohemian	0	1	1
Total	4	3	7

TABLE XXVII.—TERMINAL DEMENTIA.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	2	8	10
" " English	2	3	5
German	2	6	8
Scandinavian-Norwegian	1	2	3
" -Swedish	1	2	3
Sclavonic.			
Polish	1	0	1
Celtic.			
Irish	2	6	8
" -American	1	2	3
" -Canadian	1	0	1
Latin.			
Italian	1	0	1
SHEMITIC.			
Hebrew, Belgian-Polish	1	2	3
Total	15	31	46

TABLE XXVIII.—SECONDARY CONFUSIONAL INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon American	9	19	28
" " English	1	2	3
" " Scotch	1	1	2
" " Canadian	1	1	2
German	9	14	23
" -American	5	4	9
" -Austrian	2	3	5
" -Swiss	3	4	7
Sandinavian-Swedish	6	8	14
" -Norwegian	2	4	6
" -Danish	1	1	2
Latin.			
Spanish	1	0	1
Italian	2	3	5
French	1	3	4
" -Canadian	1	1	2
" -Swiss	1	1	2
" -Belgian	1	0	1
Celtic.			
Irish	14	27	41
" -American	5	15	20
" -Canadian	1	2	3
" -Welsh	0	1	1
Sclavonic.			
Bohemian	1	3	4
Russian	1	2	3
Polish	1	3	4
SHEMITIC.			
Hebrew-Polish	2	2	4
MONGOLIAN.			
Hungarian	1	3	4
Total	73	127	200

For the reasons already stated the proportion of secondary confusional lunatics is much smaller than it is elsewhere. The fact that the female death-rate up to 1884, exceeded the male explains why the proportion between the sexes is not the same as found elsewhere.

[*To be continued.*]

Periscope.

THE ANATOMY OF THE NERVOUS SYSTEM.

The Posterior Commissure of the Brain. L. DARKSCHEWITSCH, *Neurol. Centralbl.*, No. 5, 1886.

The posterior commissure can be separated into two parts, a ventral and a dorsal part. The fibres in the ventral part can be traced easily in fœtuses, as their calibre is greater than that of the dorsal fibres, and they become medullated at an earlier age. They arise from the upper part of the oculomotor nucleus, and from the posterior longitudinal fasciculus, and curve dorsad around the central gray matter of the aqueduct of Sylvius, thus reaching the posterior commissure. After crossing the median line in this they turn dorsad and end in the pineal gland. The fibres of the dorsal part become medullated so late that it is difficult to separate them from other systems of fibres in the human fœtus. D. has therefore traced them in rabbit's brains.

He finds that they arise from the second layer of white fibres in the corp. quad. ant., that they pass dorsad and cross in the posterior commissure, and then turn cephalad and enter the internal capsule, through which they are continued to the cerebral cortex (what region?) This statement is accompanied by two figures in which the course of the fibres described is supposed to be shown. The continuity of the fibres in the ventral bundle with those of the posterior longitudinal fasciculus is, however, by no means evident. In fact, these fibres appear to terminate in an area of the tegmentum just external to that occupied by the post. longitudinal fasciculus. It is exactly in this area that the bundle of fibres is to be found which Wernicke has described (*Lehrbuch d. Gehirnkr.*, I., 99) as entering the tegmentum from the posterior commissure, and which Spitzka has found atrophied (JOUR. NERV. AND MENT. DIS., April, 1885, p. 221,) in a cat after destruction of the opposite optic thalmus and posterior commissure. The figure, therefore, supports the statements of Wernicke and Spitzka rather than those of the author. It is unfortunate that he seems unaware of the researches here referred to, for the results of the atrophy method are fully as reliable as those of the

embryological method, and conclusions which are not supported by both methods conjointly must be looked upon with distrust.

The Connections of the Inferior Peduncle of the Cerebellum with the Posterior Columns of the Cord. L. DARKSCHEWITSCH and S. FREUD, *Neurol. Centralbl.*, No. 6, 1886.

It is well known that the inferior peduncle of the cerebellum is made up of numerous bundles of fibres collected from different parts of the cord and medulla. The authors of this article describe a cross section of the peduncle as an elliptical figure somewhat bent inward at its extremities and consisting of three parts. There is a central portion divided into a ventral and a dorsal half and a peripheral portion surrounding the two inner halves. The ventral half of the central portion is continuous with the direct cerebellar column of the spinal cord. The dorsal half of the central portion is connected with the nuclei of the posterior column of the cord. The peripheral portion is connected with the opposite olivary body. There is no difference of opinion regarding the first and third of these constituents of the inferior peduncle. Views have been held regarding the connection of the nuclei of the posterior columns of the cord with the cerebellum which are incompatible (See JOUR. NERV. AND MENT. DIS., July, 1884, p. 348, and Jan., 1885, p. 107). The authors have, therefore, reviewed the various opinions and have examined a series of sections through fœtal medullæ with the result of establishing the following facts :

The nucleus cuneatus extends cephalad for a much greater distance than the nucleus gracilis, and in its upper part lies in the dorsal half of the inner portion of the inferior peduncle, forming the "nucleus of the restiform body" of Wernicke. From it fibres enter the inferior peduncle, thus connecting it with the cerebellum. Fibres also issue from it and pass ventrad as fibræ arcuatæ. These cross the middle line in the so-called sensory decussation, but instead of turning cephalad in the interolivary tract they continue ventrad and pass between the lateral border of the pyramid and the olivary body to the surface of the medulla, where they become arciform fibres and, curving around the lateral surface of the medulla dorsad, they enter the inferior peduncle of the cerebellum. The same course is traced for a few fibres from the nucleus gracilis. Thus each nucleus cuneatus and gracilis sends fibres to both inferior peduncles of the cerebellum. This course had been previously described by Edinger, so that these researches are merely confirmatory of his position. The authors also confirm his statements regarding the entrance into the inferior peduncle of short arcuate fibres from the nuclei gracilis and cuneatus. (See JOUR. NERV. AND MENT. DIS., Jan., 1885, p. 108).

On the Effect of Hardening Methods upon the Microscopic Appearances in the Spinal Cord. After a review in *Neurol. Centralbl.*, No. 1, 1886.

Danilo and Popow having recently described certain changes in the appearance of the motor cells of the spinal cord in rabbits and dogs after poisoning with arsenic and phosphorus, F. Kreyssig has repeated the experiments under F. Schultze's direction, and observed the results (*Virchow's Archiv*, Bd. 102). In preparation for the investigation the normal appearances in the cords of these animals were examined, and certain important facts were elicited, which may have a wider application than the author suspects. He found that after hardening in Müller's fluid or any chromic-acid solution the cells of the normal cords presented great varieties of appearance, and of power of absorbing coloring matter. Some cells were colored dark and surrounded by a pericellular space; others were pale and were not so surrounded. Vacuoles were found in the cells, large cells without processes, whose body was apparently deficient in protoplasm were observed, especially in young animals. All these changes which were ascribed by the earlier investigators to effects of the poisons were therefore present in normal cords. It was found, however, that these appearances were more evident in cords which had been transferred from Müller's fluid to 96 % alcohol than those which had been placed in 10 % alcohol and then in solutions of alcohol of increasing strength till the strong solution was reached. The appearances were consequently ascribed to the effects of the strong alcohol upon the cords hardened in the chromate salts. When the proper precautions were taken with the cords of animals poisoned with arsenic and phosphorus no such appearances were found as had been described by Danilo, capillary hemorrhages being the only lesion produced by the poison.

It is not at all impossible that the same precautions would be advisable in hardening human cords, since the appearances found resemble those often observed in human cords hardened in the usual manner. M. A. STARR.

PHYSIOLOGY (INCLUDING EXPERIMENTAL PHYSIOLOGY) OF THE NERVOUS SYSTEM.

The Relation Between the Temperature Changes in the External Auditory Canal and the Circulation of the Brain. *Pflüger's Archiv*, Bd. 38, Heft 3 and 4.

Dr. Istamanoff has made a series of experiments upon the subject. The observations of Dr. Mendel have demonstrated that the temperature in the external auditory canal is about 2° lower than that of the axilla. If now we compare this statement with the results of Dr. Wassiljeff, that the temperature of the external auditory canal gives the temperature of the brain, then it may be assumed that the temperature of the brain is lower or similar to the temperature in the axilla, which is opposed to facts stated by Drs. Heidenheim and Körner, that the brain is the warmest organ in the organism. He went over Dr. Wassiljeff's experiments and found that under the influence of hot applications to the hand of

about + 35° and lower, the temperature rose in the external auditory canal; whilst under the influence of + 8° and lower, the temperature fell in the external auditory canal. Hence the conclusion that under the influence of cold applications to the hand the determination of blood to the brain is diminished, and under the influence of warm applications to the hand it is increased. In a boy who had a defect in his frontal bone he studied the changes of volume in the brain in relation to the temperature of the external ear. For this purpose the changes in the volume of the brain were estimated, and simultaneously the temperature in the external auditory canal was measured. The experiment showed that under the influence of cold applications the volume of the brain increased, and at the same time the temperature in the external auditory canal fell, an opposite result ensued by warm applications. From these experiments it follows that the external auditory canal behaves towards thermic irritations as the peripheral parts of the body, and that the temperature of the peripheral parts by the irritation stands in antagonistic relation to the vessels of the brain. ISAAC OTT.

On the Modification of the Circulation of the Blood in the Brain During Chloroform Narcosis, Accompanied with Painful Excitation. By Drs. ANTONIO CARLE and GUISEPPE MUSSO. *Rivista Clinica, Gennaio*, 1880.

The following conclusions were deduced in the investigation of Drs. Carle and Musso on this subject:

1. Under the influence of the inhalations of chloroform there was a progressive diminution of the tonicity of the vessels of the brain. The circulation of this organ is slowed and there is produced a venous congestion.

2. In the period of complete narcosis and persistent anæsthesia the diminished tonicity of the vessels of the brain and the slowing of the circulation of the blood of this organ, which was initiated during the inhalation, there exists in the place of the above-mentioned congestion in the brain, an arterial anæmia.

3. This last condition of the cerebral circulation is not modified in an appreciable manner with the return of consciousness and sensibility of the individual, whence follows that this is not enough to explain the anæsthetic action of chloroform, and therefore the hypothesis is strengthened that chloroform anæsthesia may be due to a direct action of the chloroform on the nerve centres.

4. Chloroform anæsthesia does not withdraw entirely the nerve centres from the action of the strong peripheric irritation which increases in a transitory manner the tonicity of the vessels of the brain, and increases the circulation of this organ.

GRACE PECKHAM.

Physiological Studies of the Knee-Jerk, and of the Reactions of Muscles under Mechanical and other Ex-

citants. By S. Weir Mitchell and Morris J. Lewis. (*Medical News*, Feb. 13th and 20th, 1886.)

The authors have made an exhaustive study of the knee-jerks in all possible relations. The article is so laden down with details that it will be quite impossible to write a satisfactory review of it. The majority of the facts prove this, that muscular activity in any one part of the body removes a certain amount of inhibition from reflex acts simultaneously excited in any other part of the body. We have no comment to offer, not even on the authors' daring electrical experimentation (strong and but roughly estimated currents passed through the head), but in justice to them will append their own resumé.

The k.-j. varies in health, it may be exhausted by too much use, and may increase from frequent excitation.

All volitional acts, if strong enough, may increase the k.-j. of either leg, and even such small acts as winking, etc., are competent to do so under favoring circumstances.

Weak innervation of the crural nerve increases, and strong innervation of the same, prohibits k.-j.

All sciatic innervation increases k.-j.

Volitional reinforcement lasts for an appreciable time after volition ceases.

Continued violent muscular acts, as of both arms and hands, at last enfeeble the k.-j., and this enfeeblement lasts for an appreciable time.

Passive tension is not essential for the production of k.-j.

Moderate tension mechanically favors it.

Extreme tension destroys it, even in spastic cases, and this is probably mechanical in part, but also, and to a large degree, physiological.

An act of will directed to a part which is functionally inert, or to amputated parts, reinforces k.-j. Hence it is not the muscular motion which is the essential factor.

Strong or weak stimulation of one sciatic in an etherized animal, intensifies the k.-j. of the other leg ; pressure upon the sciatic in man, causing pain and numbness, diminishes the k.-j. of that leg.

Elbow-, ankle-, and jaw-jerks obey the same laws as the k.-j.

One k.-j. does not reinforce the other.

Mere touch has no effect on k.-j. All abrupt impressions, as of pain, heat, or cold, anywhere on the skin, increases k.-j.

Gustation has no effect on k.-j.

Violent optical impressions, in sensitive cases, increases k.-j.

Nitrate of amyl has no effect on k.-j.

Etherization, if profound, abolishes the k.-j. in dogs ; has less effect in rabbits.

All short faradic currents, anywhere, if strong enough to move muscles, increases k.-j.

The wire brush, with faradism on the dry skin, is one of the most effectual of all means of addition to k.-j.

Short galvanic currents, not strong enough to move muscles, give, under certain conditions, marked increase of k.-j.

Galvanism to temples especially, but also to other regions of the head, gives large reinforcements to k.-j.

Making circuit is more effectual than breaking, and these effects soon wear out.

The negative pole to temple gives greater and more constant increase of k.-j. than the positive in same position.

There is more effect on k.-j. from pole to temple than over leg-centres. Effect the same for either k.-j.

Galvanism to temples, with violent synchronous muscle acts, very greatly reinforces k.-j.

Long ascending spinal galvanic currents give good increase of k.-j.; descending, far less.

Moderate constant currents to spine do not reinforce k.-j.

The skin-reflexes (cremaster abdominal) are not reinforced by muscle acts or by pain.

When the belly of a muscle is struck, the resulting contraction obeys all the laws of reinforcement which apply to the k.-j. Tension has upon it much the same influence as on k.-j. One muscle jerk does not reinforce another.

The movement caused by electricity seems incapable of reinforcement.

Tension lessens the effect of even quite strong faradic currents as to pain and motion. B. S.

PATHOLOGY (INCLUDING PATHOLOGICAL ANATOMY) OF NERNOUS SYSTEM.

The Histological Changes in Multiple Sclerosis. By M. KÖPPEN. *Arch. f. Psych.*, xvii., 63.

Disseminated sclerosis appears in various parts of the central nervous system without any apparent law governing its local extent. Charcot's statement that the central cortex is exempt from the process is put in doubt by the case reported by Frommann ; and Erb has shown that Buchwald's assertion that the anterior and lateral columns of the cord are the regions chiefly involved is not based on fact. Schlute has held that the process begins in the brain. Putzar, however, holds that it may commence in the cord. The changes which were at first supposed to be limited to small foci are now known to be at least in some cases diffuse. The basis substance of the focus of disease is spoken of as a semi-fluid gray sticky material, as a structureless interstitial material, and as a fibrillary tissue produced by an increase in neuroglia fibres. How the short neuroglia fibres can change into long connective-tissue fibrillæ is not explained. In the focus cells are described of large size with distinct nuclei, the giant cells of Chvostek—supposed to be collections of protoplasm about nuclei already present, and extending themselves outward in branching processes. Spindle-shaped cells are also described in the basis substance

and also free nuclei and cells lying in the interstices of the tissue, whose origin is obscure. Emigrated blood-globules are always found. All observers note a thickening of the vessel walls. And from these new formations a process of degeneration in the normal tissue is noticed in the ganglion cells and nerve fibres, as evidence of which are found fat drops, fat crystals, corpora amylacea, and nucleated cells. According to Charcot and Schultze, the nerve fibres lose their medullary sheath while the axis cylinder is not destroyed, but is either swollen or atrophied. The preservation of the axis cylinder explains the absence of secondary degenerations. Other observers have, however, disputed this assertion. The majority of authors agree that multiple sclerosis is a chronic inflammatory process, beginning either in the neuroglia or in the vessel walls.

After this review of previous writers the author proceeds to describe three cases which he has examined by aid of the new staining methods of Weigert and French.

The foci were not sharply limited, but the process faded off into the normal tissue imperceptibly, and was therefore diffuse. In the white substance the basis substance consisted of fine fibrils sharply curved, which were evidently thickened interstitial connective tissue. The vessels were greatly thickened, were tortuous, and were distended with blood. Throughout the foci bodies of shining appearance were found, not connected with the neuroglia fibres, but often lying in the interspaces, and about the same diameter as the axis cylinder. There were naked axis cylinders. No true cellular elements were to be found, nor were Deiter's cells at all prominent. On the periphery of the foci nerve fibres with very thick medullary sheaths were noticed. Near the nerve roots long fibrils of neuroglia were observed running parallel with the nerve fibres, and swollen axis cylinders were also found. In the foci in the gray substance the same network of basis substance and the same naked axis cylinders were seen. The ganglion cells were not affected.

In longitudinal sections it was evident that the basis substance consisted of long straight fibrils, and that the shining bodies were swollen axis cylinders. The axis cylinders were thicker and more swollen in the centre of the focus than at its borders. The medullary substance had at points collected in drops, leaving the cylinder bare at parts between. The cylinders were rarely broken. Such a preservation of the axis cylinders, and such swelling of the cylinders in the midst of sclerotic tissue is found only in multiple disseminated sclerosis, and must be considered characteristic of the lesion. In what relation it stands to the slight degree of paralysis present, the intentional tremor, the excessive reflex action and the other symptoms is undetermined.

The Histological Changes in Spinal Sclerosis. BABINSKI. *Arch. de Physiol. Normal et pathologique*, 1885, p. 186.

In an exhaustive article on this subject the author reviews the microscopic appearances found in disseminated sclerosis, and contrasts them sharply with those observed in secondary sclerosis. He finds that in disseminated sclerosis the degeneration resembles that occurring in the central end of a divided nerve. There is an entire disappearance of the myelin sheath in the plaque, but many axis cylinders remain intact, lying free in the sclerotic tissue. The connective tissue is much increased, and is peculiar on account of the tortuous appearance of the fibrillæ, of which the plaque is composed. The thickening of the vessel walls is intense. In secondary sclerosis the degeneration resembles that occurring in the peripheral end of a divided nerve. The myelin sheaths are not destroyed wholly, many nerve fibres with their sheaths remaining. There are no free axis cylinders to be found. The axis cylinder and its sheath perish together or are preserved together. The connective tissue is increased, but does not consist of twisted strands of fibrils. The changes in the vessel walls are slight in degree but constant.

The author advances the opinion that the sclerosis occurring in locomotor ataxia resembles in its histological characters that occurring in disseminated sclerosis more closely than that occurring in secondary degeneration. He has probably been led to this conclusion chiefly by the analogy already mentioned between the changes occurring in the two ends of a divided nerve and the two forms of sclerosis. This analogy, however, is misleading; for according to the law of Wallerian degeneration a division of the central end of a posterior nerve root between the posterior ganglion and the cord is followed by a centripetal degeneration traceable into the cord, similar in character to the degeneration occurring in the peripheral end of a mixed nerve which has been divided in an extremity. The analogy would therefore indicate that the changes in a locomotor ataxia should resemble those in the peripheral portion of the divided nerve, which are similar, as the author shows, to those occurring in secondary degeneration of the cord. No one who is familiar with the appearance of disseminated sclerotic plaques and with the changes in locomotor ataxia could confound the two, as the absence of the twisted and coeild fibrils of connective tissue in the latter lesion is very noticeable. On the other hand a comparison of the sclerosis in locomotor ataxia in the posterior root zone with the sclerosis in the posterior median column (which is admitted by all authorities to be an ascending degeneration secondary to the lesion in the posterior root zone), shows that the histological changes are very closely allied, if not identical. In both there are an increase of connective tissue, a thickening of the vessel walls, and an obliteration of many nerve fibres, the entire fibre, axis cylinder, and myelin sheath being destroyed together; but nowhere are naked axis cylinders preserved and myelin sheaths alone destroyed.

M. A. STARR.

Descending Degeneration in the Crus Cerebri. By G. ROSSOLYMO. *Neurol. Centralbl.*, Nos. 7 and 8, 1886.

It is well known that after a lesion of the central convolutions a descending degeneration can be traced through the middle third of the crus cerebri. The middle third of the crus is therefore known to be the position occupied by the motor tract.

The researches of Brissaud have proven that the inner third of the crus degenerates downward after lesions of the frontal convolutions. In it there pass the tract which joins these convolutions with the nuclei in the pons varolii. Rossolymo now reports two cases of degeneration of the outer third of the crus cerebri. In the first case an embolus in the middle cerebral artery had produced extensive atrophy of the frontal, central and anterior portions of the parietal and temporal convolutions ; the posterior half of the parietal, and temporal lobes and the occipital lobe escaping. The entire crus cerebri was atrophied and degenerated, only a few fibres in its outer third being preserved. The patient had no sensory symptoms, and lived one year after the onset of the disease. In the second case the outer and middle thirds of the crus were degenerated after a lesion of the parietal and temporal lobes.

1. Bechterew has reported a case of total destruction of one hemisphere with degeneration in the entire crus, and another case of destruction of the parietal, temporal and occipital lobes with degeneration of the outer third of the crus.

2. These observations would render probable a connection of the outer third of the crus with the posterior part of the parietal, and with the temporal lobes.

3. Charcot and his school have held with Meynert that sensory impulses are transmitted upward in the outer third of the crus. This view is opposed by the statements of Flechsig and by the facts here recorded, since in this case the outer third of the crus was destroyed without the production of sensory symptoms. These facts also confirm the position advanced by the reporter that the sensory tract lies in the outer half of the tegmentum and does not pass through the crus.—JOUR. NERV. AND MENT. DIS., July, 1884.

M. A. STARR.

A Contribution to the Comparative Study of Convulsions. By J. HUGHLINGS-JACKSON, *Brain*, April, 1886.

Among an interesting series of articles in a remarkably good number of *Brain*, this article of Hughlings-Jackson is the most remarkable. The subject-matter, the manner in which it is presented, the peculiar diction, all are truly * * * Jacksonian ! The author returns to a subject which he discussed most skilfully years ago, and he does this apparently to proclaim a change in his former views. He believed in former years that "no variety of convulsion in man arose from any sort of change below the cerebrum proper" ; but he now holds that *some* convulsions in children depend on lesion of the pons or medulla oblongata. He

refers to "inward fits" (laryngismus stridulus) or "respiratory convulsions," and these constitute the special subject of this paper. To explain *these* convulsions he has written a few pages on convulsions in general, and written in such condensed fashion that it is almost impossible to repeat this part of the paper without reproducing the original nearly verbatim. J. insists on a three-fold scale of fits: 1. Epileptic fits (epilepsy proper) depending on discharging lesions of parts of the highest level of evolution; 2. Epileptiform seizures, depending on discharging lesions of parts of the middle level of evolution (1 and 2 are cerebral convulsions); 3. inward fits (respiratory convulsions) and some other fits depending on discharges beginning in parts of the lowest level of evolution.

Now by the lowest level of evolution (in this anatomico-physiological scheme) is meant the spinal cord, medulla oblongata, and pons Varolii. "The lowest sensori-motor centres represent all parts of the body, animal and organic, 'from nose to feet' in simplest combinations." The middle level of convolution consists of the various motor centres of Ferrier's and sensory centres in the cortex. "The middle sensori-motor centres re-represent all parts of the body, organic and animal, in more complex combinations."

The highest level of evolution includes the frontal or prefrontal lobes, highest motor centres, and the occipital lobes, the highest sensory centres.

The "inward fit" is an instance of a fit on the lowest level. These fits seem to be confined almost exclusively to the period between the sixth and twenty-fourth month. Jackson explains this limitation by age in the following way:

"These inward fits occur in the very young whose lowest level of evolution is the most developed and yet most actively developing, and which from the comparatively little development of higher centres is little controlled." (These speculations are based upon the valuable researches of Soltmann, upon which Meynert also bases his views of the gradual development of the infant's mind.) But why do these "inward fits" occur chiefly in the rickety? "Excessive venosity," as we have all long since known, acts as a stimulant upon the respiratory centre, and "excessive venosity" is favored in various ways in rickety children.

"The ribs being abnormally soft in the rickety, and thus not 'holding out' during the descent of the diaphragm, the efforts of that muscle are more or less neutralized. This favors venosity. Further, the attacks of laryngismus occur chiefly at night, when, from sleep there is still less perfect respiratory action, and thus a condition for still greater venosity * * * too great venosity will stimulate the respiratory centres so overmuch as to produce not a sequence of normal respiratory movements, but that excessive development and contention of them which is convulsion." We cannot refer now to the section of "Universalisation of Convulsion beginning Respiratorily" (Jacksonian English!), and will

only add, in conclusion, that J. shows that the therapeutic measures generally employed, directly or indirectly, help to reduce excessive venosity. The author recommends cod-liver oil, cold sponging, carrying out child in cold weather as constitutional remedies; for treatment of the convulsions proper, he advises the use of musk, belladonna, chloral; and artificial respiration if the respiratory centre should become exhausted after a convulsion. The subject of respiratory fits, experimentally produced, the author promises to discuss in a future paper. B. S.

Case of Cerebral Tumor. By A. HUGHES BENNETT, M.D., F.R.C.P.—the Surgical Treatment by RICKMAN J. GODLEE, M.D., F.R.S.S.—Reprint from vol. 68 of the "Medico-Chirurgical Transactions," London, 1885.

This case, if we mistake not, enjoys the sad distinction of having made the round of the daily press in England and America.

A farmer, æt. 25, applied at the Hospital for Epilepsy and Paralysis Nov. 3, 1884. He complained of paralysis of left hand and arm. Four years ago a piece of timber fell from a house, struck him on the left side of the head, and knocked him down. Loss of consciousness for a few moments only. With the exception of occasional slight headaches, remained in good health for a year. At the end of that time he began to experience a feeling of twitching in the left side of mouth and tongue. This developed into attacks of a paroxysmal character which became more frequent. Some months afterwards he had a "fit," which began with a peculiar feeling in the left side of the face and tongue and turning of the head to the left side. The sensation ran down the entire left side, and culminated in loss of consciousness and general convulsions. These twitchings and "fits" continued for two and a half years.

Six months before admission spasmodic twitchings of left hand and arm without loss of consciousness were observed daily, alternating with the already mentioned twitchings of the face. Shortly afterwards, weakness of left fingers, hand, and forearm, which gradually increased to complete paralysis. Since August, 1884, twitchings set in in the left leg, which usually supervenes upon, and is accompanied by, similar attacks in left arm. The left leg grew weak, and the patient walked a little lame.

On deep and hard pressure there was an area in the parietal region close to the right of the sagittal suture (on a level with a line drawn vertically from the anterior portion of the external meatus), which was more sensitive than the neighborhood. Double optic neuritis, slight immobility of left side of face, distinct on attempted forced movements; tongue, when protruded, pointed slightly to the left. Hearing less acute in right ear. Knee-jerk exaggerated on left side. Patient suffered greatly from lancinating pains in head—such attacks lasting frequently twelve or more hours at a time. Attacks of vomiting not necessarily associated

with headaches. Diagnosis: An encephalic growth, probably of limited size, involving the cortex of the brain, and situated at the middle part of the fissure of Rolando—a diagnosis from which there could have been no escape in the present state of our knowledge. Operative interference determined upon. The manner of trephining is detailed with great accuracy. The tumor was found an eighth to a quarter of an inch below the surface, thinly encapsulated, but perfectly isolated from the surrounding brain substances. The tumor was removed (scraped out, chiefly), the dura mater was drawn together by carbolized silk (!) sutures, drainage-tube inserted, etc. The patient recovered well from operation, and did well for the first 21 days after the operation; at that time septic symptoms set in, to which the patient succumbed a week later. The tumor was a glioma.

For further details and the full report of the autopsy, we must refer the reader to the original. A pity it cerainly is that a surgical accident marred the results which would otherwise have proved brilliant. B. S.

Aphasia and Paresis of both Hypoglossal Nerves Due to a Small Lesion in the Centrum Semiovale. By Dr. LUDWIG EDINGER. *Deutsche méd. Wochenschrift*, April 8, 1886.

The above is a case of unusual interest. A man aged 83, who, in spite of preceding apoplectic attacks, was able to converse well and to swallow his food easily, suddenly found himself unable either to speak or to swallow. He was unable to utter any intelligible sound, although he understood every thing that was said to him. He could move his tongue a little, but could not protrude it beyond the teeth. Solid food he could not swallow. Fluids he managed to swallow under great difficulty only. No other symptoms of any importance. The patient died about 11 days after onset of these symptoms from inanition and pneumonia.

At the autopsy it was shown that these symptoms were due to a small area of softening (the size of half a dime) in the right centrum semiovale The softening was situated immediately above the roof of the ventricle, ½ centimeter to the outside of the caudate nucleus, and about ½ cm. caudate of the anterior angle of convergence of the thalamus and caudate nucleus. On the surface this would correspond to the fissure between the second and third frontal convulutions and ½ cms. in front of the præcentral convolutions.

(The chief interest of this case centres in the fact that it corroborates Wernicke's views with regard to the course of the speech-tract. It is well established that the greater part of the speech-tract starts from the third frontal convolution and reaches the bulbar nuclei. But how it gets there is still a mystery, except that this case proves that it passes through the area occupied by the lesion described above.) B. S.

Post-Hemiplegic Disturbances of Motion. B. GREIDENBERG. *Arch. für Pych.*, xvii., 131.

This article contains the most exhaustive discussion of the various symptoms which develop after a hemiplegia, and is noticeable on account of its masterly summary of the facts recorded by various authors on the subject, and by its complete bibliography. It deserves careful study and suffers in any attempt at condensation.

The first symptom discussed is *post-hemiplegic contractures.* These are classified as (1) such as appear at the time of the apoplexy or follow it immediately. They may be (*a*) temporary, lasting only during the attack ; (*b*) intermittent, in the form of tetanic spasms ; (*c*) stationary, continuing till death ; (*d*) alternating with spasms from time to time. These contractures are due to (1) a laceration of the fibres of the motor tract in their passage through the centrum semiovale or internal capsule, which produces such an irritation in the tract as to cause muscular action. They are very frequent in those cases in which a hemorrhage has torn its way into the lateral ventricles, but it is to the injury to the tract, and not to irritation within the ventricles that this form of contracture, termed apoplectic, is due. These contractures are due (2) to irritation of the cortex in the motor area, such as occur in meningeal apoplexy ; and (3) to injury in the crus or pons, which involves the pyramidal tract. Any lesion of a destructive nature which involves the motor tract may therefore cause an apoplectic contracture.

(2). The second form of contracture is termed primary or early contracture, and appears from two to five days after the apoplexy. In these cases the limbs are flaccid for a time, but then the muscles become rigid, both extremities assuming a position of semiflexion, the flexors prevailing over the extensors. This form of contracture is easily overcome by passive motion, passes off soon as a rule, but occasionally goes on to the secondary contracture. Its cause is supposed to be an irritation of the motor tract produced by the inflammatory process set up by, and in the vicinity of, the initial lesion. This irritation is conveyed to the muscles and results in an increase of normal muscular tone and consequent contracture.

(3). The third form of contracture is secondary or late contracture. This is very common, and is usually permanent. It appears from six to eight weeks after the apoplexy, although occasionally it appears earlier (twenty days). It is not a simple rigidity of the flexors, but affects all the muscles of the limbs, and is not easily overcome by passive motion. As a rule the arm is flexed, the leg extended ; but occasionally the arm is extended, and the hand may then be fixed in various positions. It is rare for the leg to be extremely flexed, and as a rule the leg is affected to a much less degree than is the arm. Contractures of the face, neck, and thorax have been noticed, but are very rare, and occasionally the opposite extremities have been involved, and a para-

plegia has resulted. Such contractures are diminished by rest, by warmth, by sleep, or by bandaging, and are increased by effort, cold, and mental emotion, or mechanical irritation. Although occasionally subsiding under favorable conditions, they are apt to return suddenly, and have therefore been called latent contractures. They are not unfrequently followed by atrophy of the contractured limbs. This form is most fully developed in infantile spastic hemiplegia. Various hypotheses have been offered to explain this form. It is due to the development of secondary degeneration in the pyramidal tract, which is supposed by some to cause an irritation of the fibres in this tract, an irritation which being conveyed to the motor cells of the spinal cord produce the contracture. Continued irritation of a normal cell, however, exhausts the cell and results in a temporary suspension of its action ; it is therefore supposed by others that the degeneration extends to the cell itself and affects its function in such a manner that it responds to all reflex impulses more readily than before, the least excitement leading to excessive muscle tone, and thus to contracture. The contracture is therefore ascribed to continued irritation of the anterior cells of the spinal cord. This is the theory most widely accepted. If these cells are actually destroyed as well as altered in function then an atrophy of their muscles results.

The second symptom discussed is the *increased tendon reflexes*, on the paralyzed side. This symptom is very constant. The statement of Meulen's, that the reflexes are slightly exaggerated during the first month after the attack, are greatly exaggerated from the second to the seventh month, and then gradually return to the normal, has not been confirmed by other observers. On the contrary, a rapid and permanent increase of the reflexes is found to occur in the very large majority of cases. The symptom has been noticed within one hour of the attack, but it usually develops from the seventh to the twenty-first day (Westphal). The theories which are offered to explain this symptom are (1), that the inhibitory influence exerted by the cortex upon the subcortical centres is removed by the rupture of the motor fibres which conduct these impulses (Westphal) ; (2) that the secondary sclerosis in the motor tract produces an irritation of the fibres, and this being transmitted to the cells of the anterior horns of the spinal cord increases their irritability (Charcot) ; (3) that the two factors already mentioned are supplemented by a third, which is the result of the first two, viz., a tension of the muscles and tendons with increased irritability of the peripheral nerve-endings (Ross) ; (4) that the lesion itself produces an irritation which is sent along the motor tract and is communicated to the motor cells of the cord (Schwarz). The latter theory is preferred by the author, who cites in its favor the recent experiments of Adamkiewicz on brain compression. When the cortex was compressed the tendon reflexes increased, but they diminished again to normal when the compression ceased. From this experiment it is evident

that permanent degeneration is not necessary to the production of the symptom, and that temporary irritation is sufficient to cause it.

Associated movements of the paralyzed and unaffected halves of the body are then discussed. Motions of the face as evidence of emotion, such as occur in laughing or crying, are often found to be not only perfectly performed on the paralyzed side, but even excessive. They are only defective where the optic thalmus is outraged (Nothnagel). So too movements of the arms in yawning are usually well executed, although voluntary motion in one arm is imperfect. Frequently, when the unaffected arm is moved, the paralyzed arm makes, or at least begins, a corresponding movement. This is seen chiefly in those cases in which voluntary motion is entirely impossible and in cases of infantile hemiplegia. Westphal has offered in explanation the theory that voluntary impulses starting from one hemisphere of the brain may be transmitted by commissural fibres to the opposite hemisphere, when inhibition is impaired, and thus give rise to these associated movements. There is another form of associated motion which Benedikt considers characteristic of infantile hemiplegia. It is well known that when the flexors are forcibly contracted there is also a slight action of the extensors, which serves to fix the points so that the maximum effect of flexor action is possible. The same is true of many sets of antagonistic muscles. In infantile hemiplegia this normal contraction of antagonistic muscles may be excessive, so that any desired motion is performed with difficulty on account of the internal resistance as it may be called. This is produced by the unbalanced action of antagonistic muscles. Such unbalanced action is not observed when the seat of the lesion is in the motor tract. It is quite characteristic (according to Benedikt) of cortical disease. Charcot and Strümpell, however, regard such movements as reflex in their origin.

Posthemiplegic tremor, resembling that of either paralysis agitans or multiple sclerosis, has been observed, but is rare, occurring in less than five per cent. of the cases. Posthemiplegic chorea is more frequent. The motions are usually more severe and constant in the arm than in the leg, and they increase on voluntary movements. The symptom is often associated with hemi-anæsthesia in the affected limbs. The same is true of hemiataxia, and both symptoms are characteristic of lesions which affect the posterior part of the internal capsule, through which the co-ordinating tracts pass. Posthemiplegic athetosis has been observed in about one hundred and fifty cases since its first mention by Hammond. The athetoid movements are usually slow and regular, but occasionally they may be rapid and jerky. They are usually so uniform as to be called mechanical, and have been likened to those of a machine. The hand-muscles are the ones involved, though the entire arm and the leg may take part in the movements. Hammond's statement that the motion continues during sleep has not been confirmed by other observers. Hemianæsthesia is not associated with athetosis as often as with chorea,

but may be present. While some cases with autopsy support the theory that this symptom is due to lesion of the optic thalamus, others fail to confirm it, and the cause is therefore still undetermined. M. A. STARR.

Paralysis of the "Glottis-Dilators" as the Initial Symptom of Tabes Dorsalis. Prof. A. WEIL, of Heidelberg. *Berl. klin. Wochenschrift*, March 29, 1886.

Prof. Weil gives a full account of a case of tabes dorsalis, which set in with peculiar laryngeal symptoms. The patient is a man, æt. forty-nine, and a pilot on one of the large rivers of Germany. About a year before he consulted W. he had passed through intense excitement from the fact that his boat was in danger of colliding with another. He issued his commands in a very loud tone of voice, and immediately thereafter had an attack of extreme dyspnœa, which lasted about ten minutes with crowing respiration, etc. Eleven months later he had a second attack of the same sort. From that time the least exertion produces a loud snoring respiration. Laryngoscopic examination showed that the margins of the glottis were two to three *mm.* apart during expiration, but during inspiration there was nothing but a cleft-like opening left. Phonation was entirely normal. These symptoms differ from those of ordinary laryngeal crises in several particulars: 1. They are not distinctly paroxysmal; there is a *permanent morbid condition* which is aggravated by physical exertion or mental excitement. 2. There is no cough. In regard to the question whether these symptoms are due to paralysis of the *ab*ductor- or spasm of the *ad*ductor-muscles, the author is not prepared to give a definite answer, but he inclines to the view that there is paralysis of the dilator-muscles. The author gives a list of published cases in which symptoms of this sort were observed. He refers very properly to the necessity of examining for tabes in all cases of sudden paralysis of the vocal cords.

On a Case of Locomotor Ataxia with Laryngeal Crises and one of Primary Sclerosis of the Columns of Goll, Complicated with Ophthalmoplegia Externa. By JAMES ROSS, M.D., LL.D., *Brain*, April, 1886.

Dr. Ross' first case is of greatest interest when compared with the one we reviewed in the preceding paragraph. Although Dr. Ross speaks of "laryngeal crises" the symptoms are remarkably like those which Weil's patient described. Dr. Ross says of his patient, "About two years before admission, the patient began to experience a crowing noise *along with inspiration.* He suffered *almost continuously* from this noisy inspiration; but in addition he had paroxysms of difficulty of breathing, each of which lasted several minutes. * * * he was told that the noise which accompanied breathing was always present in a marked degree when he was asleep."

"A laryngoscopic examination shows that the vocal cords do not attain the normal excursion during respiration, and that the chink of the glottis is much smaller than usual during inspiration."

In Ross' case, the laryngeal symptoms were preceded by gastric disturbances, and were followed by almost all the other symptoms typical of locomotor ataxia. A post-mortem examination was held.

The crura cerebri, pons, medulla oblongata, and spinal cord were examined by the author. All but the cord were studied histologically; the sclerosis had extended upward into the white substance of the slender and cuneate nuclei and the direct cerebellar tracts, which are the direct continuation upwards of the columns of Goll, the posterior root zones and the direct cerebell. tracts of the cord respectively. In addition to other changes, which the reader will find carefully recorded in the original, there was a decided alteration in the character of the descending root of the fifth nerve. The author thinks it probable that disease of this bundle gives rise to the diplopia disorder of movements of the eyeball which are common symptoms of the early stages of tabes.

Dr. Ross' second case is of interest both clinically and pathologically. The patient was a collier, æt. thirty-five; history of syphilis many years ago; well marked double ptosis; eyeballs almost completely immovable: pupils dilated; reactions good; vision impaired; pallor of both discs; loss of knee-jerks; gait uncertain, but not like that of locomotor ataxia.

Post-mortem it was found that the columns of Goll were sclerosed throughout their whole extent in the cord but no decided changes in the upward continuations of the columns. The disease had extended to the external bands up the posterior columns. That the process is unlike that of true locomotor ataxia is shown by the fact that those parts of Burdach's columns which are generally spared were the very ones affected in this case, viz.: the posterior third and the anterior portion adjoining the commissure. Ross thinks that if the patient had lived longer, the disease would have involved the external bands of the posterior columns to a much greater extent and that the later symptoms would have resembled altogether those of ordinary tabes dorsalis. B. S.

Hysteria in Soldiers. Quoted in the *Lancet*, April 10, 1886.

Dr. A. T. Ozeretskovski describes in the *Russian Medical Review*, a whole series of cases of hysteria occurring in soldiers presenting nearly all the phenomena usually associated with this affection, as it occurs in females—dumbness, deafness, deaf-mutism, various disturbances of the vision and of general condition, spasms and paralysis, troubles connected with micturition, joint trouble and elevation of the temperature.

The subsequent history of the cases in most instances left no doubt of the correctness of this diagnosis.

Hereditary predisposition, and in some cases shock were etio-

logical factors. Half of the patients were recruits which tends to show that the sudden change from domestic life and work to the arduous conditions of military service is not without its effect on the nervous condition of the Russian soldier.

The treatment of the hystero-epileptic cases was always unsuccessful, neither water, electricity, nor narcotics appearing to exert any influence over the frequency or severity of the attacks. The cure of the paralytic forms of the disease was successfully accomplished by means of exercise, gymnastics, etc.

GRACE PECKHAM.

MENTAL PATHOLOGY.

Report of a Case of Insanity Following Gunshot Injury to the Head; Cerebral Cyst; Aspiration; Recovery.

By C. F. McDONALD, M.D., Auburn, N. Y. *Amer. Jour. Med. Science*, April, 1886.

Dr. McDonald here gives an excellent report of a most interesting case. Darwin Dingman, æt. twenty-seven, a criminal, was committed to prison for having attempted to shoot his wife. The attempt being unsuccessful, he shot himself in the head with suicidal intent. The ball was removed very soon thereafter. He was sent to the State Reformatory Feb., 1883. In June, 1885, he was committed to the State Asylum for Insane Criminals. The mental disturbance had existed several weeks previously. On admission to the asylum the patient was in a state of violent maniacal excitement; he refused food, and objected to any examination of the head. An examination of the head revealed a nearly circular depression of the skull about half an inch in diameter and about a fourth of an inch deep at the centre. The skull lesion was located, as nearly as could be determined by external measurement, over the right first frontal gyrus at a point corresponding to the junction of its anterior and middle third. The least pressure upon the depression was extremely painful. The mental disturbance was not unnaturally referred to the skull lesion, and an operation was decided upon.

Finding the skull pervious (the opening in the skull was not closed by bony union), the hypodermic syringe was inserted in three several directions—no result; on the fourth insertion, however, the direction of the needle being downward, forward, and outward, clear serous fluid was discovered, and nearly two drachms of this serum were removed. As soon as the effect of the ether had passed off the patient began to converse rationally, and three hours later gave all the circumstances of the shooting, as detailed above. Several months later he is reported as being in excellent health.

Dr. McDonald draws several conclusions from this case: 1—That lesions of the præfrontal lobes are not accompanied by motor or sensory disturbances. 2—That when not in a state of inflammation, the brain substance may be punctured with a fine, clean

needle with comparative immunity from danger or disturbance of function. 3—That the recovery in Dingman's case was directly due to the operation. 4—That the prognosis in cases of traumatic insanity is not necessarily bad if surgical interference be allowed.

B. S.

Phthisis and Insanity. Dr. R. H. BENNER (*Alienist and Neurologist*, April, 1886) says that phthisis manifests itself through the mind by the presence of suspicional delusions and hallucinations and their consequences—irritability, moodiness, unsociability, and extreme selfishness. In the discussion of this paper Dr. J. P. Brown expressed a doubt as to the existence of a specific form of insanity associated with phthisis. Dr. Stearns reported a case in which the mental symptoms disappeared when the physical symptoms became prominent. That phthisis does have peculiar relations to insanity, and does often give a peculiar tinge to the psychosis coexisting with it, has long been accepted as proven by alienists. Dr. Savage ("Insanity," p. 400) sums up the relationship between the two diseases as follows: Phthisis in the insane is associated with certain groups of symptoms, characterized by suspicion and refusal of food on the one hand, and with masking of the physical symptoms on the other. Sanity not uncommonly returns before death in phthisical lunatics, and some recover sanity to die, in a year or two, of phthisis. Van der Kolk ("Mental Diseases") says that insanity and phthisis alternate, and similar observations have been made by Bucknill-Tuke, Ball, Langlois, Neumann, Clouston, Griesinger, and others. As early as 1819 Dr. Jas. S. Macdonald ("Records of the New York Hospital") called attention to the relations between phthisis and insanity, already mentioned in the case of Guiteau's paternal uncle, who died in the Bloomingdale insane hospital in 1819.

Insanity Among Convicts. The report of the Illinois State Penitentiary for 1884, shows, that of two hundred and sixty-nine convicts sent to that institution for murder, sixteen or six per cent. had ultimately to be sent to an insane hospital. The medical officer, is a homœopathic renegade from the regular profession, whose knowledge of medicine may be judged from the fact that he reports a death from "malingering," hence this precentage must be very much under the mark, more especially as the institution is run on a purely politico-commercial basis. So long as a convict is able to work there is no examination of his mental state. How rarely insanity is likely to be detected, is shown by the case of the paranoiac cited in the February number of the JOURNAL, who, despite the fact that he cut his thumb off and then his throat, was not regarded as insane. Even these meagre statistics show that insanity is thirty-two times more frequent among homicidal convicts than it is among Illinois citizens, generally speaking. There were nineteen convicts sent to the insane

hospitals out of a total average population during the last biennial period, of fourteen hundred and twenty-nine, a little less than one and four-tenths per cent. The percentage of insanity in the state is $\frac{5}{100}$ per cent.

Transitory Insanity. By Dr. C. H. HUGHES (*Alienist and Neurologists*, October, 1885,) reports the case of a thirty-three-year-old physician, resident of a Kansas city, who was much overworked professionally, this overwork resulting in insomnia, for which chloroform was taken. He started to Washington with some friends. When twelve hours from home he imagined that he emitted a fœtid odor, and stopped off at Kansas City in consequence. He was brought to St. Louis the following day, by two friends. On the way he manifested persecutory delusions, made an attack on his friends, and was in consequence lodged in jail, where he had a profound night's sleep, followed by recovery the next day. In many features the case resembles those described by Kiernan, (JOURNAL OF NERVOUS AND MENTAL DISEASE, 1880,) Brush (*American Journal of Insanity*, 1882–3,), and Schwartzer (*Die Transitor. Tobsucht*). From a forensic standpoint it is of decided value and interest.

Puerperal Insanity. By Dr. WIGGLESWORTH (*Journal of Mental Science*, April, 1886,) discussed seventy-three cases of puerperal insanity, which came under his observation. Puerperal insanity was looked upon as a genus of which the insanities of pregnancy, parturition, and lactation were the species. Dr. Wigglesworth described ten cases of the first class, thirty-one of the second, and thirty-two of the third. Insanity of pregnancy was the least frequent and least curable of the three varieties, the psychosis developed late rather than early in gestation. The influence of parturition was not immediately beneficial, in some cases having opposite effect; mania was as frequent as melancholia. The second variety—insanity of parturition—occurred most frequently in primiparæ, and in the great majority of cases commenced in the first weeks after parturition; mania was more frequent than melancholia. The precentage of recoveries was high. There was nothing peculiar in the mental symptoms taken by themselves. The thirtd variety—the insanity of lactation—had the highest recovery rate; first lactations were not specially liable to insanity, the psychosis commenced early rather than late in lactation; melancholia was more frequent than mania. Prognosis was least favorable in insanity of pregnancy, and most in that of lactation, as shown in the following percentages:

	PREGNANCY.	PARTURITION.	LACTATION.
Recovery rate	60	74	85.7
Death rate	20	14.8	7.1
Incurable rate	20	11.1	7.1
Average duration of recovered cases	1 year.	8 4-5 mos.	7 1-4 mos.

It will be obvious that the same elements of error exist here as in the researches of Schmidt. J. G. KIERNAN.

On Insanity Alternating with Spasmodic Asthma. By CONOLLY NORMAN, Superintendent of District Asylum, Monaghan, Ireland. Reprint from *Jour. of Ment. Sci.*, April, 1885.

The author reports seven cases in which there was a distinct alternating relation between the mental and pulmonary condition: "Thus in the first case we have chronic asthma vanishing when insanity comes on, and reappearing when the mental trouble becomes chronic. In the second, asthma cuts short and takes the place of an attack of insanity. In the third, perhaps the most remarkable and interesting of the series, habitual asthma disappearing, its place is rapidly taken by insanity, which again disappears immediately on the return of the asthma. When the last change occurred the patient was under close observation in an asylum, so that there can be no doubt as to the sequence of events. It would probably be carrying scepticism too far to say that the cure was due to the action of expectant attention in a patient already convalescent. In the fourth case, chronic asthma occurring in an imbecile, ceases with an acute attack of insanity, and comes on again when the latter has passed off. In the fifth the same order of things is observed as in the third. In the sixth, chronic asthma lessens in severity and finally disappears with the oncome of insanity: when the mental defect becomes chronic with some degree of amelioration, asthma returns."

In spite of the author's assurances most readers will question whether the mental impressions of a diseased physical state be not a very important factor in the case. B. S.

Cataleptic State in Acute Poisoning by Corrosive Sublimate. Reported by Dr. SANTI BIVONA in *Gaz. degli Ospitali, Marzo* 3, 1886.

A woman, aged twenty-four, miscarried in her third pregnancy at three months; a puerperal metritis. A vaginal injection of the mercury bichloride was ordered, and the sister administering it gave it as an enema instead. Half an hour after, the patient became pallid, cold, eyes half opened, unconscious, respiration fifty-six per minute, irregular, and very superficial. The pulse 135, weak, and small; the tremor subsiding, the muscular tonicity was increased, the superior extremity which was experimented with maintained any position in which it was placed for more than a minute. The more easy the position the longer it was maintained. After a while it would return as if it were insensible to the quiet state. The patient was unable to swallow. Each attempt occasioned strangling.

The tremor and the consecutive paralysis are common occurrences in acute mercurial poisoning, but the writer affirms that the cataleptic state above described has never before been noted. He thinks it an individual idiosyncrasy brought about by a neuropathic condition. He excludes from his patient the hysterical condition.

The victim of the poisoning recovered after three hours, during which the most vigorous measures were used to restore her, including antidotes, stimulants, and artificial respiration.

GRACE PECKHAM.

The Insane in Associated Dormitories. It has been claimed by Dr. LAULOR, of Dublin, Ireland (Richmond District Lunatic Hospital Report, 1878), that associated dormitories exercise a decidedly beneficial influence on the insane. Dr. Boyd (*Journal of Mental Science*, vol. xvi.) says that the abolition of solitary cells and the substitution of associated dormitories does away with the prison-like appearance, which has a depressing effect on those mentally afflicted. Dr. G. F. Blandford (*Journal of Mental Science*, vol. xxiii.) says: "The abolition of solitary cells and the association of patients by night and day also conduces greatly to their quiet and well-being." Dr. Wardner (*Alienist and Neurologist*, 1881) says, speaking of his enforced experience caused by a fire: "These patients have been contented, and the number of escapes has not been as many as in former years. Sleeping in such large associate dormitories seems to have had the effect of keeping them quieter, and with the exception of occasional excitement from epileptic attacks there has not been as much disturbance as would have occurred with the same number of sane people lodged in the same room. Those who had been seriously noisy and disturbed their neighbors, while occupying single rooms or small dormitories, out of consideration for others or in consequence of the restraining influence of numbers and the eye of the night attendant, became quiet and acquired the habit of keeping still, and of sleeping well. In fact, a general improvement has been observed in both mental and physical health." Dr. Catlett (Report for 1881) expresses very similar opinions. Dr. Kiernan, from an experience in the Ward's Island Insane Hospital, (*American Journal of Neurology and Psychiatry*, November, 1884), and a very similar experience in the Cook County Insane Hospital, is of the like opinion. Dr. R. S. Dewey, of the Kankakee Insane Hospital, in his last report says that he placed in one dormitory twelve patients who had nearly all been in single rooms before, and who had been addicted to noise, destruction, filthy habits, violence, and several of whom were epileptics. The immediate salutary effect was almost too good to believe; in a word, quiet, cleanliness, and order took the place of turbulence, filth, and destructiveness; and these patients were not only better at night, but remained better all day every day, and some of them dated from this period a steady upward progress, such as had hardly been supposed possible. There are many psychological reasons why such dormitories should stimulate the defective inhibitions of many of the insane.

Melancholia.—Dr. C. H. Hughes (*Alienist and Neurologist*, April, 1886) says that in general the insane hospital is the best

place for the majority of the melancholiacs. The worst mistake that can be made is to send them to an insane hospital within too convenient reach of the patient's family or intimate friends and relatives, where the patient may every few days get a fresh load of grief in the painful familiar faces which go to kindly, but fatally, stab him with their sympathy and keep fresh his mental wounds. . . . When a patient refuses food or medicine it is generally from the promptings of some silent delusion as to its being impure, poisoned, or sinful to take it (and in many instances the refusal and the delusion arise from intestinal anæsthesia secondary to the psychosis). A common and great error in the treatment of this disease is sending the patient off on a long journey without medical attendance, or with a medical man who has no experience in psychiatry. Probably more melancholiacs, sent away in this way for the benefit of their health, die violent deaths by their own hands than recover to return home. J. G. K.

Anomaly of the Pupillary Reflex in Dementia Paralytica.—Morselli (*Gaz. Digli. Ospitalli*, Feb. 21, 1886,) reported a case before the Royal Academy of Turin of an individual who was an insane paralytic, whose pupil dilated when exposed to the light instead of contracting as usual.

Mosso thought that this fact might prove a psychical action.

Reymond said that a trifling brief dilatation of the pupil took place in the ataxic. Also the first effect of esserine was a slight and rapid dilatation of the pupil.

Morselli observed that his case could not be explained by either of these two interpretations. GRACE PECKHAM.

Nymphomania.—Dr. Theophilus Parvin (*Medical Age*, Feb. 10, 1886,) says that in nymphomaniacs "there are generally found a coarse skin, large lips, black eyes, black hair, dark complexion, and a more or less rigid, contracted, and prominent condition of the muscles; there is usually less fat and connective tissue, so that on the whole the body is more angular and less rounded than in the perfect woman. But there are no absolute physical signs of a nymphomaniac. The condition may occur in the blonde, and the passion master the whole woman. There are three stages of nymphomania: the first, where the woman has complete control over her erratic desires, and retires to loneliness to dream over her passions; the second, where she seeks the men and by lascivious looks and gestures, maybe by a partly uncovered person, give evidence of her terrible infirmity; in the third degree the woman becomes truly a maniac and gratifies her desires by seeking connection with men and even with dogs. In some cases this condition is caused by local irritation, vaginitis, or uterine or ovarian disease may be the exciting cause. But in very many cases there is no local trouble." That there is such a thing as a

nymphomania pure and simple may well be doubted. The great majority of so-called nymphomaniacs are hebephreniacs, paroniacs, hypomaniacs, or hysterical lunatics. In no case, other than those of imperative conception or certain periodical lunatics, is nymphomania the only symptom. Dr. Parvin's idea of nymphomania here cited is clearly biased by the description of older authors who ignored the other symptoms of insanity and laid stress on the nymphomania alone. This was at an epoch when the tendency to create monomanias dominated *dilettante alienists.* The description of the personal appearance of the ideal nymphomaniac is taken from some Parisian author. The manner in which the term maniac is used by Dr. Parvin does not denote a very extended acquaintance with psychiatrical terms. Dr. Parvin says that local vaginal application of cocaine has been of value in this symptom, but that moral suasion will be far more efficacious than drugs. The arms of a nymphomaniac may be tied, and she will masturbate by wriggling motions of her legs and thighs ; her arms and legs may be tied and she will accomplish her purpose by rubbing her body against the bed, bureau, or other furniture ; nothing less than death will control this all-devouring passion unless the mind can be impressed. The talk about removing the ovaries and clitoris reminds him of the man who was going to cut off the dog's tail to prevent him from going mad, when some one said that it would be necessary to cut it off *close behind his ears.* There are but few cases which will be benefitted by operative procedure. It is desirable, however, to remove all sources of irritation, and as has been shown by Dr. Goldsmith (*Alienist and Neurologist*, October, 1882), oöphorectomy is of decided value in certain chronic cases with nymphomaniacal symptoms as an aid to moral treatment. In such chronic cases motherhood is contraindicated for the benefit of society and the patient ; hence oöphorectomy cannot be regarded as contraindicated because it unsexes the woman. KIERNAN.

THERAPEUTICS OF THE NERVOUS SYSTEM.

The Treatment of Sciatica and Neuralgic Affections by Methyl Chloride.

Dr. Henry B. Millard, in the *Therapeutic Gazette*, Feb. 15, 1886, describes this method, as Debon, the originator of it, showed it personally to him in Paris. As is known, methyl chloride is a gas, which is rendered liquid by pressure. The liquified gas directed upon the skin, produces immediate whitening and hardness by congelation ; these two effects disappear almost immediately, the skin becoming red and hot. To quote from Debon : " The jet of Methyl Chloride is directed upon the skin of the entire region which is the seat of pain. In sciatica from the sacrum to the malleolus." Patients experience a burning sensation, which is painful, but in no wise comparable to that produced by cauterization by heat, nor is it of long duration. No dressing is necessary.

Practically the effect is the same as that of an extensive, superficial, and rapid cauterization, without vesication or destruction of the epidermis. This treatment is not suited to all forms of neuralgia, (*i. e.* syphilitic), it is most applicable to those having a rheumatic basis. Dr. M. mentions seven cases which he saw treated ; and all were cured, or nearly cured, by from one to three applications. Unfortunately the cost of importing the necessary apparatus to this country would be nearly $275.

Hypnone. Hypnone as an hypnotic does not seem to "pan out" well. It has a horrible taste, is violently irritant, and should be given in gelatin capsules, or pearls : it seems to be unreliable and not free from danger.

Hopeine. It is just as well to know that "Hopeine" has been proved to be a fraud, being only morphine flavored with hops.

Urethane or Carbamate of Ethyl. Dr. HENRI HUCHARD, contributes his experience with Urethane. Translated from *Bulletin, Gen. de. Ther.*, Feb. 15, 1886, by F. R. Campbell, M.D.

The *Urethanes* form a class of ethers derived from carbamic acid, an acid which has been isolated, but which enters into the composition of several salts and ethers. This acid has certain chemical relations with urea, and it is on this account that the carbamic ethers have been called Urethanes. *Urethane*, or ethyl Urethane, in the carbamate of ethyl, or ethylic ether of carbamic acid. Its formula is, $N H_2 1 Co_2 C_2 H_2$ and is found to consist of rhomboidal crystals fusing at 55°, and distilling at 180°. It is very soluble in water, alcohol, and ether. Its taste is quite agreeable,—and resembles somewhat that of nitrate of potash. The new medicine was introduced into therapeutics by Schmideberg, of Strassburg, and has been studied by Jolly, Jacksch of Vienna, and by Riegel & Stecker, of Giessen, who recognized its sedative and hypnotic properties. H. gives the results of Dr. Elroy's experiments on rabbits and guinea-pigs with the drug, the general effect being to produce a kind of cataleptiform sleep.

One rabbit remained in a profound sleep during an entire day, under the influence of 3 gram. of urethane, given subcutaneously. In another, 9 gram. did not produce death.

Huchard, prescribed urethane in fourteen cases, suffering from various degrees of insomnia, and affected with various diseases, including heart disease and angina pectoris. The results are described as highly satisfactory, sleep, "calm, peaceful without dreams, digestion disturbance or headache," comes on in from 10 minutes to one hour, and last 4 to 10 hours. H. prefers 3-4 gram. as a dose instead 1-2 gram., directed by German writers, and gives it mixed with syr. aurant. cort.

One great advantage claimed for Urethane, is that it does not

produce unpleasant symptoms relating to the stomach, heart, or nervous system. It is superior to opium in consumption, an experience confirmed by Jacksch & Stecker: but urethane is greatly inferior to morphine, where insomnia is due to pain, as it has no analgesic power, its advantages may be summed up as follows: feeble toxic powers, great solubility in water, not a disagreeable taste, its easy administration to children, the absence of unpleasant, sequelæ, and the excellent effects produced in phthisis and heart disease.—*Buffalo Med. and Surg. Journal.*

NOTE.—In the *Neurol. Centralblatt*, No. 5, 1886. Dr. Emil Kraepelin reports very favorably upon the action of urethane. His conclusions are based upon 200 doses, distributed among 34 patients. With 1.0 doses treatment was successful in 54%, with 3.0 in 70% of his cases. In the excited state of general paresis he found large doses effective in 60% of the cases; but in a severe maniacal state, he had to resort to paraldehyde. The hypnotic effects of urethane were also exhibited in 77% of the cases of melancholia. (B.S.)

Quebracho in Melancholia. KIERNAN (*Am. Lancet*, Feb., 1886), reasoning from the fact that in melancholia stuporous insanity and other depressed states, the cerebral disease seems to exert an inhibitory action on cardio-motor innervation, causing the pulse to be small, the arterial tone low, and the capillary circulation weak, etc., concluded that quebracho might be of use in such conditions. Guided, also, by its effects in a case, where he gave it for the relief of great dyspnœa, and found that the mental condition was also greatly improved, he tried the drug in ten cases of melancholia accompanied by præcordial pain. The results were decidedly beneficial. He thinks that in melancholia, especially in the atonic types, this drug will prove of decided value.—*The Med. Analectic*, March, 1886.

The Treatment of Recurrent Headache in Children.—Dr. RUSSELL STURGIS (*Boston Med. and Surg. Jour.*) highly recommends ergot for this affection. By recurrent headache he means that form which recurs at intervals of days or weeks, often lasting several years, sometimes accompanied with optical sensations of sparks, bands of color, etc., rarely with nausea. The children who are the subjects of the disease are, as a rule, of nervous temperament, fretful, do not sleep well, and frequently have nocturnal terrors.

Dr. S. thinks the headaches are not identical with true migraine in the adult. Since 1882 he has treated sixteen cases. At first he spent a long time unsuccessfully to relieve the patient with tonic and hygienic treatment, caffeine, and guarana. Finally he employed ergot, giving m. x. of the fluid extract three times daily after meals, continuing the treatment two weeks at least after disappearance of the pain. The longest time required to free a patient from an attack was four weeks. In one case two doses sufficed.

Four typical cases are described. The total number cured is not given.

The Nutritive Value of Some Beef Extracts.—Dr. Thomas J. Mays (*Therap. Gaz.*, March 15, 1886) has undertaken to determine the nutritive value of beef extracts in general, and the relative value of some of those in the market in particular—a work which has long required an experimenter. Whether the particular mode adopted by Dr. Mays can be relied upon to determine a practical question of this kind may be questioned. His experiments, at any rate, are of considerable interest. He takes advantage of the fact that the isolated frog's heart, after being washed out with a saline solution, will beat with the same, but after a while becomes entirely exhausted. If, after it has ceased to beat with such a solution, it be filled with blood or serum, it recommences to beat, and soon does so as forcibly as before. If, instead of blood, the heart were refilled with saline, acid, or even alkaloid solution, it would show no sign of returning vitality. The explanation of this is, the saline solutions are devoid of material with which the heart can perform its functions ; and although it works while it is filled with these solutions, it does so at the expense of the nutritive material stored up in its own meshes, and not with any energy derived from these solutions. Dr. Pohl-Pincus has brought forward evidence to show that there are lacunæ in the walls of the frog's heart in which material is stored up, and as soon as this material is exhausted the heart cannot beat any more unless new material is brought to it. The only substances thus far shown to have the power of restoring an exhausted heart are blood, serum, milk, albumen, and gelatine. Dr. Mays, in his experiments, employed dilute beef-extracts, 1–1,000 (strong dilutions 1 to 100 or 1 to 500 were found to have no effect), and found that they were all capable of resuscitating the heart, and hence were true nutrients. A comparative investigation was then made as to the relative effects of a number of different commercial beef-extracts, and the following products were obtained, which indicate the mean percentage of the number of pulse beats given by each preparation, while that of blood is taken at 100. The figures are only claimed to be approximate.

Liebig's extract of beef	58
Johnston's fluid beef	59
Valentine's meat juice	60
Cibil's extract of beef	61
Sarco-Peptone (Park, Davis, & Co.)	62
Beef peptonoids (Reed & Carnrick)	74
Milk	100
Two-per-cent. solution of dried bullock's blood	100

MORTON PRINCE.

Reviews and Bibliographical Notes.

Allgemeine Diagnostik der Nervenkrankheiten. By Dr. Paul Julius Moebius, of Leipzig. F. C. W. Vogel, Leipzig, 1886.

Of writing books there is no end. So we neurologist might well think. Within the last eighteen months we have been favored with treatises on diseases of the nervous system, by Gowers, Granger, Steward, Ross, Althaus, Strümpell, not to mention a number of other works on some special branch of neurological science. And yet in spite of this vast array, this little book of Moebius (338 pages) subserves an excellent and distinct purpose.

It is evidently intended for the student and general practitioner, rather than for the specialist. Disregarding altogether the diseases of the nervous system as such, the author has set himself the task of giving a clear and concise exposition of the symptoms of these diseases, their anatomical and physiological basis, and of the methods employed in examining for these symptoms.

The book opens with few but pregnant remarks on the family and personal history of patients, on the methods of examining patients for mental and nervous troubles. These remarks are followed by chapters on affections of speech, on disturbances of the motor and sensory apparatus, of the special senses, and in each instance, the best methods of investigation are as fully discussed as are the significance and diagnostic importance of the symptoms themselves. The character of the book is best illustrated by reference to Chapter III., which treats of motor disturbances. Under this head we find a full discussion of paralysis, ataxia, tremor, fibrillary movement, choreic and tonic spasms, forced movements, associated movements, choreiform movements, athetosis, etc.

Under the head of Paralysis, we have first the methods in use for determining the amount of paralysis; then an excellent presentation of the course of motor tracts, differential diagnosis between the various kind of paralysis (cerebral, spinal, and peripheral), and so on.

In short, just the kind of information which a student would expect to find in a book of reference. We have singled out

but a single chapter, but we can assert that every chapter has been prepared with an equal amount of care.

The two appendices are as valuable as the main body of the work. The first contains a list of industries, which are a menace to the health of those engaged in them, and in the second, the anatomy and physiology of each muscle are fully discussed with reference to its action in health and disease.

We have but one fault to find with the author, and this is, that in his eagerness to be "up to date," he has occasionally overstepped the mark. In a book of this sort, facts and well-founded theories should be given and not mere speculations. Lichtheim's diagrams on aphasia are too recent to have stood the test of earnest criticism, and the author would, in our opinion, have done far better to have introduced Wernicke's diagrams, which are, moreover, much simpler than those of Lichtheim. We think, too, that it would have been wiser to have treated of aphasia—a subject as difficult as any in neuropathology—at the end rather than at the very beginning of the book. But, at most, these are simple errors in judgment, and do not detract greatly from the value of Dr. Moebius' work.

The illustrations are truly superb, many of them being reproductions from large atlases, which are inaccessible to the majority of readers. The general typographical work does honor to the publishers. The book merits translation. B. S.

Die Principien der Epilepsie-Behandlung. By Dr. ALBRECHT ERLENMEYER. J. F. Bergman, Wiesbaden, 1886.

This monograph of forty pages, is a reprint of a lecture delivered, Oct. 7, 1845, before the society of physicians, at Coblenz. It is essentially a plea for the conscientious examination and treatment of epileptics, the author insisting that, if care were taken in searching for the etiological factors in each and every case, the results of treatment would be much more satisfactory than they now are.

Among the various questions of interest which the author raises, is the one, whether in cases in which a series of attacks occur at short intervals, and these are then followed by a complete rest for several years, this period of rest in turn, being succeeded by fresh attacks, these fresh attacks may not be looked upon in the light of a primary affection, dependent upon a special cause, and standing in no connection, whatsoever, with the first series of attacks. If this can be answered in the affirmative, then the special etiological factor must be sought, and treatment instituted accordingly.

The authors views in reference to the so-called reflex epilepsies from the nose, throat, or vocal cords, are no doubt correct. He believes that many such cases are not reflex epilepsies at all, but are epileptic attacks due to carbonic acid-poisoning, in consequence of the disease of these parts,—the hypertrophy of the nasal membrane, or the new formation on the vocal cords admitting only of an insufficient supply of air.

Of internal remedies, for relief of those attacks, which cannot be attributed to any specific cause, Erlenmeyer advises the bromides in the form of Brom-water, as recommended by him in another communication. This water is a combination of bromide of potassium, bromide of sodium, and bromide of ammonium in the proportions of, 4 : 4 : 2. together with 750 parts of carbonated soda water, and a drop of ammonia.

All in all, this little book is timely, and, although, it contains little or nothing absolutely new, yet if its precepts were followed by every one, the treatment of epilepsy would soon be rescued from the empirical and unenviable position which it now occupies.

GEO. W. JACOBY.

Schema der Wirkungsweise der Hirnnerven. Ein Lehrmittel für Aerzte und Studirende. Dr. JACOB HEIBERG, Wiesbaden, 1885.

Consists of a table of the cranial nerves, showing their distribution and physiology almost at a glance. The names of the nerves and the description of their distribution are printed in colored inks, red being used for the motor nerves, yellow for the sensory, and blue for the nerves of special sense. Thus, Trigeminus is printed *Tri* in yellow, *gemin* in red, and *us* in blue. This method of instruction, by endeavoring to impress the brain by means of the eye, is one which undoubtedly has its great advantages, and this table of cranial nerves will probably aid the student somewhat in acquiring a knowledge of their distribution and function. For the physician it may be of service to refresh his memory in moments when time is valuable.

G. W. J.

L'Uomo Delinquente (Criminal Man). Br Dr. LOMBROSO, Professor of Legal Medicine at the University of Turin, Turin, Italy. Bocca Bros., 1883.

Criminal anthropology is being much studied elsewhere than in the Anglo-Saxon countries. In the United States and Canada low politics reign supreme in the legislative halls, in the State boards of charities, and science is at a discount. The ward worker, the clerical and medical politician, use institutions—which should be available for scientific purposes—as a means of ensuring the spoils to some political ring. The criminal, the pauper, and the insane are the prey of unclean beasts of politicians, and scientific studies are prevented through an endeavor to conceal malfeasance, by pandering to public prejudice against any thing which seems to disprove the modern doctrine of free-will, and this prevents the scientific study of criminals in America and the other Anglo-Saxon lands.

The present volume is divided into three parts : Criminal Embryology, Criminal Patho-Anatomy and Anthropometry, Criminal Biology and Psychology. Dr. Lombroso opens by a study of crime among animals, among savages and infants, and draws from these studies the conclusion that crime is largely the result of

criminal organization. In many respects his views, startling as they may seem to many, are accepted by the great majority of thinking penologists. The second part opens by the discussion of the findings in three hundred and fifty skulls of criminals, principally robbers. He has found submicrocephaly, exaggerated cephalic indices, especially in brachy cephalic, eurygnathism, enormous orbital cavities, enormously weighty and large lower jaws, high faces, diminution of the facial and cephalo-orbital indices, even less than the diminution observed in the insane ; the cephalo-spinal index is more exaggerated even than in the insane, Pacchionian cavities, oavites, synostoses, cranial and facial asymmetries, plageocephaly, and other signs of degeneracy are found. The brain findings closely resemble the findings in original paranoiacs, there being cases of reversion to the lower animal types as well as aberrant convolutions similar to those found by Wilmarth (JOURNAL OF NERVOUS AND MENTAL DISEASE, March, 1886) in idiots and imbeciles, and by Spitzka and Lander in original paranoiacs. Many of Dr. Lombroso's criminal brains exhibited pathological consequences secondary to the teratological defects found. It is clear from Dr. Lombroso's researches that the opinion expressed by the reviewer (*Chicago Medical Review*, vol. iii., 1881) in a review of Benedikt's "Brains of Criminals," that the connection between paranoiacs, imbeciles, and habitual criminals is a close one, is fully justified. Dr. Lombroso, however, from anthropometric study of 3,839 criminals, comes to the following conclusions : The criminal is always taller and weightier than normal man, even in youth ; his arms are longer ; he has a more ample chest, and he is more hairy. In thieves of all ages, and especially in confirmed criminals, submicrocephaly is double the normal. Dr. Lombroso is of opinion that the born criminal, with whom the present volume deals, is a case of reversion to the primitive races. The present volume is of decided interest and value, albeit Dr. Lombroso is too much biased by the idea that the born criminal is a distinct type from the original paranoiac. The criminal brains and criminals coming under the reviewer's observation were closely analogous to the paranoiacs, and when the existence of the "reasoning lunatics" and "congenital moral imbeciles" is recollected, it is difficult to see how a line can be scientifically drawn. Dr. Lombroso admits there is no difference between the congenital criminals and "moral lunatics," and between these latter and the other types of original paranoia the connection is intimate and close.

When criminal psychology is regarded in Anglo-Saxon and other countries as a branch of psychiatry, results of value to society will be obtained, but until then the present confused methods of dealing with criminals for whose crime their organization is responsible, will continue. The professional ward politician, the medical and clergyman politician will pretend to reform the class of criminals for whose crimes their own war upon society is responsible, and who differ from them only in the fact of having

been found out. With all his enthusiasm one alienist like Dr. Lombroso is of more value to society than the political clergyman, physician, and military men who are at the head of American charitable and correctional institutions. This is saying a good deal when the late revelations about the Pennsylvania soldiers' children's homes, the New Jersey prisons, and the Illinois charitable and correctional institutions are remembered.

Studies of the present type from a popular standpoint are very desirable, and the editor of the *Chicago Daily News*, who instituted a series of discussions involving criminality in its broadest aspect, and thereby acquainted the public with views as to criminality very similar to those advanced by Dr. Lombroso, rendered a service to the State. From present indications Pennsylvania seems most likely to be the first American State to utilize its prisons as sources of anthropological material. Dr. Howard, in Canada, made an attempt at the scientific study of criminality in England. Dr. Nicholson made a scientific study; but these sporadic attempts in the Anglo-Saxon countries have not received the scientific support they deserved. Dr. Nicholson's researches anticipate in many important points the results of Dr. Lombroso. The present work is well issued. J. G. K.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, March 2, 1886.

The Vice-President, LEONARD WEBER, M.D., in the Chair.

Dr. SPITZKA exhibited the brain of a porpoise, demonstrating the absence of the pyramid tract in this animal, and the enormous size of the auditory nerve, which nearly equalled the lumbar cord of the same animal in transverse section. The comparatively small diameter and shortness of the segments of the lumbar cord, as compared with the dorsal segments, was dwelt upon. The brain weighed a few drachms short of forty-five ounces ; the animal (a bottle-nosed dolphin) weighed 286 pounds, and was obtained through Mr. Eugene Blackford.

Dr. SPITZKA then presented the specimens and history of a case of neurogliomatous hypertrophy of the pons oblongata transition. (See p. 193 of this number.)

Dr. E. C. SEGUIN, being requested to open the discussion, said that he thought the paper was one which was hardly open to debate, but he welcomed it as a very valuable addition to the literature of these cases.

Dr. M. A. STARR had observed in the specimens and diagrams presented an apparent implication of the acoustic nerve.

Dr. SPITZKA said this nerve was entirely unaffected, and explained its seeming implication.

Dr. C. L. DANA made a brief preliminary report of a case which he thought would be interesting in connection with lesions in this region. The man, about forty-five years old, was brought into his ward with a history of having been ill a very short time. He was strong and robust-looking. He was found to have static ataxia ; was unable to walk or stand. There was no motor

paralysis in any of the extremities. There was anæsthesia of the temperature sense, of the tactile sense, and of the pain sense on the right side from the shoulder down, involving the right arm, right leg, and right side of the trunk ; the right half of the face was not involved. There was no anæsthesia of touch, temperature, or pain sense on the left side, but in the left hand and arm there was the most marked ataxia which the speaker had ever seen. The arm showed no tremor nor paresis, but any attempt to place it in a particular position would cause it to fly about in a most senseless manner. There were some cerebral symptoms, but the intellect was clear. In a few days there developed some signs of vagus trouble ; there was some difficulty in swallowing, partial paralysis of the vocal cords, inability to speak loud. The patient died after about three days from the entrance of food into the respiratory tract.

At the post-mortem examination there were found a small aneurism of the basilar artery, a recent thrombus of both vertebral arteries ; near the upper aspect of the right half of the floor of the fourth ventricle was a small spot of softening. The latter lesion, it seemed to him, would account for the ataxia of the left arm. He had not yet prepared the brain further than to photograph it.

Stated Meeting, April 6, 1886.

C. L. DANA, M.D., in the chair.

A NEW THERMO-CAUTERY.

Dr. GRAEME M. HAMMOND presented the instrument, consisting of a Bunsen Burner, which, by the action of a spring and clamp, throwing a platinum tip at right angles to the burner, could at once be converted into a thermo-cautery. It had the advantage over the ordinary Paquelin cautery of being always ready for use.

CASE OF MEDIAN NERVE-SUTURING.

Dr. FRASER C. FULLER presented a man who two and a half years ago sustained a glass cut wound of the lower forearm, resulting in complete division of the median nerve and a part of the sublimus flexor. The nerve was sutured with three small strands of cat-gut a few hours after the injury. The tendons were also united. The operation and dressing were absolutely antiseptic, and primary union took place.

At the time of the operation there was complete cutaneous anæsthesia of the region supplied by the nerve. After several months sensation slowly returned, and a year and a half after the operation it was perfect. Power in the small muscles of the affected hand had not entirely returned.

Dr. FULLER referred to another case in which spontaneous recovery took place, operation being refused. There occurred, however, deep necrosis of the finger-tips, and to constant care and energetic electrical treatment only could the eventual recovery be attributed.

Dr. DANA thought the case illustrated very well the result of the experiments of Johnson and Stockholm, that divided nerves, if sutured, healed in about sixty days, but if let alone they would heal in about ninety days.

Dr. B. SACHS then read a paper entitled : "Preliminary Report on a case (with autopsy) of Tubercular Disease of the Spinal Cord." (To be published in this journal.)

After a few introductory remarks the reader gave a full report of the case, of which the following is a short abstract :

The trouble, which was supposed by the patient to be rheumatic, began with pains in the left shoulder, which radiated down into the arm, forearm, and hand ; by degrees the pain became more intense ; for the first it was confined chiefly to the area of distribution of the ulnar nerve, but had gradually spread over the entire dorsal and volar surface of the left arm and hand ; in addition to this hyperæsthesia (and hyperalgesia), to puffiness of the fingers and to a glossy appearance of the skin, there was marked weakness of the grasp in this left hand, with only slight loss of power in the left upper arm and forearm. The condition of the left upper extremity remained unchanged during the entire course of the disease—a period of about two months. At the time of the first examination—five weeks after initial pains—there were no other symptoms discoverable but these ; a slight paresis and some hyperæsthesia of the left leg, exaggerated knee-jerks and ankle clonus on both sides. These symptoms, chiefly unilateral, continued so until the close of the seventh week ; meanwhile the peresis of the left leg had developed into almost complete paralysis ; in the course of another week this unitateral paralysis was transformed into a complete paraplegia of the lower extremities. The motor paralysis also affected the abdominal muscles, and to some extent the respiratory muscles of the thorax, and

the right upper extremity. Incontinence of urine and trophic changes in the skin were superadded. The sensory symptoms amounted to a general hyperæsthesia of the left half of the body below the level of the third rib; this hyperæsthesia was changed toward the end of the disease into anæsthesia, which spread from the left half, and finally involved the right leg, and to a lesser degree the right half of the trunk and the right upper extremity. A tumor pressing upon the left posterior root fibres of one of the lowest cervical segments was thought sufficient to explain the unilateral symptoms; the bilateral symptoms were attributed to a cervico-dorsal myelitis. But there was no clue during life to the nature of the tumor. The autopsy showed a solitary tubercle, situated on the left side, between the sixth and seventh cervical segments, followed by a cervico-dorsal myelitis; there were *very slight* tubercular deposits in the lungs and intestines.

In his remarks on the case the author of the paper attempted to explain the eccentric characters of the sensory symptoms (no anæsthesia on the side opposite the lesion), the exaggerated knee-jerks, and presence of ankle clonus on both sides, and then referred in detail to the behavior of the muscular sense, which was lost on the side of the lesion, and not on the side opposite the lesion. This was in accord with Brown-Séquard's views and opposed to those of Ferrier. In conclusion the reader asked for discussion of the following points: 1—Differential diagnosis between tumor and other forms of spinal-cord disease. 2—Frequency of tubercular affection of the spinal-cord substance. 3—Unilateral symptoms from spinal-cord disease with special reference to disturbances of sensibility, and of the muscular sense in particular.

DISCUSSION.

Dr. M. A. STARR had, by invitation of Dr. Sachs, made an independent examination of the patient three weeks ago. That which caused them to hesitate in making a diagnosis was the difficulty of harmonizing the sensory disturbances with the assumption of an absolutely unilateral lesion in the cord. It had been said that Brown-Séquard had shown pretty conclusively that anæsthesia on one side and hyperæsthesia on the other were due to a unilateral lesion in the cord on the same side on which there was hyperæsthesia. The autopsy in this case showed pretty evidently that

it did not fall in line with those of Brown-Séquard ; that in the early stage, at least, of unilateral cord disease anæsthesia of the opposite side of the body did not always exist. But it was impossible to draw any definite conclusions from a single case, and he had been unwilling to admit this one as being outside the usual line, because he had previously seen one at the Polyclinic, since reported by Dr. Taylor as confirming in all respects the theory of Brown-Séquard. It was true no autopsy was obtained, but the symptoms seemed to be very definite. Dr. Starr thought the whole subject of sensory conduction in the spinal cord was in an unsatisfactory state. There seemed to be no doubt that the muscular sense tract lay in the column of Goll and crossed in the medulla, not in the cord. That view was confirmed by Dr. Sachs' case. The areas of analgesia and hyperalgesia in the later stages of this case were rather irregularly distributed, thus rendering the study of the sensory tracts in the cord extremely difficult. He had been impressed with the great hyperalgesia in the arms of this patient, the slightest touch even of the nails causing great pain. If the sensory tracks crossed just after entering the cord, why was there not anæsthesia on the right side in this case? Three or four cases had been recorded in which there was some reason to believe that the sensations were conveyed upward through lateral tracts in the cord, anterior to and a little outside of the pyramidal tract. Three or four cases had been reported in which there was ascending degeneration in this tract, and he had himself seen one which was not yet reported. If sensations were carried upward in that portion of the cord it would possibly explain some of the peculiarities in Dr. Sachs' case in which that portion chiefly escaped.

Dr. L. PUTZEL said that if Dr. Sachs referred to miliary tuberculosis of the cord he could say from his own experience that it was not of very infrequent occurrence.

Dr. SACHS said he referred to tuberculosis of the spinal-cord substance, and not of the spinal meninges.

Dr. PUTZEL had seen only one case of that kind in which the disease gave rise to a myelitis with all the symptoms of myelitis. Tuberculosis of the cord had not been suspected. The case occurred in a phthisical woman. It seemed to him that the lesion in Dr. Sachs' case was so diffuse that very little could be learned from it regarding localization and transmission of sensory impressions. Concerning Dr. Starr's views as to the muscular sense

being conducted by the columns of Goll, he had seen some years ago a case of meningomyelitis in which there was considerable thickening of the membranes, posteriorly, and such degeneration of the columns of Goll that it could be seen distinctly through the pia mater, yet there was not the slightest evidence of affection of the muscular sense.

Dr. LEO had seen in institutions a number of cases of tuberculosis in patients suffering from locomotor ataxia, especially in old men.

Dr. E. C. SPITZKA reviewed some of the points in the case, and said he thought the fact that tumors in other portions of the cord had not been excluded deprived the case of much determining value as to the views entertained by Brown-Séquard and referred to in the discussion. He had been somewhat surprised at some of the revelations made during the course of the discussion. He had not considered tuberculosis either of the membranes or of the substance of the cord so common an occurrence in old age that any single observer could have seen a large number of cases, and certainly the suggestion made by Dr. Leo was worthy of following up. Regarding the muscular-sense tract, he thought that if a single case were to be accepted as disproving the supposed function of a given part of the cord there was no part which could not be regarded as without function. He remembered that specimens had been presented before this society in which it was claimed that the colums of Goll had undergone slight degeneration when it was found that there was only a slight thickening of the septum from old age.

Dr. PUTZEL remarked that the case which he referred to was also examined by Dr. Welch.

Dr. GRAEME M. HAMMOND said the question which interested him specially was whether exaggerated knee-jerks and ankle clonus necessarily indicated an organic lesion. He had under treatment a gentleman in whom there was exaggerated tendon reflex and very marked ankle clonus in both the upper and lower extremities on the left side. There was no stiffness, pain, atrophy, or other symptom of spinal disease. After a few doses of ergot the ankle clonus and exaggerated tendon reflex disappeared, to return again after quitting the ergot, and disappearing with its renewal. Two or three years ago he exhibited before the American Neurological Association a man cured of locomotor ataxia, and in whom there was entire absence of tendon reflex, yet under

the influence of ergot the tendon reflex returned. We could hardly conclude that ergot cured true sclerosis or any organic disease. The question arose, were exaggerated reflexes and ankle clonus to be regarded as the effect of organic disease of the cord?

Dr. PUTNAM JACOBI inquired as to the relation between increased patellar tendon reflex and functional disease, and referred to a marked case of hysteria in which during a protracted attack she noticed very marked exaggeration of the patellar tendon reflex.

Dr. SPITZKA said that only a few days ago a child which had polio-myelitis, the right lower extremity only being affected, received for several days in succession double the dose of strychnine which he had intended to administer, which had the effect of producing marked exaggerated knee-jerk and ankle clonus, which had previously been entirely absent. He could not understand why ergot should produce the opposite effect, although he did not doubt that it had done so in Dr. Hammond's case.

In taking up the various points raised in the discussion, Dr. SACHS wished to insist again on the importance of differentiating in his case between the symptoms due to the tumor and those due to the subsequent myelitis; the unilateral symptoms alone could be put to the account of the tumor. Addressing himself to Dr. Spitzka, Dr. Sachs said he did not think it was necessary to suppose several lesions in the cord in order to explain the symptoms at the beginning of the case; the symptoms which developed later in the disease were attributed to the myelitis. Of the initial symptoms the only one opposed to Brown-Séquard's views was the absence of anæsthesia on the side of the body opposite the lesion, and the author explained *that* on the supposition that the fibres in the sensory tract were only pushed aside, and not destroyed by the tumor, retaining their conducting power. Explained in this way the case could be made to accord with Brown-Séquard's views. As to the exaggerated knee-jerks and double ankle clonus, it was admitted by almost all that exaggerted knee-jerk might be present in comparatively normal conditions. The existence of ankle clonus without some change in the lateral column was questioned by many, but it was shown by Professor Pitres to exist as early as eight hours after an apoplectic attack, and that therefore in his (Dr. Sachs') case it would not be necessary on account of these symptoms alone to assume multiple lesions. Dr. Sachs agreed with Dr. Spitzka concerning the infre-

quency of tuberculosis of the spinal cord. The best authorities claimed that tuberculosis of the spinal-cord substance was exceedingly rare.

Dr. LEO, in reply to a question, said he did not claim that the tabes dorsalis in the cases which he had seen in old people was due to tuberculosis of the cord ; the existence of the two diseases may have been a mere coincidence.

Dr. G. M. HAMMOND thought that exaggerated tendon reflex or ankle clonus might be due to either a localized congestion or anæmia of the cord, and this would explain why in one case they were relieved by strychnia and in another by ergot.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, January 26, 1886.

(*Continued from page* 192.)

Dr. WHARTON SINKLER then reported a case of "Brain Tumor.

Miss S., aged fifty-two. Mother, maternal grandfather, great-grandfather, two maternal uncles, and two brothers died of phthisis. Paternal ancestors long-lived. No cancer. Healthy and well up to four years old. Then had a serious illness, spoken of by some as "brain fever"; another person says dysentery. Seemed to recover entirely from this, and was bright, well, and agreeable. Was not very intelligent mentally, but not by any means deficient. Catamenia first appeared at eighteen years. At seventeen years friends noticed a change in her; she was irritable, cross, and peculiar about many things. This condition of things continued through the remainder of her life, all the peculiarities becoming intensified. She was a great reader and a great eater. She was fond of literary pursuits; and spent a considerable amount of time in writing and painstaking composition. She never had convulsions as far as known. At twenty-five years she was examined as to her mental condition by Dr. Wm. Kirkbride, and he decided that, although not mentally vigorous, she was not insane.

For five or six years before her death, say forty-five years of age, she seemed to grow stouter, less inclined to work, and in walking seemed to move slowly and with difficulty. For three or four years before her death there was a drooping of the left corner of her mouth, most noticeable in smiling or speaking. This gradually increased. For eight or ten years she has had headaches.

In the summer of 1885 she was at Atlantic City, and had two or three attacks of violent headache associated with unconsciousness. These lasted several days at a time. She came under my charge in September, 1885. She then showed slight left facial paralysis, but no loss of power in arm or leg of either side. She complained sometimes of pain in the head, sometimes in the shoulder or neck. Seldom seemed to have persistent pain in one place. She had an attack of unconsciousness with severe headache. Screamed with pain, but could check herself, and when

asked what was the matter said she had pain, but could not state exactly where it was. At times she talked incoherently, and spoke of having seen persons whom I knew she had never seen. She was unwilling to leave her bed, but if she could be persuaded to get up could walk about. Her appetite was good, but there were indigestion and constipation. No vomiting. Vision seemed good, but the eye-ground was not examined. She was eccentric in her way of talking, and said many things with the evident intention of creating surprise.

At my suggestion she was taken to a country town in the vicinity of Philadelphia, and was there under the care of Dr. J. Reeve. After reaching this place she complained of pain in the right side of the head, and seemed unable to walk. Would fall into a semi-unconscious condition, which would last for some hours. The temperature became elevated. The facial paralysis became more marked, but although no paralysis of the limbs, there was general muscular weakness. The patient died on October 26, 1885.

The post-mortem examination was made by Dr. H. R. Wharton. Brain and cranial cavity; upon exposing the membranes of the brain they were found markedly congested, and the dura mater was very adherent to the petrous portion of the temporal bone on the right side. Brain removed, membranes divided and turned aside. Upon left side there was no apparent lesion. On the right some bulging of the membranes was apparent in the region of the fissure of Sylvius, and upon dissecting them off they were found very adherent to a tumor larger than an English walnut, growing from the fissure of Sylvius about the line of the fissure of Rolando. The tumor was red in color, stood out from the brain tissue, and was dense to the touch. There was some effusion into the ventricles.

Dr. de Schweinitz made a microscopic examination of the growth and sent me the following report:

"In the periphery the growth shows a structure composed of more or less perfectly developed fibrous tissue, more interiorly the stroma of the tumor is made up of numerous, variously sized, dilated blood-vessels, sometimes empty, but for the most part filled with corpuscles. Between these are numerous small round and large spindle cells (sarcoma tissue) scattered through the growth, sometimes singly, often in groups; there are round, yellowish-white bodies, which are probably amyloid in their nature. The tumor may be properly classed as an *angio-sarcoma*."

The growth, as may be seen, is spherical in shape and springs up out of the fissure of Sylvius. The points of interest in the case to me are these. First, the probable long standing of the growth. It is likely that it began at the age of eighteen years, when mental

peculiarities first showed themselves. Secondly, the absence of most of the symptoms peculiar to brain tumor. There were no convulsions, no vomiting, no defects of vision, and no pain localized in one particular spot. The facial paralysis was not noticed until three or four years before death. There were many marked hysterical symptoms which masked the true nature of the disease. These, I think, are often met with in brain tumors in women.

Stated Meeting, February 22, 1886.

S. WEIR MITCHELL, M.D., President, in the chair.

Dr. HARRISON ALLEN read a paper on the headaches which are associated clinically with chronic nasal catarrh.

The reader divided the headaches of chronic catarrh into three kinds: the reflex, the neurotic, and the inflammatory.

THE REFLEX HEADACHE.

The reflex headache is almost entirely restricted to the forehead, the temple, and the vertex. In all varieties of chronic catarrh a dull pain in the region of the forehead is complained of, which may or may not be associated with oppression of spirits and with confusion of ideas. When the disease is confined to that plane of the chamber corresponding to the middle turbinated bone the pain is referred to the temple.[1] The patient will often accompany the account of this pain by a gesture. The index-finger is drawn across the face from the middle of the nose to the temple, and thence, in some instances, to the parietal eminence. The track thus begins at the nose and ends at the temple, or a point beyond. It is not a superficial path, but is referred to a locality lying deep within in the face, and is sometimes described as lying back of the eye. The temporal artery is tense and bounding. An attack of pain, which, in a mild form, may be confined to this region of the face and the temple, will extend, in severe attacks, to the vertex, and even to the nape of the neck. In such phases it is often associated with nausea, and is said to be a "sick headache." Less frequently the pain is referred, in the first place, to the vertex. The slightest exacerbation of the catarrh causes, in some cases, a severe pain at the top of the head

[1] A probe passed in the nose, so as to enter the space defined between the inferior turbinated bone and the outer wall of the nose, often excites a sensation which is referred to the region of the lachrymal sac.

with a subjective impression of heat at the spot, and hyperæsthesia of the scalp.

From the headaches of cerebral disease the headaches of catarrh are sharply separated. The absence of any symptom referrible to cranial sources, the lack of evidence furnished by the history of the case that the complaint is of central origin, the complete control of the condition by local treatment, easily distinguish the affection last named.

Reflex catarrhal headache can be distinguished from "sick headache" of gastric origin by the absence of gastric disturbance, such as the furred tongue; from the temporal pains of eye-strain, by its persistence after the correction of the errors of refraction; and, with less exactness, from neuralgia of the head by exclusion of the rather multiform causes which are found to constitute this condition. Of necessity, it is possible to have a neuralgia of the variety last named or a condition following ciliary strain, as well as dyspepsia, in the same group of symptoms.

Cases of catarrhal headaches which are reflex in nature, are far more numerous than either the neurotic or inflammatory.

Unless the cribriform plate and the upper portions of the lateral masses of the ethmoid bone be seriously involved, either by malignant growths or by necrosis, the meninges of the brain do not suffer in any of the affections to which the nasal chambers are subject. Even the traumatic causes of meningeal inflammation, which may be referred to the nose, are very rarely met with in private practice. It may be broadly asserted that the headaches of nasal disease are not of meningeal origin, but, as a rule, are reflex in nature. A great number of the paths of clinical reflex symptoms are conjectural, and it would be a fruitless task to endeavor to set down, with any show of accuracy, the directions taken by the afferent and efferent nerves in producing, from a peripheral irritation within the nose, a painful sensation in the head. Catarrhal headache is best considered, therefore, entirely from the clinical standpoint. It is a curious circumstance that the reference of a transient pain to the teeth, or to the roof of the mouth, as well as the occurrence of suffusion of the conjunctiva and the flow of tears, which so commonly ensue upon surgical interferences in the nasal chambers, do not often find a place among the conditions complained of in the study of catarrhal headaches, excepting in the neurotic form of the so-called "hay fever." I have sometimes been induced to believe that the head-

achés might be vaso-motor reflexes, and be confined to the great meningeal artery and its branches, but this position cannot be maintained.[1]

Special stress must be laid upon the compression of the anterior end of the middle turbinated bone.

While it must not be supposed that disease of the middle turbinated bone, of necessity, creates a reflex pain in the head, yet the existence of such a pain aids the observer in locating the lesion. All things remaining the same, the presence of a broad middle turbinated bone, the covering membrane of which is in a state of chronic inflammation, and which is tightly wedged between the lateral and the median walls of the nasal chamber, will predispose the subject to attacks of cranial pain.

THE NEUROTIC HEADACHE.

By the neurotic headache, when seen in connection with catarrh, may be understood that state of the system in which a very moderate extent of local nasal or pharyngeal disease excites numerous and inconstant symptoms which cluster about the region of the head and throat. In a typical instance of the kind, the patient—a highly neurotic person—complained of a pain in the throat and ears, in the head, and back of the throat, of a dryness in the roof of the mouth and the nostrils, and of a parched feeling in the eyelids. At another time these inexact symptoms were supplanted by a sensation which was described as "drying the head up." It is evident enough that these complaints cannot be said to constitute a true headache. And yet it is of practical value to remember that the patient was greatly improved by a course of local treatment to the nasal and pharyngeal mucous membrane; at the same time due regard was given to the removal

[1] Reflex headache may have its origin in the pharynx. At least the symptoms disappear upon the relief of the pharyngeal distress. As is well known, pharyngeal disease is often a sequel of nasal disease, and the congested state, which creates the sensation of fulness and pain in the pharynx, may also cause the pain which is referred to the head. A striking example of this complaint was observed by me in the person of a lady, the wife of an army officer, who had spent several years in the high, dry altitudes of the Rocky Mountain range. When first seen, she had just recovered from an attack of neuralgia of the spine. The pharynx was the seat of deep-seated infiltrations which were accompanied by a sense of fulness and a choking sensation. Conversation was sustained with effort, while reading aloud was painful, and in a short time impossible. After the pharyngeal symptoms subsided a disposition to attacks of "sick headache," which she had had for many years, to the surprise of the patient, disappeared. No complaint at any time had been made of the nose, and certainly no evidence of disease in the nasal chambers was apparent.

of the causes which had induced the prostration of the nervous system.

HEADACHES OF INFLAMMATORY ORIGIN.

Catarrhal headaches of inflammatory origin I have never seen, except in acute congestion or inflammation of the frontal sinus. The pain is of high grade, and is, as a rule, confined to one side. In the cases I have studied there has been no elevation of the temperature of the body, and the attacks have subsided after the application of leeches.

Under the heads of *prognosis* and *treatment* little is required to be said. It is evident that if the premises assumed above be correct, that the catarrhal form of headache will continue until the local cause is removed.

All things remaining the same, the patient can be assured that the distress can be relieved by a system of local treatment. The relief is quickly attained when the diseased structures are limited to small patches of the nasal mucous membrane, but is, on the other hand, when the surfaces are extensive, reached only at the end of a long and tedious course of local treatment. I recall, in this connection, a case of a young lady, who came under my care against the advice of others, who thought that, as a result of the serious impairment of the general health, the rest treatment should precede the ordeal of cautery treatment to the nose, yet who advanced steadily in improvement under the treatment, in spite of a headache of the grayest type.

The treatment is in no way modified from that which I have advocated for the treatment of chronic nasal catarrh. The diseased structures must be removed as thoroughly and as rapidly as is consistent with all the facts, and in obedience to the general principles and practice of surgery. In two cases marked relief followed the retention of obturators of vulcanite in the spaces left by perforatin ulcers of the septum.

Dr. Carl Seiler said that we all know that there is more or less headache associated with nasal catarrh, but how far exact lesions give rise to these exact pains had not before been described in so many words. He thinks, with Dr. Allen, that the headache of reflex origin is largely due to pressure upon one of the surfaces of the nasal chambers. It is a curious fact, in his experience at least, that in most cases the headache of catarrh occurs on the left side of the head ; while, on the other hand, obstruction of the right nostril is much more common than that of the left. He

has observed several cases in which the pain was referred to the teeth and to the ear, which, as Dr. Allen has remarked, is very rare. He remembered three cases which came under his notice lately. Two of these patients complained of toothache, or rather of a peculiar pain in the upper jaw, which might be called toothache. After careful inspection by skilful dentists, the teeth were pronounced perfectly sound, and local applications for the relief of the catarrh also relieved the toothache. The third, a lady, complained of intense pain in the left exteral meatus, quite deeply situated. In this case applications to the meatus had no effect, but the pain was entirely relieved by local treatment directed to the nose. In neither of these three cases was headache present.

DR. S. SOLIS-COHEN said that Dr. J. Solis-Cohen had once told him of a book written by a charlatan, who had discovered the connection between nasal catarrh and headache, and had been quite successful in his treatment of such cases. He had not been able to find the reference to this book, but he had come across a reference to a paper in an early number of the *American Journal of Medical Sciences*, vol. v., describing a case of periodical hemicrania relieved by the expulsion of a calculus from the nose. This is referred to only in relation to the history of the subject. As to the thesis itself, his personal experience is not extensive ; but he has seen in the practice of Dr. J. Solis-Cohen a large number of cases in which headache was associated with, and frequently dependent upon, diseased conditions of the nasal passages, and in which relief followed the cure of the local affection. He had not before heard the subject presented in the systematized way in which Dr. Allen had treated it, and consequently was not prepared to discuss his classification.

DR. HARRISON ALLEN said that Dr. Seiler's remark on the connection between earache and chronic nasal catarrh interested him very much. We are not yet in possession of all the facts necessary to explain this connection. He was glad that Dr. Cohen had referred to the case of nasal calculus. The cases recorded by others were not mentioned in his paper, since he lacked the necessary time thoroughly to look up the literature. He believed that the subject of headache which is found associated with chronic nasal catarrh, has never been separately considered, nor the differential diagnosis between it and other forms of headache presented. It is well known that the majority of headaches are much alike. As stated in the paper, the brow, temporal, vertex,

and occiput are so commonly involved in all headaches that patients do not distinguish between the several sources of pain, and physicians themselves may occasionally be so far misled as to fail to determine the exact cause of the ailment. In all obscure cases of distress about the head, the nasal cavities should be carefully examined.

Dr. Charles K. Mills, at the request of Dr. C. P. Henry, of the Insane Department of the Philadelphia Hospital, exhibited "A Case Presenting Cataleptoid Symptoms, the Phenomena of Automatism at Command, and of Imitation Automatism." This patient had been recently admitted to the Hospital, and no previous history had as yet been obtained. He was a middle-aged man, not unintelligent looking, and in fair physical condition. His condition and his symptoms had remained practically the same during the short time that had elapsed since admission. He remained constantly speechless, almost continually in one position, would not open his eyes, or at least, not widely, would not take food unless forced, and his countenance presented a placid, but not stupid or melancholy appearance. He had, on several occasion, assumed dramatic positions, posing and gesticulating. It had been discovered by Dr. Henry that the patient's limbs would remain where they were placed, and that he would obey orders automatically. The case had been regarded as probably one of katatonia, but in the absence of previous history it was not known whether or not he had passed throught the cycle of mania, melancholia, etc., which constitutes this fully developed disease. He had had, since admission, attacks of some severity, probably, from description, hystero-epileptic in character.

Dr. Mills, in exhibiting the patient, first placed his arm, and legs, and body, and head in various positions, where they remained until he was commanded to place them in other positions. His mouth was opened, one eye was opened and the other was shut, and he so remained until ordered to close his mouth and eyes. In most of these experiments the acts performed were accompanied by remarks by Dr. Mills that the patient would do thus and so as he was directed.

Various experiments to show automatism at command were performed. Dr. Mills, for instance, remarked that the gentleman was a good violin-player, when the patient immediately proceeded to imitate a violin-player. In a similar way he took a lead-pencil, which was handed to him, and performed upon it as if it were a

flute. He danced when it was asserted that he was an excellent dancer ; placed his arms in a sparring position, and struck out and countered on telling him that he was a prize-fighter ; went through many of the movements of drilling as a soldier, such as "attention," "facing," "marking time," "marching," etc. He was told that he was preacher and must preach, and immediately began to gesticulate very energetically as if delivering an earnest exhortation. He posed and performed histrionically when told that he was an actor, etc. He was given a glass of water and told that it was good wine, but refused to drink it, motioning it away from him. He was then told that it was very good tea, when he tasted it, evincing signs of pleasure. During all these performances he could not be induced to speak ; his eyes remained closed, or, at least the eyelids drooped so that they were almost entirely closed. He showed a few phenomena of imitation, as keeping time and marching to the sound of the feet of the operator, etc.

After exhibiting these phenomena Dr. Mills made the following remarks :

This patient is undoubtedly suffering from some form of mental disorder. The case is probably one of those which would be classed under the head of katatonia, although in the absence of a past history, I do not think that I am entirely justified in making this diagnosis. In the affection known as katatonia first described by Kahlbaum, and in this country discussed by Hammond, Spitzka, and others, but most ably and fully by Kiernan, alternate periods of mania, melancholia, and, it is said, cataleptoid states are present.

Taking the patient as we find him, I have no doubt several views will be suggested to those present. One of the first thoughts that would suggest itself to any one is that the man is simulating. This idea, I believe, can be dismissed. He is, so far as we have been able to determine, a genuine case of mental disorder, the phenomena which have been exhibited this evening constituting an essential portion of the psychical affection. Many here present, however, are trained in the observance of mental and nervous manifestations, and I would like to hear from them as to the nature of the case.

Taking up the phenomena themselves in detail, let us question ourselves as to their nature. Have we here genuine catalepsy ? What constitutes catalepsy ? What are the pathognomonic symptoms of this ancient but not well-understood affection. I have re-

cently been interested in the subject of catalepsy, and I find some want of clearness in authorities as to its distinctive differential features. Rosenthal and some others would make waxen flexibility the *sine qua non ;* in its absence regarding the case as not one of genuine catalepsy. Waxen flexibility and unconsciousness of surroundings, are the two points upon which most stress is laid by the majority of well-known writers. What is to be understood by waxen flexibility ? I take it that it is a symptom which shows itself in the following way : A patient's leg, or arm, or fingers, his head, or his trunk, on being placed without command, or without remark, by the operator in any special position, will there remain as long as it is possible under ordinary physical laws for it to continue in that position. Such limb or part can be moulded like wax or lead into every possible shape, and will there remain, independently of, or in spite of, commands to the contrary. The true cataleptic patient, according to this conception, is in such a condition as to consciousness that he is not capable for the time being of understanding or of obeying a command. So far as mentality is concerned, he is a genuine "wax figure." This, in my opinion, is a very rare condition. I have sometimes almost doubted its existence. I have certainly seen very few cases which would answer to the picture which I have tried to draw.

Certainly, waxen flexibility, as I have thus described, is not present, or at least, not always present in this patient. It is true that even when I say nothing, his limbs will sometimes remain in the grotesque positions in which they are placed ; but he is not in a strict sense unconscious of what is being done. The very movements of my hand, my appearance, (for these patients do see, although their eyes are partly closed) may in this peculiar frame of mind suggest to him any wishes. I am inclined to think that many of the cases reported as examples of catalepsy are in reality cases which present phenomena analogous to those shown by this man.

These phenomena are those which have for many years been known and described under various names. I well remember, when a boy, attending a serious of exhibitions given by two travelling apostles of animal magnetism, in which many experiments similar to those exhibited this evening were performed upon individuals, selected apparently at haphazard from a promiscuous audience, these persons having first undergone a process of magnetizing or mesmerising. In the experiments of

Heidenhain, of Breslau, upon hypnotized individuals, many similar phenomena were investigated, aud discribed and discussed by this physiologist under the names of "automatism at command," and "imitation automatism." The hypnotized subjects for instance, were made to drink ink supposing it to be wine; to eat potatoes for pears; to thrust the hand into burning lights, etc. They also imitated all manner of movements possible for them to see, or to gain knowledge of by means of hearing, or in any other way. They behaved like imitating automatons, who repeated movements linked with unconscious impressions of sight to hearing, or with other sensory impressions. It was noted in the experiments of Heidenhain, that the subjects improved with repitition. I am inclined to believe that the patient before us performs better to day than he did yesterday, or the day before. His manifestations, although in my opinion, not simulated, have been improved somewhat by practice. Charcot, Richet, and their *confrères*, have made similar observations on hysterical and hypnotized parients, which they discuss under the name of "suggestion."

Hammond (*Med. and Surg. Reporter*, vol. xlv., Dec. 10, 1881), suggests the term "Suggignoskism" from a Greek word, which means "to agree with another person's mind," as a proper descriptive designation for these phenomena. In referring to persons said to be in one of the states of hypnosis, he says that he does not believe that the terms hypnotism and hypnosis are correct, as, according to his view, the hypnotic state is not a condition of artificial somnambulism; the subject, he believes, is in a condition where the mind is capable of being affected by another person, through words or other means of suggesting any thing. In the clinical lecture during which these opinions were expressed, Hammond is reported to have performed on four hypnotized young men experiments similar to those which have been exhibited this evening upon this insane patient. His subjects, however, were not cases of insanity. A bottle was transformed by suggestion into a young lady; sulphur was transmuted into cologne; one of the subjects was bent into all sorts of shapes by a magnet; another was first turned into Col. Ingersoll and then into an orthodox clergyman, etc. In reading such reports, and in witnessing public exhibitions of the kind here alluded to, one often cannot help believing that collusion and simulation enter. Without doubt this is sometimes the case, particularly in public exhibitions for a price. What has been shown here this evening

with this man mentally afflicted ; what has been shown again and again by honest and capable investigators of hypnotism—proves, however, not only the possibility, but the certainty of the genuineness of these phenomena in some cases.

Dr. H. C. Wood did not see that this case is closely allied to catalepsy. He had never seen a case which he considered genuine, thorough catalepsy. He had seen a number of cases occurring in the somnolent state of cerebral syphilis and various other disorders, in which there is a tendency to catalepsy—of course, using the term in its narrow sense. He believed this to be simply a case of automatism at command. He noticed when Dr. Mills raised the hand, that the man moved his hand rather by his own effort than by permitting it to be lifted by the doctor. This shows that the man interprets the muscular movement just as he interprets the command to march. The apparent catalepsy is simply the result of the command. He had seen exactly the same condition in a child two or three years of age under the care of Dr. de Schweinitz. The child could be placed in any position and would stay there almost indefinitely. He could see very little relation between this and true catalepsy. This is a psychical condition, while catalepsy is probably a disease involving lower nerve-centres than are affected in this man.

Dr. Hobart A. Hare said, with reference to Dr. de Schweinitz's case, that it exhibited more of the lead-pipe character than does this one. When the attempt was made to move a limb, it moved in a stiff way. One of the peculiar positions in which the child was placed was to seat it on the floor with the head and feet pointing toward the ceiling. It would remain balanced on the coccyx for some time, until it fell over exhausted.

The President preferred to accept the German definition of catalepsy as given by Dr. Mills. In his lifetime he had seen two cases. One for but a few moments before the condition passed off. The other was most extraordinary. Many years ago he saw a young lady from the West, and was told not to mention a particular subject in her prefence, or very serious results would ensue. He did mention this subject, rather with the desire to see what the result would be. She at once said, "you will see that I am about to die." The breath began to fail, and grow less and less. The heart beat less rapidly, and finally, he could not distinguish the radial pulse, but he could at all times detect the cardiac pulsation with the ear. There was, at last, no visible breathing, al-

though a little was shown by the mirror. She passed into a condition of true catalepsy, and to his great alarm remained in this state a number of days, something short of a week. Throughout the whole of this time she could not take food by the mouth. Things put in the mouth remained there until she suddenly choked and threw them out. She apparently swallowed very little. She had to be nourished by rectal alimentation. She was so remarkably cataleptic that if the pelvis were raised, so that the head and heels remained in contact with the bed, she would retain this position of opisthotonos for some time. He saw her remain supported on the hands and toes, with feet separated some distance, with the face downward for upward of half an hour. She remained as rigid as though made of metal. On one occasion, while she was lying on her back, he raised the arm and disposed of the fingers in various ways. As long as he watched the fingers, they remained in the position in which they had been placed. At the close of half an hour, the hand began to descend by an excessively slow movement, and finally it suddenly gave way and fell. Not long after that she began to come out of the condition and quite rapidly passed into hysterical convulsions, out of which she came apparently well. He was not inclined to repeat the experiment.

Dr. JAMES HENDRIE LLOYD related the following case : A male patient, about twenty-five years of age, presented himself at the Nervous Dispensary, University Hospital, with a history of masturbation, and was then suffering with a consequent sexual hypochondria. His manner and facial expression indicated profound melancholia. He told his story with difficulty, and tended constantly to lapse into silence and brooding introspection. While a relative, who accompanied him, was relating some details of the case, the patient was observed to fasten his eyes steadily on the blank wall and remain in a fixed attitude. On seizing his arms and elevating them above his head, they were found in a condition of true "lead-pipe" flexibility, with prolonged persistence in the positions in which they were passively fixed. At the same time the patient was apparently unconscious, or unmindful, of the experiment ; and even required a loud call, accompanied with a decided nudge, to bring him to himself. The case throws some light, possibly, upon the psychology of these interesting conditions. This patient was no doubt in an exaggerated state of what is usually called "abstraction of mind," which all persons experi-

ence in minor degrees. He was absorbed in his melancholy reflections, and oblivious to peripheral impressions. Subsequent observations failed to discover him in this condition.

Dr. HENRY said that the idea of simulation in this case, as has been stated, can unquestionably be dismissed. Before any conceivable motive could be ascribed the actions were more marked than to-night. Since his admission, five or six days ago, he has quite spontaneously assumed various attitudes—dramatic attitudes, attitudes of prayer, etc. In moving the man's limbs there is a considerable of the "lead-pipe" element brought out.

Another feature of the case is that the man has had peculiar convulsions resembling epilepsy. He, however, has not lost consciousness. By vigorous shaking and calling he could be recalled to himself, but his face is absolutely apathetic, and to all appearances he is unconscious; he also refuses to eat, which agrees with the classical descriptions of katatonia; he has to be fed either artificially, or through the fear of artificial feeding, after the nasal tube has been resorted to once or twice, he will take food without it.

Editorial Notes and Miscellany.

The editor wishes to state that he cannot make special acknowledgment of all books and reprints sent to him. He promises, however, that all publications of interest to neurologists shall be promptly reviewed in the pages of this JOURNAL.

The following named gentlemen were elected officers of the New York Neurological Society for the ensuing year: For President, C. L. Dana, M.D.; for First Vice-President, M. A. Starr, M.D.; for Second Vice-President, B. Sachs, M.D.; for Recording Secretary, G. W. Jacoby, M.D.; for Corresponding Secretary, W. M. Leszynsky, M.D.; for Treasurer, E. C. Harwood, M.D.; for Councillors, L. Weber, M.D., E. C. Seguin, M.D., W. R. Birdsall, M.D., G. M. Hammond M.D., and A. D. Rockwell, M.D.

The Council of the American Neurological Association will hold a meeting on June 16, 1886, to determine upon the time and place for the annual meeting of the Association. We understand that varions members of the Council are in favor of holding the regular meeting at Long Branch, during the month of July.

On the first of September of this year, D. Appleton & Co., will issue a translation of Prof. Strümpell's excellent work on General Medicine which includes a now famous treatise on Nervous Diseases. The translation will be done by Drs. P. C. Knapp and H. F. Vickery, of Boston, and we have reason to think that the translation will be a good one. We hope that that the publishers will make each volume saleable separately and that they will not compel the physician interested in one volume only, to purchase the entire work.

The prize essay of Luciani and Seppilli on the Localization of Cortical Functions has been translated into German by Dr. M. O. Frankel. A review of this book will appear in one of our next numbers.

The first number of the *Neurological Review* edited by Dr. J. S. Jewell of Chicago, has appeared. This number, consisting of sixty-four pages, contains the first instalment of an article by Dr. Jewell, "Clinical Contributions to the Treatment of Epilepsy," and one by Dr. Kiernan on Paranoia. Forty-four pages are devoted to Abstracts, Editorial Notes, and Reviews. This proportion will undoubtedly be changed in later numbers as the editor says that, the present issue falls far below his ideal. The *Review* is neatly printed and has a very attractive title-page. We earnestly hope that Dr. Jewell will attain the object he had in view in establishing the *Review*. There is ample room for two Neurological monthlies in this country and we trust that there will be none but the most generous rivalry between the *Review* and the JOURNAL.

Present prospects are that before long the insane will have attendants as well educated for their special work as are the nurses who serve in hospital wards. The first class of trained attendants was graduated from the Buffalo State Asylum only a few weeks ago. The physicians of that institution deserve great praise for their efforts in behalf of this cause. Two little books which have appeared almost simultaneousiy will aid the physicians in teaching would-be "attendants."

These books are: "How to Care for the Insane," by William D. Granger, M.D. G. P. Putnam's Sons. And "Handbook for the Instruction of Attendants on the Insane." Prepared by a subcommittee of the Medico-Psychological Association, appointed at a branch meeting held in Glasgow, Feb. 21, 1884. Cupples, Upham & Co., Boston, Publishers.

VOL. XIII. JUNE, 1886. No. 6.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

THE PAROCCIPITAL, A NEWLY RECOGNIZED FISSURAL INTEGER.

By BURT G. WILDER, M.D.,
PROFESSOR OF COMPARATIVE ANATOMY, ETC., IN CORNELL UNIVERSITY.

(*With five figures.*)

IT has been well remarked by Ecker (Edes, transl., 50[1]), that "the understanding of the convolutions of the occipital lobe is in itself more difficult than that of all the other lobes."

The present sources of this difficulty are five, relating respectively to: (A) the intrinsic features of the lobe itself; (B) the ordinary mode of removing the brain; (C) the way in which this region is commonly represented; (D) the prevalent belief that the human fissures may be elucidated by means of those of monkeys; (E) the custom of copying figures and accepting interpretations without critical comparison with large numbers of specimens.

To particularize somewhat:

(A) The dorso-caudo-lateral surface of the occipital region presents numerous small or secondary fissures, and even the principal ones are variable in certain respects; the whole

See Bibliography at the close of this article.

lobe has usually the appearance of being crowded into insufficient space.[1]

(B) During extraction of the brain by the ordinary method, the occipital lobe is apt to be cut with the saw, and more or less distorted or torn.

(C) As a whole the lobe is an irregular, three-sided pyramid. The base is the imaginary plane coinciding with the occipital fissure-plane; one of its sides, the mesal, is approximately flat, but the lateral and ventral are irregular, and the former, especially, changes direction constantly, and is, in fact, more caudal than either dorsal or lateral. Hence figures which represent the hemicerebrum in either of the four conventional positions, viz., from the lateral, mesal, dorsal, or ventral aspects, either exclude the group of occipital fissures and gyres altogether, or exhibit them foreshortened and indistinctly. To show them adequately in a photograph, the axis of the camera-tube should nearly coincide with the general direction of the occipital fissure, as in figs. 1 and 3. The case is similar with the frontal region.

(D) The nomenclature of the human occipital gyres (convolutions) introduced by Gratiolet in 1854 and largely followed, even up to the present time, was based, primarily, upon the condition of things in certain monkeys; yet, as Ecker says, in a continuation of the paragraph above quoted, in no part of the cerebral surface is the difference between the human brain and that of these monkeys more marked than in this very region.

(E) The reputation of Ecker, the clearness of his descriptions, and the simplicity of his figures, with the existence of both English and American translations, have caused his statements and views to be accepted, and his diagrams to be generally reproduced, not merely in compilations and clinical reports, but in the papers of original observers. But although, as I hope to show in a subsequent paper on the so-called "ape-fissure," Ecker has clearly explained

[1] May these characteristics be correlated with the fact that the occipital lobe is almost if not quite confined to the Primates, and is, so to speak, a "new thing" in Nature? The superficial smoothness of the monkey's lobe is only apparent, the poma ("operculum occipitale") really involving a very peculiar and considerable complication, which I hope to discuss in a future paper.

(pp. 56–60 and note) some of the distinctions between the human and the simian occipital lobe, yet his interpretation of the morphological relations of the parts immediately surrounding the dorsal end of the occipital fissure, which forms the natural starting-point for the study of this region, is not in accordance with what is indicated by the material examined by me, and not even, as it seems to me, substantiated by his own descriptions and figures.

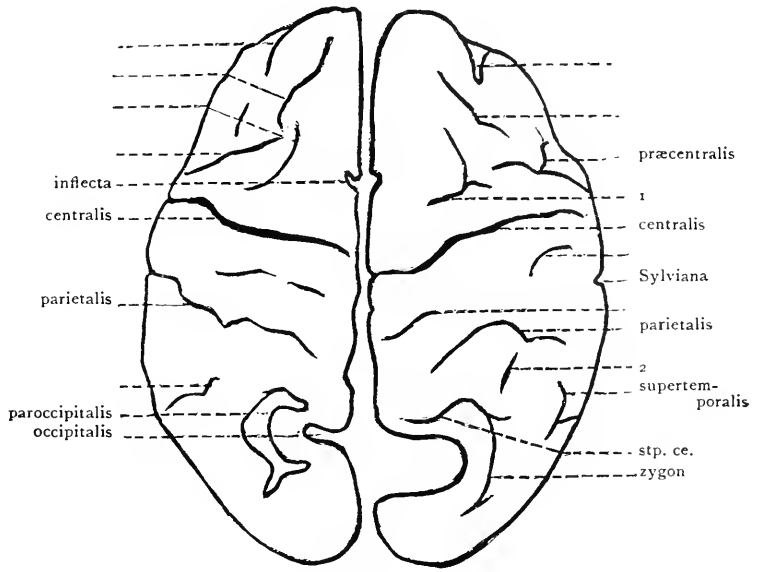


FIG. 1.—Dorso-caudal aspect of a fœtal human cerebrum; No. 734; x 1; drawn by Mrs. Gage.

Abbreviation.—The names designate fissures only, so the F. is omitted. *Zygon* and *stp. ce.* refer to parts of the paroccipital fissure (see fig. 2).

Main point.—The early condition of the paroccipital fissure as a U-shaped depression, opening toward the occipital, and deepest at its middle, with no evidence of a "transverse occipital" at its caudal end more than at its cephalic, and with no approach to the parietal. For the earliest stage see p. 308.

Preparation.—The entire body was injected through an umbilical artery, first with alcohol and then with starch-mass; (Gage, **19**). The head was sawn through at the level of the mouth, and the cerebrum exposed, the base of the cranium being retained for the support of the brain.

Defects.—Unfortunately the size and weight of the fœtus were not recorded. The injection-mass was extravasated at several points, notably so as to distend the occipital fissure on the right side and the paroccipital on the left. The brain was not very well hardened, and the right hemicerebrum is apparently tilted somewhat caudad.

Secondary points.—(*a*) The lack of exact symmetry between most of the fissures of the two sides; (*b*) the interruption of the right parietal, (*c*) and of both precentrals.

Definitions.—The fissural and gyral names employed in this paper are, for the most part, the mononyms proposed by me a year ago (*66*); to avoid misconception they are here enumerated, with some of their more common synonyms; on the figures only the Latin forms are employed but the following are the English paronyms (see my paper *64*).

Gyres.—*Paroccipital* (Fig. 2, *G. paroccipitalis*) occipital; superoccipital; first occipital; first transition convolution; superior annectant gyrus. Respecting the other occipital gyres, I am in doubt as to both their limits and their most appropriate appellations. As stated in 1873 (*11*) and 1882 ("Anatomical Technology," p. 494) I think that the fissures should first be determined.

Fissures.—*Occipital:* occipito-parietal; parieto-occipital; internal perpendicular. *Parietal:* interparietal. *Supertemporal:* parallel; supra-temporal; first temporal. *Central:* Rolandic. *Paroccipital:* the caudal portion of the interparietal with the "transverse occipital." A few self-explanatory names occur on the figures. The following term requires special definition:

Zygal Fissures (*F. zygales*): H-shaped fissures, quadradiate fissures.—A general name proposed for fissures which, like the paroccipital, present a pair of branches at either end of a connecting bar or yoke (zygon). When the earliest condition of the fissure resembles a U, the rami constituting the sides of the U may be called *stipes*, and the others *rami*. To carry out the comparison with letters, the complete or typical condition of a zygal fissure is like two y's joined by their stems, $\overset{Y}{\lambda}$, or, viewed from the side, like an expanded *H*,)—(. The orbital fissure often presents this arrangement (Ecker, Edes, transl., 33).

In Ecker's diagram of the dorsal aspect of the cerebrum (fig. 2) the right parietal ("interparietal") is made to stop nearly opposite the occipital, and there is a heavy line extending across the base of the lobe, a little caudad of the occipital, and wholly distinct from the parietal: this he calls the *sulcus occipitalis transversus*.

It is not necessary to include the figure in this paper, since the diagram has been reproduced substantially in the American translation of Ecker (Edes, transl., fig. 2, p. 19), and in the following commonly accessible works: Charcot (Fowler, transl., fig. 12, p. 53); Ferrier (fig. 65, p. 306); Huguenin (Keller and Duval, transl., fig. 37, p. 45).

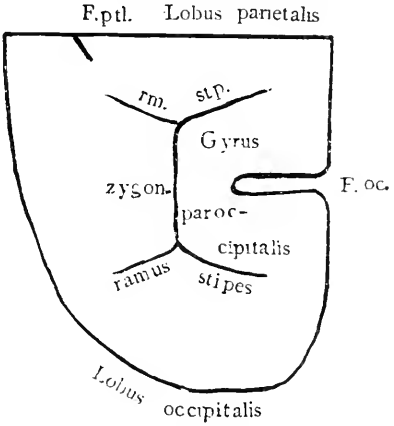


FIG. 2.—Diagram of the paroccipital fissure as a type of the zygal fissures.

FIG. 2.—The outline represents the dorsal (dorso-caudal) aspect of the occipital end of the left human hemicerebrum, including the occipital lobe and part of the parietal, with the dorsal end of the occipital fissure (*F. oc.*) and the caudal end of the parietal fissure (*F. ptl.*). The four-branched line represents the paroccipital fissure, bounding the paroccipital gyre (*Gyrus paroccipitalis*). Primitively, as in fig. 1, the figure is U-shaped, consisting of the occipital and parietal stems or stipes united by the longitudinal bar or zygon; the adult fissure usually presents, in addition, an occipital and parietal branch or ramus as in fig. 3. The parietal ramus often joins the parietal fissure, while the occipital ramus and stipe together constitute the "transverse occipital" fissure of Ecker and others.

Ecker's interpretation of the relations of the parietal and "transverse" fissures obviously depends upon the occasion-

al independence of the latter, and I was led to suppose that the small number of human brains accessible to me at the time of the publication of Ecker's work might be more or less anomalous in the occipital region. Recently, however, I have carefully examined all the brains in the Museum of Cornell University, twenty-nine occipital lobes, and all the original figures of the cerebrum in the best anatomical monographs and manuals. *In no case have I found even an approximation to an independent fissure* in the location of Ecker's "transverse"; on the contrary, in every specimen and in every figure, so far as I can judge,[1] a fissure there situated is joined by what Ecker regards as the extension of the "interparietal."

Since Ecker's reputation precludes the idea that the condition of things on the right of his diagram is altogether imaginary, or even based upon an artistic misconception, it may be supposed either that the brain there figured was exceptional, or, what is more probable, that a very different fissure, fœtal and possibly transitory, has been mistaken for the adult "transverse." Upon this point I hope to present evidence in another paper.

Whatever be the explanation of the difficulty, the "transverse occipital" of Ecker has been almost universally accepted. So far as I know, only three writers have expressed doubts as to its integrality and significance.[2] Clevenger (*21*) says that it "might be considered, and probably is, in many cases, a ramus projected forward [laterad] from the occipital termination of the parietal." Pansch holds (22) that the variability of the "*transversus*" excludes it from the category of primary fissures.

[1] The exquisite photographs in Dalton's recent work (vol. I. pl. 1, 2) represent the occipital region so much foreshortened that the relations of the fissures cannot be determined; the fissure named *transverse* on pl. 2. answers more nearly to Ecker's "occipitalis longitudinalis inferior" (his fig. 1, o $\frac{2}{}$). On the left of pl. 1. the interpreting diagram omits what appears from the photograph to be a perfectly distinct continuation of the two divisions (caudal stipe and ramus) of what, according to Ecker, would be the "transverse."

[2] Edinger reproduces Ecker's diagram in two places (21, 22) with only a comment (23) upon the difficulty of recognizing the "regulation pattern" in the occipital region:

"Dieser Occipitallappen ist aussen nicht an allen Gehirnen so gleichmässig gefurcht, dass man immer die von den Autoren angegebene erste (obere), zweite (mittlere) und dritte (untere) occipitalwindung leicht und ohne Künstelei wieder finden könnte."

The most serious opposition to its acceptance is in the following passage from Wernicke's paper (321), which did not come into my hands until after my conclusions had been formed: "Das vorkommen einer durch besondere constanz oder tiefe ausgezeichneten queren Furche (*sulcus occipitalis transversus*) kann ich nach meinen Befunden am erwachsenen Gehirne nicht bestätigen."

There may be room for discussion respecting the propriety of accepting as an integer a fissure which, like the postsylvian of the cat and the postcentral of man, is only occasionally independent; but surely we are not called upon to accept without question the integrality of a supposed fissure which, like Ecker's "transverse occipital," is independent upon only one side of his own diagram, and which, apparently, no one else has ever found in that condition.

My first conclusion is, then, that *what is commonly understood as the transverse occipital of Ecker is not a fissural integer, and that the name and its synonyms should be abandoned.*

The second question is as to the relations of the longitudinal zygon (fig. 2) to the parietal. Ecker's view is indicated in all his figures and specifically stated in the following passages (pp. 58, 38):

> "In the fœtus the two portions of the fissure, the posterior (*occipitalis superior*) and anterior (*interparietalis*) [real parietal] arise separately from each other and subsequently unite. The former is nothing but an extension [*fortgesetst*, rendered *convolution* by obvious oversight in the American translation] of the latter."

On page 38, it is admitted that the fissure is less distinct ("manchmal weniger deutlich"), because often interrupted, and this more frequently on the right side. Nevertheless, on both sides of his diagram (fig. 2), the fissure is made continuous, and his view seems to have been generally accepted.

After a careful study of all the specimens and figures obtainable, I am led to conclude that this view is erroneous; that the true parietal and the "superior occipital" do not form parts of one fissure, and that the latter is the prin-

cipal and primary constituent — zygon — of a paroccipital fissure.

The evidence is threefold: (1) as admitted by Ecker, the zygon always appears independently in the fœtus: (2) as also admitted, it often remains separate in the adult; (3) when the union does occur, in all the cases examined with reference to this point, excepting one, the combined fissure is shallower at the presumed place of junction, and deeper at or near the middle of the two constituents— the true parietal and the paroccipital zygon.

In more detail, the facts are as follows:

1. The earliest trace of the paroccipital appears upon a fœtal cerebrum (No. 1820, M. C. U.), which, as preserved in alcohol, with slight distortion, measures 61 mm. in length. The central, Sylvian, occipital, and calcarine are well-marked; the parietal is a slight depression, directed obliquely. A more distinct longitudinal depression, perfectly distinct from the parietal and opposite the occipital, is undoubtedly the commencement of the paroccipital zygon. I regret that the age and size of the fœtus are not known.

The immediately succeeding stages have not been observed, but in fig. 1 and in Bischoff's paper (**12**, figs. 10, 12) the fissure presents itself as an irregular crescent, consisting of the zygon and the cephalic and caudal stipes, with traces of the corresponding rami.

2. As Ecker has frankly stated, the true parietal often remains separate from its supposed continuation upon the occipital lobe.

Among the 29 hemicerebrums, representing 25 individuals, in the museum of Cornell University, I find the separation complete in 13 and a junction in 16.[1] If to these be added the two shown in Bischoff's evidently accurate fig. 1, and the 12 carefully measured by Jensen, we have 43 cases, of which 22 are separate and 21 united.[2]

[1] So far as these cases go, there is striking confirmation of Ecker's remark, that the interruption appears to happen more often on the right side (p. 38). The fissures are independent on 9 right sides and only 4 left; the junction occurs on 10 left and 6 right. I neglected to note this point on Jensen's figures.

[2] It seemed best to exclude from this tabulation such of my specimens as are imperfect or poorly preserved so as to be doubtful, and also most published figures, which seldom represent the occipital region with distinctness.

So far then as reliable evidence is attainable by me at the present time, it appears that Ecker's "interparietal" is interrupted as often as it is continuous.

On page 38, Ecker endeavors to diminish the force of what would be commonly regarded as evidence adverse to his view, by affirming that the interruption of his "long" parietal occurs no more frequently than in, for example, the temporal fissures. Even if this be true, it is by no means certain that the temporal fissures are the integers they are ordinarily admitted to be; they certainly, like all other fissures, need monographic treatment.

It has been shown above that the caudal part of Ecker's interparietal is constantly connected with what he regards as a "transverse." In the majority of cases this transverse is not straight, but deflected caudad at both ends, so as to constitute a bifurcation of the zygon. In like manner, in most cases the cephalic end forks, one branch (stipe) extending meso-cephalad and remaining free, while the other (ramus), which extends latero-cephalad, often joins or is joined by the true parietal.

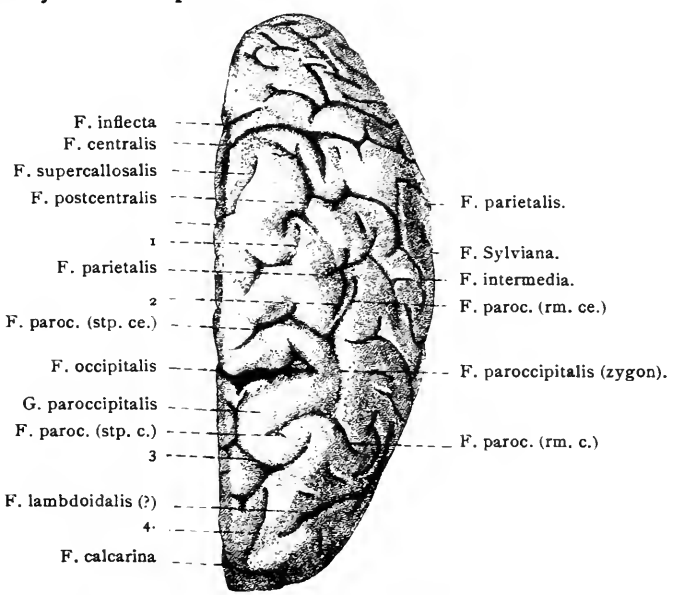


FIG. 3.—Dorso-caudal aspect of the right hemicerebrum of a child at birth, No. 478; x 1; drawn by Mrs. Gage.

Main point.—The relations of an independent paroccipital fissure to the occipital and parietal fissures. See diagram, fig. 2.

Abbreviations.—*F.*, fissure; *G.*, gyrus; *F., paroc.*, paroccipital fissure; *stp. ce., stp. c.*, the cephalic and caudal stipes of the paroccipital fissure; *rm. ce., rm. c.*, its cephalic and caudal rami. *1*, a separate fissure which may represent part of the postcentral; *2*, a separate fissure, which probably represents part of the parietal, but which joins neither it nor the cephalic ramus of the paroccipital; *3*, *4*, unidentified fissures.

Preparation.—The brain was hemisected while fresh, and the hemicerebrum hardened in alcohol while resting on the mesal surface; hence it is thinner than it should be. It was photographed while supported so that the axis of the camera coincided nearly with the direction of the occipital fissure. The mesal surface is partly seen, much foreshortened.

Secondary points.—(*a*) The presence of the inflected fissure; (*b*) the extension of the central fissure, so as to appear upon the meson; (*c*) the extension of the supercallosal, so as to appear on the dorsum; (*d*) the extension of the calcarine (dorsal fork), so as to appear not only upon the dorso-caudal aspect, but also in a lateral view of the brain (perhaps most of what shows is another fissure which is joined by the calcarine); (*e*) the presence of a fissure, independent of the occipital and paroccipital, which may be the one figured in the fœtus by Bischoff (figs. 7, 8, 9).

When the junction does not occur, the parts present more or less closely the appearance shown in fig. 3.

This simple, perfect condition of the paroccipital is represented on the right side of the orang's brain by Spurzheim (Pl. V.). In Bischoff's orang (fig. 26), the cephalic ramus is joined to the parietal.

If, as I believe, the zygon is the principal, central, and primary constituent of a fissural integer, the paroccipital, it would be expected to be deeper at or near its middle than at its ends, or than the stipes and rami. This is the case in the few brains which I have examined with reference to the point, and is exemplified in fig. 4, but numerous observations are desirable.

3. It is a generally accepted rule that a true fissural integer is usually deepest at or near the middle of its length, coinciding approximately with the place of its first appearance in the fœtus; as a corollary, it is also generally believed that any marked or frequent shallowing in the course of a supposed integer furnishes ground for inquiry into the facts

of adult condition and development. It has already been seen that, so far as known, the true parietal and the paroccipital always commence independently, and the natural inference from this fact is further supported by what has been ascertained respecting the relative depth of the continuous fissure so formed.

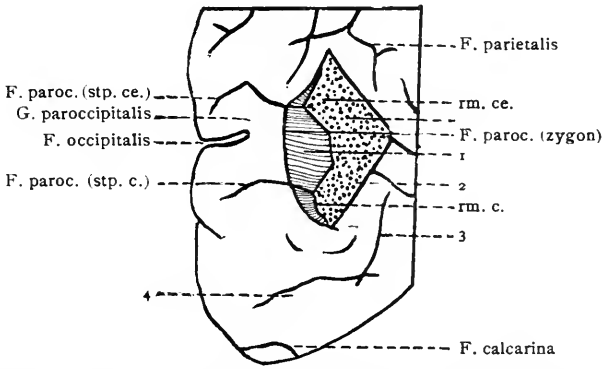


FIG. 4.—Diagram of the partly dissected paroccipital region of the right hemicerebrum (of a child at birth) represented in fig. 3; No. 478; x 1.

Main point.—The greater depth of the paroccipital fissure at the middle of the zygon than at its ends or in the rami.

Abbreviations.—*F. paroc.*, paroccipital fissure; *stp. ce.*, *stp. c*, its cephalic and caudal stipes; *rm. ce.*, *rm. c.*, its cephalic and caudal rami; 1, shaded with lines, the lateral surface of the paroccipital gyre; 2, shaded with dots, the cut surface left by removing the gyre just laterad of the paroccipital; *3*, *4*, unidentified fissures.

Preparation.—The preparation of the entire hemicerebrum is stated under fig. 3. For this figure the gyre immediately laterad of the paroccipital f. was removed by two incisions meeting at a point opposite the occipital. The preparation was photographed while tilted so as to show the lateral aspect of the paroccipital gyre more completely; hence the undulations of the dorsimesal outline.

In the few brains whose elasticity permitted the divarication of the gyres bordering the fissure in question, there appears to be a shallowing at or near the place of the presumed junction of the two fœtal fissures. With one right hemicerebrum (No. 376)—whose platetrope (fellow of the opposite side) by the way presented a total interruption, as in fig. 3—the lateral gyres were removed so as to expose the depth of the supposed single fissure throughout its en-

tire length. At the middle of the true parietal the depth is 12 mm., at the middle of the paroccipital zygon 16 mm., but at the middle of their combined length, where the depth should be greatest if it were a true integer, it is only 10 mm.

Facts of the same kind are supplied by the careful observations of Jensen, which are the more valuable in this connection, in that he was apparently inclined to regard the independence of the caudal division (my paroccipital) as abnormal. Jensen "sounded" the depth of the fissures of six brains. In the five hemicerebrums in which there was a continuity of the parietal and the paroccipital, his figures indicate a shallowing at the place of presumed junction.

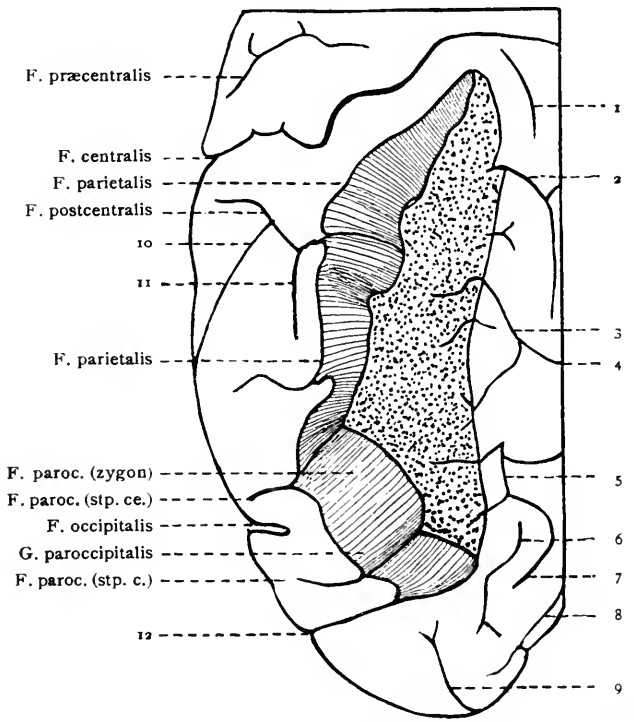


FIG. 5.—Diagram of the partly dissected paroccipital region of the right hemicerebrum of an adult man; No. 376; x 1.

Main point.—The shallowing of the parietal fissure at its junction with the paroccipital.

Abbreviations.—*F. paroc.*, paroccipital ; *stp. ce.*, *stp. c.*, its cephalic and caudal stipes ; *1*, a short, lunate fissure, which nearly joins the central ; *2*, (the line should join the short fissure which extends latero-cephalad) the Sylvian f.; *3*, *4*, extensions of the supertemporal f.; *5*, *6*, *7*, *8*, unidentified fissures ; *10*, *11*, caudal branches of the postcentral f.; *12*, an unidentified fissure which nearly reaches the dorsimesal margin of the hemicerebrum. As in fig. 4, the cut surface is indicated by dots, and the exposed sides of the gyres by vertical lines.

Preparation.—The gyres immediately laterad of the parietal and paroccipital fissures were removed by oblique incisions so as to expose the full depth of the fissures. The brain is a small one, weighing only 1115 grams when fresh, including the dura ; it was from an Irish hospital patient, thirty-seven years old.

Defects.—To avoid complication there has been omitted from the figure a fissure which extends latero-cephalad from the occipital ; on this and some other accounts the left hemicerebrum would have been preferable, but on that the paroccipital and parietal are wholly *independent.*

Finally, even Ecker himself, perhaps unintentionally, has narrowed at the same spot on fig. 2 the heavy line representing his continuous fissure.

The only difficulties encountered in recognizing the paroccipital fissure have arisen from the occasional presence of a more or less distinct transverse fissure between the occipital and one or the other ramus, so as to subdivide the cephalic or the caudal arm of the paroccipital gyre.

In these cases the gyre is wider than usual. Such intercalated fissures may be called *preparoccipital* and *postparoccipital.* I have not observed their co-existence on the same side.

SUMMARY.

Historical.—Most writers follow Ecker in recognizing a "transverse occipital fissure" and in regarding the parietal ("interparietal") as comprehending the whole length of a fissure which begins on the parietal lobe, may be interrupted in its course, but usually (*always*, I believe) joins the transverse nearly a right angle.

Facts.—1. In no figure accessible to me (excepting one side of Ecker's diagram), and in no one of twenty-nine adult occipital lobes examined, is there an independent fissure answering to the "transverse."

2. The supposed single parietal always begins in two parts: one, the true parietal, on the parietal lobe; the other, opposite the dorsal end of the occipital ("parieto-occipital") fissure.

3. In the adult these two fissures remain independent in about half of the cases, more often on the right side.

4. When there is a continuous fissure, it is, in all but one of the cases examined, shallower at the point where, if it were a single integer, it should be deepest.

Conclusions.—1. Ecker's "interparietal" includes (*a*) the true parietal; (*b*) the longitudinal part of a newly recognized fissural integer—the *paroccipital.*

2. In its typical condition, the paroccipital fissure is a longitudinal bar (zygon), opposite the occipital, bifurcating at each end into a cephalic and caudal stipe extending mesad, and a cephalic and caudal ramus extending laterad.

3. The "transverse occipital" of Ecker is the caudal stipe and ramus of the paroccipital, and does not constitute a fissural integer.

4. The paroccipital is an example of what may be called *zygal* or yoked fissures; the orbital is another.

5. The gyre intervening between the occipital fissure and the paroccipital may now be called the *paroccipital gyre*, instead of annectant convolution, etc.

6. Fissures which occur within the paroccipital gyre may be called, according to location, preparoccipital and post-paroccipital.

7. All fissures should be studied, not only in their development, their relation to other parts, and their superficial course and connections, but also in respect to their relative depth in various parts of their course.

8. The fissures of the dorso-caudal aspect of the occipital lobe are more clearly represented when the line of vision coincides nearly with the general direction of the occipital fissure.

BIBLIOGRAPHY.

Bischoff, T. L. W.: 12. Die Grosshirnwindungen des Menschen mit Berücksichtigung ihrer Anordenung bei den Affen. München Akad. Abhandl., X. 1870, pp. 389–498.

Clevenger, S. V.: 4. Cerebral topography. JOUR. OF NERVOUS AND MENTAL DISEASE, Oct., 1879, pp. 27, 3 figures.

Dalton, J. C. : Topographical anatomy of the brain. Q., 3 vols., pp. 175 ; 48 plates, with outline duplicates ; 7 figures. Phila., 1885.

Ecker, Alex. : Die Hirnwindungen des Menschen, etc. 2d ed., O., pp. 58, 7 figs. Braunschweig, 1883.

Edes, R. T., *transl.*—Ecker's The cerebral convolutions of man, represented according to original observations, especially upon their development in the fœtus ; intended for the use of physicians. O., pp. 87, 6 figs. New York, 1873.

Edinger, L. : Zehn Vorlesungen über den Bau der nervosen Centralorgane für Ærtzte und Studirende. R. O., pp. 138, 120 figs. Leipzig, 1885.

Ferrier, D. : The functions of the brain. O., pp. 323, 68 figs. New York, 1876.

Fowler, E. P., *transl.*—Charcot's Lectures on localization in diseases of the brain. O., pp. 133, 45 figs. New York, 1878.

Gage, S. H.: **19.** A starch injection mass. *New York Medical Journal*, June 7, 1884. (A slight modification of this is interpolated as a leaflet between pp. 140 and 141 of the second edition of "Anatomical Technology.")

Gratiolet : Memoire sur les plis cerebraux de l'homme et des primates. Q., pp. 104, with folio atlas of 13 plates. 1854. (The volumes bear no date, but the one above given is contained in an advertisement of the work upon a fly-leaf of Lauret et Gratiolet.)

Jensen, J. : 1. Untersuchungen über die Beziehungen zwischen Grosshirn und Geistesstorung an sechs Gehirnen geisteskranker Individuen. *Psych. Arch.*, v., 1874–5, pp. 587–757 ; 5 plates.

Keller et Duval, *transl.*—Huguenin's Anatomie des centres nerveux. O., pp. 368, 149 figs. Paris, 1879.

Leuret et Gratiolet : Anatomie comparée du système nerveux considere dans ses rapports avec l'intelligence. 2 vols., O., 1284 pp., with folio atlas of 32 plates. Paris, 1831, 1857. (Vol. I. is by Leuret, Vol. II. by Gratiolet ; as stated in a note appended to the explanation of Pl. xvii., Pl. i.–xxii. were drawn under the direction of Leuret ; but the explanations of xvii.–xxii. were prepared by Gratiolet according to Leuret's method ; the remaining plates were prepared under the direction of Gratiolet.)

Pansch, Ad. : Die Furchen und Wülste am Grosshirn des Menschen. O., pp. 51 ; 3 plates. Berlin, 1879.

Spurzheim, J. G. : The anatomy of the brain. 2d. Am. ed., 1836. O., pp. 244, 18 plates.

Wernicke, C. : 1. Das Urwindungsysstem des menschlichen Gehirns. *Psych. Arch.*, vi., 1875–6, pp. 298–326 ; 3 plates.

Wilder, B. G. : **11.** The outer cerebral fissures of Mammalia (especially of the Carnivora), and the limits of their homologies. Am. Assoc. Proc., xxii., 1873 (pt. 2), pp. 214–234 ; 19 figs.

—— **64.** Paronymy versus heteronymy as neuronymic principles. Presidential address at eleventh annual meeting of Amer. Neurol. Assoc., June 18, 1885. JOUR. NERV. AND MENT. DISEASE, xii., July, 1885, pp. 21.

—— **66.** On two little known cerebral fissures, with suggestions as to fissural and gyral names. Amer. Neurol. Assoc. Trans., JOUR. OF NERV. AND MENT. DISEASE, xii., 350–352, July, 1885.

Wilder, B. G., and Gage, S. H. :—Anatomical technology as applied to the domestic cat. 2d ed., O., pp. xxix., 591, 4 plates, and 130 figs. New York, 1886.

MASSAGE IN NERVOUS DISEASES.

By Dr. GEORGE W. JACOBY.

PHYSICIAN TO THE CLASS OF NERVOUS DISEASES OF THE GERMAN DISPENSARY OF THE CITY OF NEW YORK.

IV.

(*Continued from page* 223.)

Writer's Cramp.

UNDER this title we shall consider all affections which have been described by Benedict under the generic name of "Co-ordinatory Occupation Neuroses," without at all confining ourselves to the various forms as they occur in writers. The name "writer's cramp" has, almost since its existence, for various manifest reasons, been acknowledged as insufficient. The symptoms, for we really have only symptoms to deal with, and not a disease itself in the true significance of the term, may occur in any person who is obliged to make continuous or prolonged use of any group of muscles. These symptoms, occurring in persons occupied in various pursuits, have received the names of the occupations followed; thus we read of writer's, draughtsman's, engraver's, telegraphist's, violinist's, pianist's, blacksmith's, tailor's, sempstress', and also of photographer's and ballet-dancer's cramp. The movements employed in all these occupations are not produced by the contractions of any one muscle, but by the harmonious co-operation of several. We will, therefore, for convenience' sake, speak of writer's cramp only, but all our remarks apply with the same force to any of the so-called "cramps," as they occur in either of the above-enumerated occupations.

The word "cramp" itself is also objectionable, as the symptoms may present themselves in various forms, of which the "cramp" is only one. Benedict has classified the symptoms under the headings of spastic, tremorous, and paralytic; and these seem to us to cover the symptoms observed. Exactly where the seat of the disease is in writer's cramp, is a question which has received considerable attention, and has been the subject of great discussion, and is one which certainly cannot at present be answered in a positive or dogmatic manner. All co-ordinated movements, particularly complicated ones, can only be produced if there is no obstacle to the harmonious action of all the muscles necessary to the production of the desired act; any derangement will, according to its seat or character, be followed by an irregularity or incapacity. The various possible sites have, in this affection, been accused of being implicated. Thus, by some authors, Duchenne, Solly, Althaus, Erb, it is supposed to be of central origin, situated either in the supposed co-ordinating centres of the brain, or in the cervical part of the spinal cord. Others, Reynolds, Zuradelli, Geigel, Haupt, Meyer, Poore, Beard, etc., believe it to be of peripheral origin, and due either to an affection of the motor or of the sensory nerves.

For our purposes it is not necessary to enter into the question of the pathology or even of the mechanics of the peripheral cases at all extensively, but certainly to-day the bulk of testimony goes to prove that the majority of cases of writer's cramp, if carefully examined, will be found to be of peripheral origin. On the other hand, it cannot be denied that many cases are of central origin, and that in many also, the so-called writer's cramp is only an early symptom of some central affection of the nervous system, which might have been diagnosticated if sufficient care and attention had been devoted to it.

When we consider that the train of symptoms known as writer's cramp may be produced by a disorder of the most different parts of the nervous system, and also by the most varied causes, we can appreciate the necessity of a careful examination of every case, and, if possible, of making a

diagnosis which shall embrace more than the words "writer's cramp." This necessity becomes all the more apparent in the treatment by massage, for here in order to attain any thing like the uniform good results of which we shall speak later, it is imperative to be able to select our cases, as it is evident that a case of central origin will not be benefited by massage any more than by any other mode of treatment; and cases which are of peripheral origin, even if the central nervous system has ultimately become functionally disordered, may, nevertheless, be influenced by attacking the primary locus morbi. The very first question which we must propound to ourselves is, therefore, "Is the case peripheral or central?" If it is pheripheral, what is the direct disorder? Are we dealing with a paresis or a paralysis of certain small muscles of the hand, or with a disordered state of nutrition of single muscles, by which means is produced a hypercontraction of the muscle itself or a relaxation of its opponents, and thereby an inco-ordination, or is it an early stage of a peripheral neuritis? The answers to these questions are not always easily given, and, to insure any thing like accuracy, the examination should be a very careful one.

Particular attention must be paid to the action of each muscle separately, and also to the harmonious action of the various groups. For the latter purpose the writing itself must also be studied. This is essential to the making of a special diagnosis of the muscle or muscles affected. Painful points and indurations along the course of the nerves —in fact, in the entire arm—must also be sought after.

It is unnecessary here to enter into a consideration of the physics of the various muscles employed, as has been so carefully done by Zuradelli, but, at the same time, it is well in examining a case to remember certain facts which will enable us to detect disorder of certain muscles and to more carefully direct our treatment.

In examining the method of pen prehension, which should be done first, it will be found that, if the pen is grasped tightly, it is probable that one or more of the small muscles, —the interossei, the abductor and opponens pollicis, the

flexor brevis pollicis, and the extensor primi internodii—are incapacitated for work, and that the larger ones are being used as substitutes. If the forefinger rises or slips off the pen, a failure of action of the first dorsal interosseous is the cause. If the phalangeal angle of the thumb gives in, it is due to a disorder of the extensor primi internodii.

The various movements of the fingers must next be watched,—the up-and-down motion, the lateral movement of the hand, etc. According to Poore, inability to keep the hand upon the paper, is due to a failure of the supinator longus.

A limitation in the length of lines or letters (to about 1½ cm.) is generally due to a paralysis of the long extensors of the fingers, as thereby an inability to extend the first phalanx is produced. Inability to strike a loud note on the piano, or to hold the pen with the points of the fingers, is generally due to a paralysis of the long flexors, the first and second phalanges being found in permanent extension.

Abduction of the thumb, so that it falls in the palm of the hand and prevents flexion of the fingers, is due to paralysis of the abductor longus pollicis and flexor brevis pollicis.

An inability to touch the ends of the fingers with the thumb without flexing them in the second and third joints, denotes a paralysis of the abductor brevis and opponens pollicis.

The importance of examining carefully for these and similar manifestations of peripheral mischief in the muscles, and the frequency of occurrence of such disorders in the various artisan's neuroses, may be appreciated when the following statement of Poore is considered: "In every case of impaired writing power which I have seen, there has been evidence, more or less marked, of derangement of one or more of the muscles used in writing"; and he furthermore says: "The writers' cramp of text-books, in which failure of writing is the sole symptom, I have never seen."

We can fully corroborate both of these statements; all cases, whether of writer's cramp or of other artisans' cramp which we have seen, have, upon careful examination, pre-

sented evidence of peripheral disturbance sufficient to account for the failure in their work. Furthermore, the cramp was in none of them the primary and sole disturbance, and all of our patients were not only unable to perform their own special work, but were also unable to execute other work which entailed similar movements and similar use of the affected muscles. It is not essential that this peripheral disorder be dependent upon a paralysis or a paresis; but it may, and we believe it to be so in very many cases, be due to a faulty innervation of single muscles or groups of muscles, which causes either a failure in their action while the other muscles still exert their functions normally, or causes them to enter into a state of tonic contraction whenever an unusual amount of work is required of them.

The electrical examination will also, in many of these cases, indicate the affected muscles, showing a difference in the reaction from that of the corresponding muscles of the other hand—either an increased or a decreased irritability. Stress is also laid upon the electrical examination by Zuradelli, Erb, Gowers, Poore, and others.

Another indication of disordered nutrition of the muscles is fibrillary tremor; it is sometimes found at a very early stage, and even before any marked subjective symptoms are present.

Treatment.

Canstatt's words, written over forty years ago, "Much has been tried, nothing has succeeded," could, until a few years ago, have been repeated, and fully express the results of treatment in writer's cramp. Here and there a cure was effected by means of one remedy or other, but the great majority of cases remained unimproved.

Since massage has been systematically used in the treatment of this affection, the cures attained have far outweighed the failures. As to whom is due the honor of first recommending massage in the treatment of writer's cramp, there is some difference of opinion. Meding, over thirty years ago, used it empirically; Erb, in 1874, in the first edition of his text-book on disorders of the nervous system, speaks of "Gymnastic and massage for the cure of writers' cramp";

and Douglas Graham, in 1877, was one of the first to direct closer attention to this means of treatment.

The first reported case of cure by means of massage was published by Rossander in 1873, but, unfortunately for the claims of massage, another remedy was simultaneously employed—the subcutaneous injection of strychnine,—and for this reason it is not possible to ascribe the cure to the use of the massage alone. The massage treatment consisted in two séances daily; the muscles of the thenar and hypothenar eminences, the interossei, and the lumbricales were rubbed successively. At the same time the muscles of the thumb, of the small finger, and of the forearm were percussed with a wooden cylinder. The patient was cured after four weeks of treatment.

The next case is one by Gottlieb.

Patient, a female æt. fifty-two, came to Gottlieb August 17, 1874; had always been well until 1863. She was accustomed to write as much as nine hours a day. At this time she exposed herself to severe cold. Two years later she suddenly became incapable of writing. The pen falls from her hand, and she is unable to resume her work. Since then the right index constantly refuses to act in writing or in performing any analogous work. Then the middle finger became affected; still being able to use the thumb, she continued to write with it and the two last fingers. She then endeavored to use her left hand, and this in time became affected, but to a much lesser degree. Both hands presented a slight degree of œdematous swelling. She was treated by massage. Thirty-seven séances in all were used. Complete and permanent cure.

In 1877, Douglas Graham republished these two cases and another by Drachman, which latter, however, we do not consider as one of writer's cramp, and at the conclusion made the following remarks: "When sufficient time for rest has been allowed, and in the absence of spasm, or spasm of the flexors alone being present, I should think it might be useful to add resistive motion, so as to bring systematically into more powerful action the opposing and less-used extensors, which would tend to restore harmony

of action by a counterbalancing distribution of will, nerve, and muscular effort." There is as much of value contained in this paragraph as in all that Wolff, the self-styled inventor of a "method" of cure, has written.

Since then others have also, in a few words, advocated the use of massage in these affections, but it was not until after Schott's publication in 1882, that particular attention has been devoted to this mode of treatment. Wolff, who claims to be the originator of the method, is a writing-master, and as such, according to Stein, sees many children and adults with poor handwriting. Among these naturally there are some affected with writer's cramp, who lay the blame of their poor writing to their incapacity, whereas it is really due to their disease. Wolff, therefore, endeavored to cure them "by a peculiar system of writing-instruction, which he combined with massage and gymnastic exercises, passive and active, applied to the muscles of the arm."

Beyond any doubt Wolff has treated and cured more cases than any other single person. For that, reliable evidence is present; but he has done absolutely nothing towards communicating his mode of treatment to others. In an article published in 1884 in the *N. Y. Med. Record*, p. 205, vol. i., he says, in speaking of the priority claims of the Drs. Schott:

"Both gentlemen again and again questioned me about my method, but they never received another response to their numerous inquiries than that the same consisted in a peculiar combination of massage and gymnastics." And in a small monograph, "The Cure of Writer's Cramp, etc.," published in 1884, he evades description of the method by the same means as he claims to have employed with the Drs. Schott.

Certainly, whether Wolff or Schott is the originator of the method, to Schott belongs the credit of having communicated it to others, and Wolff is entitled only to the thanks of the patients he has cured, and not to those of physicians or scientists, for he has not advanced the cause of scientific knowledge one iota, but on the contrary has only endeavored to obscure and to hamper it. Schott's

mode of treatment consists in a combination of gymnastics and massage. The gymnastics consist of movements performed by the patient alone, and movements performed with the opposition of the operator.

The first are performed by the patient during from twenty to thirty minutes, rarely for forty-five minutes. The first movements are gymnastics of the fingers, extension, flexion, abduction, and adduction, the thumb being exercised separately; thereupon the same four motions are executed at the wrist joint, then extension and flexion of the forearm, and ultimately the arms themselves are exercised in the same manner and are to be lifted over the head. Each single exercise is to be performed from six to twelve times. After each motion a pause is to be observed.

The opposed movements are to be carried out in the same manner, except that the operator must carefully resist their executions as though he were endeavoring to force the patient to perform a motion just the reverse of his intentions. Regularity of pressure is to be observed in this, so that the same amount of force is always used, and that the pressure does not vary in intensity from moment to moment.

The time to be devoted to these opposed movements is to be the same as that for the unopposed ones. According to the intensity of the affection the exercises must be repeated two to three times daily. The massage itself consists of two parts—nerve and muscle massage. The nerve massage is effleurage along the course of the nerve trunks, the median, ulnar, and radial, going upwards to the axillary and cervical plexuses. This effleurage lasts about ten minutes. Following this is the muscle massage. This consists of petrissage, beginning with the hand and ending at the shoulder. Duration same as last movement. One sitting a day has always proved sufficient.

Schott says in from two to three weeks improvement is noticed. The treatment must not be interrupted then but must be kept up for at least six to eight weeks, which time is necessary to attain a complete cure. During the time of treatment the occupation of the patient must be discontinued. Schott has thus succeeded in curing all the neuroses which

came under his treatment, mostly pianists, and in none of them has any return been observed.

Wolff's method, according to *Stein*, for he is the only one who has given any thing like a detailed description of Wolff's procedure, differs from the preceding in so far that he makes use of "a peculiar method of writing-instruction" in addition to the gymnastics and massage.

And he says, furthermore, "the peculiarity of the method consists in that fact, that Mr. Wolff, in consequence of years of practice and special treatment, understands how to carefully isolate those muscular groups which require a special gymnastic treatment, either with his hand or by means of rubber bands, in a way that we electro-therapeutists, even with the most minute electro-diagnostic examination, are unable to do."

The accompanying illustrations taken from this article

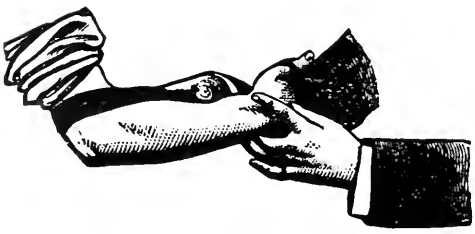


FIG. I.

FIG. II.

will give an idea as to the manner in which these bands are applied.

Fig. I. shows the patient, on whom, by means of tightly drawn rubber bands, certain muscular groups have been isolated, in the act of having passive opposed movements performed.

Fig. II. shows the same in the execution of active movements.

Wolff has certainly achieved remarkable results by his mode of treatment, and that his patients are permanently cured we must believe, for we have the evidence of men like Billroth, Esmarch, Wagner, Bardeleben, Bamberger, Hertz, Benedict, Nussbaum, Charcot, Vigouroux, and Stein; and only recently De Watteville, of London,

has published cases which have been completely cured by Wolff. From 1877 to 1882, Wolff has treated in all 277 cases of writer's cramp and similar affections—245 were writer's cramp, and of these 132 were radically cured, 22 improved, and 91 without result; 32 cases of pianist's, violinist's, telegrapher's, and painter's cramp—of these 25 were cured. In all, 157 cured, 22 improved, 98 not cured.

No other single person can hope to attain such results from want of material; but Wolff is not alone in obtaining good results; for Weiss, Podrazky, Zabludorosky, Schreiber, and Douglas Graham all report good results in the use of massage in writer's cramp.

The results obtained by us have been excellent ones, and, although the number of cases is necessarily a limited one, still it is sufficient to show that there is no secret in the method, and that good results can be obtained by any one; all that is necessary being perseverance and thoroughness, and that the "peculiar" combination and the "peculiar" system of writing instruction is not essential. As model for treatment we made use of Schott's description.

The patients were examined carefully, to localize, if possible, the exact seat of the affection: cases which were not clearly of peripheral nature were not treated at all. Particular attention during the massage was paid to the interossei, and to all the muscles of the thumb. The massage was carried out in the manner described by Schott, the idea to be kept in mind being that, by means of the exercise and massage, the weakened muscles are to be strengthened and their nutrition improved.

In order to thoroughly manipulate the interossei and lumbricales, the hand of the patient sitting face to face with the operator must be taken by him into both hands, and the metacarpal bones separated from each other as much as possible, and then moved upward and downward. The small end of Granville's percuteur will also be found very serviceable in acting upon these small and deeply-seated muscles.

The larger muscles can be manipulated as described under the general head of Technic—petrissage and massage à friction being principally applied.

The most important part of the treatment, however, consists in the active and passive movements, with and without opposition.

For the execution of the active movements we have laid down the following rules to be observed by the patient. The movements are to be executed :

1st. As slowly as possible.

2d. In a certain rhythm.

3d. With a certain amount of effort.

4th. Care must be taken not to over-exert the muscles.

5th. The movements must be executed three times daily, and each movement carried out from twenty-five to two hundred times.

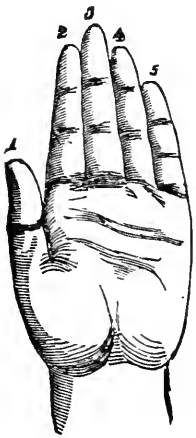


FIG. III.

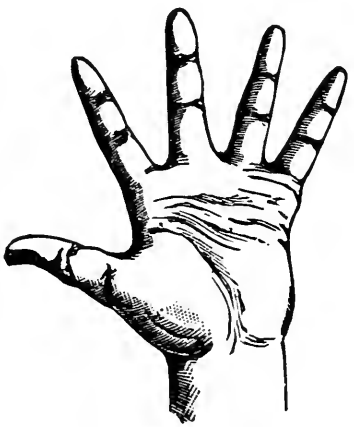


FIG. IV.

FIG. V.

Every patient is first taught the following positions of the hand by heart :

1st. Fingers extended and approximated (Fig. III.).

2d. Fingers extended and separated (Fig. IV.).

3d. Fingers flexed at right angles to the hand and approximated (Fig. V.).

4th. Same, but fingers separated.

5th. Fingers flexed at knuckle (first phalanx forming straight line with hand), fingers approximated (Fig. VI.).

6th. Same, fingers separated (Fig. VII.).

7th. Fingers flexed upon the palm and approximated (Fig. VIII.).

8th. Same, but fingers separated as much as possible.

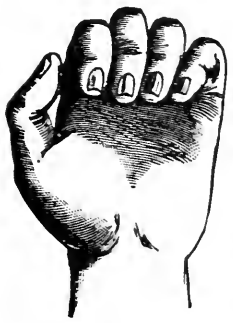


FIG. VI.

FIG. VII.

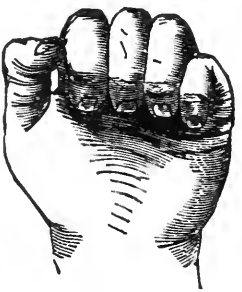


FIG. VIII.

After the patient has learned these perfectly and by number, the following exercises are given him to exercise at home.

1. The fingers are to be brought from 1st to 2d position, one after the other (muscles exercised are the interossei, volarès et dorsales and the external fibres of the extensors).

2. Fingers to be brought successively from 1st to 3d position (muscles exercised are the flexor digit. commun., sublim., and profund., principally the former). The lumbricales and interossei also assist in this movement.

3. Fingers to be brought from 1st to 5th position (muscles exercised, flexors, principally the flexor profundus perforans).

4. Fingers from 1st to 7th position (muscles exercised are the flexors equably and the four lumbricales).

5. Fingers from 2d to 3d position (muscles exercised are the same as in No. 3, together with the interossei.)

6. Fingers from 2d to 5th position.

7. From 2d to 7th position.

8. From 3d to 4th position.

9. From 3d to 5th position.

10. From 3d to 7th position.

11. From 4th to 6th position.

12. From 4th to 8th position.

13. From 5th to 7th position.

14. From 6th to 8th position.

Reversing these movements will, of course, exercise the antagonists; thus, in exercise 2, if instead of commencing with the 1st position and bringing the fingers to the 3d, we reverse matters and bring them from the 3d to the 1st, then, instead of bringing the strain upon the flexors, we do so upon the extensors.

These exercises must be chosen with care and given to the patient for execution at home, one at a time, care being taken that they are well understood and satisfactorily executed.

The following opposed movements can of course only be executed with the aid of the operator.

The opposed movements which are the most serviceable are the following:

1. Hand of patient in position 1, each finger is successively to be brought into position 3 while the operator endeavors by counterpressure to prevent it.

2. Fingers in position 3, operator endeavors to bring them into position 1 while the patient prevents it.

3. Fingers in position 1, to be brought to position 2 while operator opposes.

4. Fingers in position 2, to be brought to position 1 by operator while patient opposes.

5. Fingers in position 4, to be brought to 3 by operator. Fingers in 3, to be brought to 4 by patient.

6. Fingers in position 6, to be brought to 5 by operator. Fingers in 5, to be brought to 6 by patient, operator opposing.

In order to enable the patient to perform certain opposition movements at home and at the same time to be sure that a certain amount of equable opposition is being used, we have constructed the following apparatus. The movements which are to be executed are simply those of extension and flexion, and the opposition is furnished by rubber bands.

The apparatus consists of a metallic bracelet, the inside of which is padded. One end of it consists of a ratchet,

which admits of increasing the size of the bracelet to fit any arm, and at the same time allows it to be tightly fastened. Upon one side of the bracelet, the top or bottom as the case may be, are fastened five brass pegs. Rubber bands are buttoned to these by means of leather end-pieces. The other end of the bands terminate in leather finger-coverings, which are slipped over the fingers of the hand. The bracelet is always to be adjusted just above the joint, and tight enough to keep it from slipping. If the extensor muscles are the ones to be exercised, the

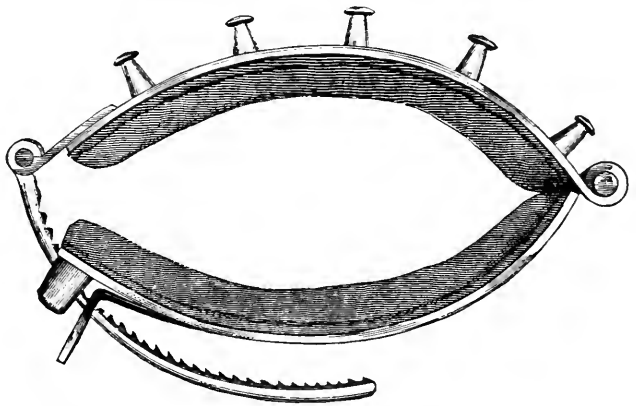


FIG. IX.

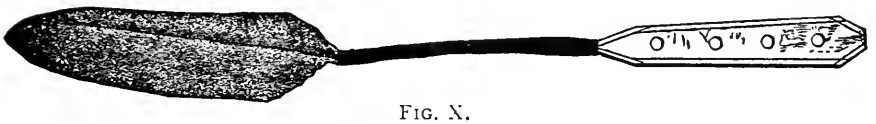


FIG. X.

bracelet must be so placed as to allow the pegs to be at the bottom. Care must be taken that the apparatus be placed on the arm in pronation for flexion movements, and in supination for extension. The kid fingers are then slipped over the fingers of the hand, and the elastic bands attached to the pegs. These elastic bands must be chosen for each case, and frequently of different strengths for the various fingers. If the flexors are to be exercised, the bracelet is applied with pegs pointing upward, and the bands then adjusted. The opposition is thus furnished, and the patient can execute the movements at home (Figs. IX. and X.). The

thumb must always be exercised separately. For opposing abduction, the band is brought from the inside of the thumb over the palm around the ulnar border of the hand and fastened to the proper peg on top. For opposing adduction, the bracelet is fastened with the pegs pointing downward. The rubber band is then brought over the back of the hand around the ulnar border and fastened to the desired peg below. The question whether patients afflicted with one of these artisan's neuroses must give up their employment during the time of treatment, is one which must be answered in the affirmative for all except writers, and these can, by means of an apparatus designed by v. Nussbaum, continue writing during the time of treat-

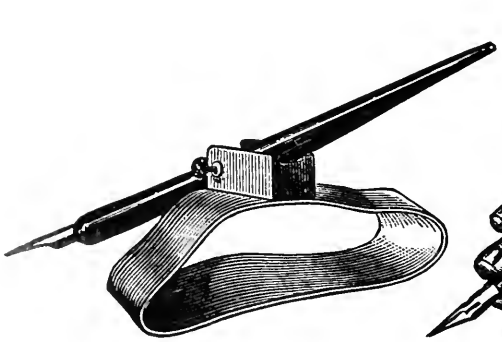


FIG. XI.

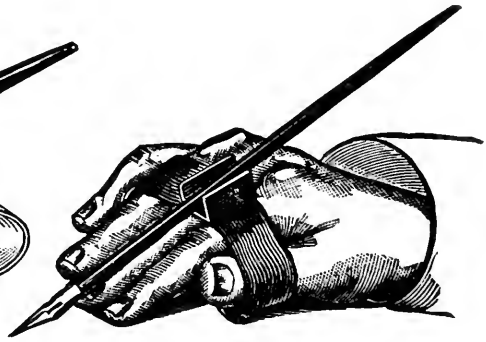


FIG. XII.

ment. In fact, the more they write with the apparatus, the more they hasten their recovery; v. Nussbaum taking the idea as a basis, that in writing, the flexors and adductors of the fingers are almost exclusively employed, and that the cramp is produced by over-exertion of these muscles, therefore the latter can be avoided by employing the antagonists —the extensors and abductors—in all writing. The apparatus constructed to effect this consists of a thin, oval, hard-rubber ring, of about two centimetres in width (Figs. XI. and XII). This ring is somewhat convex above, and is made to fit over the first four fingers (thumb, index, middle, and ring). It is passed over these fingers, the small finger remaining outside. Upon the upper surface of the ring is a movable pen-holder, which can be fastened in any

position by means of a screw. The ring must be chosen somewhat wider than the fingers to be surrounded by it. In order to keep the apparatus from sliding off the hand, the patient is obliged to keep his fingers forcibly abducted, the thumb pressing upon one side and the ring-finger upon the other. Thus the abductors and the extensors are the muscles used. For persons who are obliged to keep to their business during the course of treatment, the apparatus is invaluable; as after a very few hours of practice they can write plainly and legibly and without any exertion, and, as already stated, far from having any deleterious effect upon the results of the treatment, it may be looked upon as an ingenious adjuvant.

The following cases have been treated by us in the manner described above.

CASE I.—Nov. 1, 1883.—P. W. S. æt. forty; lithographer; has always been well. He is a draughtsman and lithographer by profession and was considered very expert in his line. Always worked very hard, generally working from eight to ten hours a day, and frequently spending his evenings in writing. About two years ago he experienced a gradual and increasing difficulty in executing his work. When drawing or writing he would have a dull, tired feeling in the entire hand, and a feeling of pain in the thumb and index finger.

The difficulty in writing and drawing increased until he was unable to work for more than half an hour at a time. Then he was unable to keep his thumb upon his pen. It slipped from it and became flexed upon the palm of the hand. Soon this occurred whenever he attempted to write. He had been unable to do any work whatsoever for over a year. Drawing, engraving, or writing were absolutely impossible.

Electrical examination showed reduced excitability to both currents in the flexor pollicis brev. and the abductor longus pollicis.

The first and second dorsal interossei were atrophied. There was pain upon pressure along the course of the radialis.

The treatment by massage and gymnastics was begun on Nov. 4, 1883, particular attention being directed to the affected muscles.

V. Nussbaum's bracelet was used for writing, with the result that after a day's practice he was able to carry on his entire correspondence by its aid. He attempted to use it for drawing but did not succeed as he could not make his movements sufficiently delicate. The treatment was carried on daily for four weeks in the manner described. At the end of this time he considered himself well, but as there was still pain upon pressure and reduced electrical excitability in the muscles, he was advised to continue treatment. After two further weeks he resumed his former occupation, and as a proof of his ability and gratitude, his first work was a crayon portrait of ourself. He has since then remained perfectly well, and has not lost a day's work, although he is more careful not to overwork himself than formerly.

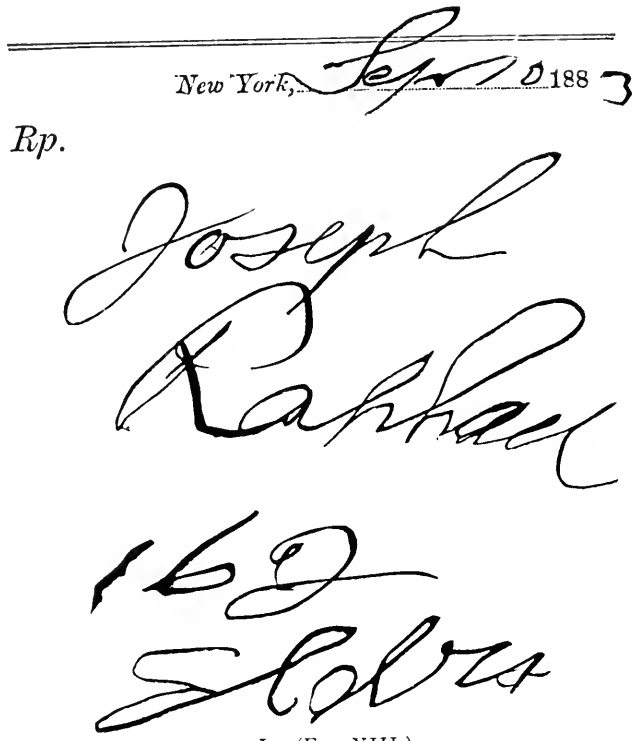
CASE 2.—Sept. 9, 1885.—J. R., æt. twenty-eight; bookkeeper. Family history very neurotic. Father had paralysis agitans when he died at age of sixty-four. A sister of his father has epilepsy. One brother in the insane asylum. Patient was always very nervous and easily excited. Frequently had cramps of various groups of muscles.

At the age of eighteen, while preparing for an examination, he had a great deal of writing to do, and finally broke down under the strain. He says for months he was unable to write at all. Had a painful, tired feeling in the entire right arm, up to the shoulder. This, however, in consequence of rest got better, and he was able to take a position as bookkeeper. He was then well until age of twenty-six, when the same symptoms again set in. They grew so severe that as soon as he had written a few words, his fingers began to tremble, and then the pen would be tightly grasped, the fingers entering a state of tense contraction. This state of affairs had lasted for nearly two years, when we saw him. During this time he was unable to do any work at all. Upon examination the entire hand seemed cold and weaker than the left, the entire nutrition

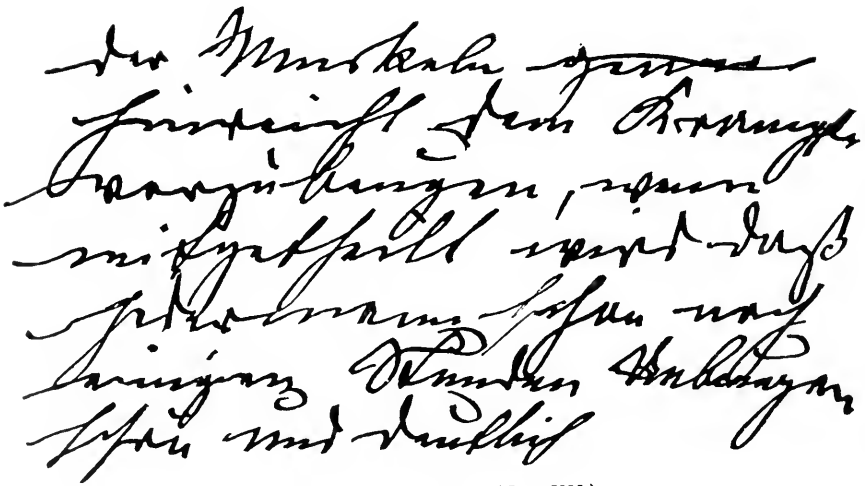
of the arm being a diminished one. The muscles particularly affected were the flexors, and the patient found it difficult to hold the pen firmly. When he had, however, by a severe effort, managed to write a few words, the flexors contracted spasmodically, drove the point of the pen into the paper, and put an end to any further attempt at writing. The first specimen (Fig. XIII.) shows this very well, and is all that he was able to write at that time. The second specimen (Fig. XIV.) was written after ten days' treatment and is also the entire amount. The third is the last part of a dictation of a page of note-paper, written after three weeks of treatment (Fig. XV.). The fourth is the last part of a dictation of a page of note-paper, written after six weeks' treatment (Fig. XVI.). The patient has since then remained perfectly well.

CASE 3.—Patient, female æt. thirty; piano teacher. Has devoted almost her entire life to the study of music, and has, from her fifteenth year, rarely spent less than six hours a day at the piano. At the age of twenty-eight she found that her left hand, which had formerly been as thoroughly developed as the right, and possessed the same amount of mechanical dexterity, was growing weak. She attributed this to an insufficient amount of practice, as she was then teaching a great deal and practising less than usually. She attempted to make up for this by increased attention and playing until very late at night. The more, however, that she played, the worse did her hand feel. She then had drawing pains along the entire arm, and a feeling of heaviness.

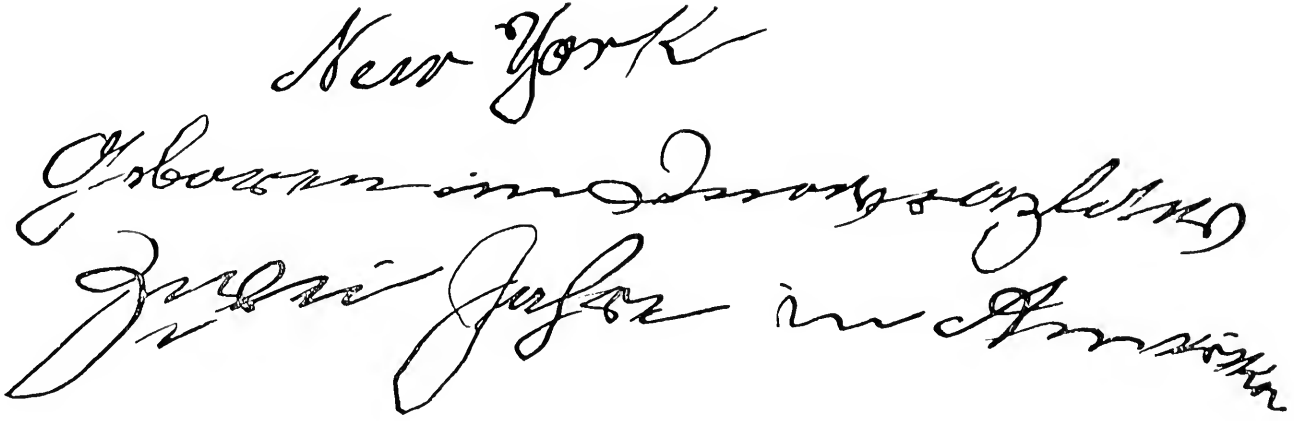
The principal trouble seemed to be in raising the fingers from the keys and in stretching any thing over an octave. This grew worse gradually, and she was ultimately obliged to give up playing entirely. When she came under our treatment, in October, 1884, she had not played upon the piano for over six months. Upon examination, it was found very difficult to localize the affection in any particular set of muscles, but the interossei and the extensors were, from the history, supposed to be specially implicated, and the treatment addressed to them particularly. After



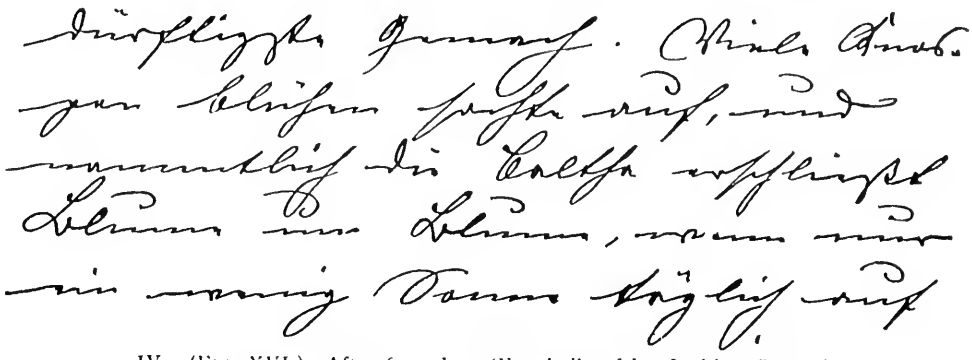
I. (FIG. XIII.)



III. After 3 weeks. (FIG. XV.)



II. (Fig. XIV.) After 10 days' treatment. (Fac-simile of handwriting Case 2.)



IV. (Fig. XVI.) After 6 weeks. (Fac-simile of handwriting Case 2.)

eight weeks of treatment she began playing again, but still felt a weakness in her hand, very much the same as when the affection first began. She, however, kept on with the gymnastic exercises for several weeks longer, and then considered herself entirely well. She has since then remained well, but says that her left hand is not as reliable as it formerly was.

Besides these three cases we have had two more treated by this means, the one a stenographer the other a merchant. These cases present nothing particular of interest: the one is still under treatment, and the other is apparently cured; but the time is still too short to be able to say if it is permanent or not.

The number of cases is too small to draw any deductions from them, but they certainly show that these cases, if properly selected, can be cured, and all that is necessary is massage and gymnastics, which are not in any way "peculiar," and without the special knowledge of the mechanism of writing and the writing calisthenics, which Wolff claims are an essential part of his "method."

OPHTHALMOSCOPIC STUDIES OF ACUTE MANIA, WITH NOTES OF CASES.*

By LOUIS J. LAUTENBACH, M.D.

(ASSISTANT SURGEON, EYE AND EAR DEPARTMENT, PHILADELPHIA DISPENSARY.)

HISTORIES OF PATIENTS.

By Dr. ALICE BENNETT,

(RESIDENT PHYSICIAN, FEMALE DEPARTMENT, STATE INSANE HOSPITAL, NORRISTOWN.)

CONSIDERING the intimate relations of the eye to the brain; its near proximity to this important centre; the short distance between the origin and the termination of the optic nerve: the fact of its venous system terminating in that of the brain; and that the arterial supply is derived directly from the same main vessel as that of the brain; that the subvaginal (inter-sheath) space is but an extension of the subarachnoid space; and that perhaps there is a canal system in the cribriform fascia which is in communication with the same space; considering also that in the last twenty years ophthalmology has advanced with such rapidity, it certainly seems remarkable that more attenhas not been bestowed upon the ophthalmoscopic examination of the mentally diseased—the subjects of insanity.

The pathology of insanity is certainly very important, and yet of it but little is known. The eye offers a means by which we may perhaps, even in the living subject, obtain some suggestion of the diseased condition; perhaps be a guide-post which, in a path involved in the mist of uncertainty, may with a gleam of light here and there serve to give a suggestion of the way.

* Read before the Phila. Neurological Society, April 26, 1886.

In acute mania the pathology is very uncertain, both by reason of the infrequency of deaths as well as by virtue of the fact that in the majority of cases when death occurs little or no change is found. This absence of any marked lesion is not conclusive that there are no lesions during life; disturbances of the vascular supply of the brain are evanescent, and when present are not striking, and are therefore either not observed or are passed by without remark.

These ophthalmoscopic examinations were undertaken, despite considerable discouragement both from ophthalmologists and alienists, with the view of determining whether the study of eye conditions might not be of service in the pathological study of insanity, and after three years' work, examining some eight hundred insane patients, it would appear that its value would soon be evident.

It is surprising that of the many text-books and treatises on diseases of the eye, of which twenty-five were examined, but two have any reference to eye conditions in insanity. Of the sixteen text-books on insanity examined (all published since 1865) several had no reference to the subject whatever, while the rest had but a few lines or at most a page or two devoted to it.

The apathy displayed is certainly, remarkable and scarcely in keeping with that spirit of investigation with which man is supposed to be blessed. There are no unusual difficulties to be encountered in the prosecution of these investigations; time and patience are the requisities; in fact there are peculiar advantages offered—the patients are not scattered here, there, and everywhere, but are collected together, many similar cases can be examined in succession, or again dissimilar cases can be immediately contrasted. A question which here presents itself is as to the reason why the medical residents at the institutions do not prosecute the work. Their excuse must be the multiplicity of their duties preventing their devoting sufficient time to become expert. While believing that it requires constant daily work for years to be able thoroughly to recognize changes in nerve structure, as well as minute vascular changes, yet, with six months' daily work, the eye can become sufficiently trained to allow of the recognition of ordinary conditions.

That the eyes of the insane should be systematically examined, seems to be self-evident. That all patients should, if possible, be examined at least once by an experienced ophthalmologist, goes without saying; that many cases should be kept under observation, appears to be a necessity.

The amount of time this would take in a large institution would be considerable—more, perhaps, than one actively engaged in his specialty could afford. What, then, is the remedy? To have the resident or assistant-resident study up all cases after the initial examination: such cases as are worthy to be frequently examined by the resident, and as soon as the least change is observed, the case to be again examined by the expert. Of course, unusually interesting cases would be kept under the direct observation of the expert.

Among those who have labored in this field may be mentioned Köstl and Niemetschek,[1] who examined 223 eyes of 134 insane patients; Köstl,[2] later, made a special study of epileptics; Sebaldi,[3] who examined 170 patients; Voisin and Galezowski[4] made examinations of 40 cases of general paralysis; Grafé and Westphal[5] examined 14 similar cases; Billod[6] is popularly supposed to have examined the fundus of paralytic patients, but only tested the acuteness of vision (finding total blindness in 3 out of 400 cases); Allbutt[7] reports examinations of 214 patients, of whom 51 were the subjects of mania; Jehn[8] reports examinations of 153 patients (36 of whom were examined by Prof. Saemisch and Dr. Mandelstaum), of which 17 were mania cases; Bouchut,[9] among his ophthalmoscopic examinations of 234 cases of cerebral disorders, includes epilepsy and idiocy, but reports no cases of mania; Bouchut and Dubuc[10] report 31 epileptic cases; Noyes[11] reports a total of 60 patients examined; Aldridge[12] reports examinations of cases of epilepsy, of general paralysis, and acute dementia; Klein[13] reports examinations of 134 patients; Dolbeau[14] reports 2 cases of general paralysis; Monti's[15] paper has been inaccessible; Schmidt-Rempler[16] examined 128 cases of insanity, in whom he found changes in but 13; Boy[17] examined 80 cases of general paralysis; Nettleship[18] ex-

amined several general paralytics; in the cases examined by Gowers,[19] he found changes in the eye-ground infrequently; Sebaldi,[3] in his examinations of idiopathic acute mania, found no decided change in the background of the eye, but yet found disturbances of the circulation in 75 per cent. of the cases; Allbutt[7] examined 51 cases of mania, and found in "25 symptoms of intracranial disease; in 13 cases it was of doubtful meaning, and in 13 no change at all was found, or only local changes, such as glaucoma, myopia, etc."

Among his 51 mania cases examined there are 21 cases of acute mania, and, from his description of the ocular condition, I have made the following table:

ALLBUTT.—ACUTE MANIA.

	Nearly Normal.	Ret. Hyper.	Ret. Cong.	Ret. Anæmia.	Papillitis.	Sl. Atrophy.	Atrophy.	Total.
Acute mania, 1st attack. . .	1	4	2	1 (during paroxysm)	2	2	1	13
Acute mania, 2d or subsequent attacks.	2	1	0	1	1	1	2	8
Totals.	3	5	2	2	3	3	3	21

As the result of his investigation, he concludes:

1st. That symptomatic changes in the eye are to be found in a large proportion of cases of mania.

2d. That if cases known to be functional only, or incorrectly named (such as erotomania, transient mania, hysteria, etc.), be omitted, the proportion of cases presenting permanent changes in or near the optic disc is still larger.

3d. That both in mania depending on organic causes and in functional mania, the back of the eye, if observed within a few days after a paroxysm, presents a vascular suffusion or pinkness so great after severe paroxysms as to obscure the disc. No exudation is seen in these cases unless there exists some permanent mischief.

4th. That during a paroxysm, on the contrary, the disc is anæmic, perhaps from spasm of the vessels.

5th. That the permanent changes in the disc are due either to stasis from obstruction to the intracranial circulation with consecutive atrophy, or to *ramollissement* ending in simple white atrophy, or they may present changes of a mixed character.

Jehn[8] reported ophthalmoscopic examinations of seventeen cases of mania, but gives the condition of the fundus in but two or three cases.

Noyes[11] reports nineteen cases of acute mania, of which he places thirteen as accompanied by retinal hyperæmia, and six as being either normal or having an anæmic retina. Upon analyzing his cases I formed the following table:

NOYES.—ACUTE MANIA.

	Nearly Normal.	Ret. Hyper.	Ret. Cong.	Papillitis.	St. Atrophy.	Atrophy.	Total.
Acute mania	2	6	8	1	2	0	19

Among Klein's[13] nineteen cases of mania he found four cases of retinitis paralytica, and one case of diffuse retinitis. In the other fourteen, while he found no special lesions, he yet found unusual circulatory disturbances, but in how many of these cases it occurred is not mentioned.

Of the several cases examined by Gowers,[19] in only one was there a pathological appearance—undue and uniform redness of the disc, with a distinctly softened edge.

It may be well to indicate here the method of work, and also the terms used to express the conditions found.

The eye-examinations have been made without a knowledge of the mental condition, which was, in fact, ignored. The examination included the taking of remote vision, testing the accommodation in about one half the cases. The lids, conjunctiva, cornea, sclera, iris, and lens were examined, and the tension taken. The ophthalmoscopic examination of the vitreous, nerve, retina, and choroid, with the measurement of the refraction, was usually made without a mydriatic, although in about one hundred and fifty cases an ex-

ception to this rule was made. All other facts concerning the ocular conditions present were noted. In the early part of the study the examination was now complete, but soon the value of an examination of the ear and throat became apparent, and for the last sixteen months has always been included.

In the tables the terms nearly normal, cataracts, iritic deposits, retinal hyperæmia, retinal congestion, papillitis and slight papillitis, slight atrophy, atrophy, choroiditis, retinitis hemorrhagica, retinitis albuminurica, retinal anæmia, and glaucoma, are all that are used. Each case is recorded but once, the two eyes not being recorded separately.

As *nearly normal* have been recorded all cases which did not present any noticeable feature in either eye.

Cataracts and *iritic deposits* are only recorded where a satisfactory ophthalmoscopic view could not be obtained.

Retinal hyperæmia expresses the presence of an increased amount of blood, with little or no effusion, while *retinal congestion* is limited to a very marked increase of redness and marked enlargement of the vessels, with effusion, with sometimes a peculiar irregular, perhaps to be termed granular, appearance (the retinitis paralytic of Klein is here included) of the retina.

The term *papillitis* is used only when the optic disc is swollen and hazy, where the outlines are indistinct, or where the disc, while but slightly raised or swollen, has an indistinct edge or is congested, the porus being more or less obliterated.

Slight papillitis sufficiently expresses itself.

Slight atrophy is but a convenience to express minor grades of atrophy, in many cases of which the vision is near normal; the color and margin of the disc, with the appearance of the vessels and of the porus, being sufficient to make the diagnosis.

Atrophy includes all cases not placed under the previous headings, in which we had degeneration of the nerve, with frequently a sharpness of outline or an increase in the extent of the porus and diminution in the size of the vessels.

The other terms used have their ordinary acceptation, and therefore need no explanation.

Up to April 12, 1886, 707 insane patients were examined at the Norristown and Philadelphia hospitals, of these 130 were males and 577 females.

The condensed table will serve roughly to indicate the class of patients examined and the results.

EYE EXAMINATIONS BY DR. LAUTENBACH AT INSANE DEPARTMENT PHILADELPHIA HOSPITAL AND AT NORRISTOWN INSANE HOSPITAL.

	Nearly Normal.	Cats. and Iritic Deps.	Ret. Hyper.	Ret. Cong.	Papillitis.	Sl. Atrophy.	Atrophy.	Choroiditis.	Hemor. Ret.	Album. Ret.	Anæmic Ret.	Glaucoma.	No. of Cases.
Mania (including monomania)	41	11	42	41	30	38	63	9	1	0	1	1	278
Melancholia	40	1	30	19	9	11	21	2	2	2	2	0	139
Dementia	33	9	12	1	5	30	93	4	1	0	0	0	120
All other cases, including epilepsy, general paresis, cerebral syphilis, imbecility, opium and alcoholic habits, moral insanity and cranks,	22	1	20	14	12	8	22	2	1	0	0	0	102
Totals	136	22	104	75	56	87	199	17	5	2	3	1	707

Of the 278 mania cases 105 were of the acute type; two cases have, however, been omitted, as no histories could be obtained.

The following table will serve to show the ocular conditions present in these 103 cases.

LAUTENBACH.—ACUTE MANIA.

	Nearly normal.	Cataracts.	Ret. Hyper.	Ret. Cong.	Papil.	Sl. Atrophy.	Atrophy.	Choroiditis.	Ret. Anæm.	No. of Cases.
Mania, acute, first attack.	3	1	8	13	10	10	3	1	1	50
Second or subsequent attacks	9	1	9	11	8	3	9	3	0	53
Totals	12	2	17	24	18	13	12	4	1	103

The following table will serve to compare these results with those of Allbutt and Noyes.

MANIA, ACUTE.

	Nearly Normal.	Cataracts.	Ret. Hyper.	Ret. Cong.	Papil.	Sl. Atrophy.	Atrophy.	Chor.	Ret. Anem.	No. of Cases.
Examined by Noyes . .	3	0	5	2	3	3	3	0	2	21
" " Allbutt . .	2	0	6	8	1	2	0	0	0	19
" " Lautenbach	12	2	17	24	18	13	12	4	1	103
Totals	17	2	28	34	22	18	15	4	3	143

It will be observed that in my observations on 101 cases of acute mania (excluding the two cases of cataract, as in these the background could not be satisfactorily examined) 11.82 per cent. were nearly normal, 16.83 per cent. had retinal hyperæmia, 23.76 per cent. retinal congestion, 17.82 papillitis, 24.75 atrophy, either slight or marked. These results are rather striking, but they are not markedly different from the results of Allbutt and Noyes, among whose cases 12.5 per cent were nearly normal.

As noted in the table, of these 103 patients 50 were first attacks. Not to occupy too much of your time, short histories of only these primary attacks will be presented. Before doing so it may be here noted that but two of our 103 patients have died; in neither has death resulted from the mental condition; in one a post-mortem was refused, in the other it revealed some fulness of the cerebral capillaries.

ACUTE MANIA CASES EXAMINED DURING THE FIRST ATTACK.

CASE 1. Female, aged twenty-eight years, married, in good health up to three weeks before admission, when, after three days of inflammation of the hand (said to be erysipelas, followed by palmar abscess), accompanied with intense pain, loss of sleep, and general debility, she developed acute mania. Symptoms in early weeks were active, but soon subsided into a condition of comparative quiet with moderate degree of intellectual derangement, from which she slowly recovered. Discharged one year and eight months after admission.

Ophthalm. exam. in eighth month of attack showed a condition of retinal congestion of both eyes with a large lymph deposit on the right nerve.

CASE 2. Female, nineteen, married. Has one child, born three weeks before admission. Previous health had been good. On the eighth day after labor, which was without complications, she had a chill, followed by fever, and on the ninth day developed maniacal symptoms of the most violent form. Admitted to the hospital on the sixteenth day of mania. Much reduced in flesh and strength; flesh bruised by throwing herself about. Vaginal examination revealed pelvic cellulitis. Kept in bed partially under influence of anodynes, with appropriate local treatment. All symptoms began to abate in two weeks. Discharged entirely well in three months.

Ophthalm. exam. in fourth week of attack showed an anæmic retina in both eyes.

CASE 3. Female, nineteen, single. Worked in factory, often did overwork, and had not always nutritious food. The physical exhaustion consequent upon this mode of life was assigned by her friends as the cause of the insanity. Admitted to the hospital on the third day of an attack of violent mania, which, after running a course of three months, terminated in recovery.

Ophthalm. exam. during the third month, when she was convalescing, and the mental symptoms had in great measure subsided, revealed retinal congestion and retained nerve-sheath of both eyes.

CASE 7. Female, forty-two years, single. Has lived without regular occupation; belongs to a family considered eccentric and highly excitable, but with no history of insanity.

At the time of admission was in a state of wild excitement. Insanity had existed for three months, beginning with melancholia and passing into mania, which had become uncontrollable only during the preceding week. After repeated exacerbations and remissions, the patient appeared to recover at the end of ten months and was discharged, but the disease has since *recurred.*

Ophthalm. exam. made in seventh month revealed atrophy of both nerves.

CASE 9.—Female, thirty-three years; married. Cause: ill-health. Admitted in sixth month of attack, of mild type. *Still in hospital;* has not recovered (twenty-seventh month). Prognosis unfavorble.

Ophthalmoscopic examination in sixth month, nearly normal; in twenty-seventh month, slight atrophy.

CASE 10.—Female, fifty years; widow. Cause: family troubles. Admitted in third week, mild type. *Still in hospital* (thirty-eighth month); now partially demented.

Ophthalmoscopic examination in eighth month, nearly normal; in thirty-eighth month, slight atrophy of both eyes.

CASE 12.—Female, thirty-two years; married. Insanity developed in third month of pregnancy; admitted in third month of attack, of mild type. Discharged, recovered (eighth month of attack); six weeks after labor.

Ophthalmoscopic examination in fourth month showed slight (syphilitic ?) atrophy of both eyes.

CASE 13—Female, twenty-seven years ; single. Cause : overwork. Admitted in third week. Recovered in one year, and no relapse since (three years).

Ophthalmoscopic examination in eleventh month showed retinal hyperæmia of both eyes.

CASE 14.—Female, nineteen years ; single. Cause : change of country. Admitted in second week. Recovered in six months.

Ophthalmoscopic examination in seventh week, retinal congestion of both eyes ; in sixth month, same condition, but less marked.

CASE 15.—Female, nineteen years ; single. Cause : measles. Admitted in fourth month. Recovered in seven months.

Ophthalmoscopic examination in tenth month, showed retinal congestion of both eyes. Re-examined five months after her discharge, when the same condition, but less marked, was observed.

CASE 17.—Female, twenty-four years ; single. Cause : ill-health. Admitted in third week. Recovered in five months.

Ophthalmoscopic examination in eighth week of attack ; showed retinal hyperæmia of both eyes.

CASE 18.—Female, eighteen years ; single. Cause : ill-health. Admitted in fourth month. Recovered in ten months.

Ophthalmoscopic examination in tenth month : retinal congestion of both eyes.

CASE 19.—Female, nineteen years ; single. No apparent cause. Mania of the hysterical type. Admitted in third week. Recovered in eight months.

Ophthalmoscopic examination in seventh month : retinal hyperæmia of both eyes ; slight papillitis, right eye.

CASE 20.—Female, twenty-eight years ; married. Melancholia, followed in three days by mania, came on eight days after labor. Recovered in five months.

Ophthalmoscopic examination in sixth week : slight atrophy of both eyes.

CASE 21.—Female, thirty-two years ; married. Father was insane before her birth. Recovered in six months.

Ophthalmoscopic examination in third month ; slight atrophy of both eyes.

CASE 23.—Female, nineteen years ; single. Cause : change of country. Admitted in fifth week. Great excitement with partial remissions. Recovered in nine months.

Ophthalmoscopic examination in seventh month (convalescent) : retinal hyperæmia of both eyes.

CASE 24.—Female, twenty-six years ; single. Cause : disappointment in love. Recovered in one year.

Ophthalmoscopic examination in seventh month : retinal hyperæmia of left eye. Re-examined four month after her discharge, and the same condition, slightly less marked, was observed.

CASE 26.—Female, seventeen years ; single. No known cause. Active manifestations of mania. Recovered in seven months.

Ophthalmoscopic examination in fourth week : papillitis, right eye.

CASE 27.—Female, twenty-one years; single. Cause: overwork. Admitted in third month. Now *in hospital* (seventh month); recovery doubtful.

Ophthalmoscopic examination in sixth month; papillitis, both eyes.

CASE 28.—Female, twenty-eight years; single. No known cause. Admitted in third week. Recovered in seven months.

Ophthalmoscopic examination in second month: slight atrophy of both eyes.

CASE 29.—Female, twenty-eight years; widow. Cause: overwork and anxiety. Admitted in third week. Recovered in six months.

Ophthalmoscopic examination in fifth month: retinal congestion of both eyes.

CASE 30.—Female, twenty-eight years; married. Cause: lactation. Admitted in sixth week. Now *in hospital;* prognosis doubtful.

Ophthalmoscopic examination in eighth week; slight atrophy of both eyes. Re-examination in seventh month (mental symptoms aggravated): slight atrophy with retinal congestion of both eyes.

CASE 32.—Female, seventy-five years; widow. Cause: family trouble. Recovered in six months.

Eye examination revealed cataracts of both eyes.

CASE 33.—Female, twenty-four years; married. Came on three weeks after labor, six months before her admission; and recovered one year later.

Ophthalmoscopic examination in sixth month: slight atrophy of both eyes.

CASE 37.—Female, twenty-seven years; married. Cause: ill-health. Developed melancholia four months previous to her admission and mania one month after admission. Now *in hospital;* prognosis favorable.

Ophthalmoscopic examination in fourth month, nearly normal; in fifth month, papillitis; in seventh month, papillitis less marked.

CASE 38.—Female, twenty-eight years; single. No known cause. Violent mania developed eight days before admission. Recovered in five months.

Ophthalmoscopic examination in first month, retinal hyperæmia of both eyes; re-examined ten months after recovery, same condition present.

CASE 39.—Female, twenty-three years; single. Cause: ill-health. Admitted in third month; recovered in eight months.

Ophthalmoscopic examination in eighth month (convalescent), retinal congestion of both eyes; re-examination twenty-eight months after recovery, retinal hyperæmia (both eyes) present.

CASE 41.—Female, twenty-five years; single. Cause: ill-health. Admitted in second month, recovered in three and a half months; but *recurred* one and a half years later, and died from the effect of hemorrhage (uterine).

Ophthalmoscopic examination in fifth week: papillitis of both eyes.

CASE 42.—Female, twenty-five years; single. Cause: overwork. A pronounced case of mania; now in fourth year, and has passed into a state of dementia. Still *in hospital.*

Ophthalmoscopic examination in eleventh month, slight atrophy of both eyes; subsequent examinations showed the atrophic condition more marked.

CASE 43.—Female, forty years; married. Developed four months after labor; now in twelfth month, and is still *in hospital;* prognosis is unfavorable.

Ophthalmoscopic examination in first month, papillitis of both eyes; in twelfth month, commencing atrophy of both eyes.

CASE 44.—Female, twenty-seven years; married. Came on five weeks after labor; now in fourth month of attack; still *in hospital;* prognosis favorable.

Ophthalmoscopic examination in fifth week, retinal hyperæmia of both eyes; in fourth month, same condition, less marked.

CASE 45.—Female, thirty years; married. Cause: overwork and anxiety. Still *in hospital* (tenth month of attack); prognosis unfavorable.

Ophthalmoscopic examination in fourth month, papillitis of both eyes; in tenth month, same condition, but less marked.

CASE 46.—Female, twenty-nine years; single. Cause: overwork. Remained in hospital for eight months without much change; then left, and has not been heard from.

Ophthalmoscopic examination in third week, papillitis of both eyes.

CASE 47.—Female, twenty-eight years; married. No known cause. Still *in hospital* (fourth month); prognosis unfavorable.

Ophthalmoscopic examination in third month: slight atrophy of both eyes.

CASE 48.—Female, twenty-five years; single. No known cause. Recovered in sixteen months.

Ophthalmoscopic examination in eleventh month: nearly normal, both eyes.

CASE 50.—Female, twenty-six years; single. Cause: phthisis. Recovered in seven months.

Ophthalmoscopic examination in first month: choroiditis, left eye.

CASE 51.—Female, twenty years; single. No known cause. Still *in hospital* (in tenth month of attack); prognosis unfavorable.

Ophthalmoscopic examination in second month, retinal congestion of both eyes; in tenth month, atrophy of both eyes.

CASE 52.—Female, twenty years; single. Cause: ill-health and family trouble. Recovered in five months.

Ophthalmoscopic examination in second month, papillitis, both eyes.

CASE 54.—Female, thirty-six years; married. Began on the

twelfth day after labor. Still *in hospital;* now, in third year of attack, in condition of chronic mania.

Ophthalmoscopic examination in sixth month, atrophy of both eyes; in third year, same condition.

CASE 55.—Female, twenty-five years; single. No known cause. Recovered in nine months.

Ophthalmoscopic examination in second week: retinal congestion of both eyes.

CASE 56.—Female, thirty-three years; married. Cause: mental shock. Now in second month of attack. Still *in hospital;* prognosis favorable.

Ophthalmoscopic examination on twelfth day: slight atrophy with choroidal congestion of both eyes.

CASE 57.—Female, thirty-five years; married. No known cause. Recovered in eleven months.

Ophthalmoscopic examination in third month: retinal hyperæmia of both eyes.

CASE 58.—Female, twenty-seven years; married. Began three weeks after labor. Recovered in three months.

Ophthalmoscopic examination in first month: retinal congestion of both eyes.

CASE 59.—Female, thirty-five years; single. Cause: overwork. Now in twenty-third month of attack; still *in hospital;* prognosis unfavorable.

Ophthalmoscopic examination in second month, retinal congestion of both eyes; in twenty-third month, slight papillitis of both eyes.

CASE 60.—Female, twenty-seven years; single. Cause: overwork. Still *in hospital* (thirteenth month of attack); prognosis favorable.

Ophthalmoscopic examination in eighth month, retinal hyperæmia of both eyes; in thirteenth month, same condition, but less marked.

CASE 93.—Male, twenty-five years; married. Cause: ill-health. Recovered in seven months, but has since *recurred* several times.

Ophth. examination in fifth month: papillitis, right eye.

CASE 94.—Male, forty years; single. Cause, intemperance. Now in thirty-fourth month of attack; still *in hospital;* in a condition of dementia.

Ophth. examination in twentieth month: slight atrophy, both eyes.

CASE 95.—Male, thirty-three years; married. Cause: irregular life. Attack came on twenty-eight months ago. He is still *in hospital*, the present diagnosis being general paresis.

Ophth. examination in eleventh month: retinal hyperæmia, both eyes, with atrophy of left eye.

CASE 96.—Male, twenty-seven years; married. Cause unknown. Recovered in fifteen months.

Ophth. examination in fourth month: retinal congestion, both eyes.

CASE 97.—Male, twenty-six years; single. Cause: intemperance. He is still *in hospital* (thirty-five months since commencement of attack), in a condition of dementia.

Ophth. examination in twenty-second month: retinal congestion, both eyes.

It will be observed that of the fifty cases twenty-eight recovered, and, so far as known, have not recurred; the termination of one case (46) is unknown. Three cases recurred, and of the eighteen still in the hospital the prognosis of four is favorable, of two doubtful, and of twelve unfavorable. Of the three patients in whom the disease recurred, two (41 and 93) presented papillitis, and one (7) a condition of atrophy. In the four cases with favorable prognoses we have retinal hyperæmia in two cases, in one (44) the disease is of four months' standing, and the retinal hyperæmia has lessened considerably during the course of the disease. The other case (60) is of thirteen months' standing, and the retinal hyperæmia has also moderated. Of the other two cases, one (37) is a case of papillitis which has become less marked, and the other (56) is a case of slight atrophy. Of the two cases of doubtful prognoses one, (27) is a case of papillitis, and the other (30) of slight atrophy. Of the twelve unfavorable cases, two (9 and 10) were nearly normal, but soon became slightly atrophic; three were accompanied with retinal congestion, one (51) becoming a case of atrophy, one (59) a case of slight papillitis, and one (97) remaining in the condition of retinal congestion; two (43 and 45) were accompanied with papillitis, three (42, 47, and 94) with slight atrophy, and two (54 and 95) with atrophy.

Among these twenty-one cases slight atrophies appeared eight times during the course of the disease, atrophies four times, and papillitis seven times.

Of the twenty-eight cases which recovered, one (48) was nearly normal; one (32) was a cataract case; six (13, 17, 23, 24, 38, and 57) were cases of retinal hyperæmia, while ten (1, 3, 14, 15, 18, 29, 39, 55, 58, and 96) were cases of retinal congestion; three (19, 26, and 52) were cases of papillitis (case 19 being classified as slight); five (12, 20, 21,

28, and 33) were cases of slight atrophy, while one (2) was a case of anæmic retina, and one (50) of choroiditis.

The case (46) of which the result was not known was a case of papillitis.

It will perhaps be interesting to contrast the eye conditions in the recovered cases with the changes in the recurred and in those who have not recovered.

	Nearly Normal.	Cataracts.	Ret. Hyp.	Ret. Cong.	Papil.	Sl. Atr.	Atrophy.	Anæm.	Ret. Chor.	No. of Cases.
Recovered cases.	1	1	6	10	3 (1 of sl. pap.)	5	0	1	1	28
Recurred and not recovered cases	2	0	2	3	6	5	3	0	0	21
Result unknown					1					1

The frequency of hyperæmia and congestion in the favorable and recovered cases is rather striking, so also is the absence of marked atrophy. In the other cases:—those which either recurred or are unfavorable, present rather a large number of cases of papillitis and atrophy.

We accepted the facts, refrained from drawing conclusions, have hoped that as our cases grew in number our knowledge would become broader, and that conclusions might develop unconsciously. In endeavoring to draw conclusions we have a difficulty with which to contend:—the undoubtedly various bases of the symptoms of mania; perhaps in one case due to an abnormality of structure, a want of development; perhaps, again, a localized congestion, or even a degeneration, or perhaps an irritation nucleus occasioning vascular disturbances by reflex action; or, again, a general vascular engorgement, or one of various diseases acting through some change in blood pressure or in the constitution of the blood or by reflex action.

A few facts have developed:—for instance, we feel that if in acute mania hyperæmias or congestions only are present, the case is apt to terminate favorably; that if papillitis be present, the chances are decidedly lessened; that this is also the case if marked atrophy be present; that if a condition of retinal hyperæmia be present, in addition to a slight

atrophy, the chances of recovery are decidedly more favorable than would be the case were the latter alone present. These general truths we believe to be evident to all who will consult our last table. That, in addition, many inferences have been unconsciously suggested during the course of this work, must be evident to all; but it is better to wait until the work is older, and the list of cases longer, and allow the inferences or theories to develop into truths before presenting them.

[1] *Prager Viertelj*, 1867, Bd. 95, p. 134.

[2] *Prager Viertelj*, 1870, Bd. 106 and 107.

[3] *Revista Clinica*, 1870. *Nagel's Jahresbericht für Ophthal.*, 1870, p. 374. *Archiv für Psychiatrie*, 1871, iii., p. 228.

[4] *L'Union méd.*, vol. xxxi., 1866, p. 404. *Schmidt's Jahrbücher*, 1869, Bd. 141, p. 79.

[5] *Archiv für Pschiatrie u. Nervenkr.*, 1868, i., p. 44.

[6] *Annales méd.-psychol.*, 1863, ii., p. 317.

[7] "Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys," 1871, p. 371.

[8] *Allgemeine Zeitschrift für Psychiatrie u. s. w.*, vol. xxx., 1873–4, p. 519.

[9] "Du diagn. des malad. du syst. nerveux par l'ophthalm.," 1866, Paris."

[10] *Prager Viertelj*, 1865, Bd. 85, p. 102.

[11] *Amer. Journ. of Insanity*, 1872, vol. xxviii., p. 410.

[12] *West Riding Lunatic Asylum Reports*, vol. i., p. 71; vol. ii., p. 223; vol. ix., p. 291.

[13] *Wiener med. Presse*, 1877, p. 89.

[14] *Archiv für Ophthalm.*, Bd. ii., p. 1. *Gaz. des hôpitaux*, 1866, No. 48.

[15] "De l'ophthalm. dans les malad. mentales."

[16] *Annales d'oculistique*, 1875, vol. lxxiv., p. 267.

[17] *Thèse de Paris*, 1879.

[18] *Ophthalm. Hosp. Reports*, vol. ix. p. 178.

[19] "Medical Ophthalmoscopy," 1882, p. 176.

Periscope.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

Upon the Character of the Muscular Contractions Which Are Evoked by Excitation of the Various Parts of the Motor Tract. *Jour. of Phys.*, vol. vii., No. 2.

Profs. VICTOR HORSLEY and SHAEFER have made a series of experiments upon this subject with dogs, cats, rabbits, and monkeys. Their results were as follows:

1. The rhythm of muscular response to electrical excitation of the nerve-centres is the same whether the excitation be applied to the gray matter of the cerebral cortex in the motor region, or to the fibres of the corona radiata emanating from that region, or to the spinal cord (but not to the peripheral motor nerves). 2. The rhythm of muscular response in the case of voluntary and reflex contractions is essentially the same as that which results from electrical excitation of the nerve-centres. 3. The rhythm of muscular responses in all cases of after-excitation (whether distinctly epileptoid in their nature or not) is fundamentally the same as that of voluntary and reflex contractions and of contractions immediately produced by a rapidly recurring electrical excitation, but the response in the case of epilepsy may present a secondary rhythmic summation which produces a clonus of a slower rate. The principal conclusion from their results, which have been supplemented by numerous observations on voluntary and epileptoid contractions in man, seems to be this: That every prolonged contraction of the skeletal muscles which is provoked by excitation, whether natural or not, of any part of the nerve-centre, is a tetanic contraction which has been produced by a series of impulses generated in the nerve-centres and passing along the motor nerves at an average of about ten per second.

As to the place of the generation of this rhythm, it is certain that in some cases it occurs in the lower nerve-centres—that is, in the motor-nerve cells of the spinal cord, medulla oblongata, pons, and mesencephalon. At least this conclusion appears to follow from the fact that when we excite the motor tract above those centres by a rapidly interrupted electrical stimulation, the excitation manifests itself by a muscular response which has a rhythm

of only ten per second, whereas it is certain that the rapid excitation of the nerve fibres of the motor tract must have caused equally frequent nervous impulses to pass along those fibres. It is clear, therefore, that these rapidly succeeding impulses have not been transmitted unaltered through the motor-nerve cells, but have become summated within them and converted into a smaller number of impulses, which are then forwarded with a constant slower rhythm by the peripheral motor-nerve fibres to the muscles.

The Rhythm of Muscular Responses to Volitional Impulses in Man. *Jour. of Phys.*, vol. vii., No. 2.

Prof. SCHÄFER and Students CANNEY and TUNSTALL have investigated this subject. They usually employed the opponens pollicis. Their results were as follows:

1. A prolonged voluntary contraction in man is an incomplete tetanus produced by from eight to thirteen successive nervous impulses per second. About ten per second may be taken as the average.

2. The average rate of muscular response to volitional impulses is approximately the same in man as in other mammals that have been examined.

3. The average rate of muscular response to volitional impulses in man is approximately the same as the average rate of muscular response to rapidly recurring excitation of the nerve-centres in animals.

4. The average rate of muscular response to volitional stimuli in man is approximately the same as that obtained in man and animals as the results of pathological or other excitation of the cortex cerebri producing epilepsy, although in the latter case the impulses tend to undergo summation and thus to cause the appearance of clonic contractions of slower rhythm.

5. The rate of muscular response to volitional stimuli in man is nearly the same as the rate of muscular response which is due to activity of the spinal cord alone.

Olfaction. *Du Bois' Archiv*, 1886, Heft 394.

Herr ARONSOHN has made a long series of experiments upon this subject in man. He found that the olfactory nerve was completely blunted for a time through the uninterrupted action of an adequate irritation in the course of a few minutes.

2. Completely exhausted olfactory nerves need at least a minute for complete recovery.

3. Different kinds of smells affect different territories of the olfactory region; one territory is excited to a maximum degree, a second territory in a lower degree, and a third territory not at all.

The Electrical Discharge of the Malapterurus Electricus. *Jour. of Phys.*, vol. vii., No. 2.

Mr. GOTCH has experimented with a fish brought from the river

Senegal, and he found that the discharge on electrical excitation of the skin is not of a reflex character, but is the result of direct excitation of the electrical organ at the point excited.

2. That the latency of the organ under these conditions is extremely short.

3. That the excitatory state is propagated through the organ at a rate of about two and a half metres per second.

ISAAC OTT, M.D.

The Cerebro-Spinal Paths of Centrifugal Conduction.

Lo Sperimentale, Marzo, 1886.

Experiments upon dogs have been made at the Physiological Laboratory of the Royal Institute at Florence by Drs. IRO NORI and DARIO BALDI, with a view of settling the direction taken by impulses travelling outward from cerebral centres. They give an interesting review of the work already done by Franck and Pitres and others, and a detailed account of their own experimentation, of which the following is a *résumé:*

1. After an excitation of the motor zone of the cortex, if the stimulus be very slight, there follows always a crossed unilateral movement; if the stimulus be stronger, there follows a bilateral movement which may be diffused to all four of the extremities.

2. When a bilateral movement occurs, the first to contract are the muscles of the part or parts opposite, and then the muscles of the part or parts of the same side as the excitation.

3. When spinal hemisection has been performed, the phenomena become inverted, so that the cortical excitation of the opposite side to the hemisection gives a movement more pronounced in the muscles of the same side as the hemispheric excitation, which then is diffused to the muscles of the opposite side corresponding to the hemisection.

4. In a dog in which one of the sigmoid gyri has been extirpated, when the other is excited the movements in the opposite side to the extirpated gyrus are much weaker than those on the same side as the excitation. Then if spinal hemisection is performed at a point corresponding to the last dorsal vertebra on the same side as the ablation of the cortex, and then the motor zone remaining be excited, they lessen, but not entirely, the movements on the same side as that to which the excitation is applied.

5. In a dog with the corpus callosum excised, bilateral movements are possible; those of the same side as the excitation are scarcely appreciable. Hemisection of the cord in this case, by almost complete abolition of the movements resulting in the two extremities of the same side where the excitation is made.

6. In a dog in which there has been a division of the posterior roots on one side which go to make the crural and sciatic nerves, the forms of movements in the two posterior extremities are different if the excitation is applied to the side corresponding to the divided roots.

From these facts they conclude that in the centrifugal transmission of excitations from the motor centres of the cortex of the cerebral hemisphere of a dog, there exists not only transverse interspinal paths of conduction, but also transverse intercerebral paths of conduction. GRACE PECKHAM.

PATHOLOGY (INCLUDING PATHOLOGICAL ANATOMY) OF NERVOUS SYSTEM.

Senile Changes in the Brain. KOSTJURIN. *Wiener Med., Jahrb.* 1886, Heft 2.

Kostjurin has examined a number of brains of old persons in order to determine the histological changes which present themselves in the various tissues in simple senile atrophy. He finds that the greater number of nerve cells in the cortex undergo a more or less marked degeneration with the production of pigment, and fat, and occasionally with the development of vacuoles. In the pericellular space numerous round cells are found in addition to the mass of detritus when the cell is entirely degenerated. The nerve fibres in the cortex are atrophied or reduced in number. The vessels of the cortex undergo an atheromatous degeneration, with the constant production of a connective tissue thickening in their walls. This may become so great as to obstruct the lumen of the vessels. The pigment deposit in the adventitia of the walls is also increased. In the place of the nerve-cells and fibres which have disappeared by atrophy there is found a slight increase in the neuroglia. On the surface of the cortex a large number of corpora-amylacea are found which may form a continuous layer covering the convolutions.

The intensity of these dangers depends less upon the age of the patient than upon the relative degree of loss of weight in the brain, so that they are more evident in light brains than in very old brains. They are sufficient to account for the mental symptoms of senility observed.

The Gulstonian Lectures on Spasm in Chronic Nerve-Disease. By S. J. SHARKEY. *British Medical Journal*, 1886, March and April.

By Spasm is meant excessive muscular contraction in defiance of the will or in excess of the intention. It may be persistent, the individual waves of contraction overtaking each other; intermittent, the individual waves succeeding each other with or without regularity; or co-ordinated, in which case groups of muscles are the seat of clonic spasms which are co-ordinated in such a manner as to produce some regularly recurring though involuntary movement. The motor mechanism through which all movement is produced consists of a cerebral part, viz.: the motor area of the cortex, and the pyramidal tract; and a spinal part, viz.: the an-

terior cornua of the spinal cord and the motor nerves. These parts originate and transmit all efferent impulses concerned in movement. Spasms may be produced by disturbances of the cerebral or of the spinal mechanisms, and by fine molecular changes which cannot at present be limited to any particular part of the motor tract.

I.—Spasm in connection with cerebral mechanisms.

Any injury to the cerebral motor tract between its cortical origin in the central convolutions and its termination in the anterior cornua of the spinal cord produces a degeneration in it below the seat of injury. This is followed by a tonic spasm in those muscles which are under the control of the injured fibres; increased tendon phenomena and rigidity being evidences of the increased muscular tone consequent upon the loss of cerebral control over the spinal centres. The partial or complete suppression of nerve impulses passing from the cerebral motor centres down the pyramidal tract may occur under a variety of circumstances, *e. g.*, acute fevers, anæmia, external debility, as well as organic disease. Its usual cause is a destruction of some part of the cerebral motor tract; *e. g.*, softening of the central convolutions in whole or in part, division of the internal capsule, sclerosis of the lateral column of the spinal cord. In these cases contractures are common. When the motor tract is pressed upon rather than destroyed tumors of a rhythmical kind appearing on voluntary movement are produced; and if the pressure produces finally a destruction of the tract, rigidity and paralysis supervene in the tremulous limbs. If the disease is within one hemisphere the symptoms are unilateral, but when it is situated between the basal ganglia and the medulla they are frequently bilateral. Bilateral tremor followed by paralysis and rigid contraction which finally become permanent, and succeeded in the last stage by occasional general tetanic spasms indicate in all probability a hard basilar tumor producing gradual pressure on the motor tracts where they lie in close proximity to one another. The diagnosis will be strengthened by the development of optic neuritis, and by paralysis of the cranial nerves. Since all these symptoms, including those of contracture and tetanic seizures, may be produced by pressure upon the motor tracts at any part of their course, it is no longer permissible to consider them as characteristic of cerebellar tumor as Hughlings Jackson supposed. They only occur in cerebellar disease when this is situated in the middle lobe and exerts pressure downward upon the pons and medulla—for where the lobe is destroyed by softening, no pressure on adjacent parts being produced, rigidity and spasms may be wholly wanting.

A number of interesting cases with records of post-mortem findings were cited to illustrate these points. Defective development of the brain involving the motor region and attended by congenital deficiency of the pyramidal tract, results in the condition of spastic hemiplegia, the mere absence of the controlling impulses, which in healthy subjects traverse the pyramidal tract,

being capable of giving rise to a spastic condition of the limbs. This condition, known as infantile spasmodic paralysis, may be unilateral or bilateral. Under these circumstances the spasm may be either mobile or fixed. If it is mobile the condition may be one of athetosis, rhythmical continuous movements of the fingers and hands or legs and toes, being kept up constantly; or the movements may resemble those of chorea, disseminated sclerosis, ataxia, or paralysis agitans. All the varieties of mobile spasm seem to result from a mixture, in varying proportions, of paralysis, spasm, and irritation, and their development depends upon lesions which interfere with the perfect functions of the motor centres and fibres, but which do not completely interrupt them. They may be situated in any part of the motor tract, and the variety of the movement does not depend upon the seat of the lesion, but rather upon the condition of the tract below the lesion. While the destruction of the basal ganglia has been attended by such spasms they are to be ascribed rather to an implication of the motor tract in the internal capsule than to the disease of the ganglia, since the latter may be entirely latent. A case was related of total destruction of the caudate and lenticular nuclei on the left side, with descending degeneration in the inner third of the crus and in the pons, but no changes whatever in the medulla or cord. In this case two attacks of hemiplegia had occured, from which the patient had recovered completely several years before his death, no paralysis or rigidity remaining. Our present knowledge of anatomy, physiology, and pathology does not justify us in concluding that there is any efferent motor connection between the brain and the spinal cord except the pyramidal tract, and this has no connection with the cerebellum or basal ganglia, and its fibres are nowhere interrupted by entering ganglionic centres, but pass straight to their several destinations in the anterior cornua. If the tract be interrupted at any point in its course in the spinal cord all the spinal centres which are under voluntary control below the point of interruption will be thrown into a state of hyperphysiological activity. The symptoms of disease in the lateral columns of the cord are the same whether it follow cerebral or spinal lesions. They differ somewhat according as the pressure made upon the lateral columns is from without inward or from within outward. When a tumor grows in the spinal canal outside the cord, it may produce but few symptoms until it presses the cord against the resisting walls of the canal; but after this has taken place the course of the disease is very rapid, as the cord is quickly flattened by the constantly increasing demands for growing space which are made by the tumor. When a tumor arises, on the other hand, within the spinal cord, it disturbs its functions even from the very commencement; but as the nerve substance is elastic and allows gradual stretching without serious interference with its functions, a tumor may go on growing for a long time before it produces striking pathological phenomena, either by pushing the cord

against the bony walls of the spinal canal, or by exhausting the elasticity of the membranes which envelope it. In a case of tumor pressing upon the upper dorsal spinal cord from without the symptoms were in the order of time, weakness of the legs in walking; increasing weakness of the legs, accompanied by cramp and shooting pains; paraplegia, cramp, and involuntary flexing of the legs; slight numbness and long-continued attacks of muscular spasm in the lower extremities, and finally persistent rigidity. In a case of tumor within the cord, in the cervical region, the symptoms were partial loss of power and sensation in the arms and hands, and some tremor in them when used; not till twelve months after the beginning of the symptoms were numbness of the legs, loss of power, and incontinence of urine, with exaggerated reflexes, observed; and these were referred rather to a hemorrhage in the dorsal region than to the tumor in the cervical region. The contrast between the two cases is striking. Primary lateral sclerosis characterized by weakness of the limbs, tremulousness, jerkings, and rigidity is easily recognized if it is not complicated by sclerosis in other tracts than the pyramidal columns. When the posterior columns are also involved the symptoms may be obscured by those of locomotor ataxia.

II.—Spasm in connection with spinal motor mechanisms.

By the spinal system it is intended to include all the nerve centres with their efferent and afferent nerves which occur between the central ganglia of the brain and the termination of the spinal cord. (1) Spasm produced by diseases of the efferent spinal nerves. In the majority of cases where muscular spasm causes distortion it is the healthy muscles which are actively concerned, while the diseased muscles are for the most part passive, *e. g.*, main en griffe, of progressive muscular atrophy; deformities of pachymeningitis cervicalis. The rigidity occuring in alcoholic paralysis is of this kind, the healthy muscles overcoming their weakened opponents, which are usually the extensors. The same is occasionally seen in lead palsy. In all such cases the existence of normal electric reactions in the rigid or contracted muscles reveals the true condition present. In some cases, however, it is the shortening of diseased muscles which produces distortion. Here the diseased muscles are rigid; when attempts are made to extend them passively they do not relax under anæsthesia, and they have usually lost their electric contractility. Chronic muscular spasm is quite the exception in peripheral nerve disease, although occasional spasm may occur from this cause. Direct irritation of motor nerve filaments rarely causes contractions in the muscle, though it is often severe enough to produce paralysis. In this connection Weir Mitchell's opinion was quoted, viz.: "The symptoms in disease of the peripheral nerves affect at first rather the sensory sphere than that of motility. We have pain and anæthesia; or hyperæsthesia, but not, as a rule, convulsions. In certain cases the nerve wound, in place of causing primary loss of motility, occasions either sudden muscular contraction, followed by instant

loss of power, or in very rare instances, long-continued spasm. Tonic contraction of muscles are occasionally met with at a later stage of these injuries, but are perhaps amongst the rarest of the secondary symptoms."

(2) Spasm produced in a reflex manner by disease of afferent nerves. Such spasm is to be referred to a hyperphysiological activity in the spinal centres rather than wholly to the stimuli applied to the afferent nerves. Muscular contraction of a reflex kind varies much in degree in different individuals and in different states of health, although the exciting stimulus may be the same. Hence it is probable that the injury which supplies the stimulus to the sensory nerve in these cases though apparently the principal agent, is really so in many instances only from a particular point of view. Had the same stimulus been applied to the same nerve when connected with a healthy and stable centre no spasm would have ensued. Reflex spasm no doubt occurs, but how frequently it does so, or how far the afferent or efferent nerves or nerve-centres take the leading part in its production are points which can scarcely be estimated. The condition is reflex when sudden pain in a part succeeds spasm. Persistent reflex spasm may be due to irritation from joint disease, as Charcot has shown, the joint being rigid until the patient is anæsthetized. The muscles normally contract slightly when they are stretched by voluntary contraction of their opponents. This reflex contraction may under certain conditions—especially overexertion—become excessive, giving rise to sudden movement of the limb in a direction just opposite to that intended. This is very probably the cause of the tremor in the direction of the movement seen in disseminated sclerosis. It often follows sprains, in which certain muscles are overstretched. Interesting cases illustrating this condition were related. What is really developed in these cases is an involuntary neuro-muscular habit, and it is probably associated with an abnormal state of the nerve-centres as well as with disease of the afferent nerves. Hence it often yields to the use of bromides.

III.—Functional spasms may be referred to molecular changes, not visible by the aid of microscope, in either the cerebral or the spinal parts of the motor tract, and therefore they can be classified in the same manner in which spasms from organic disease have been. (1) Functional disorder of the cerebellar-motor mechanism is seen in hysteria, hemispasm, either fixed or mobile, being not infrequently observed. There is this difference between the hysterical conditions and those which are seen in gross disease : the leg is affected most, the arm less, and the face not at all. Apart from this the spasms are similar. In functional diseases there is simply removal of the voluntary impulses which in health pass down the pyramidal tract. The muscles and nerves remain practically healthy, but rigidity and increased tendon reflexes are present, just as in those cases in which there is a congenital defect of the motor tract. Absence of voluntary impulses is enough to give rise

to the phenomena in question, and hence it is legitimate to conclude that suppression of the functions of this tract is the cause of functional hemispasm. Monospasm, athethosis, and allied mobile spasms which occur in hysteria are to be explained in the same way.

There is another class of functional spasms which, I believe, have their origin in suppression of the functions of certain portions of the pyramidal tract, viz.: professional hyperkineses, *e. g.*, writer's cramp, pianist's cramp, etc. When a voluntary effort is made to perform a certain act, spasm of the muscles involved occurs, and prevents further effort. Before this stage of the affection is reached, great fatigue often accompanies endeavors to work. What has occurred is probably that after long-repeated acts of the same kind, that part of the pyramidal tract which is used becomes fatigued, and its functions are partly suppressed, so that a condition of "latent contracture" of the muscles over the voluntary actions of which it presides is developed ; the lower centres are "let go," as Jackson says, and are in a state of hyperphysiological activity.

(2) Functional disorder of the spinal motor mechanism is also observed. Of course deformities due to atrophy of some muscles, and contracture of their opponents, or to shortening of diseased muscles, cannot be called functional, nor should we expect to find that irritation of the afferent or efferent nerves would give rise to functional spasms, since it rarely causes spasm when it goes far enough to produce gross changes in the nerves. The nerve-centres of the cord may, however, develop a state of hypersensitiveness and of hyperkinesis without gross lesions, and hence functional spasms from this cause are not rare. There can be no question that one of the peculiarities of nerve-centres in hysteria is their abnormal irritability, so that slight afferent impressions give rise to muscular acts which pass with great ease into neuro-muscular habits. Such a condition is well exemplified in cases due to irritation. The patient sees a certain form of muscular spasm, and the idea produces the same in her. Interesting cases of this affection were related. Persons may have those peculiarities of their nervous system which are usually embraced under the term hysteria, without ever presenting striking emotional or other tendencies which are wont to call attention to the existence of the disorder. The first evidence of the latter may be the sudden supervention of spasms from a very slight external injury. In any case, the more the cerebral control retires to the background, the more likely are spasmodic contractions to come to the fore-ground. Hyperexcitability of the spinal-centres without a diminution of cerebral control occurs under the use of strychnine ; hence it is not impossible that molecular changes may produce the same state, giving rise to spasms on any voluntary action.

In the comparison between the results of gross disease on the one hand, and molecular, or so-called functional alterations, on the other, the pyramidal tract stands prominently forth as the

great offender in the production of muscular spasm. Its action is indirect, it is true, as it only loosens the reins of the spinal centres, which it should keep well in hand. Still it rules the situation. Spasm rarely ensues directly from injury or disease of the peripheral motor-nerves, and, although it frequently results from reflex causes, it is very likely that the spinal-centres are, in a considerable proportion of cases, more at fault than the afferent nerves. Considering how late the pyramidal tract develops in man, and what a high pitch of evolution it represents, it is not to be wondered at if it is one of the first parts to suffer in the process of dissolution. It is important to remember that exhaustion in one area of the motor system seems to affect the whole system, disturbing that equilibrium which is necessary to normal action.

M. A. STARR.

On Hysterical Anuria with Secretion of the Urine by the Stomach, and Experimental Researches Made in Hysterical Anuria in Relation to Uræmia. By Dr. EUGENIO ROSSONI. *Rivista Clinica*, October and November, 1885.

The writer gives the report of two extreme cases of hysterical anuria in which the most careful observations were made. In one of these the patient vomited a fluid which in every way resembled the urine, which at times corresponding to the menstrual epoch, the menses being absent, was tinged with blood. Space would be wanting to go into the details of this extremely elaborate article, but we give the conclusions arrived at, including the results of experimentations with pilocarpine and urea. Anuria is a phenomenon which is undoubtedly more often manifested in hysteria than is generally believed. Hysterical anuria is found in a peculiar state of the physical organism, which may be recognized by a retardation and limitation of the general nutrition, together with a state of neurasthenia of the nerve-centres, more especially those which influence the secretory activity of the kidneys.

An hysterical person with anuria may live a long time without manifesting grave uræmic symptoms, this depending on the morbid condition of the general nutrition, and not of necessity giving rise to vomiting, to compensate for the suspended functions of the kidneys. This vomiting may be lacking entirely.

Not every kind of vomiting which may be manifested during hysterical anuria should be believed to depend on the anuria.

In hysterical persons, with suspension of the function of the kidneys, may be found in their stomachs a fluid more or less abundant, which has all, *ad literam*, the physical as well as the chemical properties of normal human urine.

The activity of this urine-like secretion does not stand in inverse relation to that of the kidneys.

The urine-like secretion of the stomach may be suspended for a time more or less short, without a contemporaneous return of the renal secretion, which may remain absent a considerable time longer.

The existence of hysterical anuria should not be denied when, in a given case, vomiting of urinous fluid is lacking.

Given a case of hysterical anuria in which the functions of the kidneys may be suspended, pilocarpine will bring about a temporary activity.

Pilocarpine may have an active effect in exciting renal secretion of human beings.

In some hysterics with anuria pilocarpine may determine the secretion of a saliva having all the physical and chemical characters of normal human urine.

Urea introduced artificially into the circulation in an hysterical patient with anuria and without secretion of urine from the stomach, acts perniciously—determining uræmic attacks.

Urea by itself may contribute to the development of uræmic symptoms; may be introduced artificially into the system, in quantities of sixteen grams in a short time, in hysterical subjects with anuria, if there is active urinary vomiting.

There does not exist any identity between hysterical anuria and the uræmia of nephritis and of animals with extirpated kidneys or ligated ureters. GRACE PECKHAM.

A Contribution to the Doctrines of the Innervation of Movements of Expression. By Dr. P. ROSENBACH, of St. Petersburg. *Neurol. Centralbl.*, 1886, No. 11.

It is a well-known fact that in facial paralysis of central origin, muscles which cannot be moved by voluntary effort, may contract in obedience to reflex impulses. By a comparative study of clinical and pathological data, Nothnagel concludes that this condition could exist only in case the optic thalamus and the fibres of the corona radiata joining the thalamus and the hemisphere had not been interfered with. Nothnagel's views have been corroborated by recent investigations of Bechterew. Rosenbach now reports a case in which the ordinary condition of things is reversed, for in this instance the facial paresis is not made evident until the patient begins to laugh.

The patient, a women æt. thirty six, had an attack of left hemiplegia (sudden onset without loss of consciousness) ten months ago. When the facial muscles are at rest, there is only the slightest trace of paresis of the lower facial muscles. In speaking, both halves of the face can be moved equally well. The left half of palate is lower than right half. When the patient attempts to laugh, *the left half remained altogether passive*, the left naso-labial fold disappeared, and the mouth was drawn to one side. The examination revealed also *left bilateral hemianopsia*, anæmia, and organic cardiac lesion. The occurrence of hemianopsia, together with the loss of movements of expression, leads the author to locate the lesion in the optic thalamus. No autopsy; and yet we think the auhor was justified in publishing the clinical data.

Tabes with Presence of Knee-Jerks. Discussion in the Berlin Society for Psychiatry and Nervous Diseases. After a report in *Mendel's Neurol. Centralbl.*, No. 10, 1886.

Westphal read a paper on cases of tabes dorsalis in which the knee-jerk was not at all times absent.

Bernhardt observed that he also had seen such cases, and referred furthermore to a case of disseminated cerebral tumors, in which the knee-jerks were absent. Mendel found the knee-jerk absent in the earlier stages of tabes, and either on one side or on both. He called attention to a variety of cases in which the knee-jerk is not obtained, and instanced a case of tumor of the cerebellum in which the knee-jerk was absent, without any changes having been found post-mortem in the spinal cord. The reason of this loss of patellar tendon reflex, he is not able to explain.

Thomsen referred to ten cases of cerebro-spinal and tubercular meningitis, in five of which the knee-jerk was absent; the spinal cord was examined in all these ten cases, but no histological changes were discovered. Thomsen thinks that in such cases the peripheral nerves should be examined. Westphal states that he never believed the knee-jerks to be absent in tabes only. This symptom occurs in cerebral diseases as well, in which there is considerable diminution of muscular tone. Jendrassiks' method of eliciting the knee-jerk can be explained by the fact that increased (involuntary) innervation of all the muscles, including the *quadrieps femoris*, increases the tone of the muscles, and can thus excite the knee-jerk, which could not be obtained while the leg is at rest.

Contributions to the Pathology of Tabes. OPPENHEIM and SIEMERLING. Read at the Berlin Soc. for Psy. and N. D., and reported in *Neurol. Centralbl.*, 1886, No. 11.

At the instigation of Prof. Westphal, the authors made a careful examination of the condition of the peripheral nerves in cases of tabes dorsalis. In order to have a reliable basis for comparison, they examined the peripheral nerves in a large number of individuals who had died of other than nervous diseases. The result of this study was a conviction that lesser degrees of degeneration occur in cases of infectious or toxic disease, and as the result of a general marasmus. Extreme nerve-degeneration, however, was observed only in cases of multiple neuritis, either of tubercular or alcoholic origin.

In the majority of cases of tabes dorsalis, the sensory nerves and their branches had undergone excessive changes, such as were found in no other cases but those which had presented the clinical symptoms of neuritis. This excessive degeneration was found (in seven cases of tabes) in the branches of the N. saphenus major, in the smalll branches of the peroneal which supply the skin of the toes, and in the digital branches of the ulnar nerve. Similar changes were noted in the X., and in the recurrent laryn-

geal nerve of persons who had been afflicted with gastric and laryngeal crises. In every instance the changes in the sensory nerve were found to be in excess of those in the larger branches of peripheral mixed nerves.

The authors call attention to a special form of degeneration in many cases of tabes. This was as follows: On cross-section the perineurium is seen to be considerably thickened; the nuclei very numerous. Between the perineurium and the nerve-fibres there is a vascular tissue. The blood-vessels in this layer of tissues are for the most part obliterated, and their walls have undergone sclerotic changes. The blood-vessels are so numerous that the nerve-substances appears to be surrounded by them. This condition represents an interstitial neuritis or perineuritis. This perineuritis was observed in persons in whom tabes was complicated by other diseases.

The posterior roots and the spinal ganglia were diseased in some cases of tabes. As for the connection between the disease of the posterior columns and the degeneration of the peripheral nerves, the authors deny any connection of the sort, either condition not being necessarily accompanied by the other. It is but one step further to infer that the sensory disturbances, including ataxia, and the phenomenon of delayed sensory conduction, are due to these changes in the peripheral nerves. The changes in the vagus and recurrent laryngeal will account for the gastric and laryngeal symptoms. It is worth adding that the nuclei and roots of the vagus, and the post. longit. fasciculus of the oblongata, were normal in cases in which the peripheral affection existed. With regard to the condition of the spinal cord, the authors only report that they have frequently observed atrophy of nerve-fibres in the columns of Clarke. The usual changes are not referred to. B. S.

The Effect of Extirpation of the Ovaries upon Nervous Symptoms. SCHMALFUSS. *Arch. fur Gynæcologie*, xxvi., 1.

In a valuable article upon this subject the author sums up the results obtained in Hegar's clinic at Freiburg. Only those cases of extirpation of the ovaries are considered in which nervous symptoms had predominated, and which had remained for some time under observation after the operation. Anatomical changes were discovered in the large majority of the ovaries cut out. Prior to the operation in all cases a long course of general treatment had proved unavailing. The thirty-two cases reported are divided into three groups. In the first group are placed cases in which the nervous symptoms had been confined to the sacral and lumbar plexuses and their distribution. In the second group cases are placed in which other nerve regions had been affected. In the third group the cases of general neurotic condition with symptoms of reflex disturbance and neurasthenia are classified. In all these cases, except two, a decided diminution in the num-

ber and degree of the nervous symptoms followed the operation. In twenty-four cases an entire cure resulted; all the nervous symptoms disappearing sooner or later after the extirpation. In six of the eight remaining cases a decided improvement was produced. An explanation for the failure in the two cases mentioned is found in local conditions which the operation necessarily failed to reach. M. A. S.

MENTAL PATHOLOGY.

Insanity in the United States. Dr. A. D. WRIGHT (Conference of Charities, Eleventh Session, October, 1884) gives the following table of the proportions of the insane to the sane in the United States:

NEW ENGLAND STATES.

Maine, one insane to every	421
New Hampshire, one insane to every	329
Vermont, " "	327
Massachusetts, " "	348
Rhode Island, " "	404
Connecticut, " "	361
Total population, 4,010,629; one insane to every	359

MIDDLE STATES.

New York, one insane to every	362
New Jersey, " "	470
Pennsylvania, " "	516
Total population, 10,496,878; one insane to every	424

INTERIOR STATES.

Ohio, one insane to every	439
Illinois, " "	600
Indiana, " "	558
Michigan, one insane to every	586
Wisconsin, " "	521
Total population, 13,091,477; one insane to every	610

NORTHWESTERN STATES.

Iowa, one insane to every	639
Minnesota, one insane to every	681
Kansas, " "	996
Nebraska, " "	1,095
Total population, 3,853,886; one insane to every	750

SOUTH MIDDLE STATES.

Delaware, one insane to every	740
Maryland, " "	504
Virginia, " "	627
North Carolina, one insane to every	690
Total population, 3,993,866; one insane to every	610

SOUTH INTERIOR.

West Virginia, one insane to every	630
Kentucky, " "	592
Tennessee, " "	642
Missouri, " "	655
Total population, 5,977,886; one insane in every	629

EXTREME SOUTHERN STATES.

South Carolina, one insane to every	895
Georgia, " "	908
Florida, " "	1,065
Alabama, " "	830
Mississippi, " "	987
Louisiana, " "	938
Arkansas, " "	1,017
Texas, " "	1,018
Total population, 8,499,572; one insane to every	935

PACIFIC SLOPE.

California, one insane to every	345
Oregon, " "	462
Washington, " "	556
Nevada, " "	2,008
Total population, 1,176,844; one insane to every	385

WESTERN STATES AND TERRITORIES.

Dakota, one insane to every	1,878
Montana, " "	664
Idaho, " "	2,038
Wyoming, " "	5,197
Colorado, " "	1,963
New Mexico, one insane to every	781
Utah, " "	953
Arizona, " "	1,926
Total population, 725,994; one insane to every	1,263

The proportion of insanity among the whites of the South is as follows:

Delaware, one insane to every	807
Maryland, " "	453
Virginia, " "	512
North Carolina, one insane to every	544
West Virginia, " "	627
Kentucky, " "	564
Tennessee, " "	558
Missouri, " "	639
South Carolina, " "	607
Georgia, " "	635
Florida, " "	848
Alabama, " "	597
Mississippi, " "	670
Louisiana, " "	652
Arkansas, " "	939
Texas, " "	950

It is curious that in this paper Dr. Wright should have completely ignored the influence an insane errabund tendency has in increasing the proportion of insanity when very marked in the Pacific States as well as in Maryland, where the insane immigrant very frequently remains in the city where he has landed (Baltimore).

Insanity in Italy.—VERGA (*Archivio Italiano per la Malatie Nervose*, Jan., 1886) concludes from a study of Italian lunacy statistics for the past five years, first: That the congenital types of insanity and those developing on an alcoholic, hysterical, or

epileptic basis as a rule seem to increase in frequency, but that the moral and cyclical psychose sare diminishing or remain stationary ; the sensorial, senile, and puerperal types are also diminishing or remaining stationary. Second : Mania, melancholia, monomania, and dementia remain in much the same general proportion. Third : Furious mania does not seem to increase in frequency. Fourth : Depression is most frequent. Fifth : The congenital, moral, epileptic, alcoholic, and paralytic types of insanity are most frequent among men ; furious mania, sensorial, pellagrous and senile insanity. Sixth : Pellagrous insanity is infrequent in Naples and Sicily, and absent in Venice. Alcoholic insanity is more frequent in Upper than Lower Italy. Cretinism is most frequent in Lombardy. Puerperal and hysterical psychoses are most frequent in Venice. Epileptic in Tuscany ; alcoholic in Liguria. Seventh : Two thirds of the cases admitted to Italian insane hospitals are chronic and incurable. Eighth : In Italy insanity is very prevalent between twenty-one and forty, also in unmarried persons. Ninth : Suicidal tendencies and sitophobiac propensities are most frequently manifested by the female sex.

A Seventeen-year-old Paretic Dement. (L'Encéphale, Nos. 51 and 85.)

RÉGIS has reported the case of a seventeen-year-old paretic dement, who in early infancy acquired syphilis from his wet nurse. There were very irregular symptoms, and exalted delusions were wanting. The patient sank rapidly into extreme dementia.

Paretic Dementia and Syphilis. (*Deutsche Med. Woch.*, No. 33, 1885.)

MENDEL gives the following figures as representing the percentage of cases in which syphilis was found by the authorities cited in paretic dementia and the non-paretic psychoses :

Obersteiner	$21\frac{6}{10}$	per cent.	against	$4\frac{1}{10}$	per cent.
Lange	33	"	"	2	"
Oebeke	25	"	"	8	"
Mendel	75	"	"	18	"
Nasse	35	"	"	1	"
Reinhardt, private patients	73	"	"	$16\frac{7}{10}$	"
" public "	73	"	"	$8\frac{9}{10}$	"

Insane Self-Mutilation to Secure Pecuniary Damages. —Dr. THEO. W. FISHER (Report of the Boston Lunatic Hospital, Dec. 31, 1885) reports the following case, which is of considerable forensic value, indicating a clear knowledge of the legal consequences of an act, and also a desire to profit by these consequences. A chronic, persecutory, delusional, and hallucinated lunatic escaped from the garden where he was at work with the gardener, and was concealed by his friends, who professed to consider him sane. A lawyer was employed to secure his dis-

charge. After several weeks he was arrested by the police, and while being taken to the station deliberately put his foot under a horse-car wheel, for the purpose of being taken to the City Hospital and getting damages from the city or the railroad company.

Kalmuc Idiocy.—Dr. SHUTTLEWORTH (*British Medical Journal*, Jan. 30, 1886) says: There is a variety of imbecility, probably scrofulous, which has obtained from its physiognomical characters the name of the "Mongol" or "Kalmuc" type. Dr. Shuttleworth has numerous cases of that type in his institution (perhaps 3 per cent. of its population), and in all there is a certain family resemblance, though they come from widely distant parts of the district. They all have a skin coarse in epidermis, if not furfuraceous; many have sore eyelids, some fissured lips; but one most striking peculiarity is the state of the tongue, which is transversely fissured and has hypertrophied papillæ. Many have almond-shaped eyes obliquely set, and this feature, with the squat nose and wiry hair, gives the "Mongol" aspect, whence they derive their name. Dr. Shuttleworth believes that they are, in fact, unfinished children, and that their peculiar appearance is really that of a phase of fœtal life. He does not mean that they are necessarily prematurely born, but some cause has depressed the maternal powers, and there has been a defect of formative force. It is remarkable that nearly half these children are the last born of a long family, and in more than one third a phthisical history has been traced. They are generally delicate in body, and very susceptible to cold; mentally they have good imitative powers, are often very fond of music, and dance and drill well. Comparatively few grow up to be men and women, and, as a rule, they die of phthisis before twenty. Dr. Kiernan (*Detroit Lancet*, April, 1884) suggests that in many cases these Kalmuc idiots are instances of atavism, the Aryan races having absorbed two Mongolian races, the Picts and the Firbolgs, and this ætiological element should receive some attention.

Cocaine in Insanity.—It has been stated by Dr. BROWER (*Journal of the American Medical Association*, Jan. 17, 1886) that the best results yet obtained from the drug have been in cases of mental depression. . . . Dr. A. B. Shaw speaks with some degree of positiveness of its value in the insanities with depression. Dr. Shaw (*St. Louis Courier of Medicine*, March, 1886) has, however, either changed his opinion, or Dr. Brower has misunderstood him, for he says: "Nothing can be more supremely ridiculous than the attempt to cure insanity by the use of a drug that not only induces those conditions which predispose to insanity and intensifies them where they already exist, but will, in a remarkably short time, produce insanity. I have seen a melancholiac attempt suicide by cutting the radial artery three times in ten days, while he was receiving cocaine to such an extent that it seriously impaired

his desire for food and his general nutrition and strength. While insane patients that could scarcely be induced to take nourishment have under the influence of cocaine partaken more freely of food than they did before its administration, yet the daily use of cocaine, even in small doses, seems to invariably impair the desire for food, although the contrary is generally stated by the patient, notwithstanding he is gradually losing weight and strength." Dr. Brower further says: Although the bad effect of the drug upon the digestive and assimilative processes, and upon the secretions, has frequently disappointed him in its use, he has given the drug several hours before eating, in order to avoid anorexia, but even with this precaution it was frequently found impossible, while using it, to give that great abundance of food, systematic feeding, which is the most valuable therapeutic measure in melancholia. In a case of profound melancholia in a physician aged forty-five, the result of excessive professional work in a large country practice, cocaine was given in one-grain doses three times a day, with pil. hydrargyri, aloes, and strychnia. His recovery was rapid, and has continued for four months. A melancholiac forty-eight-year-old woman under this combined treatment recovered rapidly. The drug was administered in pill form, and probably because of its combination did not interfere with the free use of egg-nog and other concentrated food in large quantities. Dr. Catlett, Fulton, Mo., (*Journal of the American Medical Association*, Feb. 6, 1886,) concludes from an extensive use of cocaine in insanity during the past six months:

1. It is an agent possessed of great potency. It acts primarily with great power and celerity upon the cerebral and spinal nervous system, and secondarily upon the heart and vascular system. 2. From its potency and rapidity of action it is liable to initiate organic lesions and functional disturbances which are uncontrollable. It is not uniform in its action, and therefore is an uncertain agent. 3. Its effects are too transient and unstable to become a reliable and efficient remedy in constitutional or organic diseases. The aphrodisiac effects attributed to it have not been observed, neither have the intense nausea and inability to vomit been observed. Its permanent beneficial effects in melancholia and allied affections are not established facts in his opinion. 4. As it is an agency of great potency, and as it is under trial to determine its limits of usefulness and danger, it should be prescribed with increasing precaution and discretion. The experience with cocaine here narrated is the experience with hyoscyamine retold. Used without due care on the extravagant laudations of experimenters destitute of analytic power, a reaction naturally occurred from its proving, like all neurotics, not destitute of danger where carelessly used. Like opium, it, *a priori*, might have been expected to lock up the gastro-intestinal secretions in some cases, but this was entirely ignored in the furor which followed the discovery of Koller. In the writer's opinion fluid extract of coca has advantages which the alkaloid does not possess,

and given in combination with drugs which act on an atonic gastro-intestinal canal is of value in true melancholia, but is decidedly contra-indicated in the epileptic, traumatic, phthisical, senile, menopause, hysterical, and other psychoses with delusions of suspicion and persecution very generally fused together under that term. At the same time it must also be admitted that in cases of melancholia associated with lithæmia and prëexisting oxaluria coca or its alkaloid would seem to be contra-indicated.

Moral Imbecility. Dr. SHUTTLEWORTH (*British Medical Journal*, Jan. 30, 1886) says :

"Primarily neurotic cases (of imbecility) depend upon inherited instability of the nervous system and are characterized by abnormal excitability. The senses and the perceptions may be sharp enough, but there is a painful restlessness, an incapacity of sustained mental application, and often strange propensities for mischief and cruelty. . . . Such cases of moral imbecility too often tend to insanity at puberty." Unfortunately for the community and themselves, the moral imbeciles do not always become demonstrably insane. Then, too often, as Crichton Brown says, they are "actuated by impulse or by the most selfish, depraved, and cruel motives"; they present, "in short, a perfect picture of the desperado and ruffian." The existence of "moral imbecility, like the existence of every thing else, has been called into question, and there are not lacking those who would send the 'moral imbecile' to the penitentiary or the scaffold." It has been denied that moral imbecility exists: on the one hand, because innate ideas do not exist; and on the other, because they do. The arguments drawn from the teachings of the contending schools sufficiently offset each other as to destroy even a theoretical basis for objection against the clinical fact. This is still more strikingly shown by the fact that two alienists who accept the clinical entity adopt opposing views of the nature of the moral sense. Bannister (JOURNAL OF NERVOUS AND MENTAL DISEASE, 1877) says: "Whether we consider this moral sense as a *primary feeling*, as seems probable for many reasons, or as a derivative one composed of still more elementary feelings, or as a necessary sequent of some other state, it does not alter the case as regards the present question of moral insanity. By this term we mean a disease of the brain affecting alone its functions as the organ of the moral nature, disordering the capacity to receive moral impressions and the ability to control conduct for moral ends. Spitzka, on the other hand ("Insanity: Its classification, Diagnosis, and Treatment"), says: The mental state of the imbecile has been very well expressed by the statement that those *mental coördinations* acquired in the course of a higher civilization have not been formed in him. Moral defect is a prominent feature of some cases, and this condition may be the chief manifestation of mental deficiency. There are subjects whose reason-

ing powers are fair, whose memory is excellent, who are, perhaps, accomplished in the arts, but in whom the moral sense is either deficient or entirely absent. The term moral insanity should be limited to this class of subjects, and a much better term would be moral imbecility."

Psychical Character of Hereditary Paranoiacs. MAGNAN (*Annales Médico-Psychologiques*, Jan., 1886) says that the predominant feature in the hereditary types of paranoia is the disharmony and lack of equilibrium, not only between the intellectual operations, properly so-called, on the one hand, and the emotions and propensities on the other, but even between the intellectual faculties themselves. A hereditary paranoiac may be a scientist, a noble lawyer, a great artist, a mathematician, a politician, a skilled administrator, and present from a moral standpoint profound defects, strange peculiarities, and surprising lapses of conduct, and as the moral element, the emotions, and propensities are the base of determination, it follows that these brilliant faculties are at the service of a bad cause—of the instincts and appetites which, thanks to defects of the will, lead to very extravagant or very dangerous acts. In other cases the opposite occurs. Hereditary paranoiacs of irreproachable character show strange lacunæ in their intellect. They have a feeble memory. Sometimes they cannot understand figures, calculus, music, or drawing. In a word, another wise modern intelligence is lacking as regards certain faculties. The perception centres are unequally impressionable, unequally apt to gather together impressions; only certain impressions are registered and leave durable images; otherwise certain relations, certain associations between different centres, are perverted or even entirely destroyed.

Insanity and Aural Disease. Dr. G. C. CATLETT (*American Journal of Insanity*, January, 1885,) Fulton, Mo., reports the following case of insanity associated with aural disease. A., twenty-one years old, single, farmer; had been a man of good moral habits, except as to masturbation. He has had double aural inflammation in both ears from scarlatina since he was eighteen years old, and pus flows from both ears since that time. His first attack of insanity of indefinite duration, since he has been very eccentric for some years. He was peculiarly restless and made constant circular motions, walking around in a circle to the right. He had tinnitus aurium, heard persons conversing with him, frequently looked out of the window to see who the persons were whom he heard talking, etc. A paternal aunt was insane. He is insomniac, self-willed, perverse, but eats well; suspicious, fearful of personal injury, very obstinate, and refuses to comply with the request of physicians or attendants. Restraint was needed to examine and treat his ears. Both auditory canals filled with hardened dried pus and cerumen. When the obstructions were

removed both drums were found to be perforated, and discharged from the internal ears. Both Eustachian tubes were free, as bubbles of fluid could be seen escaping through the ears when air was forced through the tubes. Hearing improved and the tinnitus diminished, the hallucinations became less, also the disposition to keep in motion either in the right circular direction or otherwise, as the disease in the ears improved. Without further details of the case it is satisfactory to state that this man returned home in four months recovered from his insanity and his aural disease. It is more than probable that the hallucinations only were removed and the insanity still remains.

Melancholia from Paranoia. Dr. KIERNAN (*American Lancet* January, 1886,) cites the following case: "In melancholia, stuporous insanity, and in certain phases of depression which mark other psychoses the cerebral disease seems (to use Clouston's words) to exert an inhibitory action on cardio-motor innervation, causing the pulse to be small, the arterial tone low, and the capillary circulation very weak indeed, and in many cases there are very decided thoracic symptoms accompanied by mental distress resembling attacks of suffocation, accompanied by precordial fright as it has been termed. For these reasons quebracho would seem to be indicated in melancholia and the psychoses mentioned. While aware of the theoretical basis for the use of quebracho in the psychoses named my attention was especially attracted to a case of what seemed to be melancholia with the facies, capillary circulation, and emotional depression well marked. The patient, a woman, had phthisis, and had been deserted by her husband. She ran down rapidly and at one time seemed almost moribund from dyspnœa. To relieve this, quebracho in half-drachm doses every two hours was given, with very beneficial results not only on the dyspnœa, but also on the patient's mental condition. She seemed to markedly rally from her depression, and the facies and depression of melancholia disappeared, but an insanity of manner made its appearance, and it was found on careful investigation the patient had systematized delusions of grandeur for several years before being suspected of any mental disease, and that, therefore, the melancholia was a complication of a preëxisting paranoia which had not been suspected.

J. G. KIERNAN, M.D.

THERAPEUTICS OF THE NERVOUS SYSTEM.

Production of Sweat in Various Diseases and the Effects of Pilocarpine. By E. DE RENZI. Quoted from *Gaz. Med. di Torino* in *Gaz. degli Ospitali.* 7 Marzo, 1886. No. 19. P. 149.

The author from clinical experimentations comes to the following conclusions:

1. The sweat after the subcutaneous injection of a centigram of pilocarpine appears not later than five to ten minutes in some part of the cutaneous surface, especially in the neck and breast.

2. When there exists a paralysis of the motor nerves of whatever origin, there is a lack or perceptible retardation of the appearance of perspiration in the skin corresponding to that region.

3. In three cases of ataxia the sweat was scanty, retarded, and was lacking in the parts affected with greatest intensity by the motor disorder.

4. In diabetes as can readily be conceived, on account of the dryness of the skin the sweat is small in amount, and ordinarily is lacking in the inferior extremities, especially in the feet.

5. In the intense prosopalgia the sweat is lacking in the face. In one patient it was possible to demonstrate that the sweat secretion was independent of the state of the cutaneous vessels. In fact, in spite of the extreme vascular dilatation and of the extreme reddening of the face after the injection of the pilocarpine, there was in this part of the body not a sign of perspiration.

6. Contrary to all preceding observations in which the secretion is diminished, the author cites two cases of unilateral pleuritis in which the sweat was without doubt greater on the affected side.

7. It is possible in the actual state of science to give a satisfactory explanation of these facts relative to the secretion of sweat. Alterations of the cerebral motor nerves, spinal and peripheric branches, either through paralysis or ataxia, produces always a diminution of the sweat ; the neuralgic process in the trigeminal arrests also the secretion in the face. An irritative process, such as results probably from a pleuritis on the intercostal veins, favors greatly a secretion of the sweat ; hence it may be said that the sweat secretion depends upon the nervous system, and is independent of the dilatation or constriction of the vessels, and is produced by the direct action of a fibre acting on the secreting cell.

The principal centre and point of origin of this fibre is in the spinal medulla, and also from the brain.

8. The author gives the result of the injections of pilocarpine in the blood, the sphygmograph showing in five minutes that the ascending line becomes higher, reaching its maximum intensity in fifteen to twenty minutes. After forty minutes it returns to the same height as before the drug was given. Maximum intensity of the drug is in fifteen minutes ; an increased dicrotism and increased number of the pulsations also take place.

GRACE PECKHAM.

Reviews and Bibliographical Notes.

Die Thomsen'sche Krankheit (Myotonia Congenita) Studien. By Prof. Erb, of Heidelberg. F. C. W. Vogel, Leipzig, 1886.

In this monograph of 128 pages Prof. Erb presents as complete an account as can at present be given of Thomsen's disease. It is not merely a compilation of cases heretofore reported, but an entirely independent study of the disease based upon the most careful observations of three cases—two of his own and one sent to Erb by Dr. Fischer, of Canstatt. Having all the methods of examination and investigation thoroughly at his command, Prof. Erb has been enabled to add very materially to our knowledge of Thomsen's disease. The additions he has made are indeed so numerous that a very recent and otherwise able critical digest on this disease in the April number of *Brain* must remain incomplete because it preceded the publication of Erb's monograph by several weeks.

We believe that Erb has now fixed the "type" of this disease, according to which all subsequently reported cases of Thomsen's disease must conform. In view of the general interest of the subject we propose to give a full review of this monograph. We will not pretend to write a *critique;* though we wish to record but one impression, and that is that, to our thinking, Prof. Erb has too rigidly excluded certain variation of symptoms which have been reported by other observers.

In analyzing previous cases the author admits but 23 "typical" cases among those hitherto described. Eleven others he considers doubtful, and among these he classes Eulenburg's case of hypertonia musculorum hypertrophica, about which there has been considerable dispute. He says nothing about two cases referred to by Hamilton, possibly because he has not heard of them, more probably because he does not consider them at all related to this form of disease.

As regards the name of the disease, it will be noticed by the title of the monograph that the author approves of Strümpell's term *myotonia congenita*, and yet retains the name "Thomsen's disease." There is a peculiar propriety in doing this, for the physician who first described it was (is) also a sufferer from the disease.

The chief result of Prof. Erb's studies is the discovery of *typical changes in the mechanical and electrical excitability of the motor nerves and of the muscles;* and of *marked anatomical changes in the muscles themselves*, which changes are probably closely related to the changes in mechanical and electrical excitability.

The characteristic features of Thomsen's disease are as follows (according to Erb):

The disease is hereditary, and generally affects several members of the same family; it begins in earliest infancy or as late as the age of puberty.

CLINICAL SYMPTOMS.

Motor Disturbances: After a period of prolonged rest, there is a marked *stiffness* of the muscles and *slowness of movement*, when a movement is attempted, in consequence of which the body may become rigid, the rigidity disappearing only after persistent muscular effort. The phenomena may be exhibited in walking, running, in the attempt to move the hands, and even in chewing. If the patient is asked suddenly to perform a single movement, *e. g.*, to close the hand, he does this promptly enough, but he is not able to relax his muscles at will; there is evidently a *tonic contraction* outlasting the volitional stimulus. This period of *protracted contraction* may be from 5 to 30 seconds. These symptoms Erb styles *myotonic* disturbances of motion.

The muscles are enormously developed, but their power is not in proportion. In the relaxed condition, the muscles have the normal character; during contraction they are remarkably hard and firm. Erb observed occasional *fascicular* contractions which he says are distinct from fibrillary contractions,

MECHANICAL AND ELECTRICAL EXCITABILITY.

Motor Nerves: Mechanical excitability possibly diminished, certainly not increased; *faradic* excitability quantitatively normal; sudden increase in strength of current may produce prolonged contraction, lasting from 4 to 10 seconds after the withdrawal of the stimulus. *Galvanic* excitability very nearly normal; single shocks absolutely so; a succession of shocks may produce tonic prolonged contractions.

Muscles: Mechanical excitability undoubtedly increased; simple touch with finger being sufficient to excite tonic contraction of the fasciculi under the finger. Such contractions may last from 5 to 30 seconds.

Faradic excitability very marked; muscles are quickly relaxed when weak currents are used; sudden increase produces prolonged contraction, but opening contractions, no matter how strong they may be, are never lasting. In one case, keeping the electrode fixed upon a muscle (the gastrocnemius) produced strong oscillating contractions in the muscle. *Galvanic* excitability increased quantitatively (k. c. c. and a. c. c. in different muscles with currents of ¼, 1, 2, m. a.). Qualitative changes: Both poles act nearly equally; the anode may have a slight preference. The

muscular contractions are slow, tonic in character and prolonged. Strong currents invariably produce prolonged contractions. But most characteristic of all are *rhythmical wave-like* contractions produced by stabile currents when the one pole is placed upon the muscle, and best of all, upon its tendinous end. These wave-like contractions proceed from the kathode to the anode, up or down the arm for instance, according to the character of the pole held in the hand. For the electrical phenomena described above, which are typical of Thomsen's disease, Prof. Erb proposes the term *Myotonic Reaction* (My. R.). The R. D. has at last found its mate ; the two have some features in common, but in the main they are widely different.

PATHOLOGICO-ANATOMICAL DATA.

From one of his patients Erb had a piece of muscle removed for purposes of examination. In this he found very marked changes, which he considers characteristic of the disease. On cross-section Erb found an enormous hypertrophy of muscular fibres, as compared with the appearance of healthy muscle ; the cross-section of each fibre appears well rounded, almost circular, and not polygonal like the cross-section of a normal fibre. The main change, however, is a very considerable increase in the number of nuclei in the sarcolemma ; furthermore, an increase of the interstitial connective tissue. In the case of Dr. Fischer, Erb found, in addition to the changes just enumerated, a striking vacuolisation of the individual muscular fibre. The great value of this discovery will be appreciated when we consider that a number of eminent authors were ready to class Thomsen's disease among the heterogeneous functional neuroses.

The question arises, whether or not Thomsen's disease is a disease of the muscles merely. Up to date no changes have been found anywhere else, but the nervous system has as yet been very insufficiently explored. Erb wishes it distinctly understood that he is not willing to affirm or deny that Thomsen's disease may prove to be a tropho-neurosis.

We have given the merest outline of this truly classical monograph. For an account of the differential diagnosis[1] between the clinical symptoms of this disease and those of other somewhat similar forms of disease, and between the pathological changes underlying the various troubles, and for the suggestions regarding treatment, we must refer the student to the original. The monograph is instructive from beginning to end and deserves the very closest study.

We trust that typical cases may soon be reported by American neurologists. B. S.

[1] Erb suggests, (p. 79), that hereafter a tap with the percussion hammer and a few closure contractions with the kathode or anode upon certain muscles will be sufficient to establish a diagnosis of the disease. Diseases which may bear some resemblance to Thomsen's disease are : *Tetany, pseudohypertrophy of the muscles, and chronic troubles with marked reaction of degeneration.*

Editorial Notes and Miscellany.

THE LATE PROF. V. GUDDEN.

THE whole medical world, and neurologists in particular, have suffered a severe loss in the tragic death of Professor v. Gudden. Though advanced in years (he died in his 69th year) Gudden was still adding to the great reputation he enjoyed—a reputation which was based largely, though not exclusively, upon the successful application of the "atrophy method." As a cerebral anatomist, he ranked among the first of all times, and his untimely death has undoubtedly deprived us of much light that he would yet have been able to throw upon the anatomy of the brain.

While he was devoted mainly to his anatomical researches, he was also deeply interested in cerebral physiology, and in his latest public utterances before several learned societies he declared himself decidely in favor of Goltz's views of localization and against those of Munk. But whatever his merits as a scientific investigator may have been, he was equally great as clinician and teacher. He was a man truly to "inspire" pupils, and we are confident that his teachings will bear further fruit in the labors of his scholars, some of whom stand even now in the foremost rank of German alienists.

KING LUDWIG'S INSANITY.

Our daily press has furnished some amusing information in the line of "expert" opinion on the form of insanity with which the king was afflicted. With one exception, the experts (sic!) shot wide of the mark. For the benefit of these gentlemen we wish to add that the disease was *not* general paresis, and that the commission (consisting of v. Gudden, Hagen, Grashey, and Hubrich) reported the royal patient to be "originär verrückt." The diagnosis would therefore read, Paranoia—primary insanity, if you will.

The Council of the American Neurological Association announce that the regular business meeting of the association will be held at Long Branch, N. J., on Wednesday, Thursday, and Friday, July 21, 22, and 23, 1886. Two daily sessions will be held, one in the morning from 10 A.M. to 1 P.M., and one in the afternoon, from 3 to 5 P.M. A suitable room has been engaged at the Howland House, where the meeting will be held.

VOL. XIII. JULY, 1886. No. 7

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

ON THE CAUSE OF ELECTROTONUS AND OF THE NORMAL FORMULA OF POLAR REACTIONS.

By G. BETTON MASSEY, M.D.,

ELECTRO-THERAPEUTIST TO THE PHILADELPHIA ORTHOPÆDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES.

(*With four wood-cuts.*)

WHEN a motor nerve that has been isolated from its natural bed of superior conducting materials is subjected to the action of either pole of a galvanic battery, a stimulation of its functions is produced when the circuit is closed and opened. It is well known that this stimulation varies in amount at the closure and at the opening, and with the pole that is used. Briefly, it is found that the strongest stimulation is at kathodic closure, and the next strongest at anodic opening, with anodic closure following closely, and kathodic-opening stimulation only produced by the strongest currents. Such is the substance of the normal formula of contraction as ascertained in the laboratory. It corresponds closely with the formula obtained in man, especially when the nerve is not deeply situated.

The well-ascertained facts of electrotonus show that when the isolated nerve is kept under the influence of the kathode of a constant current, any additional stimulus applied to the area directly under that pole will produce a greater con-

traction than normal; and that when the nerve is under the influence of the anode a parallel lessening of its excitability is found. This has recently been shown to be true of the embedded nerve also, as proven by the experiments of Waller and De Watteville,[1] conducted under a special grant from the Royal Society of England.

A single fact underlies, therefore, both electro-contractility from interrupted currents and electrotonus from constant currents, namely: that the kathodic condition is at least more exciting than the anodic. This preponderance of excitability to the kathode will be recurred to directly.

It is almost unnecessary at the present day to add further to these preliminary statements by saying that the action of a pole has nothing to do with the correspondence, or want of correspondence, between the directions of the elec-

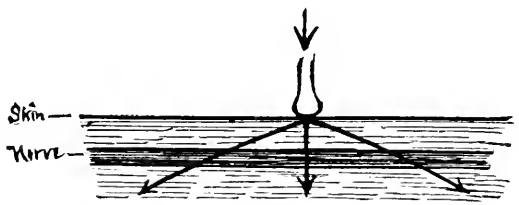


FIG. 1.—Diagram of a nerve under unipolar influence. It is traversed by the current lines in all directions.

trical current and of the current of nerve force. This assumption of the physiologists, who dealt only with uncovered nerves, in which such a correspondence between the two paths of force could be readily obtained, was disposed of by the fact that experiments on the embedded nerve yielded the same results, though the current traversed the nerve so situated in at least three different directions under the one pole (fig. 1). The conclusion was forced that the stimulation and its varieties were polar effects rather than directional effects of the current, and this view has been generally adopted by writers on the subject. I may here also remind you of an older conclusion of the same investigators that requires constant remembrance, and that is: that an excitation great enough to produce contraction

[1] Philosophical Trans., 1882, p. 961.

must be due to a current variation ; and that the variation or oscillation of current must occur within a certain time—in other words, must be sudden. It is true that we have tetanoid contractions during the continuous flow of a strong galvanic current, but these are doubtless due to those minute variations that are now presumed to exist in the most constant current, becoming visible as striæ when such currents are passed through partial vacuums in the dark. These variations, being very numerous and in rapid succession, produce, when the volume of the galvanic current is overpoweringly great, a tonic contraction of the muscle similar to that produced by the rapidly-successive faradic currents.

Electro-contractility is therefore essentially due to the act of current variation. Electrotonus is dependent only upon the period of current flow, and is probably not due to the slight variations just referred to as producing the tetanoid contraction. It is further important to remember that both these actions of contraction and tone are physiological in character, being of a different nature from the chemical or electrolytic action of the current.

With these preliminary remarks I will proceed to a discussion of the electro-physical laws governing these polar effects, and will subsequently present a new theory of their explanation that may or may not be acceptable ; being convinced that these obscure questions of the physiological action of electricity can only become comprehensible through a study of the physical conditions of currents during experiments.

The laws of current diffusion within large conductors have been indicated for years by the necessities of Ohm's law, but no actual determination by experiment was made before the investigations of Prof. W. G. Adams, the results of which are embodied in the Bakerian Lecture for 1875.[1] Buckets and tubs of salt water and various acid solutions were used for the experiments, a continuous galvanic current being sent through these large and variously shaped conductors from poles inserted in them at proper distances

[1] Proc. Royal Soc., Vol. XXIV., p. 1. See also a theoretical discussion by Foster and Lodge, Proc. Lon. Phys. Soc., Vol. I., p. 113.

apart. During the passage of the current, measurements were made of the potential of various spots in the body of the liquid by means of galvanometer electrodes insulated to the tip. These, being freely movable in any direction, permitted the mapping out of lines of points at which the potential was the same, thus determining the equipotential lines throughout the interior. If one galvanometer electrode was placed at one of these points, no deflection could be obtained with the opposite galvanometer electrode on any spot of the same line; with the opposite galvanometer electrode placed at any other point a deflection would result, indicating the difference between the potential of the two points.

These experiments proved what had previously been a mere theory, completely verifying it. The principles thus established are of the utmost importance to medical users of electricity, and I have therefore designed for the use of my classes at the Infirmary some illustrations (figs. 2, 3, and 4) showing the main facts to the eye. The diagrams are all drawn for a current of twenty volts, and show its behavior within conductors of varied thickness; the position of the potentials and lines of flow only being indicated, and no reference being had to the number of milliampères transmitted. The latter would depend on the resistance of the several conductors—a question that we are not now interested in, as the equipotential lines remain the same whatever the volume of the current, and are only dependent on the number of volts in the circuit.

You will notice that a current of twenty volts has a positive potential of ten volts at the anode and a negative potential of ten volts at the kathode. Each potential recedes to zero as the centre of the intervening resistance is approached, the points where certain potentials are found being indicated in the diagrams by dotted lines of equal potential. The current flows from potential to potential in what are called lines of flow, always crossing a potential line at right angles, just as water flows down a series of steps. The theory that prevails at present in the practical literature of electricity, assumes that electrical energy is in a state of accumulation

at the anode and deficiency at the kathode,—the anode being at the highest point; zero at the level of the earth; and the kathode at the lowest point of an imaginary incline extending from a certain distance above, to an equal distance below, the level of the earth or equilibrium. The flow is down this incline with an impetus measured by the height of the anode above the kathode.

In the narrow conductor (fig. 2) the equipotential lines, or

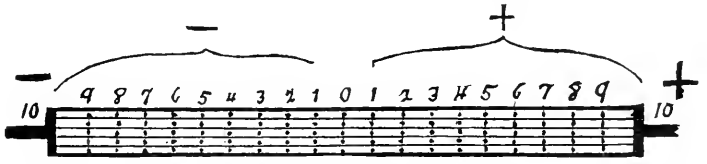


FIG. 2.—Diagram showing the distribution of a current of twenty volts within a narrow conductor. The milliampères are equally distributed in the lines of flow (represented in the cut by horizontal lines). The lines of equal potential (represented in the cut by dotted lines) are drawn one volt apart, and have a value indicated by the figures.

in illustrative language, the edges of these steps of electric level, extend straight across the conductor, the lines of flow being straight lines from pole to pole. In the wider conductor (fig. 3) the lines of potential tend to curve somewhat about the poles, hence the lines of flow on either side of the

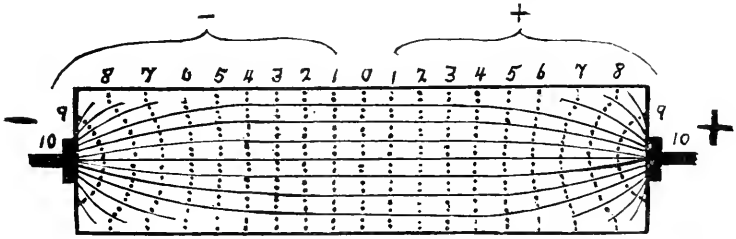


FIG. 3.—Distribution of a current of twenty volts within a wider conductor. The lines of flow on either side of the central one curve somewhat, as do also the most positive and most negative potentials.

centre one, which remains straight, curve a little also, as the potential lines must be crossed at right angles.

When the current is passed through so large a conductor as the human body (fig. 4) the potential lines become arcs of small circle, about each pole, the size of the circles rapidly increasing as we proceed away from the poles. The lines of

flow necessarily crossing the potential lines at right angles become variously curved also, those next to the straight line in the centre remaining straightest; while those most distant from the direct line from pole to pole curve the most. Prof. Adams found that all equipotential lines abandoned their regular curves when approaching the edges of the conductor, or the sides of the vessels containing the

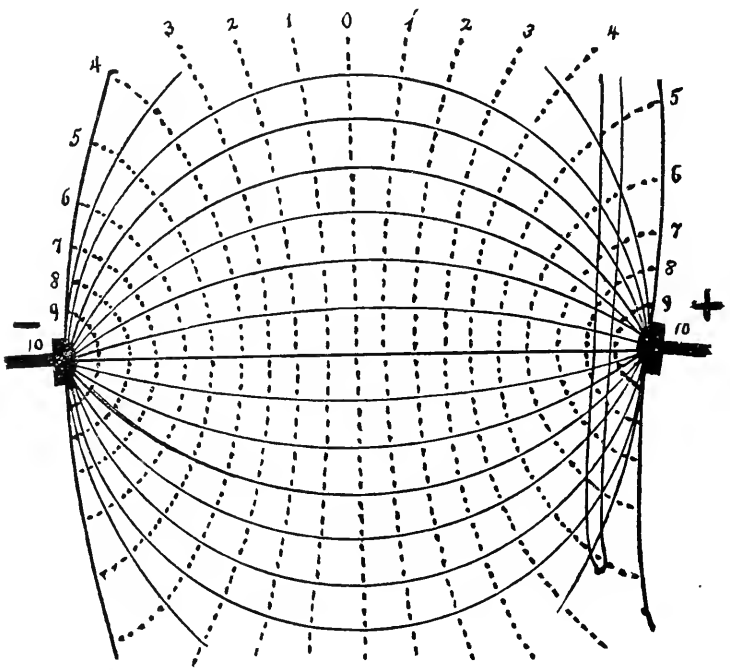


FIG. 4.—Distribution of a current of twenty volts within the human body. The equipotential lines or planes become segments of perfect circles, and the lines of flow necessarily crossing the latter at right angles, become greatly curved, and extend to all parts of the body in passing from pole to pole. The number of milliampères traversing the lines of flow differs somewhat, being inversely proportional to the length of the lines.

liquid, and cut them at right angles. This means that the lines of flow nearest the boundaries of a conductor are always parallel to the boundary edges.

When the poles are applied to opposite sides of a large thick conductor like the body the lines of flow curve between them in all directions, to the right and left, upward

and downward. Each equipotential is best conceived in the mind, therefore, as a curved plane shaped like the half of a hollow globe, placed with the concavity towards the pole of the same sign. In a model constructed to show this, the material between the planes would resemble thick cups of progressively increasing size and curve, placed in two nests, base to base, and with the smallest cups next to each pole.

It is necessary to add that these diagrams are drawn to represent the distribution in a homogeneous conductor, such as a single organ, and that slight alterations would be required to make them fit any composite conductor made up of the various human organs, differing as they do in conductivity. This would only make the lines of potential and flow more or less wavy, however, the general direction remaining the same; and as no two persons would present the same waviness, the average curve was adopted as represented.

With these facts before us let us examine the theory of polar reactions and electrotonus first promulgated by Helmholtz, and so widely published since by Erb, De Watteville, and others. A cardinal principle of this theory is that the kathodic polarity alone is exciting. On this point the theory is doubtless correct, but the hypothesis resorted to to explain it is somewhat confusing, and I think weakens the force of the main point. I allude to the assumption of a peripolar region of opposite polarity in the neighborhood of each pole. You are probably all familiar with the diagrams used to convey this idea. I have been unable to find any support for this theory outside the works of medical writers. The diagrams described in this paper show nothing of the sort. It will be seen by again referring to the latter that the potentials of either sign gradually lessen from the points of entrance to the region midway between them, where it is zero. The exact situation of zero, or the point of no potential, depends upon the evenness of the resistance of the body—it is at the middle of the resistance. Of course, any one point among the positive potentials is relatively negative to a higher potential, and conversely with the nega-

tive points; but no reasoning should assume that a positive potential is absolutely negative merely because it is less positive than a point higher up in the scale. All positive potentials are more positive, and all negative potentials are more negative, than the general body condition, which is nearer equilibrium.

The peripolar theory probably arose through the mistake of regarding the lines of flow through a nerve instead of considering the potential to which it is subjected. It was argued that if the current lines passed through a nerve their entrance at one side created an anodic polar region, and their exit from it at the other side created a kathodic peripolar region—both regions being within the diameter of the nerve. This view of the matter is seen to be faulty when you glance at fig. 4, where the cord is shown bathed in one polarity; with a potential stronger on the nearer side it is true, yet showing at the most a mere gradation of one kind. Moreover, the entrance of a line of flow into a nerve and its exit from it are not separate facts but parts of one fact, and therefore incapable of presenting the current variation essential to the production of a contraction.

It seems to the writer very difficult for the average mind to grasp these questions of the polar reactions and their rational explanations unless the deep meanings of electrotonus are clearly apprehended. That electrotonus is a physiological impression made upon a nerve, which, besides altering the nerve excitability during its continuance, will give rise to a contraction if the impression is suddenly produced or suddenly ended, is now well established. Regarded in this way, katelectrotonus, producing a heightened excitability during its continuance, causes a very great contraction at its inception and a very slight contraction at its cessation. Anelectrotonus, producing a lessened excitability during its continuance, causes a slight contraction at its inception and a great contraction at its cessation.

It is thus seen that all alterations of the electric equilibrium of the nerve will produce contractions—provided the alterations are great enough,—as has been understood for many years; and the rational cause can well be conceived

to be the mere fact of some sort of molecular change accompanying the change of potential; but who has explained why katelectrotonus will increase irritability and anelectrotonus lessen it, or why the production of katelectrotonus causes a greater contraction than its cessation, while the cessation of anelectrotonus causes a greater contraction than its production? Why, in other words, the positive pole is the most negative in its action on the body? I am not aware that this question, important as it must be, has ever been formulated before. A possible answer occurred to the writer some months ago after reading a suggestive paper[1] in an English physical journal. This paper contained an elaborate citation of facts and observations to prove that the direction of electrical flow is the reverse of that now held, being from the kathode to the anode. The kathode, accordingly, is the true plus pole, or pole of raised level, and the anode the true minus pole, or pole of depressed level. Using the convenient phraseology of physical electricians, the kathode becomes the "source" of supply to the body, and the anode the "sink."

The English observer's facts were all connected with the physical behavior of electricity of very great potential, as produced by Holtz machines, and no reference was made to the action of the poles on living tissue; yet no individual fact could bring stronger evidence in support of this theory. Adopting that theory, the laws of physical electricity remain the same, with a mere change in the method of expression—in fact, a mere change of terms. With its aid both electrotonus and the polar reactions become easily and rationably explainable. Katelectrotonus is a condition of increased excitability, because the normal potential of the nerve is exalted. Anelectrotonus is a condition of depressed excitability, because the normal potential is depressed. The sudden raising of the potential of the nerve

[1] The paper referred to in the text was only casually read by the present writer while pursuing another line of search in a library. Its reference was taken for more careful perusal, but the note-book containing it was lost, and subsequent search has failed to rediscover the paper. It is interesting to note that the *London Lancet* for May 1, 1886, contains an abstract of the Lumleian Lecture delivered since the present paper was written, in which a paragraph occurs referring to the uncertainty of current direction, but making no suggestions.

above equilibrium (kathodic closure) produces the greatest contraction; while its fall to equilibrium (kathodic opening) produces the least. The sudden depression of the potential of the nerve below equilibrium (anodic closure) produces also a small contraction, and the sudden restoration of the depressed potential to equilibrium (anodic opening) produces a contraction that is next to the greatest.

In other words, kathodic closure is in this theory the sudden application of electric energy to the part, and anodic closure is the sudden abstraction of electric energy from it. At kathodic opening the energy applied at kathodic closure is permitted to dissipate; while at anodic opening the vacuum produced at anodic closure is suddenly annihilated by the inward rush of the more normal potential of the surrounding tissues.

It may therefore be noted that a sudden application of this energy, or rise of potential, is a greater excitant than a sudden abstraction of the energy, or fall of potential—a fact that seems quite a matter of course. Additionally, if the rise is from equilibrium upwards, the excitation is greater than if it is from a depressed potential to equilibrium only; and if the depression is from equilibrium downwards, the excitation is greater than if it is from an exalted potential to equilibrium only.

The discussion of this subject could be carried much further with advantage, but I think sufficient has been said to show that the new theory, if once established, would be of great advantage in making comprehensible what is now merely a bare collection of facts.

RACE AND INSANITY.*

By JAS. G. KIERNAN, M.D., Chicago.,

late medical superintendent cook county hospital for the insane, formerly of the new york city asylum for the insane, member of the new york society of medical jurisprudence and of the chicago medical society.

THE† influence of immigration in increasing insanity in the United States has hitherto been discussed from one, and it must be admitted, rather limited standpoint, during the past five years. In 1881,[1] basing my opinion on my New York experience, I urged governmental regulation of immigration. Dr. Pratt[2] also urged governmental restriction. He, however, did not take into account an element of greater importance than the mere importation of insane individuals. A non-English speaking foreigner seems less out of accord with his environment, and hence does not very soon reach an insane hospital. He often marries and rears a family. Even after he learns English his peculiarities are condoned on the ground of being a foreigner. The greatness of the dangers from this source cannot easily be demonstrated, since the reign of the "practical Philistines" in Anglo-Saxon psychiatry has prevented the proper study of insanity from a biological standpoint. But little is available for present purposes. Morandau de

* A series of Studies in Ethnological Psychiatry.

† A number of errors have crept into the tables of the last article. Five per cent, should read sixteen (p. 236). The German female column in table XI. (climacteric insanity) should read 2 in place of 0. The German male column in table XI. (choreic insanity) should read 0 in place of 4. The negro and Bohemian figure in table XX. should be transposed. The male negro column in table XII. should read 5 in place of 2; the Holland 4 in place of 5; the American Anglo-Saxon 17, not 19. For table XVIII. should be substituted XVIII. in the present article. The Anglo-Saxon American figures in table XXVI. should read 8 in place of 9.

Monteyel[3] has called attention to certain biological facts which deserve attention in this particular, and which indicate one peculiar unnoticed danger from the imported insane. Paranoiacs are peculiarly liable to regard the United States as a fitting field for their projects, and hence their errabund tendencies direct them hither. That many of them never reach insane hospitals is obvious from the history of a Norwegian, elsewhere related,[4] who lived in the United States twenty years and procreated several children before his insanity was suspected; only an accidental rencontre with an irascible, abusive neighbor led to its discovery. The relative proportion of paranoiacs is therefore of interest. Only the German and Italian hospitals afford means of comparison.

TABLE XXIX.—PARANOIA.

Races.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American,	15	11	26
" " English	6	2	8
" " Scotch	2		2
" " Canadian	1		1
Hollanders	2		2
German	14	11	25
" -American	4	2	6
Scandinavian-Danes	4	2	6
" Norwegians	5	3	8
" Swedes	1	3	4
Celtic.			
Irish	20	12	32
" -American	8	4	12
" -Canadian	1	1	2
Latin.			
French-American	1		1
Italian	2	1	3
SHEMITIC	3	2	5
Hebrew.			
NEGRO	3	2	5
Total	92	56	148

The paranoiacs in the German and Italian insane hospitals are about seven per cent. of the resident population; in the Cook County Insane Hospital they furnished fourteen per cent. of the resident German and Italian insane.

Nor does this fully indicate the real number of paranoiacs imported, for, from reasons already indicated, a larger number will escape insane hospital treatment in the United States. No Bohemians appear in the statistics, yet I know of several Bohemian paranoiacs who died after a very checkered career without entering an insane hospital. The same is true to a lesser extent of the Teutonic and Latin races. The United States is therefore constantly receiving elements of degeneracy. The paranoiac's child may be an epileptic, a periodical lunatic, a chronic hysterical lunatic, a paranoiac, imbecile, idiot, or congenital criminal. It becomes of interest therefore to ascertain what dangers there are of children being procreated by these lunatics. The only statistics available are those collected by myself, which tend to corroborate de Monteyel's researches, and are as follows:

TABLE XXX.

Civil Condition.	Male.	Female.	Total.
Married—childless	12	8	20
" children born dead	9	2	11
" majority died infants	18	11	29
" " survived infancy	4	5	9
Unmarried	19	11	30
Total	62	37	99

TABLE XXXI.

Children in Family.	No. of families.	Survived infancy.	Died in infancy.
5 children in family	3	7	8
6 " " "	2	5	7
8 " " "	7	19	37
9 " " "	8	20	50
10 " " "	11	31	79
11 " " "	2	5	17
12 " " "	1	3	9
13 " " "	1	2	11
16 " " "	1	3	13
17 " " "	1	4	13
22 " " "	1	3	19
Total	38	102	263

TABLE XVIII.—SENILE INSANITY.

Race.	Male.	Female.	Total.
ARYAN.			
Teutonic.			
Anglo-Saxon, American	5	6	11
" " English	1	0	1
German	4	5	9
Scandinavian-Swede	0	1	1
Celtic.			
Irish	9	11	20
Sclavonic.			
Bohemian	0	1	1
Latin.			
French-Canadian	2	2	4
SHEMITIC.			
Hebrew	1	1	2
Total	22	27	49

It is obvious that the dangers of degeneracy are serious and need national attention.* The fact that the mismanagement of the Cook County Insane Hospital, elsewhere described,[5] resulted in an enormous death-rate, which, of course, affected most largely the chronic insane, shows that, seriously significant as these statistics are, they rather under- than over-rate the danger. It has been stated that the influence of heredity is not as potent as is generally believed, for otherwise the world would be depopulated. While this was stated for mercenary reasons, it is true that the conservative tendencies of heredity would soon overcome the degenerative tendencies were it not true also, as has been shown by Bannister,[6] Manning,[7] and myself,[8] that there is a very readily understood tendency on the part of the neuropathic to seek each other in marriage.

(*To be continued.*)

BIBLIOGRAPHY.

[1] *Chicago Medical Review*, vol. iv.
[2] *Journal of American Medical Association*, vol. ii.
[3] *L'Encéphale*, 1883; JOURNAL OF NERVOUS AND MENTAL DISEASE, 1883.
[4] *Neurological Review*, vol. i, No. 1.
[5] *Chicago Medical Journal and Examiner*, vol. lii., No. 4.
[6] JOURNAL OF NERVOUS AND MENTAL DISEASE, 1882.
[7] *Australasian Medical Gazette*, Aug., 1885.
[8] *Detroit Lancet*, vol. viii.

* The senile lunatics appear to be imported in great numbers, judging by the above table.

A CONTRIBUTION TO THE LOCALIZATION OF FOCAL LESIONS IN THE PONS OBLONGATA TRANSITION.

By E. C. SPITZKA.

(Continued from p. 205.)

ONE remarkable feature of the diseased area was that the topographical details, such as the natural demarcation lines separating various nerve-strands and ganglia were clearly recognizable. The disease had invaded the nerve nuclei and the sagittal nerve-tracts distinctively; but as those nerve fibres which run in the plane of such sections as are made transversely to the peduncular axis were not destroyed, the intricate fielding of the reticular formation of the raphe and its contiguous fields, as well as the arciform fibres, was not obliterated. On the contrary, it appeared even more distinct than on the healthy side; as it were, every detail being exaggerated.

MICROSCOPIC CHARACTERS.

The characteristic feature of the diseased area is the presence, in enormous numbers and enlarged dimensions, of bodies which in every respect resemble the so-called granular elements of the neuroglia. The greater number of these bodies do not exceed the maximum dimensions of normal neuroglia elements, but some are half as large again, and a few nearly double that size. Together with the stroma, they make up the mass of the new formation. The stroma, in most part, particularly in the region corresponding to the reticular field, resembles the normal neuroglia. If any thing, the molecules appear a little coarser and more glisten-

ing. In other regions, particularly where regressive changes appeared to have been in preparation, there is a well-developed supporting frame-work of colorless, transparent substance, resembling the supporting mesh-work of the retina. It cannot be stained, as a whole, by any agent, but in some places it includes faintly-stained, round nuclei in the larger trabecula which make up the mesh-work. The nuclei before referred to could be brushed out of this supporting frame-work, just as the granular and ganglionic elements of the retina may be removed from the enclosing basis-substance by a similar procedure.

The blood-vessels have undergone a considerable hypertrophy, so much so that the contour of the diseased area can be pretty accurately traced by their injection. They are everywhere more numerous than on the healthy side, and contorted, zigzag, or of a corkscrew shape. They also show considerable distension, and this not evenly, but here and there, producing fusiform expansions of the vascular contour. The increased vascularity is particularly marked in those places which are naturally most vascular; thus the lower facial and the motor nuclei of the mixed system are practically converted into a convoluted maze of blood-vessels. As these stain in clearest contrast in fuchsine, sections prepared with the latter agent were used to make the drawings of figures 3 and 4 from.

In several instances a yellowish amorphous substance is found in the subadventitial space of the blood-vessels; and this occurrence is found limited to the diseased area. Otherwise the walls are normal.

The scattered nerve elements found in the mesh-work of the reticular substance and in the upper strata of the trapezium are much larger on the affected side than on the one which in this level is unaffected. I find every transition between the smaller as well as the medium-sized ganglion cells and the characteristic elements of the new formation. Usually the processes are not as distinct as in typical nerve cells, but in a few cases even this requirement of the standard ganglion cell is complied with.

A number of foci are found within the growth which do

not stain as the rest does. On examining one of these foci, situated at the border of the subendymal gray substance, midway between the abducens nucleus and the lateral angle of the ventricle, a beautiful transition is observed between free nuclei of the neuroglia and round vesicular bodies scattered through a finely molecular substance surrounding and merging into the pale area. The transition is best expressed as follows:

1. Typical neuroglia nuclei.
2. Ditto, enlarged, pale, and surrounded by a little protoplasm.
3. Ditto, and surrounded by a clear space.
4. Vesicle containing nucleus, surrounded by a little protoplasm.
5. Simple vesicle, without nucleus.

This transition corresponds exactly to that claimed by Hubrich[1] and Forel[2] for certain clear bodies found in the gray substance of lower mammals and in the cortex of paretic dements.

As to another form of body occurring in the affected area, I am unable to offer any explanation. It seems equally to present every transition to the neuroglia nucleus, but is entirely different from the ganglionic as well as the vesicular elements just described. Such elements stain a little more deeply in hæmatoxylin and of a higher shade in carmine than the former; but to fuchsine and other anilines they react in the same manner. The smaller of these bodies contain a distinct nucleus, and traces of such may be seen in the larger; but the very largest appear perfectly homogeneous and nearly hyaline. In many places, particularly the one furnishing the accompanying illustration, they were so closely crowded as to influence their contours, and the most bizarre shapes resulted, reminding one of other forms of neoplasm (figure 2).

In some instances these bodies seem to have undergone dehiscence, leaving a crumpled, transparent capsule behind. Evidences of this kind are found in the neighborhood of

[1] *Zeitschrift für Biologie*, Band ii. page 391.
[2] *Archiv für Psychiatrie*, Band vii. page 447.

the cavities to be referred to. Clear vesicles, occasionally indenting the contour of the hyaline masses, are also noted here. They resemble those previously described.

In the greater part of the affected area not a single normal nerve fibre can be identified as divided in transverse section. But those fibres running in the plane of the section, such as the *fibræ arcuatæ* and most of the nerve-roots, are not only structurally normal, but react normally to the staining-tests of Weigert. All vestiges of the normal nerve-cells of the facial nucleus (main nucleus) have disappeared,

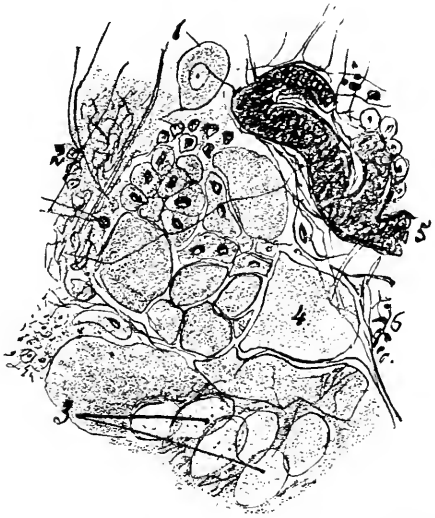


FIG. 2.—1. Element with nucleus, and process. 2. Neuroglia. 3. Vesicular elements in protoplasmic map. 4. Large irregularly contoured elements. 5. Blood vessels.

but in the hypoglossal and vagus nuclei the process of death can be distinctly studied. The cells maintain their outline, but within a transparent membrane—if it may be so called—reproducing the cell outline, the protoplasm is found in irregular and separated fragments. This is true of the processes, as well as the body. The nucleus is indistinct. As regards the majority of the normal cell-groups in the affected field, they were either destroyed or crowded out of sight by the abnormal elements. At all events, they cannot be identified.

The few nerve-tubes of sagittal direction remaining intact within the affected area have hypertrophic myelin. Between the zone where such tubes exist and the area of maximum lesion, as well as in the latter, there are a number of clear spaces, resembling those described in the spinal cord as spots of vesicular degeneration by Leyden.[1] These clear spots are a rather accurate gauge of the extent of the lesion in the great fibre systems, since they produce a peculiar spongy appearance, visible under a low power, and even to the myopic naked eye.

The ventricular granulations referred to in describing the recent anatomical appearances, have the usual structure—a fibrillar connective tissue, with included nuclei, occasionally overgrowing and imbedding the endymal epithelium.

On the unaffected—or, rather, the least affected—side of the oblongata there is an exquisite development of spider-cells throughout the entire reticular field, particularly in the triangle bounded by the raphe, vagus roots, and olive. Remarkably enough, no such bodies are found in the area of the neoplasm. These spider-cells are found in greatest abundance in the auditory and upper hypoglossal levels. None occurred in the abducens or decussation altitudes.

In several places the sections of the new formation exhibited gaps. An irregular one is situated immediately caudad of and within the concavity of the right genu facialis. It measured 2 *mm.* in its greatest diameter. A second is situated at the bend of the roots of the vagus, involving in some levels the trineural fasciculus (solitary bundle). A third is found in the reticular field just mesad of the right pneumogastric roots, measuring 3 *mm.* in its greatest diameter. A fourth lies directly in the line of the right abducens roots, interrupting their continuity. A fifth lies at the upper border of the interolivary field, opposite the hilum of the olive; this has been already referred to as being present in the recent specimens. The other four evidently resulted from the sponginess and crumbling of the tissue, and were found situated in the centre of those spots characterized by greatest vascularity. Not the slightest

[1] *Klinik der Rückenmarks krankheiten*, Band ii.

evidence of hemorrhage was found. The contents of many blood-vessels were in a condition of stasis.

TOPOGRAPHY OF THE LESION.

1. Level of the *locus cæruleus.* [Figure 3.]

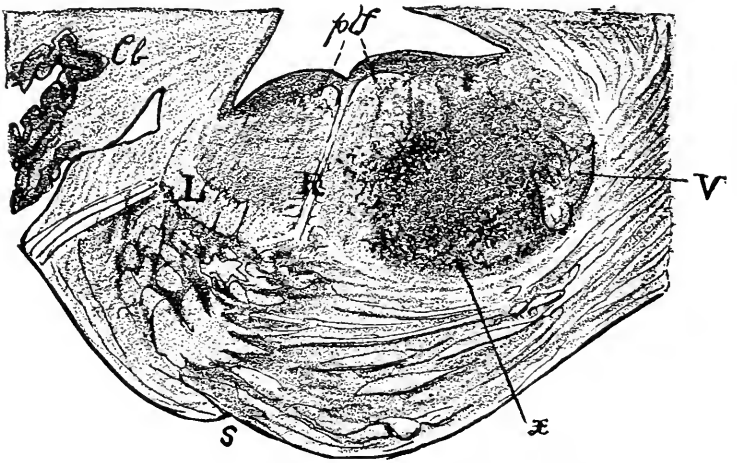


FIG. 3.—Transsection of pons, at lower level of *locus cæruleus plf* posterior longitudinal fasciculi; R, raphe; L, left lemniscus (intact); S, sulcus basilaris; V', ascending root of trigeminus, on the left side the level is a higher one owing to the asymmetry described in text; X, most vascular part of the new formation.

The diseased area is strictly limited to the right side; it occupies the reticular field of the tegmentum, and the lemniscus; it does not reach the raphe except at the junction of its dorsal third with its ventral two thirds; it does not affect the transverse fibre mass of the pons, the subendymal gray substance, the posterior longitudinal fasciculus, and the locus cœruleus. It slightly invades the right raphe root of the fifth pair. The contour of the focus of disease is therefore round, with a slight flare towards the upper third of the raphe.

2. Level of the abducens nerve-roots.

The diseased area is almost directly on the right side. There is a slight infiltration of the left internal division of the reticular field, not extending as far as the left abducens roots. The raphe is intact. The right posterior longitudinal fasciculus is partially involved by the "vesicular

degeneration "adverted to. The general contour of the lesion is the same as in the preceding level. The lemniscus in its middle third, the sagittal fibres of outer part of the reticular field, the lower facial nucleus, and the greater part of the facial root are entirely destroyed. The inner part of the right reticular field, the innermost part of the ascending root of the right fifth pair, and the right abducens nucleus are invaded.

3. Level of the *striæ medullares.*

In this level the encroachment of the lesion beyond the median line is greatest. The internal division of the left reticular field is invaded in its dorsal third; the left as well as the right nuclei fasciculi teretes are unidentifiable. The infiltration, aside from the encroachment on the left side, is bounded by the striæ medullares and the subendymal auditory nucleus above, the restiform body, and its tributary arched fibres laterally, and ventrad it is gradually lost towards the olivary halo. It sends an angular extension to the border of the interolivary layer.

4. Level of the hypoglossal nerves.

The parts destroyed in this level are the dorsal end of the raphe, nearly the entire right and the cephalic part of the left hypoglossal nuclei, the right vagus nucleus in its greater portion, the nucleus ambiguus and the trineural fasciculus (fasciculus solitarius) altogether. The limits of the focus are similar, but more contracted, except in the direction of the interolivary layer.

To complete the topographical survey of the tumor, it may suffice to add, that its cephalic end ceases as a gradual infiltration of the tegmental field at the lemniscus junction. Its caudal end, ceases in the gray matter, which is cut up by the fasciculi coming from the decussation of the interolivary layer. Both cephalad and caudad of the diseased area proper, however, there is considerable asymmetry in the sections.

We are now prepared to inquire into the effects of this abnormal growth on the normal structures of the pons and oblongata. As it involved an enormous hypertrophy of the diseased area, it caused a considerable asymmetry of the

brain axis. In the first specimen it had been observed, that while the sulcus of the fourth ventricle nowhere deviated from the straight line, and appeared to occupy the normal axial position, the basilar furrow of the pons deviated considerably to the left. The accompanying figures and the following table illustrate the asymmetry better than any description in words.

If the brain axis be supposed, for the sake of the illustration, to be composed of an expansible mass, and an area corresponding to the right tegmentum and right subventricular region be supposed to have undergone inflation, the

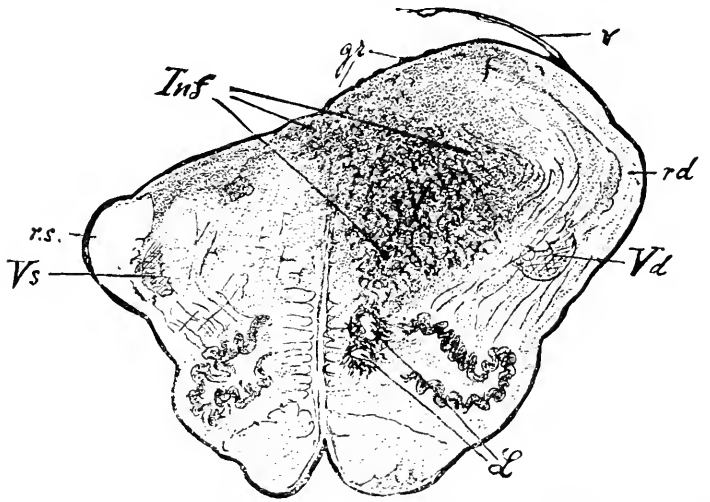


FIG. 4.—Section immediately caudad of the *striae medullares*, *v velum medullare*; *r.s.* left restiform body; *rd* right restiform column, flattened out but normal; *gr* ventricular granulations; *Vs* and *Vd*, left and right ascending trigeminal roots; *L* lacunæ at margin of interolivary larger; *Inf*, new formation infiltrating.

distortion in this case becomes clear. In the first place, as soon as the limits of intrinsic expansibility outward are passed, the neighboring parts will participate: the raphe becomes lengthened dorso-ventrad. The consequence is that the comparatively uninvaded left half of the oblongata becomes drawn out in the dorso-ventral direction, and consequently narrowed in the lateral direction. Nowhere is this illustrated more prettily than in the contrast offered to view by the restiform columns of opposite sides. (Fig-

ure 4.) On the left side, it is more compact even than in the normal oblongata; on the right side it is stretched out to a mere film on the outer side of the oblongata. Yet in absolute area the two are equal, the restiform being one of the tracts which has escaped disease entirely. The disorting influence of the neoplastic hypertrophy was not only exerted in the transverse plane. Considerable deformity resulted in the antero-posterior direction. Thus the right half of the pons, on its ventral face, appeared flattened out. In consequence it was wider cephalo-caudad than the more compact left side. Intrinsically the transverse and the longitudinal fibres of the pons were absolutely healthy.

The normal structures directly cephalad and caudad of the neoplasm had been crowded respectively forwards and backwards. Thus in strictly transverse sections in front of the disease focus, the elements on the right side represented what normally should have been a lower level; the corresponding left side representing a higher level. In the same way sections caudad of the growth represented always a higher level on the right than on the left side, inasmuch as the right side had been bulged out caudad. The olive particularly illustrated this; it had been so much compressed, that its cephalo-caudal diameter became shortened to the increase of its transverse area. In texture it, like the equally distorted right pons[1] half, is healthy.

With reference to individual parts of the pons and oblongata, the following recapitulation will show to what extent they are injured or destroyed.

LEFT SIDE. *Internal division of the reticular field:* destructively involved to the ideal continuation line of the hypoglossal nerve-roots, and to the extent of three millimetres dorso-ventrad, in the level of the medullary striæ. In front of and behind this level, a less intense affection, ceasing at the abducens and middle hypoglossal level, is found.

L. nucleus of fasciculus teres: destroyed.

L. hypoglossal nucleus: destroyed in its cephalic and middle thirds.

[1] Meaning only the pedal part of the pons, that which really represents a bridge under the tegmentum, uniting the cerebellar hemispheres.

L. glossopharyngeal nucleus: destroyed in its internal part, infiltrated in its outer part.

L. vagus nucleus (subendymal): infiltrated near the endymal border.

RIGHT SIDE. *Right posterior longitudinal fasciculus:* slightly involved on its border towards the abducens nucleus, healthy in every other level.

R. tegmentum; internal division: destroyed, with the exception of some fibres next the raphe and interolivary layer, in the transition level. In the abducens level it is nearly intact, being intensely involved, however, in the part pierced by the abducens roots.

R. tegmentum; external division: focus of disease entirely destroyed in trigeminus and facial levels, and with exception of part near olive and lateral field in oblongata.

R. lemniscus. In trigeminus levels the ectal two thirds are destroyed; the internal third[1] intact. The interolivary layer is intact in the abducens level but slightly infiltrated, and the seat of a lacuna at its dorsal-ectal border in the oblongata. (Figure 4.)

R. trineural fasciculus. Destroyed in cephalic half, greatly involved in caudal half.

R. trigeminus nerve. 1. *Raphe root:* slightly invaded. 2. *Motor nucleus:* considerably involved. 3. *Sensory main nucleus:* involvement doubtful; *ascending root:* intact, except at exit level, where about the inner one sixth exhibits a slight infiltration of the septa; the nerve fibres themselves unaffected.

R. facial nerve. 1. *Proper nucleus:* destroyed; *root:* ,' fatty " degeneration at genu and dorsal third of emerging ramus. Singularly enough, the peripheral root shows no other anomaly than a deficient reaction to fuchsine. It stains normally by Weigert's hæmatoxylin method.

R. abducens nerve; nucleus: considerably involved; *roots:* reduced in number, and interrupted in their course by a lacuna. Between the nucleus and the lacuna they are nearly as well developed as those of the healthy side.

R. hypgolossal nerve; nucleus: almost entirely de-

[1] " Bundle from pes to tegmentum " of some.

stroyed, except a few cells on the border of the vagus nucleus and the extreme caudal tip of the nucleus in the level of the interolivary decussation, no trace of its normal elements can be found. *Nerve :* Some normally-stained fibres, about one third in number those of the opposite side, preserved.

R. glossopharyngeal nucleus : destroyed.

R. vagus nucleus : destroyed in its ventral two thirds; slight infiltration of the remainder. The dark color is due to increased vascularity.

R. vagus nerve : half the normal diameter; normal fibres, except for a defective reaction to fuchsin.

R. deep nucleus of the mixed system :[1] destroyed, and the emerging rootlets cannot be identified.

Both on the left and right side, the anterior pyramids, the sagittal and transverse fibres of the pons proper, the olivary nuclei, those arciform fibres which pass through the olives, the restiform columns, the "internal division[2] of the cerebellar peduncle," and the auditory nerve-roots are entirely free from disease. The right subendymal auditory nucleus show a richer vascularization than the left, but there are no destructive changes. The right *nucleus magnocellularis* of Deiters, exhibits considerable infiltration in the molecular net-work, but the cells and fibres themselves appear as normal as those of the left side. The striæ medullares are normal up to within a millimetre of the median line, where (in the fresh specimen) they became lost in the gelatinous transformed mass. Microscopically, the fibres were found normal up to this point.

A singular feature is the healthful appearance of the peripheral nerves, considering the atrophy of some of their roots. The right facial is nearly entirely degenerated at the *genu ;* the right abducens cut across by a lacuna, and the right hypoglossal represented by fewer radicles than the left. Aside from a diminution in size and number of the nerve-tubes in the right facial, abducens, and hypoglossal

[1] The probable axial nucleus of the pharyngeal and laryngeal nerves, "Architecture and Mechanism of the Brain," JOURNAL OF NERVOUS AND MENTAL DISEASE, 1870.

[2] Regarded by some modern investigators as an auditory root.

nerves, nothing abnormal could be found in them. Probably the total destruction of the nuclei and the interrupting lesions had occurred too recently before death. In the intra-cerebral course of the facial, degenerative changes were advancing towards the periphery. As to the glossopharyngeal and vagus rootlets a number of them were lost in preparation, and nothing positively abnormal found.

In attempting to establish a relation between the symptoms and the lesion in this case, we must assume a progressive involvement of various centres and tracts, beginning at that point which lies in the centre of the morbid growth, and which corresponds to the functional disturbance. It happens that both indications point to a spot corresponding to the reticular formation of the tegmentum, near the nuclei at the floor of the fourth ventricle and in the plane of the abducens and pneumogastric nerves.

In the following table I have attempted to indicate the chronological and topographical relations of the main symptoms.

Date.	Symptom.	Explanation.
	MOTION OF THE EXTREMITIES.	
1884, Feb.	Dragging of the left leg.	The temporary paresis (?) cannot be accounted for; the ataxia of movement resulted from disturbed muscular sense; see the latter.
1884, May.	Normal again in all respects.	
1884, July.	Ataxia of movement on left side.	
	CUTANEOUS SENSATION OF THE EXTREMITIES.	
1883, Oct.	Subjective left heminumbness.	The lesion began in the reticular field of the tegmentum, probably intermediate to the raphe and ascending trigeminus root. Owing to the total destruction of this tract, the question arises, Are there other cutaneous sensory tracts, sensation not being entirely destroyed, but merely perverted?
1884, Jan.	" " sensation of pins and needles.	
	Subjective left paræsthesia (cold feeling).	
1884, May.	Pressure sense of left side impaired.	
	MUSCULAR SENSE OF EXTREMITIES.	
1884, May.	Doubtful impairment on left side.	The interolivary layer of the right side was involved late, the lemniscus earlier.
1884, July.	Decided impairment on left side.	
	CRANIAL NERVES—PUPILS.	
1884, May.	Indolent reaction to light, tendency of pupils to remain in contraction after accommodation and exposure to light. This symptom thenceforth stationary.	In most focal pons affections reflex iridoplegia has been observed; the lesion in them extended further cephalad and further dorsad in cephalic levels.
	CONJUGATE EYEBALL MOVEMENTS.	
1884, March.	Dancing motion of objects.	This case corresponds to those described by others: involvement of the reticular field near the posterior longitudinal fasciculus.
1884, May.	Associated movement to the right beyond the median plane, entirely destroyed and nystagmus. Former stationary, latter decreases.	

Date.	Symptom.	Explanation.
	RIGHT ABDUCENS NERVE.	
1883, Sept.	Blurred vision, temporary.	Early, slight involvement; later, destructive lesion in root transit and more serious involvement of nucleus.
1883, Nov.	Temporary diplopia.	
1884, May.	Paresis.	
1884, July.	Nearly total paralysis.	
	FIFTH PAIR, SENSORY BRANCH.	
1884, May.	Subjective numbness of R side of face.	Slight involvement of sensory ascending and raphe root, later, of sensory nucleus of exit level.
1884, July.	Subjective sense of heat on R side of face.	
	FIFTH PAIR, MOTOR BRANCH.	
1884, March.	Tightness of jaws.	At first, irritative lesion of; later, slight infiltration of motor trigeminus nucleus.
1884, April.	Continued difficulty of mastication.	
1884, May.	Paretic weakness of R masseter.	
	RIGHT FACIAL NERVE.	
1883, Oct.	Spasms of.	At first, irritative lesion; later, total destruction of.
1884, May.	Total paralysis of all external branches, the uvula deviating to the right.*	
	RIGHT AUDITORY NERVE.	
1884, April.	Buzzing in right ear without assignable cause.	Possibly hyperæmia of nucleus, which was found injected.
	TASTE.	
1884, May.	Slightly impaired (delayed appreciation) on right side.	R solitary bundle and R glossopharyngeal nucleus destroyed. L glossopharyngeal nucleus involved later on.
1884, July.	Profound bilateral impairment, not accurately mapped out.	
	DEGLUTITION.	
1883, Sept.	Spasmodic disturbances of.	Subendymal and insular nuclei of vagus and accessory nerves first irritated, then destroyed, or nearly so.
1884, Jan.	Pharyngeal paresis (nasal, voice, etc.) general.	
1884, March.	Eructation difficult.	
1884, July.	Deglutition progressively becomes paralyzed.	
	LARYNX.	
1884, May.	Normal.	The insular nucleus of the "mixed system" was destroyed.
1884, July.	Undetermined.	
	GASTRIC.	
1883, June.	Nausea, vomiting, and irritability of the stomach, which can be obviated to some extent, as in sea-sickness, by remaining motionless, continues nearly till death.	Subendymal nucleus of vagus nearly destroyed after preceding irritative infiltration.
1884, June.	Bulimia.	
1884, July.	Anorexia.	
	RENAL.	
1884, May.	Urine normal (Dr. Powell).	The affection of both vagus nuclei must account for the polyuria.
1884, July.	Polyuria (?).	
	CARDIAC.	
1884, March.	Intermission of the heart-beat at long intervals, increasing in frequency.	Ditto

* It is recorded in several cases of total facial palsy that the uvula deviated in the direction contrary to the one regarded as the rule.

Date.	Symptom.	Explanation.
1884, July.	Heart's action intermittent and irregular.	
	HYPOGLOSSAL NERVE.	
1884, May.	Tongue, as a whole, deviates to the right with inclination of the tip to the left.	The hypoglossal nuclei were both involved early, in their upper portion, and nearly destroyed, *sub finem*, together with their raphe connection.
1884, July.	Deviates to the left as a whole, is the seat of fibrillary tremors, is asymmetrical, the right side being the larger; protrusion is difficult; *sub finem*, complete anarthria supervenes.	
	CERVICAL MUSCLES.	
1884, July.	Oscillatory motion of head, with passive rotation of head to left.	

As I am engaged in preparing a monograph on the lesions of the oblongata, I limit myself for the present to giving a history of this case and its findings, trusting to discuss its points of agreement and contrast with the cases of others, at no remote date, in the paper referred to.

712 Lexington avenue, July, 1886.

ON CONVULSIVE TIC WITH EXPLOSIVE DISTURBANCES OF SPEECH (SO-CALLED GILLES DE LA TOURETTE'S DISEASE).

By C. L. DANA, M.D., and W. P. WILKIN, M.D.

DEFINITION.—The affection above indicated is characterized by sudden convulsive movements accompanied with inarticulate foul or profane ejaculations.

HISTORY.—Described by Bouteille in 1818 in his "Traité de Chorée."

A case reported in 1825 by Itard, quoted later by Tourette.

Somewhat similar cases referred to by Trousseau in his "Clinique Medicale," vol. ii., 5th ed., p. 267.

The Jumpers of Maine, believed to be cases of the same kind, described by Dr. Geo. M. Beard in 1880, JOURN. NERV. AND MENT. DIS., vol. vii., and by Dr. G. B. Thornton, *Med. Record*, Dec. 26, 1885.

The Myriachit of Siberia, a similar disease, was described by Dr. Wm. A. Hammond in 1884.

The Latah of the Malayese, and a similar affection in Kamskatcha, are referred to by Beard, Hammond, and Tourette. The disease has been described by a non-medical man only, Mr. O'Brien, but it is thought to be identical with convulsive tic.

In 1884, M. Gilles de la Tourette collected previous observations and reported a case of his own (*Arch. de Neurol.*, July, 1884).

In 1885 this same author (*Archives de Neurol.*, January and March, '85) presented and analyzed nine cases observed in France.

He described his cases under the title, "A Nervous Affection Characterized by Motor Incoördination, Accompanied with Echolalia and Coprolalia."

In January, 1886 (*Rev. de Médecine*), Dr. Geo. Guinon reported four more cases. These included two cases on whom M. Charcot delivered a clinical lecture published in *la Riforma medica*, Nos. 184, 185, and 186. Guinon's cases resembled Tourette's, but showed more mental impairment—two of them having *idées fixés*.

In March 1886, Dr. I. I. Pautynkhoff reported (*Russkaya Meditizina*, March 16th) "a case of imitative disease," resembling those of Tourette's, the patient showing echolalia and echokinesis (imitation of speech and movement). Patient had defective intelligence.[1]

M. Lannois ("Nosographie des Chorea," 1886) makes some comments upon this affection, but contributes nothing new.

Dr. T. C. Railton (*Medical Chronicle*, April, 1886) describes a very typical case.

As no case of the *tic convulsif* observed and described by Tourette and Guinon has as yet been reported in this country (aside from the general descriptions of the Jumpers), we have thought that the following case would be of interest and might call the attention of physicians to a disorder of a quite well-marked type, but as yet, not at all well or widely known.

The history of our patient is as follows:

CASE I.—M. W., boy, twelve years, U. S. Parents healthy; brothers and sisters healthy—excepting one sister who is of a nervous hysterical temperament.

This boy has been backward in development, both physical and mental. Has always been of an irritable, excitable, and impetuous temperament. Has had three convulsions, the first in first year, the second in second year, and the third when six years old; none since that time. In his studies he is very backward; was unable to advance beyond the alphabet for several years.

Last Christmas he fell and sprained his foot, since when he developed the trouble which forms the subject of this paper.

[1] I am greatly indebted to the courtesy and linguistic attainments of Dr. Thomas A. Stedman, who translated this interesting case in full for me.—C. L. D.

His parents gave the following history : He had never been profane or obscene in his language up to about December last. It was then noticed that when sitting quietly he would suddenly and involuntarily burst out into expressions of the most profane and obscene character ; repeating them rapidly for a few moments and then stopping. A surprise or sudden noise of any kind was liable to bring on an outburst. He did not seem to understand the significance of the words used. It was necessary to send him from school, as besides disturbing the other scholars his example was, of course, hurtful.

The word which he used oftenest, repeating it rapidly and automatically, was the equivalent of the French word "merde," which is the word quoted by Tourette as most frequently expressed by his patients. He used to be much worse mornings, and before or during his toilet would regale the house with his automatic blasphemies so loudly as to disturb the neighbors.

Besides this condition of coprolalia he developed echolalia, though to a lesser extent.

He would repeat not only the last words but the ends of sentences ; and do it automatically imitating to a considerable degree the tone and accent as well as the words of the speaker.

Besides this the patient seemed forced to utter confessions of the things that he most wanted to conceal. When he found that he has thus automatically exposed his own misdeeds, he tried to make up for it by denials. The boy was a good liar. As an illustration of the forced confession which he made, his brother stated that on one occasion when his mother was away he broke a dish. He asked his brother not to tell about it, and a plan of concealment was arranged. The moment the mother appeared, however, the boy burst out : "I broke the dish." Then realizing his mistake, he added, quickly : "No, I did n t, either."

When examined by us he was observed to be anaemic, but fairly well grown and nourished. He has choreic movements in the face, and slight convulsive movement in legs and arms. When he is making a strong effort to repress the obnoxious words, or when he is under any restraint, the movements are more prominent. The choreic movements, as stated, affect his face and shoulders most. He had also a kind of expiratory spasm, producing a noise like a half-developed cough. If more marked, it might be put down as an illustration of laryngeal chorea.

The history showed that at first the speech disturbances were most marked, later the convulsive movements were prominent, and finally the speech disturbance nearly disappeared and the movements alone were noticed. This is the reverse of the order noted in Tourette's cases.

He has no ataxia or other sensory disturbances.

His reflexes were normal. His vision was apparently normal to ordinary tests. Urine normal. Appetite and sleep normal. The fact that the boy's language and behavior were the evidence of disease became very manifest to the parents. At first he was

soundly and systematically punished. It was found, however, that just as soon as the flogging was over, the boy would look up and repeat the same obscenities.

The treatment has been simple and quite effective ; it consisted essentially of iron, arsenic, and bromide potassium.

The GENERAL HISTORY of the disease is as follows :

It begins, as a rule, in children between the ages of six and sixteen years, and affects, by preference, the masculine sex. There is almost always a neurotic family history. In Maine, the disease is an hereditary, or family one.

The exciting cause is generally unknown, but sometimes powerful emotions start up the symptoms. Tickling is said, by Dr. Thornton, to be the cause of the Jumpers.

If we admit the unity of the various disorders already referred to, climate and race plainly have something to do with its prevalence.

The disease begins almost without exception by attacks of motor incoördination, affecting generally the head, face and upper extremities first, then involving the whole body, The movements can be controlled for a time by the will, only to break out with increased violence later. They cease entirely during sleep, which is generally very profound. M. Gilles de la Tourette states that the disease always begins with the motor disturbances, and that the symptoms may remain limited to the motor sphere. But this is not always the case evidently, as was shown by our patient, in whom the psychical symptoms developed first, and at first greatly predominated.

The physical condition of these patients is good ; sleep, appetite, and general nutrition are not seriously impaired.

The mental state is also, as a rule, good, but a few of the patients have some mental defect. The patients are perfectly aware of the incongruity of their expressions. They can even control their movements and language for a time. No disturbance of the special senses, and no other neuroses, such as hysteria and epilepsy, are known to be associated with the disease.

The most striking and peculiar feature of the disorder is that which relates to the speech.

After having suffered from attacks of motor incoördination for a time, the patient will, with the attacks, utter inarticulate cries, or he may begin to repeat or echo the words that he overhears. All this is done automatically and suddenly, with the accompaniment of grimaces and muscular contortions.

This echoing of speech is accompanied by an echoing of gestures (echokinesis).

Tourette records the curious illustration of a young woman suffering from this disorder who retired to rest one night, when a dog began to bark under her window. The unfortunate patient echoed the bark, which the dog took up in turn, and, against her will, she was kept barking the greater part of the night.

A still further peculiarity of this disease, and one which Tourette regards as pathognomonic, is the sudden interjection by the patient of obscene words and expressions. This symptom is the last in the series, and was observed in five out of nine cases. It was not noted by Beard or Hammond, but has been observed by Mr. O'Brien in his description of Latah.

It appears to us that the explosive and automatic character of speech disturbances is more the essential feature than the echoing or obscenity. It is as if the inhibition were removed from the lower and interjectional speech centres, leaving them over-sensitive and responsive.

A characteristic not noted by Tourette is the imperative and explosive utterances of things most desired to be kept secret, as was illustrated in our case.

The disease is a chronic one, beginning insidiously and lasting for years, sometimes for a long lifetime.

Beard said, "Once a Jumper, always a Jumper." None of Tourette's cases were cured, though some were very greatly ameliorated.

The diagnosis of the disease is not difficult. From chorea it is distinguished by the suddenness and larger range of the involuntary movements, and by the fact that a muscular explosion is followed and preceded by complete quiet. From so-called diaphragmatic and laryngeal chorea it is di-

agnosticated, according to Tourette, by the fact that in the disease he describes the cry is always accompanied by a muscular convulsion.

Echolalia and coprolalia may form part of the symptoms of insanity, and coprolalia has been observed in post-hemiplegic aphasia. It is hardly necessary to show how easily these conditions can be differentiated.

The coprolalia is pathognomonic of the disease, according to Tourette.

As to the pathology of the disease, from its long continuance we can exclude any organic lesion; and from the general history it is apparent that it belongs to the neuro-degenerative disorders. It is an evidence, like epilepsy and paranoia, of family decay.

While the disease is certainly far removed from the ordinary chorea of Sydenham, which is a subacute disease, and runs a definite course, yet it does seem that some of these cases are closely allied to certain aberrant and peculiar forms of chronic chorea not unfrequently met with. Thus, one of us has now under observation a boy with chronic chorea, which has lasted, with some intermission, for three years. This boy, at one period, was a "jumper," although his movements were not nearly so violent as those described by Beard and Tourette. In another case of chronic chorea, the boy was often seized with violent incoordinate movements. It may be, therefore, that we shall find cases shading all the way from well-marked Latah or Myriachit to something like chronic chorea.

In the treatment, one measure has proved of marked value, and that is isolation. Tonics, bromides, arsenic, electrotherapy, have caused some amelioration.

In our own case, bromides, arsenic, and iron have produced great improvement.

Periscope.

PATHOLOGY OF NERVOUS SYSTEM.

Histological Changes in the Muscles in Cases of the "Juvenile Form" of Dystrophia Muscularis Progressiva. By W. ERB (*Neurolog. Centrbl.*, July 1, 1886).

In the last number of this JOURNAL we reviewed Erb's monograph on Thomsen's disease, and in that review referred particularly to the histological changes in muscles affected by that disease. In connection with that monograph the above article is of commanding interest. During the past few years Erb has attempted to rearrange and properly classify the large number of diseases included under the term *Progressive Muscular Atrophy.* For those forms of progressive muscular atrophy which are due to disease of the anterior horns of the spinal cord, Erb proposes the name of amyotrophia spinalis progressiva. To this he opposes what he calls cases of *dystrophia muscularis progressiva*, under which head he includes cases of infantile pseudohypertrophy of the muscles, of what Leyden termed hereditary muscular atrophy, and Erb's juvenile muscular atrophy. Duchenne's "Atroph. muscul. progressive de l' enfance" would probably fall under the same head.

It is the "juvenile form" which we are now concerned with; the histological changes of the muscles in this disease have hitherto been but imperfectly known. The condensed history of Erb's case is as follows:

Bohemian Butcher, æt. forty-one, in Erb's clinic from Feb. 19th to March 18, 1886. No hereditary trouble; no syphilis; had conjunctivitis, typhoid, dysentery, carbuncle, hemorrhoids. At the age of thirty-four fell into a quarry to a depth of sixty feet; struck in the small of back by a stone; was unconscious; was in hospital six weeks; no fracture; was able to work after ten weeks. One to one and a half years later present trouble began with weakness in the shoulders, emaciation of upper arm, inability to use arms; is easily fatigued on walking; a feeling of tightness in legs; no other pains in arms or legs; no rigidity; entire nervous system normal. The following changes had taken place in

the muscles : Atrophy and paresis of the pectorals (with exception of small portion of the pars clavicularis), of the trapezii, the rhomboidal, the serrati antic. mag., of both latissimi, of the biceps, brach. antic. and supinator long. of both sides, of both triceps, and of the extensor muscles of the back. There was *hypertrophy* of the deltoids, the flexors of the right forearm, the supra- and infra-spinati, and possibly of the subscapular and of the mm. teretes. Atrophy of left thigh and right gluteal muscles. All other muscles mormal or nearly so. Sensation normal everywhere, cutaneous reflexes normal. Tendon reflexes present but weak. No triceps reflex. Sphincters normal. No fibrillary movements. Mechanical and electrical excitability simply diminished ; no R. D.

A small piece of muscle was removed from the hypertrophied right deltoid and from the slightly atrophied right biceps. Deltoid exhibited, on cross-section ; all muscular fibres considerably hypertrophied, with the exception of a few that are atrophied. Average diameter 15–170 μ. (normal average 40–60 μ.). The largest ten fibres measured 130–170 μ., the smallest ten fibres 15–40 μ. The fibres are well rounded instead of polygonal, and are farther apart than normally ; some fibres exhibit the peculiar vacuolization referred to in the review of " Thomsen's Disease." Connective tissue is slightly increased ; the blood-vessels exhibit thickened walls and increased numbers of nuclei. About the same changes are exhibited on longitudinal sections.

The biceps seems to exhibit the disease in its more advanced form ; some of the bundles resemble those of the deltoid ; other bundles exhibit a smaller number of hypertrophied fibres and a larger number of smaller fibres (very much below the average). The individual fibres are widely separated from one another ; vacuolization has not been observed in these. Connective tissue increased and a segmentation of the muscular fibres. Vessels thickened ; no fatty infiltration.

The important points to be noticed are that the chief changes are in the muscular fibres themselves and not so much in the connective tissue, and that these changes consist in simple hypertrophy, proliferation of the nuclei, division and vacuolization of the muscular fibres. Later on in the disease simple atrophy is superadded, but there is no fatty or other degeneration of these fibres. The histological changes in the juvenile dystrophy Erb considers very similar to those commonly found in cases of infantile pseudo-hypertrophy. B. S.

De l' Atrophie Musculaire dans les Paralysies Hystériques. Dr. J. BABINSKY. *Progrès Médical*, 1886, I., p. 329.

From Charcot's clinic B. reports four cases of muscular atrophy occurring in hysterical paralysis. The absence of trophic symptoms has always been considered a cardinal diagnostic point between paralysis due to hysteria and paralysis due to other causes.

Now, however, in view of the present facts this negative symptom loses part of its value. In two of these cases the paralysis was monoplegic ; in the other two hemiplegic, without facial paralysis. The characteristics of this hysterical muscular atrophy are : 1st. It is more or less extended. 2d. There are no fibrillary twitchings. 3d. The idio-muscular excitability appears to be normal. 4th. Electrical excitability diminished in proportion to the amount of atrophy, but no degeneration reaction. 5th. The atrophy may come on very rapidly. 6th. Its retrogression appears also to be rapid. B. looks upon this atrophy as a simple one, that is not dependent upon any material lesion of the gray matter of the cord or of the peripheral nerves. He also believes it to be distinctly trophic, the nervous system however not presenting any change recognizable by our methods of investigation ; in short, a purely dynamic alteration, analogous to the changes which Charcot believes take place in the spinal cord in the atrophies consecutive to joint lesions. The important part of this communication is not so much in the theory of production as in the fact that an atrophy of a paralyzed muscle may be due to hysteria.

Note Relative à l' Existence de la Névrite Segmentaire Périaxile, à Propos d' un Cas de Paralysie Diphtheritique. A. GOMBAULT. *Progrès Médical*, 1886, p. 472.

A recent article by Pitres and Vaillard in the *Archives de Neurologie* again directs attention to the lesions found in peripheral nerves in diphtheritic paralysis. These investigators found segmentary neuritis, together with lesions indicative of Wallerian degeneration. The segmentary neuritis was not periaxial—that is to say, that in every place in which the myeline was affected the axis-cylinder had disappeared. Gombault now shows that besides this form of neuritis there is also another which is the periaxial one. In the specimens which form the basis of the communication, the axis-cylinder could be traced through the affected part of the nerve and was in direct continuity with the healthy part. It had, however, not completely preserved its normal characteristics. In parts it was swollen and flattened. In the three cases which the author has observed the neuritis was distinctly periaxial.

G. W. JACOBY.

Some Forms of Paralysis Depending upon Peripheral Neuritis. By THOMAS BUZZARD, M. D., *Lancet*, 1885.

Neurologists in Germany and France within the past few years have been devoting considerable attention to the various forms of peripheral neuritis and the paralyses resulting therefrom. An impetus was given to the study of these pathological conditions by an outbreak, within the past few years, of endemic paralysis that occurred among Chinese and Japanese coolies, wherever these laborers were to be found. Scheube was among the first to make

careful autopsies of fatal cases. He almost invariably found an intact brain and cord, while the nerve trunks and branches were in a condition of parenchymatous degeneration. The disease in China and Japan is known as Beri-beri, and has occurred as epidemics in these countries for a great number of years.

Dr. Thomas Buzzard, in the series of Harveian lectures, 1885, treats the subject of paralysis depending upon peripheral neuritis rather from a clinical than from a pathological standpoint (*Lancet*, Nov. 28, Dec. 12 and 19, 1885). A typical example of localized peripheral neuritis is first portrayed, followed by a series of clinical views of cases that have occurred under the author's observation. He distinguishes, pathologically, two forms of neuritis: interstitial neuritis, in which the connective tissue of the nerve is the primary seat of inflammatory changes, the essential element being secondarily affected; and parenchymatous neuritis, in which there is destruction of the essential element of the nerve fibres, with but little or, perhaps, no recognizable alteration in the interstitial tissue." The prominent symptoms for a typical case of peripheral neuritis are the following: marked paralysis, loss or diminished electrical reaction, agonizing pain of a lancinating character, a sodden, œdematous appearance of the affected member, the skin being glossy in patches and exhibiting a purple discoloration and occasionally a bulbous eruption. But cases are not always typical, and even localized neuritis may at times resemble a central disease. An instructive history of such a case is narrated. The diagnosis must then rest chiefly on concomitant circumstances. Electrical examination will afford great assistance in most cases, but the author emphasizes the fact that the resistance offered by the skin and subjacent tissues to the electrical current may sometimes vary in the two sides of the body. The strong relationship between gout and peripheral neuritis must be borne in mind in diagnosticating an obscure case of paralysis. The difficulties encountered in diagnosticating multiple neuritis are extremely great and at times almost impossible. As a rule, however, in multiple neuritis the sphincters of the bladder and rectum remain intact. That form of progressive multiple neuritis due to alcoholic intoxication possesses some special clinical features. The patient usually exhibits some mental disturbance, the memory is especially weakened, and there is a tendency to incoherent talk. Diphtheritic paralysis is held by the author to be dependent upon peripheral neuritis.

It is difficult to do justice to the many valuable and frequent observations that the lectures embody in a short abstract, and a careful perusal of the original is highly commended.

H. N. VINEBERG.

Absence of Patellar Reflex as the only Symptom of Locomotor Ataxia. Société de Biologie. Meeting of April 10, 1886.

DÉJÉRINE reports the case of a tuberculous patient who during life presented absence of the patellar tendon reflex. He had never presented any other symptoms of locomotor ataxia, no lightning pains, no ocular symptoms, no incöordination, etc. At the autopsy, the spinal cord, which was examined with the greatest care, as also the peripheral nerves, were found to be absolutely normal. This case proves that the patellar reflex may be absent without any other symptom, or any characteristic lesion, of locomotor ataxia being present.

Ablation of the Motor Centres. Meeting of April 17, 1886. DUPIN, presents a dog in whom he has removed the motor centers. This dog is able to execute all movements. Laborde, remarks that the dog presents a certain amount of ataxia of gait.

Radial Paralysis due to Compression.—DÉJÉRINE communicates the results of researches made by himself and Vulpain upon six patients affected with radial paralysis due to compression. The cause of this paralysis is always the same. All the muscles are affected, except the triceps, which generally escapes. The duration is always long, at least five to six months. The faradic excitability of the radial nerve is normal. Excitation above the point of compression does not elicit any contraction of the muscles. The nutrition of these muscles is not changed ; they are not at all atrophied, except, perhaps, the supinator longus, which is slightly diminished in volume. Besides this, the muscle presents the signs of degeneration reaction. The main characteristic of this paralysis, then, is a nerve which does not allow the will to pass, but which preserves its faradic excitability.

Vulpian and Déjérine have endeavored to reproduce this paralysis upon animals, but they have not been able to succeed. The question as to the exact nature of the lesion is still an open one. It is certain that the axis-cylinder cannot be affected. Brown-Séquard believes that in these cases it is a peripheral excitation which produces an inhibition upon the spinal cord. The compression, which is insufficient to produce a degeneration of the nerve, is, however, sufficient to produce an effect upon the nervous centres. It is probable that in such patients, secondary alteration of the spinal cord will be found.

Physiological Action of Hypnone.—QUINQUAND presents a communication relative to the action of hypnone upon the blood. As a result of injecting 2 to 3 cubic centimétres of hypnone into the circulation of a dog, an increase of carbonic acid in the arterial blood was found ; also an increased consumption of oxygen. Therefore if a sufficient quantity be used, all the phenomena of asphyxia are produced. All the animals experimented upon died. In practice, therefore. hypnone must be used with the greatest caution.

Alteration of Peripheral Nerves in Chronic Articular Rheumatism. Meeting of June 12, 1886.

PITRES and VALLARD communicate the results of their studies

upon this subject. Chronic articular rheumatism, they say, by its clinical course and symptomatic manifestations, recalls certain forms of trophic affections. It is generally acceded that the nerves are not affected in this disease, but P. and V. now show that this is not always the case. In two of their cases deep and diffuse changes were encountered. These two patients, during life presented all the pronounced symptoms of arthritis deformans, with severe osseous changes and also trophic affections of the skin and nails. The nerves were found to be the seat of parenchymatous neuritis. Whether the peripheral nerves are always thus affected, or whether the neuritis is simply due to a special localization of the rheumatism, are questions which the authors do not attempt to solve. G. W. J.

MENTAL PATHOLOGY.

Paretic Dementia and Cerebral Lues.—Dr. Hugo Engel (*Medical Bulletin*, July, 1886) in defence of his position. J. McC——, after having suffered from headache, worse at night, for a considerable time, about a year or so ago, upon one night appearing before the public, had suddenly shown symptoms of aphasia, and of a loss of memory in general. From that time his mental faculties began to decline. After these symptoms of mental decay had been in existence for some time, paresis attacked various groups of muscles, and more decided psychical disturbances made their appearance, but he never exhibited any ideas of magnificence or grandeur. A person standing by the bed of the patient, while the latter was dozing, would have never imagined that he had a very sick man before him. The face looked well nourished, and there was nowhere to be seen that emaciation usually observed in far-advanced cases of paretic dementia. Instead of it, and while his complexion was almost florid, paled somewhat by long confinement, there was noticeable, especially between his brows, that peculiar dirty-yellowish discoloration which he had never found absent in cases of chronic syphilis. All the facts pointed to this malady: the history, its mode of commencement, its progress with the absence of hallucinations of magnificence, the apoplectic seizure, the well-preserved general nutrition, the characteristic discoloration over the forehead. It is unnecessary to say, basing the opinion only on evidence here given by Dr. Engel himself, that he has never seen a case of paretic dementia, and furthermore that he is totally unacquainted with the literature, otherwise he would know that, as a rule, while true paretic dements are well nourished, luetic cases are as frequently of an emaciated type. Furthermore, apoplectic attacks are a characteristic phenomenon. Delusions of grandeur are frequently absent in pure paretic dementia on the one hand, while in syphilitic cases they are as frequently present. The results of treatment neither prove nor disprove any thing, as chronic cerebral syphilis in which secondary degenerative changes

have occurred is intractable to any treatment. Dr. Kiernan (*Alienist and Neurologist*, 1883) pointed out that there was no means of distinguishing luetic from pure paretic dementia, and in an article evidently suggested by this, Dr. H. C. Wood (JOURNAL OF NERVOUS AND MENTAL DISEASE, Periscope, 1885) enunciated the same opinion. John McCulloch, the patient referred to, is known, and can be proven to have exhibited grandiose delusions. So Dr. Engel is evidently unacquainted with the patient's full history. The discourtesy with which he publicly treated Dr. Nichols, of Bloomingdale (second to no American alienist), deserves a rebuke. No true physician would have, in the public press, proclaimed his ability to cure an affection pronounced practically incurable by the best authorities.

Surgery Among the Insane.—In 1881, Dr. W. A. Hagenbach (JOURNAL OF NERVOUS AND MENTAL DISEASE) called attention to the peculiarities of surgery among the insane. In 1882, Dr. Schule (quoted in JOURNAL OF NERVOUS AND MENTAL DISEASE, 1883) made an extended study of the subject. Dr. GINE Y PARTOGAS (*La Independencia Medica*, Año xxi.) calls attention to the various surgical affections found among the insane. Erysipelas, especially facial erysipelas, is common, and sometimes favorably influences the psychoses. Furunculosis and abscesses are common. Eczema and acne are to be very frequently found. Various self-mutilations from castration to œsophagotomy require attention. Herniæ are frequent. Insane patients do not very frequently complain of somatic affections, and hence constant care is needed.

Epileptic Insanity.—Dr. RODRIGUEZ (*La Independencia Medica*, June 21, 1886) finds that the types of epileptic insanity observed by him tend to corroborate the views of Fabret and Souet that there are ante, post, and equivalent, types of epilepsy. He finds, also, that the equivatent may take the form of an imperative impulse.

Mental Excitement after a Cataract-Operation.—Dr. THEO. W. FISHER (Report of the Boston Lunatic Hospital, 1885) reports the following case, which tends to corroborate the views expressed in the January, 1886, number of the JOURNAL OF NERVOUS AND MENTAL DISEASE as to the relation between senility, psychical disturbance, and cataract-operations: "Last summer an old lady, who has been an inmate of the hospital for thirty years, and who still retains considerable intelligence, was operated on for cataract by Dr. Wadsworth. She had been fond of reading and sewing, and the entire loss of sight by double cataract was a great deprivation to her. The operation was painless, under the use of cocaine, and was entirely successful, in spite of an

attack of excitement, which complicated the after-treatment. She is now able to read and sew, and enjoy her walks in the garden."

Increase of Insanity in the Aged.—A private insane hospital affords a good means of testing such an alleged increase. Dr. H. P. STEARNS (Report of the Hartford Retreat, 1885) says that the percentage of admissions of persons above sixty years, during the last ten years, has considerably more than doubled, as compared with that of the ten years from 1850 to 1860, or any earlier decade. This large increase of admissions among elderly persons may be due to any one or more of several causes. It may possibly indicate that more old people become insane than formerly. Or it may be due to the fact that friends and relatives are at the present time less tolerant of the eccentricities and peculiarities of persons affected by a decay of their mental faculties; or, again, it may arise from the fact that, owing to the increase of longevity during the last twenty or thirty years, there exist in communities a larger number of persons relatively to the whole population who are past sixty years, from which to recruit members as candidates for asylums. Dr. Clouston (Morningside Insane Hospital Report, 1885) calls attention to a similar fact. J. G. KIERNAN.

Reviews and Bibliographical Notes.

Nosographie des Chorées. Dr. Maurice Lannois. Pp. 172. Paris, 1886.

In this monograph the author makes use of the word chorea to embrace "almost all involuntary muscular movements, which are incessantly renewed, without rest or intermission, and only ceasing during sleep." The acceptance of this far-reaching definition of the word necessitates the description of a multitude of conditions which, although having certain symptoms in common, nevertheless are very far from being analogous.

These various conditions are divided by the author into three main groups: rhythmical choreas, pseudo-choreas, and arhythmical choreas. The first group embraces epidemic chorea, so called, and the actual rhythmical choreas.

Under the title of epidemic chorea are classed such affections as "tarentism," a name given on account of its supposed dependance upon the bite of a tarentula, "tigretier," an affection occurring along the course of the Tigris, and the "Jumpers." The "Jumpers" which are here referred to are certain sects of Methodists found early in the present century in Wales and Cornwall. They must not be confounded with the Jumpers of Maine. The jumping among the former constitutes part of their religious worship.

"The camp-meetings of the American Methodists of the far West" are also classed under this heading, and are described as an aggravated form of epidemic chorea.

Under "actual rhythmical choreas" are classed chorea magna and reflex saltatory spasm. In this chapter a very interesting case of hystero-chorea is described and illustrated by twelve instantaneous photographs. The execution of these pictures leaves a great deal to be desired.

The second group, the pseudo choreas, includes the tic de Salaam, electrical chorea, chorea of the larynx, and chorea of the diaphragm (singultus), the various tics convulsifs under which title Gille de la Tourette's disease is also classed, and lastly paramyoclonus multiplex.

Laryngeal chorea is justly acknowledged by the author as not

deserving the name ; chronic singultus is so well known as an hysterical symptom that the term diaphragmatic chorea is rather startling.

The third class, the ahrythmical choreas, embraces the chorea of Sydenham (sempichorea of Gowers and West, chorea of pregnancy, chorea of old age, and hereditary chorea), also hemichorea and hemiathetosis.

To each of these affections a special chapter is devoted, and it would be difficult to surpass the author in his accuracy of research and his exposé of the present state of our knowledge upon each of these points. Particular attention is also paid to the etiology and to the various symptomatic points of differentiation. The chapter on pathological anatomy, although containing nothing new, is an excellent resumé, facts being clearly separated from theories. All in all, the author has succeeded in producing a book which is replete with facts, of actual and historical interest, and full of bibliographical references. No future writer on this subject can well do without this monograph. G. W. J.

Des Vertiges. Dr. E. WEILL. Paris. J. B. Baillière et Fils, 1886.

This monograph of 120 pages treats of the various forms of vertigo, taking the word in its narrowest sense, for the definition adopted by the author is that of Grainger Stewart, namely, "The sensation of the instability of our position in space, in reference to surrounding objects." The classification of the book is not based, as is generally done in treating of this subject upon etiological conditions, but according to physiological principles. The physiology, and particularly the literature of the subject, is very complete, but certain unaccepted physiological experiments are taken as facts and deductions made from them which consequently can also not be accepted.

Vertigo is here classified into :

1. Vertigo dependent upon a disorder of the reflex apparatus of equilibration. Under this title is included vertigo due to organic lesions (Ménière's disease, lesions of the cerebellum, etc.), also V. dependent upon functional affections (neuroses, affections of the circulation, reflex, diatheses, toxic).

2. Vertigo dependent upon affections of the sensory mechanism of equilibration. Includes visual V.

3. Vertigo of complex causes. Includes rotary V. and seasickness.

The chapter devoted to Ménière's disease is the most complete.

In speaking of auricular, traumatic, gastric, and neurotic vertigos, the author does not enter at all into the manner of production, and although he seems to attribute vertigo in all its forms to an action, either direct or indirect, upon the cerebellum, he leaves us completely in the dark as to how this action is supposed to take place.

To the form known as neurasthenic vertigo, a form which is very frequently met with, only a few words are allotted. The chapter on diagnosis, especially from a differential point of view, is not as complete as it should be. The entire impression left by reading the book is tbat of a good resumé of the subject, interestingly written, but containing little new. G. W. J.

On Some Forms of Paralysis from Peripheral Neuritis of Gouty, Alcoholic, Diphtheritic, and other Origin. By THOMAS BUZZARD, M.D., London. J. and A. Churchill, London, 1886.

In the Periscope department of this number will be found a review of Dr. Buzzard's lectures on paralysis from peripheral neuritis, published originally in the *Lancet*. As we go to press we receive a copy of these lectures in book form which contains a "considerable amount of material which was unavoidably omitted from the 'Lectures,' as they were delivered at the Harveian Society of London, and afterwards published in the *Lancet*." This little book (of 142 beautifully printed pages), which any student can read in a couple of hours, will, we believe, contribute greatly to the proper understanding of certain forms of disease which are often falsely interpreted by neurologists and invariably so by the general practitioner. This is not to the discredit of the general practitioner, for it is only within the last year or thereabouts that any one of us has been able to formulate his views on this subject. Dr. Buzzard's book is, therefore, particularly timely.

Not wishing to repeat what has already been said in the Periscope department, we will simply direct attention to a few additional points. After giving a clear definition of simple neuritis, with some illustrative cases, the author refers at once to neuritis in the gouty. The cases given are convincing. We believe that many of our so-called lithæmic and some of our reflex paraplegic cases would be classified more properly in this way.

The chapter on multiple neuritis seems to us to have brought order out of chaos. Buzzard includes under the terms a group of symptoms which may be present in subjects of syphilis, of chronic alcoholism, of lead poisoning, and of Kakké or Beriberi. The remarks on alcoholic paralysis are among the most interesting in the book. The preponderance of paralysis in the lower extremities in this form of neuritis is insisted upon again and again, and the author seems to consider the foot-drop as characteristic of alcoholic paralysis as the wrist-drop is of lead-palsy.

"The existence of foot-drop is not alone a proof of habits of excess, but the symptom is so extremely frequent in cases of alcoholic paralysis that we should be wanting in our duty if we failed to bear this in mind and direct investigation accordingly."

Buzzard, as Dreschfeld does (see *Brain*, 1885, 1886), attaches great importance to the peculiar mental condition of alcoholists: "they describe the presence of their friends as if they

saw realities, and reason tolerably clear upon false premises." The third and last chapter is concerned chiefly with diphtheritic paralysis.

In this form Dr. Buzzard is inclined to regard a multiple neuritis as the essential lesion of the disease. There is ample pathological proof of this. The book closes with short sections on diagnosis (in which the various points of differential diagnosis are carefully considered), prognosis, and treatment.

The book is a great credit to the author and publishers. We have read it eagerly and with pleasure. B. S.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of May 4, 1886.

C. L. DANA, M.D., President-elect, in the chair.

ADDRESS OF THE RETIRING PRESIDENT.

The address of the retiring president, Dr. W. R. Birdsall, absent through sickness, was read by the secretary, Dr. George W. Jacoby. Dr. Birdsall reviewed the work of the Society for the year, and expressed satisfaction at the number and quality of the scientific contributions and the interest which they had awakened, at the increased attendance of the meetings, and at the harmony which had prevailed in their counsels. He had not shared the fears of those who had believed that the organization of a section in neurology in the New York Academy of Medicine would have a detrimental effect upon the activities of this Society. The year's work had shown that such fears were not well founded ; that this city could support two societies in neurological research without the one detracting from the merits of the other. Both societies had thrived, and the one over which he had the honor to preside had been able to receive one half the number of papers offered. Dr. Birdsall thanked the Society for the honor which it had conferred upon him, and expressed regrets that unavoidable absence deprived him of the pleasure of introducing the President-elect.

INAUGURAL ADDRESS.

Dr. C. L. DANA, the President-elect, recommended to the society that it consider the question of abolishing the custom of having either a retiring or a formal inaugural address unless these be made the means of presenting some scientific question. The

suggestion might also be made whether it would not be wise to limit the membership of the society, or else to establish some qualifications for membership. Dr. Dana said he would call the attention of the members to a large gap which existed in our knowledge of the etiology of nervous disease, and in the need of closer examination into this branch of that specialty. At present our knowledge of the etiology of nervous diseases (leaving out poisons) might almost be summed up: heredity, syphilis, and rheumatism. Was it not possible that neurologists had neglected to apply the ideas with regard to micro-organisms and infection which were now dominating pathology? It was true, however, that Leyden had found a micro-organism in cerebro-spinal meningitis, Rosenbach the bacillus of tetanus, and Strümpell, had urged the view that acute anterior poliomyelitis was an infectious disease, etc.; yet these points were not solidly established, and the relation of infectious poisons or parasites to nervous diseases deserved closer study. It had seemed to him that many cases of the chorea of Sydenham were really infectious in origin. He would also call attention to the possibility of a parasite being at the root of some of the neuro-degenerative disorders, such as ophthalmoplegia externa, bulbar paralysis, and progressive muscular atrophy, The necrobiotic process which took place in these disorders was often so steadily and frightfully progressive, so nearly malignant in the fatal course, as to suggest some active agency behind it.

History of a Case of Primary Labio-Glosso-Pharyngeal Paralysis:

Dr. E. D. FISHER presented a patient whose history was as follows:

Mrs. H., æt. 43, has always enjoyed good health up to July, 1885. At this time she lost her oldest son, who was accidentally drowned. She was much affected by the loss, and was constantly crying and calling for her son. The following September she first noticed some difficulty of speech and inability to move her tongue freely, with also some difficulty in swallowing. Dr. Fisher saw the patient for the first time in February. She then presented the following symptoms: Inability to protrude the tongue beyond the teeth, to form the lips so as to whistle or blow, the lower lip being down, and the saliva ran freely from her mouth. The lower part of her face was expressionless. No loss of power of the upper muscles of the face. The patient was unable to pronounce linguals or labials, and also, as the palate was

partially paralyzed, was unable to pronounce the explosives ; all her tones were decidedly nasal. Her food had to be pushed with her hand to the back of her mouth, when with difficulty it was swallowed. There was no tendency for liquids to return through the nose, but they would come out of the mouth. There was no loss of sensation or taste.

The faradic current was somewhat decreased in reaction, but there was no reaction of degeneration to the galvanic current.

These symptoms have all increased since first seeing the patient, and she has lost about twenty pounds in weight. There are no signs of paralysis of the upper extremities ; the disease is located entirely in the bulbar nuclei.

The interest of the case lies in the fact that the cause can be clearly traced to the excessive grief at her loss.

Dr. Fisher suggested, in the discussion of the case by the Society, that, recognizing the lesion as seated in the fourth ventricle, involving the hypoglossal, facial, vagus, and glosso-pharyngeal, the question of the situation of the facial nuclei be taken up. Clarke has mentioned that the facial has a lower nucleus for the orbicularis oris, and Gowen thinks that fibres for this muscle are given off from the hypoglossal nucleus. Either of these theories would explain the escape of the upper muscles of the face, as is usual in this disease.

DISCUSSION ON DR. FISHER'S PAPER.

The President said there were several obscure points for discusssion which Dr. Fisher's case had suggested : among others, the question of the etiology of labio-glosso-pharyngeal paralysis, some features in its symptomatology, and its treatment. Regarding the etiology, it was once claimed, he believed, that the disease was always of specific origin. He had had three cases under observation the past two years, and of those only one gave a pretty clear specific history ; in the other two, no such influence could be detected at all. In the one, although the patient gave some evidence of having had specific disease, yet it was simply assumption that this was the cause of the bulbar affection. In his opinion, we could only place specific disease among the predisposing causes.

Dr. PUTNAM-JACOBI asked whether the patient had heart disease.

Dr. FISHER replied that the heart had been examined, and no evidence of cardiac disease could be discovered.

Dr. B. SACHS thought the case was one of great interest to all. Bulbar paralysis, he thought, was more common in Europe than in this country ; there was scarcely a clinic at which one or more cases did not present themselves during the year. He had seen a number at the medical clinic at Strassbourg, under Professor Kussnaul. The etiological factor which Dr. Fisher had mentioned, particularly in his own case, deserved consideration. It was further interesting from the fact that the central lesion in this disease, and in diabetes, was near the same region, and that many cases of diabetes had been observed by him in which the etiological factor was intense emotion.

Dr. SACHS thought it was difficult to explain why, in an affection like that from which Dr. Fisher's patient was suffering, in which the pathology was similar to that of progressive muscular atrophy and polio-myelitis anterior, consisting of an affection of the nerve nuclei, there was not the re-action of degeneration in the muscles supplied by the affected nerve nuclei. But it was possible that the action of degeneration would appear later.

Dr. PUTNAM-JACOBI thought the suggestion made by Dr. Sachs as to the analogy between bulbar paralysis and diabetes as far as their possible origin in emotional influences was concerned was worthy of consideration ; and the question had arisen in her mind whether such emotional influence may not have first influenced the cardiac centre in the medulla oblongata and secondarily contiguous centres. Dr. Jacobi spoke of certain anatomical considerations in connection with bulbar paralysis, and referred to several cases reported by Eisenlohr. It seemed to her that exemption of the upper branches of the facial nerve in typical bulbar paralysis was an extraordinary circumstance, and one which she would be glad to have explained. It seemed remarkable that in Dr. Fisher's case the symptoms should have remained so limited for so long a time.

The PRESIDENT had examined the urine for sugar in two cases of bulbar paralysis, but with negative results. With regard to the affection of taste, it is well known that that sense was not usually involved in bulbar paralysis. He had thought that the glossopharyngeal nerve at its nucleus was purely a sensory nerve, and that it received its motor fibres from the spinal accessory ; that it supplied taste to the posterior, and perhaps to the anterior part of the tongue. The question as to whether it supplied general sensation to the fauces or posterior part of the tongue it seemed to

him was involved in considerable obscurity. The cases which he had seen had given no positive evidence that the glosso-pharyngeal nucleus was involved except in one in which there was disturbance of the sense of taste, and there had been two other cases reported in which this sense was involved. With regard to the seventh nerve, and involvement of its nuclei, he thought that in some cases the branches of that nerve were involved. In one of his cases the upper portion of the face was not wrinkled, the eyelids could scarcely be approximated, showing that the facial nuclei were becoming involved. Regarding the reaction of degeneration, it was never present except in the later stages. There might be partial reaction of degeneration at an earlier date. The explanation which he had given was that the trophic centres of the nerve were involved, causing atrophy, to which the paralysis was due.

As to treatment, he thought he should adopt a radically different form from what he had hitherto employed. It seemed to him that the cases improved for a while under electrical treatment, and then such treatment seemed to make them worse. He would give the affected muscles complete rest if possible, and confine the electrical treatment to the stabile galvanic current.

Dr. SACHS remarked that the phenomena of the reaction of degeneration might be present at first only to a limited extent, developing more completely as the case progressed.

Stated Meeting, June 1, 1886.

The President, CHARLES L. DANA, M.D., in the chair.

Trigger Finger (*Doigt à Ressort*).

Dr. GEORGE W. JACOBY read a paper on this affection, which he said was, strictly speaking, one of a surgical and not of a neurological nature; that is, if its pathology, as at present accepted, was correct. These cases, however, when encountered by the general practitioner, were liable to be referred to the neurologist; hence, the importance of being able to diagnosticate the condition. Doigt à ressort was the name given by Nélaton to a peculiar inhibition of motion in fingers otherwise normal. Flexion and extension were arrested at a given point, and if completed by force, the movement resembled the closure or opening of the blade of a pocket-knife. Sometimes only extension was inter-

fered with. As a rule, muscular effort alone was sufficient to overcome the obstacle. Generally the entire motion was painful, particularly at the time of the snap. The patient usually located the pain in the interphalangeal joint, but a careful examination would show that it was at the metacarpo-phalangeal articulation. Externally the finger presented nothing abnormal, but pressure over the last-mentioned joint almost always produced pain, the painful point being usually confined to a small place upon the volar surface of the flexor tendon. In all cases except those of Busch and his own, a hard lentel-sized body, which was particularly painful to pressure, was found attached to the tendon about two centimetres above the digito-palmar fold. All authors laid stress upon the presence of this body, as it was, according to all theories of the mechanics of this phenomenon, essential to its production. In Dr. Jacoby's first case, he did not remember to have found any nodosity, but as he did not pay particular attention to it, it may have been overlooked. In his second case, however, knowing of the cases of Busch and of Marcano's criticism on them, he made a very careful examination, and could say positively that there was no nodosity or abnormality of any kind discoverable. He saw his first case in 1881, but did not make a diagnosis. The patient was a female servant, who almost continually had her hands in water. She had had vague rheumatic pains for years, but had never had an attack of acute articular rheumatism. About six months prior to her visit to Dr. Jacoby, she began to have a peculiar tingling sensation in the ring finger of the left hand, with shooting pain upward in the arm ; she also complained of weakness of the finger and difficulty in flexing it. There was, however, no distinct ressort until two months before he saw her ; then she was unable one morning to close the finger, and in attempting to aid herself with the other hand, the finger suddenly snapped shut. Dr. Jacoby saw her only once.

The second case was that of a clerk, aged twenty-eight, whom he saw in November last. The middle finger of the right hand was affected. There was no apparent cause ; the patient had never had rheumatism, nor sustained an injury of the finger. The phenomenon came on very suddenly while he was engaged in writing, and was very much fatigued. He made his own diagnosis of writer's cramp, and a physician whom he consulted coincided with this diagnosis. Upon examination, Dr. Jacoby found the peculiar snap to be well marked, and the patient as unable either

to fully extend or flex the finger without the aid of the other hand. Both flexion and extension caused severe pain. Pressure over the metacarpo-phalangeal joint was painful. Repeated and careful examinations failed to reveal the presence of a nodosity or irregularity whatsoever. The treatment consisted in the application of the galvanic current, but, after a few sittings, the patient disappeared from under observation.

The affection had been described and cases published successively by Notta and Nélaton, by Fenerly, Arrachart, Busch, Annandale, Dumarest, Hahn, Menzel, Fieber, Vogt, Blum, Felicki, Herræz, Leisrink, Marcano, and Larheau. The only reference to it which Dr. Jacoby had been enabled to find in any English or American periodical was a translation of Menzel's article, published in the Boston *Medical and Surgical Journal*, 1874, and the description of a case by Annandale, which, however, he evidently did not recognize as a case of doigt à ressort. Dr. Jacoby gave tables of 33 cases by different authors; 21 cases were in women and only 10 in men, in 2 the sex not being specified. All the cases were in adults excepting 2. Occupation seemed not to have any influence in the production of the malady. The fingers affected were the thumb, 16 times; the ring finger, 15 times; the middle, 6; the small finger, twice; and the index finger, only once. In five cases more than one finger was affected. The ætiology must, in the majority of cases, be sought in rheumatism; next in traumatism. In some cases no direct cause could be found. The diagnosis was easy; the prognosis was generally fair, as the symptoms usually disappeared after several weeks of appropriate treatment.

Dr. E. C. SEGUIN said he had never seen a case of true doigt à ressort. He had seen two cases which resembled this condition, but which were of an entirely different nature in their ætiology, They verified the author's statement that such patients were likely to be sent to the neurologist, and he should therefore prepare himself to make a diagnosis. In one of the two cases to which he referred the patient was unable voluntary to flex the terminal phalanx of the thumb, and if it were forcibly flexed it would go back with a jerk. There had been section of the long flexor of the thumb.

Dr. M. A. STARR, who had also seen the second case referred to by Dr. Seguin, said the difficulty, which was due to section of a tendon, had been mistaken for paralysis, but Dr. Seguin cor-

rected the error in diagnosis. A surgeon had sent another patient to Dr. Starr within a week, who at his work was accustomed to make firm pressure with his hand, and suddenly he experienced difficulty in flexing his fingers, being entirely unable to flex the little finger. The faradic current caused flexion of all the fingers but the little one. He sent the patient to a competent surgeon, who made the diagnosis of rupture of the long flexor tendon of the little finger.

Dr. WILLY MEYER had seen two cases of doigt à ressort in Europe. One came to the surgical clinic at Bonn while he was assistant. In both patients the middle finger was affected. One patient was a man, the other a woman. In the case of the man a very thorough examination was made, but no apparent pathological change was present. He was able to use his hand, but with some inconvenience. He wore a splint four or five weeks, which left the finger a little stiff, but this was overcome by prolonged warm baths and passive motion. The woman had complained about two months of pain along the flexor tendon from the middle of the palm of the hand to the tip of the finger, the pain having grown steadily worse. A small painful nodosity was felt just below the metacarpo-phalangeal joint. As there was no doubt that this nodosity was the cause of the affection, they advised its removal, but the patient refused. There were two interesting cases of trigger finger published in the *Centralblatt für Chirurgie* 1884, No. 18.

Dr. Meyer thought there was always a mechanical cause of the disease, for even in those cases in which no particular pathological symptom was observable, there might be something wrong within the articulation. As to treatment, were there no apparent cause for the difficulty, he would use the plaster-of-Paris splint, massage, prolonged hand-bath, passive motion, and perhaps electricity would be advisable. If a nodosity were found it should be removed.

Gilles de la Tourette's Disease.

By C. L. DANA, M.D., and W. P. WILKIN, M.D. (See p. 407)

The PRESIDENT remarked regarding the case that the patient had improved very much under treatment. It was one of the cases in which moral agencies had the power of suppressing the symptoms for a time. The audience had probably observed that while sitting quietly the patient had exhibited a peculiar kind of cough which was about the only thing noticeable.

Dr. GRÆME M. HAMMOND asked whether the boy was cruel.

The President replied, not to his knowledge. The only bad trait which the boy had manifested was the disposition to lie.

Dr. JULIUS RUDISCH thought it probable the disease, as described by Hammond, was the same as that prevailing in Kamtchatka. Persons suffering from acute or chronic belladonna poisoning exhibited this jumping tendency. A further interesting fact was the similarity between the symptoms manifested by this class of patients and those of certain persons sensitive to tickling. Some persons suffering from mental weakness or a mild form of insanity were disposed to pronounce very obscene words, to manifest twitchings of the face and other involuntary movements which they sometimes took pleasure in observing in the mirror. It hardly seemed to him that the description of the case presented to-night accorded with that given by Beard and of those in Siberia and similar ones in Java.

Dr. E. C. SEGUIN thought it might be questioned whether the case presented was like Tourette's cases on the grounds expressed by the author, namely, the different mode of development, the order of development, and the fact that this patient had a defective mind, whereas Tourette's patients had a normal mind. Still, this might be a case in which there was simply an inverse order in the development of the phenomena. He would like to enter a protest against the nomenclature of the disease, especially as it presented no definite clinical history. In some of Tourette's cases there was absence of echolalia or of copsolalia. He preferred Charcot's definition, that of tic convulsiv, which might include quite a variety of jerking affections, or another term embracing all possible varieties of these cases might be employed, as abnormal chorea. An interesting, but almost forgotten, French monograph of about 150 pages on these jerking affections was published at Strasburg in 1850. An interesting case had been reported by a distinguished surgeon of New York.

Dr. M. A. STARR referred to a case related by Dr. Mills, of Philadelphia, in which a tumor involved the second frontal lobe on the left side and pressed upon the third. One of the prominent symptoms was the tendency on the part of the patient to use profane and obscene expressions, apparently without any power to control it. The case suggested the question, why, since irritation of the central convolutions would produce involuntary motion, irritation of the third frontal convolution should not produce involuntary speech.

The PRESIDENT was perfectly aware that the case was one difficult to classify, but he felt positive that if there was such a disease as that pictured by Tourette this patient had it. In one journal which he consulted the name Gilles de la Tourette was given it by Charcot, and he was much surprised to hear Dr. Seguin say that he did not approve of that name. However, he agreed with Dr. Seguin that there were objections to the name. He could not understand how some of the gentlemen arrived at the opinion that the condition in this case was due to insanity, for the boy, although there seemed to be some defect in his mental nature, did not manifest any symptoms of insanity.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, March 22, 1886.

S. WEIR MITCHELL, M.D., President, in the chair.

Dr. H. C. WOOD read a paper on "Landry's Paralysis."

In the course of the paper the symptomatology and diagnosis of neuritis and poliomyelitis were to some extent considered.

Dr. G. BETTON MASSEY said that in regard to pain on pressure in poliomyelitis, that it is of interest to recall the fact that in this affection the skin is unusually sensitive. In children, for a year after the attack, there is hyperæsthesia to tactile impressions and especially to the battery. Hyperalgesia would probably better describe the condition. These cases have presented marked reactions of degeneration, and it has seemed to him that the greater the reactions of degeneration the greater the sensitiveness to the galvanic current in children. An autopsy is by no means necessary to make out the diagnosis, for the clinical histories of these cases are almost identical. It should be as easy to diagnose acute poliomyelitis in a child as to diagnose smallpox. He thought it might be possible in the cases referred to to remove a portion of the nerve for examination during life.

Dr. WHARTON SINKLER, in reply to Dr. Wood's statement that the diagnosis cannot be regarded as proved unless there has been an autopsy, said that we cannot make autopsies in all cases, and we must therefore infer that our diagnosis is correct when the symptoms are identical with those of other cases in which autopsies have been made. Competent observers in this country and abroad have made post-mortem examinations in cases of infantile paralysis, and have found distinct lesions in the anterior horns of the cord. Hence we may assume that there is such a disease as poliomyelitis anterior. He had seen children, as described by

Dr. Massey, in whom there was hyperæsthesia to electricity, especially to the galvanic current.

At least twelve or fifteen years ago Barnwell advanced the view that infantile paralysis was a disease of the peripheral nerves; the later observers, supported by autopsies, seemed to have disproved his theory. Kennedy has described a variety of infantile paralysis under the name of temporary paralysis. We believe that this disease is distinct from "myelitis of the anterior horns," and it may be the result of a diffused neuritis, as suggested by Dr. Wood. He had no doubt that some of the cases of the so-called spinal paralysis of adults, those of short duration, may be of a similar nature.

Dr. FRANCIS DERCUM had lately seen a case in private practice which was of interest in this connection. A man who had been exposed to damp and cold had a chill followed by fever. He was called to see him, and found him suffering with intense pain affecting both brachial plexuses, followed by rapidly developing paresis of both arms. This was evidently a case of peripheral neuritis. The electrical reactions were examined by Dr. Lloyd, who can better give the results of this examination.

In such a case, if the patient does not die early, we may expect the cord to become involved, and then, in the absence of a knowledge of the previous history, it would be impossible for any one to say which was the primary lesion.

He thought it possible that we may have a disease in which both the centres and the nerve trunks are affected simultaneously. Diphtheritic paralysis is probably a case in point.

Dr. JAMES HENDRIE LLOYD thought that the case referred to by Dr. Dercum was of interest, especially with reference to one or two points which had been brought forward in regard to the reactions of degeneration. This man was completely paralyzed in both deltoid muscles, and in the right infraspinatus and the right teres major and minor muscles. The left infraspinatus and teres muscles were perfectly normal. If this was a case of peripheral neuritis, we have facts contradicting some observations made to-night. In this case, typical reactions of degeneration were present. The changes were both serial and modal, and the faradic irritability was almost extinct. There were no fibrillary contractions in this case. In regard to this subject of reactions in wasting muscles, it seems to him that where there have been marked fibrillary contractions, the reactions of

degeneration are not as distinct as we should expect to find them, and this is probably due to the fact that such cases are caused by a slowly advancing anterior poliomyelitis, in which the trophic centres are very gradually destroyed ; whereas, in peripheral neuritis the lesion would be more quickly established, and the characteristic electrical changes would be earlier observed.

Dr. CHARLES K. MILLS thought that while multiple neuritis may occur more frequently than we have been led to think, still, we have as yet few positive diagnostic points. Pain on pressure over a nerve trunk is perhaps one of the best signs of neuritis of all forms, but this is also sometimes found in subacute myelitis of the anterior cells, and in neuralgias there is hyperæsthesia at limited points. In genuine posterior sclerosis at times limited areas of pain on pressure are present. In regard to Landry's paralysis, it seems that subacute myelitis of the anterior horns would simulate it more closely than any thing else.

Some years ago, Leyden advanced the view that locomotor ataxia was due to neuritis, and since then this view has been advocated by different authorities. We find tenderness over the nerves in various spinal and cerebral affections. In some cases of brain tumor, hyperæsthesia is a marked feature. The same is sometimes seen in spinal tumors. It might be said that in these cases there was also neuritis, but in some cases of brain tumor this is impossible. The tumor may be so high as to be remote from the nuclei of origin of the nerves. Leaving post-mortems aside, the uniform bilateral character of many of the cases classed as subacute anterior poliomyelitis is, so far as it goes, in favor of a central origin of the disease.

The suggestion of Dr. Dercum that the disease may simultaneously affect the nerve trunks and the centres, seems more philosophical than to attribute all these cases to multiple neuritis. In arsenical paralysis, he believed that the nervous protoplasm was everywhere affected.

Dr. MORRIS LEWIS read a paper on "The Use of Nitrite of Amyl in the Severe Paroxysms of Whooping-cough."

E. B., female, æt. thirteen weeks ; breast fed ; a well-formed, healthy baby ; rather small, and weighing about seven and one half pounds.

At the age of seven weeks, having just recovered from the effects of a perfectly normal vaccination, she contracted whooping-cough from her brother, whose case had been one of the lightest

character, rendering the diagnosis impossible for over two weeks. The infant's cough for the first week was but slight; but during the second week it began to show the characteristic symptoms.

The child was placed upon a mixture of belladonna and alum every three hours, and progressed favorably until the night of February 12th, which was towards the end of the second week of the disease; it was then seized with a violent paroxysm of coughing, became purple in the face, and finally, according to the mother's statement, ceased to breathe. I was immediately sent for; but before I could answer the summons the mother had thrust the child out of the window into the cool, damp air of a foggy night. This procedure was immediately followed by an inspiratory effort, and the child breathed again. When I arrived the child was in an exhausted state, and was breathing regularly. After this, until the 22d of the month, but one other slight attack of this nature occurred. By this time the child was coughing in a perfectly typical manner. The medicine was continued, but in slightly larger doses, the child taking about the $\frac{1}{12}$ gr. of extract of belladonna in the twenty-four hours.

On the night of the 22d there were three severe paroxysms, during all of which I was present.

The child would awaken with a series of violent expiratory coughs, with scarcely an inspiratory effort between them. Finally, an expiratory spasm would occur, lasting fully fifteen seconds, during which the child would struggle and become perfectly livid. This would be succeeded by complete collapse, with entire suspension of respiration, due probably to exhaustion of the respiratory centre.

During the last two of these attacks I administered ether by inhalation, and believe that thereby the spasmodic stage was somewhat shortened, but the subsequent collapse was so severe that I was obliged to resort to artificial respiration, and once had to continue it for ten minutes, as during that time there were but one or two feeble attempts at inspiration. The evening before I had placed the child on the $\frac{1}{600}$ of a grain of sulphate of atropine every three hours.

Dr. William Pepper then saw the case with me in consultation, and suggested combining a small amount of nitrite of amyl with the ether to prevent if possible the stage of exhaustion. This combination seemed to have a good effect; but, as the amount of ether required seemed to render the child drowsy and

disinclined to nurse, I gradually diminished the proportion of ether until the mixture contained one fourth part of nitrite of amyl. This mixture was kept in a small vial, and with each cough the end of the finger was wetted with the mixture and held close to the child's nose and mouth, so as to catch the first inspiratory effort. Thus administered, the child practically got nothing but nitrite of amyl. After the commencement of this treatment the child never had another attack of exhaustion, and the severity of the paroxysms seemed to be lessened, although the child coughed just as frequently, the number averaging fifty in the twenty-four hours, the amyl being given each time.

No untoward effects were at any time noticed, even when once I held the bottle, containing at least ten drops of amyl, to the child's nose.

It was difficult to judge of the amount of flushing produced by the drug on account of the flushing caused by the cough.

I am confident of the beneficial effect of the nitrite of amyl in this one case, and, although one swallow does not make a summer, I think the drug will be found of use in analogous cases.

Dr. John M. Taylor, who assisted me in watching the case, is also positive of the good effect produced.

In the short time that I have had to look up the literature of the subject, I cannot find that nitrite of amyl has been used in whooping-cough, nor has anybody that I have spoken to on the subject known of its use.

By diluting the nitrite with ether or alcohol it can be administered in any dose required, and could more easily be placed in pearls than the pure nitrite.

By placing it in a bottle with a small top, that can be covered easily by the finger, it can be almost immediately administered by simply wetting the tip of the finger by inverting the bottle.

The child is now doing well, and has had no complication other than a slight umbilical hernia.

Stated Meeting, April 26, 1886.

S. WEIR MITCHELL, M.D., President, in the chair.

Dr. H. F. FORMAD, by invitation, read a paper and presented specimens illustrating different forms of "Cerebral Hemorrhage."

Dr. DERCUM said that Dr. Formad states that in no case can the blood find its way from the pia mater into the ventricles. That

is true of the general surface of the brain, but not of the base. Profuse hemorrhage at the base may find its way into the lateral and third ventricles through the transverse fissure of the brain ; of this he had seen an instance.

Dr. CHARLES K. MILLS thought that perhaps the statement in regard to the first class of cases may be misleading, if he says absolutely that small hemorrhages in the fourth ventricle and in the lateral ventricle occur only from traumatism. These are of frequent occurrence, particularly in the floor of the fourth ventricle in many diseases. They probably take place at the time of death. He has seen them in cases dying in the epileptic status. They have been found in cases supposed to be hydrophobic.

The occurrence of hemorrhage in the second class of cases opposite the point of application of the force is interesting, and he thinks is best explained by Duret in his papers on traumatisms. He attributes it to displacement of the cerebro-spinal fluid with the formation of numerous vacuoles and the rupture of the blood-vessels from within outward, the largest vacuoles usually occurring opposite the seat of injury. In those cases in which the hemorrhage is in the lenticular body and works its way into the lateral ventricle, numerous ecchymoses occur in various parts of the membranes and of the brain. These occur for the same reason as when the skull is struck from the outside.

He believed that in children hemorrhages occur beneath the pia mater without any special recognizable cause. He has seen one case in which meningeal hemorrhage occurred in a new-born child, probably as result of traumatism from the use of forceps. Where children die soon after delivery, he believes that death is sometimes due to this cause.

Dr. CHARLES K. MILLS reported a case of "Unilateral Sweating."

The patient was an unmarried woman thirty-four years old, sent to him by Dr. Deakyne. Eighteen or nineteen years ago she began to have spasmodic attacks which seemed, from description, to be epileptic. About fourteen years ago she had a stroke of left-sided paresis. About the same time she began to perspire excessively on the side of the paresis. This unilateral sweating has continued up to the present time and is more marked than before. The secretion on the right side is normal. There is also increased lachrymation on the left side. The saliva seems to be about in equal amount on both sides. Before the occurrence of the spasms she complained of a peculiar taste, probable metallic. She occasionally has slight attacks of dizziness, is somewhat

absent-minded, and at one time had severe pain in the head. Four years ago the spasms ceased. The left side of the mouth is visibly drawn a little upward. There is slight paresis of the muscles supplied by the facial nerve. She cannot draw up the left side of the mouth volitionally as well as the right. The slight loss of power in the limbs of the left side is more particularly in the arm.

She has had no trouble with the bladder or bowels. At times the left side of the face reddens very much and remains so for some time. She has some pain on the left side. She has been chiefly treated for uterine trouble. She does not present any signs of the hysterical temperament. Against hysteria are the facts that in the first place, she has paralysis of the muscles supplied by the facial nerve; second, she has contracture on the left side, which is sometimes present in old cases of facial paralysis; and, third, the persistence of the condition for fourteen years.

Dr. EDWARD T. REICHERT thought that it is not only probable that sweat centres exist in the spinal cord, but also that there is a dominant centre in the medulla oblongata. The latter is no doubt bilateral, and while it generally acts as an individual centre, affecting in like degree sweating on both sides, the centre on one side only may be in a condition of over-excitement, as is probably the condition in the present case, thus causing unilateral sweating. The lesion must, he thinks, be above the middle of the pons in order that there may be paralysis of the face and limbs at the same time.

Dr. FRANCIS DERCUM believed that the supposition of some lesion of the cortex going on slowly and leaving a sclerotic patch, and followed by degeneration involving these so-called sweat centres, might explain the case. As regards unilateral sweating, he observed it for four or five years in a case which subsequently terminated in apoplexy. No autopsy was made.

Dr. CHARLES K. MILLS said that Dr. Reichert's remarks would indicate a possible cause for this case. It seemed to him that it might be due to a very small tumor or patch of degeneration of some kind high up in the pons. He found, in looking up the subject, one case of unilateral sweating reported in which degeneration of one of the cervical ganglia was found. He attached but little importance to this. He had recently looked up the subject of unilateral progressive facial atrophy, of which more than fifty cases have now been reported. In a majority of these there has been diminution or entire absence of sweat on the atrophic side

Editorial Notes and Miscellany.

THE annual meeting of the American Neurological Association was held at Long Branch, July 21st, 22d, and 23d. The meeting was in most respects a successful one, but it did not have the appearance of a meeting of a *National* Association. With the exception of two gentlemen from Baltimore, one from Cincinnati, and one from Ithaca, the members present were members either of the New York or Philadelphia Neurological Society. We had an idea that Boston contains not a few neurologists, and it was certainly a matter of regret to those present at the meeting that the HUB was in no wise represented. Perhaps our Boston friends will join us next year at Washington !

The following papers were read and discussed at these meetings :

July 21st.—Morning Session.

Retiring Address of the President, Prof. Burt G. Wilder : " The Collocation of a Fissure and Suture in the human fœtus."

Address of the President-elect, Dr. C. K. Mills, of Philadelphia : " Fissures, and their Relations to Crime and Insanity."

Afternoon Session.

Dr. L. C. Gray : " Lesion of the Temporal Lobe without Sensory Aphasia."

Dr. V. P. Gibney : " Pseudo-Hypertrophic Paralysis."

Dr. Sarah J. McNutt : " On a Case of an Infant with Multiple Tumors of the Cerebrum, probably of Specific Origin."

Dr. G. Betton Massey, of Philadelphia : " Exhibition of Diagrams illustrating the Laws of Ohm."

Dr. G. B. Massey : " On the Laws of Electrotonus and of the Normal Formula of Polar Reactions."

July 22d. Morning Session.

Dr. Burt G. Wilder, Ithaca : "Notes on the Brain."

Dr. Leonard Weber, of New York : "On the Psycho-Neurotic Affections which Accompany and often Mask Phthisical Disease."

Dr. Philip Zenner, of Cincinnati : "A Case of Auctioneer's Cramp."

Afternoon Session.

Dr. C. L. Dana : "Pseudo-Tabes from Arsenical Poisoning."

Dr. Burt G. Wilder : "Exhibition of a Living Frog which was decerebrized more than Seven Months Ago."

Dr. Wharton Sinkler, of Philadelphia : "Treatment of Facial Tic."

Dr. E. D. Fisher, of New York : "Some Remarks on Epilepsy."

Dr. B. Sachs : "Intracerebral Hemorrhage in the Young."

July 23d. Morning Session.

Dr. Burt G. Wilder : "Exhibition of the Medisected-Alinjected Head of a Murderer."

Dr. J. Rudisch, of New York : "Exhibition of a new Galvanic Battery."

Dr. J. Hendrie Lloyd, of Philadelphia : "Moral Insanity."

The majority of the papers read at the meeting and the entire transactions will be published in this JOURNAL.

Volume V. of "Pepper's System of Practical Medicine" has been received. It is a stately volume of 1326 (!) pages on diseases of the nervous system. As a matter of interest to our readers we append the list of articles and contributors, reserving an adequate review of this volume for some future number.

"General Semeiology of Diseases of the Nervous System ; Data of Diagnosis," "The Localization of Lesions in the Nervous System," by E. C. Seguin, M.D. "Mental Diseases," by Charles F. Folsom, M.D. "Hysteria," "Hystero-Epilepsy," "Catalepsy," "Ecstasy," "Progressive Unilateral Facial Atrophy," by Charles K. Mills, A.M., M.D. "Neurasthenia," "Acute Affections Produced by Exposure to Heat," "Syphilitic Affections of the Nerve-Centres," by H. C. Wood, M.D., LL.D. "Sleep, and its Disorders," by Henry M. Lyman, A.M., M.D. "Vertigo," by S. Weir Mitchell, M.D. "Headache." "Tremor," "Paralysis Agitans,"

"Chorea," "Athetosis," by Wharton Sinkler, M.D. "Local Convulsive Disorders," "Epilepsy," by Allan McLane Hamilton, M.D. "The Neural Disorders of Writers and Artisans," by Morris J. Lewis, M.D. "Tetanus," by P. S. Conner, M.D. "Disorders of Speech," by Edward P. Davis, A.M., M.D. "Alcoholism," "The Opium Habit and Kindred Affections," "Chronic Lead-Poisoning," by James C. Wilson, A.M., M.D. "Diseases of the Membranes of the Brain and Spinal Cord, "Tubercular Meningitis," "Chronic Hydrocephalus," "Congestion, Inflammation, and Hemorrhage of the Membranes of the Spinal Cord," by Francis Minot, M.D. "Spina Bifida," by John Ashhurst, Jr., M.D. "Anæmia and Hyperæmia of the Brain and Spinal Cord," "The Chronic Inflammatory and Degenerative Affections of the Spinal Cord," by E. C. Spitzka, M.D. "Concussion of the Brain and Spinal Cord," by William Hunt, M.D. "Intracranial Hemorrhage and Occlusion of the Cerebral Vessels, Apoplexy, Softening of the Brain, Cerebral Paralysis," by Robert T. Edes, M.D. "Atrophy and Hypertrophy of the Brain," "Disease of One Lateral Half of the Spinal Cord," "Progressive Labio-Glosso-Laryngeal Paralysis," by H. D. Schmidt, M.D. "Tumors of the Brain and its Envelopes," "Tumors of the Spinal Cord and its Envelopes," by Charles K. Mills, A.M., M.D., and James Hendrie Lloyd, A.M., M.D. "Infantile Spinal Paralysis," by Mary Putnam Jacobi, M.D. "Diseases of the Peripheral Nerves," by Francis T. Miles, M.D. "Neuralgia," by James J. Putnam, M.D. "Vaso-Motor and Trophic Neuroses," by M. Allen Starr, M.D., Ph.D.

Dr. Ireland's book, "The Blot upon the Brain" (G. P. Putnam's Sons), is to be translated into German. The French translation of this book, which is being prepared by Dr. Edgar Berillon, of Paris, is nearly finished. The "Blot" has been prohibited by the Russian Censorship. This is no doubt owing to the chapter on the hereditary insanity of the Romanoffs, and the historical illustrations about the harm insane monarchs have caused to their subjects.

VOL. XIII. AUGUST, 1886. No. 8.

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

A CLINICAL STUDY OF LATERAL HEMIANOPSIA.*

By E. C. SEGUIN, M.D.

CONSIDERABLE progress has been made in the attempt to locate the cortical centre for vision, and the path of the fasciculus opticus from it to the primary optic centres. Indeed, a recent analysis of cases[1] of lateral hemianopsia would seem to justify the proposition that in the cuneus and adjacent cortical gray substance there is in either hemisphere a centre for half vision. If one cuneus is destroyed, lateral hemianopsia results, and if both cunei are disorganized, we have complete blindness, at least for higher organized vision. The state of our knowledge is such as to render every new case of lateral hemianopsia with autopsy of extreme interest and scientific value; yet it does not, I think, render quite useless the publication of cases without post-mortem study. A number of points in the clinical history and symptom-grouping of hemianopsia may be illustrated by such cases, and the diagnosis *intra vitam* of other cases facilitated. Such is my apology for presenting this paper to the Association.

* Read before the Association of American Physicians at its first meeting, in Washington, D. C., June 19, 1886.

[1] A Contribution to the Pathology of Hemianopsia of Central Orgin (Cortex Hemianopsia), this JOURNAL, Jan., 1886.

The cases are nine in number, and are naturally divisible into two groups: First, a group of three cases, in which lateral hemianopsia was one of a considerable symptom-group of what I may call hemi-symptoms; second, a group of six cases, in which lateral hemianopsia was the sole, or at least the strikingly predominant, symptom of organic cerebral disease.

GROUP I.—Cases of lateral hemianopsia, with hemiplegic symptoms, probably due to lesion of the outer edge of thalamus and of the internal capsule in its caudal part.

CASE I.—Male, æt. fifty-three. Seen in consultation with Dr. P. C. Cole.

Constitutional syphilis, contracted about ten years ago; secondary symptoms.

On March 4th, developed right hemiparesis, right hemianæsthesia, and aphasia; attack not apoplectic.

Examination showed ataxic aphasia, alexia, partial paralysis, and anæsthesia of right face and limbs. Right lateral hemianopsia, vertical line passing to right of point of fixation. Central vision good with glasses for presbyopia.

Rapid improvement under thorough mixed treatment; KI up to 350 grains a day.

Re-examined May 29th. Speech about normal. No agraphia, but handwriting is awkward. Only a trace of right-side paresis. Sensibility of right hand (and of face) normal to tests, but patient declares that it is not as fine or distinct as upon left side. The hand and forearm exhibit, in voluntary movements, well-marked ataxic-choreic movements. Central vision good; color-perception unimpaired. Right lateral hemianopsia persists. Near median line of darkened half fields is a zone of imperfect vision —mere perception of object used in testing.

Pathological Diagnosis.—The lesion in this case was very probably syphilitic arteritis of the left posterior cerebral artery, with obliteration of the small branches supplying the caudal segment of the internal capsule and the outer part of the thalamus opticus.

CASE 2.—Mr. H., aged sixty-two years, seen January 3, 1881. Is a man of unusual intelligence and culture; master of several living languages. Enjoyed good health; had no premonition, and on May 19, 1880, returned home from business with a numb and queer feeling in right arm. Was not well for three days, and on morning of May 23d awoke with right hemiplegia and incomplete aphasia. Was semi-comatose later in the day, and remained so for several days. There must have been marked hemianæsthesia, as patient did not know when his right foot and hand lay in or out the bed. He spoke much, but mis-

called things. Vision was imperfect to the right of the patient. No serious general symptoms. Improved rapidly as regards power of motion, speech, and writing. The only remains of the attack are right paresis with ataxic movements and partial anæsthesia, right lateral hemiopia and alexia. Has learned to write with left hand.

Examination.—Very slight right hemiparesis. Awkwardness and ataxic movements of right hand and foot; marked tactile and caloric anæsthesia of right hand; feels pain or a simple touch well, yet he is unconscious of passive movements of fingers and hand when his eyes are closed (so-called loss of muscular sense). Has incomplete homonymous (or lateral) hemiopia; right temporal and left nasal fields darkened. No positive contracture. Heart and kidneys normal.

Speech is a trifle thick, but not aphasic to any extent; occasionally uses a wrong word; writes well with left hand, and composes a letter in French, German, or English as well as ever; no omissions. Sees letters and numerals, but can't read except by a laborious process of spelling; some few short and familiar words he reads at once. Hemiopia does not cause this alexia, as shown by his ability to read any word by spelling it. The blindness is for words, or rather for the images or concepts which words represent. There is no word-deafness, and, beyond some hysteria, mental action is good.

Pathological Diagnosis.—Atheromatous degeneration of left posterior cerebral artery, causing softening of the outer part of the thalamus, and partly of the caudal division of the internal capsule. Path for muscular sense specially involved.

CASE 3.—A male patient, æt. twenty-six, referred to me by Prof. Edward Curtis. On April, 16, 1876, sudden attack of right hemiplegia in the street; was only partially paralyzed. Next day, well-marked amnesic (?) aphasia; right arm powerless; much numbness of right cheek and arm. "Could see only with inner half of right eye," as patient expresses it.

Rapid improvement in speech and walk. Right arm slowly regained its power.

Since then, irregular jerking movements have appeared in right arm (not in face and leg); marked numbness of entire right side of body; defect in vision has persisted; has had several epileptiform attacks.

Probably had syphilis six or eight years ago; chancres; and, later, ulcerated legs.

Examination shows right hemianæsthesia, partial in degree. Paresis of right upper extremity, with marked ataxia during volitional efforts. At rest there are small quasi-rhythmical movements of the hand and fingers. The movements are therefore of the mixed ataxic-choreic type. The pupils and eye muscles are normal; no ophthalmoscopic lesions visible. The fault in vision is a right lateral hemianopsia, not quite reaching the point of

fixation, and not with a vertical line; about one third of the visual field is obscured (two thirds of each half field).

The diagnosis recorded at the time of observation (1877) was a simple hemorrhage just outside the left thalamus; and in publishing the case[1] I pointed out that it contradicted Charcot's dictum (since abandoned) that hemianopsia could not be produced by a strictly cerebral lesion.

I see no reason to alter this diagnosis. The patient was given the benefit of the doubt as to syphilis, and treated with Hg and KI, but without relief to the hemianopsia and post-paralytic chorea.

GROUP II.—Cases of bilateral hemianopsia without hemiplegic symptoms. Lesion probably in one cuneus and adjacent gray matter.

CASE 1.—Male, æt. fifty-two. Seen in consultatation with Dr. H. H. Tinken, June 13, 1882. Former health good, with exception of severe attack of migraine with vomiting frequently in the last ten years. Has had rheumatic manifestations, but positively denies any venereal disease.

On February, 23, 1882, in a severe attack of migraine, after violent vomiting, suddenly found that he could not see on his left. Later in day was completely blind. No paralytic phenomena or unconsciousness; for a week or ten days was ill with slight fever (100°, 112°); after this, sight improved, but blindness to left remained.

On April 20th, in the midst of a debate, without loss of consciousness or paralysis, suddenly found himself "mixing up his words." Was able to write at once after attack. When seen later on same day by Dr. Tinken there was slight hemiparesis. Speech has since greatly improved.

Examination.—The only trace of right-sided paresis is a slight hanging of the lower part of the face; grasp normal; no hemianæsthesia; speech good. Pupils of the eye muscles normal; no lesion of fundus; with glasses for presbyopia reads easily; has typical lateral hemianopsia to left, with a concentric limitation of nasal half field of the left eye. Heart large, but without murmur. Albumen in each of three samples of urine.

Pathological Diagnosis.—Two separate attacks of cerebral hemorrhage: 1st, with destructive clot in right cuneus, and a slight ecchymosis (?) in the left cuneus (total obscuration of fields for a few days); 2d, clot in or near the speech centre of Broca in the left hemisphere.

CASE 2.—V. S., male, æt. fifty-seven. Seen in consultation with Dr. Herrick, of Paterson, Oct. 17, 1884. A prematurely

[1] A Contribution to the Study of Post-Paralytic Chorea, "Opera Minora," p 197.

senile man, who for two years has had polyuria (without albumen), sense of cerebral exhaustion, frontal headache, frequent attacks of dizziness, and marked loss of memory. Denies syphilis.

Eyes were examined by Prof. H. Knapp on August 30th, and he has kindly sent me the following memorandum: "V = $\frac{20}{20}$, fields complete." Not long afterward patient noticed that he could not see well to his right.

Examination.—Mental state very dull; memory feeble; no hemiplegia; good equilibrium; no anæsthesia. Pupils and eye muscles normal; no retinal changes; optic nerves abnormally whitish, but not distinctly atrophied; has right lateral hemianopsia with vertical limits; left half fields slightly contracted.

Pathological Diagnosis.—Atheromatous degeneration of the cerebral arteries, blockading the left occipital artery (branch of posterior cerebral artery), and consequent suffering of the cuneus and adjacent nervous substance.

CASE 3.—A. B., female, æt. thirty-four, referred by Dr. C. R. Agnew, Oct. 24, 1885.

A healthy woman, who has had four confinements. All normal. At the close of the third, in August, 1883, just after the child was born, had a peculiar attack, in which she experienced a "snap," or sudden pain, in the left temple, and felt giddy. For several days afterward she had severe pain in the head, and could not see objects to her right. At the same time that she first noticed darkness to her right, there were a few simple hallucinations (a chair, chickens, etc.) in the dark half fields. Vision was also generally dim.

No distinct hemiplegia, or aphasia. Has since suffered from more or less painful headaches, and has had a number of epileptic attacks, of grand-mal and petit-mal. Has had two distinct "auræ," one consisting in a sense of churning in the head, the other of numbness and stiffness (subjective) in the right arm and hand, with inability to speak or to use the right words.

The fourth confinement (perfectly normal) occurred in February, 1885.

Examination.—No distinct hemiplegia, or anæsthesia, or ataxia. Pupils small, but active. Eye muscles normal. Color-vision good. V = $\frac{12}{xx}$ with either eye alone, and $\frac{12}{x}$ with both eyes when page of book is held to her left. There is right lateral hemianopsia, with vertical division line passing a trifle to right of the fixation point. No ophthalmoscopic lesion. Heart has a basal systolic murmur, not transmitted upward.

Pathological Diagnosis.—Embolism of left occipital artery (branch of post. cerebral), with consequent softening of cuneus and adjacent nervous substance.

CASE 4.—Male, æt. fifty-six. Seen January 25, 1886. Also under the care of Dr. E. Gruening and Dr. H. Gulecke.

Only distinctly syphilitic affection is an indurated chancre, observed and treated by Dr. Gulecke, in spring of 1884. Mixed

treatment ordered, but not followed with any persistence. No secondary manifestations have appeared.

In spring of 1883, failing sight and pain about eyes. Glaucoma diagnosticated. Double iridectomy performed in August, 1883, by Dr. Gruening, with relief. Dr. Gruening informs me that just before the operation the left eye presented an incomplete lateral hemianopsia. After the operation the right eye also exhibited one-sided limitation of its field. There was not, however, true geometric lateral hemianopsia. There was no color-blindness, and the optic nerves were not atrophied.

In November, 1884, true lateral hemianopsia to the left was discovered, and has persisted unchanged.

The patient has complained of headache, and of inter-pressure. He has had several attacks of right-sided supra-orbital neuralgia; but he has had no paralysis, spasm, or anæsthesia. Depressed, but not demented.

Of late legs have been weak, and locomotion slow and uncertain.

Examination.—Left lateral hemianopsia, with vertical line passing a little to left of point of fixation. Central vision fairly good with glasses (not exactly tested). Optic nerves cupped, rather whitish, but not atrophied. No hemiparesis or anæsthesia. Very marked weakness of both legs, shown especially in rising from a chair; this is increasing. Staggers slightly, but the walk is not quite that of cerebellar disease. It is to be observed that the chancre occurred about six months before the true lateral hemianopsia was observed.

Pathological Diagnosis.—Either a tumor of right occipital lobe, or syphilitic obliteration of right occipital artery, with softening of cuneus. Glaucoma can hardly have produced hemianopsia.

Case 5.—Male, æt. fifty-one. Referred to me by Dr. Webster, at the Manhattan Eye and Ear Hospital, February 8, 1886.

A large, strongly-built man, who has enjoyed good health.

For a week at close of last year had occipital headache, aggravated by coughing. There was also pain over the left eye. On Jany. 13th, arose well, except as regards the headache, which was severe On the street, at 7½ A.M., suddenly experienced a sort of shock, felt dizzy, but did not fall. It seemed as if every thing was going round for a few moments. No aural phenomena. Next day noticed that he could not see persons standing near him on his left.

Examination—No paralytic phenomena, or anæsthesia. No head symptoms. Heart acts rapidly, and is rather large, but presents no murmur. The only symptom now present is left lateral hemianopsia. The vertical line is a little zig-zag in its course, and passes just to the left of the point of fixation. The upper quadrants of the right half-fields are somewhat limited. Central vision good. No ophthalmoscopic changes. Pupils and eye muscles normal.

Pathological Diagnosis.—Hemorrhage in right occipital lobe ; in its apex, or near mesal aspect, involving the cuneus.

CASE 6.—Male, æt. forty-one. Seen Oct. 7, 1885. Former health good. Denies any form of venereal disease, and injury to head. One of his brothers is in last stages of tabes (was under my care some seven or eight years ago). Four weeks ago, in the country, had two severe attacks of vomiting. After the second, noticed diplopia for distant objects. Has also noticed an increasing weakness of both legs and awkwardness in gait. No vertigo, and only trifling headache since.

Examination —Walk a little uncertain with tendency to right. Not an ataxic nor yet a cerebellar walk. Patellar reflex and station normal. No paralytic symptoms or anæsthesia.

Left pupil is a trifle larger ; both active. No diplopia to-day. Typical left lateral hemianopsia, with vertical line passing a little to left of point of fixation.

Oct. 12th. Dr. E. Gruening saw patient and reported central vision good, no diplopia, no changes of nerves or retinæ ; hemianopsia as above.

Nov. 7th. Diplopia for distance present of late (due to weakness of left external rectus). Ophthalmoscope shows slight swelling of optic disks. Hemianopsia

On Nov. 10th, Dr. Gruening verified diagnosis of double neuro-retinitis, and found both external recti weak.

When last seen (Nov. 13th) patient exhibited failing vision, the same hemianopsia, convergent strabismus (both eyes turned), and marked weakness of both legs. Patellar reflex much increased. Left arm weaker (?) Abnormal drowsiness is a symptom which has existed from the beginning. Can sleep at any time in chair. Mind clear.

Pathological diagnosis.—Tumor of right inner occipital lobe extending forward and upward. Centres for legs involved (?) Pressure on lobus opticus (?)

I reject basal tumor because of late appearance of neuro-retinitis, preserved pupillary reaction, and absence of crossed hemiplegia.

REMARKS. 1.—The relatively frequent association of lateral hemianopsia with hemiparesis, hemianæsthesia, and hemiataxia is very interesting. Exclusive of the hemianopsia such cases constitute the symptom-group known as post-hemiplegic chorea, in which the abnormal movements (in our experience) may vary from choreic tremor to ataxia, and athetoid movements. It has been quite well settled that in such cases the lesion is to be found in the lateral part of the thalamus, and that it often impinges on the caudal segment of the internal capsule (*carrefour sensitif* of French writers). We may therefore reasonably conclude

that the super-addition of lateral hemianopsia to the above symptom-group indicates an extension of the lesion dorso-laterad so as to involve the fasciculus opticus.

Alexia was present in two of the cases.

2.—In the second group the hemianopsia stands out as almost the only symptom of gross (or focal) cerebral disease. Consequently, from the results of autopsies in other cases,[1] we are justified in locating the lesion either in white substance of one occipital lobe (injuring the fasciculus opticus), or in most cases, we think, in one cuneus and adjacent gray matter (cortical visual centre).

3.—In one case (No. 1 of group II.) there was temporary complete blindness, which resolved itself into permanent hemianopsia. In this case it is probable that both cunei were injured at first. In the same case a second fresh lesion developed two months later, causing temporary aphasia and right hemiplegia.

4.—In two cases (Nos. 4 and 6 of group II.), marked and progressive weakness of the legs would suggest an upward or dorso-frontal extension of the lesion (tumor) so as to involve the paracentral lobules (centres for the legs). In such a manner a strictly cerebral paraplegia might arise.

5.—The ophthalmoscope revealed changes only in two cases (Nos. 4 and 6 of group II.). In the former were seen the cupping and partial atrophy of the optic nerve, due to glaucoma. In the second typical neuro-retinitis was developed while the patient was under observation.

6.—In no case was Wernicke's hemiopic pupillary reaction observed.

7.—A very interesting symptom, not heretofore described (to my knowledge), occurred in one case, viz., No. 3 of group II. This consisted in hallucinating images in the half-fields which had just become blind. The images were few and simple, such as a chicken, a chair, etc., and rapidly passed away. It seems to me that these hallucinations represented the irritation of the cortical visual centre just previous to its

---

[1] *Vide*, A Contribution to the Pathology of Hemianopsia of Central Origin (Cortex-Hemianopsia), this JOURNAL, Jan. 1886.

destruction ; and that they are the analogues of the localized convulsions which are now generally spoken of as indicating an irritating lesion of the cortical motor centres. That "discharges" or excitation symptoms should occur when a sensory cortical centre is irritated by disease, as well as when a motor centre is similarly affected, is perfectly reasonable ; being, moreover, in strict analogy with the results (peripheral symptoms) of pressure and irritation of nerve trunks. It is probable that further inquiry will show that hallucinations occur not infrequently at the outset of hemianopsia.

8.—An important point in semeiology, and one which I propose studying further, is the invariable (?) preservation of central vision. It make no difference whether we have to deal with right or left lateral hemianopsia (in the foregoing series there were five on right side and four on left side), the vertical division line always passes a little away from the point of fixation, *i. e.*, a little toward the blind half-field in either eye. Practically it is found that vision is often perfect directly in front of the patient, No. 1 Jaeger being read. It follows that in the fundus of the eye the region of the macula escapes the paralysis of the half of the retina. Can this be due to the peculiarity of structure of the macula, or is there a special set of nerve fibres connecting the macula with the cortical visual centres? It is premature to speculate on these alterations, and much will yet have to be done before we possess the anatomical and pathological data for the solution of the problem.

9.—The anatomical or pathological diagnosis in living cases must always be stated with reserve. Yet, guided by results already obtained, an attempt should be made in all cases. In the foregoing cases the lesions were probably as follows :

Syphilitic arteritis with obliteration, and consequent softening	1 case.
Senile arteritis with obliteration, and consequent softening	2 cases.
Cerebral hemorrhage	3 "

Tumor of one occipital lobe	2 cases.
Embolism of occipital artery	1 case.

10.—A special pathological question of much interest presents itself in case 4 of group II. Can glaucoma cause true lateral hemianopsia? While ready to admit that glaucoma can produce extensive breaks in the visual fields, in some cases approximating hemianopsia, I cannot understand how an intra-ocular pressure could be so distributed as to affect only homonymous halves of the retinæ, with a vertical division line between the light and dark half-fields. And how explain the preservation of central vision for nearly three years on the hypothesis of an intra-ocular disease? That an irregular partial lateral hemianopsia existed in case 4 at the time of the iridectomy is established, as a fact, but it is also established that true geometric lateral hemianopsia was not discovered until fourteen or fifteen months afterward. I think that in this case we must admit the coexistence of the diseases, glaucoma, and (probably) tumor in one occipital lobe.

II.

ON THE PSYCHO-NEUROTIC AFFECTIONS WHICH ACCOMPANY AND OFTEN MASK PHTHISICAL DISEASE.

By LEONARD WEBER, M.D.

IT is a daily observation that there is a special condition of the mind associated with pulmonary tuberculosis. Frequently it consists in a peculiar cheerful hopefulness, the *spes phthisica*, which seems strangely out of harmony with the unmistakable signs of an advancing fatal disease by which it is accompanied. But there is also a state of mental depression which has been noticed in intimate association with the disease. The peculiar hopefulness is most frequently met with in the acute form of phthisis, and it is often so irrational and persistent as to amount to an insane delusion, growing in strength in spite of the accumulating evidence of its baselessness. In the last stage of such cases the religious and emotional exaltation is often extreme, and actual delirium not unfrequent. To give examples of cases of this kind is quite unnecessary; every practitioner has seen them, and the mental condition described is not present before the objective signs of phthisis are well marked, and consequently need not come in question in making out a diagnosis.

An opposite mental condition is met with in chronic phthisis, more especially in that form which has been called latent. All through the course of this disease there is a prevailing depression and distrustfulness, though the physical symptoms are neither so distressing nor so obvious as in the acute forms. The mental symptoms sometimes precede

the physical. Langour and depression, mingled with waywardness, are characteristic of the initial stage, and are usually accompanied by general functional debility—Neurasthenia—which is often attributed to mere disturbance of digestion and assimilation. The skin is habitually pale and the circulation feeble. In many cases the physical signs of pulmonary phthisis when present are apt to be overlooked; in others they escape observation for years, though repeated and careful examinations may have been made by competent observers. Again, where the mind is much affected, the ordinary symptomatic cough, expectoration, and dyspnœa are often absent; and this is the case sometimes where physical exploration reveals the existence of pretty large excavations and other characteristics of advanced disease.

As the further stages are reached the mental condition becomes less one of depression and more of distinct enfeeblement; occasional fits of considerable irritability and excitement may vary the picture. There is a disinclination to enter into any kind of amusement or continuous work; and even if it be overcome there is no interest manifested in the employment. This condition might be called a mixture of subacute mania and dementia, being sometimes the one and sometimes the other, the symptoms of dementia predominating as the disease advances towards the last stage. If there is any tendency to periodicity at all in the symptoms, the remissions are not so regular nor so complete, nor so long, as in ordinary periodical insanity. The mental depression in these cases is accompanied with irritability and the want of any fixed depressing idea or delusion. If there is any single tendency that characterizes the mental state of these cases, it is to be *suspicious*.

Quite an interesting case in this respect is that of Mary B., æt. 36, married, multipara, who came under my observation in Jan., 1880. Her father died of cerebral apoplexy; her mother, an energetic but excitable woman, is alive and well; two sisters in good mental and physical condition; another one is somewhat weak-minded, and has made an attempt at suicide, under the influence of some insane delusion. Mrs. B. has lived in comfortable circumstances; her

husband has always been kind and indulgent to her; her three children are healthy and well-behaved, and until her twenty-ninth year nothing unusual had occurred in her physical or mental condition. In 1878 she underwent an operation for lacerated perineum. The wound healed by primary union, but during the nine or ten days she was confined to bed she had some febrile exanthema, which was pronounced to be urticaria, and passed off quickly enough. Soon after she began to be troubled with malaria-like symptoms, on account of which she took quinine, and finally moved out of the house she then occupied, though no other inmate of the same had intermittent fever or other maladies which could be traced to any house-poison. During the winter of '78 she was not well, and went to Europe in '79, where she continued to have frequent and irregular attacks of slight fever, alternating with gastric disturbances; also an acute attack of extensive febrile erythema, ending with desquamation, as in scarlatina; and short periods of well-being. Never any cough or expectoration, but a good deal of worry and fretful speculation about her peculiar malaria or blood-poisoning, and occasional attacks of slight depression. She returned late in autumn, '79, and when I first saw her I found her comparatively well nourished, rather pale, with good, somewhat rapid, pulse, normal temperature, normal respiration, no cough nor expectoration, appetite uncertain, bowels regular, physical ability good, but great disinclination to pursuit of pleasure or work; nothing she loved so well as to talk and argue about her case from morning till evening with any one who chose to listen. Physical examination negative as to respiratory organs; spleen and liver neither enlarged nor tender; her retroverted uterus was easily replaced, and held in position by a pessary; her stomach improved by a proper regimen. However, I soon noticed that Mrs. B. had occasional slight rises in pulse and temperature, and quinine having no apparent influence in combating this febrile condition, the diagnosis "intermittent fever" was soon abandoned. In the course of the next two years her appetite continued to be capricious; she lost in weight slowly, but perceptibly; became more pale, but not en-

feebled; had crying spells and fits of depression, but never coughed; febrile movements were frequent. Although I strongly suspected the development of disease of the respiratory organs, neither myself nor anybody else was able to detect any thing by repeated and searching examinations. A complete change of climate to Davos or some other suitable place, with the intention of having the patient stay there, could not be carried out then nor subsequently, when more urgent indications arose, owing to an utter want of fixity of the patient's mind, as well as the want of firm support on the part of her friends.

In March, 1883, I noticed fine crepitant râles and slight wavy breathing and a little dulness on percussion over right apex for the first time, but these symptoms went and came in an irregular manner, and so did a slight cough and expectoration of a little bluish-white sputum. So insignificant and transitory were the physical signs, that as late as July of that year an eminent European authority had to keep the patient under observation for nearly six weeks, before he was able to find symptoms which induced him to agree with me in the diagnosis of latent phthisis. While abroad again she had a severe attack of febrile erythema, ending with extensive desquamation over upper extremities and the body; also periods of hysterical excitement alternating with depression, and anorexia and diarrhœa alternating with ravenous appetite. On her return to the city she did not look much worse than when she left; latterly disbelieved the idea of her lungs being affected, but had a growing conviction that her whole trouble was due to blood-poisoning brought about by the perineal operation performed some years before. Some friend telling her that all her troubles might be taken away by her having another child, Mrs. B. responded to this advice very promptly, and late in 1884 I delivered her of an apparently strong and well-formed boy. All during her pregnancy she had little to complain, but four weeks after delivery she experienced her old malarial symptoms again, and felt altogether wretched and despondent. She went and consulted the physician who had performed the operation for lacerated perineum upon her, who,

being ignorant of the state of her lungs, unfortunately encouraged her in her notion of blood-poisoning, and treated her for some weeks with antiseptic hypodermics. As soon as possible these proceedings were discontinued, but the patient not feeling any better after the treatment, immediately became suspicious of having now been thoroughly poisoned by these injections. This suspicion quietly developed to an insane delusion with wild maniacal propensities, and her temporary confinement to an asylum became necessary in March, 1885. On her discharge from it six weeks later she was quiet, but continued to nurse the former belief, and had grown quite suspicious towards some of her friends, whom she supposed to have conspired with the doctor in poisoning her. The latter suspiciousness continued its hold upon her up to the fatal end of her disease.

During the summer of 1885 the pulmonary phthisis progressed bilaterally and extensively. There was considerable expectoration, and blood, elastic fibres, and Koch's bacilli could be demonstrated in the sputum.

In August there occurred another short period of mania that required asylum treatment for two months. In October she showed all the signs of a hectic, but became more quiet as her feebleness increased, and gradually sank and died from exhaustion in December.

A case where the pulmonary signs had been overlooked and the patient treated for melancholia, supposed to be due to amenorrhœa, is that of Sister B., of the order of St. Francis. She is thirty-two years old, tall, pale, and thin, her pulse feeble, appetite and digestion quite irregular. No menstruation for twelve months. Expression of countenance very markedly that of melancholia. Family history phthisical. Though I had no difficulty in making out the signs of bilateral pulmonary phthisis on first examination last March, she presented none of the general signs of tuberculosis, but she had fever of irregular type and gradual loss of flesh like the first case. By nutritious food, cod-oil, the hypophosphites, and occasional doses of antipyrin and complete rest, a marked improvement in the patient's mental and physical condition was obtained in two months.

The symptoms of melancholy passed away, and she cheerfully did her work again, which she had lost all ability and inclination to do before. The improvement, however, was transitory, the mental condition of the patient grew worse again in May, she refused food, lost all interest in her work, had delusions of a religious character, and made several attempts to hang herself. In June she was taken to another place out West belonging to these Sisters, but is no better so far as I know.

Of vasomotor neuroses, it is the mild and graver forms of hysteria under which tubercular phthisis loves to hide its insidious work. Female patients, generally with some hereditary taint, I have seen to present various spinal and arthropathic symptoms of an hysterical character for some years before the presence of pulmonary phthisis could be demonstrated.

The case of Mrs. F., hereditaria, æt. twenty-five, primipara, is interesting in this respect. She is a very stout woman, and has been under my treatment for two years, principally for rheumatoid joint-affections and neuralgias, which, I believe, are due to the action of the tubercular poison. She has never shown any fever, but has slight hæmoptysis from time to time, and whenever that occurs I am able to detect abnormal conditions in left or right apex, which disappears again with the hæmoptysis. She has no cough or expectoration generally, neither bacilli nor elastic fibres have been found in the sanguinolent sputa, but I am sure it is only a question of time in this case for destructive changes to develop in the lung-tissue.

Another case, which I saw in November, 1885, in this city, was that of Mrs. W., aged thirty-five, multipara, hereditaria, who, besides having slight cardiac disease, had been troubled for many months with hemianæsthesia, hemiparesis, transitory unilateral amaurosis, various neuralgias and arthralgias as we often see them in the graver forms of hysteria. Of late considerable fever had set in, the articular pain in lower extremities increased, and she had been confined to her bed for some weeks when I was called in to give advice as to the rheumatic fever from which she was

supposed to suffer, and which had thus far been refractory to quinine and the salicylates. There was but little cough and expectoration, yet there was no difficulty in proving disease of the apices and probably general tubercular infection, simulating rheumatic fever. Upon inquiry I have ascertained that since March last her neurotic and rheumatoid troubles are in abeyance, but the phthisical pulmonary process has become progressive.

The cases of phthisical insanity and phthisical neuroses I have seen thus far concerned mainly women; in nearly all of them the hereditary element played an important part, and the nervous symptoms were not traceable to other influences besides the tubercular infection, which led to malnutrition and functional disorder of the cerebral nervous system, often long before any pulmonary or intestinal lesion had been produced by it. I see no reason why the tubercular virus, having lain dormant, for instance, in the bronchial and other lymphatic glands, in hereditary cases, may not on entering the circulation affect the nervous system as well as the joints, bones, and other tissues, though I concede that many of the symptoms above described may be due to malnutrition and anæmia accompanying the tubercular disease. Almost always there is a perverse relation to be noticed between the pulmonary affection and that of the nervous system; while the former may be insignificant, often hardly suspected, the latter may alarm and worry the patient and his friends; and *vice versa*.

The course of phthisis, thus complicated, or marked is slowly but surely progressive; the prognosis as to permanent relief or even considerable improvement, worse than in ordinary cases, for the reason that there is so much difficulty on account of the fickleness of mind, feebleness of will, and marked distrust on the part of such patients to carry out a definite plan of treatment for a proper period of time. Besides, there is the hereditary element which so often is athwart all therapeutic measures.

So long as we do not know of a remedy which will be at least as good an antidote to the tubercle-virus as mercury is to syphilis, we had better give no drugs to such patients,

except for special purposes. But as much good has been accomplished in phthisis and therapeutics by sea and mountain air, the patient ought to be sent to a sanitarium selected with due regard to his individuality and the peculiar features of his case; and into the care of an accomplished physician who has learned to treat and manage phthisical patients rather than phthisis. I am afraid that many physicians are not aware that the phthisical patient needs often a good deal of moral treatment and management before we can depend upon him to carry out systematic and painstaking treatment for months—for years. A careful hygiene, continual supply of fresh air night and day, and yet scrupulous care in avoiding cold in the routine of daily life, proper clothing, frequent baths, and even douches to counteract the bad effects of and limit the profuse sweating, gentle and graduated exercise to invigorate the heart, I consider to be among the principal measures to help the patient in his fight with the disease. Of the utmost importance is the frequent supply of nutritious food by the mouth, by the rectum, combined with the judicious use of stimulants. Feeding, nay, over-feeding, as Debove advises, is indeed the *sine qua non* of an improvement or a possible cure in the case of a patient suffering from so chronic and debilitating a disease as pulmonary tuberculosis.

THE COLLOCATION OF A SUTURE AND FISSURE IN THE HUMAN FŒTUS.*

By BURT G. WILDER, M.D., of Ithaca, N. Y.

AT the meeting of this Association over which I had the honor to preside last year, I delivered a somewhat extended address upon a general question.[1] In retiring from office, therefore, it may be permitted me not only to make my remarks brief, but also to confine them to the statement of a single fact, with commentaries thereon. So far as I am aware, it is entirely new, which, as most of us know to our mingled chagrin and gratification, can seldom be claimed for any observation or idea in these days of intense encephalic activity.

Although, from the practical standpoint chiefly, considerable attention has been given to the topographical relations of the sutures and fissures in the adult, I am not aware that their fœtal relations have been noted or even mentioned as worth noting.

Statement of Facts.—In three alcoholic human fœtuses in the museum of Cornell University, estimated at from three to seven months, the lambdoidal suture directly overlies a short but deep fissure.

I submit herewith a photograph and drawing of the dorso-caudal aspect of the cerebrum of one of these.[2]

---

* Remarks of the retiring President of the American Neurological Association, July 21, 1886.

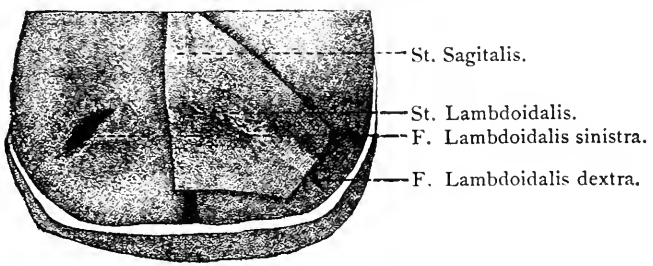
[1] See Bibliography at the end of this article.

[2] The photograph was taken by my colleague, Prof. S. H. Gage, and the drawing was made by his wife. The accompanying figure represents the preparation of natural size. The fissure should appear deeper and more sharply defined.

Before photographing, the line of the lambdoidal suture was inked on the pericranium for the sake of distinctness; in the drawing, the locations of the sutures are indicated as slightly raised tracts about 3 mm. wide.

From the left side the calva and dura have been removed, excepting a narrow strip along the meson; on the right there has been retained a strip 12–25 mm. wide, including most of the lambdoidal suture and part of the sagittal.

On the right the point of a fissure projects slightly beyond the cut edge of the calva, just entad of the lambdoidal suture; on the left the same fissure is wholly exposed, but it is antitropic with the right suture, and was situated directly entad of the left at the time I exposed the brain. The fissures are about 1 cm. long, and nearly half as deep. The edges and sides are smooth and natural, and there is no indication that they were artificially produced.



Among three other fœtal brains, estimated at from three to seven months, which had been previously exposed so that the existence of the collocation cannot be determined, the fissure is distinct in one, but absent or irregular in the other two. It is not represented in the figures of fœtal brains by Schwalbe (p. 574, fig. 352.), or Pansch (Taf. iii., figs. 7 and 9). Evidently numerous observations are needed with this special point in view.

In fœtuses of the last two months, and after birth, no collocation exists. If the fissure persists, it lies caudad of the suture.

The Name.—In 1868, Owen applied the name *lambdoidal* to the *dorsal end of the occipital fissure*, but the appellation has not been adopted by any subsequent writer, and the

correlation between the suture and the fissure here discussed is so marked and perfect that I hope the distinguished English anatomist will not object to the transfer. For the present, at least, I shall speak of it as the *lambdoidal fissure*.[1]

Not a New Fissure.—Although, as I am led to believe, but little known, the lambdoidal fissure is not altogether new. In 1868, Bischoff represented (**12**, Taf. iv., figs. 7–10, pp. 58–60), under the name of "fissura perpendicularis externa," what seems to me to be the lambdoidal, and it is referred to by Huxley, Kölliker, and Ecker: but these authors have, I think, mistaken it for other fissures, as will be more fully stated in connection with the question of its homology. The fissure is also shown, but not named, by Mihalkovics (Taf. iii., fig. 26), and by Gratiolet (Pl. xxix.). Finally, this is the fissure referred to in my recent paper "On the Par-occipital" (**76**, p. 6) as "fœtal, or possibly transitory."

Location.—The fissure may be described in the fœtus not only as situated at the lambdoidal suture, but also as coinciding with about the middle third of a line extending obliquely across the hemicerebrum from at or near the dorsal outcrop of the occipital fissure, forming an angle of 45 degrees with the meson, and related sagittally to the caudo-lateral outline. Approximately, therefore, it forms one of the two straight sides of an octant of the dorso-caudal surface of the hemicerebrum, when that surface is perpendicularly regarded.

Form.—Unlike the earlier states of most fissures when no longer than this, the lambdoidal is deeper than wide, and half as deep as long; unlike them, also, the sides and ends are nearly vertical, and the bottom approximately flat and pointed at each end.

An Early Fissure.—With the possible exception of the dorsal end of the occipital, there are no other fissures on the convex surface of the parieto-occipital region of the ex-

[1] On page 126 of the work above named, Owen speaks of the correspondence in position between the coronal and lambdoidal sutures and fissures; but he is treating of adult monkeys, not human fœtuses, and his "coronal" fissure it the central or Rolandic, while his "lambdoidal" is part of the occipital, on the one here under consideration.

ample figured; the lambdoidal therefore appears before the parietal and paroccipital fissures.

Ental Correlative.—So early and deep an ectal depression might well be expected to represent an ental or ento-cœlian elevation, like the hippocampal and calcarine fissures and some others which will be mentioned in the "Notes on the Brain," which I shall present later in this meeting. So far, however, no ental correlative has been observed, but the earlier fœtuses in which the lambdoidal fissure exists have not yet been sufficiently examined in this respect.

Homology.—By Bischoff (**12**, 58–60) and Huxley (p. 492) this fissure is regarded as representing the "external perpendicular or occipito-temporal fissure" of apes, and the latter states that its "filling-up" is, aside from size, the chief characteristic of the human brain. Ecker (46) and Kölliker (Fig. 359) regard it as the first appearance of the "transverse occipital fissure," which I have recently attempted to show (76) is really part of the paroccipital. The special subject of these remarks, namely, the collocation of the lambdoidal fissure with the suture of the same name, need not be affected by the question of its homology, so I content myself on this occasion with dissenting from the interpretations of the anatomists just named, and hope on a future occasion, in connection with the so-called "ape-fissure" of man, to give the grounds for the view that the fissure persists but becomes obscured and almost lost among the numerous and complex fissures of the caudal end of the adult occipital lobe.[1]

The Collocation Transitory.—Although, as just stated, I am inclined to think that the lambdoidal fissure itself is persistent, yet its attribute of collocation with the suture of the same name is certainly temporary, and thus, like all transitory conditions, interesting as suggesting a permanent ancestral feature.

Another Case of Collocation.—In at least two of the fœtal

[1] It is possible, though I think improbable, that the lambdoidal fissure becomes what Wernicke and Schwalbe (p. 550, Fig. 342, *O A.*) have called "occipitalis anterior," but for which, in a paper (**81**) prepared since this address was presented, I have proposed the mononym *exoccipitalis*, for distinction from the *præoccipitalis* of Meynert.

brains examined, the coronal suture on each side was found to overlie directly a well-marked fissure, which was apparently the precentral. There was, however, some laceration of the surface in these cases, and I refrain from enlarging upon the small number of observations on imperfect material.

Questions.—The following questions naturally occur in connection with the subject:

1. At what age does the lambdoidal fissure first appear?
2. Is it from the first collocated with the suture of that name?
3. How generally is the fissure present?
4. When the fissure is present, is the collocation invariable?
5. What is the proximate cause of the collocation? Is it mechanical, or vascular? or is this a mere coincidence with no relation of cause or effect, or even of relation to some common cause?
6. At what age does the collocation cease to exist?
7. What is the proximate cause of the loss of collocation?
8. Has this fissure any relation with the occipital?
9. Does the fissure ever disappear entirely?
10. If not, does it remain independent or become continuous with others?
11. Does the collocation exist in other mammals, especially in the primates?
12. For many reasons all facts bearing upon the physical peculiarities of man are of interest. Hence we wish to ascertain whether either the fissure itself, or its collocation, or the temporary existence of the fissure, or the cessation of the collocation, are peculiar to man or to the primates.

Until these have been satisfactorily cleared up, no opportunity should be lost for observation. Hence I venture to offer a few

Suggestions.—1. The fœtuses should be weighed and measured, and notes made of the various conditions by which unknown ages are estimated.

2. Fœtal heads, or entire bodies, or the brains alone, should be injected with alcohol, and then with red starch mass, as directed by S. H. Gage.

3. The brains should be exposed with the scissors and nippers, carefully, and in brine if necessary; the parietal bone of one side should be removed, beginning near the meson, and leaving the frontal and occipital bones to support the corresponding regions of the brain until the relations of the sutures and fissures have been determined.

4. Preparations should be preserved and photographed.

5. If possible these brains should be exposed by the anatomist himself or by a trained preparator under his eye. The present remarks might not have had an occasion if the exposure of the brain had been delegated as a purely mechanical operation.

In resigning the chair to my successor,[1] I desire to congratulate him and the Association upon the choice, and to express my personal pleasure in the change of date, by which my own attendance upon the meetings will not, as heretofore, be rendered difficult or impossible on account of annual duties elsewhere.

BIBLIOGRAPHY.

Bischoff, T. L. W.: **12.** Die Grosshirnwindungen des Menschen mit Berücksichtigung ihrer Anordnung bei den Affen. München Akad. Abhandl., x., 389–498. 1870.

Ecker, A.: (Trans. by Edes.) The cerebral convolutions of man, etc. O., pp. 87, 6 figs. New York, 1873.

Gratiolet: Mémoire sur les plis cerebraux de l'homme et des primates. Q., pp. 104, 13 plates. 1854.

Huxley, T. H.: Manual of the comparative anatomy of vertebrated animals. O., pp. 431, 110 figs. New York, 1873.

Kölliker, A.: Entwicklungsgeschichte des Menschen und der hoheren Thiere. O., pp. 1033, 606 figs. Leipzig, 1879.

Mihalkovics V. von: Entwicklungsgeschichte des Gehirns, etc. Q., pp. 105, 7 plates. Leipzig, 1877.

Owen, R.: Comparative anatomy and physiology of vertebrates. O., 3 vols. pp. 2155, 1471 figs. London, 1861, 1868.

Pansch, A.: Die Furchen und Wulste am Grosshirn des Menschen. O., pp. 51, 3 plates. Berlin, 1879.

Schwalbe, G.: Lehrbuch der Neurologie. O., pp. 1026, 319 figs. Part of Hoffmann's "Lehrbuch der Anatomie des Menschen." Erlangen, 1881.

Wilder, B. G.: ***64.*** Paronymy versus heteronymy as neuronymic principles, Presidential address at the eleventh annual meeting of the Am. Neurol. Assoc., June 18, 1885.—JOUR. NERV. AND MENT. DIS., July, 1885, pp. 21.

——***76.*** The paroccipital, a newly recognized fissural integer. JOUR. NERV. AND MENT. DIS., xiii., 301–315, 5 figs., June, 1886.

——***81.*** Human cerebral fissures: their relations and names, and the methods of studying them. Am. Assoc. Adv. Sci., 1886.

[1] Dr. Chas. K. Mills.

NOTES ON THE BRAIN.*

By BURT G. WILDER, M.D.

ADDITIONAL Case of Independence of the Paroccipital Fissure.—In my recent paper "On the Paroccipital Fissure" (*76*, 308), I gave the result of a tabulation of the reliable specimens and figures accessible to me as follows: among 43 hemicerebrums, the parietal and paroccipital are continuous in 21 and separate in 22. Since the publication of that paper I have prepared the perfectly fresh brain of a negro child (No. 1834 of the Museum of Cornell University) that died at birth. In addition to other interesting facts which I hope to present at a future meeting, this brain has the two fissures totally independent on the right and barely united by a very shallow junction on the left. Even, then, if the latter be admitted as technically an example of continuity, the totals are now 22 unions to 23 cases of independence.[1] This brain, it will be seen, also illustrates Ecker's generalization, that the interruption of his "interparietal," which he regards as normally a continuous fissure, occurs more frequently on the right side. The interruption has now been observed on 10 right sides and only 4 left, while the junction occurs on 11 left and only 6

* Read at the meeting of the Am. Neurological Association, July, 1886.

[1] Since this paper was read Dr. C. L. Dana has kindly put in my hands his notes and diagrams of twelve hemicerebrums in his possession: in seven the parietal and paroccipital are connected, how deeply not ascertained; in one the junction is by a "very shallow depression"; in the other four, the separation is complete. The paroccipital is likewise wholly independent on both sides of a second brain of a negro child at birth (No. 2041, M. C. U.) which was removed by Dr. Formad and sent to Dr. C. K. Mills who generously turned it over to me. Admitting that Dana's case of shallow union should be placed with the series of continuity, the totals are now thirty cases of continuity to twenty-nine of complete independence.

right. It remains to ascertain the cause and significance of this difference between the two sides. As stated by me in 1873 (11, 219) a fissure represents more cinerea than a superficial gyral surface of equal width, so we may associate this greater frequency of cinereal increase in a limited locality with the commonly superior weight and activity of the entire left hemicerebrum.

An Ental Correlative of the Occipital Fissure in the Early Fœtus.—As shown by the exhibited photograph and figure, in a fœtus (No. 1816 in Mus. of Cornell University) estimated to be about four months old, there is, immediately opposite the somewhat deep occipital fissure, a distinct elevation of the mesal wall of the postcornu, just dorso-cephalad of the calcar. The wall here is but little thinner than elsewhere, so that there is the appearance of a wrinkle or fold in the entire thickness as with the calcar. After the observation was made, this spring, I noticed that a similar elevation corresponding with the occipital fissure is figured by Tiedemann (Pl. IX., Fig. 2), who, however, does not mention it in the text, and that an ental elevation corresponding to the occipital fissure is mentioned by Mihalkovics (146), who implies that it is mentioned by His. Although I have not had an opportunity of looking in other fœtal brains, the distinctness of the elevation in the example here described, leads me to anticipate that it will be found a constant feature of the early fœtal brain.

Besides its constancy in the fœtus, three other questions arise in connection with it: 1. Does it persist in the adult? 2. If so, has it been already recognized? 3. How shall it be designated?

None of these questions can be answered satisfactorily with the information now at hand. At present it seems probable that with some brains the general thickening of the postcornual parietes renders this early ridge hardly, if at all, distinguishable; but that in others it may be recognized as the oblique elevation dorso-cephalad of the calcar, which was called by Henle (p. 168, Figs. 91–93) *bulbus cornu posterioris*, by Quain (11, 346) *bulb of the posterior cornu*, and by me, in ignorance of the foregoing,

eminentia splenialis (58, 234, 273, Fig. 44). If it should prove to be distinct from the postcornual bulb, it may well be named *eminentia occipitalis*.

Meantime, its relation to the occipital fissure in the fœtus justifies the inclusion of the occipital among the *total fissures*, or foldings of the entire thickness of the cœlian parietes, as distinguished from *cortex fissures*, or depressions reaching to a certain depth only. The other such cases of correlation between ental elevations and fissures are: calcar and calcarine fissure; hippocamp and hippocampal fissure; eminentia collateralis and collateral fissure; striatum and Sylvian fissure. In addition, the following are cases of structural correlation although the part does not project into the encephalocœle as an elevation: olfactory lobe and fissure; amygdala and amygdaline fissure, or postrhinal fissure. See my paper (***66***).

The Fœtal Extension of the Proplex to the End of the Postcornu.—In the adult the proplex or procœlian (lateral) portion of the prosoplex is practically confined to the cella and medicornu, with occasionally a pointed projection into the other cornus, as represented in my preparation, No. —, M. C. U. Since, however, the early hemicerebrum is nearly accurately filled by the plexus, it might be expected that intermediate stages would be found on careful examination. In the photograph here shown, enlarged about two diameters from a horizontal section of a fœtal brain estimated at —— months, the plexus is seen to extend to the end of the postcornu. Most of the postcornual portion, however, seems thinner and less substantial than the rest, as if in process of atrophy. Whether it comes off or simply shrinks cannot be ascertained as yet. There were indications of a similar precornual prolongation.

Points Illustrated by the Transection of a Fœtal Brain.—As shown in the exhibited photograph, a transection of the brain of a fœtus, No. 1816, estimated at four months (the same which presented the ental correlative of the occipital fissure), exhibits several features which are morphologically suggestive.

1. The medicommissure is perfectly distinct and well preserved.

2. The procœles (lateral ventricles) are higher than wide, and half their height lies dorsad of the level of the callosum.

3. The caudatum (caudate portion of the striatum) forms a marked projection of the lateral wall.

4. The fornix at this level occupies about one third of the entire width of the cerebrum, whereas in the adult it measures not more than one fifth and probably more nearly one seventh.

5. The fimbria, constituting the margin of the fornix, reaches the *sulcus limitans*, which demarcates the thalamus from the striatum, the diencephal from the prosencephal. In other words, each hemifornix is equal in width to the corresponding thalamus.

6. As corollaries to the preceding: (*A*) the dorsal surface of the thalamus is wholly pial, with no endymal portion as in the adult; (*B*) the thalamus does not, in any sense, enter into the composition of the procœlian floor. This point is discussed at some length in my paper, ***56***, pp. 460, 461.

It will be seen that most of the foregoing points are more or less distinctly related to the increase in width of the entire brain, and specifically to the lateral extension of the thalami during the later stages of fœtal life.

The non-appearance of the thalamus in the procœle, and the concomitant narrowness of the rima, are also significant, because it is the permanent condition in all other mammals, with the possible exception of some other primates. Hence these facts bear upon the important question as to the nature, extent, and significance of human or primatial peculiarities.

BIBLIOGRAPHY.

Mihalkovics, V. von: Entwicklungsgeschichte des Gehirns, etc. Q., 195, 7 plates. Leipzig, 1877.

Wilder, B. G.: **11.** The outer cerebral fissures of mammalia (especially of the carnivora), and the limits of their homologies. Am. Assoc. Proc., xxii., 1873 (pt. 2), pp. 214–234; 19 figures.

——***56.*** Methods of studying the brain: The "Cartwright Lectures" for 1884. *N. Y. Med. Jour.*, xxxix., 1884, pp. 141–148, 177–183, 205–209, 233–237, 373–377, 457–461, 513–516, 653–656; xl., 113–116; 64 figs. Abstracts in *N. Y. Med. Record*, xxv., 1884, pp. 141–143, 197–199, 225–227, 365–367, 449, 450, 545, 546.

——***66.*** On two little known cerebral fissures, with suggestions as to fissural and gyral names. Amer. Neurol. Trans.; JOUR. NERV. AND MENT. DIS., xii., 350–352, July, 1885.

——***76.*** The Paroccipital, a newly recognized fissural integer. JOUR. NERV. AND MENT. DIS., xiii., pp. 301–315; 5 figs.; June, 1886.

THE EVOLUTION OF THE SPECIAL SENSES.

By W. C. CAHALL, M.D.

BY sensation the mind is cognizant of an outer world; by expression the outer world is cognizant of the mind. All grades of animal life possess in some form organs of sensation. We do not care to except even that substratum of life—protoplasm. This curiously vitalized structure lives and reproduces itself without organs; moves without limbs; takes food without a mouth, and digests it without a stomach. It will not be a surprise when we find that it has sensation without nerves. More properly, it might be considered as a unit of being, functioning as an organ. Of the nature of this sensation nothing definite can be said. It is known that it is irritable, and able to discriminate between food and indigestible substances. Among the unicellular organisms, sensation is of a higher degree, though of the same character as that we find in the indifferent protoplasmic bodies, and the sensation even here is limited to the protoplasm of these organisms. As we ascend the scale of life it is not until we reach the medusa family that we meet the sensory organs sufficiently developed to deserve the name, for here do we find the first evidence of a separate nervous system. Hitherto, throughout the preceding groups, the differentiation had attained only to the neuro-muscular tissue, in which the cell acted in the dual rôle of muscle and nerve. Evidently here was the first opportunity for the appearing of special organs of sense, since sensory organs are but the terminal organs of a sensory nerve. These sensory organs of the medusa are extremely

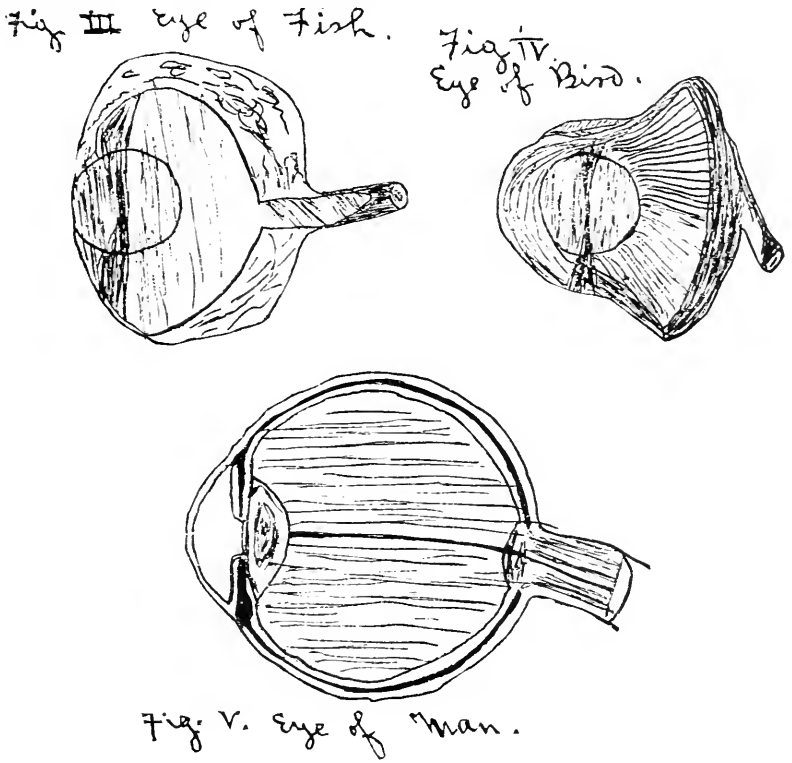
rudimentary, but they are a forecast of those perfect organs which are to come. The tentacles of the medusæ (jelly fishes) are abundantly supplied with tactile organs, which greatly assist them in their selection of food. An additional amplification of the sensory apparatus appearing at this stage are the "marginal bodies," which are situated upon the margin of the bell of the jelly-fish, and are of sufficient development to manifest the rudiments of an ear and eye. The "ears" are vesicles or sacs filled with a fluid substance, and containing concretions or crystals of lime, attached to the walls, from which run nervous filaments to the ganglionic centres. As the jelly-fish lives in the water, it is readily seen how the vibrations of sound, travelling through the water, could be transmitted to the fluid contents of the vesicle, and how this agitation may cause concussion of the particles of lime, and how, from the connec-

Fig. 1.—Marginal body of medusæ. (After Gegenbaur.)

tion of the nervous fibrils with these concretions, the organism becomes conscious of the sound. The "eyes" are patches of pigment, in which nerve-fibres terminate. Surmounting and imbedded in each of these pigment spots is a transparent, highly refractive lens-like body. Simple as is this apparatus, it possesses the essential elements for vision, truly not as we know it, but sufficient for all the needs of the creature.

Among the vermes, or worm family, may be seen examples of the gradual evolution of the organs from this simple condition to that of a really complex structure. In the lower forms, the eye remains a pigment spot, while, by slight gradations, it becomes in the higher forms an apparatus possessing an expanded optic nerve, vitreous humor, and a crystalline lens. The auditory organs of the

crustacea furnish an example of a direct adaptation of organs of general sensation into those of a special order. These organs lie in a cup-shaped indentation of the integument. The cavities in some instances are closed, and in others open. In the latter case the otoliths are grains of sand brought in from the exterior, which are attached to hairs of the integument enclosed by the indentation. These



hairs are but the modified hairs or tactile rods, which are found scattered over the surface of the body, and with whose roots or bulbs nerve filaments are connected.

In the insecta the development continues. Over the opening is stretched a tympanum or drumhead, and instead of the hairs and sand, there is connected with this drumhead a ridge of regularly-arranged club-shaped rods of nervous structure. Here is the first appearance of the drum-

head. In the eye of the higher mollusca the noticeable features are a recession of the lens from the cornea, dividing the cavity of the eye into the anterior and posterior chambers, and the presence of an iris and pupil. The auditory organs show an approach toward a membranous and cartilaginous labyrinth.

The eye of the vertebrate has three stages in its evolution before it reaches the perfect type of the mammal. The first stage is that of the fish; the second that of the bird, and the third that of man. Although among the invertebrates there is a certain mobility of the eye, only in the vertebrates is found that complex muscular attachment by which the eye is so readily directed in any direction Here also are perfected the integumentary folds—the eyelids,—designed to protect the delicate organ from injury. Since the eyes of the fishes and the cetacea of the mammals are constantly bathed by the water in which they live, it is obvious that any lachrymal apparatus would be superfluous. Consequently they are here absent, but are found among the higher vertebrates. The ear of the fish has a labyrinth and semicircular canals, where the nerve filaments terminate as the end organs or otoliths. The labyrinth undergoes gradual but great modification as we ascend the scale of vertebrates. The most important one is the development of the cochlea. In the fishes it exists as a small eminence or diverticulum; in the birds as a straight cone; while in the mammals this cone is turned upon itself in a spiral manner, and becomes the seat of the organs of Corti.

The tympanum is almost universally present in the vertebrates, and so early as the saurians has receded from the surface, thus forming an external auditory meatus. Connecting the tympanum with the labyrinth is a chain of auditory ossicles, intended to convey and magnify sound. These bones also run from the simple to the complex, as the examination extends from the fishes to the mammals.

The external ear is not met with until a high stage of vertebral development is attained.

I have purposely refrained from noticing the development of the gustatory and olfactory senses, for it is difficult

to speak definitely of these senses at any distance beneath the vertebrates, nor do I conceive it greatly necessary, for the differentiation of the organs of these senses is so slightly above that of the general sense of touch, that when one is satisfied of the evolution of the higher senses, there would be little hesitation in accepting it in regard to the lower. It will be noticed that nature was consistent in her process of evolution, and did not depart, even in the most complete organs, from the first principles with which she started, for the lens-like crystal and pigment spot of the lowest

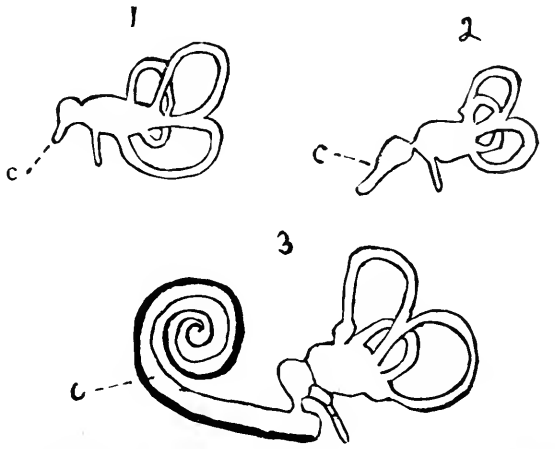


Fig. 2.—Diagrams of labyrinth. 1. Fish. 2. Bird. 3. Mammal. c. Cochlea. (After Waldeyer.)

orders have their analogues all the way up the scale, whether in the crystalline cone and pigment layer of the eye of the insect, or in the crystalline lens, rods and cones of retina, and choroidal (pigment) coat of the human eye.

THE PHYSICS OF SENSATION.

Now that science is conversant with the physics of sensation, it is easy to understand how such an evolution was possible. Sensation is vibration, and the modifications of sensation are but modifications of vibrations. There are no more mathematically exact branches of physics than acoustics and optics, and the essential basis of these is vibration. General sensibility is only possible by actual contact

of bodies in mass, producing a low grade of vibration. This is also true, to a great extent, of the senses of smell and taste, although the particles of the impinging body are more minute. There is associated with general sensation the appreciation of heat, and every student of physics knows that heat is but vibration. Thus we have the senses of touch, taste, and smell, of a group, of low stage of evolution from general sensibility, and necessitating actual contact of bodies or their molecules in order to produce that peculiar vibration which is translated into sensation. It remains to speak of those more elaborately differentiated special senses, viz., hearing and sight. For hearing, the sensory apparatus must be modified to receive vibrations of greater amplitude than those of heat; and for sight, those of lesser amplitude. In the structure of the end organs of these senses, we see the adaptation. In the hairs and grains of sand of the lower forms of life, or the nervous fibrilla and otoliths of the higher forms, we see an organ peculiarly adapted to receive the grosser vibrations of the conducting medium. The refining evolution of the sight organs is not so difficult. The vibrations of light are more closely associated in rapidity to those of heat than are the vibrations of sound. A lens will condense both heat and light vibrations. Indeed, heat is associated as the invisible rays of the spectrum, and lying just beneath the light rays. The apparatus of sight consists of a lens, which condenses the rays, and a nervous plate, which responds to the lowest vibrations of light, yet will not respond to those of heat.

Thus I have attempted to show that consciousness of the material world comes through vibration, and that the special senses are but modifications of the undifferentiated nervous structure, each adapted to vibrations within a narrow limit, and developed for the purpose of widening the horizon of knowledge, and rendering possible a life upon earth other than a purely vegetative one.

THE EFFECT OF USE AND DISUSE UPON THE ORGANS.

While use and necessity develop the special organs of sense, disuse will as certainly allow them to degenerate and

lose their distinguishing characteristics, and the instances illustrating this law of adaptability to environment from trifling changes to complete annihilation are numerous, and it is interesting to note the methods to which nature resorts to overcome or compensate for the obstructions in her path. At great depths in the ocean the light is extremely feeble. Indeed, some animals have given up the use of their eyes entirely, and depend upon their other senses, which, by use, become so acute that they do not miss their eyes, and perhaps consider as myths the traditions of their ancestors that they once saw. Among others, as the crabs, there is an effort made to fight the darkness of the ocean valleys by an enormous development of the eyes, which are sometimes four or or five times as large as those of crabs living at the surface. In addition to this great development of the eye, some marine animals have their eyes phosphorescent, which glow through the dark waters, and light their way ahead, like the head-light of a locomotive in miniature. As if not satisfied with this, some are arrayed with rows of phosphorescent spots along their bodies, which, if the simile may be carried further, are like the lights shining through the windows of the cars. The Niphargus and the Onesimus are animals whose eyes are no longer used, but have the organs of smell and touch highly developed. These organs—and here is the beautiful part of it—are situated upon the antennæ of those which live in the water and where the antennæ are used in the selection of food, and upon the blades of the foot-jaws of those which live and dig for food in the mud of the bottom, where the antennæ could be of no assistance.

There are numerous cases on record where men, having lost their sight or hearing, have developed the acuteness of the remaining senses to a marvellous degree.

The skill of reading by means of the elevated type, and the power of recognizing acquaintances after long intervals by the slightest touch of the hand, are familiar examples of the degree of touch which the blind display. Professor Carpenter, in his Physiology, mentions the case of a blind man "who had acquired a very complete knowledge of

conchology, both recent and fossil, and who is not only able to recognize every one of the numerous specimens in his own cabinet, but to mention the nearest alliances of a shell previously unknown to him when he has thoroughly examined it by his touch"; and that of John Gough, who, though blind, was a noted botanical collector; but what is most wonderful of all, and there are too many instances to leave any doubt, the blind, by means of the touch, have been enabled to distinguish colors, and that upon surfaces similar in other respects.

REMARKS ON EPILEPSY.

By Dr. E. D. FISHER, New York,

LECTURER ON NERVOUS DISEASES, N. Y. UNIV. MED. DEPT.

IT is with considerable hesitancy that I approach this subject, fully comprehendiug its difficulties and my inability to adequately handle it.

The character of the times seems to render it almost impossible, in the great number of conflicting duties and occupations of the hour, for one to concentrate his time and mind on any one subject.

In the department of diseases of the nervous system, one at present finds it necessary to know more of general medicine outside of any special knowledge of this department itself than what a few years ago would have constituted a liberal education in medicine.

The object of my paper shall be to emphasize the idea of the cerebral character of epilepsy, and to place it in the class of organic diseases, rather than its generally accepted classification as a functional disorder.

As Gowers, in "Diseases of the Spinal Cord," well says: "Strictly speaking, a functional disease is one which consists in a disorder of function without any preceding alteration of nutrition," and affections of the nervous system which can be included under this head are very few.

In most cases of so-called functional disease we must assume changes in nutrition.

Ross, in the last edition of his work on nervous diseases, defines epilepsy as a chronic functional disease of the nervous system, characterized by loss of consciousness, and convulsions.

Krafft-Ebing, in his "Lehrbuch für Psychiatrie," describes it as a disease marked by repeated attacks of complete or incomplete loss of consciousness, or even slight dulness of intellect, accompanied by partial or general convulsions dependent on spasm of the cerebral arteries.

Brown-Séquard has, perhaps, given us the most satisfactory definition. He says: "Epilepsy seems to consist in an increased reflex excitability of certain parts of the cerebro-spinal axis, and in the loss of control that in the normal condition the will possesses over the reflex faculty."

He places its cause as in the medulla.

The disturbance of the functions of the cerebral lobes during and immediately after a fit, and in the interparoxysmal period, he ascribes as due to alteration in the cerebral substance taking place during the seizure.

The hitherto mysterious coincidence of loss of consciousness, or, in other words, loss of function of the cerebral lobes, with increased action of the medulla, in an epileptic attack, is thus easily explained.

In describing epilepsy, great stress is laid on the character of the seizure, the attending epileptic cry, the unilateral or bilateral form of the convulsion, and but little attention is paid to the previous mental state, or to the condition in the intervals between the attacks—that which is called by Nothnagel the epileptic change, or, still better, by Griesinger the epileptic condition.

I do not refer to the status epilepticus, that condition immediately following a paroxysm, in which the patient may be in a state of mania, or in which paroxysm may follow paroxysm for hours or days, but I mean the period intervening between the attacks, be it six days or as many years.

The question is, then, What produces this epileptic condition, and what the outbreak of the seizures?

It seems to me the confusing of these two has caused the confusion in our ideas of the nature and seat of the disease.

Unconsciousness and convulsions may be produced by various causes. We know excessive loss of blood, ligature of the carotids, local or general cerebral anæmia, may account for them.

Bernard, on cutting the sympathetic in the neck and extirpating the superior cervical ganglion, caused an increase of temperature on that side of the brain.

Nothnagel found that, after performing the above experiment, irritation of the crural nerve caused contraction of the vessels of the pia mater, and while cutting of the sympathetic caused the dilatation of the vessels, faradization of the cut extremity caused narrowing. Van der Beke and Callenfels obtained similar results. The author then states:—these experiments prove three things: first, that the vasomotor nerves for the vessels of the pia course in part through the fibres of the sympathetic in the neck; and, second, that perhaps another set, more important, pass through the superior cervical ganglion; and, lastly, that above the ganglion are fibres probably accompanying the cranial nerves.

This would explain, in the reflex epilepsy following teething, intestinal irritation, and injuries, the contraction of the vessels of the brain which follows.

There may be other causes, however, for convulsions besides cerebral anæmia. The cerebrum can be removed, and yet convulsions follow. Marshall Hall long ago stated that anæmia of the medulla oblongata was the cause of convulsions. There is no doubt that irritation of the medulla can cause them, and were this all of epilepsy, we might well rest our case with this part of the nervous system as the seat of the disease. Nothnagel has proven the existence of the convulsive centre in the medulla, and the presence also of the vasomotor centre explains, by its producing cerebral anæmia, the accompanying unconsciousness, but this does not explain the intellectual deterioration present, the epileptic condition to which I have already referred, and which I regard as the most essential feature of the disease.

Cerebral congestion was for a long time held as the cause of epilepsy, but Brown-Séquard, Bernard, Schiff, and others have proved that this is secondary to the convulsion.

Trousseau says of this:—there are therefore two very distinct conditions in an attack of eclampsia or epilepsy,

whether idiopathic or symptomatic: first, a cerebo-spinal modification unknown in its essence and its nature, which in a second abolishes all the manifestations of animal life; and, second, a secondary cerebral congestion, which, although less important, may in some extremely rare cases be carried so far as to produce subcutaneous ecchymoses, cerebral capillary hemorrhage, and even meningeal hemorrhage. In the interval between the seizures there is a change in the individual. We may have mania or delirium continuing for weeks; there is a marked loss of intellectual power, and of memory, an indefiniteness of ideas, an irritability of temper, or, as Esquirol puts it, if not insane, the character is peculiar, irritable, capricious, paradoxical, the features become coarse, a precocious senility sets in.

Who has not observed the obstinacy of the epileptic, the look of vacancy or fatuity, the mental weakness?

In the cases that have come under my observation in the last five years, few if any have preserved their full intellectual power. This would speak against the theory of regarding the seat of the disease as in the medulla.

Extensive implication of the medulla, as in primary and secondary bulbar paralysis, does not involve the intelligence. I have two cases of that nature at present under observation, where the intelligence is absolutely intact.

I also protest against calling a single convulsive seizure caused by teething or indigestion, as epileptic.

Should these attacks continue, then, as a result of repeated sensory irritation conveyed to the brain, the cerebral cortex may become involved. Chronicity must be present in this disease as a factor.

The cortex is the location of all psychical processes which are present to our consciousness; it is the seat of memory and all acts of the will (Edinger).

In general paralysis Tuczeck shows the first layer of the cortex is first involved, and so in order to the fourth. Not until the first month of life, however, do the cerebral fibres assume a sheath, and until then the child's acts are all reflex.

The fibræ propriæ or association fibres are the first to as-

sume this sheath, and these fibres play an important part in the extension of an epileptic seizure.

When we have simply loss of certain mental functions, as paroxysmal attacks of loss of words, or loss of the power of reading, which may precede the seizure or be present without the latter, we may well conceive that certain convolutions containing the centres of these attributes are affected.

Should this extend its irritative action, the surrounding centres controlling motor or sensor functions might be involved, and thus lead to a more extended class of symptoms.

Epilepsy *per se* is a chronic condition or disease of the cerebral cortex, manifesting itself by periodical attacks of convulsions, etc., with a progressive intellectual decline and tendency towards insanity, and belongs to the class of cases coming under the head of general paresis.

Simple convulsions are distinguished by their constant relation to some known cause, and by their yielding to treatment directed against it. Hitzig speaks of the continuance of the epilepsy after the removal of the irritation. I think this sustains the idea of a morbid process having been established. The cause may have been removed; the condition, however, has been induced, and now we find the permanent symptoms of the disease present.

The tendency at present is to regard the loss of consciousness, be it slight or great, with or without spasm, even dizziness,—in fact, any alteration of mental activity occurring paroxysmally, as epilepsy. I certainly hold with this view. I would look upon the epileptic seizures as simply expressions of the disease as existing in the degeneration of the gray matter of the cortex. It may involve any part, as the centre of speech or a motor centre, causing loss of control over the lower centres in the medulla, and thus lead to convulsions. Just as Bright's disease is accompanied by uræmic convulsions, the latter not being the disease but simply an expression of the diseased condition of the kidney, which may produce a large number of other changes in the system, so in epilepsy, we have various

consequences of the morbid state express themselves in convulsions, mental weakness, or even insanity. In the one we have arrived at a definite knowledge of the pathological changes, in the latter we have not as yet, but should look for them in the cortex. If in the frog, after removal of the cerebrum, the reflex acts are increased, it is fair to presume that in epilepsy, the cerebral centres being for the time in abeyance, the lower spinal and medulla centres, being no longer inhibited, are subject to increased reflex action on the slightest external irritation. Let the cerebral loss of function be produced by anæmia, or otherwise, does not affect the principle. If we consider that cerebral irritation is capable, independent of lesion of the medulla, of producing convulsions and loss of consciousness, it seems more probable, in consideration of the many mental symptoms present in epilepsy, and which can only have their origin in the cortex, that the cerebrum is also the original seat of the convulsions.

Hysterical attacks, although often as violent as epileptic convulsion, do not lead to any condition similar to that of epilepsy.

Who has not observed the tonic and clonic contractions in hysteria, involving the face, extremities, and trunk, and even partial loss of consciousness? and yet, although this may be often repeated, the after-effects never resemble those seen in epilepsy. The definition of the disease as given in the earlier part of this paper, in which it was described as a sudden but temporary loss of function of any or all the cerebral centres, is hardly correct, as implying that between these attacks a condition of restoration of cerebral function exists. As Krafft-Ebing has well put it, the epileptic shows a psychical degeneration, a loss of intellectual power up to complete imbecility, showing itself by forgetfulness and slowness of judgment and perception. I would, therefore, define epilepsy as a disease of the cerebral cortex, attended by a progressive decline of the intellectual powers and by paroxysmal attacks of partial or complete loss of consciousness, accompanied by convulsions involving a part or the whole of the body.

In reference to the treatment of epilepsy I depend on the bromides exclusively, usually commencing with thirty-grain doses three times a day.

I prefer, as a rule, the mixed bromides, in the proportion of four parts of the potassium bromide to two parts each of the sodium and ammonium bromide; when cardiac weakness is present, employing digitalis or aromatic spirits of ammonia.

I have not found, as some affirm, that iron acts deleteriously—that is, when anæmia is present. Nor does the continued use of the bromides for years even, in my experience, lead to any mental disturbance; in fact, I find a gradual improvement in this respect whenever I succeed in controlling the disease.

In regard to cure of the disease, in my opinion that is less often accomplished than amelioration of the condition.

Clinical Cases.

REPORT OF TWO SUCCESSFUL CASES OF TREPHINING FOR TRAUMATIC EPILEPSY.*

By CARLOS F. MACDONALD, M.D.,

MEDICAL SUPERINTENDENT N. Y. STATE ASYLUM FOR INSANE CRIMINALS.

The operation of trephining the skull having been performed at the State Asylum for Insane Criminals under my charge, in two cases of traumatic epilepsy during the past year, and in each instance with favorable result, as regards the epileptic seizures, I venture to occupy your attention with a brief recital of the cases, in the hope that they may prove clinically interesting, and also for the purpose of adding them to the list of recorded cases of trephining operations upon the skull for the relief of epilepsy of traumatic origin.

CASE I.—J. M.; male; age twenty-nine. Convict; peddler by occupation, and of intemperate habits. Admitted to the asylum Nov. 6, 1885, as a case of acute mania. Certified cause of insanity, a blow on the head; which, patient claims, was inflicted during a street fight in New York City, in the year 1880; he at that time having been struck by a policeman's club and knocked senseless. He was picked up and removed to a hospital in an unconscious condition; he regained consciousness soon after admission to the hospital; but was ill for about two weeks, as

* Read before the Third District Branch of the N. Y. State Med. Ass'n at Binghamton, N. Y., June 17, 1886.

an effect of the blow. Subsequently, and up to the time of his reception in the asylum, he suffered much from attacks of vertigo, was irritable and quarrelsome; and frequently became a participant in fights and brawls. On admission he exhibited delusions of persecution and poisoning; was excited, noisy, and seemed to be suffering from great pain in the head. His pupils were unequal and markedly dilated, the right the more, and it was with difficulty that he was prevented from doing serious injury to himself by striking his head violently against the wall and floor; which he frequently and determinedly endeavored to do.

An examination of patient's head revealed an accentuated depression of the skull, irregularly circular in shape, and about $1\frac{1}{4}$ inches in diameter. It was situated above and to the left of the right ear, three inches superior to the external occipital protuberance, and two inches to the right of the median line, approximately covering that part of the posterior segment of the superior parietal lobule lying just above the deep interparietal fissure.

Dec. 2, 1885.—After breakfasting the patient fell in a fit, while still in the dining-room; he was convulsed and unconscious for a few minutes, then, partially regaining consciousness, he attacked the attendants who were caring for him, and violently resisted their efforts to remove him to the ward. On the evening of the same day he suffered a similar attack, and remained excitable for some time after emerging therefrom. During the interim he was irritable for a great part of the time and disposed to attack, without provocation, those about him. At this time he was seen by Dr. Stephen Smith, State Commissioner in Lunacy, who concurred in advising trephining as a probable means of relief, if not cure. I might state that the patient, when free from the epileptic paroxysms, was anxious to have the operation performed.

Dec. 15, 1885.—Patient suffered from three distinct and separate attacks on the 12th inst., also a single seizure on the 13th and 14th, respectively. Each attack was described by the patient as having been preceded by a well-marked aura, likened by him to the sensation of having received a blow on the head at side of depression, with sharp, lan-

cinating pains in superior posterior cervical region. These prodromic symptoms were, in each instance, followed by vertigo and loss of consciousness; also pain in the region of temples on recovering.

The seizures were not usually of the complete *grand mal* type, but rather that of minor epilepsy or *petit mal.*

On December 16th the operation of trephining was performed; previous to which, however, the head was shaved, and a photograph thereof, showing site of depression, taken.

Ether having been administered, a crucial incision was made over the depression and flaps reflected back. A conical trephine was then applied just above the point of greatest depression. With little difficulty the disc of bone was removed. At the point trephined the skull was found to be nearly three eighths of an inch in thickness, quite vascular, and much roughened on the under surface by rudimentary osteophytes, to which the dura mater appeared to be slightly adherent. Proceeding to remove the remaining portion of depressed bone by gnawing with the rougeur, that instrument was broken before much had been accomplished. Twenty minutes elapsed before another could be procured; and in the interim a second opening, the site of which was contiguous to and overlapped the first to nearly a third of its extent, was made with the trephine. Then, with the new rougeur, the bone was gnawed away until all of the roughened portion of the inner table was removed, leaving an opening in the skull which measured approximately one and a quarter inches in length by one in breadth. In removing the second button of bone the dura mater was slightly wounded by the trephine. The exposed portion of the dura also presented a congested and thickened appearance. After having carefully trimmed the sharp edges of bone, the scalp wound was closed by horse-hair sutures, dressed with iodoform and lint, and covered with absorbent cotton and a skull-cap bandage applied.

Owing to breaking of the rougeur the operation was somewhat tedious, the patient's pulse flagging toward the latter part thereof, which necessitated the hypodermatic injection of whiskey.

Dec. 17, 1885.—Patient rallied well and quickly from the operation, but suffered from nausea all night and got little sleep in consequence. This morning feels cheerful. Temp., 99.4°; pulse, 100; respiration, 20. At 7 and 10 A.M., respectively, morphiæ sulph., gr. $\frac{1}{4}$, was administered; also calomel, gr. $\frac{1}{2}$; the latter to relieve nausea. Evening temp., 99°; pulse, 84; resp., 20. Patient is rational and cheerful, and says he feels much better.

Dec. 18, 1885.—Patient was given chloral, gr. xx., last night, and slept well for about four hours. This morning his temp. was 99°; pulse, 88; resp., 20. Evening temp., 99.4°; pulse, 100; resp., 24. Complained of some pain at seat of wound. Was given chloral, gr. xxx., at bedtime.

Dec. 19, 1885.—Temp., 99°; pulse, 88; resp., 20. The dressings were removed to-day; incisions found to have healed by first intention. No unfavorable symptoms have yet developed. He has been taking quiniæ sulph., gr. ii., in half an ounce of whiskey t. i. d. Appetite improving.

Dec. 23, 1885.—The stitches were removed to-day, the wound having thoroughly healed.

Dec. 28, 1885.—Patient sat up for a short time to-day. No pain in the head. Is doing well in every respect.

June 16, 1886.—Patient's improvement has been steady and uninterrupted. He has been under close observation day and night since the operation, and up to date has had no return of the epileptic attacks. The marked irritability and want of control from which he was suffering when admitted to the asylum have subsided, and his bodily and mental conditions are much improved.[1]

CASE 2.—J. C., male; aged twenty-four, convict. Driver by occupation; native of New York. Admitted July 11, 1884, as a case of epilepsy. Of intemperate habits. Physical condition fair. Has a depression on skull, the result of falling down a flight of stairs when about six years old.

July 11, 1884.—Patient is quiet. Pupils dilated. Claims he has no delusions, but that he has been insane several times. Confined in Ward's Island Asylum three times.

[1] J. M. was discharged recovered and returned to the prison, June 24, 1886, where he has remained free from epilepsy to the present time, Aug. 1, 1886. —C. F. M.

First and second times remained two years: third time over three years. Was discharged from that institution in the spring of 1884. Depression in the skull, referred to above, is behind and above the right ear, two and one half inches superior to external occipital protuberance, and one inch to the right of the interparietal commissure, covering the posterior segment of the superior parietal lobule lying just above the deep interparietal fissure. Patient exhibited no motor disturbance or impairment of the general or special sensory functions.

Patient's history previous to admission to asylum is that of a chronic epileptic, and is as follows: Fits began at sixteen years of age, and have continually recurred up to the date of operation. Longest interval of quiescence was while under treatment at the asylum on Ward's Island, when a period of fifteen months passed without recurrence of fits. Subsequently they reappeared at frequent intervals, the last occurring just previous to date of this entry. The seizures usually occur at night, and are marked by convulsions and complete loss of consciousness, and followed by a state of active mental excitement, during which patient, as a rule, becomes noisily maniacal and pugilistic.

July 14, 1885.—Patient has been quiet and well-behaved since last entry; had a fit last night while in bed, but it was not followed by the usual mental disturbance.

From this time until Nov. 20, 1885, he suffered from frequent nocturnal seizures, which left him each time in a disturbed mental condition, though not violent. He gradually became very whimsical, irritable, unreasoning, and discontented. This condition persevered until February 18, 1885, when he began to complain of pain in the right occipital region, most intense over the above-described depression. From this up to the day of the operation the frequency of attacks increased, always nocturnal, but evidenced by a bitten tongue, and by his having been frequently found by the night watchman out of bed on the floor in a dazed, semi-conscious condition, and incapable of telling why he was out of bed. On the 25th of August, 1885, the operation of trephining was performed. It was

similar in character to that in the preceding case, except that in this there was but one button of bone removed. The inner surface of the bone was smooth and non-adherent and the dura mater was not wounded. Nothing occurred during the operation worthy of special comment.

At 5 P.M., patient, who had rallied well, was given ℥viii. of Magendie's solution of morphia, hypodermatically. At 9 P.M., morphia sulphate, gr. $\frac{1}{4}$, was administered per os. A little milk and lime-water was given during the evening.

August 26, 1886.—Temperature, 98.5°; pulse, 65 ; respiration, 16. Appears in good condition.

Aug. 27, 1885.—Complained of headache during the night. Temp., 97.5°; pulse, 56; respiration, 16.

Aug. 29th.—Temperature has not been above 98.5°, in axilla, since the operation. Dressings removed. Primary union of wound has taken place; not a drop of pus is to be found. Wound thoroughly cleansed and iodoform dressings reapplied. A little puffiness of scalp in vicinity of wound was observable, but there were no evidences of suppuration.

Aug. 31, 1886.—All stitches removed, and head dressed with carbolated cosmoline and lint. Puffiness, remarked at last dressing, has entirely disappeared. Sat up in bed for a little while.

Sept. 15, 1885.—Has been about the ward the past week. Is out in the yard to-day. Has complained occasionally of slight headache.

Nov. 18, 1885,—Since the operation, has exhibited very little of his former irritability. Complains of headache occasionally, but not nearly so frequently.

Feb. 3, 1886.—Much less irritable, less frequent headaches, no recurrence of fits.

June 16, 1886.—Up to date patient has remained free from epileptic seizures, nocturnal or diurnal, while at the same time there has been a considerable degree of improvement in his mental tone. He now controls himself fairly well, and appears to be free from delusions, but exhibits a certain amount of weakness of mind, the result of long-con-

tinued epilepsy, and from which a complete recovery is not to be expected, even though there should be no return of fits.[1]

Applying the rules of cranio-cerebral topography to these cases, it will be seen that the lesion, in each instance, was located over a region of the brain to which no definite function has as yet been assigned, namely: the parietal lobules. Hence the absence of motor or sensory disturbance such as might have been expected from a similar lesion located in those areas of the brain which are now generally believed to govern motion and general sensation—that is, in the central region surrounding the Rolandic fissure.

While the existence of organic lesion of the cerebral cortex in either of the foregoing cases is extremely doubtful, it is, nevertheless, a well-established fact that those portions of the brain lying outside of the so-called motor zone may suffer extensive destructive lesion without giving rise to paralysis or other special symptoms of organic cerebral disease, though lesions of these unexcitable or non-motor regions, limited to or involving the dura mater, not infrequently give rise to localized headache, vertigo, convulsions, etc. Irritation of the nerves of the dura mater interferes with the blood supply by causing spasm of the dural vessels, and this in turn excites reflex convulsions of a general character, whereas convulsive manifestations arising from irritative lesions of the cerebral cortex are, generally speaking, definitely localized. Epileptic attacks may also result from a lesion of any part of the skull, whether within or without the motor centres. In fact, epilepsy has been known to arise from the reflex irritation following a fracture of the outer table with no direct pressure on the subjacent parts. Mr. James F. West reported to the Royal Medical and Chirurgical Society, in 1879, a case of this kind, of seven years' standing, which he cured by trephining.

Surgical history tells us that formerly it was customary to trephine in cases of epilepsy that resisted medical treatment, but the practice gradually fell into disuse, probably

[1] August 1, 1886.—Patient is still under observation. He has had no fit to date.—C. F. M.

on account of the numerous failures resulting from want of discrimination in the selection of cases upon which to operate, as well as the gravity of such operations before the dawn of antiseptic surgery. In 1852 Prof. Stephen Smith, of New York, in an article on the Surgical Treatment of Epilepsy, collected and analyzed 35 cases of trephining, giving the results of the operation and ably advocating its adoption in cases dependent on depressed fracture of the skull. Prof. John B. Roberts, in his recent valuable essay,[1] refers to 92 American operations collated by Stephen Smith, Billings, and Echeverria, of which 63 were cured, 13 improved, 2 unimproved, and 14 died. This author also cites 130 cases collected by Walsham, of which 75 were cured by the operation, 18 improved, 7 unimproved or worse, and 30 died; also "30 cases of epilepsy from old injuries of the head have been operated upon by Briggs,[2] of these 25 were cured, 3 ameliorated, 1 not changed, and 1 died."

That the cessation of the fits in the two cases here reported was directly due to the operation, will not, I believe, be questioned. In this connection the question naturally arises, would the mental impairment resulting from the long-continued epilepsy have been prevented by an earlier resort to the trephine? The writer is of the opinion that it would.

In view of the high ratio of recoveries in the comparatively large number of cases here cited from reliable sources, together with numerous other successful cases which, for want of time, I have not attempted to bring to your notice, it is not surprising that both surgeons and neurologists of the present day, almost without exception, should not only sanction, but advocate, an operation which promises so much in the way of cure, and which, with proper antiseptic precautions, is attended with so little danger to the patient.

---

[1] "The Field and Limitation of the Operative Surgery of the Human Brain," 1885.

[2] Transactions of the Am. Surgical Association, 1884.

Periscope.

PATHOLOGY (INCLUDING PATHOLOGICAL ANATOMY) OF NERVOUS SYSTEM.

Recent Observations on Degeneration, and on Nerve Tracts in the Spinal Cord.—A Critical Account. By J. N. LANGLEY, M.A., F.R.S. *Brain*, April, 1886.

In view of the great importance of the facts collected and the conclusions reached by Langley, we propose to devote several pages to the consideration of his paper. It is a "critical digest" and does not well bear further condensation. For this reason we shall reprint the original (contrary to our usual custom) with but a few unimportant omissions. Langley's results are based upon articles by: 1, Langley and Sherrington; 2, Pitres; 3, Löwenthal; 4, Sherrington; 5, Homén.

The following may be taken as a rough summary of the chief points connected with the observation of secondary degeneration, although no doubt several of the statements are open to discussion. Secondary degeneration is best observed with the microscope four to five weeks after the injury. The degeneration can be made out near the lesion in a week or less. It spreads fairly rapidly—probably in less than twenty-four hours—along the whole course of the nerve fibres. It can be earliest seen by the alteration in the neural axis, a day or two later by the change in the myelin sheath also. In one to two weeks it can be observed by the eye in the cord hardened in potassium bichromate. In about three weeks it can be seen by the eye in the fresh cord. The neuroglia now begins to increase, and the nerve fibres to be absorbed, although probably none disappear for some weeks. In consequence of these changes, the degeneration is, from the sixth week onwards, more and more noted under the microscope by the change in the neuroglia, and less and less by the presence of altered nerve-fibres. In the lateral pyramidal tract altered nerve fibres are seen for three or four months. The length of time after the lesion during which the area of degeneration can be detected by the "scar," varies with the number of fibres which have degenerated; in the lateral pyramidal tract the scar can be seen for a

year, and probably for much longer. The degeneration can be seen in the fresh cord for four to five months ; during the last six weeks or so of this time, it changes from milky-white to grayish. Possibly in the cerebellar tract the change takes place earlier. In time (? six months) no change can be seen in the fresh cord. In the cord hardened in potassium bichromate, the area of degeneration becomes more distinct for four to six months ; later than this it becomes less and less distinct ; in the lateral pyramidal tract it may be seen for about a year. In its earlier stage, the area of degeneration may appear larger than it really is, in consequence of a spreading out of the connective tissue change ; in its later stage it becomes smaller, in consequence of a retraction (? partial disappearance) of the connective tissue.

Whilst all observers have paid considerable attention to the time after the lesion at which secondary degeneration makes its appearance, comparatively little attention has been paid to the time at which the degeneration of the nerve-fibres is complete. But this is a point of some importance. According to Löwenthal, the degenerating areas in the region of the lateral pyramidal tract, which result from sections of the cord in the cervical region, consist chiefly of altered fibres three months after the section. He finds, too, that the number of altered fibres is greater in twelve weeks than in six, and greater in six weeks than in three. Sherrington describes a few altered fibres as being present in the lateral pyramidal tract seven months after cortical lesion. But from the description of most observers, we should gather that some fibres disappear altogether in about a month. Homén, in three weeks, finds spaces from which fibres have disappeared ; but it is not clear whether the fibres had been absorbed from these spaces, or simply dropped out of the section. If fibres do disappear so early, and if Löwenthal's statement regarding the increase in the number of altered fibres for two to three months after the lesion is correct, it follows that some fibres have disappeared, whilst others have scarcely begun to degenerate. This seems extremely unlikely, if all the fibres are undergoing true secondary degeneration.

It seemed possible that there might be some differences between descending and ascending degeneration in these respects ; but I cannot find that any one who deals with both degenerations points out any difference. The only clear statement as to the time of disappearance of nerve-fibres in ascending degeneration which I have been able to find is given by Singer. According to him, in *young* dogs after section of the cord in the dorsal region, the nerve-fibres of the cerebellar tract and of the medial part of the posterior column completely disappear in four to five weeks. In the adult dog, the process is slower ; unfortunately he does not say how much slower. There seems to me, however, to be *primâ facie* ground for thinking that, at any rate in the pyramidal tract, some fibres do not begin to degenerate until several weeks after section of the cord.

Turning now to the nerve-tracts in the cord, we may first consider :

The Pyramidal Tracts.—Flechsig mentions that in the lateral pyramidal tracts there are a certain number of fibres which do not come from the opposite pyramid. But in addition to these fibres, it is probable that the lateral pyramidal tract, as described by Flechsig, contains many other fibres which do not belong to this tract.

Bouchard, in 1866, noticed that in man, the descending degeneration in the lateral column was considerably greater after injury to the medulla than after injury higher up in the brain.

Bouchard describes the area of degeneration which appears in the cord in the posterior part of the lateral column after injury to the medulla, as extending to the surface. Since the cerebellar tract occupies the periphery of this part of the cord, and does not undergo descending degeneration, Bouchard's statement regarding the peripheral strip probably rests on a misinterpretation of the appearance. According to Schiefferdecker, the sclerosis may extend beyond the area of the degenerated fibres ; to this the sclerosis seen by Bouchard at the periphery of the cord was probably due.

The fibres which degenerated after injury to the medulla, but not after injury to the brain, were situated on the lateral border of the pyramidal tract. These fibres he called "fibres commissurales antérieures longues." This observation, has passed with little or no notice.

A somewhat similar difference, however, is described by Löwenthal, as occurring in the dog, after section of the cord in the cervical region and after removal of the motor area of the cortex. According to Löwenthal, the long commissural fibres lie between the cerebellar tract and the lateral pyramidal tract ; they are chiefly large fibres ; whereas, as we know, the pyramidal tract fibres, like those of the pyramid, are chiefly moderate-sized and small. Löwenthal points out also that, after section of the cord in the cervical region, the degenerated area in the lateral column has a fairly sharp boundary on its lateral edge. In degeneration after removal of the cortex of the brain, this is certainly not the case, the degeneration becomes quite diffuse as it approaches the cerebellar tract.

Sherrington also has come to the conclusion that, at any rate in the cervical region of the cord, the real pyramidal tract occupies a part only of the pyramidal tract as figured by Flechsig. This conclusion is, however, based upon a comparison of the area of degeneration which he obtains in the dog, with the area of non-medullated fibres seen in the developing cord of the ape, and with the area of the pyramidal tract as given by Flechsig in the cord of man. The basis is somewhat uncertain. Proceeding, however, in this way, he concludes that the ventral and the latero-dorsal parts of Flechsig's pyramidal tract do not degenerate from cortical lesions, and hence are not continuations of the pyramid fibres.

On the whole, it seems probable that the pyramidal tract is much smaller than that given by Flechsig, that it does not form a compact bundle of fibres, but on its anterior (ventral), lateral, and posterior (dorsal) borders it becomes diffuse, its fibres being mixed with other fibres, many of which, especially on its lateral border, degenerate on section of the spinal cord. In its inner (mesial) side its fibres, as described by Flechsig become mixed with the small fibres of the "boundary layer." It will be noticed that, as far as Löwenthal's experiments extend, the "anterior long commissural fibres" might be "commissural" fibres of the cord. From Bouchard, however, we should conclude that they run from the medulla to the various segments of the cord, and hence we might call it the descending medullary tract.

Bilateral Degeneration of Lateral Pyramidal Tract in Consequence of Unilateral Cortical Injury.—It has been known for some time, that in man an injury confined to one side of the cord, might cause a degeneration in the lateral pyramidal tract of the opposite as well as in that of the same side of the cord. Charcot suggested as an explanation of this, that part of the lateral pyramidal tract crossed in the anterior commissure, especially in the upper dorsal region, to the lateral tract of the opposite side. Thus the fibres would first cross in the pyramidal decussation, and then again in the anterior commissure.

A case of bilateral degeneration in the cord of the dog, as the result of unilateral cortical injury, was described in 1880 by François-Franck and Pitres.

Moeli, in 1883, found after unilateral injury to the mid-brain in dogs, that besides the degeneration of the pyramidal tract of the opposite side there was a degeneration of a few scattered nerve fibres in the pyramidal tract of the same side.

An instance in the ape has also been described by Schäfer (1883); the examination was made seven months after the cortical injury. In the cervical region of the cord there was sclerosis in both lateral pyramidal tracts, the area of the two sides being about equal; but the sclerosis was less in intensity on the same side as the lesion, and on this side it could not be traced beyond the cervical region.

The bilateral degeneration of the pyramidal tract has received a good deal of attention from recent observers. In no case apparently does unilateral brain-injury cause degeneration of the opposite pyramid, so that whilst one pyramid only degenerates, there is degeneration in the region of both the lateral pyramidal tracts in the cord. We will, for lack of a better term, call the degeneration which occurs on the side of the cord opposite to the sound pyramid the *re-crossed degeneration.*

Pitres examined the spinal cord in forty cases of unilateral brain-injury in man. In ten of these there was on both sides sclerosis of the lateral pyramidal tracts. In some cases this could only be made out on microscopical examination. In six out of the ten cases the sclerosis was symmetrical and equal on the two

sides, and on both sides it occupied a larger area, chiefly in the ventral direction, than that occupied when sclerosis occurred on one side only. In the remaining four cases the re-crossed degeneration was of less intensity than that of the opposite side. According to Pitres, the re-crossed degeneration stretches throughout the cord, reaching the surface of the cord in the lumbar region. No relation could be made out between the presence or absence of a re-crossed degeneration, and the presence or absence of a degeneration in the medial part of the anterior column (the direct pyramidal tract, Türck's column). In six cases the direct pyramidal tracts showed some sclerosis; this, whilst varying in amount, was in all cases slight, and in one case only could it be traced in the lower dorsal and in the lumbar region. In three cases there was sclerosis in *both* direct pyramidal tracts.

Pitres considers the re-crossed degeneration to be due to the degeneration of fibres coming from the pyramid of the same side; *i. e.*, he extends to these fibres the view advanced by Bouchard and by Flechsig with regard to the direct pyramidal tract.

There are on this view four ways by which the fibres of each pyramid can proceed to their ending in the cord. They proceed along (1) the lateral pyramidal tracts of the opposite side; (2) the lateral pyramidal tract of the same side; (3) the direct pyramidal tract of the same side; (4) the direct pyramidal tract of the opposite side. Most commonly all the fibres take the first course; not infrequently they run both in the first and second courses; less frequently some fibres proceed by the third, the greater number running either in the first or in the first and second course; rarely some fibres may in addition take the fourth course. If this description is the true one, the term "re-crossed" degeneration, which I have used above, is obviously inapplicable; but there are reasons which will be mentioned later, against accepting Pitres' views. Charcot's suggestion, that the fibres of one lateral tract cross to the other by the anterior commissure, has, as Pitres points out, no satisfactory basis, for no degenerated fibres have as yet been described as occurring in the anterior commissure.

Bilateral degeneration in dogs after unilateral cortical injury has been described both by Löwenthal and by Sherrington.

Löwenthal mentions it as occurring in two out of about twenty cases. In each of the two the degeneration (sclerosis) was diffuse, and could not be traced farther than the mid-dorsal region. Sherrington found bilateral degeneration in each of twelve cases observed three to twelve months after the lesion, but found unilateral degeneration only, in two cases observed seven and fourteen days respectively after the lesion. The re-crossed degeneration appeared to him to be always somewhat less advanced than the degeneration on the opposite side of the cord. Hence he concludes that the re-crossed degeneration begins three or more weeks after the degeneration in the opposite lateral pyramidal tract. The conclusion is somewhat hasty; for in the two

cases in which re-crossed degeneration was absent, it might not have occurred had the animal been kept alive; and further, Sherrington does not appear to be quite certain that the nerve-fibres in the re-crossed degeneration were really in an earlier stage of degeneration than those on the opposite side of the cord. It may be noted, however, that if his conclusion is right, it disposes at once of the views both of Charcot and of Pitres as to the origin of the fibres in the re-crossed degeneration; for if the fibres which are affected in the re-crossed degeneration were directly continuous with the pyramid fibres, they would certainly not degenerate weeks later than the rest of the pyramid fibres in the lateral pyramidal tract.

Sherrington also finds that the re-crossed degeneration does not usually diminish regularly in its course down the cord, but is greater in the upper lumbar and lower dorsal region than in the mid-dorsal region, and is commonly greater between the third to seventh cervical nerve-roots—where, indeed, it is earliest seen—than between the first to third cervical nerve-roots. If this is the case the bilateral degeneration cannot be due to a crossed and an uncrossed portion of the pyramid, and the re-crossed degeneration cannot be due to pyramidal fibres. It is on this account that I have used the term "re-crossed degeneration."

If the pyramids send no fibres to the lateral column of their own side, some other explanation of bilateral degeneration must be sought for. Let us consider in what other way it could be brought about. Since in the dog there is no evidence of a direct pyramidal tract, we have for the dog the simplest scheme, that all the pyramid fibres running to the cord decussate in the pyramidal decussation, and run down the cord in the lateral pyramidal tract of the opposite side. Since these fibres end in the gray-substance of the cord, it is quite possible that their degeneration should cause more or less pronounced degeneration in some parts of the gray substance. And, in fact, one or two cases have been described of atrophy of cells in the anterior cornu in consequence of degeneration of the lateral pyramidal tract, and of atrophy of Clarke's column in consequence of degeneration of the posterior roots. And there is some evidence that in man the gray substance in lateral sclerosis is not infrequently affected in a less degree; for on the non-hemiplegic side it is not uncommon to find increase of reflexes, or general weakness without any discernible change in the columns of that side of the cord. These cases, although admitting of other explanation (see below), are most satisfactorily explained by supposing that the gray substance of the cord has been affected by the degeneration of the pyramid fibres. If the change proceeds further than this, viz., to degeneration, it is to be expected that some fibres proceeding from the gray substance to the columns of the cord, *i. e.*, connecting fibres, will degenerate. Such fibres, we may fairly conclude—bearing in mind the assumption with which we started—are the fibres of the re-crossed degeneration.

But if the disappearance of the pyramid fibres on one side can affect the gray substance sufficiently to lead to the degeneration of connecting fibres on the opposite side, we should certainly expect that it would cause some degeneration of connecting fibres on its own side. There seems to me to be some evidence that this does take place. We have seen above that there is some reason to believe that within the area of the lateral pyramidal tract some fibres show signs of degeneration very much earlier than others. This is not easy to explain, if all the fibres are the direct continuation of the fibres of the pyramid; whereas if the fibres which are first affected are pyramid fibres, and the later ones connecting fibres of the cord, the protracted period of degeneration presents no difficulty.

Sherrington and myself have used the term "tertiary" to denote that degeneration which is brought about by a change in gray substance as a consequence of nerve-fibres in it degenerating. We might then state the conclusions of the foregoing argument in the following way: injury to one lateral column of the cord (or unilateral injury to the motor area of the brain) causes secondary degeneration in the crossed pyramidal tract, and not infrequently tertiary degeneration of connecting fibres of the cord within the area of both crossed pyramidal tracts.

This view affords a basis for understanding how the re-crossed degeneration can, as described by Sherrington, be greater in the lower dorsal than in the mid-dorsal region; why it occurs later than the 'normal' degeneration; why on both sides of the cord fibres in an early stage of degeneration are found long after the injury; and why in bilateral degeneration the area is often, as described by Pitres, greater than in unilateral degeneration. It does not satisfactorily explain how the sclerosis can be—as described by Pitres in certain cases—equal in intensity on the two sides of the cord.

But with all this it must not be forgotten that the statements on which the view rests require confirmation; and, further, that so far no relation has been observed between bilateral degeneration and atrophy of gray substance. I have stated the view with some fulness, on account of its importance if true; but I need hardly say that I regard the questions here raised as at present quite open.

One other question of importance arises out of bilateral degeneration. This is whether each pyramid is connected with the gray substance of both sides of the spinal cord.

Flechsig, in his "Plan des menschlichen Gehirns," figures the "direct" pyramidal tract as ending in the gray substance of the same side of the cord; those who accept Pitres' view of the meaning of bilateral degeneration might consider that here, too, each set of fibres ends in the gray substance of its own side.

That is to say, the extent of the uncrossed tracts of the pyramid would be a measure of the number of its fibres which end in

the cord on the same side as it. The variability of the course of the pyramid fibres in the cord would be associated with a variability in their termination.

It is clear that there is no necessity that this should be the case. All the fibres of one pyramid might run in one lateral column, and yet end in the gray substance of both sides of the cord ; and, on the other hand, the fibres of one pyramid might run in both lateral and in both anterior columns, and yet end in the gray substance of one side only of the cord.

And that this is the case seems to me to be in the highest degree improbable. It requires a great deal of evidence even to make it likely that the individuals of one species vary so much, that in one the cortex of the brain is connected with one side only of the cord, and in another is connected with both sides ; that, in fact, in different individuals the end stations of homologous fibres are indifferently on the right or on the left side of the body.

What, then, is the proof of this variation ? It is partly that the pyramid fibres are said to vary in their course in the cord ; this, even if we accept the fact, is, as I have said, no proof at all. It is partly that in some cases in which a direct pyramidal tract has been found, there has been weakness of body or increase of reflexes on the non-hemiplegic side. But this proves nothing either, for the affection of the non-hemiplegic side of the body often occurs without any abnormality being perceptible on that side of the cord.

The proof of the variation is then of the most tenuous character, and bearing in mind the objections to it on general grounds, we may safely regard it as purely hypothetical.

And we may go further, and say that in the present state of our knowledge there is little justification for believing that the pyramid fibres end on both sides of the cord. For in certain cases of hemiplegia from cortical lesion, no clinical symptoms can be observed on the non-hemiplegic side, so that we must conclude that the pyramid fibres end on one side only of the cord. When clinical symptoms are observed on the non-hemiplegic side, they are in all probability due to an alteration of the gray substance, brought about by the degeneration of the pyramidal fibres.

The Posterior Columns.—Our knowledge about the direct continuation of nerve-fibres of the posterior roots of the spinal nerves into the medulla, we owe chiefly to the observation of Bouchard (1866), of Lange (1872), of Schultze (1883), on man, and to the experiments of Singer (1881) on dogs. Schiefferdecker (1876) was the first to give a detailed description of ascending degeneration after section of the cord in dogs. From these and other observations we gather the following account of the course of the nerve-fibres of the posterior root. Each posterior root on entering the cord forms the most lateral portion of the posterior column, that next the gray substance ; a considerable number of its fibres at once enter the gray substance, the rest continue

upwards, becoming, as they go, more mesially situated, in consequence of the entrance of other nerve-roots, and giving off, in their course, fibres of the gray substance of the cord. But not all the nerve-fibres end in the cord ; some run on to end in the clava ; the course of these fibres has been worked out for the sacral and lumbar nerves only. From what has been said, it is evident that the several posterior nerve-roots form ascending laminæ of fibres, so that in the upper lumbar region the nerve-root which has last entered forms a lamina in contact with the posterior cornu, and each nerve-root below this forms a lamina immediately mesial to that of the one above it, the meso-dorsal angle of the posterior column being occupied by fibres from the last sacral nerve. As these laminæ ascend in the cord, they give off at intervals, and chiefly in the dorsal region, a considerable number of their fibres to the gray substance of the cord. In the cervical region of the cord all the fibres which have reached it from the lumbar and sacral nerve-roots are found in Goll's column occupying the same relative position as they occupied lower down. These fibres, without much diminution in number, run on to the medulla, and end in the cells of the clava ; they form thus an ascending medullary tract.

Hence then, broadly speaking, the nearer the mesial part of the posterior column is to the medulla, the more completely does it become an ascending medullary tract, and the less does it consist of cord fibres simply ascending from the posterior roots. It seems to me probable that it is on this account that in the developing cord—as described by Flechsig—there is in the dorsal cord no area of fibres in which the medulla is developed late, to correspond to Goll's column in the cervical cord. At the same time, one would expect that further observation on the time of development of the medulla of the nerve-fibres would enable the ascending medullary to be distinguished from the ascending spinal fibres in all regions of the cord Schiefferdecker and Singer with some reservations, and Schultze unreservedly, consider Goll's column to be made up of fibres which come from the sacral, the lumbar, and perhaps from the lower dorsal region. The corresponding fibres of the upper part of the cord they apparently consider run in Burdach's column. Since there are ascending medullary fibres in the lower nerve-roots, we may take it as certain that there are similar fibres and of similar functions in the upper nerve-roots also. On the above view, then, Goll's column contains medullary fibres from the lower part of the body, and Burdach's column contains medullary and spinal fibres from the upper part of the body. The medullary fibres of Goll's column end in the nucleus of the funiculus gracilis ; the fibres of similar function in Burdach's column must then end in the cells of similar function to those of the nucleus of the funiculus gracilis ; thus they either leave the funiculus cuneatus for the funiculus gracilis, and end in its nucleus, or they end in the nucleus of the funiculus cuneatus ; in which case the latter—in part, at any rate—has the

same function as the nucleus of the funiculus gracilis, the one being the centre for the medullary fibres of the upper part of the body, the other for the medullary fibres of the lower part of the body.

Schultze's opinion is based upon an observation in man in which the middle of the cervical enlargement has been completely destroyed; the degeneration of the posterior columns, complete just above the lesion, was in no part of the cord up to the medulla confined in Goll's column, but occupied also a considerable part of Burdach's column. A similar account is given by Löwenthal of the effect of section of the posterior columns in the cervical region in the dog. These observations are not very conclusive as to the point in question; and it may be noted that no one has obtained complete degeneration of Goll's column in the upper cervical region by section of the cord in the lower dorsal region, and further, that Goll's column certainly increases in its way up the cord. We must be content to wait until observations have been made upon the effects of section of the posterior roots of the spinal nerves in the cervical region.

Since the fibres of Goll's column consist of small nerve-fibres, and since there is ground for believing that medullated nerve-fibres do not alter in size in their course in the cord, we may conclude that the medullary fibres of the posterior column consist of a greater or less part of the small fibres of the spinal nerves, and that the spinal fibres of the posterior columns (except perhaps in the upper cervical region) include all the large fibres of the spinal nerves.

The question of the presence of commissural fibres in the posterior columns we may omit, since the recent work on the spinal cord does not deal with it.

The Cerebellar Tract.—Singer found, on section of the cord in the dog between the dorsal and lumbar regions, that in the mid-dorsal region, besides the degeneration of the tract ordinarily recognized as the cerebellar tract, there were degenerated fibres scattered over the whole of the antero-lateral column, these fibres being fewest at the periphery of the anterior column. In the cervical region, the degenerated fibres were much less in number; they were present at the periphery of the lateral column only, but stretched from the anterior to the posterior nerve-roots, although they were much more diffuse ventrally of the ligamentum denticulatum than dorsally of it. These fibres in the cervical region were considered by Singer as belonging to the cerebellar tract.

According to Flechsig, the cerebellar tract in man stretches as a compact bundle of fibres about as far as the ligamentum denticulatum; at its ventral end it becomes diffuse, and scattered cerebellar fibres are found for a short distance in the antero-lateral column. Making the fullest allowance for the scattered fibres mentioned by Flechsig, there still remains a considerable difference between his description for man and that of Singer for the dog. The more recent observations on ascending degenera-

tion, both in man and in the dog, have tended to confirm, in the main, Singer's account. That in man the ascending degeneration stretches more ventrally than is described by Flechsig, has been pointed out by various observers, and especially by Schultze. Löwenthal has observed the asending degeneration in the dog after section of the cord in the cervical region ; according to him, the cerebellar tract is fairly compact up to the denticular ligament ; beyond this it becomes much more diffuse, but stretches along the periphery of the cord as far as the point of exit of the anterior nerve-roots. Near the denticular ligament, it extends some little distance into the lateral column.

The fact, however, that these ventrally placed fibres degenerate in an ascending direction is alone sufficient to show that they belong to the cerebellar tract. To show this the fibres must be traced up to the cerebellum. According to Löwenthal, the ventral fibres do not run in the restiform body, as do the dorsal fibres, but run in the lateral region of the pons—probably as the "aberrirende Seitenstrangbündel" of v. Monakow,—twist round the superior peduncle of the cerebellum, and thence run to the cerebellum.

Löwenthal, like all previous observers, finds that the degeneration of the cerebellar tract diminishes in its way up the cord ; that is, that a considerable part of the "cerebellar" tract consists of connecting fibres of the cord. Flechsig, on account of the steady increase in size of the cerebellar tract upwards, considered that it received fibres from the cord, but gave none to it ; but the fact does not warrant the conclusion, for the tract must increase in size as long as it receives more fibres than it gives off. It may be mentioned that the most of the scattered degenerated fibres—probably connecting fibres—described by Singer in the dorsal region, were not observed by Löwenthal in the cervical.

Fibres proper to the Cord which undergo Descending Degeneration. —These fibres are called by Singer, who restricts the meaning of the term, as used by Bouchard, "commissural" fibres. The term is not, I think, a very happy one. In anatomy the word "commissure" has come to mean the connecting portion between bilaterally symmetrical parts ; in this sense fibres which connect the cervical with the lumbar region of the cord do not form a commissure any more than fibres which connect the spinal cord with the cerebellum or with the cerebrum. If "commissure" is used in its wider meaning, then all these fibres are equally commissural, the fibres of the pyramidal tract and of the cerebellar tract, as much as those connecting the different segments of the spinal cord. But it would, I think, be better to restrict the term "commissural." The fibres which degenerate can conveniently be designated according to their connection and their mode of degeneration, whether above or below the point of injury. Thus we have ascending and descending spinal, medullo-spinal, ascending cerebello-spinal, descending cerebro-spinal fibres in the cord. But there are many nerve-fibres in the antero-lateral columns

which do not degenerate either above or below the place of section. These might conveniently be called commissural fibres; and the designation would have some justification. The fibres which degenerate have a trophic centre at one end only, and it is very likely that this is due to the nerve-fibres in life conveying impulses in one direction only, for there seems to me no reason to suppose that the fibres of the posterior roots or the fibres of the pyramidal tracts ever receive impulses from the spinal cord. On the other hand, the fibres which do not degenerate on section must have a trophic centre at both ends, and in view of what has just been said, it is probable that this is due to their receiving impulses at both ends. If this is so, these fibres are more properly commissural than those which degenerate.

The proper cord-fibres which degenerate in an ascending direction have already been mentioned in connection with the cerebellar tract, since the two sets have not as yet been with certainty distinguished.

Of the proper cord-fibres which degenerate in a descending direction, those which lie in, and in the neighborhood of, the lateral pyramidal tract have also been mentioned. The remainder are scattered throughout the ventral part of the antero-lateral column. In the dog the longer fibres form a moderately compact zone at the periphery of the anterior column. The observations of Schiefferdecker and of Singer, from whom chiefly the above statements are drawn, were made after section of the cord in the lower dorsal region. In Löwenthal's experiments the spinal cord was cut through more or less completely in the cervical region; he finds throughout the lower cervical, the dorsal, and the upper lumbar region, as far as the lumbar swelling, a degenerated tract of fibres round the whole periphery of the anterior column like that previously described by Schiefferdecker and by Singer, after section of the dorsal cord. But, unlike previous observers, he finds that this tract, at the junction of the anterior with the lateral column, continues dorsally for some little distance into the lateral column, although leaving the periphery and becoming more diffuse. Since these fibres diminish in number in passing down the cord, they must run to different cord segments. Since, further, the zone in the lumbar region is less compact than that previously described after section of the cord in the dorsal region, we may conclude that it receives from, as well as gives off, fibres to all parts of the cord. Some of them probably arise in the medulla, *i. e.*, are descending medullo-spinal fibres.

For the fibres of short course which undergo descending degeneration, Löwenthal finds much the same distribution in the cervical region as that found by Schiefferdecker and by Singer in the lumbar region—viz., in the non-peripheral parts of the anterior column, and in "anterior mixed zone" of the lateral column.

Tertiary Degeneration.—Sherrington and myself found that after injury to the brain in the motor area, there was, besides the secondary degeneration in the pyramidal tracts, a degeneration of

a different nature, more resembling chronic myelitis. The altered fibres were chiefly in the anterior columns, varying somewhat in position in different parts of the cord. Since this was apparently the result of an alteration in the gray substance of the cord in consequence of the secondary degeneration of the pyramidal tracts, we called it a tertiary degeneration, at the same time pointing out the possibility of its having been produced by the hardening agent. Sherrington has since found two other cases in which, after a cortical lesion, a similar change had taken place in the spinal cord. As he points out, the entire absence of altered fibres from the cerebellar tract is strong reason for regarding the "myelitic" appearance of certain fibres in the rest of the cord as being due to degeneration, and not to the hardening agent.

The Parts of the Cortex of the Brain which are Connected with the Lateral Pyramidal Tracts.—It is well known that a lesion which affects a considerable part of the motor area of the cortex of the brain causes a degeneration in the lateral pyramidal tract of the cord, and that a considerable lesion in other parts of the cortex—such as that of the occipital and temporal lobes—causes no degeneration in the cord.

We know very little more than this, but there has been a great tendency to consider this knowledge as much more precise than it really is. This is so because it is commonly assumed: 1st. That the motor area of the cortex, as it has been determined by electrical stimulation, coincides with the "cord area," *i. e.*, with the area of the cortex, destruction of which causes degeneration in the cord. 2d. That each "centre" in the motor area is connected with its corresponding local centre in the spinal cord, and with that only; for instance, that the cortical area, stimulation of which causes movements of the fore-arm, is connected with the "arm centre" in the cervical region of the cord, and with no other part of the cord.

Ferrier and Yeo, in a very interesting paper on the physiological effects of extirpation of the cortex in the ape, touch somewhat incidentally on the question of secondary degeneration. In the cases in which the cord was examined, secondary degeneration was only found when the cortical lesion had taken place in the motor area (the convolutions bounding the fissure of Rolando). These observations are distinctly in favor of the view, that the "cord area" coincides with the motor area for the limbs and trunk, but the details given are not sufficiently full to prove that this is the case.

The more recent experiments in the dog are difficult to harmonize with the scheme given above.

Sherrington and myself, from a comparison of the area of greatest degeneration on the two sides of the cord consequent on destruction of the cortex of the brain to unequal extents and at different times, were inclined to believe that the anterior part of the motor area was connected with the dorsal part of the lateral pyramidal tract, and the posterior part of the motor area with the

anterior part of the lateral pyramidal tract; and further, that the destruction of the cortex behind the motor area as figured by Hitzig and by Ferrier would give rise to degeneration in the cord. The nature of our observations did not, however, allow of conclusions being drawn with certainty. It will be noticed that our tentative conclusions involved: (1) That fibres from each "centre" of the motor area run in a fairly compact bundle. (2) That the cord received nerve-fibres from parts of the cortex which are considered to be motor centres for certain of the muscles of the head. (3) That the "cord area" stretches farther posteriorly than the described motor area.

The posterior part of the cord area—as given above—lies in part, at any rate, in the region which, in Ferrier's observations, produced, on stimulation, movements of the eye. Ferrier considered it, however, to be a part of the "sensory" area of the cortex. This region is called the "Fühlsphäre" for the eye by Munk, who puts it in the same class with the cortex of the sigmoid gyrus, which he calls the "Fühlsphäre" for the limbs and neck.

Löwenthal has observed five cases in which the coronal and anterior ecto-sylvian convolutions—the anterior parts of the 3d and 2d convolutions—were largely removed; in two of these cases a part also of the median ecto-sylvian and of the anterior sylvian convolution were also removed. In each case, however, a small part of the postero-lateral edge of the sigmoid gyrus was affected; in one case this appears to have been a mere fraying of the edge of the sigmoid gyrus. In every instance there was a degeneration of the lateral pyramidal tract of the spinal cord, a degeneration which could be followed a variable distance in the dorsal region.

If the degeneration was due to the destruction of the coronal, ecto-sylvian, or sylvian convolution, it follows that the destruction of the motor centres for certain muscles of the head, causes degeneration of part of the lateral pyramidal tract of the cord. It is unlikely that the slight and superficial injury to the sigmoid gyrus should have caused so considerable a degeneration as occurred in these cases; but even by granting this, the difficulty is not removed, for the part of the sigmoid gyrus injured is the motor area for the fore-limb, and the destruction of this ought not, on the generally accepted view, to cause degeneration in the dorsal region of the cord.

It may be, however, that in each of these cases the lesion was carried too deep, and affected part of the corona radiata containing fibres coming from the cortical "centre" for the hind-limb. Löwenthal mentions this possibility, but does not say that there was any evidence for it in the sections of the brain.

Sherrington gives an observation which is more difficult to explain on the ordinary view of the connection of the motor centres with the cord. He finds that a lesion which does not reach farther forward than the posterior edge of the ecto-sylvian

fissure, produces degeneration throughout the cord, *i.e.*, a lesion which lies posteriorly to the motor area as given by Hitzig, Fritsch, and by Ferrier,—though including part of the "Fühlsphäre" of the eye of Munk—causes degeneration in the lateral pyramidal tract. In his case it is very difficult to see how fibres from the sigmoid gyrus could have been affected.

Further, Löwenthal finds that lesion of the anterior limb of the sigmoid gyrus—the posterior lateral border being left intact—leads to degeneration as far as the upper dorsal region. No one, I believe, has considered the anterior limb of the sigmoid gyrus to contain a motor area for any of the muscles of the trunk or of the hind leg.

From the destruction of certain parts of the cortex, Löwenthal has observed no degeneration in the cord. In a case in which the lesion was confined to the postero-dorsal part of the sub-orbital lobe degeneration was absent. Sherrington also found no degeneration from injury of the sub-orbital lobe. According to Munk, the sub-orbital lobe is the "Fühlsphäre" of the trunk.

The tentative conclusion mentioned above, that the fibres from different parts of the motor area run in different parts of the lateral pyramidal tract, has received no confirmation. Löwenthal examined the cord in seventeen cases of cortical lesion affecting the sigmoid gyrus in different degrees and in different regions. He mentions no difference in the cord degeneration, except a difference in intensity. Sherrington finds the position of the cord degeneration to be the same whatever the cortical lesion which has given rise to it. If this is so, it shows a very remarkable intermingling of the fibres of the pyramidal tract in their course from the cortex.

A limited intermingling of fibres is suggested by the observations of Hitzig, who found that movements of the muscles of the trunk could be obtained by stimulation of the motor area for the fore-limbs; and a greater intermingling is suggested by the recent observations of Paneth, made under Exner's direction. Paneth finds that there are small motor areas for muscles of the fore- and of the hind-limbs interspersed over a considerable area of the posterior limb of the sigmoid gyrus.

But both as regards this point, and as regards the connection of the part of the motor area with the lateral pyramidal tract, we must wait for further investigations. The experiments which have been mentioned above suggest conclusions which are not in accordance with those which we should on general grounds be inclined to accept; for this reason, then, we are justified in reserving a definite opinion until fuller and more satisfactory evidence is forthcoming.

Embolism of the Medulla. By GEO. B. SWASEY, M.D. *N. Y. Med. Rec.*, August, 1886.

Dr. Swasey reports an interesting case of what he considers to

be "Embolism of the Medulla." The main points in the history are as follows: J. D. Warter, æt. thirty-two, single, heavy drinker had chancre three years ago. Evening of April 9th, drank freely; next morning found that he had lost power of speech, unable to swallow, and had pain in region of sternum; was dizzy and staggered; ringing in his right ear. Pulse that same day, 108; temperature, 99.5° F. Some paresis of lower extremities, most marked upon right side. Could walk quite well with eyes closed, and could stand with heels together without reeling. (This in the afternoon.) Stood on one foot with eyes closed, but with much difficulty upon the right owing to its weakened condition. There was some paresis of thigh muscles, more marked on right side than on left. He was able to open his mouth over half the distance, but was unable to protrude his tongue; mucous membrane of palatine arch was anæsthetic. Facial nerve upon right side was paretic, most marked on its lower fibres of distribution; left facial less paretic than right. Respiration, 22, "slightly Cheyne-Stokes." Frequent gaping. Six days later: Temperature, 98½°; pulse, 94; respiration, 16. Paresis of both forearms, greater on right side. Plantar reflexes lessened; left cremaster lessened, right absent; patellar reflexes exaggerated; left ulnar and radial reflexes present, right absent; sensibility normal, bladder normal, bowels constipated. In the course of a month or two all symptoms disappear, with the exception of occasional difficulty of speech and some slight trouble in holding his water. Patellar reflexes exaggerated, left more so than right. Both radial reflexes normal[1]; others not tested. The author believes that an embolic obstruction occurred in one of the vertebrals, but he is not willing to decide whether upon the right or the left side. The writer makes out a fairly strong case in support of his diagnosis. We do not feel quite so certain that this is a case of medullar disease. The amount of paralysis that existed was slight, and neither respiration nor the pulse was at all seriously interfered with. The chief symptoms were those of aphasia and hypoglossal disease. We ask the writer's attention to a paper by Edinger reviewed in this JOURNAL —page 256, of this volume. B. S.

Symmetrical Vaso-Motor Paralysis of Both Hands. *Gazette des Hôpitaux*, p. 501, 1886.

The patient, male, æt. fifty-four, entered the hospital on the 1st of May. No hereditary history. Health generally good; never had any acute disease; no tendency to nervous affections; well nourished. Without any assignable cause, the patient, four years ago, at the beginning of the winter, noticed that his hands were swollen; this swelling, which was not painful, lasted a fortnight. Five months ago the same swelling, again without pain, returned. Upon examination both hands are found red, hot, and swollen. The skin is thick and hard and immovable. The dorsal surface

[1] Present or absent (?—Reporter).

is somewhat œdematous, and the veins are enlarged. The lower part of the arms is somewhat hot, otherwise the arms are normal. Appetite good, but a certain amount of polydipsia, with corresponding polyuria. No sugar in the urine, but slight traces of albumen. Heart-sounds normal. The axillary temperature is 37.4°, that of the forearm 34.6°, of the dorsum of the hand 35.5°, of the palm 35.6°, and between the index and middle finger 35.8°. The œdema began to disappear after a few days, and after three weeks the hands were again in their normal condition. The diagnosis in this case wavered between an œdema due to a disorder of the kidneys or heart, erythema, acrodynia, rheumatism, and the one which was made of vaso-motor paralysis.

Poisoning by Carbonic Oxide—Paralysis of the Extensor Muscles of the Left Forearm—Rapid Cure. SCHACHMANN. *La France Médicale*, July 1, 1886.

Various theses published within the last ten years have again called attention to the influence of carbonic oxide gas upon peripheral nerves. The subject was not new, but forgotten, for as early as 1843 Bourdon minutely described the accidents due to the action of this gas. The number of well-observed cases is not large. S. here describes the case of one of three persons who attempted suicide together by asphyxiation with this gas. Two of them succeeded in the attempt, and one survived.

B. C., female, æt. twenty. Family affairs led her to attempt suicide. The attempt was made on the 8th of May. All the apertures of the room were carefully closed, and the open stove with charcoal was lighted. She was discovered in an unconscious state and taken to the hospital.

May 10th.—Patient seems to have entirely recovered from the effects of the gas, but upon examination a complete extensor paralysis of the left forearm is found. Also about the middle of the arm a now fluctuating swelling is found. No electrical examination of the muscles.

May 11th—Condition of paralysis and swelling the same.

May 12th and 13th.—No change.

May 14th—Slight movements of extensor with fingers. Diminution in size of swelling.

May 15th and 16th.—Improvement.

May 17th.—Almost complete restoration of functions.

May 20th.—Complete cure.

(The author's theory that the paralysis was due to the action of the carbonic oxide gas is certainly not proven by the history given. The general impression left is that of a traumatic radial paralysis.)

G. W. JACOBY.

THERAPEUTICS OF THE NERVOUS SYSTEM.

A Case of Epilepsy, with Peripheral Aura, Cured by Blistering at the Seat of Aura. DIGNAT (*Progres méd.*, 1886, No. 18) after reviewing the literature of the subject, reports a case of this kind.

A shoemaker, twenty-one years old, had an epileptic attack, for the first time, Jan. 1, 1880. The attack began with a sensory and motor aura in the thumb of the left hand. He lost consciousness, the head was turned to the left, the eyes to the right; general convulsions and sleep followed. During the following seventeen months he had twenty-one such attacks in the daytime, and a very large number of lighter ones by night. In the intervals, with the exception of a decided increase of the knee jerks, there was no disturbance of sensation or motion, only in the bend of the left elbow there was a painful point in the course of the median nerve. He was admitted, in the summer of 1882, to the Pitres Hospital, at Bordeaux. A large blister was twice applied (July 8th and 15th) at the above tender spot. No other treatment was employed. From this time till 1884, the last time he was seen, he was entirely free from attacks.—*Neurol. Central.*, No. 12, 1886.

The Influence of Treatment on Chorea, with Special Relation to the Full Use of Arsenic and its Results.—Dr. W. B. CHEADLE combats the opinion that chorea is better influenced by treatment, and argues that chorea must be favorably influenced by treatment, inasmuch as there is a certain number of cases which tend to run on indefinitely when *not* brought under treatment, but that they do not so run on, or for an extended period, when they *are* brought under sytematic treatment. If treatment does not influence the course and duration of chorea, *some* of the cases, which come under treatment in the early stages, would run on indefinitely, and continue for months and months, just as untreated cases do. But C. claims they do not. Analyzing 167 cases, which have come under his own observation, he finds that some have run on 13, 14, 15, 17, 20, 34, 38, 47, 52 weeks, and longer, before coming under treatment, while the longest duration after coming under treatment was 12 weeks and 1 day, and that in a single instance; and yet a large number came under treatment at a very early stage. The following statistics are given as proof of the value of arsenic, which he thinks is the only drug capable of shortening the duration of the disease, as well as mitigating the symptoms.

Of 62 cases treated otherwise than by arsenic, the average time under treatment in hospital was 36.01 days; while of 105 cases, treated with arsenic, the average time of patients in hospital is 26.6 days. The average duration of the disease, in both classes of cases, before admission, was 63 days.

[One fallacy in such statistics seems, to the Reporter, to be in taking the period of stay in the hospital as a measure of duration

of disease, especially when the cases compared occurred in different years, as was the case with those reported. In some hospitals, at least, there has been a tendency of late years to discharge patients at the earliest possible moment, and not allow them to prolong their stay unnecessarily, as was formerly frequently the case. Furthermore, it is not easy always to say exactly when a chronic patient is cured, and one is not apt to be as exact in recording the precise time of cure in cases treated at an earlier period, and by methods in which one has little interest, as in cases treated by a method in which one is interested.]

The Physiological and Therapeutic Effects of Adonidin. Dr. H. A. HARE. *Therapeutic Gaz.*, April 15, 1886.

The adonis vernalis has for many years been used empirically in Russia for all forms of cardiac failure ; but until the clinical observations of Bubnow, of St. Petersburg, were made in 1879, nothing was known of the action of this drug. Since then a number of observers have clinically studied it and given favorable reports of its action as a cardiac stimulant.

The writer, has supplemented these, for the most part, clinical observations by an elaborate study of its physiological action on animals, using for the purpose frogs and dogs.

The conclusions he arrives at are : Adonidin in all doses increases arterial pressure by stimulating the vaso-motor centres, and by increasing the cardiac force.

In moderate doses it increases the pulse rate and force from the first, but when large toxic doses are given, it primarily slows the heart by stimulating the pneumogastric, and then increases the pulse rate by depressing the inhibitory nerves and stimulating the accelerator apparatus.

The slowing of pulse rate is also, in all probability, due in part to increased arterial pressure ; as under these circumstances the blood paths are greatly diminished in calibre. On the nervous system the drug has but little action, unless the quantity administered be enormous. Under these conditions it paralyzes the sensory side of the cord, but has no effect on the motor tract, or on the efferent or afferent nerve-trunks.

As to the practical use of the drug, Durand and others all declare that adonidin is possessed of far greater diuretic properties than is digitalis. Thus Traversa says that it increases the urine from 300 c. c. (the total quantity passed) to 2,000 c. c., the sp. gr. being diminished ; œdema is rapidly diminished under its influence. While Durand does not believe that its cardiac effects are equal to those of digitalis, he concludes that it is preferable when a long course of medication is necessary, as it is not cumulative.

The indications for its use are the same as for digitalis.

Houchard's observations agree with those of Durand and others, viz., that adonidin diminishes the frequency and increases the force of the heart's action, arterial tension, and diuresis.

The dose is one fifteenth of a grain in pill form four or five times daily. MORTON PRINCE.

A Few Words About Hypnotism from a Therapeutic Point of View. Dr. FOURNIER. *Gazette des Hôpitaux*, p. 536, 1866.

The author in this article, in opposition to the views of Charcot, Bernheimer, Richet, Dumontpallier, and Battey, sanctioning the therapeutic use of hypnotism, takes a directly opposite stand, and publishes two cases in which direct injury was done by its use. The first case was that of a man, æt. thirty-six, who has suffered from occasional epileptoid attacks for the past seven years. Since the last year these have given place to well-marked epileptic attacks, which took place about every twenty-eight days, so that in all he had had fourteen attacks during the year. The first hypnotic séance took place on the 11th of May. By means of fixation of a bright object, closure of the lids was obtained after half an hour. All the experiments dependent upon nervo-muscular hyperexcitability were performed with success. After awakening he complained of heaviness of the head, which, however, soon left him.

May 13th.—Severe epileptic attack. Hypnotism.

May 14th.—No attack. Hypnotism.

May 15th.—No attack. Hypnotism.

May 17th.—Two epileptoid attacks since last visit. Hypnotism.

May 18th.—No attack. Hypnotism.

May 20th.—Yesterday, at 4 P.M., had an epileptic attack of the utmost severity.

May 22d and 24th.—Hypnotism.

May 25th.—At 8 P.M., exceedingly severe attack of epilepsy.

Hypnotism discontinued.

The second case:

Mrs. C., æt. thirty-four, has since two years, attacks of hysteria, which come on at time of menstruation. All remedies having proven futile, hypnotism was tried.

May 30th, at 2.30 P.M., first séance. A few minutes sufficed to produce sleep. Various experiments. Awakening after a few minutes.

The night from the 30th to the 31st, she had a most severe attack. This attack differed from the preceding ones. It began by intense hallucinations, and finished by severe attacks of dyspnœa and palpitation of the heart.

These cases not only lead the author to cautious experiments in the use of hypnotism upon patients, but also upon people who are perfectly healthy. The number of hysterical, hypochondriacal, and neuropathic individuals is already large enough without increasing the number through any fault of our own.

G. W. J.

The Electrical Resistance of the Body.—DE WATTEVILLE (*Neurol. Central.*, No. 9, 1886) states that for a long time he has noticed a marked variation between the deviation of the needle of an absolute galvanometer and the number of the elements

employed. He at first sought for an explanation of this phenomenon in the inaccuracy of the instrument. After obtaining, however, an Edelmann's galvanometer, he convinced himself that the cause of the difference lay in variations of the resistance of the body. Waller and Stone arrived independently at the same conclusions.

A simple method of observing the fact is the following : Place two large electrodes upon the skin, and after its resistance has been reduced to a minimum, observe the deviation of the needle after each group of a given and equal number of elements. Then add to the first group, the second, third, and fourth successively, and mark the deviation after each addition ; it will be found that the total deviation after each addition is greater than the sum of each group taken separately. For example : de W. found that each of four groups, containing three Leclanchés each, gave a current strength of two milliampères. After joining successively the groups, he obtained with 3, 6, 8, 12 elements, not 2, 4, 5, 8 M.-A. respecting as he should by Ohm's law, but instead an increase from 2 to 5, 8.5 and 11.8 M.-A. A control experiment with a metal resistance coil gave an increase in arithmetical progression according to Ohm's law, showing that the above variation was due to a variation in the resistance of the human body. De Watteville concludes that we can lay it down as a law that in the usual medical uses of electricity the resistance of the human body diminishes with the increase of the electro-motive force.

Again, it is well known that when a current, passing through the body, is suddenly reversed, there is an increase in its strength shown by the galvanometer. For example : a current of twenty milliampères, on being reversed, will more or less quickly increase to 23, 24, and 25 M.-A. After some moments the needle of the galvanometer will be seen to return to the original number 20. The amount of increase of deviation and rapidity of return to the original strength will depend upon the duration of the current before being reversed. By a control experiment it is shown that a reverse current due to polarization does not correspond to the above amount of increase, but only amounts to a fraction of a milliampère. De Watteville concludes that the electrical current as usually applied for medical purposes causes certain alterations in the tissues, which in turn give rise to a temporary increase in the strength of the current on being reversed. This increase may be explained in two ways : Either the resistance of the tissues for the reversed current is diminished, or there is developed an electro-motive force through polarization. The second hypothesis is the more acceptable. Nevertheless there are opposed to it the facts that, first, the supplementary deviation of the needle does not appear immediately with the change in the direction of the current ; and, second, as the above control experiment shows, the tissues do not give a current as the result of polarization sufficient to account for the increase.

MORTON PRINCE.

VOL. XIII. SEPTEMBER AND OCTOBER, 1886. NOS. 9 AND 10.

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

PRESIDENTIAL ADDRESS.

INTRODUCTORY REMARKS ON THE WORK OF THE AMERICAN NEUROLOGICAL ASSOCIATION.

ARRESTED AND ABERRANT DEVELOPMENT OF FISSURES AND GYRES IN THE BRAINS OF PARANOIACS, CRIMINALS, IDIOTS, AND NEGROES. DESCRIPTION OF A CHINESE BRAIN.*

BY CHARLES K. MILLS, M.D.,

PRESIDENT OF THE AMERICAN NEUROLOGICAL ASSOCIATION; VICE-PRESIDENT OF THE PHILADELPHIA NEUROLOGICAL SOCIETY; PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE, ETC.

PART I.—Published work of the members of the American Neurological Association—Remarks on Membership and on the By-Laws—Congress of American Physicians and Surgeons—Objects, Duties, and Dangers of Special Associations—Personal and Impersonal Criticism.

PART II.—Arrested and Aberrant Development of Fissures and Gyres—Importance of the Study of the Comparative Anatomy of the Brain—Some Points from the Brains of the Primates—Simian and Human Brain Compared—Indications of a Low or Aberrant Type of Human Brain—Description of the Brains of Burk, Taylor, Beach, of a Feeble-Minded Youth, and of a Negro — Indications of Atypical Asymmetry — The Brain of Guiteau—The Question of a Criminal Type of Brain and the Anatomical Basis of Crime.

PART III.—Preliminary Study of a Chinese Brain, by A. J. Parker, M.D., and Chas. K. Mills, M.D.

I.

ALTHOUGH prevented from attending the last meeting of the Association, you did me the honor to unanimously elect me President for the year which begins

* Presidential address, delivered at the meeting of the American Neurological Association held at Long Branch June 21, 22, and 23, 1886.

to-day; and for this honor I cordially thank you, although I feel poorly fitted to occupy the chair which has been filled by such distinguished predecessors.

The Association may well congratulate itself upon the large amount of published work which has been done by its members since the last annual meeting. In Pepper's "System of Practical Medicine by American Authors," of the 1326 pages of the fifth volume, which now constitutes the greatest general treatise on nervous diseases which has ever been written, no less than 843 have been contributed by members of this Association. American medical periodicals, both neurological and general, have also amply exhibited in their pages the work of members of this Association.

I am glad to note a tendency to the filling of our ranks with new members. Much in this direction can be done by individual effort on the part of those who have the interests of the Association at heart. Our membership ought to be at least doubled by the addition of acceptable physicians from all sections of the country; for the American Neurological Association should be in fact, as well as in name, the great national representative body of those members of the American profession who are interested, either practically or theoretically, in neurology and psychiatry, and the branches of science which are especially correlated and auxiliary to these subjects. It might well include in its membership some anatomists, zoölogists, histologists, and psychologists, etc., who are particularly concerned with a study of the nervous system, either in man or the lower animals, even although not physicians. We have not in this country, as in Great Britain, and in several nations of Continental Europe, a great Medico-Psychological Association. In place of it we have only the Association of Medical Superintendents of the American Institutions for the Insane; and the National Association for the Protection of the Insane and the Prevention of Insanity, which is not confined to the medical profession in membership, and which has been a society rather of a humanitarian character than one which has either called forth or sustained scien-

tific enterprise in neurological fields. These associations may have distinctive and useful spheres of their own; they may serve a good purpose in some directions; but such of their members as are professionally and scientifically worthy of the honor, should be admitted to this Association; and so should those medical jurists who take an active part in special investigations into the mind and nervous system: although I would not, for a moment, advocate the cheapening of membership, or the enlargement of the opportunities for obtaining it beyond reasonable limits.

The adoption of the amendment to Article IV. of the By-Laws, by which it has become the rule that all business, not of a scientific nature, shall be transacted in executive session, will do much towards making our meetings more interesting. It will insure to those who are on the programme for papers or discussion at certain times, that their work shall not be excluded or postponed by tedious discussion of unexpected matters of policy or business. The amendment to Article VI. of the Constitution, adopted at the meeting held June 21st of the present year, by which that Article is now made to read, that "the annual meeting of the Association shall be held on any two or three days in the month of July in each year, as may be chosen by the Council, which shall also designate a place of meeting," will, also, I believe, have a favorable influence upon the welfare of the Association. The holding of meetings in different sections of the country, a plan which is inaugurated with the present session, will tend to make our organization more thoroughly national.

I wish now to refer briefly to a matter which I have deeply at heart. It is well known to a majority of the members of this Association that a project is on foot and well under way for the organization of a Congress of American Physicians and Surgeons. This measure originated in the American Surgical Association, with the distinguished Southern surgeon, Dr. C. H. Mastin, of Mobile, Alabama. A memorial and accompanying resolutions regarding it have been transmitted to me by Dr. Mastin, and also by Dr. J. Ewing Mears, Secretary of the Committee of the

American Surgical Association. The memorial and resolutions will come before you for formal action at the executive session to-day. The details of the project are doubtless familiar to most of you.

It is proposed to unite the following-named associations into a Congress to be called "A Congress of American Physicians and Surgeons": the American Surgical Association, the American Ophthalmological Association, the American Otological Association, the American Neurological Association, the American Laryngological Association, the American Gynæcological Association, the American Dermatological Association, the American Climatological Association, and the Association of American Physicians.

The plan of organization proposed embodies the following points: each society is to select its own officers, hold its own sessions apart from the others at the time and place of meeting of the Congress, publish its own transactions, and do all other acts which by virtue of its constitution and by-laws it has the inherent right to do, thus preserving its own autonomy; the Congress to be composed of these special societies when in session, and its meetings to be held in the city of Washington; the constitution and by-laws of the Congress to be formed by a committee of the like number from each special society; the opening session of each annual meeting of the Congress to be devoted to such general business as might pertain to the interests of the association as a whole; the Congress to be presided over by a president, elected annually, who must deliver an opening address on the first day of the session; the president to be chosen by a nominating committee of one from each special society; the presidents of the special societies to be *ex-officio* vice-presidents of the Congress; membership in the Congress to be acquired only by virtue of fellowship in one or other of the special organizations; the other officers to be determined upon by the convention in session.

Personally I am heartily in favor of this movement, and I trust that the American Neurological Association will unanimously indorse it, and appoint a committee of confer-

ence. Of the associations which it is proposed to unite to form the Congress, the American Surgical, the American Climatological, and the American Laryngological Association, and the Association of American Physicians, have already indorsed the project and appointed committees.

Some difficulties may arise in connection with the initiation of the Congress; some differences of opinion there may be as to the plan of organization and methods of holding the meetings; but with committees composed of men who have both the interests of the special organizations which they represent, and the general welfare of the entire project at heart, I have no doubt that such difficulties can be readily overcome and such differences promptly arranged. One danger apprehended by some is that the Congress may drift, insensibly, perhaps, into the arena of medical politics; but, if wisdom prevails during the development and crystallization of the plan of organization, the rocks and shoals so much dreaded can, with comparative ease, be avoided.

The plan submitted in the memorial of Dr. Mastin is, on the whole, a good one, but might, it seems to me, be improved in some respects. It would, I believe, be better for the meetings of the Congress to be biennial, thus allowing the associations as special organizations to have entirely separate and independent meetings on alternate years. Among other advantages, this would allow the separate associations to meet every two years in different sections of the country —at the seashore, in the mountains, or in some great city— according to the wishes of a majority of the members. The meetings of the Congress as such, however, should be held at a fixed place; it should have a local habitation as well as a name, and should not allow itself to degenerate into a wandering, social body. Some differences may arise as the most suitable place to hold the meeting of the Congress. Personal preference should yield to the will of the majority of those who confer. Washington being the national capital, having within its limits the great Congressional Library and Army Medical Museum, being geographically best situated to the different medical sections of the country, should receive the preference, but this is a matter which can be decided

satisfactorily by a wise committee of conference. It would be well if some plan could be adopted by which the presidency of the Congress should be held successively by a member of each different organization entering into its composition. No one should be reëlected to the position. During the days of the sessions of the Congress, both general and special meetings could be held; for example, a general meeting of the Congress with a carefully arranged programme in the mornings of each day, and special independent sessions of each constituent organization in the afternoon. This plan would permit the coming together for mutual advantage and criticism of individuals representing all branches of the science of medicine, and would allow, at the same time, the special organizations to do much of their own work in their own way after the manner of the sections of the American and the British Medical Associations. The proceedings both of the general and of the special meetings should be published in one volume. The authors of addresses, papers, and all forms of scientific contributions, should, however, be allowed to publish their production, at least in abstract, in medical periodicals, and not wait for the slow process of publication of an annual volume.

In concluding these introductory remarks, a few words with reference to the object, duties, methods of conduct in a society of this kind may not be out of place. I cannot do better than repeat the words of one of the original members of this Society, Dr. S. Weir Mitchell. In discussing, before the Philadelphia Neurological Society, the object and duties of special societies, he said very truly that if they have any real reason for existence, it is in the fact that they bring together in groups men having common interests, so that these men stimulate one another by example and criticism, and by the sympathy arising out of unity of pursuits; that it is never well to be absolutely isolated in our pursuits; that all men do more and better work amidst the competition of other workers; that some men can do no work unstirred by the ferment of companionship in like efforts. "In these subtle agencies lies the value of associations like ours—in the examples they offer, the dis-

cipline they teach, the criticism they afford, the sympathy they evolve. The advantage of small societies is that they secure definiteness of aim, and that we hear only what interests all who are present." The same distinguished physician points out that the danger of such bodies is narrowness.

An association like the American Neurological Association, any scientific body that deserves the name, should not degenerate into a mutual admiration society; but, on the other hand, it should not become an arena for the settlement of either past or prospective personal disputes and difficulties. Papers read, and subjects introduced for discussion, should be subjected to careful, severe, and, if called for, even hostile criticism; but such criticism should always be exercised and administered from the scientific and impersonal standpoint.

II.

The President of this Association owes a duty to its members from the scientific side. He should contribute, not only as a member of the Association but also in reciprocation of the honor conferred upon him, some new facts to the proceedings of the meeting at whose sessions he presides. It is my purpose, in addition to the remarks which have just been made with reference to some of the special interests of the Society, to present for your consideration a paper upon the study of arrested and aberrant fissures and gyres, which will be illustrated by descriptions and references to the brains of paranoiacs, criminals, idiots, negroes, and apes.

My study will be confined to the topography of the cerebral surface. This is but a limited portion of the great field of cerebral anatomy. The practical importance of such a study to neurologists, and more particularly to general practitioners, may to some, at first sight, appear to be slight. Looked at closely, however, such will be seen not to be the case. Much of the confusion and many of the controversies in connection with the great subject of cerebral localization, which has to so large an extent occu-

pied the medical profession, particularly neurologists, neuro-physiologists, and neuro-pathologists, during recent years, have arisen from an imperfect acquaintanceship with cerebral morphology.

My object being to direct attention to certain conditions of arrested and aberrant development in the specimens to be exhibited—to demonstrate abnormalities and peculiarities in the formation of gyres, fissures, and lobes,—a few facts of comparative anatomy and of human cerebral anatomy, a comprehension of which is necessary to an understanding of the questions discussed, will be first briefly considered. The specimens present in striking degree such characteristics as atypical asymmetry of the hemispheres in bulk and in fissural and gyral arrangement, ape-like similarities and affinities, and a persistence of embryonic and fœtal peculiarities.

The physician should, by a study of the brains of primates, of fœtal brains, and of brains supposed to be normal, obtain a good working knowledge of the average arrangement of the hills and valleys of the cerebral surface. Observations have not been sufficient to enable us to say with great positiveness what we would expect to find in every brain which is alleged to be normal, but scattered contributions on the subject of cerebral morphology permit us to arrive at some approximate conclusions. In the literature of the subject a few names are prominent, such as, for instance, in Germany and Austria, Bischoff, Virchow, Ecker, Meynert, and Benedikt; in France, Gratiolet and Broca; in Italy, Lombroso; in Great Britain, Huxley, Turner, and Rolleston; and in America, Parker, Wilder, Osler, and Spitzka. The treatise of Gratiolet, "On the Convolutions of Man and the Primates," is among the greatest of the contributions to the study of the cerebral surface.

While we have not as yet what might be termed an average standard adult brain, what can be hoped for is that the observer, even the practitioner of medicine of ordinary attainments in cerebral anatomy, shall have a fair idea of brains of different types or degrees of inferiority or superi-

ority of development. Some authorities deny that the fissures and gyres of the human brain can be elucidated by a study of the lower animals, and perhaps in some respects it would be best always to describe and figure what is found without reference to any views as to affinities with or differences from other animals. Even so high an authority as Meynert, however, takes as his starting-point the brain of a monkey. Bischoff holds that the monkey brain is not a miniature model of the human brain, but represents arrested stages in the development of the latter. Equally important is the study of fœtal brains in different stages, and of the post-natal brain at different ages.

In the higher forms of quadrupeds, fissures and gyres are arranged in certain types—oblique, longitudinal, and transverse; and a few well-marked, so-called, primary fissures are common to all types. In the quadrumana, new features appear, among the most important of which are primary fissures similarly disposed, and the gradual development of secondary and tertiary fissures, dividing the surface of the brain into lobes and gyres according to a new but constant pattern, like that of the human brain. The principal of these in the primates are the Sylvian, the central, the external perpendicular, the supertemporal, the parietal, the occipital, the hippocampal, the calcarine, and the supercallosal.[1]

A study of the size, development, branchings, and surroundings of these great fissures in man and in the primates gives points of departure in the consideration of the question of highness and lowness of type—that is, of relative superiority or inferiority.

---

[1] In describing fissures and gyres I shall in the main use the nomenclature of Prof. Burt G. Wilder of Cornell University.

Prof. Wilder, (JOUR. OF NERV. AND MENT. DISEASES, vol. xii., No. 3, July, 1885) has suggested:

1. That all the indentations of the cerebrum be called fissures (abbreviation, F.), and that this word be restricted thereto.

2. That, excepting a few parts (*insula*, *cuneus*, *præcuneus*, *paracentral lobule*, *uncus*, *subiculum*, etc.) which have received special names, all the inter-fissural elevations be called gyres, Latin gyri (abbreviation, G.), and that this word be restricted thereto.

3. That, so far as possible, mononymic names for the fissures and gyres be selected from among those which are in use, or formed therefrom by substituting prefixes for words indicating relative position.

The most important suggestions as to naming fissures and gyres are shown in the following table:

One of the most important fissures in the ape is the external perpendicular; in the man-like apes it is particularly well-marked, a deep gash separating the parietal from the occipital lobe, chiefly on the lateral aspect of the hemisphere, but reaching well up to the median edge, with a continuation on the inner aspect, known as the internal perpendicular fissure. In its depths are certain gyres, con-

	FISSURES, MAINLY OR PARTLY MESAL.	
Name (*Wilder.*)	*Abbreviation*	*Synonym*
Callosal,	CL,	Callosal.
Supercallosal,	SPCL,	Calloso-marginal.
Occipital,	OC,	Parieto-occipital.
Calcarine,	CLC,	Calcarine.
Collateral,	CLT,	Collateral.
Hippocampal,	HMP,	Hippocampal.
	GYRES, MAINLY OR PARTLY MESAL.	
Callosal,		Gyrus fornicatus.
Paracentral,		Paracentral lobule.
Precuneus,		Precuneus.
Cuneus,		Cuneus.
Subcalcarine,		Lingual lobule.
Subcollateral,		Fusiform lobule.
Hippocampal,		Hippocampal.
Uncus,		Gyrus uncinatus.
Subiculum,		Subiculum.
	FISSURES, MAINLY OR PARTLY LATERAL.	
Sylvian,	S,	Posterior branch of S.
Presylvian,	PRS,	Ascending branch of S.
Subsylvian,	SBS,	Anterior branch of S.
Basisylvian,	BS,	Basal portion of S.
Olfactory,	OLF,	Olfactory.
Orbital,	ORB,	Orbital or Triradiate.
Central,	C,	Fissure of Rolando.
Precentral,	PRC,	Precentral.
Superfrontal,	SPFR,	First frontal.
Subfrontal,	SBFR,	Second frontal.
Postcentral,	PC,	Retrocentral.
Parietal,	PTL,	Interparietal.
Supertemporal,	SPTMP,	Parallel or first temporal.
Subtemporal,	SBTMP,	Second temporal.
Exoccipital,	EOC,	Wernicke's.
	GYRES, MAINLY OR PARTLY LATERAL.	
Insula,		Island of Reil.
Subfrontal,		Third frontal.
Superfrontal,		First frontal.
Precentral,		Precentral.
Postcentral,		Postcentral.
Parietal,		Superior parietal lobule.
Marginal,		Inferior parietal.
Angular,		Angular.
Supertemporal,		First temporal.
Subtemporal,		Second temporal.

volutional masses which tend to fill up the bridge over the chasm as brains advance in grade. In man usually the chasm becomes nearly or quite filled with brain substance.

Rolleston,[1] referring to this fissure, says that "in one part of the brain, where two of the five great masses into which its convoluted surfaces may be mapped out abut

Besides these names which correspond to fissures and gyres described by writers and anatomists generally, Wilder has introduced several new terms descriptive of what he regards as new fissural integers and gyres. With some of his suggestions I accord, with others I do not. The chief of these new suggestions are as follows: *Paracentral fissure*, PARC, the terminal portion of the supercallosal fissure; *Precuneal fissure*, PRCN, a fissure crossing the precuneus; *Paroccipital fissure*, PAROC, the posterior portion of the parietal fissure with the transverse occipital of Ecker; *Postcalcarine fissure*, PCLC, a fissure in the extremity of the occipital lobe; *Amygdaline fissure*, AMYG, a fissure at the tip of the temporal lobe near its median border; *Postcalarine gyre*, a gyre in the occipital lobe posterior to the calcarine fissure; *Medifrontal fissure*, MFR, a somewhat common fissure of the frontal lobe between the superfrontal and subfrontal fissures; *Meditemporal fissure*, MTMP, a fissure between the supertemporal and subtemporal; *Paroccipital gyre*, the first occipital gyre of Ecker, or the superior external *pli de passage* of Gratiolet; *Medioccipital gyre*, the second occipital gyre of Ecker; *Suboccipital gyre*, the third occipital of Ecker.

In some cases in the text the names of Gratiolet, or others, have been used in describing parts, as, for instance, the *plis de passage*, which are not recognized or named by Wilder.

The following are the most important publications of Prof. Wilder on the subject of encephalic nomenclature:

A Partial Revision of Anatomical Nomenclature, with Especial Reference to that of the Brain. *Science*, ii., No. 38, pp. 122–126; No. 39, pp. 133, 138; March 19 and 26, 1881.

On Encephalic Nomenclature. Am. Neurol. Assoc. 1884, JOUR. NERV. AND MENTAL DISEASE, July, 1884, 18, 50; abstract.

Methods of Studying the Brain. The "Cartwright Lectures" for 1884. *N. Y. Med. Jour.*, February 9, 16, 23; March 1; April 6, 26; May 10; June 14; August 2.

On Some Points in Anatomical Nomenclature. Am. Assoc. Proc., 1884; abstract.

Encephalic Nomenclature. 1. Cœlian Terminology; the Names of the Cavities of the Brain and Myelon. *N. Y. Med. Jour.*, xli., pp. 325–328, 354–357, March 21 and 28, 1885; 8 fig.

Paronymy *versus* Heteronymy as Neuronymic Principles. Presidential address at eleventh annual meeting of Amer. Neurol. Assoc., June 18, 1885. JOUR. NERV. AND MENT. DISEASE, xii., July, 1885, pp. 21.

On Two Little Known Cerebral Fissures, with Suggestions as to Fissural and Gyral Names. Amer. Neurol. Assoc. Trans., JOUR. OF NERV. AND MENT. DISEASE, xii., 350–352, July, 1885.

The Paroccipital: a Newly Recognized Fissural Integer. JOUR. OF NERV. AND MENT. DISEASE, xiii., No. 6, June, 1886.

Prof. Wilder has also sent me manuscript notes of a revised list of fissures, presented at the last meeting of the American Association for the Advancement of Science; and to be published with figures in the *American Naturalist*, October, 1886.

[1] "On the Affinites and Differences between the Brain of Man and the Brains of Certain Animals," by George Rolleston, M.D., F.R.S. Two Lectures delivered at the Royal Institution. *Med. Times and Gazette*, vol. I., Feb. 22, 1863, and March 15, 1863, p. 181 and p. 259.

upon each other, what are but connecting spurs in the ape's brain, overhung and concealed by the beetling parietal and occipital lobe, rise in man to the dignity of connecting table-lands, filling up and bridging over levelly what is a valley, or rather a chasm, in most simious encephala." Gratiolet has discussed these bridging or transition gyres under the name of *plis de passage*. He attached great importance to them in the study of the problem of development, and distinguished altogether six of these transition gyres—four external and two internal. The four external pass from the lateral portion of the occipital lobe to join the parietal and temporal lobe. He named the uppermost of these the first or superior external *pli de passage*, and the others, the second, third, and fourth, respectively. The two internal transition gyres he named the superior internal and the inferior internal *pli de passage*. These, according to him, connect the cuneus with the precuneus. Parker[1] has also called attention to the importance of these bridging convolutions in a valuable paper.

Rolleston indicates clearly as follows the chief points in which the human cerebral surface and the cerebral exterior of the ape coincide and differ. The human and simian cerebrum viewed from above presents an ovoid shape, the human blunt, the simian taper; the human outline is irregular from elongation; the outline of the ape conforms more closely to the egg type; the lines limiting the superior edge of the simian brain are all but a semicircle, while below, regularity and evenness have been removed from the lower and become the characteristics of the higher brain; unevenness in the ape's brain is due rather to defect in the development than the reverse; the human brain has more altitude than that of the ape, and is always wanting in internal convolutions; the simian brain is very frequently wanting in external convolutions, filling up certain fissures, especially the external perpendicular fissure; the frontal gyres have in man an enormous development as compared with those in the ape; and it is especially in the uppermost of these that the widest differences are noted.

[1] "Proceedings of the Academy of Natural Sciences of Philadelphia," vol. xxx., 1878, p. 159.

"The occipital lobe is separated from the parietal lobe," says Parker, "by a well-marked transverse fissure, the so-called perpendicular fissure; the mesial portion, corresponding to the fissure known in the human brain as the parieto-occipital, is called the internal perpendicular, while the lateral portion is known as the external perpendicular fissure. In most of the monkeys, such as Cebus, Cynocephalus, Cercopithecus, Macacus, etc., these two fissures are continuous; but in man and the higher apes, such as the Orang, Chimpanzee, etc., they are separated into distinct fissures by the development of a bridging convolution, the so-called superior external transition or connecting convolution, the *pli de passage supérieur externe* of Gratiolet. The same condition is also found in Ateles and Hylobates. In man this convolution is largely developed, and alters, to a great extent, the appearance of this region as found in the ape. This convolution was found invariably smaller, less developed, and simpler in the negro than in the white. In one negro brain it was so imperfectly developed that the internal and external perpendicular fissures were superficially continued. The fissure corresponding to the external perpendicular is also better developed in the negro."

With these facts before us, the peculiarities which probably indicate a low or aberrant type of human brain can be recognized. In general, simplicity of structure, with well-defined and comparatively little-complicated fissures and gyres, point to a low type; simplicity of the frontal fissures and gyres in particular indicates an inferior order of brain. Unusual symmetry and atypical asymmetry are indicative of a low type. Normal human brains present a typical asymmetry. Coming to special details, Benedikt refers to a fissure, which he terms the external orbital, very constant in apes, and not commonly distinct in the human brain, but rarely absent in criminals' brains examined by him. Partial or complete uncovering of the insula, that is, its exposure to sight without pulling apart the margins of the Sylvian fissure, is to be expected in white brains of inferior grade. The Sylvian fissure in the average brain is

of moderate length; the higher the type, other things being equal, the more likely it is to be short. The central fissure in inferior types will be less sinuous, and less likely to be fully separated above from the sagittal or longitudinal fissure and below from the Sylvian. With reference to the central fissure, the question of complete or partial confluence, either above or below, or laterally, is of considerable interest. These confluences are comparatively rare and probably indicative of low type. It seems, however, unphilosophical to speak with Benedikt of a "confluent fissure type of brain." Fissures run together in brains of low organization because of the want of development of connecting or encircling gyres, and the stress should be laid upon this absence of connecting gyres. Bridging of the central fissure has been observed in rare instances. This might be thought to indicate rather a high order of individual brain, since it gives additional convolutional substance, but it is an aberrant or unusual condition. In poorly developed hemispheres the gyres bounding the central fissure will not be sinuous and complicated—that is, will present little elaboration. An unusually well-defined, elongated, or unbridged parietal fissure; a small marginal gyre, as when the parietal fissure approaches closely to the Sylvian; a much elongated, so-called retrocentral (postcentral of Wilder) fissure; and an occipital fissure which opens well out on the lateral surface, with the superior *pli de passage* below the brain level, are probably indications of inferiority. Shortness of the Sylvian fissure, in more highly evolved brains, tends to confer a corresponding shortness in the temporal lobe; the opposite is seen in brains but little evolved. Other peculiar conditions of the temporal lobe seem to be indicative of low type; as great length, particularly of the posterior vertical arm of the supertemporal or parallel fissure, with a tendency to confluence with the Sylvian, occipital, or parietal fissure. Such a tendency indicates a lack of development of Gratiolet's transition gyres of the temporo-occipital and parieto-occipital regions. On the median surface of the hemisphere, some indications of inferiority are smallness of the paracentral lobule and precuneus, confluence

complete or incomplete of the calcarine and hippocampal fissures, and unusual definition of the median portion of the occipital fissure, although, curiously, in a few brains of low type an unusual development of the so-called gyre of the cuneus, or inferior internal *pli de passage*, is observed. This is an aberrant or peculiar condition, certainly not usual in the average human brain.

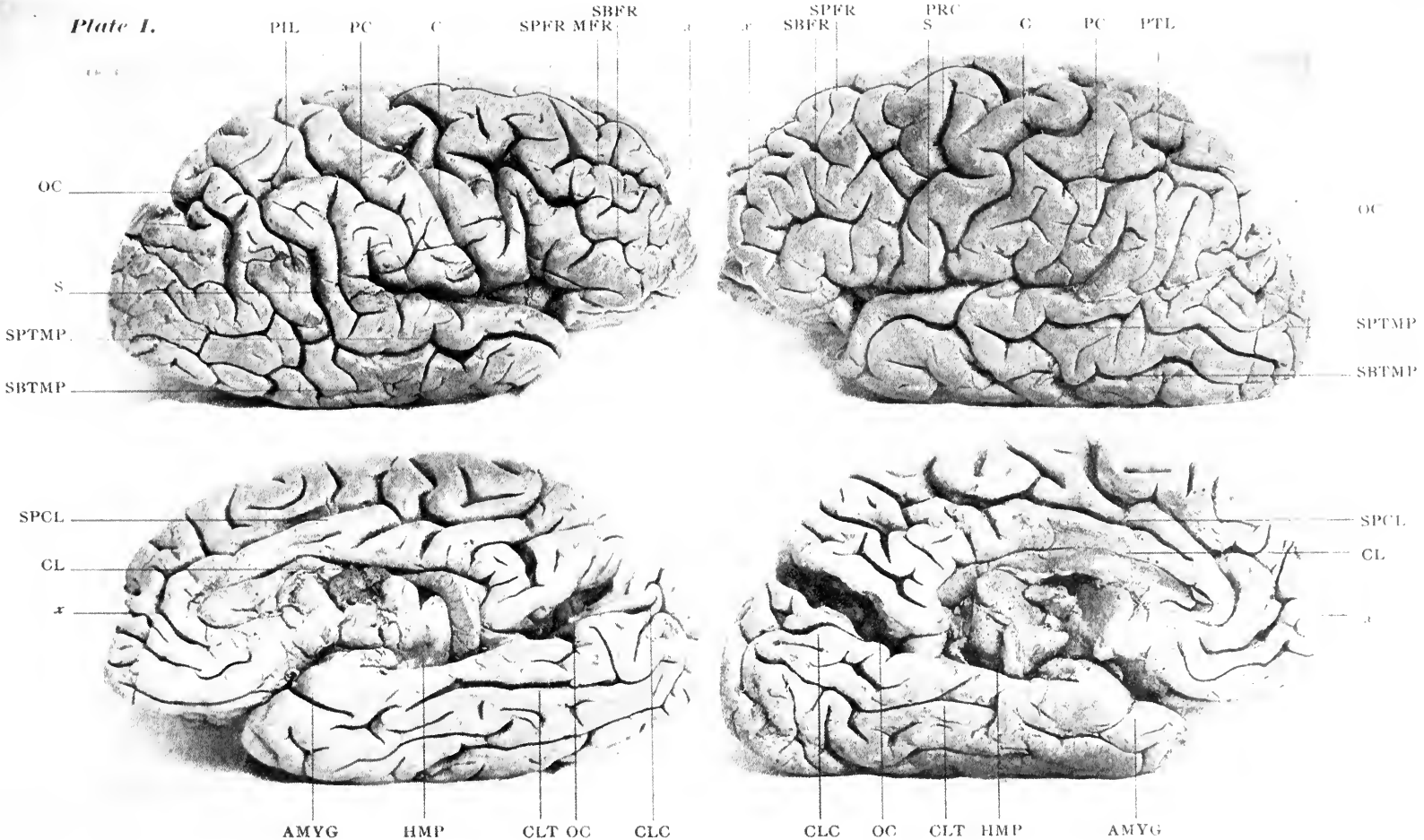
The specimens which I exhibit are: (1) The brain of James Burk; (2) The brain of Joseph Taylor; (3) The brain of a feeble-minded youth. I have also notes on (4) The brain of Dr. L. U. Beach; and (5) The brain of Edward Ford. In addition, I exhibit, through the courtesy of Dr. A. J. Parker, (6) One hemisphere of the brain of a negro; and (7) A Chinese brain.

The first specimen which I exhibit is the brain of a man named Jas. Burk, who was either burned to death or suffocated in the fire which destroyed a large portion of the Insane Department of the Philadelphia Hospital in 1885. He was about 70 years of age at the time of his death. He was admitted to the Insane Department of the Philadelphia Hospital more than twenty years since. It is told of him that forty years ago, in Ireland, he shot a watchman who attempted to arrest a friend, and fled to America to escape punishment. In 1856, or about then, he attempted to cut his throat, and, a few months later, killed a man and a woman who were employed as cooks in the hospital. He was evidently a delusional lunatic who believed that it was his duty to kill. He sometimes said that he was followed by an evil spirit who carried a long chain, and when this spirit said "kill" he felt that he must. After the killing of the cooks he was, for a time, especially violent and dangerous. For two years he was shackled with heavy chains, and after these were removed his hands were kept muffled. After the removal of the chains he believed that both his legs were cut off, and walked about with great caution for fear of injuring himself. He sometimes asserted that if his hands were free he would kill anybody that came in his way. He was a finely built man and is said to have had a remarkable memory.

In Plate I, we have two views of each of the hemispheres of the brain of Burk—from the dorso-lateral and meso-ventral aspects. The lettering corresponds with the nomenclature of Wilder. A careful comparison of the following description, with the figures and symbols, will serve to bring out the peculiarities of the brain. Small blocks of brain tissue were removed for microscopical examination from the tips of each frontal lobe at *x*. The photographs were made after the brain had been hardened in zinc chloride and alcohol. Fissures only have been indicated, in order to save too much interference with the appearance of the phototypes. The letters are those given as abbreviations by Wilder.

In the *right hemisphere* of the brain of Burk the Sylvian fissure is distinctly shorter than the corresponding fissure of the left hemisphere. The insula is exposed in its anterior portion with the brain in ordinary position, although it is not as distinctly visible as in the brain of the feeble-minded youth and the negro, which I will exhibit later. The central fissure is confluent with the Sylvian. The central gyres are well developed, except a depression about the middle of the precentral gyre, where is shown a tendency to confluence with a vertical secondary branch of the superfrontal fissure. The precentral fissure is short and does not communicate with the Sylvian, but is confluent with the subfrontal fissure. The right frontal lobe presents three well-defined fissures running in a sagittal direction, corresponding to the superfrontal, subfrontal, and medifrontal fissures of Wilder, and giving, if these are regarded as primary fissures, four well-demarcated gyres (the four frontal gyres of Benedikt). Otherwise the markings of the frontal lobe are comparatively simple. The orbital surface shows in addition to the common H-shaped or zygal fissure, an exact duplication of this H-shaped fissuration, anterior to it, and also a well-marked fissure sagittal in direction external to both H-shaped fissures (the external orbital fissure of Benedikt). The parietal fissure is in two parts, the separation being by a narrow gyre which apparently corresponds to the second external *pli de passage* of Gratiolet. The posterior branch of the parietal fissure (paroccipital of Wilder) almost completely merges with the occipital fissure, from which it is separated only by a very much depressed anterior arm of the superior *pli de passage* of Gratiolet (first occipital gyre of Ecker, paroccipital of Wilder). This posterior branch of the parietal communicates with a well-marked transverse occipital. The so-called retrocentral fissure (postcentral of Wilder) is large and clearly defined. Both the parietal and marginal gyres are well developed. The angular gyre is small. The occipital fissure shows in itself, and

Plate I.



BRAIN OF JAMES BURK.

in its communications and surroundings, marked ape-like conditions. As already stated, it partly communicates with the posterior branch of the parietal, the superior external annectant gyre being very small and much depressed in its anterior portion. The posterior gyral margin of the occipital fissure has a true pent-house appearance. The medioccipital and suboccipital gyres are well demarcated by fissures which are in the same lines with the supertemporal and subtemporal fissures. The calcarine fissure is crossed about its middle by a narrow gyre. The occipital, the calcarine, and the hippocampal fissures are almost completely confluent. The collateral fissure and its bounding gyres are well defined and simple in type. The supertemporal fissure is clearly defined, its posterior ascending arm being nearly confluent with the parietal fissure. The four fissures of the temporal lobe are all particularly well marked, reminding one of the diagrammatic representations of Ecker. Wernicke's fissure [1] is well defined, communicating with the supertemporal and subtemporal fissures. The supertemporal gyre narrower than usual, particularly in its anterior half. The supercallosal fissure is well defined, with a shallow bridge about the junction of its middle and posterior third. The inflected, the precuneal, and the amagdaline fissures of Wilder are all well shown in this hemisphere.

In the *left hemisphere*, the Sylvian fissure is longer than on the right side. The insula is exposed, but not as much so as on the other side. The central fissure shows a superficial tendency to confluence with the Sylvian, but the two are separated by a small bridge. The precentral and postcentral gyres are both strongly developed. The left frontal lobe is not clearly separated, like the right, into four gyres by three distinct fissures. The superfrontal fissure and subfrontal fissure are well outlined, but in place of the medifrontal fissure which is present on the other side, is an undulating arrangement of fissures and gyres. The super- and subfrontal gyres are well demarcated and simple in aspect. The orbital fissuration is similar to that on the other side, except that the fissures are deeper and more elongated. The parietal fissure, as on the other side, is in two parts separated by a small sunken gyre near its middle. The posterior branch of the parietal is connected with an illy defined transverse occipital. The superior external *pli de passage* is narrow in both of its arms and slightly depressed, but the depression and pent-house appearance are not so marked as on the other side. The retrocentral fissure (postcentral) is poorly outlined. The medioccipital and suboccipital gyres, with their corresponding fissures, are well defined. The calcarine fissure is crossed by a bridge below the surface. The occipital, calcarine, and hippocampal fissures show the same tendency to confluence as on the other side, but the hippocampal is separated from the other a little more completely, although by a depressed gyre. The collateral and subtemporal fissures are

[1] This fissure will be more fully discussed when the brain of Taylor is exhibited.

strongly outlined, as are also the subcalcarine, subcollateral, and subtemporal gyres. The cuneus in this, as in the other hemisphere, is small. The supertemporal, or parallel fissure presents a very unusual appearance : instead of being, as it is commonly, a long, deep, unbridged furrow, it is divided about the middle of its infra-Sylvian portion into two parts by a comparatively large gyre. The posterior extremity of its anterior division runs superficially into the Sylvian. The supertemporal gyre is unusually narrow in its anterior and posterior portions. The angular gyre is also narrow. The subtemporal fissure is long and well defined, reaching into the occipital lobe. Wernicke's fissure is small and not well defined. The supercallosal fissure is well marked, but is separated into two parts, the anterior portion forming a fissure similar to the posterior, giving the appearance on the median surface of two supercallosal fissures, one above and somewhat anterior to the other. The inflected, the precuneal, and the amygdaline fissures of Wilder are well shown in this as in the other hemisphere. The inflected fissure in particular forms a deep indentation. The precuneus is of fair size, and is strongly marked with fissures.

In brief, the most striking indications of inferiority in the brain of Burk are the comparatively simple type of the frontal lobes ; the partial exposure of the insula ; the confluence of the right central with the Sylvian fissure ; the confluence, almost complete on the right and less perfect on the left, of the calcarine with the hippocampal fissures ; the ape-like similarities shown by the annectant gyres of both sides ; the shallowness, shortness, and complete bridging of the right calcarine fissure ; the imperfect angular gyre of one side ; and the simplicity of the temporo-occipital and parieto-occipital regions.

A cast was made of the skull of Burk, and this showed, among other things, that the right anterior, middle, and posterior fossæ were more capacious than the left. The calvar was also decidedly asymmetrical, the left side being more capacious than the right.

The confluence of the right central with the Sylvian fissure in this brain is worthy of particular attention. Benedikt claims that in thirty-eight cerebral hemispheres he found this complete union eighteen times. These results are startling, when it is considered that prior to the observation of Benedikt very few instances of confluence of these two great fissures had been reported—among others, one by McDonald, Parker, and myself respectively. In Zernoff's collection of one hundred brains, referred to by Benedikt, it occurred in but one instance. Ecker states that he had never seen an example. Since the publication of Benedikt's work, Osler has reported that out of sixty-three hemispheres, from thirty-four individuals, he found the central fissure communicating with the fissure of Sylvius three times completely and seven times incompletely.

The second brain is that of a man named Joseph Taylor,

who killed one of his keepers while serving a term in the Eastern Penitentiary, Philadelphia. He was defended on the plea of delusional insanity, but was convicted of murder in the first degree and hanged. Dr. H. C. Wood, H. Preston Jones, and myself, were retained as medical experts for his defence. He was, I believe, a delusional monomaniac of the criminal or prison type. His delusions chiefly had reference to the putting of injurious or poisonous medicines into his food or drink, and to persecution and bad treatment by the warden, keeper, doctor, and prison officials generally. Articles upon his case have been published by Dr. Wood and myself.[1]

This brain was first exhibited and verbally described by Dr. A. J. Parker, at the Philadelphia Neurological Society.

In *The Polyclinic* for September, 1886, a plate is given, showing four views of this brain.

The brain of Taylor shows, in the first place, a difference in bulk and in the general appearance of the two hemispheres. The right hemisphere is shorter and higher than the left. The fissural and gyral conformation in general is of comparatively simple type, both hemispheres showing many ape-like and fœtal similarities.

In the *right hemisphere* the Sylvian fissures is more vertical in direction than is usual; both its anterior and posterior arms being well defined. The insula is barely exposed at its anterior part. The central fissure is unbridged, not confluent, and very slightly sinuous. The precentral and postcentral gyres are moderately well developed; the precentral fissure is confluent with the Sylvian and also with the subfrontal. The entire frontal lobe has an unusual simplicity of aspect, and it is so fissured in a sagittal direction as to give, like one hemisphere of Burk's brain, an appearance of four gyres. The superfrontal fissure is straight, and clearly defined, with a larger posterior vertical secondary branch. The subfrontal fissure is also well defined. All the frontal gyres are comparatively simple, with short secondary markings. The parietal fissure, beginning close to the Sylvian, extends uninterrupted upward and across the parietal lobe into the transverse occipital of Ecker. Its beginning portion is so developed as to constitute a well-marked postcentral fissure. The marginal gyre is small and narrow. The occipital fissure is wide on the median surface, and notches also the lateral surface very distinctly. The superior external annectant gyre (paroccipital of Wilder, superior occipital of Ecker) is well up to the brain level. The superior in-

[1] JOURNAL OF NERVOUS AND MENTAL DISEASE, vol. xi., No. 4, Oct. 4, 1884, and *The American*, vol. ix., p. 88.

ternal annectant and inferior internal annectant gyres (gyrus cunei), can both be distinctly and separately made out, although well below the surface of the cuneus and præcuneus; they arch downward acutely with longer posterior than anterior arms. This development of the inferior internal annectant gyre causes a partial separation between the occipital and calcarine fissures. The calcarine fissure extends to the extremity of the occipital lobe, communicating in front imperfectly as just stated, with the occipital, but completely with the collateral fissure, giving this portion of the brain a most unusual appearance, as if the whole of the occipital and occipitotemporal regions, on the median and lower surfaces, were cut off from the parts of the brain anterior. The supertemporal fissure is well marked and has a short posterior vertical arm, which terminates just below the posterior extremity of the parietal fissure. The meditemporal fissure is short. This fissure communicates with a very well-defined, almost vertical fissure, which separates the temporal from the occipital lobe.[1] The supertemporal as well as the meditemporal fissure communicates with this fissure, the vertical extension of the supertemporal fissure being, in fact, continuous with it. The orbital fissuration is radiate, showing four radii, and one secondary fissure between the orbital and the olfactory. The olfactory fissure merges distinctly with the basisylvian. With the exception of the collateral fissure, which communicates with the calcarine, the lower portion of the temporal lobe is difficult to describe, being atypical in its markings. The supercallosal fissure is continuous, its posterior arm turning up just behind the central fissure and strongly indenting the median edge of the hemisphere. Above the supercallosal fissure the median surface of the hemisphere presents a succession of shallow parallel, chiefly vertical markings, the vegetative repetitions of Parker.

In the *left hemisphere*, as on the other side, the insula is barely exposed in front. The central fissure is straight, uninterrupted, and separated from the Sylvian or sagittal fissures, but is superficially confluent with the superfrontal fissure. The precentral gyre is wider and more fully developed than the corresponding gyre of the other hemisphere. The precentral fissure is well defined, but not confluent with the Sylvian as on the other side. The markings of the frontal lobe are simple, although a little more complicated than upon the other side. The four frontal gyres of Benedikt are not so readily made out. The superfrontal fissure is well marked. The orbital fissuration is more irregular than in the right

[1] This fissure is what has been termed by Benedikt Wernicke's fissure; and Benedikt says that the meditemporal gyre is always situated in front of it. He describes the fissure as lying in an imaginary arc situated between the occipital fissure above and the meditemporal fissure below. It corresponds to the inferior portion of the external perpendicular fissure in the ape. Parker, in an unpublished paper, proposes to call this the occipito-temporal fissure. It is better defined in the brain of Taylor than in any human specimen that I have ever seen, easily demarcating the parietal and temporal lobes from the occipital upon the lateral aspect of the hemispheres.

hemisphere: instead of a distinctly radiate or H-shaped appearance, one deep fissure extends antero-posteriorly with two posterior arms, giving a Y-shaped fissuration, the arms of the Y being towards the Sylvian fissure. The parietal fissure, beginning between the central and the posterior arm of the Sylvian, extends backward to the transverse fissure of Ecker, with only one bridge well below the surface at its anterior part. The marginal gyre is small. The occipital fissure is well developed. The superior external annectant gyre is level with the brain surface. The inferior internal annectant gyre can be barely made out. The calcarine fissure does not communicate with the collateral as on the other side; it is not confluent with the hippocampal. The cuneus is distinctly smaller and of a different shape from the cuneus of the right hemisphere. The collateral fissure and its adjoining convolutions can be better made out than on the opposite side. The supertemporal fissure is well defined. On this side, as upon the other, Wernicke's fissure is clearly outlined, communicating with the supertemporal. The supercallosal fissure of this hemisphere is continuous. Above it are both horizontal and vertical markings, somewhat different in appearance from those of the opposite side.

The third specimen, the brain of a feeble-minded youth, was obtained from one of three cases of progressive muscular degeneration, described in a paper presented by Dr. I. N. Kerlin and myself to the American Medical Association in 1879, and printed in the transactions of the Association for that year. The brain exhibits, among other things, the superior external *pli de passage* of one side sunk below the surface, the next fold partially covering it.

I have made some studies in cerebral morphology at the Pennsylvania Training-School for Feeble-Minded Children. Photographs of some brains there studied I present, through the kindness of the superintendent, Dr. I. N. Kerlin. Dr. A. W. Wilmarth, assistant superintendent of this institution, has published, under the title of *Notes on the Pathology of Idiocy*,[1] descriptions of the arrangement of the gyres and fissures of several idiot brains.

In the arrangement of the gyres some of the most striking peculiarities were found. In congenital idiocy and imbecility, particularly when of a low grade, the difference in gyral arrangement was great. Simplicity, especially in the frontal regions, was the rule. The frontal lobes were often

[1] *Alienist and Neurologist*, vol. vi., No. 3, July, 1883.

narrow and pointed. Wilmarth says of one variety of brain, that it may be well called the atypic brain. An extreme case of this kind was a brain in which the callosum was wanting.

Dr. L. U. Beach was executed at Hollidaysburgh, Pennsylvania, February 12, 1885. His brain was sent by Dr. Smith, of Altoona, to Prof. James Tyson, of the University of Pennsylvania, for examination. When it arrived the lower portions of the brain were much softened and decayed, so that it was impossible to study thoroughly the lower temporo-occipital and basal regions. The other portions of the brain had also undergone some change, but were sufficiently well preserved to allow of a study of fissural and gyral arrangement by Drs. Tyson, Parker, Lloyd, Dercum, and myself. The case of Beach attracted much attention in Pennsylvania and throughout the country, and papers on it have been published by Clark Bell, Esq., Dr. E. C. Mann, and others. The crime for which he suffered the death penalty was the killing of his wife in 1884. The history of the patient's family was filled with evidences of imbecility and insanity. Strong evidence as to his insanity was brought at his trial and subsequently. The circumstances of the commission of his crime were extraordinary. Beach walked to the house of his wife's brother and told him that he had killed his (Beach's) wife, who was found with her head nearly severed from her body, and with other deep cuts. Beach said that he only remembered that an immense snake seemed about to attack him, and that he was compelled to cut off its head.

The *right hemisphere* of the brain of Beach showed the fissure of Sylvius and central fissure separated by a very small gyre. The insula was exposed and large, having six gyres and five fissures. The frontal lobe was of simple type. The subfrontal fissure had two fully developed bridging gyres, a fœtal condition. The super and medifrontal gyres were well defined ; the subfrontal, narrow and slightly arched ; the secondary markings of the frontal lobe unusually simple ; the precentral gyre simple and unusually narrow. The precentral fissure ran superficially into the Sylvian and more deeply into the subfrontal. The orbital surface presented a single fissure parallel with the olfactory, instead of the usual triradiate or H-shaped fissure. The upper extremity of the

occipital fissure almost merged with the parietal. An unusual condition was an easily-determined superior internal annectant gyre arching inwards and downwards on the median surface.

According to Bischoff, as stated by Parker, the superior external and the superior internal annectant gyres are identical. Ecker, however, opposes this view, holding that in the brains of various apes both are clearly developed, and that also not infrequently in man a gyre which rises at the posterior extremity of the precuneus with the superior external gyre runs backward in an arch convex inward and downward, while the superior external gyre makes an arch outward, the two gyres again coalescing in the cuneus. I was inclined to adopt the view of Bischoff, not having observed, in any specimens examined, a clearly-defined superior internal gyre until the specimen from Beach was observed.

The inferior internal annectant gyre was feebly developed. The parietal fissure was long and well marked, having a single small and bridging gyre, and communicating with a moderately well-developed transverse occipital fissure (paroccipital of Wilder). It communicated superficially with the Sylvian fissure. The postcentral fissure was large and well defined, and the parietal gyre of nearly the average size. A well-marked secondary fissure crossed the marginal gyre vertically, branching from the parietal fissure and communicating superficially with the supertemporal. The supertemporal fissure was remarkable in that it was completely confluent with the Sylvian fissure, extending from the point of the confluence as a deep, well-defined trench backwards and upwards nearly to the sagittal fissure, crossing and confluent with the parietal.

In the *left* cerebral *hemisphere* the Sylvian fissure, which was more nearly vertical than usual, passed upwards to within about an inch of the sagittal fissure, a remarkable aberration. The central fissure presented, at the junction of its middle and lower thirds, a well-marked bridging convolution. The insula could be readily seen without separating the margins of the Sylvian fissure.

Edward Ford, a negro, about sixty-five years old, was sentenced to death for murder, but was never executed, successive governors, for some reason, refusing to sign the warrant for his execution. Two or three years before his death, and subsequent to the appearance of some paralysis of the left side, he was pardoned, and soon after sent to the nervous wards of the Philadelphia Hospital, where he remained until the day of his death. He was a quarrelsome, peculiar man, in the habit of drinking, before the commission of his crime. During his stay in the hospital he was clearly out of his mind. The paralysis of his left side became quite profound before his death. The brain

of Ford was exhibited at the Philadelphia Pathological Society in 1882, and a brief account of it published in the *Philadelphia Medical Times* for May 20th, but its morphological peculiarities were not detailed.

I was assisted in my examination of this brain by Dr. James Hendrie Lloyd.

In the brain of Ford the insula was visible on both sides without pulling apart the Sylvian fissure. The Sylvian fissures were more nearly horizontal in their anterior portions than is usual in the white brain.

I will describe first the *left hemisphere*. The superfrontal fissure was deep, well defined, running from the anterior tip of the hemisphere backward to the precentral gyre, with one bridge at about the junction of the first and second thirds. The medifrontal fissure was less well marked, although easily recognized, and was nearly confluent with the first about its middle, the two being separated only by a shallow bridge. The precentral fissure was well defined and crossed at right angles by a large secondary fissure. The presylvian, or ascending branch of the Sylvian fissure, was also well marked. The gyres of the frontal lobe were more readily defined than is usual in white brains. The central fissure was a deep, unbridged, comparatively straight furrow, not confluent with any other fissure. Both central gyres were simple in type. The parietal fissure ran from the junction of the lower and middle thirds of the postcentral gyre, curving backwards to a point only about one half inch from the extremity of the occipital lobe. It had only one slight, bridging convolution which did not come to the surface. The superior external annectant gyre (paroccipital of Wilder) was depressed. The transverse occipital fissure was well developed. The supercallosal fissure was divided almost exactly into thirds. In the paracentral lobule was a deep Y-shaped depression. Above the supercallosal fissure ran a series of short, parallel fissures almost the entire length of the supercallosal itself. The supertemporal fissure was a deep, straight furrow bending up abruptly one fourth inch back of the Sylvian fissure. The meditemporal fissure was unusually well defined, as indeed were all the temporal fissures and gyres. The hippocampal communicated with the calcarine fissure.

The *right hemisphere* showed a marked pathological condition—one of atrophy. The postcentral gyre of this side was only about one half the width of that of the left side. The precentral and the parietal gyre were also visibly atrophied. The cerebellar hemisphere of the opposite side was also uniformly atrophied. This pathological condition may have rendered somewhat uncertain the morphological comparisons. The medifrontal fissure was unusually well defined, running back without a bridge nearly to the paracentral. The subfrontal fissure was also straight, short,

and better defined than usual. The central fissure was separated from the Sylvian by a narrow, shallow bridge. The parietal fissure, unlike that of the left hemisphere, at its anterior extremity, formed, an unusually well-marked postcentral fissure. There was an unusual depression or absence of gyral substance in the parietal gyre. The supercallosal fissure of the right side was not divided into thirds as upon the left, but was a deep, continuous furrow with a very slight bridge, far below the surface, near its middle. On the whole this brain presented, in a marked degree, the peculiarities observed by Parker in his study of negro brains, with a more than usual atypical a symmetry of the hemispheres.

The single hemisphere of a negro brain which I exhibit is one of a number examined and described by Parker,[1] who has made a special study of the cerebral convolutions of the negro brain. He has found, as in the Hottentot Venus of Gratiolet and the Bushwoman of Marshall, that the island of Reil was distinctly visible in the normal condition of the brain. This was well marked in some negro brains, and therefore he justly concluded it to be characteristic of the race. In the brain of a mulatto the same peculiarity was present, although not so marked. This condition is not found in the adult brain of any monkey described up to the time of Parker's contribution. The Sylvian fissures of the negro brain, instead of ascending in the usual oblique upward and backward direction, were found by Parker—again as in the Bushwoman and Hottentot Venus,—to assume at their anterior portion a horizontal direction, the posterior portion taking a direction nearly perpendicular to this. He found the average length of the Sylvian fissure in 19 white brains to be $3\frac{5}{8}$ inches; in 13 negro brains, 3 inches; in the mulatto, $3\frac{1}{4}$ inches. The frontal gyres were, as a rule, simpler and better marked in the negro than in the white, the lower seeming to be as well defined as the upper. The central fissure was simpler, straighter, and less undulating in the negro than in the white, its bounding gyres partaking of the same character. The parietal fissure was found remarkably well developed and more tortuous than in the white. In five instances it was not bridged over at any point, presenting the same appearance as in the higher apes, except that

[1] "Proceedings of the Academy of Natural Sciences," Philadelphia, vol. xxx., 1878, p. 11.

its direction was more curved. In six cases only one bridging convolution existed, and in the remaining two one well developed and another imperfectly developed. In the mulatto this fissure was continuous, but much more undulating and tortuous than in the negro. The parietal and marginal gyres were simpler and less marked with secondary fissures than the white. In one negro brain, the specimen which I exibit, the marginal gyre was entirely absent, the brain showing a deficiency in this region greater in proportion than is found in apes. The parietal fissure in this case ran directly into the upper end of the Sylvian. In the occipital lobe he found that the negro brain displayed its ape-like peculiarities to a greater extent than in any other portion of the cerebral surface. The superior external transition or connecting gyre of the occipital lobe he had found invariably smaller, less developed, and simpler in the negro than in the white.

In all of the specimens atypical symmetry as to gyral and fissural development is present. Undoubtedly in normal brains, even of individual of great capacity, marked asymmetry is sometimes found. Some of the features which may constitute atypical asymmetry are the following: the existence of a Sylvian fissure shorter on one side than the other, both absolutely and comparatively, and also a more vertical direction of the fissure on one side than on the other; greater exposure of the insula on one side, with marked differences in the development of its fissures and gyres; confluence of the central fissure with the Sylvian on one side only, and great tortuosity, or bridging of the former fissure in one hemisphere; unusual narrowness, straightness, or complication of the precentral or postcentral gyres on one side; marked differences in the simplicity or complexity of the frontal lobes; great simplicity of the orbital surface on one side; difference in the parietal fissure as to length and interruptions; a smaller parietal, or marginal, or angular gyre on one side; very great differences in the degrees of confluence and interruption of the fissures in general; exceeding great length vertically of the supertemporal or parallel fissure on one side; unusual differences in the size of the precuneus and cuneus.

The autopsy of Guiteau showed, among other things, well-marked atypical asymmetry of the hemispheres. Great differences were found between the insulas and between the fissural and gyral conditions of the median surface of the two hemispheres. The right postcentral gyre was much narrower than the left. The parietal region was flattened, and the right paracentral lobule was quite small as compared with the left, which was large and well developed.

The comments of Spitzka[1] have a certain applicability in the explanation of the peculiarities of the specimens here described.

"Asymmetry of the character found in Guiteau's brain," he says, "is congenital, and differs from the atrophy of one hemisphere, which is known to be the result of various acquired disease processes. In the latter case, as long as any convolutional type can be distinguished in the smaller hemisphere, it deviates no more from that of the opposite side than one hemisphere usually differs from the other in normal subjects. And when a destructive lesion is associated with the atrophy, the former manifests its past existence by unmistakable signs. It is to be further stated that a lack of development of one hemisphere, manifesting itself in a reduction of important gyri and lobes, and combined with an atypical course of a fundamental fissure, must have its origin in an aberrant development instituted at an early period of fœtal life. It is this fact which lends such atypical developments their peculiar significance. Destructive lesions may occur in the postnatal period, a whole hemisphere may be destroyed without necessarily producing insanity; in fact, the further advanced towards maturity the individual is, the better able is the cerebral mechanism to endure extensive injuries without reacting in the direction of pronounced mental derangement. But the nearer the time of the injury approaches the natal period, and still more true is this if it anticipates that period, the less likely is it to leave the mental mechanism intact. An aberrant development is in this respect analogous to an early injury, but with this difference, that while the injury is limited in its effects to the functions of the special region destroyed, irritated, or hampered, it is impossible to say how deeply the error in development, manifesting itself in a surface anomaly of the hemispheres, may involve that subtle architecture of the transmitting and associating tracts which a newer and rational psychology teaches us to regard as the basis of the logical mechanism. It is in harmony with this explanation that aberration in development, of the kind discovered in Guiteau's

[1] *American Journal of Neurology and Psychiatry*, vol. i., 1882, p. 386.

brain, has not yet been found in others than persons of unsound mind. The unsoundness associated with such analogous errors of brain development covers an extensive range, including 'original,' or congenital and hereditary, imbecility, and chronic insanity with systematized delusions, morbid projects, and moral perversion. The most reliable and thorough investigators, such as Starke, Schuele, Sander, Muhr, and Jensen, have noted the presence of convolutional anomalies and asymmetry in such subjects; and in a work of no less prominence than Ziemmsen's Cyclopædia, the author of the article on insanity expresses it as his belief that the only finding in the brains of constitutional lunatics of monomaniacal tendencies (Primär Verrückte) which promises to establish a relation between the insanity and the state of the brain, consists in such architectural anomalies."

Striking differences can be detected between these brains and what is commonly regarded as the average normal human brain, and the brain of high development. Here we have the brain of Burk, recognized by all as a delusional lunatic; of Taylor, whose life was a sickening tale of lust and violence; of a feeble-minded youth, the victim of a form of neuro-muscular degeneration; of Beach, who showed evidences of imbecility, paranoia, and epilepsy; and finally of Ford, an ignorant negro, with a record both of criminality and insanity. In all these brains are points of affinity which put them in a class together; in an emphatic sense they represent the brains of low and aberrant development. The specimens taken from individuals of the white race exhibit negro, simian, and fœtal similarities, resemblances, and reversions in an unusual degree. With reference to the criminals, or so-called criminals, of the series, it may be said that to conclude from such a study that the brains of all criminals are distinctive, that we have a "criminal type" of brain which can even be separated from that found in cases of idiocy, imbecility, paranoia, and other illustrations of psychical degenerative states, is not a truly philosophical generalization. The proper ground to take is probably that indicated by Kiernan and others, namely, that between the true criminal type, the idiot, the imbecile, and the paranoiac, the psychologicalrel ations and their anatomical bases are intimate and close.

The truth is that some idiots and paranoiacs have much

in common. Benedikt's position may have been partially misunderstood by some of his severest critics. What he does say in his preface to his book on the *Brains of Criminals*, is that crime is in no way analogous to monomania; that it results from the physical organization as a unit, and that its particular form of expression is determined by social circumstances.

To attempt to construct a theory as to the anatomical basis of crime is, in some respects, unphilosophical. Crime being technically the transgression of the law, criminals must be of the most diverse character. Almost any one may become a criminal under the stress of peculiar circumstances, and therefore he who would attempt, in a general sense, to establish a criminal type of brain might be led into gross error; but no matter what subdivisions of criminals may be made, a certain number will always be found who are criminals as the result of their organization, because of retarded, defective, or aberrant brain development. Whether even such criminals should be technically regarded as insane, is a further question; certainly all of them need not be so classified; more certainly many of them must be so classified.

Some authorities are strongly inclined to doubt whether studies of this description into the gross peculiarities of gyral and fissural development, or of any other gross abnormalities, can give us any result of value, so far as determining the mental type of the individual. Clevenger is inclined to doubt whether a special study of criminals' brains would afford any results apart from investigations among any other classes of men.

The error should not be on either side. On the one hand, we should not, with Benedikt, and his followers and admirers, fall into the error of supposing that we have an almost absolutely fixed type of criminal brain; on the other hand, we should not be too broad in our denunciations of those who look to a study of original organization for their conclusions as to the mental state and responsibility of those who commit crime. If we admit with physiologists and anthropologists that a certain number of the people of

this world are criminals as the result of inherited organizations, in that admission we show the necessity and the great value of studies into the conditions of brain development.

Dr. Johann Badik has for a number of years occupied himself with the question of the skulls and brains of criminals. His results were published in Virchow's Archives, and were summarized in 1884, by Dr. J. C. Wilson.[1] His investigations were conducted in the great Hungarian prison at Illava. Dr. Badik classified criminals into four typical groups, a division based upon the association of certain mental and moral qualities with definite abnormalities in the skull and corresponding lesions in the brain and its coverings. Anatomically the groups arranged themselves into: first, those with symmetrical skulls; and, second, those with asymmetrical skulls. Under symmetrical skulls he found, first, those with small developed skulls without pathological changes anywhere. These were simple-minded persons with evident and incurable lack of mental development and moral perception. Second, were those with skulls of medium development, with lesions of the brain and its membranes. These individuals were intellectually more developed; they improved mentally and morally; and their crimes were usually committed as the result of emotion, temptation, or necessity. Under asymmetrical skulls were included, first, those in whom the brain and its membranes showed no pathological changes. Badik forcibly asserts that the majority of the inmates of the prison presented this skull malformation. These were the incorrigible criminals, who were benefited neither by instruction, counsel, religion, punishment, nor any thing else. They belonged to the class of cases represented by some of the individuals from whom the brains here described were taken. Under asymmetrical skulls a second class of cases was found in which with the asymmetry were associated pathological changes in the brain and its coverings. These criminals had the conditions of faulty development shown by the last class, and, in addition, pathological changes. They were often epileptic;

[1] *Philadelphia Medical Times*, vol. xv., Oct. 18, 1884, p. 50.

their crimes had been committed for the most part, if not indeed always, in a state of perverted consciousness, and were remembered only as a dream.

In these last two classes belong those of whom Dr. J. S. Wight[1] strongly discourses as follows: "The concurrent and unanimous testimony of those who are, from their experience and their knowledge, most competent to judge, is: That the great under class of criminals have more or less defective organizations, especially as relates to their nervous system, and more especially as relates to their brain; that they are more or less deficient in moral sense, showing in this respect the lack of development or the results of decay, the best and last-developed sense, the moral sense, disintegrating first of all; that they are perversely wicked and indomitably inexpedient, committing crimes when doing right would be of more use to them; that they are as passionate as the wild beasts of the forests, and as restless as the ocean that heaves at every gust of the wind; that they are at war with mankind and ever in commotion with themselves; that they are, like the ship, beaten about by the storm—the ship without compass, rudder, or captain; that they are formed and fashioned by the hand of an evil genius, whose name is bad heredity, and whose hand-maid is ignorance; and that they cannot be very much reformed, and that their reformation ought to have been begun in their ancestors."

In concluding this paper I desire to make a few special remarks in connection with the consideration of the peculiarities of fissuration presented by the specimens exhibited. More particularly I wish to call attention to the views of Dr. A. J. Parker with reference to the vegetative repetition of cerebral fissures. Parker,[2] after considering the question whether the fissures of the cerebro-cortex are due to mechanical causes entirely, or represent lines of retarded growth, or arise through both of these methods, discusses the question whether we are to regard each fissure as produced by a distinct and separate process of formation, or

[1] *American Journal of Neurology and Psychiatry*, vols. ii. and iii., p. 135.

[2] "Proc. of Acad. Nat. Sci. of Philadelphia," vol. xxx., page 148.

that some of the fissures are only repetitions of fissures previously formed.

"In studying the cerebral fissures as presented in the brains of different animals," he says, "especially amongst the Carnivora and Ungulata, it had appeared to him that many of the fissures should be regarded in the latter light, that is, as vegetative repetitions. Viewed in this way, many difficulties in regard to the identification of homologous fissures in different brains disappear. According to the mechanical theory, a deep and distinct fissure having been formed, there would be a tendency to produce other fissures following the same general direction, and having the same general appearance, and depending for their formation on the one originally laid down. According to the view that fissures are the result of retarded cerebral growth, we may expect to find, especially in lower forms of brains in which much fissuration exists, vegetative repetitions of the same lines of growth. In either case the fissures which appear after the original fissure, and which follow its general contour, should be considered as belonging to one group with that fissure, and to be of secondary importance in relation to it. Hence, in many cases, instead of seeking fissures separately homologous to each other, we will be obliged to consider certain groups to be homologous to certain other groups, the number of separate fissures of which may be more or less numerous. Owen, in founding his nomenclature of the cerebral fissures in the Carnivora and Ungulata, gave a distinct and separate name to each fissure, and he endeavored to point out the homologue of each of these in different brains. If, however, we are to regard that at least some of these fissures are entirely secondary and to be considered as merely vegetative repetitions, then we must not seek, nor is it possible to find, homologues for each fissure, even in closely related brains."

Parker gives various illustrations of these vegetative repetitions found in his studies of the Carnivora and Ungulata. He also studied these fissures in monkeys and apes and in the negro. In all of his investigations the supercallosal or calloso-marginal fissure was particularly considered. In the

monkeys and apes, as in lower forms, he found a marked tendency in this fissure to split up in two or more similar fissures. In man it often consisted of several distinct parts. He had observed that the repetition of this fissure was especially regular and well marked in the brain of the negro. It is very observable in connection with several of the hemispheres studied in this paper; and supposing this view as to vegetative repetition of fissures to be generally applicable in a study of the cerebral surface, many examples can be found in various localities, more particularly upon the orbital surface and in the temporo-occipital region.

This view of the subject of cerebral fissuration is important in connection with the question of new fissural integers, to which Prof. Wilder has recently given so much attention. To cite but one example, Wilder regards the supercallosal fissure, which Parker has carefully studied, as not being a good fissural integer. At some future time I hope to be able to consider this question of new fissural integers more at length.

Although in the paper, partly from necessity and partly from choice, I have confined myself to a study of gyral and fissural arrests and aberrations, I wish emphatically to call attention to the fact that it is not by a study of fissures and gyres alone that the entire truth can be determined with reference to the questions of capacity, intelligence, and responsibility of the individuals whose brains are the subjects of examination.

In our more perfect studies we must go further and deeper than this. As shown by Turner, we must study the depths of fissures, the thickness of gray matter, the quality of tissues. The microscope must lend its help. It would be a most interesting, valuable, and thoroughly scientific investigation to take a human brain of the kind exhibited, and with it make an elaborate painstaking investigation from every point of view. Such specimens should be prepared carefully by a method of hardening which would allow, subsequently, both microscopic and megascopic investigation to be thoroughly made. The plan of study should be to first compare weights and general

features of the hemispheres; to examine lobes, fissures, and gyres; to study questions of simplicity and complexity in fissural and gyral development; to ascertain the relations and degrees of development of annectant gyres, and all other special points in cerebral topography. Great sections should be prepared with large microtomes, and these should be examined and comparisons made of sections of different hemispheres and different positions in the same hemisphere. Differences in ventricles, the condition of the ganglia, capsules, and corpus callosum, the relations of the cerebellum to the cerebrum, the size and development of the peduncular tracts, and of the tracts and centres in the pontile and oblongatal regions should be carefully studied. Such careful and elaborate work, although it required weeks, or months, or years, for its thorough accomplishment, would be of inestimable value to anthropology and medicine, and would reward with abundant laurels the patient and careful investigator.

III.

PRELIMINARY STUDY OF A CHINESE BRAIN.[1]

By A. J. PARKER, M.D., and CHAS. K. MILLS, M.D.

The brain was removed from a middle-aged Chinaman of the coolie class.

The first peculiarity observed is the more than usual outward and upward sloping of the orbital surface of both hemispheres, which is probably in correlation with the upward and outward inclination of the palpebral orifice in the Chinese.

Right hemisphere.—The Sylvian fissure shows no special peculiarity. It seems to make shallow connection with the vertical arm of the supertemporal, but this is a superficial appearance. The central fissure is nowhere confluent. The insula is not exposed. The precentral fissure is well defined, communicating below with the Sylvian, and above with the subfrontal. The medifrontal fissure is not distinctly out-

[1] This brain was first exhibited and described by Dr. A. J. Parker at the Philadelphia Neurological Society.

lined, and there are not, therefore, four distinctly demarcated frontal gyres. The superfrontal fissure is well defined in the posterior two thirds of its usual length. The subfrontal is clearly marked, and communicates, as stated, with the precentral. The primary fissures of the frontal lobe present a curious regularity of undulation. This brain generally has a peculiarity of this kind difficult to describe. The orbital fissuration is peculiar. About the middle of the orbital surface is a large H-shaped fissure,[1] the cross bar of the H being sagittal, and its lateral arms much curved. In front of this fissure is a small fissure which repeats the cross bar in one of the arms of the H. Posterior to the H-shaped fissuration are three shallow sagittal markings. The parietal fissure is somewhat perplexing. The so-called retrocentral fissure (post central of Wilder), which is usually regarded as a vertical elongation of the anterior extremity of the parietal, forms a fissure extending across the lobe from the Sylvian to the sagittal fissures, which is parallel with and of about the same length as the central fissure, as clearly demarcating the postcentral gyre behind as it is demarcated in front by the central fissure. Taking the lower half of this postcentral fissure as the beginning of the parietal, the latter extends a short distance backwards and is then interrupted by a well-developed gyre. It then has a posterior branch which is joined in front by the supertemporal and runs behind into the transverse occipital.[2] This posterior extension of the fissure is not well shown in the figure. The parietal gyre is of moderate size; the marginal gyre is small The occipital fissure is well outlined, and is in communication with the calcarine, but not with the hippocampal. The cuneus is of peculiar shape, and is crossed by a fissure which runs across the median edge of the hemisphere. The first occipital gyre of Ecker (paroccipital of Wilder) is developed to the common brain level. None of the so-called *plis de passage* can be made out as such. One of the remarkable

[1] Wilder has proposed the general name *zygal* (yoked) for all H-shaped fissures like the orbital and his paroccipital.

[2] The terms "transverse occipital," and "parietal" (interparietal") have been used as employed by Ecker, as the views of Prof. Wilder with reference to these "fissures" are still under discussion.

peculiarities of the brain is the great extent of the supertemporal fissure of this hemisphere, which, beginning near the anterior extremity of the temporal lobe, passes backwards and upwards across the entire extent of the parietal lobe and over the median edge of the hemisphere for the distance of half an inch, terminating in the precuneus just in front of the occipital fissure, and merging in its course, as already stated, with the posterior branch of the parietal fissure. This at least seems to be the proper interpretation to put upon the fissuration, although a backward extension of the horizontal portion of the fissure might be regarded by some as its true continuation. The subtemporal and collateral fissures are well defined. The supertemporal gyre is of good size. Wernicke's fissure, *exoccipital* of Wilder, *anterior occipital* of Schwalbe, is well shown, communicating in front with the supertemporal. A well-marked fissure passes across the occipital lobe just below its shelving extremity. The amygdaline fissure of Wilder is present. The supercallosal fissure is not continuous; it is divided into two distinct parts, both having the same shape, but the anterior is a little longer than the posterior. The anterior bends upward in front of the line of the precentral fissure; the posterior, just behind the central. Some might regard this fissure as divided into three or even four parts. The conditions as to fissuration present on the whole upper portion of the median face of this hemisphere illustrate Parker's "vegetative repetition" of fissures. The inflected fissure of Wilder here appears to be simply one of these vegetative repetitions. The precuneus is crossed by a fissure deep and well defined, which passes far over into the lateral aspect of the hemisphere.

Left hemisphere.—The Sylvian fissure is longer in its horizontal arm than the same fissure of the right hemisphere. Its ascending branch, as on the other side, is illy defined. The central fissure, clearly marked, is non-confluent. There is a depression about the junction of the middle and upper thirds of the postcentral gyre; the gyre is poorly developed in this position, as also at a point near the junction of the middle and lower thirds. The precentral fissure has a

deep communication with the Sylvian. The superfrontal fissure is deep and well defined, but neither a medifrontal nor a subfrontal are well outlined, the markings being as positive in a transverse as in an antero-posterior direction. The frontal lobe is comparatively little elaborated. The orbital surface presents one H-shaped and several small, irregular fissures. The parietal fissure, passes uninterrupted across the parietal lobe to the so-called transverse occipital, beginning close to the Sylvian fissure, just in front of its posterior bifurcation. The parietal and marginal gyres are of fair size and development. The occipital and calcarine fissures and cuneus are similar to the homologous parts on the other side. A fissure begins in the fork formed by the posterior extremity of the calcarine fissure and reaches around the tip of the occipital lobe. The supertemporal fissure is very well developed, but has not the great extent of the same fissure in the other hemisphere, its posterior vertical arm stopping a little short of the parietal fissure. The subtemporal and Wernicke's fissure can be well made out. The collateral fissure is well marked, but turns upward across the temporal lobe about its middle. The supercallosal fissure is long, continuous, and typical in appearance, very different from the other side, but the surface of the hemisphere above it presents a series of short, nearly vertical, vegetative repetitions. The amygdaline and inflected fissures of Wilder are present.

LESION OF BOTH TEMPORAL LOBES, WITHOUT WORD-DEAFNESS OR DEAFNESS.*

By LANDON CARTER GRAY, M.D.

IN June, 1885, there was admitted to the Hospital for Nervous and Mental Disease a man of about fifty, of whom his friends related that he had had a severe convulsion a few days before, this convulsion having lasted over an hour. Tested by the proper instruments of precision—such as the dynamometer, the æsthesiometer—no abnormal motor or sensory symptoms were detected. He spoke distinctly and expressed himself correctly. His pupils were normal, both in appearance and in their reflex responses. Opthalmoscopic examination revealed nothing abnormal. No cephalalgia. But there was a most remarkable and absolute loss of memory for all events since about the date of his convulsive attack, and also a slight, not very marked, loss of memory of most events of his previous life. He could not remember any thing from one moment to the other. For example: I would go into his room and ask him why he had hammered so noisily upon the door. He would answer that it was because he wanted to get out, as he could see no reason for his confinement. "Where am I? why am I detained here? what have I done?" I would explain to him that he had been sick, had lost his memory, and was therefore shut up in his room for his own good. Then he would beg my pardon, express regret that he had disturbed anybody, and would earnestly promise to bear my explanation in mind. I would then

* Read before the American Neurological Association, July, 1886.

leave the room, and by the time I was twenty feet away the hammering on the door would be renewed with all its former vigor. Returning to his room, he would be asked the same questions, make the same explanation, have the same regrets expressed, and have the same assault on the door echo through the building before I had been out of the room a minute. Finally I made him write these words on a large sheet of paper, in a large hand: "Mr. B. has promised Dr. Gray that he will not hammer on the door, as this is a hospital for sick people." I made him pin this paper on the wall just in front of where he generally stood or sat. I then left the room. The hammering occurred as usual. Going back to his room, I pointed out to him the sheet of paper, upon which the ink was still wet. As he read it, his countenance expressed the utmost surprise. "Did I write this? When?" he inquired. And so it went on from day to day. At first he could not remember me or my name; then he began dimly to remember my face, without being able to recall when he had seen it; and finally, after the lapse of several weeks, during which I saw him almost daily, he was able to recall my name after long meditation. It was the same way with his attendants. He promptly named all objects that he saw. He recognized the meaning of sounds, such as those of language, of a machine-saw, snoring, wagons in the street, etc. His sense of smell was perfect. His mental faculties, other than the peculiar loss of memory, seemed to be intact. Of course, there was a certain timid, questioning air, as of a man who has lost his self-confidence, such as he expresses in his letters which I am about to read. Although I could obtain no positive evidence of any specific disease, his life, as I came to learn, was certainly such as to have exposed him to great danger of it. For a year or two past he had suffered considerably from insomnia and occasional slight headache, and during this time he had been growing to be fretful, although not sufficiently so to create remark or to interfere with his avocation, which was that of a clerk in an insurance office.

It soon became impossible to keep him in the hospital, as he hammered so persistently on the door of the room to

which the overcrowded condition of the hospital rendered it necessary to confine him. Dr. E. E. Smith, of the Morris Plains Asylum, very kindly undertook to take charge of him, in view of the great interest of the case. Thither, then, he went, in the latter part of June, 1885. The following letters will indicate his mental condition until within a week of his death:

July.

read the other 1st, I don't know when I wrote this.

DEAR P.

To begin with, this pen is bad. There is a good deal written to you in the hierogliphics, but I 'll just say a few words here right straight out of my little head. For the date of this my memo. says 13th July but you 's better try the post mark for that. I, in a former letter I think, asked you to bring a pr of scissors to trim down my whiskers a little. if you only knew what a bad pen this is, you 'll bring me a fine Gillott, too. The scissors not to be left here. The stamped envelope however goes ahead of everything else, and is a good idea on your part.

Bless " heart)

The 1st remark must explain the blots. The umbrella is exquisite and the Straw Hat is simply lovely. The Morristown paragraph of your letter is cut out and placed in the lining of my hat, where I can see it. (after breakfast) I think it is the 10th July. I received a Box, with HAT, SOCKS & HANDKERCHIEFS, and I also got a NICE LITTLE UMBRELLA. I will not attempt to make any excuses for not sending you more letters, and will only say that I have torn up a good deal written to you. I have yours of June 25th & July 5th. Next autumn I hope to be nearly well, perhaps quite. I have no doubt about your taking me out of this hospital, as soon as it is proper for me to leave it, and therefore I am willing to abide my time, and my conscience reproaches me not for any wrong doing as the cause of my incarceration. " There 's no place like home," and I now feel that I have one yet. (later) same day I think. I 'm lying on a sofa, and have just done the inky erasing on the other side of this leaf. when I alluded to your Morristown paragraph overleaf, I certainly told a fib or else my imagination deceived me, for the paragraph in *re*—your going to visit Mrs. C., is not cut out at all nor anything else. The pencil is decidedly the best for me to conclude this letter & it can only be concluded at intervals, so peculiar is my nervous condition.

late.

I don't know when I wrote this, but I'll send it.

P. The date I know not. It is 5 o'clock A.M. a little later and I am in bed, and have been awake some time. While there 's life there 's hope, and mine is preserved by it. I do not know how many times I have made the same remark. The recent past is to

me as nothing. I find myself here in Morris Plains Asylum, I think, and know not how long I 've been here, but remember that you placed me here, by the advice of Dr. L. Carter G., and, though I may repeat, I 'm contented, if you did it. I do not remember when I saw you last, but I know you will come when you can.

September.

DEAR P.

I have yours of Sep. 20—85 dated No. 124—I also have Sep. 6th from you. As in the first mentioned you give me, I stopped to make as near as I can, a quill pen, out of a tooth pick. You can see that it is a poor success. This pen is a split tip of a tooth pick to which a rubbing on the stone window sill has given a point, and which is stuck in my fountain-pen handle, with which I am writing, though I cannot shade with one nib—as the pen is only *one* split tip of a tooth pick. Now, P., I 'm in my room, and I have before me your letter of the 20th Sep. from the house of your old friend Mrs. P., where I shall expect to address you all winter—and I 'm glad you are where I 've been to see you and I hope I may yet see the day, and a not far distant one, when I may either visit you again, or, better, live with you. "All ready + + + for a walk" has sounded—after the three crosses I am returned from the walk & you see what a different hand I 'm writing. As I have no blotting paper (of which you might bring me a piece) I find it *so* very convenient to dip the point of the tooth pick, with which I am writing, in the blot, that you must not be surprised hereafter if I make blots on your letters, for tooth picks do not take up the ink of my fountain pen as well as the steel pen does. In fact I 'm almost ashamed to send even my own sister such a blotted letter. So, P., a pen would be crushed in a letter I suppose, but I 'll try to get one here. The trouble is it would not be as *fine* as I like. Now in the twilight I will glance over your sweet little letter of Sep. 20th—On the 3d line of the 1st page of this letter—I don't know now what I intended to say. I 've since been walking, and did not see that omission when I began again. The last glimpses of day light are going fast & the ½ doz red lines of cloud right under the moon in front of my window are very pretty, and the view is very extensive & I can now see nothing but trees & green grass & corn ripening & I feel homesick for a sight of YOU, as you once said in one of yours to me, I think. I *can* see more than that stated 4 lines above but I 'll only mention the pretty little church—I 'll put a piece of newspaper on that last blot 2 lines above, and its too dark to write any more.

October.

I began (don't now remember *what*, as I stopped).

I write this by gas—and found the above two words "I began," (and all that is below—As the gas flickers, I think I had better defer till to-morrow.

M. P. September? 1885.

(There 's a good deal more written to you than this and to-morrow I 'll read it all over).

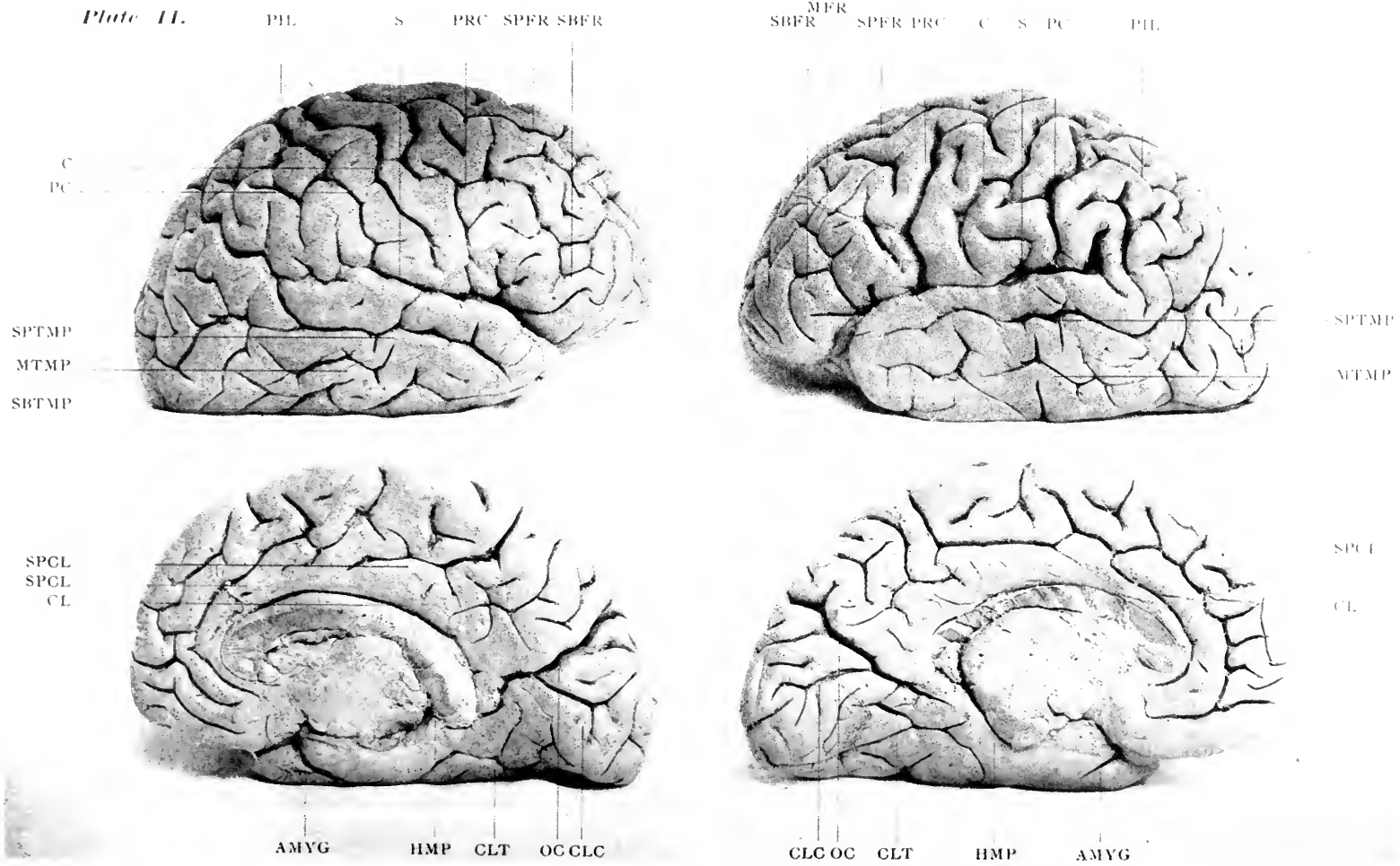
Dear P.

I have here yours of Sept? 25th, and it is all closely interlined, and the blank part filled up, and it does not, today, look to me, in proper condition to send to my sister, and as you have given me such a *sweet tablet*, and such a nice letter of the 25th Sep., I am going to try it again, and keep your pretty note, which is not much impaired by my fine pen. The quills you so kindly sent me are a comfort to me, and although I do devour almost everything I can, I will not "chew up" as you say, *these* picks, as I remember I used to do. When I first read your letter, I fancy I may not have been quite as much myself as now—Right you were about this air, and you did right to send me here—My recollection of Dr. S. is, of a slender, tall, exceedingly gentle & kind hearted man, and the last time I had the pleasure of writing to you, I could not I think, remember him quite as distinctly as now. I hope his sojourn in Europe will send him back cured. I must economise room. You encourage & comfort me when you tell me how much I am improved. It is lucky for me that I remained, or say, that I returned home, where you could look after me—for you could not have done better. My right eye is a trifle inflamed, (only for short time), at any rate, I have no doubt that it is only temporary & and so it was. You must refresh my memory : and you can laugh at my mistakes, but Mrs. C,, how is she connected with us. (a 2d cousin—I 'd tell you all about her if I dared) I know she 's one of your "boosom" friends, as Uncle called them. Was not she a W ks? & a sister to Mrs. M.? I, of course, could never forget such a name as Bridget Britt, but although I must have made Bridget's acquaintance, I cannot recall *her*. You must have bought the undershirt I have on, and, if you remember the length of it, do not let it be *quite* so long, for when I sit down I have to draw it up sometimes, as there are things more agreeable to sit upon than its border. Never mind, P., that 's much better than scantness : it is much better to be on the safe side.

Glad I am that you 'll write me "another and a longer letter soon." I have your sweet little envelope all safe—I also have, I have just found, another long letter to you & I 'll now read over you know why, but never mind—It is not as easy as usual for me to write, as my desk is my left hand only and I have one of those deep splits in the right thumb, just above the upper edge of the nail, where the pen rests, and another split (or chap) in the right fore finger, right in the crease of the finger, above where the thumb nail goes on the pen so writing is not easy today.

If you would send me *a small* piece of cort plaster in a letter. It is now evening, and so I 'll say very little—I will probably cut the sheet in half. The rackett is so great that I will pospone this.

Plate II.



CHINESE BRAIN.

It is now morning again. The hills are becoming variegated again. I guess I 'll wait now, until I 've got a cup of coffee inside, before finishing. If you ever receive any of this you can call it the lazy man's letter, for I 've been pretty long at it There 's a big man *snoring*, within five feet of me, & so I 'll change my base. Between that and the two gaps in thumb and fore finger, & the promenading (not mine) in the hall, I 'm nearly wild. never mind, I 'm beginning now to treat the whole thing as a huge joke.

I never before got a letter in such a confused state, and am being paid for the torture I once inflicted myself.

(Later) never mind, let us hope—The sun, now pleasant, will be obscured in about 5 minutes, and I 'm ready for some tea, and perhaps under its invigorating effect, I may say a few more words. I cannot afford to waste room, but this line is not on same day written as the previous. You will probably be able to know pretty nearly the date by its being the 1st day of a heavy rain storm, probably a N.E.er—I get my letter paper badly mixed up, and at the present moment I am conscious that I 'd better pospone writing this, through nervousness.

October 11, 1885.

DEAR P.

Do you know who wrote my name on this tablet? perhaps I 've asked before.—Its a treasure, it is just lovely. On a later day I answer *myself*—it must be *you*. Its one of those cloudy-sunny days—alternating. My young little old rosy cheeked friend, Willie Hearney, who was and is Mr. Madden's assistant, and who has lately had the Typhoid fever, is back again—He is not quite so stout, but looks well. You must know him—he is quite young. When did I last see you? There is a middle aged gent 'n here, rather stout, (none other could possess such a robust voice) who fills the hall with three or four base notes, which, while they make the building almost shake, you can not help thinking them to be musical—and we have a good tenor too; or we *had*. (he may be gone). The sun is a little too warm here, but I can use the curtain. I am writing on my hand—There is hardly any place on the hall but where some nervous fellow, who cannot keep still, drives me away—middle aged men act like children, sometimes. I now, for a little while only I suppose, find quiet in my own room, and have the benefit of a table. Your blank page I filled up closely, and then drew my pen through every line of one page, but there are three pages that I may send. I have written, but keep here, much to you; between lines of Sept. 25th, but now I have this sweet little tablet, I wont send it, and now I can keep your letters to myself, and they will be valuable, to refresh my memory, as well as to feast my eyes. It is hard enough to have to use hen tracks for stammering old colonels, like Johnston & Dickinson. "morning N. Y. papers" is just sung out, but not a "red" have I., but then, I 'm pretty sure, we can see them in the reading room, if we want to badly. We 're on a walk and taking

a rest—so called—but a pistol has just been discharged—& we 're now again making another move. I 'm now enjoying a quiet rest in the parlor, which is quiet, notwithstanding 6 or 7 people. The day is fine and just about as much or rather a little bit more of a strong breeze is blowing on me than I can well stand ; but it easy to move. It is easy to find a warm place in the sun, & it is 20m past 12, so dinner hour is near—When did I last see you & when again ? I suppose I can easily find a memorandum. Did you not bring me something nice to read in the way of a novel or story. and would it be easy for you to do so ? I fear not, oh how grateful the sun is on my back.

Not so, however *to-day* an apple tree trunk *is* though, against which I 'm standing. again we 're resting and the view, though not grand is pretty on a-c of the different colors presented by the changing leaves & I 'm making the fountain pen *fly* over the paper on my hat crown. There is but one very small cloud.

There are only six of us out walking including Mr M. you mustn't be surprised if I repeat—when were you & when will you be, here—we now resume our walk—au revoir—I think there is a change for the better in me. I will enquire the date—I 'll review this & note if any repetitons occur. Its after tea, during which I upset tea on table cloth. Writing by flickering gas. I 'm meditating a diary—When did I see you ? & when will I ? Old Time, with *me*, I think, does his work, always for the better, and when I call it Time, you know what I mean—Refresh my memory in regard to yourself—& tell me what changes have taken place with you—You know I 'm forgetful. I had a shorthand indexed & several books, whieh I kept, which I 've managed to hold on to—Also tell me when I will be likely to see you again. I 'm now going to postpone this for I don't want to lose my good sight and the gas is jumping Mr Madden has just called out " All ready for the ball room (Jim Crow. I don't care to go to the Ball room

There are two pages written that I think I 'll send with this, but as this is evening, I will leave the balance till I 've had a night's sleep. so good night.

Oct. 11th, 1885. This is another fine day Sunday. I 'm going to ask you again for a piece of *cort plaster* for splits one of which is now very bad. I will not feel like myself till I 've taken some medicine. " All ready for a walk—"

Again I think I 've written more than I 'll send—As it is gas light & *before* supper, I will postpon till, either after a cup of tea or that & a night's sleep.

Here I am again, before dejeuner, on a magnificent morning, and I 'm in a rocking chair, writing on the convenient flat arm. The sun is pretty well up, and has raised a mist all over the land in the distance, and has made the leaves of the trees, dazzling. The day continued perfect.

Somebody has given me something which takes the place of cort plaster, color of which is white, and sticks in the same way—I just made the remark, what a perfect conductor of sound this hall is—

Oct. 12th, You see my letters have taken the shape of a diary but I do not mean to keep up that system.
Refresh my
P, While I think of it,

These letters constitute very interesting reading to me. The last one was written about a week before the patient's death, and it is the first one in which there can be detected any mental confusion, over and above what may be attributed to the peculiar loss of memory. They furnish, moreover, curious intrinsic evidence of this loss of memory, as will be observed.

Through the kindness of Dr. Edwin Everett Smith, Dr. E. C. Booth, and Dr. C. L. Wertenbaker I am enabled to state that the patient remained in the same condition until October 15th, although I am not able to state as to whether any precise tests of motility and sensation were applied to him during this period. I myself never saw him alive after he left Brooklyn. Upon October 15th, he had a series of convulsions, some nineteen in all, and when these subsided he remained unconscious. The next day he was delirious, talking incoherently, and would, if not watched, get out of bed and wander around the room. He continued in this state until October 19th, strength gradually failing him, and died without further convulsion.

Autopsy twenty-four hours after death. Present: Dr. E. C. Booth, Dr. A. H. P. Leuf, and myself. My friend, Dr. Leuf, very kindly made the autopsy for me with his usual skill and thoroughness. Skull normal. Dura mater normal. Extensive lepto-meningitis, with adhesions and ecchymoses, the details of which are as follows:

In the left hemisphere, marked lepto-meningitis over the lower third of the ascending frontal and parietal convolutions, with strong adhesions; small adherent meningitic patch over base of second frontal; several small adherent patches in tip of first frontal; large, adherent patch over third frontal, which was softened; large adherent meningitic lesion of first and second temporal convolutions. There were several minute hemorrhages in the first frontal convolution, upper parietal lobule, and the occipital lobe.

The left temporo-sphenoidal lobe was much softened. Upon the left mesial surface, the gyrus fornicatus was softened and its surface was covered by a slight lepto-meningitis. On the right hemisphere, there was an adherent lepto-meningitis over the lower third of the ascending frontal and parietal convolutions, over the gyrus marginalis, and curving forward again over the first temporal convolution to its very tip. Upon the right mesial surface, there was a very adherent lepto-meningitis over the gyrus fornicatus. The softening presented no special characteristics. Outside of the details mentioned, there were no abnormal appearances whatsoever.

It will thus be seen that the lesions on both sides were almost entirely in the area of distribution of the Sylvian artery, although the mesial meningitis was within the territory supplied by the middle internal frontal branch of the anterior frontal artery.

This case is in entire accord with the one lately reported by Professor Westphal, of Berlin.

C. Westphal: *Ueber einen Fall von Zerstörung des linken Schläfenlappens durch Geschwulstbildung ohne aphasische Störungen. Berliner klinische Wochenschrift*, Bd. 21, No. 49, S. 777, Dec. 1, 1884.

Adult, male: attack sudden, and without apparent cause. Onset was sudden; nausea, pain in forehead and temples, tinnitus aurium, and a pulling and twitching sensation in the right extremities; also biting of the tongue, then sudden darkness, a fall, and unconsciousness. Remained so for fifteen minutes, after which he retained a headache. Repeated attacks, exactly as narrated, occurred at varying intervals of from one or two days to fourteen days. This continued four years, the patient meanwhile attending to business (character not stated). At the end of this time he gave up, because walking made him fall over backwards. Twelve days after stopping work, on Feb. 26, 1883, he came to the hospital, where he became comatose, would not respond if called to, occasionally uttered a few monosyllables, had to be fed with a tube, and held his teeth close together. Urination involuntary. From this attack

he recovered, but had a number of others. During the intervals his only symptoms were aching headache and sacral pain on laying down. Locomotion was a little difficult, but there was neither motor nor sensory paresis. Attempts at walking (a little later), and sometimes, no matter how carefully done, would cause him to promptly fall backwards. March 8th: He had clonic spasms of all the extremities, trismus, stertor, and bloody foam at the mouth. This lasted one hour, and he was then found to have a double optic neuritis. April 18th, he had a sacral bedsore as large as a hand. Would answer correctly but slowly and only after a time. Forehead and temples were painful on percussion. Tongue scarred. No disturbance of facial or hypoglossal nerves. Speech and swallowing normal. Eye motions free. On looking to the left, the eyes are not as easily rotated back as when he looks to the right. Right arm slightly resists passive motion; it is not as strong as the left, but manifests no tremor or ataxia. Relations of the two lower extremities same as the upper. Has cincture feeling at right knee and shin. Can walk a little, but right leg is a little unsteady. Retinæ marked by distinct white plaques and small hemorrhages, all limited to the place of entrance of the optic nerve. Veins of retinæ varicose, and papillæ of a decidedly grayish-red. According to the subsequent history of the case, the intervals between at tacks became less frequent and of shorter duration. Spasms of the arm increased and sometimes affected the whole body. Standing and walking became impossible. He became totally blind. Nov. 9th, Slight paralysis of left arm, which was complete on Nov. 14th. Had developed pneumonitis, of which he died the following day. *Autopsy:* Left temporo-sphenoidal lobe generally enlarged. Tip grown fast to the basal dura. The lobe contained a glio-sarcomatous tumor extending back into the posterior horn and contained myxomatous particles. The surrounding brain tissue was softened. The frontal lobe was pushed forwards and to the right. The ventricles were much dilated and filled with clear fluid. The large ganglia on the left side were enlarged and pushed well forwards. Nothing else of interest at examination.

As Professor Westphal's case, however, was one of lesion of the left temporo-sphenoidal lobe, whilst the patient was left-handed, it was not as decisive as mine. In mine, in the left temporal lobe there was a sufficient impairment of the parts involved to lead to a severe lepto-meningitis, with adhesions; moreover, the lesion had evidently been in existence some four months, and, although it was only a meningitis of the pia, was yet sufficient to cause the persistence for this period of this peculiar loss of memory; and yet there was neither ataxic aphasia nor loss of the memory of words (amnesic aphasia in the more limited sense), nor word-deafness, nor paraphasia, nor agramatismus, nor akataphasia, nor bradyphasia, nor deafness. There was simply a general loss of memory of events, and of the persons and things involved in events.

In view, therefore of Westphal's case and mine, I think that we must abandon or modify the theory, which had been growing out of Munk's clever and famous experiments on dogs and monkeys, to the effect that the human temporal convolutions were the so-called "centres" for the mental reception of sounds. It is very probably true, as Professor Westphal suggests, that the cases hitherto reported as confirming Munk's experiments have been cases of lesions of multiple convolutions, from among which the lesion of the temporal convolutions has been arbitrarily selected as responsible for the word-deafness. These two cases also mitigate against the classification of aphasia which has been suggested by Wernicke, viz.: a division into sensory, motor, and conductive (Leitungsaphasie) aphasia, the first being due to lesion of the temporal and occipital convolutions, the next to lesion of the third frontal convolution, and the last being caused by impairment of the commissural fibres between the sensory and motor areas of the cortex.

TRANCE CASES IN INEBRIETY.*

By T. D. CROTHERS, M.D.,

SUPERINTENDENT WALNUT LODGE, HARTFORD, CONN.

SUDDEN loss of consciousness, of variable duration, in which both act and language are automatic, occurs in a large proportion of cases of inebriety. As in epilepsy, these sudden palsies or trances vary in duration, prominence, intensity, and also completeness. In many instances they are unnoticed, and preceded by a state of half consciousness, which slowly deepens into a full trance or blank of memory, or like a shadow which deepens into totality, then gradually fades away. In some cases these blanks are sudden and profound, with no adumbra periods. In others they are partial, and of short duration. Most of the cases published have been noted for the prominence of the trance state, and its long duration and well-defined symptoms. Thus an inebriate suddenly changes in conduct and language, without any cause or reason, and without the usual symptoms of intoxication; or he follows an accustomed line of action in such an automatic and mechanical way as to excite attention, and yet not be intoxicated. After he claims not to have remembered these periods, and his conduct and language sustain his statement. He may enter upon a line of conduct that has been considered before, but laid aside as impractical, then after a short period of great activity stop abruptly, indicating the close of the trance state, and go on after with no reference to what he had done. The patient's statement of no recollection is always

* Read before the American Association for the Cure of Inebriates, June, 1886, New York City.

entitled to great consideration; for, no matter what personal motive or object he may have in denying all memory of the past, he may be stating a fact, which can be confirmed by other evidence than his word. An inebriate shot another man and had no recollection of the event. It was shown in the trial that, when drinking to excess, and not intoxicated, he had frequently fired his gun at different objects with no purpose that could be ascertained. The man he shot was a stranger to him, and he could have no motive. Here the circumstances and facts in his previous history sustained his assertion of no memory. Another man killed his wife and plead no recollection of the crime. It was ascertained that he had many times before, when drinking to excess, and not intoxicated, yet under the influence of spirits, expressed a strong desire to die with his wife and go to heaven together. They lived most amicably, and no motive could be assigned. After killing her, he made an ineffectual attempt to kill himself, then awoke, not realizing what he had done. All the facts sustained his statement of no memory, and yet both of these cases were punished as sane and responsible. Prominent crimes and acts committed under strange, unusual circumstances by inebriates, may furnish good evidence of these trance states; but when the conduct and circumstances point to some line of reasonable motive for the act, the question of a trance may seem doubtful. In my experience, a large number of inebriates affirm that at certain times they did not know or remember acts or words of their lives. These periods occurred when using alcohol to excess, and in many cases where they were acutely intoxicated; in others when no intoxication was present. These blanks of memory or trance states pass unobserved when followed by no unusual acts or words, and are considered as states of alcoholism, and their real significance overlooked. In the following case great injustice and suffering followed from ignorance of this state. A lawyer of wealth and clear judgment, who was an inebriate, after a period of excessive drink, planned most disreputable schemes of deception and fraud, and a day or more after denied all recollection of what he had done, and refused to

be held for his acts at this time. He was not intoxicated, but was drinking continuously, and seemed fully aware of all he said and did. This continued for several years, the blank periods increasing in length and his acts becoming more and more dishonest. No one conceived it possible that he was not fully sane and knew what he did, but the mystery of short periods of the most dishonest acts, which he denied after, could not be solved. In another case the president of a bank became a tract distributer in the lowest dens of the city. He is now a steady drinker, and at long intervals uses it to great excess at home, then suddenly buys quantities of tracts and goes out to give them away with words of exhortation. Another bank president, who is an inebriate, suddenly manifests extreme suspicion of every thing, will not sign any papers, or pay any bills, and in a short time recovers and goes on as usual, not realizing that he has been strange in manner or conduct.

These are called drunken freaks, but they occur after attacks of acute intoxication, and when the person is apparently conscious. Other and more common cases are those who follow some accustomed line of action and have no memory of it. I have published many prominent cases of this type, and now add some histories less noted.

A manufacturer noted this fact—that his leading machinist, after a few days of drinking, could not take up new work, and seemed confused when he attempted it. He could complete any work which had been determined before, but new work was impossible. These periods of stupidity lasted from a few hours to two or more days, and were noted by no memory of them; also a sudden coming on and breaking up. They were trance states. A judge of a lower court consulted me with this history: After drinking for a few days, not to intoxication, he has distinct blanks of memory, or periods when he is unconscious of what he does. He has to ask some one to tell him what has occurred. So far nothing has been done which has attracted attention or been unusual, but he is frightened at this state. These trances are from one to three or four hours' duration.

A travelling man who drinks steadily has similar trances while at work on the road, and cannot remember any thing from one point to another, an interval of a day or more. These cases are by no means uncommon, but where they are of short duration attract no attention. As an illustration, a hotel clerk will, after periods of excess of drink, wake up in a turkish bath, and have no idea how he came there. A farmer has found himself working on the farm, and the last event he could recall would be at a distant bar-room. How he came home and why he began to work are blanks to his memory.

Periodic drinkers will have trances during the drink period, and often when the period has subsided, and be unassociated with narcotism or stupidity. Some of these cases have a distinct blank, which breaks up partially as they become sober, or deepens again into a total blank when they begin to use spirits. A physician who drinks at intervals will forget every thing after a certain time, then recover in some hotel, and begin to realize his situation and desire to go home; then drink again and have another blank, which breaks as he becomes sober. Sometimes he is stupid during these blanks, at others he is apparently sober and conscious of his surroundings. During these apparently conscious periods he rides about aimlessly, talking about buying houses and lots or horses, but never makes any purchase. In other cases of periodic inebriates these blanks or trances begin with the narcotism of acute intoxication, and after this passes off, the trance continues for a variable time. A merchant, who has regular drink paroxysms of from two to three days' duration, will begin suddenly and drink to great stupor. Then, after two days, maks an effort to stop by going in the country, or where it is difficult to get spirits. The effort to break away from this paroxysm will last a week; then he will come back sober and remain so for many months. The trance blanks begin when the stupidity passes off. He will recover from the narcotism perfectly conscious of what he is doing, and be aware that he has been stupid from the spirits, then drink to stupor again. Finally, he will become alarmed at his state

and resolve to stop and drink brandy and mineral water. His anxiety and fear will increase, then another trance state will come, which will be a perfect blank of memory for two or three days. He will, during this time, drink less and less, and seem fully aware of the nature and character of his acts. The only unusual thing will be his morbid anxiety to recover, with emphatic protestations never to use spirits again. He will not transact any business, and does not talk much, but rides and walks continually; is hysterical in manner and language and very nervous. This condition may last two days; then he will awake in the morning and find that a long interval has passed which he cannot recall, or has any recollection of what he said or did. Here the trances come on when the drink paroxysm subsides. The stupidity of intoxication is not unusual; he is dimly conscious of falling asleep and awaking to get more spirits. But when he becomes alarmed at his case he then loses all recollection of what takes place; has no other idea but that he has been asleep; and cannot realize that some days have passed in which he has been about with apparent sanity and judgment.

Another case of similar character has been reported to me, where a lawyer, who is a periodic inebriate, has refused to do any business for some days after the drink narcotism has subsided. He says, in explanation of his conduct, that his head is not clear, although he is temperate and apparently fully aware of what he is doing; yet he complains that for several hours or days he cannot remember any thing he has done or said. In dipsomaniacs this blank may come on after the first drink of spirits, and break up soon or last a long time.

A dipsomaniac under my observation will suddenly feel an intense desire for spirits, which grows in proportion to the difficulties of gratifying it. After the first one or two glasses all memory of time and place vanishes. This lasts for three or four days, during which he is often stupid, then going about acting and talking as if he fully realized all his surroundings. He finally recovers, when the drink paroxysm begins to subside and memory and consciousness

come again. A dipsomaniac will have a trance state of variable duration, and recover; then, after two or more weeks, gradually recall many of the events of the past, or he may only remember parts of this period. His memory will always be very imperfect and unreliable, and, no matter how clear he may be concerning these events, a large margin of error will be present. These trances vary widely and should be studied carefully in all cases where the question of consciousness of right and wrong, and knowledge of the circumstances and surroundings, are called up. In my experience an inebriate, who has been so for any length of time, is a most incompetent witness of any facts concerning himself or surroundings. He is a mental waif, subject to every influence and impression that may happen to cross his horizon. In a recent murder trial, the prisoner, an inebriate, who committed the crime while in a trance state, was told at the police station by an officer some details of the act, which he afterwards recited as events he could remember. On this he was convicted. It was ascertained after, that these details were false and unsupported by the facts. They were the views of the officer, and the prisoner did not recognize their origin or inconsistency. In another case, where an inebriate was convicted of stealing, and where the history of trance blanks of memory was very clear, he made a confession of the details of the crime, and was convicted; later it proved to be incorrect in every particular. He had embodied the particulars of a fellow prisoner's crime as his own. He was confined in a cell with a burglar, and the first two days talked freely with him, hearing graphic details of crimes, which he had incorporated as part of his own history. It would seem from the history of many cases, that inebriates who suffer from trance states are peculiarly susceptible to the surrounding, and are likely to reflect the thoughts and acts of their associates. Hence the confession or statements of such cases are of no value unless sustained by other evidence. In almost every direction the range of clinical facts accumulate so rapidly in these cases as to bewilder the observer. The reader has only to study a single

case to realize the vast range of unknown facts that stretch out in all directions. Some of the conclusions which seem supported by the history of cases may be stated thus:

First, the trance state is common to inebriety, and may exhibit all degrees of completeness and intensity. *Second*, the memory in these trance states may be totally obliterated, never clear up; or it may, after a time, become restored, and events of the past come back either dimly or clearly. Sometimes it comes back suddenly, like a cloud passing off from the face of the sun. *Third*, this failure of memory is always followed by a faulty consciousness of the nature and character of his acts, and the consequences following from them. *Fourth*, the responsibility of these cases is a most fertile field for future investigation. It can only be determined by a full study of each case, and not from any preconceived theory of responsibility in disease.

IS THERE A PRIMARY CORD LESION IN PSEUDO-HYPERTROPHIC PARALYSIS?

By V. P. GIBNEY, M.D., New York.

THIS is not a paper presented to set forth all that is new or interesting on this subject, but is merely the record of a case to supplement the appearances of some microscopic specimens Dr. Amidon has kindly made for me. Neurological medicine still possesses charms for me, but my work for the past two years has been confined to a different branch, yet a branch intimately associated with the one that calls us together on this occasion. I must, therefore, ask indulgence if I fail to have informed myself on the results of important investigations. The record of cases completed by autopsy, and an autopsy "worked up" by one so well known in neurology as the gentleman who shares this report with me, cannot fail to elicit interest and to excite discussion.

Professor Charcot, in a communication to *Le Progrès médical*, No. 10, 1885, and his pupils, Marie and Gerinon, in the *Revue de Médecine* for October, 1885, have classified the primary muscular atrophies, and in an editorial in the *Medical News* for December 19, 1886, this classification is discussed.

Until this had come under my notice I confess that I entertained the belief that the lesion in pseudo-hypertrophic paralysis was more frequently located in the spinal cord than these distinguished authors seem to think.

They recognize two distinct groups of cases: the spinal, and the primary myopathies: the lesions of the former being in the spinal cord, while the lesions of the latter are in the

muscles themselves. One includes all those pathological processes which involve the gray matter of the cord directly or indirectly. Amyotrophic lateral sclerosis and chronic polyomyelitis anterior are in the one group, while the three following are included in the latter:

Pseudo-hypertrophic paralysis, the juvenile form of progressive muscular atrophy of Erb, and the hereditary infantile form of paralysis of Duchenne.

Charcot's school endeavors, and the writer of the editorial above mentioned thinks quite successfully, to prove that these diseases are one and the same, differing in clinical features at some time during their course, and they should, therefore, be designated as *myopathie progressive primitive*. The lesion is thus regarded as an interstitial myositis, there being a variable production of adipose tissue and occasionally an hypertrophy of individual muscular fibres.

For many years I have been able to distinguish quite readily between a progressive muscular atrophy and a pseudo-hypertrophic paralysis. The clinical features are sufficiently differentiated in the text-books. The later stages are strikingly similar in the completeness of the atrophy and in the consequent helplessness of the victims. What surprises me is that the spinal cord has not been found diseased.

The case on which this paper is based came under my observation April 10, 1878, remained under my daily observation for one year, lacking two days, was reported to me from time to time subsequently, and terminated in death from catarrhal bronchitis December 30, 1885.

The patient was a male eight years of age when he first came under my care in the hospital. The loss of power dates from the time he began walking—the second year of life. The mother did not become alarmed, however, and did not seek medical advice, until 1875, his sixth year—now ten years ago.

The family is decidedly neurotic. The mother and all the children I have seen—four or five in number—have some peculiarity of speech. Three children have died of tubercular meningitis, so reported. One brother, two years

younger than the subject of this history, is affected in the same manner, and his case is progressing the same way. He had at one time large calves, but when he ceased walking the hypertrophy began to fade, and now the muscles are flabby and small, like those in other parts of the body.

When Dr. Amidon told me that he had partially examined the specimens I understood him to say that the findings would show marked and undeniable pathological changes.

His final report, however, leaves me still in doubt as to whether the changes are primary or secondary.

DR. AMIDON'S REPORT.

Cord examined from the filum to the decussation of the pyramids.

The only lesion appeared to be in the ganglion cells of the anterior horns. As compared with corresponding sections from a healthy cord, about one half the cells seem to have disappeared—leaving no trace. Those that remain are most of them poorly defined, small, and in many instances processless. This lesion is more marked in the dorsal and lumbar regions. The cells of the columns of Clarke seemed normal.

ON A NEW PORTABLE GALVANIC BATTERY.*

By J. RUDISCH, M.D.,

ATTENDING PHYSICIAN, MOUNT SINAI HOSPITAL; PHYSICIAN, DEPARTMENT FOR NERVOUS DISEASES, GERMAN DISPENSARY.

IN an article published by Dr. Geo. W. Jacoby and myself (JOURNAL OF NERVOUS AND MENTAL DISEASE, 1884, No. 1), after having passed in review the galvanic batteries available for medical use, we came to the conclusion, that for a stationary battery the Leclanché is the best, and that the chloride of silver battery would answer best for portable use, were it not for its original high cost, its comparatively short life, and the difficulty attending its refilling with fresh chloride of silver, when exhausted.

Since that time nothing occurred to necessitate a change of opinion on our part in regard to the batteries mentioned in our article. During the last two years I have made various experiments with the object of finding a galvanic element which would answer the purposes of the general practitioner and not be open to the objections which are attached to the chloride of silver battery, and I trust I have now succeeded in finding a satisfactory answer to my inquiries.

Before presenting this battery to you it will be necessary to enter upon the train of thoughts which led to its construction.

1st. The polarization *in* the cell is inversely proportionate to the resistance either external or internal. Thus by making the resistance very high we may lower the polarization in the cell so much that a good galvanometer will be nearly or altogether insensible to it. By this means, as may be

* Read before the American Neurological Association, July 23, 1886.

readily perceived, the necessity of a depolarizer is obviated.

To elucidate this point, let us take the case of a zinc carbon couple immersed in a watery solution of sal ammoniac. If we connect the zinc with the carbon by a conductor outside the cell, chemical action is set up; the chloride of the sal ammoniac as well as the oxygen of the water combine with the zinc. Ammonia and hydrogen accumulate on the carbon. Now, if the connecting conductor be of a low resistance, the chemical action will be a rapid one, the hydrogen will not have time to either dissolve in the surrounding fluid or to escape in bubbles, and being of a more electropositive nature than zinc will set up a counter current that will weaken the principal current (from zinc to carbon) or even completely neutralize it.

The accumulation of hydrogen, as in the case described, is called polarization in the cell.

To get rid of this hydrogen we use a *depolarizer—i. e.*, a substance that readily gives up, say chlorine or oxygen, to combine with the hydrogen as soon as it is evolved.

If, then, on the other hand, the chemical action is diminished by increasing the internal or external resistance, less hydrogen will evolve in the same unit of time; it will dissolve in the surrounding fluid, and less or no polarization at all, will occur.

2d. A high electromotive force being requisite for our purpose, zinc and carbon, as standing very far apart in the electromotive series, were chosen.

3d. The exciting fluid, a solution of sal ammoniac (1 to 4) recommended itself for the following reasons: it has no reaction on the zinc, when the battery is not working; it is not corrosive; and it easily dissolves the oxychloride of zinc formed during the action of the cell.

The result of all these considerations has been the battery I now have the honor to present for your inspection.

This battery contains thirty couples of carbon and zinc of the type used in the construction of Waite and Bartlett's bichromate battery.

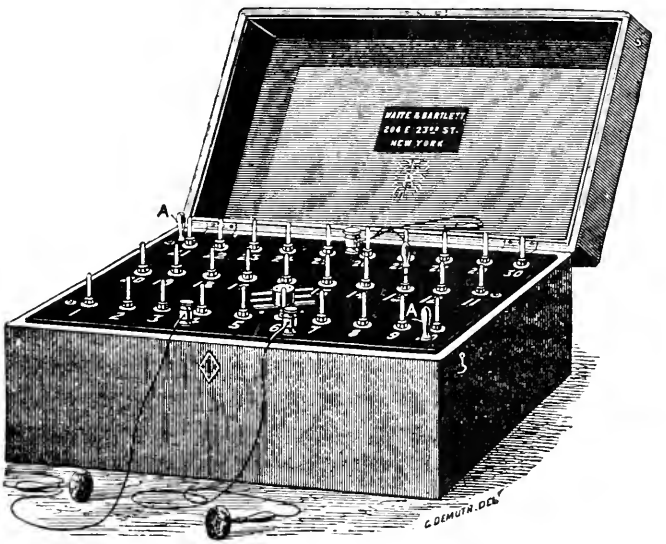
The couples are connected by screws to a hard-rubber

switch-board, having a plug and socket arrangement by which a number of cells can be thrown into the circuit.

On the switch-board are also a pole changer and a pair of binding posts.

Between the carbon and zinc of each couple a piece of stout asbestos paper is fastened by means of a few thin rubber bands.

The lower part is a trough composed of thirty hard-rubber cells, cemented one to another so as to prevent any fluid from



getting in between them. For further protection the trough is put in a tray of wood covered with an acid and water proof coating.

To fill the battery make a solution of a pound and a half of sal ammoniac to six quarts of water, fill all the cells nearly full, then by tipping the tray to a certain angle so that the superfluous liquid will run out, all the cells will be filled nearly to the same level.

This manner of filling is both expeditious and rational. By filling in the ordinary way you can hardly expect to fill all the cells to the same level. Now it is well known that one poorly filled cell tends to lower the efficacy of

the whole battery. It is also of importance to have all the zincs in the battery corroded uniformly.

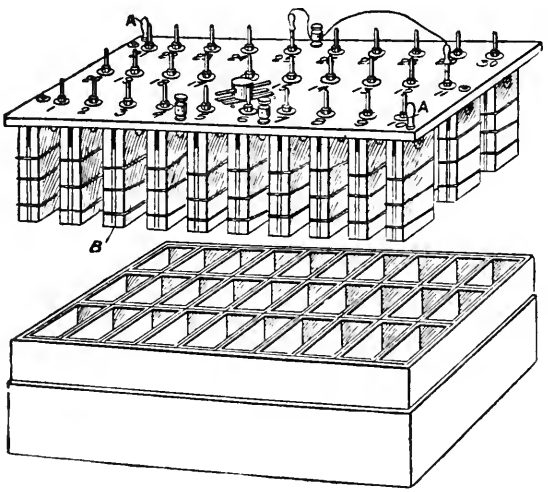
It remains to explain the rôle of the asbestos paper diaphragm between the zincs and the carbons.

Its purpose is threefold:

1st. It increases the internal resistance of the cell, which is, as I have already explained, of advantage.

2d. By its rough surface adhering to the carbon it facilitates the escape of the hydrogen bubbles, and thus acts as a depolarizer.

3d. It enables us to use the battery without the trough, the asbestos paper retaining sufficient fluid to keep the couples charged for a considerable time.



This last point is of extreme importance; it gives us a *really* transportable battery, without liquid liable to be spilled, and able to stand any position and any kind of jolting.

For the purpose of transportation, Messrs. Waite and Bartlett, who constructed this battery for me, had it so arranged that the lower part containing the tray with the cells, is left out, and the switch-board with the elements so fastened in the box as to be able to stand any amount of handling.

Now about the practical working of this arrangement. We have had this battery in daily use at the German dispensary for four weeks with the same filling of sal ammoniac.

About 7 A.M. this morning (July 23, '86) the cells were emptied and the box sent over to the meeting in Long Branch, and now, at 12.30 P.M., as you see, the battery gives a current sufficiently strong for all practical purposes.

Periscope.

PHYSIOLOGY OF NERVOUS SYSTEM.

The Physiology of the Hippocampus Major. Experimental Researches by Dr. GIUSEPPE FASOLD. *Rivista Sperimentale di Freniatra e di Med. Seg.*, anno xi., Fasc. iv., 1886.

This elaborate and interesting article is the result of experimentation performed at the physiological laboratory at Florence, in the Royal Institute, under the direction of Prof. Luciani.

The writer first reviews the various anatomical notions held in regard to the region of the hippocampus, and next the function of the hippocampus as set forth by different authorities, when he records his methods and experimentations, from which he deduces the following conclusions :

1. The hippocampus major stands in direct relation with the function of vision, of hearing, and of smell. It is one of those regions in which there is an accumulation or partial confusion of the diverse sensorial centres.

2. The sensitive fibres emanating from the hippocampus major and destined to the organs of vision have a partial decussation, in order to have a peripheric analogous distribution in the retinal field, with a predominance of the crossed fibres over the direct.

3. So the auditory fibres coming from the hippocampus major undergo a partial decussation like the visual, with a predominance of the crossed over the direct fibres.

It is probable that the olfactory fibres derived from the substance of the Ammon do not undergo decussation, or it may be such as to divide them equally between the two sides.

GRACE PECKHAM.

On Heat, Considered as the Retinal Intermediate of Light- and Color-Sensation. By L. WEBSTER FOX, M.D., and GEO. M. GOULD, A.B., of Philadelphia. *Am. Jour. of Ophthal.*, July, 1886.

The aim of the authors is to advance, and in part substantiate, the thought expressed in the title. Previous theories, especially the Young-Helmholtz and the Hering theories, have been found

essentially deductive, mutually destructive of one another, and altogether unsatisfactory in giving one the least glimpse of the *modus operandi* of the transmutation of ether-waves into the sensations of light and color. Indeed, any attempt at an explanation of the production of these psychical phenomena is expressly disclaimed by the authors, who hold that these are purely cerebral products, resulting from neural transmissions of retinal indications and changes. To throw light on the nature of this retinal function is the limited object, and, so far as the present goes, the only one promising any success. Thus the old doctrine of the specific or differentiating functions of the peripheral end-organs, upon which all previous theories of chromatic and achromatic sensation are based, is frankly discarded and the retina is reduced to a quantitative transfer of the stimulus, coupled with the fruitful theory of Local Signs. The essential nature of this transmutation or transference is considered to be a refined and delicate sensation of slight temperature changes. The ground for this conception is found to consist especially in the law of physics whereby the kinetic energies of the ether-wave, itself a product of atomic or molecular activity, can only produce mechanical energy to which nervous force is held to be allied rather than to atomic or (to chemical forces) by inducing atomic or molecular activity in a receiving medium (the retinal pigment layer). The height of this aroused molecular activity is, of course, proportioned to the ethereal wave-length, and the rods and cones transmit to the brain, just as the sensory nerves of the skin, the quantitative indications of these differing elevations of molecular energy. Many interesting facts are adduced from allied fields of research in support of this view. Especially noticeable is the experiment conducted by the authors upon themselves, by which chromatic sensation was quickly chilled into non-existence by the application of a crayon of ice to the sclerotic opposite the seat of retinal activity, the heightening of the same process by the application of heat, *etc.*, results which must follow from the premises. The additional time required for cerebral response to chromatic stimulus over that required by the mechanical stimulation of dermal end-organs is, also considered to point to the difficulty the fragile ether-wave finds in arousing the molecular agitation of the retinal reception medium in a state of unstable equilibrium, and of the transfer of this force to the optic-nerve end-organs.

This conception of the essential nature of the retinal process cannot be considered as any thing less than highly ingenious, logical, and suggestive, and our readers who feel any interest in the subject are advised to turn to the full treatment of the authors, rather than trust to the meagre outline above given. It seems from a note to the article that the idea and its working-out was wholly original with the authors, who subsequently found that the central thought of the theory had been already advanced by others, though reached by utterly different routes from as independent starting-points.

PATHOLOGY OF NERVOUS SYSTEM.

Beiträge zur Pathologie der "Multiplen Neuritis" und Alkohol-Lähmung (Contributions to the Pathology of Multiple Neuritis and Alcoholic Paralysis). By Dr. H. OPPENHEIM. *Zeitschrift f. kl. Mediz.*, vol. xi. p. 233, 1886.

Dr. H. Oppenheim has proved himself to be an active investigator and a somewhat prolific writer. His present contributions consist of six cases of multiple neuritis, one occurring in a tuberculous patient, and five in chronic alcoholists. The first case is by far the most interesting, for it shows that spinal-cord lesions and multiple neuritis may co-exist in the same individual. The author's resumé of this case is as follows: A stone-cutter, æt. forty, was taken down with weakness of both lower extremities; paralysis increased rapidly, and when taken into the hospital, about two months after initial symptoms, was not able to stand or walk. Atrophy of paralyzed muscles. Electrical reactions altered quantitatively in all paralyzed muscles, and R. D. in tibialis anticus and adductor magnus. Knee-jerks abolished. Patient complains of all sorts of disturbances of sensation. No objective sensory disturbances, except that he does not quite appreciate position of foot and toes. Pain along nerve-tracts. No disturbance of bladder or rectum. Patient is apathetic, and his memory weak. Cranial nerves not involved, except that pupils react sluggishly. Pulmonary tuberculosis. Death ensues five months after onset of disease.

The autopsy revealed, among other things: Atrophy of muscles (particularly those of lower extremities), pulmonary tuberculosis, atrophy of cerebral cortex. Nerves of lower extremities had undergone considerable degeneration, the muscular nerve-fibres in particular exhibited these changes—viz., atrophy of the nerve-fibres, and only very slight interstitial changes. The spinal cord was healthy throughout, with the exception of a very small focus of disease in the upper portion of the lumbar segments, effecting marked atrophy of the right anterior horn, partial degeneration of anterior and posterior roots, and a very trifling increase of connective tissue in the posterior columns at the same level. The author decides (and we think justly enough) that the symptoms observed during life cannot be referred to the spinal-cord lesion. He holds to the view (which seems to be obtaining general credence, and which the reporter advocated in a recent discussion) that any toxic agent may affect several portions of the central nervous system at one and the same time. In the case under consideration, it is a fair question whether the multiple neuritis is to be put to the account of the tubercular diathesis alone, or whether alcoholism was a potent factor in this as in the other cases reported by the author. The five distinctly alcoholic cases add but little new to our knowledge of multiple neuritis, though they are all well discussed. In several cases the differential diagnosis had to be made between tabes dorsalis and multiple

neuritis ; in No. VI. the difficulty was greatest, for, in addition to other symptoms, the pupils reacted imperfectly. This case went on to recovery, but when last observed the pupillary phenomena were not markedly improved, intense light producing only "eine minimale Pupillenverengung." Query : Was this not altogether independent of the chief trouble ? Oppenheim's cases are in entire accord with Gowers' opinion, that alcoholic neuritis is most frequently located in the lower extremities. It is difficult to determine in advance the duration of alcoholic neuritis ; prognosis favorable if alcohol is discontinued. O. recommends warm baths and electricity (undoubtedly the galvanic current) in the later stages of the disease. B. S.

Ueber die multiple Neuritis der Alkoholisten : Beiträge zur differentiellen Diagnostik dieses Leidens von der Tabes, der Poliomyelitis subacut. u. der sogenannten. Landry'schen Paralyse. By Prof. M. BERNHARDT (*Zeitschr. f. kl. Mediz.*, vol. xi., p. 363, 1886).

Prof. Bernhardt goes over much of the ground referred to by Oppenheim, but his object is rather to sum up present views on the subject of multiple neuritis than to add new facts to those that have now become tolerably familiar to us. The article is well written and has an admirable list of references to the literature of the subject. We cull the following points as particularly worthy of mention :

First of all, after discussing a typical case of alcoholic neuritis with characteristic mental symptoms, Bernhardt records the impression that it is only in very exceptional cases that either the cortex or the spinal cord is found diseased in these cases of multiple neuritis. He then distinguishes between cases of alcoholic paralysis and alcoholic ataxia (nervo-tabes peripherica, etc.) The latter cases are those which are most likely to be mistaken for cases of tabes dorsalis (p. 371). In the majority of cases there will be no difficulty whatever in distinguishing between these two forms of disease ; in cases of alcoholic ataxia all the symptoms develop with greater rapidity than in cases of locomotor ataxia ; in the former too there are distinct atrophic paralyses with RD of affected muscles. Alcoholic symptoms may be common to both conditions ; and Bernhardt reports two cases of true tabes in which matters were complicated very much by the presence of peripheral paralysis, due probably to a neuritis of both peroneal nerves. In these cases there were some atrophy of the muscles and altered electrical reactions.

Special attention is called to the fact that presence of optic neuritis (*not* optic atrophy) would argue rather in favor of alcoholic ataxia than of true ataxia. In a discussion on the symptomatic value of the knee-jerks, the author states cases showing that these may be present in some cases of alcoholic

ataxia, and then again refers to cases of true ataxia, reported by Hert and Westphal (reviewed in this JOURNAL), in which the knee-jerk was well preserved. Hereafter, even the diagnosis *tabes dorsalis* must not be made too hastily.

The case is even worse with regard to the differential diagnosis between poliomyelitis and multiple neuritis. In some few cases difficulties may arise, for paralysis of functionally related muscles is not altogether pathognomonic of poliomyelitis, for Eisenlohr has shown that in disease of the peripheral nerve-tracts, functionally related nerve-fibres may be affected, while other, though neighboring, fibres may remain healthy.

The author has grave doubts as to the advisability of attempting a differential diagnosis between Landry's paralysis and multiple neuritis, for the very good reason that the pathology of Landry's paralysis is still an unknown quantity, and that a rapidly spreading multiple neuritis may give rise to symptoms exactly resembling those of so-called Landry's (acute ascending) paralysis.

Bernhardt's contribution is a valuable one; yet we regret its iconoclasm. Unusual complications arise in all diseases, and it seems to us that the author has created no little confusion by laying undue stress upon these complications. In the matter of multiple neuritis we need much more building up before we begin to pull down. The difficulties in diagnosis are well illustrated in the following article.

Degeneration of the Columns of Goll in an Alcoholic Subject. By Dr. OSW. VIERORDT. *Arch. f. Psychiatrie*, vol. xvii., p. 365, 1886.

A. M., workingman, æt. 30: phthisical; had been drinking to excess for years; gastric disturbances in consequence. March, 1884, was suddenly seized with stabbing (lightning) pains ascending from feet to trunk; periodically weakness and ataxia of lower extremities with anorexia and vomiting; weakness increased up to June, 1884; from that time onward, great improvement in this respect. All symptoms returned in October, to which formications in the legs were superadded. Middle of September patient became bedridden. Venereal infection denied; no chest trouble at the time was received into clinic (at Leipzig), Jan. 22, 1885. Examination revealed slight inequality of pupils; light reflex normal; evidences of pulmonary trouble; testicles small; no objective symptoms of syphilis. Considerable uniform atrophy of all muscles of upper extremities, with corresponding diminution of muscular power. Toxic condition of muscles about normal; no ataxia, and no disturbance of muscular sense. No hyperæsthesias or paræsthesias anywhere; peripheral nerves neither thickened nor painful. Tendon phenomena very weak; muscles sensitive to slightest touch. Lower extremities: muscular status about the same as in upper extremities; atrophy more pronounced in lower legs than in thighs. Muscular power in keep-

ing with atrophy (excepting disproportionate weakness in flexing left thigh). Legs fall asleep; also formications; no marked sensory disturbances, and no hyperæsthesia.

Muscular sense normal; knee-jerks abolished; cutaneous reflexes fairly preserved. Distinct ataxia. Romberg's symptom present. Bladder and rectum normal. Increased excitability of muscles in upper and lower extremities. Percussion of nerves produces contractions in muscles supplied by these nerves. (*Nervi peron. et tibial.*) Electrical reactions unaltered. All symptoms progressed; occasional variations in amount of paresis; ataxia less marked (in upper extremities?—Rep.). In the later stages hyperæsthesia of the skin of lower extremities. Knee-jerks remain absent; plantar reflexes normal. No vesical or rectal trouble at any time, and no changes in electrical reactions. Patient developed pleuritis with effusion, and died April 12th.

In the ante-multiple-neuritis days we think most neurologists would have made a diagnosis of locomotor ataxia with some unusual symptoms (atrophy of muscles, which might possibly have been put to the account of the general (tubercular) emaciation). Dr. Vierordt made a diagnosis (with considerable reservation, however) of multiple neuritis. The autopsy revealed, besides evidences of pulmonary tuberculosis, etc., a degeneration of the columns of Goll, most marked in the oblongata and cervical spinal cord. In the middle and lower dorsal segments very slight disease of posterior root-zones (p. 371) Lumbar segments entirely normal. In the medulla oblongata the degeneration of columns of Goll was complete; peripheral nerves, normal. Vierordt looks upon the disease of the columns of Goll as a primary degeneration, and not secondary to the affection of the posterior root-zones in the lower and middle dorsal segments; but the affection of the posterior roots might have followed upon disease in the columns of Goll. Strümpell is quoted in support of such a *descending* progression in centripetally conducting nerve-tracts. Dr. Vierordt presents many problems of interest to which we have not the time nor space to refer. B. S.

On a Family-form of Congenital Paramyotonia Occurring in Six Successive Generations. By Prof. A. EULENBURG, of Berlin. *Neurol. Centralbl.*, No. 12, 1886.

This "muscular idiosyncrasy" had been introduced into a German family by a mother born in Rome of Italian parentage. Not a single one of five generations had been skipped, and no distinction was shown with regard to sex. The disease could be detected (by those who were acquainted with this peculiar muscular affection), even in the new-born, by the length of time the eyes remained closed after washing them with cold water. The entire voluntary muscular system is subject to spasm, rigidity, or paresis brought on mainly by exposure to cold or wet. In the upper extremity, for instance, the spastic condition is a transient

one, followed by a condition of paralysis which may last hours and even days; in the lower extremities there may be simple rigidity, or rhythmical oscillations of the thigh-muscles; and at other times the muscular disturbance may be so great that the patient falls down and is not able to rise again without assistance.

If the trouble arises in the facial muscles (as it does easily from cold) the patient may not be able to speak until he returns to a warm room. The reader who will refer to the review on Thomsen's disease (this JOURNAL and volume, No. 6) will recognize certain points of resemblance between the two forms of disease. The differences between the two are equally marked. The increased muscular excitability, the prolonged contractions, the wave-like contractions from anode to kathode—all these characteristics of Thomsen's disease are entirely wanting in the cases Eulenburg describes. On the other hand, he found that faradic excitability was diminished, more particularly during the stage of rigidity, and that there was a tendency to tetanic contraction following upon K. C. as well as A. C. In spite of such differences as we have referred to, Thomsen's disease and paramyotonia congenita appear to be closely allied. While it may be important to insist, on an accurate differential diagnosis (as Erb would have it), we must not forget that the pathological conditions underlying these various diseases may have much in common. Eulenburg hazards the opinion that a temporary spastic condition of the blood-vessels in the muscles (excited by cold, etc.) may furnish the solution to the present mystery.

It is worth while adding that in the family Eulenburg refers to intermarriages have occurred but twice. The disease is evidently on the wane in this family. B. S.

MENTAL PATHOLOGY.

Visceral Lesion or Disorder and Mental Disease.—Dr. JAMES ADAMS (*British Med. Journal*, May 29, 1886) calls attention to the fact that mental alienation is often associated with disease or disordered function of the bodily organs. He observes that although it cannot be stated, with perfect certainty, that these stand to each other in the relation of cause and effect, this much may, at least, be affirmed, that bodily disease or disorder not unfrequently determines the character of delusion or the form which mental disease assumes. Thus the childless wife, past her climacteric, finds, as the effect of an epitheliomatous uterus, the pains which she fondly brings herself to believe are the long-looked and wished-for pains of labor; whilst the maniac of persecution traces, in the agonies of pain which he actually undergoes from organic visceral disease, only the cruel influences and machinations of his enemy, or the evil one, upon him.

Dr. Adams reports a case of profound melancholia of about three weeks' duration, and said to be due to "anxiety in family

matters," in which convalescence was established, apparently, through the removal from the rectum, by means of spoon-handles, of "a dense and hardened mass of scybala," and subsequent complete evacuation of the lower bowel by enemata. The patient, a woman aged fifty years, with no inherited tendency to insanity, was greatly depressed, violently resisted all efforts to control her, refused to eat or drink, requiring to be fed with the tube thrice daily; was almost constantly agitated, sleepless, incoherent, confused, and stupid These symptoms began gradually to disappear soon after the bowels were freely emptied. Progress toward recovery was steady until, at the end of the third week, the patient was fairly convalescent and ready to be discharged.

While it may be said that the case here reported by Dr. Adam reveals, to the experienced asylum physician, nothing of a novel character, its recital, nevertheless, is of use in serving to call attention to the important necessity of regulating the bowels in almost every case of insanity—a point too often overlooked by the family physician and friends. In fact, every asylum physician frequently receives cases in which there has been no evacuation of the bowels for days, or even weeks, and in which the exhibition of a brisk cathartic is not seldom followed by a marked amelioration of the mental symptoms. It is quite possible that failure to overcome the inactivity of the bowels, in these cases, not infrequently arises from inappreciation of the fact that, owing to blunted nerve sensibility, the insane require relatively large doses to produce a given therapeutic result, as compared with the sane.

CARLOS F. MACDONALD.

County Care of the Insane.—The Committee on Lunacy, in their report to the State Board of Public Charities, Pennsylvania, 1885, say:

"There are very few almhouse hospitals in the State where these unfortunates can receive proper care. In most of them the only attention they receive is that afforded by the steward and his wife, with such aid as the paupers of the house afford; and it is at least doubtful whether it is desirable to facilitate this rather than that the State hospitals should assume whatever provision is required for all who must have remedial treatment, as in some of the largest States is the case.

"We do not propose here to enter at length upon the question whether the insane can be treated best in State hospitals or county. Admitting it is an open question, the weight of sentiment is unquestionably in favor of State treatment at the present time. Only one State, Wisconsin, has adopted the county system, and while there are those here and there who favor the abandonment of State hospitals, such opinion is practically limited to that State. The advantages claimed are, first, nearness of patients to their friends; and second, the better individual care that can be

given to a small number of patients. As to the first, it may be said that it is quite as likely to prove a disadvantage remedially as an advantage, because the presence of their nearest kin is often a most exciting incident in some cases of insanity. There is more force in the second reason assigned ; but, in the opinion of this committee, the same end will be better attained by adopting the cottage or detached building system of construction for State hospitals, and increasing the number of medical assistants, so that one or another of them shall be familiar with the daily condition of every patient. The attainment of this is the key to the whole question. In our opinion, classification and the separate treatment of different classes are of great importance. This cannot be attained in very small institutions, while the increased medical attention claimed for them *can* be attained in the large hospitals. The latter is likely also to be better equipped, better provided with occupations and amusements, freer from pettiness, and from political management, and conducted more on a medical or remedial basis.

"One thing is sure : an almhouse, not specially designed for hospital treatment, is no place for acute insanity, and very few of our poor-districts are provided for its cure. Moreover, the retention of insane persons at the almshouses greatly increases the cost and the difficulty of inspection, and diminishes its thoroughness ; for while the visiting official can easily visit the patients in four or six State hospitals, and as many private institutions, if he had no other visitations to make, he could not so visit them in sixty county institutions, in addition to performing his other duties. So far, therefore, as the capacity of the State hospitals will allow, the committee have continued to urge the removal of the insane from the counties, and their concentration in the large institutions."

The committee, in referring to the management of the State asylums, alludes to the varying success of these establishments, which they ascribe not so much to difference in age or condition of their buildings, but rather to variance in their official *personnel*. "You cannot," they say, "secure all superintendents with the qualities of the best, nor can a superintendent, however good, obtain the necessary number of attendants all of entire fitness for the very difficult duties of their occupation."

"We would wish, nevertheless, to couple with our advocacy of the State system, insistance upon the best officering possible. Sufficient salary should be paid to superintendents to secure high talent, and it is false economy to stint too much either the number or grade of attendants, by keeping them low. The superintendent should have an ample corps of medical assistants ; and we see no reason why the custom in vogue at hospitals for the sick and wounded should not be extended to these, of receiving graduates in medicine, for the first year or two of their medical career, as residents, for the sake of practice in nervous diseases."

CARLOS F. MACDONALD.

Reviews and Bibliographical Notes.

Die Functions-Localisation auf der Grosshirnrinde, an Thierexperimenten und klinischen Fällen nachgeinesen von Dr. Luigi Luciani und Dr. Giuseppe Seppilli. Autorisirte Deutsche und Vermehrte Ausgabe von Dr. M. O. Fraenkel. Leipsig, 1886. pp. 407.

THE LOCALIZATION OF FUNCTIONS IN THE CEREBRAL CORTEX.

To venture into the much-contested field of brain localization at the critical stage which the problem has now reached, requires no little courage ; to come out of the contest victorious with some of the old problems conquered and a general clearing of the field for future contests, requires at least equal amount of scientific ability and logical acumen. To both these claims for the highest praise Dr. Luciani and Dr. Seppilli are well entitled. The authors approach the subject with a full knowledge and a worthy appreciation of the literature ; they recognize that the solution of the problem not only requires difficult experiments and careful observations, but also a logical and unprejudiced interpretation of the results.

It is of course impossible and would be foolish to approach the problem as though we knew nothing of the cortex, and would consider any result as *a priori* probable as any other ; but one must carefully refrain from bringing to the subject any preconceived notion of the nature or arrangement of the centres. To find an area, the removal of which will cause blindness, and call that a sight centre, is to assume that centre is a definitely limited, circumscribed collection of gray matter. If such is the case it must be established by comparison of a large number of varied experiments ; not be assumed at the outset.

The problem, though apparently simple, is really very complex. To begin with, the important distinction between the phenomena due to irritation (as the effect of the lesion) and those due to the removal of a portion of the cortex (extirpation phenomena) must be made. Our authors enumerate five criteria to aid them in this problem : (1) Finding a certain disturbance as the result of a lesion, we know that the remaining functions have nothing to do with that area. This does not carry us very far. (2) By comparing the variation in the effects of the same operation in different individuals, we can arrive at the phenomena probably due to irritation, because it is not to be supposed that the irritative

effect of the lesion will be the same in each case. (3) The irritation phenomena will probably affect functionally allied centres, and may thus be detected by their nature. (4) By successive operations on the same animal one not only sees the irritation phenomena change, but the compensation of function by other parts of the brain may also be detected. (5) When a small lesion produces a serious loss of function it is naturally an extirpation phenomenon.

All the effects resulting from an operation must be noticed, not only the principal one. All the senses of the animal must be carefully tested, and this is really one of the chief difficulties. A convenient set of tests applicable to the senses of a dog or ape, would be a valuable contribution to the subject. The animals must be kept alive as long as possible, and treated as one would a convalescent patient. In this way the gradual waning of the irritation phenomena comes under observation. The usual precautions during and after the operation need not be detailed here.

The arrangement of the work is admirable. The experimental and physiological portions are due to Dr. Luciani; the clinical and pathological portions to Dr. Seppilli. The two parts are separate, and each is preceded by an historical sketch, and includes the protocol of each day's experiments and record of clinical cases.

The first chapter is devoted to the centres of the special senses, and begins with the most important and most disputed centre—the centre for vision. As the result of their experiments on dogs and apes, they conclude that if you call the sight centre all that area the destruction of any part of which will cause visual disturbance, then (in agreement with Goltz) this centre is too extended to be localized at all. But if we distinguish between transitory and permanent (though gradually decreasing) impairment of vision, we at once have the occipital lobes with a small part of the adjoining parietal marked out as the focus of the sight centre; this centre, however, is not sharply defined, but extends to and is anatomically connected with other parts of the cortex in the direction of the frontal and temporal lobes. In this way the variations in intensity and permanence of visual disturbances following the removal of different parts of the cortex are easily explained. An injury to the "periphery" of the visual area causes less severe and less permanent loss of sight than an injury to the "focus" of the area. In apes the sight centre is more definitely localized than in dogs, and its focus is confined to the occipital lobes (in general agreement with Munk). The angular gyrus forms a part of the periphery of the centre.

The problem of the relation of each cerebral hemisphere to each half of the body is especially interesting and important in the case of the relation of each sight centre to each retina. The constant fact (first pointed out by Munk), that the extensive destruction of one occipital lobe produces *homonymous bilateral hemianopsia* (greater on the opposite eye), shows that the centre of the left side, for example, is connected with the outer (smaller) part of

the left retina and the inner (larger) part of the right retina. This furnishes a simple scheme of decussation somewhat different from the current one due to Charcot. Munk's further localization of a centre for the fovea, and his view of the separate orderly projection of each point of the retina on the cortex, receive no support whatever. On the contrary, there is every proof of a thorough commingling and redistribution of the optic fibres on their way from the retina to the cortex (agrees with Goltz and Loeb). The function of the centre is not to receive the crude retinal impression, but to elaborate and associate visual images; it is perceptive, not sensational. The general results are represented in a diagram (*v. Brain*, July, 1885) in which the crowding and size of the dots represent the "intensity" of the centre, while the shaded dots distinguish the proportionate connection of the centre with the same side of the body from its connection with the opposite side.

The centre for hearing likewise has a focus and a periphery. The focus is in the temporal lobe (the usual view); the periphery in the direction of the parietal and frontal, of the hippocampus, and the cornu ammonis. It is also definitely ascertained that each centre is connected with both ears, but more with the opposite, and is perceptive in its character.

As regards smell the experiments are not as conclusive. The parts adjoining the Sylvian fissure, together with the hippocampal region, are most closely related to this sense. As regards decussation the olfactory mechanism forms an exception (at least in dogs), such centre being connected to a greater extent with the nostril on the same side.

No definite results in localizing the taste centre were reached. It is evidently closely allied with smell. On the pathological side the evidence with regard to sight is in entire agreement with the experiments on animals, and thus makes these doubly valuable and doubly certain. As regards hearing, the important symptom of word-deafness (showing the perceptive character of the centre) proves to be most frequently associated with lesions of the first and second convolutions of the left temporal lobe. There is seldom a lesion on both sides and never on the right alone (in right-handed persons).

The clinical evidence regarding the olfactory and gustatory centres is as indefinite as the experimental.

The second part of the volume is devoted to the sensori-motor area. In the first place the mixed character of the area is distinctly proved. The sensory affections are mostly tactile, but also diminish sensibility to pain, to heat and cold, and to muscular inconvenience. The permanence of the effect varies as the extent of the lesion. With regard to a more definite localization of the motor centres, that is possible only when you take the degree of the effect into account. Extirpation of the fore-leg centre will affeet the hind leg as well, but less so than the fore-leg (agrees with Munk; opposed to Hitzig and Ferrier). The sensori-motor area includes almost the whole anterior half of the brain, and is

larger than the motor-excitable area. It is exclusively connected (in dogs and apes) with the opposite half of the body.

It is in this part of the subject more than in any other that pathology helps out physiology. The careful analysis of clinical cases is particularly instructive and important. It would be impossible to enter into details ; the main conclusions reached are that while the areas for the sensibility of different parts cannot be definitely mapped out, yet it is made probable that these centres have separate foci, with more or less overlapping peripheries. In this way the lower part of the motor zone is indicated as the center of facial sensibility, the upper as the centre for the sensibility of the limbs.

With regard to the separate motor centres we have another important source of evidence : in animals, electrical stimulation of the cortex ; in man, the spasms accompanying epileptic discharges. The importance of the former is well understood. The chapter treating of the latter is perhaps the best statement of the significance of cortical epilepsy to be had, and is an original contribution to the German edition. Its point of view is that of Hughlings-Jackson, and the originality and suggestiveness of that view are fully brought out. The conclusions reached agree with those of Dr. Jackson.

One general point with regard to the sense centres of the dog remains to be noticed. A small area (in the posterior part of the parietal ; Munk's zone F) is common to the centres of sight, hearing, smell, and touch. Goltz noticed the great change of character resulting from removal of this area.

The general impression of Luciani's and Seppilli's work is a decidedly hopeful one. It gives one the feeling that after all we do know something of the brain, and are on the right path to know more. When one remembers that only sixteen years ago was the electrical excitability of the cortex discovered ; and that now the combined forces of anatomy, physiology, and pathology are applied to the solution of the vexed problem, the real significance of the modern biological movement is apparent.

To those who want one single book as a compend of the best views of localization of function in the cortex, Luciani's and Seppelli's work is decidedly the one to be recommended. It represents the high-water mark of the discussion.

The important view which they hold in common with Exner, Goltz, and others, is that the brain is not composed of strictly mapped out, functionally different centres, but that it is an organ, the several parts of which are intimately and complexly interconnected in such a way that one portion is more closely related to certain functions than another. They also deserve special credit for the method of successive operations on the same animal —a method which rules out many disturbing influences and will doubtless be adopted by others.

Dr. Fraenkel has performed a valuable service in his admirable translation of this important work into a language in which it is more apt to gain due recognition. JOSEPH JASTROW.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

TWELFTH ANNUAL REPORT.

First day, morning session.

The American Neurological Association convened at the Howland House, Long Branch, N. J., July 21, 1886, and was called to order by the President, Dr. Burt G. Wilder, of Ithaca, N. Y., who delivered the address as retiring President, on "The Collocation of a Suture and Fissure in the Human Fœtus" (see JOURNAL OF NERVOUS AND MENTAL DISEASE, vol. xiii., p. 463).

At the close of his address the President introduced the President-elect, Dr. Charles K. Mills, of Philadelphia.

On motion, the reading of the minutes of the last annual meeting, already published, was dispensed with.

Dr. R. W. Amidon, of New York, Secretary and Treasurer, read the

REPORT OF THE TREASURER,

prepared by Dr. G. M. Hammond, of New York, Secretary and Treasurer for last year. On motion the report was accepted.

THE REPORT OF THE COUNCIL

was then read, and it was recommended that the following candidates be elected to active membership: James Hendrie Lloyd and F. X. Dercum, of Philadelphia; B. Sachs, J. Rudisch, and E. D. Fisher, of New York.

They were elected by an affirmative ballot cast by the Secretary.

MEMBERS PRESENT.

The following members were present during either the whole or a part of the meeting:

Drs. Burt G. Wilder, of Ithaca, N. Y.; Charles K. Mills, of

Philadelphia ; R. W. Amidon, of New York ; G. M. Hammond, of New York ; L. C. Gray, of Brooklyn ; Philip Zenner, of Cincinnati, O.; R. L. Parsons, of Sing Sing, N. Y.; John Van Bibber, of Baltimore, Md.; W. R. Birdsall, C. L. Dana, L. Weber, V. P. Gibney, E. D. Fisher, S. J. McNutt, B. Sachs, and G. W. Jacoby, of New York ; F. X. Dercum, J. H. Lloyd, G. B. Massey, and W. Sinkler, of Philadelphia.

The President, Dr. Charles K. Mills, of Philadelphia, after the transaction of the above preliminary business, delivered his Inaugural Address. (See JOURNAL OF NERVOUS AND MENTAL DISEASE, vol. xiii., p. 517.)

Remarks on the President's Address.

Dr. BURT G. WILDER, of Ithaca, N. Y.: Inasmuch as you have asked me to open the discussion on the paper, I will do so to a slight extent, although there are some parts of it on which I do not feel competent to make any remarks.

The subject I regard as one in every way worthy of the importance which our President has assigned to it as the subject of his inaugural address, and I cannot but feel that it is substantial encouragement in the way of exact and philosophical, and, in the highest sense, scientific methods of studying neurology and psychiatry together. I feel very sure that there are more psychologists than I have had the misfortune to meet, who do not think it necessary to study the brain at all. And, on the other hand, important as is the subject, there are practising physicians and surgeons who are not so familiar with this organ as they should be.

It seems to me that the highest compliment that our President could pay to this Association is in the straightforward way which he has adopted of telling us these facts with regard to these brains, as if he took it for granted that we would know what he meant. I venture to say that not in every gathering of practitioners in the United States would it have been possible to present a paper of this kind and have it at all intelligible to the hearers.

Personally I feel an intense interest in all that he has said, and I only wish that I had had opportunity to examine the brains more fully in order to confirm some of his statements, and I trust, with some diffidence, to object to some others.

In the first place, he has called attention to the exposure of the

insula in the negro's brain. In the only negro's brain which I have, the insula was exposed much more than in any of the brains showed to-day. I speak of it because it was hardened within the skull, and there was no possibility of an abnormal covering or uncovering from exposure.

In the Chinaman's brain it will be noticed that the insula is abnormally covered, but it will not do to credit the Chinese, on account of this, with having a superabundance of that material which is present in the brains of white men, because it has been flattened upon the side, pressing the supertemporal gyre over it.

What the President has said with regard to the unphilosophical —and I will go further and say unscientific—character of some of Benedikt's generalizations concerning the type of the brains of criminals, I approve of most heartily. It seems to me that Benedikt's statements merit the most careful scrutiny, because they are sensational in character, and I doubt whether all his statements will bear scrutiny.

With regard to the confluence of the central fissure with the Sylvian, our President stated that in but few does this occur, and while I should hesitate to say any thing on this point, except that the brains which I have examined confirm this statement, I may say that in between twenty and thirty brains I have not seen such confluence.

A good deal was said about the ape's brain, the extension of the parietal fissure, and the relation of the subject to *plis de passage*, which is perhaps true in fact, but I interpret them otherwise and should employ somewhat different terms; so I will reserve my criticism for a future time.

Finally, I should like to add a word with regard to the probability that a large number of human brains are more or less peculiar in some respect. It has been my fortune never to have had any but faulty brains, and I suspect that it is the case with a large number of those who have studied the brain carefully. But the majority of brains, which are studied carefully, I think are intrinsically more or less peculiar in some respect, which is noteworthy. As a singular illustration of the embarrassment to which a teacher may be put, I succeeded in obtaining half a dozen human brains for students to study, but when I came to examine them I found that I could not afford to allow them to dissect a single one, but I kept them all, because each showed some special striking peculiarity.

In conclusion : I should like to ask our President whether, supposing he had not known that the brain he was examining was that of a Chinaman, had not known but that it was the brain of an ordinary Caucasian, he would have felt disposed to regard the peculiarities which he has pointed out as any thing more than might exist in exceptional cases. In other words, is there any thing about the Chinaman's brain which would lead him to suppose that it belonged to a different race? For my own part, I should hesitate a good deal either to say their fissural type is unusually small, or that the relations of the fissures are peculiar in any marked degree. This is hardly a fair question to put, because the doctor knew that it was a Chinaman's brain, and I did also. However, I would like to see more Chinamen's brains.

There are, then, two points on which I wish specially to insist : first, that in two negro brains I have seen the same uncovering of the insula referred to ; and second, I have never but in one specimen seen fusion of the fissure of Rolando with the fissure of Sylvius.

Dr. F. X. DERCUM, of Philadelphia : I would like to make mention of one brain which I saw in the hospital for the insane, and which was taken from the body of an epileptic female. In this brain the fissures of Rolando and Sylvius were confluent on one side and nearly so on the other ; and the insula was covered on both sides. In some respects the brain was abnormal, while as to some other points it resembled the normal brain.

While listening to Prof. Wilder's paper, a communication made to the Philadelphia Neurological Society by Dr. A J. Parker was called to mind, in which he described several fissures on the mesial surface in children which afterwards disappear as the brain becomes adult. These fissures he described as being due to a mechanical cause, the brain growing more rapidly than the cranium does, and therefore is puckered up at various situations, and that these puckerings or creases unfold and disappear as the cavity of the skull enlarges. It seems to me that the peculiar relation of the fissures and the sutures would suggest also some mechanical relationship.

Mr. JOSEPH JASTROW, Ph.D., of Johns Hopkins University, was present, and was invited by the President to take part in the discussion. He thought it possible to make a picture of the human brain by the process of composite photography, which would locate certain convolutions as normal without producing such an

arbitrary map of the brain as had been given by Ecker, and he believed that the credit of discovering certain peculiarities belonging to the brains of criminals did not belong so much to Benedikt or to the German school, but rather to Lombroso of Italy. Their general conclusions are very much the same—that the brains of criminals do decidedly differ from the normal, but the differences are not sufficient to account for the types of crime so frequently manifested.

At present anatomy and physiology do not exactly fit each other, but perhaps with increasing knowledge of the functions of different parts of the brain these small differences, now regarded as unimportant, may be finally regarded as important, and then we need not look further than the brain to explain the differences in action between the normal sane man and the criminal.

Dr. W. R. BIRDSALL, of New York : The address of our President has brought up so many interesting points in connection with this vast subject, that one hardly knows what to leave out and what to mention. I simply wish to refer to a point with regard to Benedikt's conclusions. Some years ago, when his book first appeared, I had the opportunity to read it, and to write a review of it, and in that review I criticised his confluent-fissure theory as representing a type of the criminal class. It seems to me now that that has been the general opinion with most neurologists ; that is, that these views are hardly warranted by the facts. I believe, however, that the stimulation to study which Benedikt's book gave has been beneficial. The view, however, that we can look, to any great extent, for the origin of moral defects represented in criminals, to the variations in the convolutions of the brain, seems to me to be a narrow one. That certain classes of criminals do have brains which represent a peculiar and imperfect development, must be granted ; but that any particular type can be represented, the President has already opposed, and I agree with him in that particular.

I think, however, that the study of the cranium as to the different types of skull in different races, and their relations to fissures and convolutions, should be more thoroughly done than it has been. Of course, the immense number of brains which must be studied before definite results can be reached only shows how much must be done before reliable data can be reached.

Dr. B. SACHS, of New York : Greater importance must be attached to the development of primary than to secondary fissures ; the latter are controlled by mechanical influences.

The question suggests itself with reference to the brains presented by the President: Whether there was any peculiarity about the skulls in these cases.

The subject is undoubtedly a large one, but before we can decide with regard to mental conditions and their relations to fissures and convolutions, we should know first the dependence of these variations upon simple mechanical conditions.

Dr. WILDER: I think it is proper to say that the discussion should apply to primary fissures only. I do not think that we shall gain much in speaking of secondary fissures.

The PRESIDENT: With regard to Dr. Wilder's remarks, I would say that I fully agree with him that it would be better to harden brains within the skulls, and to prepare our specimens in that manner. But in many cases it would be impossible to apply this method. With reference to two of these specimens, they were obtained under *some* difficulties, and one or two of them under *great* difficulties.

I would say here that the fissure of Rolando, in one case, was crossed by a convolution instead of being confluent with another fissure. It is true, as he has said, that many brains are peculiar. This fact may be due to various causes. First, the majority of brains which are obtained for study are from persons of low mental capacity, and often of a low moral organization. I wish to impress the fact that the peculiarities in these brains are decided and unusual.

As to the Chinaman's brain, whether I would have been able to say that it belonged to a different race from the Anglo-Saxon? No! But my attention was directed to a peculiar appearance which seems to be different from that shown by the others, although I was unable to express it technically. The only special difference noticed first by Dr. Parker,—and I would be pleased to have Dr. Wilder examine the brain further and then tell us whether or not he believes that it is present—is an unusual obliquity of the orbital surface, which attracted attention.

It is wonderful how few brains of different races have been carefully studied.

With reference to Benedikt, while we should criticise him seriously, we should not be so absolutely severe as I have now and then known some to be. Many things which he says are facts, but I would say that he has a bad way of generalizing.

As to the brains of criminals, I believe that those of a certain class do give us a peculiar conformation and development.

As to standards of comparison, referred to by Dr. Gray, we need some one to carry on a work of that kind. It seems to me that the method mentioned by Mr. Jastrow would be interesting, nevertheless it would be questionable whether putting together a number of brains and getting a composite picture, would not be as tedious as the study of a number of brains and then getting a diagrammatic standard. Dr. Wilder has already answered Dr. Sachs' remark with regard to primary and secondary fissures. As to the skulls which held these brains I have a cast of that of Burk, but no observations were made with reference to the skulls in the other cases.

The Association then adjourned to meet at 3 P.M.

Wednesday, first day, afternoon session.

The Association was called to order at 3.10 P.M. by the President.

Dr. L. C. GRAY, of Brooklyn, read a paper entitled "A Case of Lesion of both Temporal Lobes Producing General Loss of Memory of Events without Word-deafness and without Deafness." (See JOURNAL,[1] vol. xiii, p. 554.)

Remarks on Dr. Gray's Paper.

Dr. PHILIP ZENNER, of Cincinnati, O.: I would like to ask Dr. Gray to what extent the cortex was affected?

Dr. GRAY: Both temporal lobes were softened and the layer of gray matter was evidently much thinner than in other portions of the brain.

Dr. ZENNER: Was there any affection of speech?

Dr. GRAY: I am told that there was not the slightest; there was no lesion whatsoever of the faculty of speech or the perception of words; the patient's letters did not give evidence whatsoever of any disturbance of this kind.

Dr. ZENNER: I would not like to accept a case of this kind as one which refutes the ideas almost generally accepted, that one of the temporal convolutions is the seat of the reception of speech, if I may so speak; that is, that lesion of one of the temporal convolutions usually causes word-deafness.

The reason why I would not like to accept it as conclusive evidence is because there have now been so many favorable reports. So many cases of this kind have been reported in which the lesion has been situated here that it seems fairly established

[1] Refers to JOURNAL OF NERVOUS AND MENTAL DISEASE.

that it has a causal relation to word-deafness. But we cannot say exactly to what extent this convolution was destroyed in the case reported, and the meningitis does not necessarily destroy the functions of the cortex.

With regard to the temporal convolutions being the seat of hearing, that has been more or less definitely established, and that is one of the questions in physiology now quite generally accepted. But there is nothing, so far as I know, in pathology which would establish the view that the temporal convolutions are the seat of hearing.

Dr. V. P. GIBNEY, of New York, then read a paper on "Pseudo-hypertrophic Paralysis" (JOURNAL, vol. xiii., p. ~~357~~). 572

Remarks on Dr. Gibney's Paper.

Dr. GEO. W. JACOBY, of New York: Last winter I had the pleasure of administering to a case of pseudo-paralysis. At that time I looked up the literature of the subject quite carefully, and then I found in the *Glasgow Medical Journal*, for 1884, seventeen cases reported, and in none of those cases were there any changes in the spinal cord, so that the report in Dr. Gibney's case would be unique in that particular.

On the other hand, the muscles exhibited changes well known—fat in the muscular spaces, shortening of the muscular fibres, and increase of connective-tissue. But in the same patients there was no reference to the condition of the motor nerves of the muscles, except in one, and in that there was atrophy of the terminal filaments of the motor nerves. I would ask Dr. Amidon if he has examined the muscles in this case.

Dr. AMIDON answered that Dr. Gibney submitted a portion of a dorsal muscle for examination, but that he had not yet examined it.

Dr. SACHS, of New York: The subject of Dr. Gibney's paper is one of interest at this time, as diseases of the muscles are made the subject of very careful research.

In answer to some of the questions suggested, I wish to say that in contradistinction to the Charcot school we should take into consideration what Erb and his students have done in this field, and I would simply refer to some of the points to which he has called attention. I presume it is well known that in addition to the pseudo-hypertrophic paralysis which is common in the young, there is another form of muscular disease which has been

made out, and which is known as the juvenile form, as Erb calls it, of muscular atrophy ; and these two forms are classed under the term "progressive muscular dystrophy." He claims that we must distinguish between certain spinal-cord troubles and purely peripheral muscular troubles.

The ordinary form would belong to the purely muscular affection ; whereas the juvenile form of muscular atrophy would probably be classed as muscular atrophy due to spinal-cord affections.

There is a clinical point which would aid in making the differential diagnosis, and that is the electrical reactions in the case. In those cases where the peripheral muscles are affected only, the electrical reaction may be slightly diminished or increased, but not altered in the sense of the reaction of degeneration, as they would be in the cases of spinal-cord disease.

This entire question has an additional interest with special reference to another muscular disease known as Thomsen's disease.

There seems to be little or no doubt that all these diseases have certain points in common. It is a fact also that so-called peripheral muscular troubles exhibit changes in the muscles themselves. So far as I am able to make out, the changes are myositic in nature. Whether there be changes in the spinal cord also, remains to be seen.

Dr. ZENNER, of Cincinnati, O.: Of course, in the muscular nerves and the gray matter of the anterior horn, we have one single system, and it is important to know in what part the disease may be situated. Disease of the ganglion cells will produce secondary changes in the muscles and nerves, and we know now that disease of the muscles and the nerves will produce disease in the ganglion cells, and so it becomes important to determine which is primary.

It occurs to me that, in a case of this kind, where there is a great deal of external disease, disease of the muscles, and probably of the nerves also, that slight changes in the cord, changes apparently only in the size and perhaps the prolongations of the ganglion cells, and no changes otherwise, are probably secondary, and that this extensive peripheral disease is the more important factor.

Possibly, and of course, this is only a surmise, we can discover which is primary and which secondary, by noting where the most

extensive disease exists. In pseudo-hypertrophic paralysis, especially where no changes in the cord have been observed hitherto, it would appear that such slight changes would be the secondary and not the primary condition.

Dr. L. C. GRAY, of Brooklyn : It is easy to accept the views that peripheral lesions may cause central trouble, but usually when they do the symptoms are not purely motor. But there is no decisive proof that peripheral lesion is capable of producing central lesion, except where the suppurative neuritis is very marked and produces a very large peripheral lesion. For instance, in some cases neuritis has been set up in animals by caustic, which has set up a violent suppurative neuritis ; and so have some operations in the human subject, particularly some operations on the testicle and scrotum, and in one case an operation on a deep stricture.

Dr. F. X. DERCUM, of Philadelphia : There is probably no doubt that Dr. Zenner's views are most plausible, and certainly the life of the muscles is dependent upon motor cells, and if there are no more cells and the changes are such as we would expect, it seems to me that it is forced upon us as an inevitable conclusion. If a motor cell constitutes an important organ ; destroying one destroys the other.

Dr. ZENNER : I spoke of changes in the central organs as the result of mutilation of a leg or an arm, giving rise to extensive changes in the muscles and nerves.

The PRESIDENT : Did not Dr. Gibney ask a question as to whether pseudo-hypertrophic paralysis and progressive muscular atrophy are clinically distinct in the later stages ?

Dr. GIBNEY : Yes, sir ; that was one of the questions suggested.

The PRESIDENT : This question has not been discussed, or but very little. I would like to call Dr. Gibney's attention to the fact that one of the brains which I have presented to-day was removed from the body of a young man who exhibited both progressive muscular atrophy and pseudo-muscular hypertrophy. He was one of three brothers, in all of whom the symptoms of the two diseases were joined, and I had opportunity to see these cases from time to time for several years, and removed portions of muscles several times for microscopic examination. A paper on this subject was written and published by I. N. Kerlin and myself in the proceedings of the American Medical Association

some years ago—the year that it met in New York City. At that time there was discussion to some extent on the question of myositis and of the identity of the two affections, and my conviction was that they were the same disease practically, and that they were muscular rather than neurotic in character. Of the identity of the two diseases I am still convinced. In our cases portions of muscles were removed from the calves of the legs and from the back, and in the former the appearances were those of pseudo-hypertrophic paralysis, while in the latter they exhibited the appearances belonging to progressive muscular atrophy.

Dr. J. H. LLOYD, of Philadelphia : I reported to the Philadelphia Neurological Society several years ago the case of a girl who is yet under my care, who combines these two diseases in a most remarkable degree—pseudo-hypertrophic paralysis and progressive muscular atrophy. I have not sufficient details in memory to enable me to report the case fully here, and can only refer to the paper.

I wish to state, with regard to differential diagnosis between cord and muscle lesion, that it seems to me to be an exceedingly difficult thing to do clinically. In the case of the girl, she has great brawny calves, but distinct atrophy—of the forearm muscles especially, with fibillary contractions which are indicative of cord lesions. She also has slightly the reactions of degeneration, etc., and she seems to confute any effort at differentiating clinically between atrophy of the muscles, pseudo-hypertrophy, and true anterior poliomyelitis.

Dr. SACHS, of New York : This case illustrates what is well known, that two diseases occur combined in one person. For that reason it seems to me to be improper to speak of it as belonging to either. Why not speak of it as a case of progressive muscular dystrophy ?

Dr. J. H. LLOYD : I think the entire neuro-muscular apparatus is physiologically united, and that it is exceedingly difficult, if not impossible, to differentiate disease in one part from disease in another. There may be differences, but if you have fibrillary contractions and simple wasting of muscle, with a certain amount of electro-contractility intact, you probably have a cord lesion in which the entire apparatus is not involved, or wholly limited to the nerve centre ; whereas, with the brawny muscles of the calves we have a more decided muscular affection.

Dr. L. C. GRAY, of Brooklyn : I think that clinically there are

three affections. First, progressive muscular atrophy pure and simple ; second, pseudo-hypertrophic muscular paralysis pure and simple ; and third, a mixture of the clinical phenomena belonging to each of the others. It is very often a mistake to assume that a case is one of pseudo-hypertrophic muscular paralysis because there may have been some accident which has made the patient lead an inactive life, while at the same time the processes of digestion and assimilation may have gone on with their usual activity. I have a patient who has grown enormously fat under such circumstances, and the case, at first sight, might look like one of pseudo-hypertrophic muscular paralysis, which I doubt very much if it is. He has been, probably about ten years, almost helpless in his lower extremities and nearly so in the upper extremities, and at the same time has taken large quantities of food.

Dr. R. W. AMIDON, of New York : I would call attention to Dr. Zenner's remark that the lesion is too trifling to be one of the spinal cord, as compared with the changes outside of the cord. But in this case the proportion of ganglion cells is decidedly different from that seen in the normal cord ; the disproportion is very great indeed. The columns of Clarke seem to be about alike.

Dr. G. M. HAMMOND, of New York : I would like to ask Dr. Amidon if such destructive change had occurred, and if it began in the spinal cord, would it not almost necessarily have produced destruction of the contour of the cord ; that is, produced atrophy of the anterior horns themselves ?

Dr. AMIDON : I have thought that this case was very much more like those of progressive muscular atrophy than those of anterior poliomyelitis, and therefore there would not be any caving in of the cord, so to speak.

Dr. HAMMOND : A disease which begins in the cord and produces atrophy of the cells would be sufficient to produce caving in of the anterior horn ; whereas in these specimens the anterior horns seem to be in very good shape, the entire change apparently being in the cells.

Dr. DERCUM : I can hardly see that that point is well taken ; for I have the spinal cord of a case of progressive muscular atrophy in which there is, especially in the dorsal and cervical regions, absolutely no change in its contour, and yet there is marked lesion of the ganglion cells. In some of these cases there is rather an atrophic disease of the cells—simply a gradual disappearance of the cells.

Dr. GIBNEY: I have nothing further to add to the discussion, except to say that I am not sure that the lesion in the cord was sufficient to account for all the symptoms. I was in hopes that that question would have been settled definitely by Dr. Amidon and the other members.

Dr. Sarah J. McNutt, of New York, then read a paper entitled "On a Case of an Infant with Multiple Tumors of the Cerebrum, Probably of Specific Origin."

Syphilitic lesions of young children are rarely diagnosticated. They may occur as (1) generalized sclerosis, (2) miliary granules, and (3) as single gummatous nodules of large size.

In the adult the changes in the liver, the heart, the lungs, or the brain almost always assume the form of tubercles; their progress is slow; they do not appear until a very advanced stage of the disease; and they belong as much by their date as by their character to the tertiary period.

The new-born, on the contrary, present them very early, often even as the first symptoms of the disease. The organs most frequently found affected in hereditary syphilis are the liver and the lungs, and the other organs more rarely present evidences of the disease.

According to Parrot the liver is most frequently found diseased in infants who die six weeks after birth, and it is upon the disease as presented in this organ, that the most reliable observations have been made.

Dr. McNutt reported a case, which was a marked one, of generalized gummatous infiltration; the liver and the spleen contained miliary gummata; in the lungs there was one nodule of the size of two peas; and in the brain there were multiple tumors, two being about the size of small marbles.

As in this case, syphilitic tumors rarely give rise to marked symptoms, and probably this explains why a diagnosis is so seldom made.

The autopsy was made by Dr. T. E. Satterthwaite, and the microscopic examination by Drs. W. H. Porter and W. A. Shufelt. The nodules were found to be composed of an abundant cellular formation interspersed with thickened blood-vessels and cheesy zones. A striking peculiarity of all the sections was a more or less thickening of the blood-vessels, with a marked tendency to a circular arrangement of the cell growth around the swollen and thickened vessels. Most of the cells were of the small round con-

nective-tissue type, with an occasional spindle-shaped corpuscle. Another striking feature of the growth was the fact that the cells filled all the intra-fibrillated and intra-vascular spaces.

Dr. G. BETTON MASSEY, of Philadelphia, then read a paper "On the Cause of Electrotonus, and of the Normal Formula of Polar Reactions." (JOURNAL, vol. xiii. p. 379.)

Thursday, second day, morning session.

The Association was called to order at 10.30 A.M., by the President.

REPORT OF THE COMMITTEE ON ELECTRICAL DOSAGE.

Dr. GEO. W. JACOBY, of New York, read the report of the committee appointed at the last annual meeting.

"The Committee on Electrical Dosage reports that special rules applicable to all cases, for the use of the current strength, or for the length of time to be devoted to each application of electricity, do not exist, and can in the nature of things not be formulated. That all conclusions arrived at by any special investigator or series of investigators, no matter with what mathematical accuracy these conclusions be expressed, can only be arrived at empirically, and are dependent upon private experience and personal views. Therapeutic experience is too weak a foundation upon which to build an edifice of such magnitude as electrical dosage. Individual differences between patients and diseases would also render such a work of investigation futile.

"The committee, however, recommends the employment of all possible accuracy in concordance with present scientific knowledge in the application of electricity. It advocates the use of the measures adopted by the International Electrical Congress of 1881; also the use of an accurate galvanometer divided according to this system. Furthermore, the committee recommends the adoption of electrodes of certain diameters, with their square distinctly marked upon them in square centimeters (Erb's normal electrodes). This is necessary so that the current density may be known, for with a permanent current strength the density is inversely proportionate to the diameters of the electrodes, as expressed by the formula $D=\frac{I}{q.\,(\text{diameter.})}$ Knowledge of the current strength alone is not sufficient to enable us to gauge the efforts of the current upon the body, and therefore the proportion which

the current bears to its place of entrance and exit must be considered. If the current strength is represented by x, and the diameters of the electrodes by y and z, then the density of this current would be $\frac{x}{y \times z}$, or in figures, let current strength equal 6 milliampères, electrodes 6 x 12 cm., or 72 sq. cm., then $D = \frac{6}{72}$ or $\frac{1}{12}$, which means that upon 12 square centimetres of electrode 1 milliampère of current is spread.

"Therefore, it is also recommended that a system of expressing the current used, in concordance with these facts, be adopted, and that a fraction be always used of which the numerator represents the number of milliampères employed, and the denominator the number of square centimetres contained in the electrodes.

(Signed.) "Geo. W. Jacoby.
"W. R. Birdsall.
"R. W. Amidon."

The report was accepted and ordered to be printed in the transactions. It was then discussed by the Association.

Dr. L. C. Gray, of Brooklyn: I think, as this report receives the endorsement of the Association, that some other recommendations should be made; for example, as to what the covering of the electrode should be and what it is to be made of, as it will make a certain amount of difference in calculations, I think. For instance, an electrode of carbon will be much more conductile than an electrode made of sponge, and a metal electrode covered with absorbent cotton must be more conductile than one covered simply with sponge. So I think that these items would enter largely into the calculations, and that it would be well to incorporate something of that kind into the report to make a normal electrode still more useful.

Then I think it would be well to incorporate some suggestions as to what material for wetting the sponges should be employed, as it may make a good deal of difference, whether hot or cold water is used, etc. In using electricity myself I have been accustomed to have my electrical board over a sink which contains both hot and cold water, and my rule is to use water as hot as the patient can comfortably bear it. I would suggest that some such facts should be incorporated. And then I think it should be wisely incorporated as to the use of a galvanometer, and as to what kind of a galvanometer should be employed. There are in use a number of instruments, and there is a good deal of difference as to

which should be used. While we are making recommendations, I think that we should incorporate such little details as these.

The PRESIDENT : I would suggest that there is nothing in the resolution which would preclude the continuation of the committee another year.

Dr. J. H. LLOYD, of Philadelphia : I would like to say a few words on this subject. I have experienced a great deal of dissatisfaction in the use of electricity, especially in gynecological cases, because I do not know what strength of current will produce certain effects. We know that by covering the electrode with different substances a difference in the current strength can be produced, and this difficulty can be readily overcome. But the point to be known is, how much strength of current is required to do so and so ; say, to produce a catalytic action ; to produce, for instance, absorption of an inflammatory deposit in pelvic cellulitis ; or modify various neuralgias ; relieve dysmenorrhœa, etc. It is not what is to be the character of the cell to do these things, or the shape of the electrode, or its covering, but how much current strength do you require to produce therapeutic action. And this is where the Committee has not given us any information, yet it is where we require decided information. It is with reference to this subject that we have so much nonsense written with regard to electrolysis in the treatment of strictures of the urethra. If there is any such thing as electrolysis in strictures of the urethra we should know what current strength is required.

Dr. L. WEBER, of New York : So far as my experience goes with regard to electrodes, their size and kind, I have found that they may be oval, but that they should be, if possible, square. But it may be necessary to make some variations according to the part of the body under treatment. For instance, if the anode is applied to the thyroid, the electrode should be a band adapted to the size of the neck and head, giving a fixation surface, and should occupy a certain portion of the head. If I apply the cathode to the neck I use a long, square-cornered one, smaller or larger, according to the size of the patient.

The oval electrode will be useful, particularly, as far as the electrodes are concerned, when made out of charcoal. If we have the privilege of selecting, according to the statement referred to by Dr. Jacoby, and according to the results obtained by Erb's electrodes, we should have one as near square as possible, smaller or larger, according to circumstances.

As to the quantity of electricity to be used, that is as much an individual matter as any other remedy. There are no laws which can be laid down in this respect. It depends upon the individual and the amount of pain in which he receives the current. Central neurasthenia in one person will get along with two or three elements of ordinary size, while in another five or six or even more will be required. So it is impossible that any committee can be able to lay down any laws with regard to the quantity of electricity to be given. I think nothing better has been written in a long time than Müller's book, and he reccommends daily sittings of short duration and the use of very small currents.

Dr. G. BETTON MASSEY, of Philadelphia: I think that the committee has devoted too much time to the therapeutic aspect of the subject. It seems to me that the point to be gained is the determination of the number of milliampères required to move certain muscles, how many are required to produce a contraction, what the cathode closure is for certain muscles for comparison in cases of other muscles when diseased. I have recently made some experiments with regard to the number of milliampères required to move the extensor of the index finger for instance, taking a number of observations in order to compare them with normal muscles, with the muscles in writer's palsy, progressive atrophy of the hand, etc. It seems to me that a certain number of observations made upon normal muscles to determine the minimum number of milliampères required, would be of great value. According to my experience there is but little difference practically whether we use Erb's fine or Erb's small electrode, although the fine is doubtless preferable for muscles, as it limits the action to the muscles which we are experimenting upon. At the same time I do not wish to be understood as belittling the importance of the size of the electrode.

With regard to the remarks of Dr. Lloyd announcing the desirability of more accurate knowledge as to the measurement of electrolytic effects, I think that we have in the skin beneath the electrodes the nearest approach to the depth to which the current goes, or some idea with reference to electrolysis. If you apply an electrode upon a square inch of surface and send a current through it which shows a density on a galvanometer of ten milliampères, we have a density of ten to the square inch and we can note the electrolytic effects at any point. Suppose we wish to reach a tumor, how much of the electrolytic process can you

bring to bear upon it as compared with that in the skin? This can be determined by calculation. If, for illustration, the tumor has a central area of four hundred square inches and an electrode occupying one square inch of the skin upon the surface of the body is used, the density acting upon the square inch in the tumor is $\frac{1}{400}$ of what it is upon the skin at the surface. The only calculation required then is to calculate for the average spread of the current in the central plane, because the current will flow through these various lines (illustrated in a diagram), but it will be divided unequally between them; those lines which are most direct will carry more current than those which are indirect. But the difference is not so great as represented in works upon this subject; it depends simply upon the difference in length: if twice as long, it will carry twice as much current; consequently the density of the flow is not very great in a direct line, in a deep body, in a large conductor. The density of electrolytic influence to which a tumor or other body can be subjected by the constant current, through unbroken skin or mucous membrane, can be calculated by comparison with the plane of the body at which it lies.

Dr. JACOBY: I think that the primary idea in the appointment of this committee was to obtain a report on electrical dosage, and not to be a committee on galvanometers. If it had been a committee on galvanometers its work would have been less difficult; for it could have selected one of the many instruments and recommended it to the Association. The question of electrical dosage is a matter of personal experience, and such experience is not scientific, and is inaccurate. The mode of action of electricity does not enter into the duties of the committee; Ohm's law covers that.

The reason that no galvanometer was recommended was because the committeee did not wish to recommend the instrument made by any one maker. As to Dr. Massey's suggestion, that a standard galvanometer should be made, it would be difficult to find such an instrument; difficult to find a galvanometer that will remain exactly the same for a year. They will change, and are subject to magnetic influences, no matter how slight, which will lead to error in studying physiological problems; and for therapeutical use we need no such instruments.

As to the amount of current strength required to do a certain thing, if the committee were to inform the Association that a

certain number of milliampères will cure certain diseases, that again would be the result of individual experience simply ; and so far as electrolytic action is concerned the committee was not appointed for any such purpose.

As regards the question of electrical diagnosis, there again you will obtain contractions in different cases with a larger or smaller quantity of current. I do not think that any two cases of progressive muscular atrophy will give the same electrical reactions. Nor do I believe that absolutely normal muscles will give the same reactions to the same strength of current in different individuals.

The report of the committee was purposely that they were unable to do any thing on the question of electrical dosage ; they simply recommended accuracy in the application of electricity ; that is by milliampères and by regulated sizes of electrodes, and that when you do describe the amount of current it shall be in the use of these accurate terms. The question of current density will eventually lead up to it, as one of the scientific accurate methods to be pursued.

Dr. AMIDON, Secretary, then read a letter received by Prof. Dr. Aug. Forel, of Zürich, an associate member, and presented a photograph, which he had sent of a "Microcephalic Girl," fifteen years old, who had three microcephalic sisters who are dead. The father and the mother are normal.

Dr. WILDER'S paper was next in order, but before reading it he took occasion to make a few remarks retracting in part at least what he said concerning the Chinaman's brain, presented by the President at the first session of the Association.

I am prepared to say that it is the most remarkable human brain I have ever examined. And although we have no data which would enable us to say that it was the brain of a Chinaman —nothing in the shape except possibly a slight obliquity of the orbital surface, and which needs further comparison—nothing to point to its Mongolian character, it presents us with most extraordinary features, particularly the fact that the first temporal fissure extends not only as far as it usually does, but it actually sweeps up to the longitudinal fissure and into the mesal aspect of the cerebrum. This is distinctly a monkey feature. I do not think we are justified in making any derogatory comparisons, but it is distinctly one of the features which point to our ape ancestry. I have not seen this so well marked in any other human brain.

Dr. WILDER then read his scientific communication entitled "Notes on the Brain."

1. ADDITIONAL CASE OF INDEPENDENCE OF THE PAROCCIPITAL FISSURE.—In his recent paper on the paroccipital fissure, Prof. Wilder stated that among forty-three reliable specimens and figures accessible to him, the paroccipital is continuous with the

parietal in twenty-one and independent in twenty-two. The brain of a negro child at birth lately prepared by him had the two fissures wholly independent on the right and barely united by a shallow junction on the left.

2. AN ENTAL CORRELATIVE OF THE OCCIPITAL FISSURE IN AN EARLY FŒTUS.—In a fœtus estimated to be about six or seven months old, as shown in an accompanying photograph and figure,

opposite the occipital fissure there was a distinct ental ridge, so that the entire thickness of the wall was there folded. A similar ridge is figured by Tiedemann, but not named or described. Without further observations, it cannot be determined whether it disappears in the adult or persists as the more or less distinct elevation known as the *bulbus cornu posterioris* or *eminentia splenialis*. Even if transitory, it adds another to the list of "total fissures," including the calcarine, hippocampal, collateral, and Sylvian.

3. THE FŒTAL EXTENSION OF THE PROPLEXUS TO THE END OF THE POSTCORNU.—This is the case in a fœtus estimated to be five months old. The postcornual extension is apparently in process of atrophy. There were indications of a like extension into the precornu. These extensions might be expected in view of the great volume of the plexus at a still earlier stage.

4. POINTS ILLUSTRATED BY THE TRANSECTION OF A FŒTAL BRAIN.—Six points of morphological interest were noted, most of them indicating that the thalami increase in width as development proceeds, so that, in the adult human brain, they form, or seem to form, part of the floor of the procœles or lateral ventricles, which is not the case in other mammals, excepting, perhaps, the primates. (See also JOURNAL, p. 463, *et seq.*)

Remarks on Dr. Wilder's Communication.

Dr. L. C. GRAY, of Brooklyn: I was under the impression that some authors have made mention of the fact as to the thalamus not coming to the surface, and I was a little astonished to hear that such was the normal fact. I think it is alluded to in the recent work by Meynert and quite fully brought out.

Dr. WILDER: I will not deny that it is not so, but I have examined those works, as I have also almost all other works, and while it may be that it has somewhat obscurely been called attention to. I do not think that it has made sufficient impression upon anatomists to form a part of anatomical instruction. And I do not wish it to be supposed that I bring it forward as a new fact, because I spoke of it two years ago. But the point now is that I have seen it in the fœtus, and it has been claimed that no such thing occurs in the fœtus. Hadley and one or two others have called attention to the fact that the endyma is continuous over the surface of the thalamus, but nine out of ten teachers talk about the thalamus as a part of the floor of the lateral ventricle.

Dr. GRAY: I suppose it may be said that thoroughly accurate anatomical descriptions in text-books of the internal anatomy of the brain and basal ganglia do not exist.

Dr. WILDER: Quain may be taken as a type of the high-water mark of English anatomy. But I think, while it is amusing on the one hand and melancholic on the other, that he states the fact of the continuity of the endyma over the thalamus, the only figure in the book representing a trans-section of this part of the brain, leaves the student totally in the dark, and there is no attempt apparently to indicate that the endyma is continuous over the thalamus, but there is a vagueness which will not enable the student or teacher to make out what the figure is intended to illustrate.

The PRESIDENT: With reference to the nomenclature in connection with Dr. Wilder's first remarks on the "paroccipital" fissure, and also with reference to the physiological point coming out of the brains examined, it seems to me to be important to keep the developmental features in view in naming the various parts. Keeping such names as bridging convolutions and similar terms is of importance.

Then, with regard to the interparietal fissure, which Ecker represents as a single continuous fissure, as it is in apes and often in human beings.

Dr. WILDER: I must take issue there. It has not been seen in fœtal human brains.

The PRESIDENT: I think it has been so reported; the interparietal fissure passing back as a continuous fissure to what is commonly called the transverse occipital fissure, is a condition which I am sure I have often seen, but I do not say in the fœtus.

With regard to the difference between the left and the right side of the brain in a case of this kind, the view which I would take is that it can be explained upon a physiological and developmental basis.

Dr. WILDER: Supposing it to be true, does it not indicate a greater amount of cortex upon this side?

The PRESIDENT: Certainly it does.

Dr. WILDER: In other words, the fissure represents more cortex than the corresponding one of the other side.

The President presented the brain of a baboon, and also that of a new-born negro child, sent to him by Dr. Formad, of Philadelphia.

Dr. L. Weber, of New York, read a paper on "The Psycho-Neurotic Affections which Accompany and often Mask Phthisical Disease" (See JOURNAL, vol. xiii., p. 455, 1886).

Remarks on Dr. Weber's Paper.

Dr. L. C. GRAY, of Brooklyn: I think Dr. Weber's experience has been that of most of us who have seen many cases of tubercular nervous disease; in fact, the nervous symptoms preceding the detection by auscultation and percussion of pulmonary trouble.

I know that has been my experience in a large proportion of cases. In many cases also it has puzzled me so much that I have taken pains, and have sent my patients to gentlemen who have made a special study of auscultation, to see if they could detect any evidence of pulmonary disease.

Now, I suppose there is such a thing as tuberculous poisoning of the blood, the same as the syphilitic virus poison, long before it produces any organic changes, and long before it produces those changes in the small vessels of the pia mater which are particularly involved. I know of one case which is very interesting as an illustration of this fact, a case which I saw in consultation, of well-marked pulmonary tuberculosis. The individual began to manifest mental symptoms, and the question was with regard to the nature of the trouble. Death occurred, and the post-mortem examination was made by Dr. Leuf, whose reputation as a pathologist is well known, and who is competent to speak, and he found no changes whatsoever in the brain, although the person was delirious for four or five days before death. No microscopical examination of the walls of the blood-vessels was made.

With regard to treatment, I think that it is looked at in entirely too despondent a way. I have not seen a case cured, perhaps, but I have seen patients, for a time, have an absolute intermission of symptoms, produced by means of careful treatment. In a number of cases the symptoms have ceased and the patients have enjoyed excellent health, but in some cases they have relapsed.

I have found that the best treatment is some modification of that recommended by Weir Mitchell, of Philadelphia. Of course the patients cannot be placed in bed, but you can carry out the principles which underlie this treatment. The principle of absolute restriction of expenditure of energy to the greatest possible degree, and giving the largest quantity of food which the patient

can take, and that will be increased enormously if you restrict the expenditure of energy. If I can get these patients in the early stage, my prognosis is usually a good one.

Dr. WEBER : I am glad Dr. Gray has emphasized what I drew attention to in the therapeutic part of my paper, namely, the great importance of hygienic measures, particularly of the feeding of the patient. I would say in this connection that abroad, particularly in Germany, they are well aware of Mitchell's treatment of nervous disorders, and there are half a dozen institutions established already on that plan, and connected with large cities.

As to what can be accomplished by proper hygienic measures, by feeding or over-feeding in cases of tuberculosis, and by looking after the welfare of the patient, particularly in going into the minutest details, I have seen nowhere better examples than in a sanitarium near Frankfort. A similar institution has been established by Brehmer in Silesia, and most remarkable therapeutical results have been obtained.

Dr. JOHN VAN BIBBER, of Baltimore : I have had some experience in the treatment of melancholia, and I am accustomed to have the lungs examined most carefully. I fully agree with what Dr. Weber and Dr. Gray have said with regard to the desirability of appreciating properly the therapeutic measures that can be adopted in the treatment of melancholia, or the lung trouble which produces some mental derangement. And I should like to say here that I have used, with good results in such cases, the Sanguinis Bovinis Exsiccata prepared by Parke, Davis, & Co., as a concentrated food ; in some cases the results have been remarkable.

Dr. RALPH L. PARSONS, of Sing Sing : Having had charge of a number of insane patients, I have observed an intimate relation between melancholia and tuberculosis, to such an extent that for years, at regular intervals, I have carefully examined all the persons under my care for pulmonary difficulties. The frequency with which these patients will become phthisical without any thing except physical signs to indicate the existence of the pulmonary disease—even to the second and third stages—is remarkable.

Dr. PHILIP ZENNER, of Cincinnati, then reported a case of " Auctioneer's Cramp," and presented the patient.

This was a professional neurosis, but differed from others in affecting some of the muscles of articulation. No similar case had been reported, so far as the speaker knew. He termed the

disease "auctioneer's cramp," on account of the occupation of the patient. The latter was forty-five years of age, and had always been in good health previous to his present trouble. He had been an auctioneer for many years, and applied himself with unusual diligence to his business. In March, 1885, he was confined to his house for a month with an ulcer of the leg, but during this time his general health was good. At the first sale he cried after returning to his business he observed a slight but scarcely noticeable difficulty of articulation. This occurred only when a word—as five—was frequently and rapidly repeated, as often occurs in crying a sale. This difficulty rapidly increased in succeeding sales, so that after crying four or five more he was compelled to discontinue them altogether. He now began to observe that something of the same character of disturbance would occasionally occur in ordinary conversation, and he would have a sense of painful fatigue about the left corner of the mouth, which soon became an almost continuous sense of discomfort. The speaker first saw the patient about a month after the beginning of his trouble. He was under his observation then for about a week, and has been again the last few months. His condition during that time was about as it is at present, though the symptoms seem more marked at the present than any previous time.

The patient was then presented to the Association, and his condition demonstrated. In a state of repose there is nothing noticeable excepting that the naso-labial furrow on the left side is a little deeper than on the right, and the left angle of the mouth seems a little drawn in, as if pressing against the teeth—a condition due to a slight tonic contraction of the muscles. Usually there is nothing special observable in ordinary conversation, so that many of his acquaintances have never noticed any thing peculiar about him. But occasionally, when he speaks, a contraction of the muscles on the left side of the mouth is noticeable. The special symptoms are brought out by rapid speech. When he repeats the word five rapidly, as in crying a sale, a state of spasm of some of the muscles ensues, which soon makes further speech almost impossible, though the spasm ceases when the effort is discontinued. During this effort the muscles of the left corner of the mouth, chiefly the orbicularis oris, contract very forcibly, pressing it firmly against the teeth, and at the same time the muscles on the right side draw the right corner of the mouth upward and to the right. The chief spasm is in the muscles at the left

corner of the mouth. This part feels, to the examiner, as hard as a board, and at this place the patient has a feeling of very painful fatigue. The upper lip and part of the face takes no part in the spasm, and the upper lip and cheek on the left side of face are flaccid, while the right corner of the mouth is drawn upward and outward, that it appears as though the left side of the face were paralyzed. But this is easily disproved when the state of spasm has passed away. The patient can then whistle, expose the upper or lower teeth, as well on the left side as on the right, or make any movements of this kind forcibly when resistance is made by the examiner.

The patient can prevent these spasms by a very simple measure, lifting the left corner of the mouth during the act of speaking. When he does this he can repeat the word five rapidly without any facial spasm ensuing. The speaker thought this act prevented spasm through keeping the orbicularis oris, the muscle chiefly affected, in a state of extreme extension.

During the facial spasm, and for a short time afterward, the patient has a very painful sensation, a sensation like that of fatigue, especially at the left corner of the mouth, but extending beyond this. But a certain sense of discomfort is more or less constantly present on the left side of the face. Also after the facial spasm passes off, one may observe at times slight contractions of the muscles about the left eye and cheek, contractions almost fibrillary in character.

The patient suffers a good deal with mental depression, general nervousness, etc., but further than this there is no sign of disease. The fundi oculi and patellar tendon reflexes are normal; no paralysis of sensation or motion can be detected anywhere, etc.

Only the orbicularis oris was carefully tested with electricity, as this was the chief muscle affected with the spasm. The muscular contractions were sharp and quick like those of normal muscles, and were produced when the electrode was applied to the muscle or to its nerve. There seemed to be lightened faradic contractility on the left side, and heightened sensibility to the current. Tested with the galvanic current, the anode closure contraction was produced as quickly as the kathode closure contraction; otherwise the reaction appeared as normally.

The speaker had classed this case among the professional neuroses, because it has the distinguishing features of that group of diseases, its chief or only cause being the excessive use of the af-

fected muscles, and the special symptoms being evoked only when those muscles are called into play by a voluntary act. The patient believes there was some further cause than excessive use of muscles, catching cold, or the like. But no other cause has been found.

The speaker believed in this case the cause was the excessive use of a few muscles. The crying of a sale consists largely of the frequent and rapid repetition of certain words, as five, five, five, etc., etc., which keep in almost constant activity a few muscles, while there is not a continual change in muscular activity as in ordinary conversation.

The treatment has consisted chiefly of the use of the galvanic current and internal administration of arsenic and iodide of potash. The patient has discontinued his crying of sales, but it seems as if mere conversation kept the affected muscles in sufficient activity to maintain the constant sense of discomfort in the face, and to prevent a cure. If other measures will not avail, the speaker proposed the stretching of the left facial nerve.

Remarks on Dr. Zenner's Case.

The PRESIDENT : This case is a highly interesting one, and we are greatly indebted to Dr. Zenner for having brought it before the Association. I am inclined to the view that it is a real professional neurosis, although it opens up several points for discussion.

I might relate a case, which I think has not been published, similar in character. The gentleman was one of Dr. Charles S. Turnbull's patients. He was an actor and was compelled to put his face into peculiar positions, and in consequence of keeping his face in these forced and distorted positions—that is, we concluded it was in consequence of this—he came to Dr. Turnbull with paresis of the external rectus and distinct double vision. There was this peculiarity about it, namely, that the man, while vision was distinctly double, could with increased voluntary effort pull the eyeball around—he could throw power into the affected nerve muscle. This shows the truth of the view that some of the ocular movements are due to true central control of a peculiar kind. That patient has almost entirely recovered, although his difficulty lasted for some time. The case presented by Dr. Zenner suggests several things which are similar to what was seen in Dr. Turnbull's case.

The secondary contractures of Hitzig are well known—in facial paresis we have the occurrence of secondary contractures, or a combination of tonic and clonic spasmodic movements, which are apt to be continuous and difficult to alleviate. The appearance of this man's face is precisely like contracture which I have seen after facial paralysis.

It is not impossible that there exists a slight peripheral neuritis, and it may have been the starting-point of the special phenomena now present, but the character of the spasmodic movement has been implanted by the habit to which these muscles have been subjected. It shows that it is possible that there are cases of central overwork, volitional overwork, but such cases are more likely to occur amongst those of other employment than auctioneering, where the movements are almost automatic.

I believe that the line of treatment suggested by Dr. Zenner is a very good one. Certainly the indications would be to give complete rest to the centres, to restore the nutrition of the parts affected, and to diminish the mental irritability as far as possible. The last suggestion should be resorted to, namely, that of stretching the facial nerve, but all other measures should be used first. Static electricity and galvanism would probably be beneficial, but faradism would be likely to do harm, or at least would not be so likely to do good. The cautery to the face or neck might also be used.

Dr. L. C. GRAY, of Brooklyn: I think the diagnosis in this case lies between a cortical lesion, polio-encephalitis, and lesion of the facial nerve. Of course the differential diagnosis would have to be made by more careful electrical examinations than have been made, because there might be some slight difference between the two sides with the galvanic current and some possible alteration in the progression of the poles which would throw some light upon the subject. I think that the man is paralyzed on one side of the face. I do not think he closes his eye as firmly upon one side as upon the other, and when the eyes are open there is a distinct paralysis of the lower portion of the face. When he closes his jaw he overcomes the resistance upon the right side, but he had to make quite a distinct difference upon the left side. There is a distinct difference in the motor power upon the two sides.

The case differs from the ordinary forms of writer's cramp at all events. I should recommend the trial of the galvanic current, trying the positive pole. In the treatment of the superficial

muscles of the face I believe in the special efficacy of the positive and the negative poles. I would also recommend the use of the instrument of Adamkiewicz, and the application of an anæsthetic through which the current is passed, and by which you carry the liquid into the tissues. There has been much dispute as to whether that can be actually done ; but I know that it can be done, as I have obtained complete anæsthesia with a ten-per-cent. solution of cocaine in the parts surrounding the poles.

Dr. L. WEBER, of New York : In looking at this case and examining it, many points must be taken before the examination is complete. But certainly the case does not impress the impartial observer as one which began as a neurosis, because it is unilateral, the muscular movements are not tonic, but rather clonic, and are more secondary, as are often seen in post-hemiplegic affections. The activity of the entire right side of the face is undeniable. A positive paralytic condition is present on the left side, and whatever there is interesting as a case of neurosis, there must have been a certain amount of neuritis with regard to the left facial. Even the patient has the recollection that he felt a lameness on the left side of the face before these peculiar spasmodic contractions of certain muscles occurred.

Therapeutically, be it a combination of a neurosis and a neuritis or not, the galvanic current is indicated, used in such way, perhaps, as Dr. Gray has stated.

Dr. ZENNER, in closing the discussion, remarked that he would not deny that there was a neuritis present, but there had been no evidence of it—no paresis, and no reaction of degeneration. Whether a neuritis could be detected or not, would not affect our considering the case a professional neurosis, for such a classification is based chiefly upon the cause of the symptoms. The true nature of the professional neuroses, at least in most instances, is obscure, just as it is in this case. In some instances of writer's cramp a neuritis has appeared to be the cause, but the diagnosis of such a condition does not necessarily remove it from the group of professional neuroses. Possibly such a condition is the basis of them all. The apparent paralysis of the left side of the face, during the facial spasm, is not real. That the muscles retain their power is easily shown when the patient is in a state of calm, but not so easily shown in his excited state when before so many physicians, though it was then shown how he could expose his teeth equally on all sides, etc. The speaker had examined him very

carefully for paresis of the facial muscles, especially the first time he saw him, but never detected any.

The Secretary then read the following translation of a letter from Prof. Gudden, made for him by Dr. Jacoby.

MUNICH, July 15, 1885.

Highly Respected Colleague :

Herewith acknowledging the receipt of your letter of the 30th of June, I beg of you to transmit my best thanks to your respected Society for the honor which they have done me in electing me to their corresponding membership.

With highest respects,

GUDDEN.

The Association then adjourned to meet at 3 P. M.

Second day, afternoon session.

The Association was called to order by the President.

Dr. BURT G. WILDER, of Ithaca, exhibited a living frog which was decerebrized more than seven months ago.

The animal had enjoyed perfect health ever since the operation was performed, which was on the 9th of December, 1885. He had been fed on small fish or pieces of meat twice a week, but two persons were required to feed him, as he would not open his mouth voluntarily. Just here a curious phenomenon presented itself ; that is, the frog did not know any better than to attempt to do two things at the same time, which were the reverse of each other in design. If a piece of a minnow's tail projects from his mouth, he would make an effort with one or both of his fore feet to remove the fish from his mouth, while at the same time he endeavored to swallow the other end. He would change his position slightly, would balance himself, wink with one eye, make the retrograde movement, and when irritated would wink with both eyes. Dr. Wilder queried whether the frog ever slept, whether he could be hypnotized, etc. It was a question also whether he was capable of any kind of education. He would lie upon his back, although not hypnotized. It would also be interesting to know whether or not a pair of such frogs could procreate, or whether this frog was capable of procreating.

Dr. SACHS, of New York : With regard to some of the questions suggested by Dr. Wilder, there is one essential difference between the manner in which he has decerebrized his frog and the manner

in which the operation is done in Goltz's laboratory. There, nothing is removed; the parts are simply severed subcutaneously. Dr. Wilder's method is much more thorough, and I am surprised that the frog has lived so long. There is one point which corroborates what Goltz has found, and that is with reference to laying the frog upon the back; he noticed that, and also that if the legs be drawn out slowly they will be retained in any position, however uncomfortable that may be. This it is also possible to accomplish in functionally decerebrized animals of much higher order than the frog.

Dr. JASTROW, of the Johns Hopkins University: I can answer one question suggested by Prof. Wilder, and that is with reference to hypnotism, or rather catalepsy. I have a series of frogs which were operated upon in the following manner: First, normal; second, frogs in the condition of Dr. Wilder's; third, with the optic lobes also removed; and fourth, the ordinary reflex frog, with every thing removed above medulla. The very last thing that a frog gives up, is lying upon the back. None of these frogs, except the reflex frog, will, under ordinary conditions, submit to being laid upon the back.

Apparently this action, whether it is an hypnotic influence or not, will be left out of account. At all events this cataleptic condition remains as long as any thing of the brain is left at all. Furthermore, these frogs can be etherized.

Dr. WILDER: Did Dr. Jastrow succeed in cutting across the brain between the cerebellum and optic lobes?

Dr. JASTROW: Of course you cannot always be certain that this condition has been obtained.

Dr. WILDER: Has the condition of the brain, in such experiments, been observed to see whether there is any repair or reproduction? has there been any indication of reunion after transsection made subcutaneously?

Dr. JASTROW: I know of nothing with regard to the frog, but with regard to dogs it has been carefully studied, and no such union has been observed.

Dr. GRAY suggested the acetic-acid experiment, and Dr. WILDER said that it had been applied with the usual result.

Dr. JASTROW: I accidentally found a frog with one leg bitten off, and when the acid was applied to his back, he not only attempted to brush it off with the sound leg, but when that was held, he made the same attempt with the stump of the other leg.

Dr. C. L. DANA, of New York, then read a paper on "Pseudo-Tabes from Arsenical Poisoning."

The object of his paper was to report two somewhat unique cases of arsenical paralysis, presenting the symptoms of tabes dorsalis; next, to show that arsenical paralyses, like those from diphtheritic poisoning and alcohol, present two types, one of which might be called the mixed or ordinary form, and the other the ataxic form; and to show also, if possible, that the ordinary teaching that arsenical paralysis is due to a diffuse myelitis is not correct, and that these paralyses are really the result of a multiple neuritis. The conclusions arrived at were: 1. That a disease resembling locomotor ataxia may be caused by arsenic given medicinally, absorbed from wall papers, or taken in a single large dose. 2. That arsenical paralysis of this type and arsenical paralyses of other types are not due to a diffuse myelitis, as has been taught, but to a multiple neuritis. 3. That arsenical paralyses, like those from diphtheria, alcohol, and probably other infections and poisons, are of two types: *a*. The ordinary mixed, motor, and sensory paralysis; *b*. the pseudo-tabetic form, in which there is no pronounced motor paralysis, but marked sensory troubles, especially ataxia.

Remarks on Dr. Dana's Paper.

The PRESIDENT: The subject of arsenical paralysis is one in which I have been practically interested. Dr. Dana has had the kindness to refer to a paper which I wrote several years ago, which was based upon careful examination of one case that I saw in consultation, and afterwards, through the kindness of Dr. Mitchell, had opportunity to study it for months until finally the patient recovered; and also the study of the history of other cases which were affected at the same time. Since that time I have had other cases of arsenical paralysis. One of these cases I reported in a series of lectures delivered on the differential diagnosis of myelitis.

In the paper referred to by Dr. Dana I expressed the opinion that the evidence was in favor of diffused myelitis, but I also stated there what I wish to restate here, and enlarge upon, namely this point, that in cases of so-called arsenical poisoning, in the cases which I have seen, not only the nervous system has been affected, but other parts of the body have also been affected, as in cases of poisoning by mercury, or through the infectious diseases,

such as diphtheria, etc.; and that the true light in which to look upon the subject is not to regard these cases as either instances of myelitis or neuritis or myositis, but, taking the idea expressed first by Ringer, to consider that they are cases of protoplasmic poisoning, affecting not only the nervous protoplasm, but the tissues of other organs in other parts of the body. Therefore, while Dr. Dana asserts his reasons for regarding the disease as a neuritis, I wish to recede from the ground, if I ever took it, that it is solely a myelitis. This is true of a great many of these diseases. Just now we are in an era of multiple neuritis, and able investigators have proved sufficient to show that there is truth in these views. Take a case of alcoholic paralysis. Dr. Sinkler will recall a case which was also seen by Dr. Reed, and which was variously diagnosticated by different physicians, all men competent to reach a correct diagnosis. This man, however, after having all the spinal symptoms of arsenical poisoning, after a time developed cerebral symptoms, and then it was called a case of paretic dementia; he probably developed an encephalitis. Take the cases referred to in my paper on arsenical paralysis. One died with all the symptoms of diffused encephalitis; one or two were nearly permanently paralyzed with chiefly spinal symptoms; others had more or less symptoms indicating neuritis. Now what we wish is more satisfactory general considerations. Dr. H. C. Wood holds largely to the doctrine of neuritis, in connection with cases of this kind. If you come to a strict analysis of these cases the evidence of neuritis depends upon one or two things, especially pressure upon nerve-trunks. My own experience is that we have a diffused hyperæsthesia in many cases not neuritic, and this may be mistaken for nerve pain. A hyperæsthetic condition is undoubtedly present in subacute myelitis of the anterior horns, of Duchenne. I simply throw out this point for discussion.

Dr. GEO. W. JACOBY, of New York: I think that if we should look for similar cases as the result of poisoning they would be found. I once wrote a paper on lead-poisoning, and I reported in it a case of tabes due to poisoning by lead, and it was almost impossible to make a differential diagnosis between it and true tabes. The only astonishing part was that the man had paresis of the extensor muscles of the middle fingers of each hand, and ultimately the case was diagnosticated as pseudo-tabes due to lead-poisoning. I then expressed the opinion that I was more inclined

to the view of ataxy being due to lesion in the cord, if lesion it might be called, but I would now report it as a case of neuritis, due to lead-poisoning, and I think that Dr. Dana's ideas, in the main, are correct on that point. I would also add that the patient is absolutely well now, with the return of the patellar reflex.

Dr. B SACHS, of New York : I rise to enforce one point : We speak of these cases as arsenic and lead cases, apparently implying that they are all very much alike. That would seem to imply that the pathological lesion would be similar in all these cases. The analogy has been referred to between these cases of arsenical and lead-poisoning on the one hand, and on the other the poisoning of diphtheria and other infectious diseases. We know with regard to diphtheria that its poison may attack different parts of the body. Now it seems to me unreasonable that the effect of arsenical and lead-poisoning should always be exhibited at one and the same place in the body, or in any one organ only. I do not see why the infection may not affect the peripheral nerves in one case and the spinal cord in another. A large number of investigators have found in post-mortem examinations distinct lesions of the anterior horns of the spinal cord in lead-poisoning, and an equal number of other reliable observers have found lesions of the peripheral nerves. I cannot see why arsenical poisoning may not be productive of myelitic changes in one case, and of neuritis in another. The clinical symptoms will be very much alike, and cases of one kind have been mistaken for those of the other very many times. I would furthermore call attention to the fact that in cases of genuine tabes changes have been found *post mortem* in peripheral nerves. Recent experiments would lead us to suspect that many peripheral symptoms of tabes, such as pain, are possibly not due to change in the spinal cord, but rather to changes in the peripheral nerves. I would ask Dr. Mills for his opinion with regard to the toxic agent affecting different organs and parts of the body.

The PRESIDENT : I would say that the position which I hold practically is that which Dr. Sachs has just expressed. I am not attacking the view of Dr. Dana. We have neuritis in some of these cases, perhaps in the majority ; but I wish to take the position that arsenical poisoning, or poisoning from lead or mercury, or from infectious diseases, may give us neuritis, instead of myelitis or encephalitis by preference perhaps, but that they may give us one or all of these affections.

Dr. DANA: I perhaps put my cases rather more strongly than I felt myself, for the reason that the ordinary teachings are that arsenical paralysis is due to diffuse myelitis, so far as I could find them.

When I first saw these cases of arsenical poisoning I made up my mind that they were cases of neuritis. I then looked up the literature of the subject and found that Dr. Seguin and Dr. Mills and others did not believe that they were cases of neuritis. Therefore I put my cases rather strongly, perhaps. I believe that the main thing to be kept in mind is the fact that there is a form of tabes, resembling locomotor ataxy, which may be caused by arsenic, and that it may be mistaken for locomotor ataxy. Consequently I wish to bring out the fact that, so far as evidence goes, in the cases of arsenical poisoning these symptoms are mostly due to multiple neuritis. I did not say that all cases were due to neuritis; I do not suppose that that has been proved; but I think that the views expressed by our distinguished President and others, that mineral poisons sometimes affect the central and sometimes the peripheral parts, are plausible, but it seems to me that in most cases it is a peripheral neuritis.

Dr. Wharton Sinkler, of Philadelphia, then read a paper on "The Treatment of Facial Spasm." (See *Medical News*, Sept. 24, 1886.)

Remarks on Dr. Sinkler's Paper.

The PRESIDENT: Dr. Keen sent this patient to me within two or three weeks, and I found that the woman was doing very well.

First, a word with regard to the method Dr. Keen adopted to determine absolutely when the nerve was obtained; it certainly is a very practical point. I resorted to this method six or seven years ago in the Hospital of the University of Pennsylvania. We had then a case of neuralgia affecting the hand, and we determined to stretch the musculo-spiral nerve. The operation was performed by Dr. Ashhurst, and I suggested that a weak current be applied as we proceeded with the operation, to determine what nerve was being stretched. In another case which I recall, something which was not the nerve at all was resected, and a second operation was performed and the nerve stretched. This mistake might have been avoided if the battery had been used.

Dr. C. L. DANA, of New York: With regard to the statistics

offered in the paper, it seems to me that too great value is attached to them, for statistics are extremely fallacious. We have with this form of neuralgia, as with others, different causes and different peculiarities in different cases, and perhaps some of the cases would have gotten well under a slight operation of any kind, and other cases would not have improved under any treatment whatever.

As the statistics were put, it would seem that five out of twenty-one were likely to get well, but when analyzed it would seem that one or two out of the twenty-one got well. The case reported has been operated on only *four* months, and it is possible, or even probable, that the woman will return within a year.

Dr. E. D. Fisher, of New York, then read a paper entitled "Remarks on Epilepsy" (see JOURNAL, vol. xiii., p. 481, 1886).

Dr. B. SACHS, of New York, read a paper on "Intracerebral Hemorrhage in the Young." (To be published in this journal.)

After referring to the increased attention neurologists were paying to the cerebral accidents of children, the writer recorded his conviction that intracerebral hemorrhage is more frequent in children than is generally supposed, and that many cases of this sort are classified under the head of meningeal hemorrhage, Dr. Sachs then reported two cases of intracerebral hemorrhage : one in a boy two and a half years of age, and the other in a young man of nineteen years. The first case was given in full, in order to place the diagnosis upon a firm basis. The child had typical right hemiplegia with aphasia, without coma convulsions at the time of onset. The onset was slow, aphasia setting in first, paralysis of the arm and leg some hours later. The recovery was typical of that which takes place in many cases of adult hemiplegia from apoplexy.

The writer gave the reasons why he held that in this case the apoplectic attack was due to hemorrhage rather than to embolism or thrombosis. As regards the differential diagnosis between meningeal and intracerebral hemorrhage, the lack of convulsions seems to be of unusual significance. In meningeal hemorrhage convulsions are invariably present, and their absence might argue, other things being equal, in favor of intracerebral hemorrhage. In the second case the young man had had two apoplectic attacks exactly one year apart. The histories of these attacks were very similar to the one given in the first case. In the first mild attack all symptoms developed and receded typically ; in the

second attack the onset was slow, there was coma lasting for over eighteen hours, and recovery is not yet complete, contraction having set in in the affected arm. No convulsions at any time. There was no specific history.

Using these two cases as a basis, the author of the paper entered upon a discussion of the changes in the walls of the cerebral arteries, permitting an effusion of blood into the brain substance. Autopsies on this condition are very scarce, but there is good reason for supposing (reference was made to some recently reported cases of Dr. Osler) that miliary aneurisms occur in young children, and that fatty degeneration of the cerebral arteries, permitting transudation of blood through the vessel-walls (Recklinghausen), is a not infrequent condition.

Discussion was invited on the following points :

1. Frequency of intracerebral as compared with meningeal hemorrhage in young persons not the subject of specific disease.

2. The value of convulsions as a factor in differential diagnosis.

3. Pathological changes in the walls of the cerebral arteries in the young.

Remarks on Dr. Sachs' Paper.

The PRESIDENT : As to the occurrence of intracerebral hemorrhage in the young, I think it is not of such infrequent occurrence, although there are but few reports of autopsies. I have made autopsies in three cases. The first was in a very young child. Three children of the same parents had, in succession, died almost immediately after birth. I made an autopsy in the third case, and found extensive hemorrhage. In another case I found meningeal hemorrhage, much diffused. This case presented a peculiar condition, which was of interest physiologically, and a point worth looking for in very young children, and possibly may have some diagnostic significance. In ordinary hemiplegia we have paralysis of the arm and leg and slightly of one side of the face ; in other words, the muscles of the trunk and abdomen are not very much affected. In young children the hemorrhage is likely to result in paralysis which involves the muscles of the chest and abdomen, and especially the face.

Dr. Lloyd will perhaps give us a word about a case which he related to me yesterday, although it was in an adult.

Dr. J. H. LLOYD, of Philadelphia : At the University Hospital Dispensary, some years ago, I saw a young man who had distinct right hemiplegia with aphasia, following diphtheria. I was not

the physician in attendance, and did not see him until the hemiplegic condition had intervened. I had reason to doubt its being an ordinary diphtheritic paralysis. Probability was that this case was one of embolism causing hemiplegia with aphasia.

The other case to which the President has referred, was that of a young man eighteen years of age, to whom I was called and heard the statement that he had been sick all day, had sat down upon the sofa, and had rapidly passed into the condition of coma with convulsions, more especially on the right side. After the boy's death I got a history of former alcoholism. He lay in this comatose condition about twelve hours, and, so far as I could judge, with this comatose condition he had right hemiplegia. In addition, he had sweeping over the paralyzed side, waves of clonic spasm. Besides, he had extreme right lateral deviation of the eyes, and also high temperature. He also had the appearance of right facial paralysis ; the face was distinctly drawn to the left side.

I was a littled puzzled in making a diagnosis. I was unable to find out any thing with reference to history of specific disease. I concluded that he had meningeal hemorrhage. Dr. Formad made the post-mortem examination, and nothing could be found to account for the bilateral convulsions. There was an œdematous condition of the membranes, and a very decided congestion of the entire brain ; an ecchymotic condition of the skull, especially over the motor regions, as though bruised, and presenting a bluish color, as though saturated with venous blood. He also had decided venous congestion of the kidneys. There was no hemorrhage anywhere in the brain. I finally regarded the case as one of acute exacerbation of alcoholism engrafted upon a chronic alcholic condition probably favored or complicated by uræmia.

Dr. ZENNER, of Cincinnati : I would like to say, with reference to the first case, that it seems to me to be of far greater importance to recognize the frequency or the infrequency of such cases than the nerve symptomatology presented.

In cases of central hemorrhage, embolism, and thrombosis, usually no positive diagnosis can be made from the mere symptomatology. The mere fact of paralysis coming on slowly would not be sufficient to make a diagnosis, because embolism might occur so that only a branch of an artery would be plugged up at a time. The mere fact of the absence of any definite cardiac symptoms

would not be sufficient to exclude such a diagnosis, because slight changes in the heart are not revealed by physical signs. Furthermore, the heart is not the only source of emboli. Therefore, the mere symptomatology alone will scarcely enable us to determine whether we have a case of embolism, or thrombosis, or hemorrhage before us. When we take this fact into consideration, and the fact that hemorrhage in the young is so very rare—a fact not based upon speculation, but upon examination of a large number of cases, perhaps it may be found more frequently than has yet been determined,—taking these facts into consideration, it would be scarcely wise to accept cases like these as subverting previous views concerning the great rarity of this disease, and it would look like embolism rather than hemorrhage, because of the rarity of hemorrhage in the young. At present the probability is in favor of the former.

Dr. SARAH J. MCNUTT, of New York: I have not seen a case of intracerebral hemorrhage in young children. I will mention a case of paralysis on one side preceded by convulsive movements of the same side of the face, and very marked in the eye. The child died, and I expected to find some lesion in the brain, but nothing except marked œdema in all parts was found.

Dr. R. W. AMIDON, of New York: This paper has interested me very much. It seems to me that the lesion must be looked for in the circulatory apparatus, and more likely in the arteries. The most natural lesion would be, perhaps, the result of periarteritis, and then miliary aneurisms referred to, but according to my experience and reading, I should be inclined to think that periarteritis resulting in miliary aneurisms is a disease of adult life, and usually of advanced adult life. Of course I except the periarteritis which occurs in tuberculous children. Endarteritis from syphilis in children is rare, and also from any other cause. Therefore, I should throw out the supposition that miliary aneurism could cause these hemorrhages. But there is one cause of cerebral hemorrhage in young persons, to which none of the speakers have called attention, and that is the condition which has been well described by Barrie in the *Revue de Médecine*, in several articles, which are summed up in the July number—namely, congenital narrowing of the aorta. I have seen pathological specimens taken from the bodies of two boys, sixteen and nineteen years old, who had congenitally small aortas (N. Y. Path. Soc.), and both of whom died of intracerebral hemorrhage, and another case has

been reported to the same society, but I did not see the specimen. Barrie reports twenty-seven cases of narrowing of the calibre of the aorta, and he says that it is almost always below the origin of the left subclavian artery, which would give a stronger reflux into the left carotid than into the right. He says that this construction or narrowing is of three forms: (1) funnel-shaped; (2) cul-de-sac formation; and (3) interposition at certain points of an almost complete diaphragm with a small circular or lateral opening. In his cases, some of which never gave rise to symptoms, the patients having died of something else, of the pathological appearances found at autopsy, cerebral hemorrhage, while not the most common, was observed occasionally, and that the two conditions stood to each other in the relation of cause and effect is very plain indeed. Collateral circulation may compensate somewhat, but it can never take the place of a normal circulation.

Dr. SACHS: With regard to the interesting paper by Dr. Barrie, referred to by Dr. Amidon, it is especially interesting in one respect. No doubt that this is a frequent primary cause, yet I question very much, and in this Dr. Amidon will probably concur with me, whether that condition of the aorta would have caused cerebral hemorrhage if the arteries in the brain had not been previously diseased.

Dr. AMIDON: In most of the cases the patients were subject to hemorrhages, but these were not by any means restricted to the brain. They had epistaxsis, vicarious menstruation, etc. He does not call attention to any change in the blood-vessels.

Dr. SACHS: With regard to the case referred to by the President and Dr. Lloyd, I have simply to add that I do not doubt that meningeal hemorrhage is exceedingly frequent. I do believe, however, that a fair number of such cases are in reality cases of intracerebral hemorrhage, and there are, furthermore, a large number of cases of alcoholism, in which hemorrhage occurs, but in such cases as mine alcoholism would not apply.

With regard to the remarks made by Dr. Zenner, he has turned things upside down. I had weighed all points carefully, and had taken great pains to show that there were certain points in differential diagnosis as between hemorrhage and embolism, and I thought that all the facts pointed towards hemorrhage rather than embolism. The chief object of my paper was to call special attention to one point, and that is the *absence* of convulsions in these cases. My conviction is that presence or absence of con-

vulsions is a strong point in making a differential diagnosis between meningeal and intracerebral hemorrhage.

The Association then adjourned to meet on Friday morning.

Friday, third day, morning session.

The Association was called to order at 10.30 o'clock by the President.

Dr. BURT G. WILDER, of Ithaca, exhibited the medisected alinjected head of a murderer.

The features of the brain, which was thus hardened and exposed *in situ*, are to be discussed at a future meeting in comparison with another murderer's brain; the specimen is shown in illustration of the value of the method of *continuous arterial alinjection*, which has been applied to other heads, to brains, and to entire bodies (children, a chimpanzee, etc.), in the anatomical laboratory of Cornell University. The main features of the method are (*a*) the reception of the head within twenty-four hours; (*b*) preliminary washing out of the vessels with chloral (to which, perhaps, weak alcohol might be preferable; (*c*) continuous alinjection for a week; (*d*) continuity secured by a pressure of 80 *mm.* of mercury, which was reduced to 40 *mm.* when the flow became somewhat free; (*e*) gradual increase of the alcohol from 65 to 94 p. c.; (*f*) maintenance of a low temperature (8–11, C.); (*g*) accurate division of the head with a fine saw acting in a mitre-box; (*h*) the small cost. The injected alcohol represented 41.5 litres of 95 p.c., but about two thirds was regained, so at $3 per gallon it would cost $11, and at 75 cents (free of tax) only $3.

Dr. JAMES HENDRIE LLOYD, of Philadelphia, then read a paper entitled "Moral Insanity: A Plea for a More Exact Cerebral Pathology." The writer referred to John Locke's criticism of those who confound, in psychological analysis, the *agent* with the *powers* of the agent. This philosopher inculcates the individuality of the mind, and cautions against granting to the faculties each its own autonomy. The faculties "are not so many distinct agents in us, which have their several provinces and authorities, and do command, obey, and perform several actions, as so many distinct beings." It is this principle of abstracting the mere *qualities* or *actions* of a thing and then personifying them as distinct *substances* (against which Locke cautions) that the writer

proceeded to show had been the bane of the metaphysico-theological methods of studying mental functions. These errors have more than the mere speculative interest which attaches to the thoughts of the philosophers, because, as was shown, they have a practical application in almost every hospital and court-house of our land. They have crept into our text-books, confused our science, decided our practice, and unfortunately sometimes vitiated our testimony. The writer briefly alluded to Peats, and especially to the modern Scotch school of psychologists, for examples of the metaphysical imagination which has converted vast generalizations into potent personifications (or real existences), so that men have from them attempted even to construct a science of biology (as Plato's "ideas"), or have worshipped what has no substantive existence whatever. This tendency is shown by the Scotch school in their use of the words "consciousness" and "mind," which they abstract from the brain (of which they are, in reality, but the faculties and modes), and make distinct entities of them. The method of medical writers and alienists is too often inherited from the philosophers, and this is shown especially in their making of the faculties—judgment, emotion, and will—distinct beings, more or less independent of each other, and each having its own diseases. The writer taught that this was a vital error. These faculties are but different *modes* of action of one substance, which is the brain ; and are not localized in it but universal in it and all its sensori-motor centres. Reference to Carpenter and J. Stuart Mill brought out the distinction between the metaphysical and material views of relations of mind and brain. A brief review was made of this subject as it had been handled in the courts of law—where distinctions, affecting life and property, had been equally artificial and untrustworthy. The writings of Coke, Hale, Blackstone, Shelford, and Chitty exhibit the divisions into "total and partial" and "civil and criminal" insanity ; and the tests relied upon (knowledge of right and wrong and presence of delusion) were shown to be rooted in this wrong method of looking upon the mind as something capable of numerous divisions and subdivisions, some of which remained healthy while others were given over to ruin. The writer desired to make complete the illustrations of man's futile attempts to artificially construct our science. We must listen to kindred attempts often on the part of our own profession. An analysis was then made of the subject "moral insanity" as found in the

writings of Kuch, Prichard, Ray, Winslow, and Maudsley. Moral insanity proceeds upon an abstraction, just such as Locke warns his readers to avoid. It teaches that there is a moral "faculty" in the sense of a distinct agent, which has its own powers and its own diseases, and which may remain undeveloped in a "mind" otherwise healthy, or may become diseased without at all affecting the health of the other "faculties." It is nearer the truth to say that the whole brain-act of an insane man is wrong—judgment, emotion, memory, and will. To say that a man's intellect, for instance, is sound and his will diseased, is a sophism, which has more sound than reason, and is no better than to say that a Laclanché cell has electro-motive force but no current-strength. It is impossible to conceive of an emotional state of the cerebrum which does not include as essential the state also known as the intellect. Both intellect and emotion are but states or modes of the brain. It is not to the credit of psychiatry—which is the science of a diseased cerebrum—that this faulty method, inherited from the metaphysicians, should confuse its results. Moral insanity, and its big brood of special manias, is but the creature of bad science,—but the unfortunate insane, who are stigmatized by the term and robbed of sympathy and justice have, too truly, an existence. The subject was largely illustrated, and especially in conclusion, from the writings of the modern experimental physiologists, who are represented by Ferrier.

The PRESIDENT: I have great respect, both for the metaphysical and the practical knowledge of Dr. Lloyd, but at the same time I feel constrained to differ with him, with regard to some of the statements which, if I understood him correctly, he appears to make in his paper. Perhaps the difference between us, as is often the case, may be rather with reference to the use of terms and methods of expression, than a difference in point of fact.

The first general remark which I would make is that I do not see exactly what we are to gain, as practical physicians, from carrying out of the ideas expressed by Dr. Lloyd with reference to nomenclature and classification. What, after all, are some of the purposes of a classification, whether of diseases of the mind, or of any thing else? One of the purposes is to assist in the work of life, to help to clear the way for us, to aid us to understand more details with greater ease. The burden of the practical portion of the paper would seem to be that we should not separate moral insanity from intellectual insanity; that we

should not use terms of this kind ; that they are not only misleading, but actually injure the cause of science.

Then in his details he says that we cannot have a moral insanity without intellectual indisposition. Now any one who has made a practical study of insanity recognizes the truth of such statements, and therefore there is no difference in general terms ; and when we use such terms as monomania, moral insanity, kleptomania, etc., we do so understanding what we are doing, deprecating it perhaps, but doing it largely as a matter of convenience—the same as the scientist uses botanical or any other terms. Further, there is reason for the use and the existence of such terms as these, because the reports of cases are sufficiently numerous, and they have been seen by many of us in practice, to lead us to say that we have the right to believe that there are cases which deserve the designation of moral insanity, for the reason that we have in them an exemplification of a defect which predominates, sometimes exists almost exclusively, in the case.

The question of impairment of intellect is only a confusion of terms. Those who have used the term moral insanity are perfectly well aware that in every case, unless there is general paralysis, physical and mental, you must have exhibited more or less of what might be intellectual, as well as moral, impairment. Under this term only a certain number are included, such as cases which their whole lives have exhibited a preponderance of moral defect, and give decided evidence of what we know and recognize as moral impairment. In many of these cases you will find simply a little intellectual impairment, but we do find cases, however, where the moral impairment is so great, so extreme, and where the intellectual condition is so good, that, practically, using the terms in the way in which they only can be used, the insanity is only moral.

I believe that the term moral insanity, and there I agree with Dr. Lloyd in part, must be used with restriction, and perhaps some other term would be better. In the case of hysterical insanity you have moral depravity, etc., yet I would not rank it as either moral insanity or imbecility. The point which I wish to make is that we have just as good right, scientifically, to regard a case as one of moral insanity, as we have to use any one of the multitude of names which are used by all of us in describing spinal, or cerebral, or hepatic, or other diseases. I think that the Doctor puts himself into the same position as that occupied

by some of the metaphysicians whom he calls to an account. Now he speaks of disease of that unit the brain, and where does that lead us? It takes us way back to the days before Hippocrates, without any classification of insanity at all. Perhaps we have differentiated too much, but the Doctor's idea would simply turn back what has already been accomplished.

Dr. LLOYD: I distinctly stated in my paper that classification is necessary, and that differences must be recognized.

The PRESIDENT: I must contend that it is a fair inference, coming from the general argument in the Doctor's paper, when he says, with regard to insanity, that it should be spoken of as a disease of that unit the brain,—it is a fair inference that he will set us backward in this direction of scientific study.

While I cannot subscribe to all the views expressed by Dr. Lloyd, and while I believe that some of them will retard rather than assist experts and jurymen, I must pay a high tribute to the literary and scientific merit of his paper.

Dr. L. C. GRAY, of Brooklyn: I was glad to learn from Prof. Jastrow that metaphysicians are beginning to learn what they seem to have been oblivious to before. There is a book published by a physician of Edinburgh, in which he goes quite extensively into the subject of the modern experimentation with regard to localization, and leaves one to infer that it will be really a reunion of the practical physician and the abstract metaphysician, but when he draws his conclusion he is absolutely dominated by the old views of the metaphysical school, and the effect is to confirm all these old theories. I think while it is true of more metaphysicians than this one, still it is a dangerous thing to attempt to learn much from them. My ideal of a lecturer on moral philosophy would be first to make him a practising physician in mental and nervous diseases, presupposing that he should be thoroughly acquainted with the modern doctrines of localization, etc., and then, when his mind had become imbued with what are facts to a large extent, I would let him loose into the field of metaphysics. I think the trouble with most metaphysicians is that they study the moral aspect a great deal, while the physiological aspect receives only a very limited amount of attention. It is like studying the question of localization by *physiological* experiment alone; and we know how pathological data have modified the statements thus obtained.

I agree with Dr. Lloyd in one way and with Dr. Mills in another,

and I think that they do not disagree as much as they seem to. I think Dr. Lloyd means to state very clearly that we should not have the name moral insanity put upon these cases, and that if by the name moral insanity was understood simply moral depravity with intellectual disturbances, Dr. Lloyd and Dr. Mills would be in harmony.

As to the practical instances of moral insanity in which the moral symptoms are strongly predominant, and as to the existence of cases in which the moral symptoms are almost entirely intellectual symptoms, I have no doubt whatever, because I have seen such cases again and again. Suppose you take the case of a boy whom I saw some time ago, bright, quiet, intelligent, but taking pleasure while at the table in thrusting a fork into the chest of another boy. What is that but moral insanity? Take also a case reported by Hack Tuke, and one from an English asylum; I do not see how we are to classify such cases under any other head.

Of course a great deal of fuss has been made about the plea of moral insanity in the courts, but I do not think that any scientific body should be influenced by the courts, especially when such experts are brought into court as are frequently seen. Of course it is a very difficult thing sometimes to draw the line sharply between moral insanity and a case in which there may be a large amount of insanity, but in which the immoral symptoms are largely predominant. But practically, I think, according to my own experience, we should be at very great loss if we were to throw away so significant a term as this.

Dr. RALPH L. PARSONS, of Sing Sing: I suspect the particular trouble in the use of this term has been that it has in some way, in the popular and in the legal mind, become mixed up with the idea of responsibility and irresponsibility; and to the popular mind the term is objectionable because it means two different things; it may mean emotional insanity or it may mean simple wickedness.

With regard to moral insanity I am just as certain of its existence as I am of any of the other forms of insanity. We speak of different physical diseases, as disease of the liver, disease of the teeth, etc., but we do not mean that the patient is not a sick person. It is not simply the liver or the teeth, but the entire system which is affected. In a still stricter sense, I think we may say that there is a form of insanity which cannot be placed in any of our divisions of insanity, yet which exists really, but affecting the

other faculties of the mind more or less, whether the intellect, the emotions, or the will. Certainly we say that if a man wills, his mind is willing ; if he remembers something, it is his mind remembering : and that is very true. But with regard to mind remembering, he remembers certain classes of things, names, words, facts, mathematical truths, historical facts, better than some others.

My own convictions have been so well expressed by Dr. Mills and Dr. Gray that there is no necessity for extending my remarks, except simply to reiterate that in almost all forms of insanity with which I am acquainted, while there is sufficient distinction to call for classification, yet all the faculties of the mind are more or less involved.

The PRESIDENT : I would like to ask Dr. Lloyd two or three questions. I would like to ask him whether, in discussing the subject of insanity, we have any right at all to use such terms as moral or immoral ? Whether such terms should be employed in any sense ?

If so, in what sense should they be employed in discussing questions of this kind ?

Do not those who see cases of insanity frequently see those who present an overwhelming predominance of moral symptoms, of so-called moral or immoral phenomena ?

Now, then, suppose you have, in a genuine case of insanity, represented by ten, nine parts of moral defect and one of intellectual defect, where would he place the case ?

Dr. GRAY : I would also like to ask Dr. Lloyd a question. Why can't we have localization in regard to the action of the mind as much as we have central localization in purely somatic matters ? Why may we not have localized insanity by certain localized areas becoming involved ? What is there to prevent us from saying that it is possible ?

Dr. LLOYD : I feel much gratified at the politeness with which I have been treated. I can only say with reference to Professor Jastrow's remarks that I cannot get any satisfaction from the expression "line of least resistance." I prefer to say that I do not understand it. I do not believe that anybody does. How do we get an idea ? What is the exact condition of the cerebral matter when an idea is obtained ? I do not know. Nor do I believe any one else does.

With regard to moral insanity, all I can say is that it is more

than a mere difference in terms, and when the gentleman uses the term "moral" as distinct from "intellectual," and says that a man took a fork and stabbed another man, and took pleasure in it, and that he would call it moral insanity without disturbance of intellect, he speaks of something which has not been proved. Nobody has witnessed his mind with reference to the act. It is impossible to conceive of a boy stabbing another boy without cerebral intellection.

I think that it will be a sorry day when we get to despising our courts and our courts to despising us. I have studied law myself, and I do not propose to trifle with judges or juries. They have the right to insist that we should express ourselves, not paradoxically, but in intelligent, consistent language.

Dr. Mills asks what would you do with such cases, and where place the cases in which the bulk of the derangement is moral, or in which the bulk of that moral derangement is intellectual. I do not understand the moral to be apart from the intellectual. If the man's brain is affected, all the qualities of the faculties which come from it are also affected. Therefore I would say it is mere word differentiation, and not a differentiation of things. This is the only answer which I can give the question.

With regard to kleptomania or paranoia, I would be the last to say that such a person was *only* morally insane. The very idea involved is that the man has a congenital defect in his mental organization, and it has come from his mother's womb. Such cases derive their trouble from bad heredity.

With regard to classification I did not say that there is nothing in it; but I do say that you must get at truths before you can classify them.

Dr. Geo. W. Jacoby, of New York, presented a new portable galvanic battery, described in a paper by Dr. J. Rudisch, of New York (see JOURNAL vol. xiii., p. 575).

Read by title: "Facts and Deductions Bearing on the Action of the Nervous System."[1] By F. X. DERCUM, M.D., of Philadelphia.

The following amendments to the By-Laws and the Constitution were affirmed:

By Dr. R. W. AMIDON, of New York: "The officers shall enter upon their duties immediately after the adjournment of the annual meeting at which they are elected."

[1] Will appear in JOURNAL, Nov., 1886.

By Dr. C. K. MILLS, of Philadelphia : "Two Vice-Presidents instead of one Vice-President."

By Dr. G. W. JACOBY, of New York : "They (officers) shall be nominated by the Association on the *second* day of the annual meeting" (instead of the first day).

"There shall be two sorts of members, namely, active members —not exceeding at any time one hundred in number" (instead of fifty as at present).

Resolutions.

Dr. L. C. GRAY, of Brooklyn, introduced a resolution endorsing the proposition of the proposed "Congress of American Physicians and Surgeons," and moved that a committee of conference of five be appointed by the President. The resolution and the motion were adopted.

The PRESIDENT appointed Drs. L. C. Gray, of Brooklyn ; J. Van Bibber, of Baltimore ; W. Sinkler, of Philadelphia ; E. C. Seguin, of New York ; and Philip Zenner, of Cincinnati.

Officers for the Ensuing Year.

President.—L. C. Gray, of Brooklyn.

Vice-President.—John Van Bibber, M.D., of Baltimore.

Secretary and Treasurer.—G. M. Hammond, M.D., of New York.

Councillors.—B. Sachs, M.D., of New York, and Wharton Sinkler, M.D., of Philadelphia.

The Association adjourned to meet in June, 1887, the date and place to be designated by the Council.

In Memoriam.

Dr. T. A. McBRIDE.

In the sudden death of Dr. Thomas Alexander McBride, not alone has American neurology suffered a serious reduction of its ranks, but each member of our little community of specialists has lost a cherished and warm-hearted friend. There are few to whom it is given to pass through life, as the deceased had done, without provoking a single unfriendly criticism, and still fewer whose loss is felt as keenly by so many.

Dr. McBride was born in 1844, at Sandusky, Ohio. He received a liberal education, taking the degree of Master of Arts, and after completing the usual term of study at the College of Physicians and Surgeons, he graduated in medicine in 1871. He then entered Bellevue Hospital, and commenced the practice of his profession after his hospital term was completed. He was at this time an assistant at the medical clinics of the college from which he had graduated, and early distinguished himself as a thorough and searching investigator in the field of physical diagnosis. He later became the first assistant at the clinic of Dr. Seguin, and after resigning this position he was appointed to the lectureship on clinical medicine at the same school. Through his extensive professional and social connections, Dr. McBride early attained the position of a consulting physician, and during the last years of his life he was one of the most sought for consulting specialists. Among other prominent persons regarding whom his opinion was taken, were the late Samuel J. Tilden, and Secretary of the Treasury Manning.

Dr. McBride united in the happiest way the qualifications of the general clinician and the neurologist. He specially devoted himself to the elucidation of the problems connected with the nervous manifestations of diseases of assimilation, and more particularly to the application of instruments of precision to their study. He was not a prolific writer. He matured his observations slowly, but what he has published will, we believe, prove

valuable when the ballast of neurological literature in the midst of which it appeared buried shall have been disposed of. Among his papers, "The Slow Pulse and Disturbances in the Rhythm of the Pulse," "The Temperature in Uræmia," "The Utility of the Sphygmograph in Medicine," "Auscultatory Percussion," "Coma in Chronic Bright's Disease," and the "Early Diagnosis of Chronic Bright's Disease," are the best known. In addition, he reported a number of interesting cases; among others, a "Case of Hysterical Anuria Cured by Restoring a Lacerated Cervix Uteri," and a "Tumor of the Optic Thalamus Perforating the Corpus Callosum." Several of his papers were contributed to the JOURNAL OF NERVOUS AND MENTAL DISEASE. In 1880 he founded the *American Journal of Neurology and Psychiatry*, which he conducted for three years, and in which he published many of his observations, chiefly in the editorial columns.

Dr. McBride had been aware, for many years, that his kidneys were in a pathological condition. He was afflicted with a gouty predisposition, and the form of his disorder was suspected by himself to be the cirrhotic. In 1878 he contracted a bilious remittent fever, which left his constitution greatly impaired for a time. But he recuperated to all outward appearances, and to all but his most intimate friends the serious state of his health or the occasion of his last trip to Europe was not even suspected. He left Carlsbad, where he had been in the habit for the last three years of passing the summers, in a very low condition of health, and on arriving at Southampton he was seized with uræmic convulsions. Dr. Roosa, a passenger on the same vessel which Dr. McBride had engaged his passage on, was called in to treat him. His friends engaged a nurse and took him on board the North German Lloyd steamer "Aller" on August 28th. Two days later he sank into coma and died August 31st. The distance from New York, the weather, the crowded state of the vessel, and the absence of appropriate caskets and embalming material, rendered it impossible to bring his body home, and it was accordingly committed to the deep. Both his aged parents and a brother survive him.

His kindly and generous disposition to all, his loyalty to his friends, his liberal scientific spirit and unselfish devotion to every thing that seemed true and noble, have enshrined him in the memory of most of us so deeply that we may say, even in this fast-living age, he cannot be replaced, and will not be forgotten.

***

Editorial Notes and Miscellany.

OWING to inadvertence on the part of the proof-reader a number of errors have crept into Dr. MacDonald's report of cases in our last number.

On page 489 read *site* instead of side.

On page 490 read *rongeur* instead of rougeur.

On page 493 read August 26, 1885, instead of 1886.

On page 493 read Aug. 31, 1885, instead of 1886.

Sharkey's lectures on Spasm in Chronic Nerve Diseases have been issued in book form by J. & A. Churchill. These lectures received a very thorough review in the Periscope of No. 6 of this JOURNAL. We have nothing to add to-day with regard to the subject-matter of these lectures. We refer to this book merely to commend the spirit of the publishers who have ventured to reprint these lectures of Sharkey, as well as those of Buzzard on Multiple Neuritis, though the sale of such works is necessarily somewhat limited.

It is a fact worth noting that Dr. Hammond's well-known treatise on "Diseases of the Nervous System" has reached its eighth edition. A chapter has been added on "Certain Obscure Diseases of the Nervous System," including Tetany, Thomsen's Disease, Myriachit, and kindred affections. If the book reaches its ninth edition, as it undoubtedly will, Dr. Hammond will probably speak with more positive knowledge regarding some of these disorders. He will by that time have been induced to strike one of them from his present list, viz., Thomsen's disease.

At the Congress of German Scientists and Physicians, now in progress at Berlin, the following papers were announced to be read

before the Neurological Section : Prof. Binswanger, " On Aphasia " ; Dr. Remak, " Faradic Reaction of Degeneration " ; Drs. Oppenheim and Siemerling, on " Acute and Pseudo-Bulbar Paralysis " ; Dr. Meschede, on " A New and Well-Characterized Form of Mental Disease " ; Prof. Fürstner, of Heidelberg, " Some Experimental Investigations on the Central Nervous System " ; Prof. Benedict, on " The Value of Kephaloscopy, etc., in Neuroses "; Dr. Müller, of Gratz, on "Arsenic and Sabine Paralysis "; and a paper on " The Juvenile Form of Tabes Dorsalis " ; Dr. Smidt, " Cocainismus, and the Action of Cocaine in Cure of the Morphine Habit " ; Prof. Mendel, " Contribution to the Pathology and Treatment of Epilepsy " ; Prof. Adamkiewicz, on " Multiple Sclerosis," and on the " Diffusion-Electrode " ; Dr. M. Meyer, " Neurotic Exudations, the Cause of Neuroses."

VOL. XIII. NOVEMBER, 1886. No. 11.

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

A CONTRIBUTION TO THE STUDY OF TUMORS OF THE SPINAL CORD.*

By B. SACHS, M.D.

THE study of tumors of the spinal cord and of its meninges has been considerably neglected of late years. This neglect must be attributed in part to the general belief (which I shall not be able to discredit) that it is difficult to establish a differential diagnosis between tumor and other chronic affections of the spinal cord, and that the close study of these tumors is, therefore, of little *practical* value. No less an authority than Erb[1] expresses this opinion. The difficulties become still greater when we attempt to differentiate clinically between tumors of the spinal meninges and tumors of the spinal-cord substance. The latest authors on this subject, (Drs. C. K. Mills and J. Hendrie Lloyd[2]) have abandoned the attempt altogether. The great interest now being felt in syringomyelia will probably direct attention once more to the symptomatology of spinal tumors, for there is no doubt that there is an inti-

* A preliminary report on the case here reported was made before the N. Y. Neurological Society at the stated meeting, April 6, 1886. Numerous other duties have prevented the earlier publication of this paper.

[1] Ziemssen's Handbuch. Erb: "Diseases of Spinal Cord," vol. xi., 2, p. 340, Germ. ed.

[2] Pepper's System of Medicine, vol. v., p. 1091.

mate clinical (and probably also pathological) relation between these two forms of spinal disease.

Those who will not be satisfied to pursue clinical studies unless some practical good shall be the outcome of such studies, should recognize the importance of differentiating for purely therapeutic reasons, if for no other, between chronic transverse myelitis, for instance, and tumor. Every case of tumor of the spinal cord, if verified by post-mortem examination, may help to clear up the mystery surrounding the symptomatology of these diseases. The case which I propose to discuss has special claims upon our attention for the following reasons. 1. It was a tumor of the spinal-cord substance without involvement of the spinal meninges. 2. The tumor gave rise to the first and most prominent symptoms of a general tubercular diathesis. 3. The myelitis following in its wake was of unusual severity. 4. The case presented unusual sensory symptoms.—First, as to the history of the case:

F. F., aged thirty-two, a man of considerable intelligence, unmarried, a dealer in bric-a-brac, presented himself at the Polyclinic for the first time on February 7, 1886. He complained of pains and weakness in his left arm, of inability to move the fingers, and of the glossy appearance of this left hand. The man was made the subject of a clinical lecture, in the course of which I elicited the following details: He was born in Brussels, where he lived until he came to this country six and a half years ago. His mother had epileptic fits consequent upon a severe mental shock; cause of her death unknown. The father, a physician, was a heavy drinker, and died at the age of forty-two; some heart trouble the immediate cause of *his* death. The patient has several sisters and brothers, all of whom are enjoying good health. Patient himself had typhoid fever when six years old; no venereal diseases, except gonorrhœa; syphilis absolutely denied; acknowledges excessive sexual and alcoholic indulgence for many years; he was in the habit of drinking daily from twenty to thirty glasses of beer, and had a special fondness for absinth. Two years ago he had a severe attack of articular rheumatism, which affected all

the joints of his extremities, and kept him in bed for some weeks: states that he has had rheumatic pains ever since.

History of Present Trouble.—Four weeks ago (about January 8th) he felt what he termed rheumatic pains in the left shoulder; these moved down to the left arm; the hand grew weaker after the lapse of a week or more, and as the weakness increased, the fingers became puffy and the skin glossy.

Present Condition.—Feb. 7th.—The patient has a gluttonous and rather bloated look, but otherwise of excellent physique. On comparing the two arms, we observed that both arms could be raised above the head, but in dropping them the left arm dropped more readily than the right; the upper arm and forearm offered considerable resistance to passive movements, and were in marked contrast to the muscular power of the hand and fingers, which was almost nil; the fingers were slightly flexed; voluntary extension or further flexion was impossible. The sensory disturbances were even more marked; there was hyperæsthesia of the entire fore-arm and hand, which was greatest, however, over the distribution of the ulnar, and particularly over the outer dorsal surface. The mere touch with the finger was painful; the prick with the pin was so disagreeable that the æsthesiometer test had to be abandoned; cold water dropped on the arm produced excessive pain (this the patient had noticed in washing also). Pressure with the finger over ulnar and radial nerve was objected to, whether or not in consequence of the cutaneous hyperæsthesia, it is difficult to say. The vaso-motor or trophic symptoms noticeable during this first examination were a swelling of the skin *between* the joints of all the fingers of the left hand and a peculiar œdematous appearance of the skin of the entire hand including fingers. The electrical conditions were quite normal. Both nerves and muscles responded to faradic current of moderate strength; in consequence of hyperæsthesia this examination had to be made hastily. The galvanic reactions were entirely normal; no unusually weak or strong currents were required to produce contractions, and the order of contractions was not

reversed in the case of any nerve or muscle of the left arm. There was no ataxia in lower extremities; no incoördination of movements in right arm; superficial reflexes could not be elicited, except, possibly a very slight scrotal reflex on right side. The other parts of the body were subjected to a careful examination; there were no other symptoms discoverable, except these: 1. A slight dragging of the left foot, but no very marked diminution of gross muscular power, as tested when the patient was lying on his back. 2. An increase of both knee-jerks, and presence of ankle clonus on *both* sides. In view of this peculiar complexity of symptoms, a definite diagnosis was not attempted at this time.

In the course of the next two weeks I saw the patient at irregular intervals. During this time the symptoms in the left upper extremity remained unchanged. From February 15th on (about a week after first examination) the impairment of power in left leg became more noticeable; this increased until February 26th, when the patient could move the left leg with the very greatest difficulty only; resistance to passive movements almost nil; the loss of power was distributed equally over thigh and leg. The right leg remained normal. In addition to the other symptoms general hyperæsthesia of left leg had been developed, but the various forms of sensibility in *right* leg was in no wise impaired (tested with æsthesiometer points and cotton). Subjectively there was no pain in left leg, but a feeling of cold throughout the entire left lower extremity. These unilateral symptoms continued uninterruptedly until March 2d (three weeks after first examination).

In order to make a complete examination, I arranged to examine the patient at my leisure in the office. Up to Feb. 26th he had succeeded in walking about with the aid of a stick. March 2d he could walk about only with the support of a friend.

On this day, March 2d, his condition was noted as follows: no head symptoms; pupils equal, and react promptly to light and during accommodation; all ocular movements perfect; tongue straight when protruded; no dis-

turbances in articulation or deglutition; no headache; sleeps well; no atrophy of any muscles of body; can raise both arms fairly well, but left arm behaves as on first examination; no changes as regards either upper extremity since first examination; can still offer considerable resistance to passive movements with left upper arm and forearm; the fingers are completely motionless, possibly in consequence of pain, and swelling of the skin; no incoordination of movement of right arm; pricking of pin felt as distinct pain over distribution of l. ulnar nerve; a cold sponge passed over this area causes him to cry out with pain; fingers as puffy as ever; no reflexes to be elicited in this upper extremity. Faradic response from nerves and muscles fair to moderately strong currents; on this day galvanism was not tried. Left leg extremely paretic; he can stand on it without using stick, but in trying to move it requires additional support; his left leg, he states, feels like a piece of wood on a string, by which he means that it is entirely beyond his control; he cannot tell whether his foot is right in the shoe or not; there is continued hyperæsthesia of whole leg; patient stated to-day for the first time that his right leg is growing weaker also, and on examination resistance to passive movements in the leg is found to be diminished as compared with the condition of the right leg at the preceding examination; he complains also of weakness of abdominal muscles; cannot press properly when he goes to stool; sensation in right leg not disturbed at this time. The patient was urged to have himself taken into hospital; he had his own peculiar reasons for not wishing to go there. I ordered him to go home, and to remain strictly in bed, and put him on increasing doses of the iodides of potassium and sodium. I endeavored to have him as well cared for as his poverty-stricken surroundings would permit. I saw him again two days later, and found him, contrary to my orders, sitting upright in a chair, smoking a pipe, reading a French novel, and hopeful that he would be all right in a few days. With the aid of chairs he moved toward the bed; his left leg could barely be dragged along; sitting on the edge of the bed he was this

day able to swing himself around, and to lift the left leg into bed with the assistance of the right. The left arm in *statu quo;* fingers slightly flexed, but flexion can be easily overcome, though the mere touch of the fingers, and particularly of the finger-tips, causes intense pain. Forearm and hand tested again with æsthesiometer; number of points could not be readily distinguished, as the pain-impressions were predominant on entire dorsal and volar surfaces; cold water as painful as ever; left leg cannot be lifted at all from bed. Muscular sense grossly at fault; cannot estimate the distances to which left leg has been raised from bed; acknowledges his surprise when he opens his eyes and sees where his leg is; gauges the position of right leg very accurately; marked hyperæsthesia to touch or cold over entire left lower extremity; knee-jerks and ankle clonus about as before. One symptom which I observed this day deserves special notice. A plantar flexion of the left foot produced distinct contractions of the tibialis anticus; once or twice this flexion movement was followed by a distinct ankle clonus, which was therefore evoked in a way diametrically opposed to the customary method. This contraction of the tibialis anticus I was inclined to regard at the time as an instance of paradoxical contraction, though this contraction generally ensues when the extreme ends of a muscle are approached to one another, and not removed from one another as they are in this case. Prof. Westphal,[1] in a recent article on a curious group of symptoms associated with sclerosis of the posterior columns, refers to a paradoxical contraction produced in this way. I did not succeed in producing this phenomenon in the course of subsequent examinations.

March 9th.—No marked change in the symptoms as detailed above, except that incontinence of urine has set in; patient feels that he is growing worse. Left arm unchanged; left leg absolutely paralyzed, and the right growing rapidly weaker; is not well able to use his right leg in lifting the left into bed, as he was two days ago. While there is still marked hyperæsthesia of the left leg, there is no percep-

[1] *Arch. f. Psych.*, vol. xvi., 1885.

tible sensory disturbance on right leg. Muscular sense disturbed as above. On the trunk of the body, the limit of hyperæsthesia extended, exclusively on left side, as high up as level of third rib. Examined with electrical current again: no change in galvanic reactions, and faradic responses from nerves and muscles of all extremities prompt to currents of moderate strength; even the ulnar responded in this way.

March 11th.—A slight cough, which the patient has had for a few days past, begins to annoy him as he has not sufficient muscular power to expectorate freely. Lungs and heart examined as carefully as the now helpless condition of the patient would permit: there were the usual catarrhal râles but no other physical signs of any import could be made out; the action of the heart was rather feeble, but sounds were clear. The paralysis has markedly increased; the patient is totally unable to move himself in and out of bed; when raised in bed he complains of weak back; the head can be moved well from side to side, but not so well from before backward. There is some tenderness on pressure over the seventh cervical vertebra, and when the head is moved backward the pain is stated to be at the level of the vertebra and along both trapezii. The left hand unchanged; but the grasp of the right hand is far weaker than it was, and the patient states that there is now also a numbness over the dorsal surface of right forearm. No puffiness of the fingers of the right hand; the right leg has become very much more paretic; even this leg can now be lifted but three or four inches from the bed; the muscular sense, as tested by raising leg from bed, is beginning to be impaired on right leg also; the hyperæsthesia of left leg and of the left side of trunk as high up as seventh rib is giving way to loss of tactile sensibility; there is an area of hyperæsthesia still between seventh and third rib on left side. Tactile sensibility of back well preserved. Tenderness to touch most marked on the outer dorsal aspect and the whole of the volar surface of the forearm. Touching fingertips with pin produces intense agony. (Eyes remain closed during test). Warm water applied to back does not excite

pain at any particular point. No bed-sores though there is slight reddening of skin over sacrum. Weight-sense was tested without any satisfactory results. Bowels have not moved in several days. Two days later, the only changes noticeable were the increasing paresis, amounting to complete paralysis of right leg, and a suspicious redness of the skin over the internal malleolus of the right foot, against which the left foot had pressed. Knee-jerks are still exaggerated, though not as markedly so as they were; ankle clonus is not as easily excited as in the past.

The day following, March 14th, I invited Dr. Starr to visit the patient with me. Our notes made independently agreed in all particulars and brought out the following facts regarding disturbances of sensibility. The hyperæsthesia had given way still further to a gradually increasing anæsthesia to touch; while the hyperæsthesia had been confined exclusively to the left extremities and to the left half of the trunk, the anæsthesia now involved both sides of the body, but not all parts equally; the only parts which at this time retained normal sensation were the neck, face, and head. Left arm and hand: persistent hyperæsthesia to mere touch, to hot and cold; cold produced the greater pain; warm was painful also but was recognized as "warm" (in these tests, a silver spoon dipped alternately into hot and cold water was used). Left leg: sensation to touch below the knee almost wholly abolished; above the knee very nearly abolished; light touch of the finger and stroking with cotton were not perceived; the finger drawn across thigh was felt and direction in which it moved was correctly stated. The following day touch of finger was perceived on dorsal surface of both feet; hot or cold could not be appreciated.

Temperature sense distinctly altered; heat was not felt, cold produced pain; muscular sense (sense of position of muscles) entirely abolished. Right leg: sensations of touch and of pain more acute than in left leg, but less acute than in right arm and in face. Heat could not be appreciated while cold was painful; muscular sense far better than in left leg, but nevertheless considerably impaired. Trunk:

left side, which was at one time hyperæsthetic, has had its sensation to touch and pain somewhat dulled; the cotton and pin tests were felt, and correctly located, but were not as distinctly felt as on the right side of the median line. Heat and cold could not be appreciated as such on left side, but were recognized on right side. Scratching left side of abdomen with a pin produced more redness than on right side. R. arm: much more sensitive to touch than left, yet the sensations were not as acute as on the neck and in the face. Muscular sense well preserved. In left arm it could not be tested, as the mere attempt to move any finger or the whole hand was exceedingly painful. Next day, no distinct tenderness could be perceived over any part of spinal column; cotton was perceived equally on both sides of back. The motor disturbance had increased to a complete paraplegia; in the l. upper extremity grasp of hand and all movements of hand almost nil, but patient retained considerable power in the pectoralis, deltoids, biceps, and brachialis; grasp of right hand much weaker than it has been; abdominal muscles almost powerless and action of inspiratory muscles of thorax very weak; complete relaxation of muscles of back; head could be moved freely in all directions; all ocular and facial movements perfect. The knee-jerks and ankle clonus not as marked as they were; cutaneous reflexes could not be elicited.

I saw the patient again and for the last time two days later. No marked change had taken place. I took occasion to make an ophthalmoscopic examination of the eyes which I neglected to do in the earlier history of the case, but could detect no important changes. I may say again that the man continued reading in bed until a few days before death, and in answer to my questions invariably stated that vision was good. A few statements, which were not included in this chronological record of the case, will help to complete the history. The temperature was taken at different times during my visits and was never found to have exceeded 99½° in the axilla; this would of course not exclude possible rise of temperature at other times; my visits were made generally late in the evening. Pulse small

and accelerated, often about 120. Respiration varied very much, generally about 25; on the last day I saw him they had gone up to 45. The urine was examined several times and found free from sugar and albumen. There was no distinct history of night-sweats; but according to his statement the patient had for a long time been subject to unilateral sweating of the right side. At the very first examination the beads of perspiration on the right side had attracted my notice.

The patient's objections to being taken to a hospital were finally overcome, and he was removed Wednesday, March 17th, to the German Hospital. He arrived there feeling weak, but not particularly exhausted. On Thursday morning about seven o'clock, he asked his neighbor in the ward to read a newspaper to him; while this was going on, the patient died, and quietly at that, for his neighbor continued reading until an attendant who stepped up to the bed found the man dead.

Summarizing the main points in the history of this case, we may say that the trouble, which was supposed to be rheumatic, began with pain in the left shoulder which radiated down into the arm; the pain became more intense, and for the first was confined chiefly to the area of the ulnar, spreading later on over entire dorsal and volar surface of left forearm and hand; in addition to this hyperæsthesia and to puffiness of the fingers, we found considerable weakness of grasp in the left hand, with only slight loss of power in the muscles of the arm, forearm, and shoulder. The condition of this left upper extremity remains unchanged during the whole course of the disease—a period of about two months. A slight paresis, some hyperæsthesia of the left leg, exaggerated knee-jerks and ankle clonus were the only other symptoms discovered during the first few weeks of the disease. These symptoms, chiefly unilateral, continued so until the close of the seventh week. Meanwhile the paresis of the left leg had developed into an almost complete paralysis, and the unilateral paresis is transformed in the eighth week to a complete paraplegia. The motor paralysis increasing affects also the abdominal muscles, and

to some extent the respiratory muscles and the right upper extremity. Incontinence of urine and bed-sores were superadded. As for the sensory disturbances, there was developed by degrees a general hyperæsthesia of the left half of the body below the level of the third rib ; this hyperæsthesia was changed later on into an anæsthesia which spread from the left half and finally involved the right leg and, to a lesser degree, the right half of the trunk and the right upper extremity.

Now as to the interpretation put upon these symptoms during life. That the symptoms of the last two or three weeks were those of a cervico-dorsal myelitis there could be no manner of doubt, but the troublesome question was to decide the origin of the myelitis and to explain the persistent hyperæsthesia and paresis of the left upper extremity, coupled as these were, strangely enough, with slight paresis of the left leg and with exaggerated knee-jerks and ankle clonus on both sides. With the exception of these reflex movements, all symptoms were strictly unilateral for nearly four weeks after first examination. At the outset I was inclined to the belief—and in view of the patient's rheumatic record it was not an irrational one—that his was one of those rare afflictions, so rare that their existence may still be questioned, of an ascending neuritis giving rise to a myelitis. I soon abandoned this diagnosis, for the condition of the electrical reactions and the slight amount of, and unequally distributed, paresis made one rather sceptical as to the existence of a *neuritis*, and I could not imagine a *myelitis* spreading across the cross-section of the spinal cord so slowly that it should for weeks give rise to unilateral symptoms only. It was my surmise that a lesion involving the posterior root-fibres of the lower cervical segments could possibly explain all symptoms, but to the nature of the lesion I had no direct clue. Giving patient the benefit of the doubt, from the wild life he had been leading I suspected the possible existence of a gumma in this vicinity, but the therapeutic results did not strengthen this suspicion. Tubercular disease, or any cachectic diathesis, I did not suspect; the physical signs were certainly not

marked enough,[1] and the general appearance of the man, a strapping fellow, did not point that way. The autopsy has cleared up the mystery, and after I have given a short account of the post-mortem appearances, it will be in order for us to inquire whether this case furnishes sufficient points for a differential diagnosis between tumor and other affections of the cord.

Post-mortem Examination.

The autopsy was made eight hours after death by Dr. Waldstein, pathologist to the Hospital; the microscopic examination of the cord was made by the writer.

The skull was thickened but symmetrical. The dura mater was easily detached from the brain, except over parietal vertex, where it was slightly adherent to the pia. The sinuses were well filled; the vessels of the pia were somewhat more congested than normally. All blood-vessels were found normal in structure. No hemorrhage into the brain-mantle or the subcortical ganglia, and no malignant growths anywhere in the brain. The convolutions were a trifle narrrow, but not otherwise changed.

No disease of the vertebræ. The spinal dura appeared entirely normal. The cord with its meninges was removed easily, except at its upper end. On opening the dural sac, a large amount of fluid escaped, and the vessels of the pia, along the entire length of the cord, were seen to be greatly congested. The lower portion of the cord exhibited no macroscopic changes except innumerable calcareous (not tubercular) deposits scattered over the pia of this portion of the medulla spinalis.

The chief trouble was evidently limited to the lower cervical and upper dorsal segments. *From the seventh cervical to the fourth dorsal segment* the cord appeared to be a diffluent mass, revealing no trace of structure. Throughout this softened portion of the cord, whitish nodules appeared to be scattered here and there. As soon as the cord was partially hardened it was evident that the myelitis

[1] The very frequent respiration in the latter stage of the disease might have been caused by direct interference with the respiratory nerves.

had involved the posterior columns, part of the gray matter, and a small portion of the lateral columns, leaving perhaps half of the area of the lateral columns intact, as well as the anterior (ventral) portion of this region of the cord. Between the sixth and seventh cervical segments (area determined by the character of the emerging root-fibres) we found a round tumor of the size of a hazel-nut pressing closely upon the emerging posterior fibres, without displacing these, and extending inward as far as the median line; at its caudal end the tumor had exceeded the median line by a small fraction of an inch. The dura and pia had been readily detached from the spinal cord at the level of the tumor. There were no visible changes in the dura or pia except that a few very small tubercular deposits were found in the pia over the middle dorsal region.

The *tumor* consists of an outer harder portion, encircling a soft, cheesy mass within. Inspection of the cross-section of this tumor in a comparatively fresh state, and later microscopical sections, proved this tumor to be a typical solitary tubercle of very respectable dimensions. Although the tumor takes up the entire thickness of the cord (dorso-ventrad), the amount of destruction is not as great in its immediate vicinity as it is at a level about one inch caudad of the lower end of the tumor. Unfortunately I am not able to state how far cephalad this myelitic process extended, for, on removing the cord, this part was somewhat (perhaps unavoidably) lacerated. It is surely speaking within bounds to say that the myelitic process stopped short at about the third cervical segment. On the fresh cord no positive evidence of ascending or descending degeneration could be discovered. A portion of the left ulnar, median, and radial nerves were removed for microscopic examination; but the result was an entirely negative one.

For the rest, the autopsy revealed very minute tubercular deposits scattered here and there throughout both lungs; in both apices there were incipient cavernous spaces; there were also old pleuritic and pericarditic adhesions. The valves and tissues of the heart proper were entirely normal. In the intestines there was evidence of a general miliary

tuberculosis. Spleen was enlarged ; liver and kidneys were normal. No evidence of alcoholism and none of syphilis.

The post-mortem diagnosis would read : General tubercular diathesis, with chief deposits in spinal cord, lungs, and intestines. In the spinal cord a solitary tubercle followed by a cervico-dorsal myelitis.

Microscopical Examination of Spinal Cord.

For this purpose the cord was suspended at once in Müller's fluid. It was watched most carefully, the fluid was changed frequently, and yet the hardening was not entirely satisfactory. The myelitic portion could not be rendered fit for cutting, and the exact amount of destruction could not be determined more accurately than in the comparatively fresh specimen. Fortunately, the symptoms to be referred to this cervico-dorsal myelitis were so clearly marked that a pathological examination could not have been expected to throw much light upon them. The rest of the cord, after it had been sufficiently hardened, was subdivided and the single segments imbedded in celloidin. The sections were then stained either according to Weigert's hæmatoxylin (old) method, or with acid fuchsin. Picrocarmine was used for sections through the level of the tumor. The sections most thoroughly examined were from the upper cervical, the mid-dorsal, and the upper lumbar levels.

Sections at the level of the tumor showed that the tumor not only occupied the entire left side of cord but that it exceeded the median line more than inspection of the fresh specimen had led us to suppose. With its lower (caudal) end it had insinuated its way into the posterior columns of the right half of the cord, leaving the columns of Burdach almost undisturbed. It had grown around the central canal destroying the greater portion of the commissural gray matter, but left the greater portion of the gray matter of the right side entirely unmolested. The lateral columns of the right side and the entire ventral half (gray and white) of the right side were normal. Of the tumor itself, of which sections were made and stained in picrocarmine, little is to

be said, as the appearances of a solitary tubercle are sufficiently well known and do not require accurate description.[1] The rest of the cord yielded the one negative fact that there were no traces to be found either of any ascending degeneration in the posterior columns or direct cerebellar tract above the level of the tumor, or of descending degeneration below the level. This fact was a great surprise to me. I can offer no other explanation but that the rapidly spreading myelitis effaced all traces of an ascending or descending degeneration. The myelitis was unusually severe and extensive, and before descending degeneration had time to follow upon the lowest level of myelitic change, death supervened. This explanation would not hold good if, as some authors would have it, degeneration, if it takes place at all, affects an entire system at once. But this whole question is still *sub judice*. (See Langley's Digest, *Brain*, April, 1886, and JOURNAL OF NERV. AND MENT. DIS., Aug., 1886, p. 496.)

Remarks.

It will now be our duty to review the clinical symptoms of this case in the light of the autopsy. In analyzing these symptoms it will be important above all things to differentiate between the symptoms due to the tumor and those due to myelitis.

The unilateral symptoms are to be placed entirely to the account of the tumor; the bilateral symptoms are the expression of myelitic changes, except that the extension of the neoplasm into the right half of the cord (surely a matter of the last few weeks only) may have given rise to sensory disturbances in the right extremities also. It is well in this connection to recall the following facts and dates: From Jan. 8th until March 1st the symptoms continued one-sided (the period of tumor symptoms); from March 1st to March 18th they were distinctly bilateral (the period of advancing myelitis). Unilateral symptoms during eight weeks, bilateral symptoms during two and a half weeks.

[1] The outer harder portion was characterized by small cells scattered here and there among the fibrous tissue; within the caseated mass there were found various cells in all states of decay, giant-cell formation, and a lot of detritus.

Regarded in this light, the symptoms would point to the fact that the myelitis spread almost as rapidly as any acute myelitis would. The question whether this myelitis was due solely to the presence of the tumor, or whether there was not possibly a tubercular myelitis in addition to the tumor and the myelitis directly caused by it, I must leave undecided for the present.

The tubercle was first deposited in the left half of the cord, at the level of the sixth or seventh cervical segment, and in the immediate vicinity of the posterior roots. The pains in the shoulder, which radiated down the arms (and possibly the vaso-motor symptoms), must be ascribed to irritation of the posterior root-fibres; that the ulnar should have been chiefly involved is not surprising in view of the spinal-cord origin of this nerve. With the growth of this tubercle—a relatively slow growth at best—there was a gradual increase in the paresis of the muscles of the left hand and fingers; as the tumor increased still further in size, it encroached upon the lateral columns, and caused a paresis not only of the left hand and fingers, but also of the left leg.

This slowness of growth is well proven by the fact that pains in the arms preceded the (motor) leg symptoms by several weeks, and that these motor symptoms progressed very leisurely during several weeks more, while the arm (ulnar) symptoms remained stationary. Pathological appearances would indicate that the tumor had occupied fully one half of the cross-section of the spinal cord. It would be fair to ask, Why (in this case) paresis and not complete paralysis of the left leg? A double answer might be given: First, in its slow growth the tumor displaced rather than destroyed the motor-tract fibres; and secondly, post-mortem appearances are apt to deceive us as to the number of fibres or amount of spinal-cord substance capable during life of conducting volitional impulses.

A similar explanation must be sought for the peculiar behavior of the sensory symptoms. I am now referring to those which preceded the onset of the distinctly myelitic symptoms. A tumor occupying one half of the cross-section

of the spinal cord would naturally interfere with conduction in that half of the cord; the symptoms following in its wake should be very much the same as those following upon hemisection of the cord, viz.: motor paralysis of the same side and anæsthesia of the opposite side. The doctrine of crossed anæsthesia from spinal-cord lesion, first enunciated by Brown-Séquard, has been confirmed an innumerable number of times; the facts of human pathology and the results of experimental physiology are entirely in accord with this view, not even Ferrier,[1] in his article on hemisection of the cord, having any fault to find with this portion of Brown-Séquard's views. In the face of so much corroborative evidence I should be loath to attempt a contradiction from the facts of a single case, however convincing such facts might appear to be. Moreover, if we assume that in my case the tumor increased very slowly in size, and that it pushed aside some instead of destroying all the sensory paths, the fact of a partial and not a complete anæsthesia will be satisfactorily explained. It is strange, however, that the side which, according to Brown-Séquard, should have been anæsthetic retained greater tactile sensibility at a time when the originally hyperæsthetic side had become anæsthetic in consequence of the spreading myelitis. I remind you of the statement in the notes of March 14th, that the sensibility to touch in the right leg, right half of trunk, and in the right upper extremity, though less acute than in the neck and face, was more acute than in the corresponding left members.

The behavior of the muscular sense calls for explanation also. On this point the views of Brown-Séquard and Ferrier are diametrically opposed to one another. According to the former, the muscular sense is abolished on the side of the lesion, while Ferrier's hemisection of the spinal cord of a monkey would go to show that the muscular sense is impaired on the side opposite the lesion. Ferrier says:[1] "The manifest difference between the use of the right (opposite) leg, with and without vision, clearly indicated the abolition of the sense of muscular contraction, and ina-

[1] *Brain*, vol. viii., 1885. [2] *Loc. cit.*, p. 4.

bility to appreciate the position assumed by the leg except with the aid of vision." When the animal was blindfolded, it was utterly unable to extricate its right leg from any opposition made to its intended movement; but if I understand Ferrier correctly, he makes no statement with regard to the leg on the same side as the lesion. The facts of my own case would argue in favor of Brown-Séquard's and against Ferrier,[1] for the patient exhibited in the earlier stages of his disease a grossly defective muscular sense on the left side, and normal muscular sense on the right side. Ferrier criticises Brown-Séquard for including under muscular sense simply the power to direct movements, and to this criticism I subscribe, for if Brown-Séquard were right, every paralyzed limb would necessarily be minus the muscular sense; but while I accept the same definition of muscular sense as Ferrier does (the sense of the position of the muscles, and knowledge of the manner in which any member has been moved passively), I find that in my case the muscular sense was abolished on the side of the lesion and not on the opposite side, as Ferrier would have it. We should come to some definite understanding regarding the use of the term muscular sense, and the tests to be applied in examining for the presence or absence of this form of sensibility. For the present we are grouping a number of heterogeneous phenomena under this term. This case certainly raises doubts whether all sensory fibres cross at once to the opposite side of the cord, and whether there be not sensory fibres in the lateral as well as in the posterior divisions of the cord. The other sensory phenomena observed in this case point to the necessity of a more careful study of the various forms of sensibility and their relations to spinal-cord lesions. As long as so little is known of the sensory parts of the spinal cord, I cannot do more than to refer to the eccentric condition of the temperature-sense, for instance, without attempting an explanation of

[1] Ferrier says: "So far as I have been able to discover by examination of the published cases of hemispinal lesion, the assertion that the muscular sense is lost on the side of lesion and retained on the opposite side, is not supported by any satisfactory evidence." Such evidence and such a case are here presented.

these facts. You will remember, for instance, that while heat was not felt as such in the lower extremities in the later stages of the disease, cold was distinctly recognized and produced a sensation of pain.

One other set of symptoms demands our attention. How can we explain the exaggerated knee-jerks and the presence of ankle clonus on both sides in the very earliest stage of the disease, at a time when there was only very slight paresis of the left leg and no paresis whatever of the right leg? Had the knee-jerk and the ankle clonus existed on the side of the tumor only, I should have said that the tumor during that period had been sufficiently large to remove some inhibitory influence without interfering much with the transmission of volitional impulses. It is barely possible that there may have been another small tubercle somewhere in the lateral column of the opposite side, and that this tubercle has been lost sight of in the vast destruction of cord tissue. In this connection I wish to recall the fact that Prof. Pitres,[1] of Bordeaux, published not long ago, a short article on the early appearance of ankle clonus in which he records two cases of hemiplegia in which ankle clonus was observed eleven and fifteen hours respectively after the onset of the apoplectic attack,—at a time, therefore, at which there could not have been degenerative changes in the lateral columns. If there be, as Langley suggests, commissural fibres connecting the lateral columns of one side with those of the opposite side, then lesion of one lateral column might remove enough cerebral inhibition from the opposite side to increase the deep reflexes on that side also. I question, moreover, whether the patient may not have had exaggerated knee-jerks and double ankle clonus before the onset of the disease. My suspicion is based upon observations I have made on a number of cases of neurasthenia from sexual and alcoholic excesses. In all of these cases I have found the knee-jerks markedly exaggerated; indeed I consider it a valuable symptom of the neurasthenic state, and can recall some cases in which I found even ankle clonus. It was stated above that no traces of chronic alcoholism were

[1] *Brain*, p. 310, 1885.

found *post mortem;* this, of course, referred to the condition of the large abdominal organs. That the long-continued abuse of liquor may have exerted its influence mainly upon the spinal cord, is more than likely. I prefer to leave this field of speculation. There are several plausible explanations,[1] and yet not one which is absolutely satisfactory. It is well for us to become conscious of the difficulties we may meet with in attempting to assign all clinical symptoms to their anatomical and physiological causes. With the exceptions just referred to, all other symptoms can be readily explained as dependent either upon the tumor or the subsequent myelitis.

The subject of tumor of the spinal cord is treated very briefly by our best authorities; Charcot and Erb giving the fullest account of the symptoms due to such lesions, while Leyden, Bramwell, Wilks, and even Strümpell and Ross pass the subject by in great haste. It would not be wise for me to attempt a full *résumé* of the entire literature of the subject, nor to analyze a large number of cases with a view to the differential diagnosis of tumors of spinal cord. This work has been well done by Dr. C. K. Mills and Dr. Lloyd, who have not only discussed the subject carefully but have tabulated fifty cases in the most painstaking fashion. In conclusion, only a few remarks, which will be designed to secure for this case its proper position in the rank and file of tumors of the spinal cord.

Extra-medullary tumors are by far more frequent than tumors of the spinal-cord substance; among intra-medullary tumors gliomata and tubercles[2] are more common than gummata, sarcomata, or cancerous growths. Our case was one of intra-medullary tumor. These are most frequent in the cervical and lumbar segments, to which our case does not

[1] Strümpell and Moebius, in a recent short article in the *Münchener med. Wochenschr.*, refer exaggerated deep reflexes to hyperexcitability (irritation) of the ascending (sensory) division of the reflex arc.

[2] This statement is contradicted by Mills' and Lloyd's table, but this is due to the fact that these authors do not distinguish between extra- and intra-medullary growths, and that they have tabulated *some* fifty cases and by no means *the* fifty cases of spinal tumor. Glaser (*Arch f. Psych.*, vol. xvi., p. 89) claims that tubercles have no great clinical interest, because they simply occur in the course of a general tubercular diathesis; in my case the spinal symptoms preceded the appearance of general symptoms by a period of nearly two months.

form an exception. The symptoms will naturally vary according to the position (level) of the tumor in the cord, and according to the histological character of the neoplasm. Extra-medullary tumors are generally characterized by pain in the back and by sensory disturbances due to compression of the posterior root-fibres; in my case there was no lasting pain in the back, but there were marked sensory disturbances, for the posterior root fibres were affected by an intra-medullary tumor, so that this one point of differentiation between extra- and intra-medullary growths is not supported by my case.

The focal symptoms[1] will vary greatly according to the seat of the tumor; there may be but slight sensory disturbances in a single area or in one limb; the sensory disturbances may be unilateral; and these may or may not be associated with partial, with unilateral, or bilateral paralysis. The number of symptoms may vary from one to a legion of such. The differential diagnosis can not well be referred to such an indefinite basis. Any set of spinal, and more particularly, hemi-spinal symptoms, whether of a motor, sensory, or vaso-motor order, if they be slowly progressive, might suggest tumor. There will be *good* reason to suspect tumor if there be in addition to some of the symptoms just referred to—some marked constitutional diathesis. In my case, I feel confident that no one who had seen the patient would have diagnosticated general tuberculosis, and I find that in one of two cases of tuberculosis of the spinal cord reported by Chvostek[2] some years ago, the tubercular diathesis was not diagnosticated during life, and the chief mischief was done, as in my case, in the spinal cord. The protracted course of the disease is supposed by some authors to be pathognomonic of tumor of the cord. The average duration is from six months to several years; my case ran its full course in a little more than two months. Such statements as these could be easily multiplied to prove the difficulty in diagnosticating this special form of disease; thus

[1] Indeed there may be no symptoms at all as in Simon's case. *Arch. f. Psych.*, vol. v., 1875.

[2] *Med. Presse*, 1873.

Richard Schultze,[1] a good observer, has recently published a case of tumor surrounding the cord from the cauda equina to the medulla oblongata, which was considered during life to be a case of myelo-meningitis. By a combination of favoring circumstances, I believe that a diagnosis of tumor of the spinal cord can be safely made; but in many cases, it will probably be, as Leyden says, a matter of chance whether or not the diagnosis can be made.

From my experience with this case, and my present knowledge of the literature of the subject, I should be inclined to make a diagnosis of intra-medullary tumor of the cord if a case presented motor, sensory, or vaso-motor disturbances over a limited area of the body below the head, if such symptoms remained unchanged or progressed during a long period of time, if these symptoms were followed after weeks or months by symptoms pointing to an advancing myelitis, and if there were reason to suspect some constitutional diathesis. In the case of extra-medullary tumors, evidences of disease of the vertebræ would greatly assist in establishing a diagnosis.

[1] *Arch. f. Psych.*, 1886, p. 592. A similar mistake was made by Friedreich in the case reported by Schultze. *Arch. f. Psych.*, vol. viii., 1878.

MORAL INSANITY: A PLEA FOR A MORE EXACT CEREBRAL PATHOLOGY.*

By JAS. HENDRIE LLOYD, A.M., M.D.,

INSTRUCTOR IN ELECTRO-THERAPEUTICS IN THE UNIVERSITY OF PENNSYLVANIA.

IN his "Essay Concerning Human Understanding" the master psychologist of our language, John Locke, has referred to those, who would be thought to deserve Midas' Ears, "who, knowing that rich was a denomination for the possession of riches, should demand whether riches themselves were rich." In this confusion of language our philosopher sees the fertile soil in which has flourished the confusion of ideas, which has so conspicuously marked the metaphysical and theological controversies about the mind and its faculties. He insists upon the distinction between a "power" and an "agent," and would have his readers plainly to understand that it is the *man* who perceives, judges, and wills, and not that there are "so many distinct agents in us, which had their several provinces and authorities, and did command, obey, and perform several actions, as so many distinct beings."[1] In other words Locke inculcates the unity and individuality of the mind, and cautions against granting to the faculties each its own autonomy. There is no such agent as a "will," he says, which acts and is free, but it is the "mind" which *wills to act*, and either is, or is not, free. *Powers* are merely the attributes of *substance*, and it is in this sense alone that faculties come from the cerebrum. It has seemed to me

*A thesis for admission into the American Neurological Association, read at its annual meeting at Long Branch, N. J., July 21–23, 1886.

[1] Bk. 2, Cp. 21, Sect. 6.

that in this part of his essay, this wise man has not only called the metaphysicians to account, but has clearly indicated a whole catalogue of errors into which many of our medical psychologists have too easily fallen. These errors have thus more than the mere speculative interest which attaches to the thoughts of those who dwell "above and outside of material things," but have a practical application, not only in every hospital, but in almost every court-house in our land. They have crept into our text-books, decided our practice, confused our science, and, unfortunately, sometimes vitiated our testimony.

It is quite possible for us to separate by a mental act phenomena which are quite inseparable in nature. Thus we can discourse about the *color* or *shape* of an object, quite apart from the object itself; but if we desire to alter that shape or color, or to deal with it practically in any way in the arts or sciences, it is very certain that we must leave off talking about the quality and must deal materially with the substance. This power of abstracting *qualities*, and then treating of them as though they were in themselves *substance*, has often been the bane of metaphysics; giving it that fatal immunity from the usual conditions of existence which has permitted it to dissolve itself in the mists of speculation. Nature differentiates by changing the substance of things; men, often by merely disarranging their qualities by an act of mind. Thus in the history of philosophy we see where vast generalizations of the metaphysical imagination have been converted into most potent personifications, so that men have from them attempted to erect even a science of biology, or have fallen down to worship what has no substantive existence whatever. I think we cannot lay claim to be free from this error even in our practical age—because it is a fault which only careful training seems capable of averting—and where we have such brilliant and learned examples of it as we see in the philosophers, who devote themselves to the study of truth, it behooves us, as mental physicians, to see to it that we do not fall into some of these very snares. I no not know in what other sense we can regard the celebrated *ideas* of

Plato, which he proclaimed as real, and the only real, existence; unless we look upon them as conspicuous examples of that particular perversion of thought which converts subjective mental processes into objective realities. I know that there has been a vast deal of speculation about what Plato's exact meaning was in his ideas, and one critic has even said that Plato himself was never quite sure, nor always the same in his definitions, but I know that this eminent philosopher's system has exerted a powerful sway upon the most illustrious minds (not merely because of its dramatic force and its ethics), and continues to absorb men in the fascination of its mystical ideas. They have even proposed to substitute it for Christianity; and one eminent Platonic scholar, Dr. More, is said to have been so imbued with it that he believed he was himself partly idealized, and much improved in his material components thereby—which was shown, among other things, by the fact that his urine exhaled the odor of violets! But the elder Disraeli[1] observes of this, that it was probably not so much due to Platonism as to diabetes!

The Scotch school of psychologists have even attempted to improve on Locke, but have met with so little success that their writings are probably among the last to which the modern school of alienists would look to sustain facts as they are actually found in the physiology and pathology of the "mind." This certainly seems a strange paradox—that those who devote their lives, and often signal abilities, to the study of the functions and qualities of man's highest organism, should find so little to illustrate its derangements and its essential characteristics! This fact has induced me to refer to their system in a paper addressed purely to physicists (among whom they do not count themselves to be), and this the more so because I believe that they exhibit most persistently and designedly these very methods of abstraction and personification to which I have referred. Sir W. Hamilton[2] defines psychology as the "science of mind," which in turn is the "conscious subject"; thus

[1] "Curiosities of Literature," Art.: "Modern Platonism."

[2] "Metaphysics," Bowen's edition.

mind is the *substance* (as he distinctly states) and consciousness is the essential *quality*—just as extension is to body. This evidently narrows psychology down to the science of self-consciousness. He makes mind the substance, entirely distinct and apart from brain, which he makes a different entity. Now what he defines as "mind" is only a congregation of the intellectual and moral faculties (in their highest stages of activity), which faculties literally are manifestations of the true substance, which is the brain. This criticism may be materialism—but truth itself is material! "Mind," as this school uses the word, is an abstraction. We cannot handle, gauge, nor understand it. They themselves are driven to inapt metaphors to explain its relation to the cerebral masses (as the double shield, one side of which was gold, the other silver; or the two clocks which keep time together), or else they calmly confess that this thing which they proclaim as a dogma is unintelligible to themselves! It would seem from Hamilton that all the phenomena of unconsciousness, partial consciousness, and perverted consciousness, and especially those brain (organic) phenomena which underlie consciousness—and are as yet unknown—have no place in his science or in fact. Carpenter, who seems to have been influenced very much by the Scotch school, has made a most fantastic attempt to harmonize "mind" and "matter." He states that mind is a "force," and that "forces" can be conceived of as independent of "matter," upon which they act;—instead of the true statement, that any and all forces whatsoever are manifestations of matter (unless we submit them to this by-play of mental abstraction). He goes on to say[1]: "The actions of our minds, in so far as they are carried on without any interference from our will, may be considered as 'functions of the cerebrum.'" In the name of natural science what are these "functions of the cerebrum" *when they are* interfered with by our will? Does the cerebrum then cease to act? Does it sleep, or does it collapse, or does it shrivel up? It cannot get out of the skull-cap. It cannot retreat down the *foramen magnum*—like a servant dismissed down the back-stairs.

[1] "Physiology," p. 543.

Where is the will when it exerts its baneful interference with the cerebrum? What is its exact anatomical and topographical location? How can the "actions" of one thing—"the mind," become the "functions" of another thing—"the cerebrum"? Can the *actions* of one thing ever become the *functions* of another thing?

> "What do you read, my lord?
> Words, words, words!"

In this convenient shifting of functions to and fro between the "mind" and the "brain," it looks as though the reconcilers were bent upon making the despised brain do all the work, while the exalted "mind" gets all the credit for it. But, unfortunately, this poor brain goes mad, and we have the grave responsibility of studying the truth about it! It might be easy for some to say with Heinroth[1] that "insanity is equivalent to sin." But nowhere else does it appear so plainly as in the Scotch school, that insanity is, in fact, bad metaphysics.

Let us refer, for the moment, to the use by this school of the word *consciousness*. They have made of it, in truth, a term to conjure with, and this, too, by this very process of personification, which I contend is so potent to spoil our investigations of the action of the brain. Sir William Hamilton attempts to erect it into a synonym for all brain action, a something apparently sacred, inscrutable, and, as he says with emphasis, something which cannot be defined; moreover, which never sleeps, which is the identity, and coincident with the soul of man; again, something immaterial, subjective, and in a peculiar sense the "Ego." To prove that it never sleeps, he had himself suddenly awakened from slumber, and claims that he always found his "consciousness" had been at work all the while! Dr. McCosh says[2] that "self-consciousness" is the one instrument of research in studies of the "mind," and apparently would acknowledge no other. But it appears to me that the "consciousness" of one man becomes *objective* to another

[1] Quoted by Bucknill.
[2] "Psychology," p. 2.

man, and that it is in these objective studies of "mind" that the alienist proceeds almost entirely in making his examinations of the insane. Reid believes self-consciousness a "separate faculty," by which the mind knows its own operations. McCosh again attaches a mystical sense to the word, and believes that "consciousness" has in some ways a peculiar quality "more of the essence of the soul." The truth seems to be that the etymology of the word supplies the best meaning—that is, "to know thoroughly," and thus applies to all mental acts which lead to knowledge. In fact, *it is* these acts, or only another name for them. It is thus present in different degrees in different acts of the brain, and in some cerebral acts is but dimly, if at all, present, because such cerebral acts are not very active, or are abnormal—as in dreams and in imbecility. The more thoroughly a man knows his present status and its surroundings the more thoroughly is he conscious. If he is absorbed in study he is not as thoroughly conscious as a moment later, when he is suddenly aroused by the inquiry —"What are you doing?" and, recovering himself, answers: "I am studying consciousness." Thus, in its highest activity it embodies distinctly self-knowledge; it is the much-lauded "self-consciousness," the mind-speculum of the philosophers. To put it into materialistic phrase, it is the highest ideation of the cerebrum. The self-consciousness of the insane is certainly a very different factor, both in philosophy and morals, from the "thorough self-knowledge" which, I suppose, Sir William Hamilton endeavored to exert on all occasions, even, as he says himself, when he was asleep. Homer gives a more curious example than Sir William Hamilton. I refer to the death of Rhesus, when stabbed as he slept by Tydides.

> "Just then a dreadful dream Minerva sent;
> A warlike form appeared before his tent,
> Whose visionary steel his bosom tore:
> So dreamed the monarch, and awaked no more."

The dream was *true;* the visionary steel was *real.* Was Rhesus conscious or unconscious when he dreamt the exact facts and allowed himself to be slain?[1]

[1] Iliad (Pope), Bk. 10.

Now in all this, as it appears to me, the word as used by the Scotch school is only another evidence of abstracting a *quality* or *mode* and making a distinct *agent* or *being* of it. When they say that they use "consciousness" as an instrument of research in psychology, they can mean in fact nothing more than that the brain, by its faculty of general knowledge, has also the faculty of special self-knowledge; and that it is not using any instrument, but is simply using itself. It is obvious that, by this method of self-inspection alone, we cannot formulate a complete science of the "mind." John Stuart Mill [1] believed in a separate "science of mind" as distinct from a cerebral physiology—but in a very different sense from the Scotch school, and chiefly because of our ignorance of the connection between mental states and changes in the brain substance, and not because those changes are not important, even identical with these mental states. All students of insanity avail themselves of the *phenomena* without being always able to give exact accounts of the *noumenon*, or substance, and its changes. We investigate clinically the expression, language, gestures, and actions of a patient; his habits, antecedents, and heredity, and so construct a diagnosis; but we acknowledge that this is often empirical, and we would much prefer, if we could, to give the morbid anatomy of our patient. This certainly does not prove that the science of gymnastics does not depend upon the anatomy of the body, even if it be not necessary to study Gray in order to exhibit upon the trapeze!

It is probably not too much to say that the most artificial abstractions (as mistaking a part for the whole, or a quality for the substance) and the most dangerous personifications (as erecting a single symptom into a disease, or narrowing a whole diseased organ into one "mental" faculty) have occurred in our courts of law. This is not always more the fault of the lawyers than it is of the physicians; but as it is the lawyers who have had to prepare and formulate the legal tests of insanity—to which the rest of the world, both mad and sane, must conform,—it happens that they have

[1] "System of Logic," eighth edition, vol. ii., p. 430.

especially exposed both their learning and their critical acumen to the study of mankind. The physicians are particularly interested in these tests, as they are the only prescriptions which a doctor is expected to take which are not of his own writing. The tests were somewhat crude, no doubt, in the olden time both for the disease and the irresponsibility which results from it. Fitzherbert defines an idiot as one "who knows not to tell 20 *s.*" Coke tells us[1] that once the king's safety was the test of madness: "In some cases *non compos mentis* may commit high treason, as if he kill, or offer to kill, the king." Thus a man could be mad on every subject but in that particular brain-centre which had regard to the personal comfort of kings. Hale says of this test:[2] "This is a safe exception, and I shall not question it, because it tends so much to the safety of the king's person," which is a safe conclusion on the part of a chief justice who, having originally taken his seat under Cromwell's government (which had cut off a king's head), was probably desirous to keep his place upon the accession of Charles II. to the throne—even, if necessary, by sentencing every madman in the realm. This method of determining insanity was almost as good as Hamlet's diagnosis of his own madness by the compass:

> "I am but mad north-northwest;
> When the wind is southerly I know a hawk from a hand-saw."

Hale, again, who was a fertile psychologist, said that lunatics had their lucid intervals "which ordinarily happen between full and change of the moon," during which time they are responsible. He would make the understanding of a child of fourteen years a test for responsibility; whoso had less than this should escape, and whoso had more should be hung—but he does not say who would determine these points of comparative psychology. He it was, apparently, who originated the division into "total" and "partial" insanity—as used to convey the idea that a part of the brain was diseased and a part not, like a half-rotten apple, or a

[1] "Institutes of Laws of England," 3d p., cap. i., p. 6.
[2] "Pleas of the Crown."

"mildewed ear, blasting his wholesome brother." Every student of insanity knows that "total" insanity is an impossibility—as it could mean nothing but the total abolition of all brain functions—and that "partial" insanity is a misnomer for *symptoms* and *degrees* of brain-illness, in which respect it would be hardly possible to say that any two cases were alike or exactly on a level. It is but just to Sir Matthew Hale to say that while he contends that some idiots—"*surdus et mutus a nativitate*"—have enough understanding to warrant their trial, still "great caution is to be used" in executing them! I believe it was in Hadfield's case (he who shot at the king) that the notorious division of the subject into *civil* and *criminal* insanity was made. "If, in the former," says Shelford,[1] "a man appears upon the evidence to be *non compos mentis*, the law avoids his act, though it cannot be traced or connected with the morbid imagination which constitutes his disease, and which may be extremely partial in its influence upon conduct; but to deliver a man from responsibility for crimes, above all for crimes of great atrocity, this rule does not apply, however well established when property only is concerned, but the relation between the disease and the act should be apparent." It is not probable that an English lawyer's veneration for property, and disregard for human life, could be more strikingly displayed. I am fully aware that I am not treating here a novel theme, but it is one so germain to the general drift of the subject that I do not resist the temptation. I desire to make complete these illustrations of man's futile attempts to artificially construct our science. We who listen to kindred attempts on the part of our own profession can at least derive thus some benefit from the study of law, of which, Blackstone says[2] he sees no special reason, that it should be pursued by "gentlemen of the faculty of physic." It is from this very philosophy that have sprung our *nymphomania* (insanity of the clitoris), *kleptomania* (imbecility of the pocket-book), *monomania*, in which not the patient, but *one* of his ideas, is insane, and *moral insanity*, in which nothing is insane about him but his sins.

[1] "Treat. on Law Concerning Lunatics," p. 42.

[2] "Commentaries," Introduct., p. 8.

The gentlemen of the law, however, have probably risen to their boldest speculations upon psychiatry in some of the more modern tests which they have proposed. These are especially (1) delusion and (2) knowledge of right and wrong. These tests are so narrow that they cannot cover the subject, and so incomplete that they do not touch it even in the right way. A delusion is not the cause of insanity, but insanity is the cause of the delusion. "Loss of will-power," which some medical authorities insist on, is equally abstracted and artificial. It is nearer the truth to say that the whole mental act of an insane man is wrong (judgment, conscience, memory, and will). In the ideo-motor reflex these are but different *modes* of action of the one *substance*. They can be separated in speculation, but neither in physiology nor pathology can they be so dissected, and one part held up as normal and another part as abnormal. To say that a man's intellect is sound, and his will diseased, is a sophism, which has more sound than reason, and is no better than to say that we have his light without the sun himself, or that a Leclanché cell has electro-motive force but no current strength. Thus the word *delusion* is constantly misused; and is really so vague and generalized a term that no one has yet succeeded in giving a definition of it. With some, it is any kind of impaired action of a sick-brain; with others, it is an elaborated and systematized complex idea. The physicians, repelled by the *doctrinaire* tests of the lawyers, have flown to the other extreme. Thus Blandford speaks of "homicidal insanity without delusion." If this means any thing it must be a condition of impaired brain (memory, intellect, emotion, and will) which has not yet originated a systematized, elaborated, symmetrical delusion, such as an erroneous belief, scheme, or suspicion. This impaired brain, if expressing itself in an act of violence, no doubt need not necessarily have elaborated an harmonious, far-reaching design, but acts from ill-contrived, suddenly conceived, or even fragmentary ideas, often of the class of thoughts which we call emotional or passionate. This is the nature of *insane* thoughts, to be both illogical and sporadic—but it would be

advancing to an extraordinary extreme to say that such a brain in such an act had no element of intellect or judgment in it; and it would remove the lunatic not only out of the pale of justice, but out of the realm of nature, to say that he had "impulses" without "motives," if by that is meant that it were possible for him or any man to have even a weak or perverted volition without also some weak or perverted thoughts from which such volition sprang. The will is the intellect in action. The latter may be called cerebration *in esse*, the other *in motu*.

Knowledge of right and wrong, as a test of mental health, indicates this tendency to artificial distinctions or "localizings," as it presupposes a strictly moral insanity, a lesion of the *conscience*, or judgment of moral things. It is an extreme case of "moral insanity." It requires the existence of the most illogical "partial" insanity—an insanity of that part of the judgment alone which has cognizance of things good and evil. To be able to reason correctly upon the abstract subject of right and wrong, is very different from the state of ideation which may exist in regard to one special, concrete act, even the worst imaginable. Into the latter act comes the personal equation—the *man* with his diseased brain. To him it may seem, perhaps,

Dulce et decorum est pro patria mori,

but it may, therefore, appear also proper to shoot at a President. It is indeed a peculiar reflection that the "knowledge of right and wrong" should be put as a test to a poor lunatic, when sane men have been fighting each other for ages to decide the difference between the two, and the first parents were even expelled from Paradise for their too great curiosity on the subject. "My child," said the learned judge to a six-year-old witness, "do you know where you will go when you die if you tell a lie?" "Please, sir," said the witness, "I do not." "Well," observed the Judge, "neither do I."

"Non compos mentis," has long been a term in the law, and is often used to designate something which is conceived

to be quite distinct from "insanity." This has drawn forth an able criticism by Dr. Forbes Winslow. Senile dementia and the gross forms of brain degeneration, as embolus, hemorrhage, multiple sclerosis, and dementia paralytica, are such cases as would probably come under this class. Here the most patent forms of brain deterioration, those upon which we can literally place our fingers, are put out of consideration as quite too material to be classed with the "insanities." Such men have only a disease of their *bodies*, and not of their "minds," hence they do not suffer opprobrium. For what says Chitty?[1] "The malicious, untrue written assertion, that the king or any person is affected with insanity, is considered a criminal and indictable act, since it imputes to the party a malady generally inducing mankind to shun his society, though, as no one is of perfectly sound mind but the deity, it is not libellous merely to say that a man is not of sound mind."

I have been led thus far in the discussion of the philosophy and law of this subject, because it is from the former that we have much of the theory, and from the latter much of the practice, of this question. I come now to offer some consideration of the medical, or physical, side of the subject. Whoever has taken the pains to read Rush, will observe, perhaps with surprise, that he distinctly outlines the "moral insanity" of later authors. Prichard was long thought to be the originator in English of this generalization, but it is evident that he must yield the distinction to the American physician. The doctrine has had the most brilliant support, and it is only necessary to mention with Rush and Prichard the names of Ray, Winslow, and Maudsley. This question therefore has vitality (and so needs no excuse for its discussion) because (1) it has the approval of such eminent names, and (2) because it is constantly relied upon to acquit a certain class of lunatics in our courts, and almost invariably proves to be a "broken reed," which will not support him who leans upon it. I have always been reminded, when reading the authors mentioned, of Locke's criticism of those who maintained the doctrine of *innate*

[1] "Med. Jurisp.," vol. i., p. 353.

ideas, *i. e.*, that they do not tell us what these particular ideas are which they thus claim to be innate. So with those who uphold the doctrine of a "moral insanity," they do not give us the cases which prove this disease; because it is evident upon close inspection of their illustrative cases that they all exhibit various proofs of intellectual disorder, such as expansive ideas, imperative conceptions, suspicions, and a general course of depraved, or excited, or morose conduct which it is impossible to conceive of as existing without derangement of the intellect. These cases, moreover, range at large through all the classifications now in vogue, and include, promiscuously, cases of imbecility, hysteria, katatonia, melancholia, mild mania, paranoia, and even some specimens of delusional insanity. When their "moral idiots" commit suicide or homicide they say that it was done "without motive," to which the criticism applies that (1) it is difficult to penetrate into the minds of such patients; (2) that motive is shown frequently by a strict analysis of former and subsequent language and actions; (3) that no such analysis has evidently been attempted in many of these reported cases; and (4) that it is nearer the truth, and therefore a better plea, to show an insane mind with insane motives, than a "moral" lunatic without any motives whatever.

The doctrine of "moral insanity" proceeds upon an abstraction just such as Locke warns his readers to avoid. It teaches that there is a moral "faculty" in the sense of a distinct agent, which has its own powers and its own diseases, and which may remain undeveloped in a "mind" otherwise healthy, and may become diseased without at all affecting the health of the other "faculties." Yet so artificial is this abstraction, that there seems to have been as much difficulty with the philosophers to give this "faculty" a correct place in classification as if it had been the 4th quality of space. Dr. McCosh[1] cannot put it in either the "cognitive" or the "motive" powers, but gives it an intermediate place between the two. But Meynert,[2] with great precision, says: "It is taking altogether too simple a view of things to regard morality as one of man's talents,

[1] *Op. cit.*, p. 15.

[2] "Psychiatry," trans. by Sachs, Preface.

and as a definite psychical property which is present in some persons and lacking in others." He quotes Weissman: "Talents do not depend upon the possession of any special portion of the brain; there is nothing simple about them, but they are combinations of many and widely different faculties." That is to say, that talents or powers (moral, intellectual, emotional, volitional, mnemonic) are complex processes and obscure modes of *one substance*—the brain. The conscience, which, by a scholastic definition, becomes a distinct "faculty" in some systems of philosophy, is in reality nothing but a judgment of things good and evil, and has no more claim to its present distinction than has taste as a judgment of things beautiful. It does not refute this to say that conscience is more than judgment because it is emotional, for such a criticism goes to prove the unity of "mind." All ideation has its distinct modes, and may pass from one to another with great rapidity. An idea which at one time may seem to be of the judgment, does not essentially change its nature, or become another thing, by putting on an emotional phase in another moment. Some have contended that there is no act of "mind" which is entirely indifferent—*i. e.*, without emotion. Sir Wm. Hamilton says: "Cognition and feeling are always co-existent."[1] Even if there be such indifferent cerebation, it is certain that a very slight cause may put it in an emotional mode. Emotion, as originally signifying a *moving* of any idea or mental state, is but a *mode* of ideation, not a distinct department, faculty, localization, or division of man's cerebrum. Some ideas are not apt to be thus moved, being almost or entirely disassociated from pleasure or pain, while others are almost of necessity constantly in this state, because they have to do entirely with things good or bad. But a *moral* perception, again, has sometimes but little of the emotional mode, the brain being rather then contemplative. And on the other hand, emotional states of the brain are not always of things good and evil in the sense of *morals*. It is impossible for me to conceive of an emotional state of the cerebrum which does not include as essential the

[1] See Laycock: "Mind and Brain," vol. i., 171, 172.

state also known as the intellect. This confusion of speech is no doubt helped on by the personification of the word "idea." Mental acts are abstracted into distinct entities or ideas, and these again are labelled and classified like butterflies in a museum; some are "emotional," and some are "sensational," and some are "cognitive." Some become sick, and others flourish like bacteria in beer. They fall together into amazing and entrancing shapes, like bits of glass in a kaleidoscope, or they are tumbled into inextricable confusion, like the blocks of a toy-house. They are dissevered from the brain, and even the "mind" is set to looking at them as something apart from itself. Instead of the *brain* which knows *objects*, it is the "mind" which is cognizant only of "ideas." It reminds me of an insane man who was once confined in an asylum near Philadelphia, who had separated his *identity* from himself, and then accused somebody of having stolen it, and spent a whole day searching over the town for the thief. It cannot be wondered that, with such speculations, exhausted philosophy falls into utter skepticism, or rushes into the shelter of some materialistic theology. It is not to the credit of psychiatry—which is the science of a diseased cerebrum—that any such methods should confuse its results. Moral insanity, and its big brood of special manias, is but the creature of bad science; but the unfortunate insane, who are stigmatized by the term, and robbed often of sympathy and justice, have only too true an existence.

The modern experimental school of brain physiologists, represented in England especially by Ferrier, have perhaps given us more solid information about the cerebral masses than the great bulk of philosophical speculation on the subject during all the ages. These physiologists have demonstrated, or indicated, the only "localizations" which are likely ever to be demonstrated, because they are probably the only ones which exist. Their studies enforce the distinction between a "localization" and a "mode of action." In other words, they have demonstrated that the brain is a great *sensori-motor* organ, and have succeeded in mapping out pretty accurately the areas in which sensations first

strike upon the brain, and the areas from which motor impulses pass out of it. They thus not only confirm the analogy of the brain to the lower ganglionic centres—from which it is probably an *evolution*—but they furnish a solid basis for the system of Locke, who taught that sensations (impressions from without) were the origin of "ideas" (cerebral activities), which are expressed in action by motor impulses. But the grave question arises—What is there between these sensory areas and motor areas? Where are the centres for judgment, imagination, and emotion? I have already said that these are not capable of localization, that they are essentials of *all* cerebration, they are modes of action, and that such cerebration exists both in the sensory and motor areas. Ferrier implies that every sensory area is both retentive (has memory) and comparing (has judgment). It must therefore also be emotional. It is possible, indeed, that some of these areas are more active in one of these *modes* than the others, but it is not possible that any one area should have the monopoly of any one of them, any more than the lungs should display distinct activities in one part to the exclusion of such activities in another part. Ferrier reduces all ideation or cerebration to two elements—sensory and motor. Every "idea" exists in *all* sensory and motor areas that have ever been concerned with it. Thus, he illustrates an orange, which, in idea, exists in optic, gustatory, olfactory, and certain motor centres (those concerned with dimension). This fact is so strikingly displayed in the phenomena of *aphasia* that it seems to be the key to the whole situation. Such a patient has not lost a "speech centre," because he understands spoken and sometimes written language. But he has lost the use of the motor area from which language flows out of his brain, and just that much ideation and no more. He forgets this much of his motor power. He no doubt continues to think to himself in language, just as he understands it when it flows from the mouth of his doctor in upon his brain at his healthy auditory area. Ferrier[1] sums up the subject: "Ideas, therefore, except in so far as they are simple revivals of definite and uncomplicated sensory

[1] "Functions of the Brain," p. 267.

impressions or motor acts, have no circumscribed habitation in the brain, but are the re-excitation of each and every one of the sensory and motor centres which are especially concerned in their acquisition."

The application of these important facts to the study and differentiations of insanity seems to me to be evident. It must tend to do away with the sophistical and artificial distinctions which are largely our inheritance from the philosophic schools, and to regard insanity as a disease of that unit, the brain. There are important and vital distinctions as to the forms the disease may assume, but they are not to be regarded as the expression of any individual "faculty." That such a new philosophy begins to prevail is not a little to the credit of our alienists and neurologists.

The term "ideation," which has been used in this paper, may be defined as that specific act of the organic cerebrum which is usually known and defined by its different modes —sensation, memory, judgment, emotion, and volition. The minute, histological changes which underlie or constitute this ideation, or cerebration, are as yet unknown, as well as many of their morbid actions which lead to or constitute insanity. In this respect our knowledge of ideation is in neither a better or worse state than it is concerning electricity, heat, gravitation, or life. A poor explanation of these phenomena is worse than none. It does not help us to say, with Luys, that they are "purified vibrations," because how does a *purified* or any other *vibration* explain memory and judgment? Neither does it aid us to talk, with Lewes and Gowers, about "lines of least resistance." What is a line of least resistance in a nerve tract or series of tracts? Why does it resist less, and what does it resist less, than other tracts? Is it a "physiological" line, or is it a "topographical" line? Can the mere *direction* of a force, with or without regard to its *resistance*, explain the mechanism of an idea?

Let us rather rest content in our ignorance for a while, conscious that words in themselves have a strange power to propagate error, and fearful lest the few shells we have already found upon the shores of the ocean of truth should after a while hatch out a barnacle-goose.[1]

[1] Max Müller, "Science of Language," second ser., p. 555.

FACTS AND DEDUCTIONS BEARING ON THE ACTION OF THE NERVOUS SYSTEM.

By FRANCIS X. DERCUM, M.D.,

INSTRUCTOR IN NERVOUS DISEASES UNIVERSITY OF PENNSYLVANIA, PATHOLOGIST TO THE STATE HOSPITAL FOR THE INSANE AT NORRISTOWN.

TO the student of the physiology of the day, the nervous system is apt to appear as a collection of complex and independent structures. He hears so much about centres and about localization, so much about the function of this part and the function of that, that he is apt to acquire a confused notion of so many *separate* things upon which the will plays very much as a man plays upon the keys of a piano. When, too, he comes to study nervous pathology the impression is apt to be confirmed, and his knowledge of detail displaces or rather suppresses ideas of a general nature. It has been partly to place in a prominent light the erroneous nature of such impressions, and partly to present some new views on the action of the nervous system, that this paper has been written.

The nervous system develops, as we know, from a simple longitudinal involution of the epiblast, and the various parts of the tube thus formed differ at first in no essential points from each other. It is only by a gradual change, here a dilatation, there a constriction, that the various segments of the cerebro-spinal tract arise, and the further differentiation of its parts proceeds in the same gradual manner without leap or break. Evidently, then, when development has been completed, when the highest degree of complexity has been reached, the nervous system, though made up of a myriad members, is still a *unit*, is still *one*

whole. Again, in the beginning, all of its parts are in undeniable relation with each other, and in the adult state these relationships must still be maintained, though here and there they are less intimate.

Facts of comparative anatomy lead us in a similar direction. We have, to begin with, the undifferentiated simple nervous axis of the amphioxus, which corresponds to the primitive neural tube of the embryo. Then, by a gradual transition, we have differentiation of the encephalic and spinal portions up to the condition in which we find them in the higher mammals. The inference again is obvious that the mechanism, though complex, is still *one* and *not multiple.*

How is it that the nervous system, at the same time complex in structure and manifold in function, preserves its primeval character of a simple unit? Let us inquire more closely into the development of its parts, the segregation of its centres.

It is extremely probable that in a simple nervous system, such as we find in the amphioxus or see represented in the early stages of the embryo, the various functions are performed equally and indifferently by all portions. Now, evidently, the first step towards specialization must have been the *concentring* or *focussing* of certain functions in certain areas. This concentring or focussing of functions was probably determined by such simple facts as the mechanical relations of the nervous system to the other structures, certain functions being performed more readily in certain positions than in others. Again, given a concentring of function sufficiently well-defined, it must, in the course of time, have been followed by a change of structure more or less marked. It would readily appear, therefore, that the complex and highly differentiated nervous apparatus of mammals has arisen by a constant and cumulative repetition of this process. In some portions *great* structural modification has resulted, in others *little* or *none that is demonstrable;* as witness the various centres which physiologists are unable to fix and describe anatomically, but of the existence of which they feel no less certain.

Again, it is very evident that while in certain localities centres are being developed and structure correspondingly modified, there must still remain vast areas or tracts of nerve tissue in which little or no change has taken place and the function of which is still of a *general* character. That such areas whose functions are general or relatively general, are still present in every nervous system can, I think, be shown to be extremely probable. Let us take up, for instance, the much-discussed question of cerebral localization. Physiologists, as we know, have fixed upon certain portions of the cortex to which they ascribe the property of originating the voluntary muscular movements, and upon certain other portions to which they ascribe the property of perceiving the various sensory impressions. Now, as is well known, these areas, though distinctly defined physiologically, are poorly defined anatomically. A portion of cortex distinctly irritable passes without breach of continuity and without obvious or marked change of structure into portions of cortex that do not respond to any stimulus whatever. Again, this apportionment of the cortex leaves out the bulk of the vast surface of the brain, to wit, the frontal lobe, the island of Reil, the occipital lobe, all of the mesial and the greater part of the basal surfaces.

What then is the meaning of these vast areas? Attempts have been made, and upon unsatisfactory grounds, to ascribe the intelligence to the frontal lobes, and still less clear notions prevail with regard to the occipitals. As we proceed, such views will, I think, be shown in their very nature to be untenable. Obviously these large areas *are portions of the cortex whose functions are still relatively general.* The so-called motor and sensory areas, on the other hand, are nothing more than *points where functions general to the brain are concentred, and this concentring takes place because these points happen by their anatomical positions to be gateways of ingress and egress to the general cortex.*

Let us approach the subject from another standpoint.

The life in a child at birth is almost purely spinal. We know, from the researches of Flechsig and of Parrot, that, as regards the development of nervous tissue, its brain is in

an embryonal condition, and that "it is the motor zones, so-called, that are first developed, and are the first to place themselves in relation with the bulbo-spinal system, by the intermediation of the pyramidal fasciculi. . . . It is only at the end of a month that the substance of the occipital lobe begins to whiten, and only after four months, and close to the fifth, do the anterior regions begin to be developed, and this development will not be accomplished until towards the ninth month."[1]

Now, to ascribe to the pyramidal cells of the motor areas *purely motor* ideas, is to my mind absurd. We must remember that although the cortex of the motor area is the first to develop, that, hand in hand with this development, the child is evolving, not only in the direction of voluntary movement, but also sensorially, intellectually, and emotionally, *psychically in every respect.*

Before leaving the subject of central localization, another point which appears inexplicable on any other view than that here maintained in relation to the development and integration of centres, is the localization of the faculty of speech in the left third frontal convolution. The question at once arises, What is the function of the *right* third frontal convolution? Obviously it is one of those areas possessing a function of merely general value, and, if the observation of Gratiolet, that the left frontal convolutions are developed before the right, be correct, we have a ready explanation as to why concentrating of function should have gone on in the left gyrus far in advance of the right.

The ideas thus far developed naturally suggest that the nervous system, in its simplest states at least, is *vicarious* in its action. As it becomes more complex such action must become more limited, yet even in highly differentiated forms evidences of the occurrence of such action should not occasion surprise. We are all aware that some physiologists claim its occurrence for the cerebrum, and that they ascribe the temporary character of the paralysis following ablation of certain cortical areas, to the function being taken

[1] "Lectures on the Path. Anat. of the Nervous System—Diseases of the Spinal Cord," by Charcot. Translated by Cornelius G. Comegys, 1881, pp. 30, 31.

up by corresponding parts in the other hemisphere or by neighboring areas in the same.[1] Certain facts of pathology also suggest this view. Extensive lesions caused by tumors of gradual growth are, as is well known, frequently unattended by noticeable symptoms. The obvious explanation is, of course, a supplementary or vicarious action by other parts of the brain.

Again, in such states as represented in the embryo and in amphioxus, the nervous system is undeniably a simple whole, and every one must admit that there is a maintenance of equilibrium among the parts of this whole. Now, whatever disturbs the equilibrium of a part, disturbs the equilibrium of the whole. Therefore, in this primitive nervous apparatus, changes taking place in any one part would affect all other parts as well. Now, if this be true of so simple a nervous system, it must unavoidably be true of one more complex. Though complex it is still a unit, and it must react as a unit. Facts depending upon this law of the maintenance of the equilibrium, have for a long time been recognized by physiologists, and have been described by them as phenomena of "inhibition." They tell us that certain centres are "inhibited" by others. In part they are right, in part they are wrong, for the action must be mutual—must be from higher to lower and lower to higher centre as well. That the brain inhibits the spinal cord is readily demonstrable, and this fact is not be wondered at. We have, on the one hand, a mass relatively large and complex; on the other, a mass relatively small and simple. In consequence, the latter is, as it were, overbalanced; and yet relatively small and simple as the cord is, it must have a counterbalancing action on, the brain, though this is, in the very nature of the arrangement, not demonstrable. However, brain and cord are but parts of one thing, and it is but a question of action and reaction between parts.

Thus far we have endeavored to show, from general considerations alone, that the nervous system acts as a whole. Let us now turn our attention to other fields of evidence.

Several years ago, together with Dr. A. J. Parker, the

[1] See Exner, Hermann's "Hdb. d. Phys.," vol. ii., p. 332.

writer made some interesting observations on the artificial induction of certain convulsive seizures.[1] As these experiments are of special importance to the subject in hand, a short account of them is here inserted. They were performed by subjecting one or more "groups of muscles to a constant and precise effort, the attention being at the same time concentrated upon some train of thought. . . . After the lapse of a variable period of time, . . . *tremors* commenced" in the part subject to the experiment, which was most frequently the hand. "These tremors became rapidly magnified into rapid movements of great extent, sometimes to and fro, sometimes irregular. If the experiment was now continued, the muscles of the arms, shoulders, back, buttocks, and legs, became successively affected, and the subject was frequently thrown violently to the ground in a strong general convulsion. The muscular contractions frequently became tonic, so that opisthotonus, emprosthotonus, and the most bizarre contortions were produced in various degrees."

The general explanation of these phenomena, given at the time, is briefly as follows: "A person places his hands, or any other part of his body, in a position of effort. . . . What must happen? Evidently, . . . there must be a rhythmical series of motions taking place between the antagonistic groups of muscles, because the rhythms, "*i. e.* of the susurri," of these cannot be synchronous.

At first the will restrains any tendency to marked vibration in the part, but the restraining influence being gradually diminished or held in abeyance, "we find, as a result of the exhaustion of neuro-muscular protoplasm, that a disturbance must take place. This disturbance must necessarily be rhythmical. Hence we have a rhythmical motion of the part, as shown by increased tremor. The action continuing, the tremor is succeeded by to-and-fro movements. At first it affects only the parts under strain, but this disturbance, which might perhaps be considered a purely local phenomenon, speaks through the mobile channels of the

[1] "On the Artifical Induction of Convulsive Seizures," by F. X. Dercum, M.D., and A. J. Parker, M.D., JOURNAL OF NERVOUS AND MENTAL DISEASE, vol. xi., No. 4, October, 1884.

neuro-muscular appatatus, and affects it as a whole. Thus we see that, beginning with tremor of the hand, we finally have the flexors and extensors of the forearm thrown into violent clonic contractions. Next the muscles of the arm and shoulder are involved. At last, such a violent explosion takes place that the neck, the back, the legs, the diaphragm, the heart even, is affected."

Now, that tremor should follow upon placing opposed groups of muscles in a position of strain, is perhaps nothing more than what we could expect from *a priori* consideration, and is indeed a fact in accord with common experience, but that *distant parts* not primarily involved in the experiment should become affected, is indeed remarkable. Evidently a general cause, one that acts upon the *entire nervous system*, comes into play. In the first place, the power of the brain to inhibit or overbalance the spinal cord, is through its expenditure in concentration of thought gradually lessened, until not only the muscles under strain are beyond the influence of the will, but also the entire muscular apparatus. Now, as we shall presently see, the states of tonus of the various muscles are closely correlated. A change of tonus in any one muscle affects the tonus of all the rest, and we can readily understand how such marked changes of tonus, as tremor, clonic movement, or tetanic contraction, are transmitted to different parts.

That the states of tonus of all the muscles are closely correlated, is a legitimate inference from the remarkable observations of Drs. S. Weir Mitchell and Morris J. Lewis[1] on the reinforcement of the knee-jerk by coincident muscular movement in distant parts, and also on its reinforcement by coincident sensory impressions. These studies have already become so widely known that it is perhaps unnecessary to give an account of them, however brief, here. Suffice it to say, that movements of the limbs, or even such slight movements as winking or swallowing, when coincident with

[1] "Physiological Studies of the Knee-Jerk, and of the Reactions of Muscles under Mechanical and Other Excitants." By S. Weir Mitchell, M.D., and Morris J. Lewis, M.D. *Medical News*, vol. xlviii., p. 169 and 198. Jendrassik also observed increase of the knee-jerk by coincident clinching of the hands.

the blow upon the tensor, produce increase of the knee-jerk. This increase is also present when a marked and coincident sensory impression is made, such as twitching a hair, pinching or pricking the skin. We have reason, then, to believe that whenever any one muscle has its tonus increased by a volitional effort, there is an overflow to the other muscles likewise, and also that the inflow of molecular movement caused by a sensory impression, is followed by an outflow, not through any one channel, but through many, probably through all. To quote the language of Drs. Mitchell and Lewis: "Under this view we conceive of the nervous force as not confined entirely to the direct paths between the centres and the muscle to be moved, but as overflowing so as to pass through numerous ganglia, adding a certain small increment to their effect when in a state of such activity as the spinal toning centres must be at all times." Certainly, it must be in some such way as this, that in our artificially induced convulsions, tremor and clonus are communicated from one group of muscles to another.

The facts brought to light by our experiments on artificial convulsions and the discoveries of Drs. Mitchell and Lewis can be explained on no other supposition than that the nervous system acts as a unit. The close and varied relation between distant parts demonstrated in so forcible a manner, admits of but one interpretation,—one, too, that is pregnant with thought. Inasmuch as every voluntary movement presupposes a corresponding mental action, the general mental state must be in more or less intimate relation with the general muscular tonus, and special mental states must stand in intimate relation with the tonus of special groups of muscles. A moment's reflection will convince us that this is the case. We habitually judge of a man's mental state by his facial expression, and this we know to be dependent upon the tonus of the facial muscles. We unconsciously judge of his personal traits, character, and disposition by his carriage and gait, which in turn we know to be dependent upon the tonus of the muscles of the trunk and limbs. Thus the varying states of the muscular tonus, though there are other factors, constitute a means by which

we habitually acquire a knowledge of the inner nature of our fellows.

Again, facts physiological and pathological, show that the nervous system is related intimately to almost all, if not all, the other tissues of the body. We know it to be the case with the glands, the bones, the skin, and the appendages of the skin. We know that certain nervous affections are accompanied by disease of the bones, that the activity of certain glands is absolutely dependent upon their innervation, and varies markedly with mental states, that the skin sloughs in certain nervous lesions, etc., etc.

Standing in intimate relation to the nervous system, the activity of these tissues, as well as their nutrition and growth, are probably subject to conditions similar to those which govern the muscles. Doubtless, too, the activities of all the structures of the body are more or less closely interrelated. We are all familiar with the harmony of action existing among various viscera, with the atonic digestive troubles associated with depressive mental states, and with such facts as the arrest of digestion by violent physical exertion. Furthermore, the activities of the glands may at times be in even *intimate* relation with the muscular tonus, as can be inferred from the occasional behavior of the lachrymal glands and kidneys during the artificially induced convulsions.[1]

In this brief essay important facts have been merely mentioned and important principles merely touched upon, yet in summing up we may safely say that considerations of embryology and comparative anatomy, and various facts derived from other sources, point infallibly to the conclusion that the nervous system, though inextricably complex and composed of an almost infinite number of parts, *acts as a whole;* that it constitutes an engine the various parts of which are so closely inter-related, so mutually interdependent, that no one part can move unless every other part, no matter how slightly or how profoundly, moves also.

[1] *Loc. cit.* The strange and mysterious facts of the correlation of nutrition collected by Darwin, may also find here a key-note of explanation.

KATATONIA.

By W. P. VERITY, M.D., Chicago, Ill.

CONSULTING SURGEON COOK COUNTY HOSPITAL.

THE present form of insanity was first demarcated by Kahlbaum [1] in 1874, and his results were confirmed by the researches of Kiernan,[2] who was the first American to recognize the affection. Observations were subsequently published by Brosius,[3] Hecker,[4] Schüle,[5] Arndt,[6] Hammond,[7] Spitzka,[8] Hardy,[9] Clevenger,[10] and C. K. Mills,[11] which still further supported the Prussian alienist's position. Cases clinically meriting demarcation under this group have been reported by the author of "Sketches in Bedlam," by Krafft-Ebing, Sankey, Culléré, Legardelle, and Adam. Kiernan states that "the first symptom like that noticed in the inception of most other psychoses is a change in the temper of the individual. Katatonia presents at times well-marked motions of rhythmical character, always under the control of the will. In this respect, these, while bearing some resemblance to, are very distinct from, those of chorea. Another characteristic, but which is not noticeable unless the case be observed from the inception to the close, is its cyclical character—maniacal, melancholic, and cataleptoidal conditions alternating with more or less perfect convulsive attacks; there are also pathetic delusions of grandeur, and a tendency to act and talk theatrically. Erotic manifestations of some kind frequently occur, and, as is usual under such circumstances, the patient's ideas have a religious tinge. At any stage, as in other nervous diseases, remissions, or, as is claimed by Kahlbaum, complete recovery, may occur. If

the case is to end unfavorably, periods of excitement and stupidity recur with more and more frequency, and the patient dies with terminal dementia."

As a means of comparison, I cite two of Kiernan's cases. T. R., age thirty-six, policeman, single, common-school education, intemperate, as were also his parents. The patient had been a masturbator, and had indulged in sexual excess. He was at first melancholic, subsequently maniacal, but recovering therefrom, became what his fellow-policemen called "stuck up." His temper changed from good-humor to irascibility, and asylum treatment was at length rendered necessary. He was admitted to the New York City Asylum for the Insane March 17, 1873. A week previous he had gone to church, but soon returned, saying he had been followed by "droves" of dogs. He was a tall, powerful, good-humored man, and, though he asserted he would not commit suicide, he had cut off the tip of his ear in an attempt of this kind. He was somewhat subdued in manner, and had had hallucinations of sight and hearing. The day previous to admission, he was affected with a spasm of the muscles of the extremities. Five days after admission, he manifested delusion that he had committed a great crime, and refused food, but said: "This is not a penance for the crime." He required artificial feeding for three days, took food voluntarily on the fourth, and again refused it on the fifth day. A period of excitement then occurred, and he became the subject of hallucinations, differing from those he had on admission. After treatment a short time with opium and hyoscyamus, he grew quiet and took food voluntarily, but very suspiciously. In about a week after, a spasm of the muscles of the neck, followed by slight unconsciousness and slumber, occurred, the pupils dilating widely, and so remaining for a few days. Two weeks after, he had very sluggish movements of the lower extremities, bearing a suspicious resemblance to functional paraplegia, but this was really an incomplete cataleptoid condition, involving also the muscles of the neck and upper extremities.

The patient opened his mouth, and performed other simple actions of that nature; these, however, were not

ideational, but sensory-motor acts, as his attention to the subject was nil, and he was in a peculiar emotional state. That all the mental faculties were not in abeyance, was shown by the fact that he involuntarily raised his hands in an attitude of supplication, or as an acknowledgment of a favor just received. His pupils responded to light, and the organic functions were performed as usual. This condition continued for three days with very little change, except that when asked to perform a simple action the request would be obeyed, and the action continued indefinitely in an automatic way.

Five days after the beginning of the condition just mentioned, the patient had a rapid, feeble pulse, the beats of which ran into each other and did not correspond with the heart's action, which, though rapid, was otherwise normal. His eyelids and lower extremities soon became œdematous, and the cataleptoid condition disappeared. The heart's action grew more irregular, *the first sound alone being audible*, and accompanied with a *loud, blowing murmur* heard at the base. Pulse 132 and more rapid in the neck than at the wrist; respirations were increased, the lungs and temperature being normal. The heart's action soon returned to its normal condition, and the murmur disappeared. The treatment was directed to the alimentary canal only.

The patient then became entirely unconscious as to his surroundings, though taking food and performing other actions involving only the organic functions normally, and so continued for about a week. He then began to have tonic contraction of the muscular system, followed by lessening of the œdema, which finally disappeared. The cataleptoid condition then returned, and was accompanied by considerable waxy mobility. Two days after, his muscles were extremely rigid, and he remained apparently unconscious for some time. One morning he suddenly spoke, and on being asked his reason for not speaking before, said: "They told me not to." On being asked who told him not to, replied "God and others," and began to weep. The following day he had a return of the cataleptoid condition, in-

which he remained for some time. These alternations continued for three months, when he became suddenly violent, tore off a bar from the window, and tried to make his escape. The excitement continued three days, the patient then passing again into the cataleptoid condition, and very formal in conversation. This manner of speaking and acting continued for three months. He then had another cataleptoid relapse, succeeded by an attack of melancholia attonita. Then followed a condition during which his pupils at first contracted and then dilated, his left arm contracted firmly, and from it a quivering motion extended over the left side, and gradually involved the entire body. The irregularities of circulation formerly observed once more appeared, and as before went away without special treatment. Melancholia attonita became the predominant condition, accompanied, however, by increased susceptibility to external influences. This remained four months, and was followed by a cataleptoid condition with much waxy mobility. While in this state he was found to be developing phthisis. The disease ran a rapid, somewhat irregular course, terminating life, twenty-six months after his admission to the institution.

The second case is as follows: W. H. G., aged twenty-six; colored, laborer, married, intemperate, and syphilitic. Mother had been insane, but recovered. The patient one day while at work fell down suddenly, and his face and arms began to twich; from this he soon recovered, but in two months became much depressed, and was placed in the City Lunatic Asylum, where he soon became maniacal and violent, which condition was followed by a period of depression with hallucinations. He suddenly refused to eat, and soon after passed into a cataleptoid condition, from which he emerged one morning; said he "was equal to any white man," and spoke very precisely. He was afterwards taken out of the asylum by his wife, and two months thereafter was readmitted, and after having remained two months was discharged improved. He was readmitted during 1874, then in a condition of melancholia attonita, out of which he gradually passed. When speaking he always

observed great precision, and if he supposed the expression used was not correct, would alter it until he found one that might with propriety be substituted for it. He remained in this condition till July of that year, and was again discharged. He was readmitted March, 1875. Held his head up in a very consequential way, and prefaced every reply to a question by the phrase, "I-do-not-doubt-but-what." "What is your name?" "I-do-not-doubt-but-what-it-is-William-Henry-G." How old are you? "I-do-not-doubt-but-what-I-was-born-in-the-year-1838,-so-my-mother-said." Where were you born? "I-do-not-doubt-but-that-I-was-born-in-some-part-of-the-world." What part? "I-do-not-doubt-but-what-I-do-not-know-what-part." His memory was somewhat deficient but not materially so, as he remembered when he was there before, that he went out on a furlough, and the physician's name. He was well-built and comparatively strong, and while speaking wrinkled his face very much; this was somewhat of a sensori-motor act, and under the stimulus of some emotion, at variance with his "verbigeration," disappeared. Patient retained his peculiar manner of speaking and acting, but grew less inclined to walk about, would remain for hours in an upright position, staring straight ahead at vacancy. He manifested moderate erotic desires.

My own case, the report and treatment of which are biased by the view that the motor symptoms were tetanoid, is as follows:

F. B., a German, aged thirty, married, shoemaker, was admitted into Cook County Hospital, May, 1880. The patient arrived in United States nine days ago; habits, moral and temperate. No trace of venereal disease; was never ill until the present attack. On arriving in America, not obtaining work at once, he became despondent and melancholic. Three days ago found him incoherent, excitable. He gradually grew worse until he had been in the hospital some six hours, during which time he had not slept any. The day previous to admission he began to have spasms and paroxysms at intervals.

On Admission.—While the hospital employees were at the wagon to receive him, he appeared rational and quiet, when suddenly his body became rigid; opisthotonos and episthotonos, also; he began to cry and bark like a coyote. These symptoms continued at short intervals, then would relax into somewhat quiet and

atonic spasms of respiration, when he would fill the intervals between the opisthotonos with cries and barks in the above-mentioned manner, sometimes changing his cries into a very high-pitched key and occasionally a rapid succession of snorting respiration (160 per min.); pupils natural and sensible to light; P. 128°; T. 102 (in rectum, he having trismus); resp. very fluctuating. On questioning his friends they said he was able to walk six hours before admission, and that during his illness he had not had a chill. May 4th.—*Treatment.*—At 5:50 P.M., he was given ten minims of tincture of physostigma every half hour hypodermically, also chloral hydrate, gr. v., administered in the same manner, but as often as found to be needed; and during the succeeding hour he had been given gr. xv. chloral hydrate and 3 ss. tincture physostigma; still boisterous. At 6:55, administered gr. v. chloral and ℥. x. tinct. physostigma. At 7:10.—P. 132, spasms of opisthotonos lasting forty seconds at intervals of one minute; spasms 25 seconds, intervals 25 seconds. At 7:15—administered gr. v. chloral and ℥. x. tr. physostigma, and repeated at 7:35. At 8:30—T. 102° (rectum-trismus) gr. v. chloral. At 9—P. 108; resp. 20; slept a few moments; no opisthotonos; lies on his back quietly. At 9:30—P. 106; answers yes to a question, the first sensible word; just had 15 minutes natural sleep. 10—He got up from his bed, walked some 20 feet towards the door, when he turned his head half way to the right; trismus as well as rigidity of the whole body took place; after remaining in that position for 40 seconds he was removed to bed, by main strength, and several spasms of marked opisthotonos followed in quick succession; he was given gr. v. chloral. 10:30—Makes night hideous by his howling; gr. v. chloral. 10:45—P. 136; T. 103½° (rectum); perspires profusely; spasms every other minute; gr. v. chloral; howling. 11:15—gr. v. chloral. 11:30—P. 102; pupils contracted; quiet; rests; pupils slightly dilated; drank cup of water. 11:44—Drank another cup of water; stood erect and micturated; urine high-colored; drank more water.

May 5th.—12:40 A.M.—P. 100; resp. 26; has been sleeping quietly for past 20 minutes; pupils small and respondent to light; lifting the lids does not wake him; sleeping.

Temp. in axilla, 100; sleeping well; no chloral since 11:15.

At 1:10 A.M.—He now wakes up with a "Ha! ha! ha!" and asks for a cup of water. Throws his head from side to side; body quiet, hands and arms straight by his side, and muscles rigid. No opisthotonos. Snorts and howls. Rapid resp. Now holds his head still and rolls his eyes. Injected chloral hyd. gr. xi.–v. in one arm, and remainder in the other. At 4:30 A.M.—Slight opisthotonos. 4:35.—Marked opisthotonos. 4:40.—Quiet, slight trismus, sleeps a little every two or three minutes. Turns his head quickly right to left. Snorts occasionally. Pupils respond markedly to light. At 4:55 A.M.—Cries and whines faintly, sleeps. At 4:57 A.M.—Marked opisthotonos; chloral, gr. 7½. At 5:05 A.M.—Same condition, same treatment. At 5:08 A.M.—Gave

him ʒ i. kali brom. Arms have been rigid at his sides and motionless for hours. At 5:15 A.M.—Opisthotonos as frequent, but not as marked. Trismus, howling, and clonic spasms of face continue. At 5:25 A.M.—Rectal injection of chloral hyd. ʒ ss., kali brom. ℈iv., aquæ ℥iiss. Still howls, opisthotonos less frequent and less marked. At 5:55 A.M.—Still howls. Pupils equally dilated and respondent to light. Places his left hand on his chest, but immediately replaces it at his side, this being the only movement of the extremities for many hours. Occasional pleurosthotonos. At 6 A.M.—A large portion of the last rectal injection has just passed into the bed. Now holds his arms upwards, and gradually, very slowly, raises his body, until he is in a sitting posture; then returns in the same manner. This he repeats. Next holds his hands together, directly upwards, with eyes intently fixed upon the ceiling, as though in prayer; remains in this attitude for four minutes, then sits up as before. Now kicks the bed-clothes off, lies naked three minutes, turns on his side, falls asleep.

At 6:20 A.M.—P. 112. T. at axilla 100½°. Resp. 32. Still sleeping. Wakes up and asks for water.

At 6:30 A.M. injected per rectum, kal. bro., ʒ i., aquæ, ℥ iii., and applied pad and bandage to retain the injection. Slight opisthotonos; howls, cries, and laughs a little; is becoming more quiet; moves hand slightly; mutters and whines. At 7 A.M., sleeps quietly now. At 8:05 A.M., injected gr. 7½ chloral, hypodermically. At 8:50 A.M., trismus continues; injected kali bromid. ʒ i. in rectum and retained as before. At 9:05, more quiet; sleeps at intervals. At 9:15, is now sleeping soundly. At 9:40, P. 112, T. 99½°, resp. 28; moves in his sleep; fearing opisthotonos, injected ʒ ss. chloral; gets up out of bed and walks about his room with staggering gait; micturated; urine normal but high-colored: returns to bed; no trismus; opisthotonos mild; rests quietly and breathes easy. At 10:50 A.M., P. 98, skin cool; sleeps. At 1:10 P.M., just awoke; moves about quietly in bed. At 6:40 P.M., has been moving about in bed and room since 1:30 P. M., and howling in the meantime. At 6:55 P. M., injected gr. 7½ chloral, hypodermically; no opisthotonos; clonic movements of face; trismus; snorts, laughs, groans, and barks. At 11:35 P.M., turns his eyes from side to side, and will imitate any sound he hears.

May 6th.—At 1 A.M., just awoke; quite restless; had been sleeping a few minutes; gave gr. 7½ chloral as before. At 2 A.M., has talked for the past hour; breathing natural, then irregularly for about ten minutes, and finally goes to sleep in two minutes after an injection of gr. 7½ chloral. At 5 A.M., has slept and rested well for nearly three hours; he now wakens, grating his teeth, howling, snorting, and crying; gave gr. 7½ chloral hypodermically. At 7:30 A.M., injection repeated. At 2 P.M., has drank little and eaten nothing for the past two days; gave O ss. milk per stomach-pump; owing to trismus find great difficulty in

getting tube of pump into the mouth. At 7 A.M., P. 124, T. per rectum 103°, resp. 32 ; gave O i. milk in same manner as before ; falls asleep normally. At 10:40 A.M., gave O ii. milk in same manner as before ; rests well.

May 7th.—At 3 A.M., P. 116, T. per rectum 102°, resp. 28 ; he seems now to rest quietly but wakeful ; groans a little. At 3:30 P.M., P. 132, T. 102°, resp. 20 ; gave him ℥ ii. milk per rectum, also O ii. by stomach pump ; rests well.

May 8th.—At 8 A.M., pulse 104, temp. 101° ; shakes his head from side to side ; nods sometimes ; trismus ; talks if any one is near him. At 3 P.M., P. 104, T. 99°, resp. 20. At 7 P.M. T. 100° ; rational ; talks German ; quiet ; says that he wants water, then asks for milk, of which he drank one pint, but vomited some of it.

May 9th.—Does not talk any more ; gets up and defecates ; utters unarticulated sounds ; T. 100° ; whistles at times ; has cataleptoid symptoms ; he will retain his extremities in any position they are placed for two to three hours ; he will then get upon his knees, and with clasped hands for two to three minutes look upwards as if in prayer.

May 10th.—Is in the same condition.

May 11th.—About 2 A.M., P. 110, T. 99.5° ; walks about his room ; sits at table to eat, covers his face with his hands, and cries frequently ; goes to bed and howls ; is rational at short intervals.

For the next three days he remains in the same condition.

May 15th.—Patient taken home by his friends ; eats well ; has more strength. He now claims that he is a sailor. Departs peacefully.

May 19th.—He is brought back to the hospital on a bed by his friends. They put him on his feet, thrust him into the corridor (against the warden's orders), and then drove away. He now assumes the rôle of a soldier—standing perfectly erect, with hands at his sides and eyes front. On the motion being made for him to follow his friends and their wagon, he marches after them, and when they motion him back, he stops short, turns about face, as if he were on dress parade. These actions he repeats several times. Finally he was taken to the examining room, but resisted all attempts at examination ; would not allow any physician to approach him. Ultimately he had to be held by force, howling loud and long about the same as when he left the hospital, but weaker. P. 108, T. 99.4°, resp. 20. Was placed in his old quarters ; occupied most of his time in prayer ; at bedtime he threw the bedding about his room ; prays most of the time in German ; but, although he cannot speak English, he sometimes repeats the Lord's Prayer in that language plainly ; slept on the wire mattress all night ; in the morning he ate a good breakfast of oatmeal and milk.

May 20th.—He was taken to the insane court, and at court hour was with difficulty removed from the wagon, struggling much

at times, then making his body rigid. He was eventually committed to the Cook County Hospital for Insane, where he died about a year after; but there is no history of his condition while there.

Ætiology.—Kiernan [12] is of opinion that the characteristic pathological condition is an inertia of the vaso-motor centres, whose consecutive injurious effects were concentrated on the parts lying at the depths of and around the fissure of Sylvius, and that any influence tending to cause such inertia will produce katatonia. He is also of opinion that scrofulous conditions are often associated with katatonia. Clevenger [14] expresses the opinion that "katatonia seems allied ætiologically to some rheumatoid disease though its origin may be in nerves or blood." One case cited by Kiernan [13] under insanity due to rheumatism at first sight seems to bear out this opinion.

CASE 1.—J. G. Ger., æt. fifty, was admitted to the N. Y. City Asylum in a violent excited condition. The patient's wife gave the following history: The patient's father and grandfather died during an epileptic attack, and the patient's eldest brother is an epileptic. The patient has been perfectly well up to three weeks before admission, when he was attacked by acute articular rheumatism. The swelling of the joints was at times extreme, but after a month's duration suddenly disappeared, to be followed by a change to the mental condition in which the patient was admitted. The patient continued excited and violent, the violence being rather of the nature of melancholic frenzy. There were marked hallucinations present of a very distressing character. The patient continued excited for about three weeks after admission, when he suddenly passed into a cataleptoid condition with great waxy flexibility. In this state he remained for three years, when his pupils became unequal, his tongue tremulous, and an expression of content pervaded his face. He did not, however, speak until about three months had elapsed, when he talked loudly about his wealth in Germany; his speech was hesitant, and he had a great tendency to omit words. He passed through the usual stages of parei dementia, dying a year after the appearance of the paretic symptoms. No autopsy was obtainable.

This case is far from being a typical one of katatonia, nor is it cited by Kiernan as such, and while it shows that rheumatism, or the nervous condition to which rheumatism is due, may produce katatonic symptoms, the usual outcome of such cases is in other directions, and does not

demonstrate any analogy, nor more than the fact that rheumatism may cause vaso-motor inertia. Careful analysis of the cases already reported by the observers cited tends to bear out Kiernan's opinion.

Treatment.—Kiernan[12] says: "The medical treatment should be in a great measure regulated by the symptoms and should be of a tonic character, as the katatoniac is always more or less debilitated. The motor disturbance points to the use of conium. Alcoholic stimulants have had at times what could be nothing less than a food value, and have aided in sustaining the diminishing vitality of the patient. Stimulant enemata have been occasionally of service, and frequently prevented the return of a cataleptoid condition. The vaso-motor anomalies seem to indicate the use of nitrite of amyl and glonoine. He is satisfied that amyl nitrite is of value. Ten cases have certainly improved under its use, and it has caused a pleasurable feeling in all cases of katatonia where it has been given. Moral treatment, of course, in a great measure, revolves itself into the consideration of the question of asylum treatment. This is of advantage, as it affords a means of isolation from friends, always the most disturbing influence in treatment. Change of scene, and travel, under charge of a sensible educated man, not a pedant, would benefit many, as it would enlarge the patient's ideas and stimulate him to a healthy tone of mind—in short, stir him up. If the case be a boy, and he has a doting, foolish mother, removal from her should be the first step in the treatment, as her sympathy would undo all otherwise beneficial measures; a remark that applies with equal, if not greater, force in the case of a wife and husband. Balls and entertainments of a purely sensuous nature should be avoided, and all things of an intellectually stimulating nature brought as much as possible in contact with the patient. Faradization of the muscles of the chest, as a prophylactic against tubercle, is one means of treating probable somatic complications to be recommended. The general treatment by tonics, etc., is of course indicated in this and all other atonic physical conditions occurring during an attack of insanity. The preferable method of arti-

ficial feeding often required in cases of katatonia, is by means of a Davidson's syringe, the use of which is unattended with the danger that accompanies the use of the elastic but stiff tube of a stomach-pump, or the misadventures that follow the clumsy funnel method of feeding."

Spitzka expresses very similar opinions as to treatment. Hammond concurs in Kiernan's view of the pathology and hence in the treatment, but lays great stress on the use of sodium bromide.

Frequency.—Kiernan [11] has found that two per cent. of the insane admitted to the New York City Asylum for the Insane were victims of this psychosis. Clevenger [16] found that one and a half per cent. of the patients under treatment at the Cook County Hospital for the Insane were katatoniacs.

Medico-Legal Relations.—As has been pointed out by Kiernan: [12] "From the irregularity of the symptoms, which set at defiance the dicta of the forensic alienist, it would seem as if the disease could be easily feigned. Apart, however, from the improbability of a criminal being so keen an observer as to attempt feigning so complicated an affection, one symptom could scarcely be feigned with even the slightest probability of success, namely: the cataleptoid condition. The failure in the simulation of this symptom, with a close examination of his antecedent history, would soon detect any attempt of this kind. The crimes that a katatoniac would be likely to commit are murder, arson, and rape—the murder in obedience to an hallucination, the arson for a similar cause, while the rape would be an expression of his excited erotic condition. If these crimes, however, were committed during a remission, the patient should be held responsible, as he would, for the time being, be capable of acting logically on any conclusion arrived at in a logical manner. An instance where katatonia has been brought under cognizance of law occurred in a fanatical religious organization in Germany. Two ministers of this organization believed they had received, during a cataleptoid condition, a command from God to kill a certain man and raise him from the dead. The former they succeeded in doing, but in the latter they failed. In this case, which illustrates the circumstances

under which crime might be committed by a katatoniac, the accused were declared irresponsible. Any person, however, who has been acquitted on these grounds should be immediately sequestrated for the safety of the public."

Pathology and Pathological Anatomy.—I have already given Kiernan's view of the pathology of the disease, which is that adopted by the majority of authorities. Meynert [16] has said: "Katatonia is characterized by a series of fluxionary excitations, toned down by co-existent cerebral pressure, microscopic exudations, ventricular dropsy, and (perhaps) premature ossification of the sutures. From these would result forced and theatrical activities on the part of the patient. The convulsive state indicates the control of the irritative factors; the cataleptoid condition, the triumph of the depressing factors. The ideas of grandeur following close upon stupor, are the results of ideas previously caused by fluxionary conditions."

BIBLIOGRAPHY.

1 *Klinische Abhandlungen über die Psychischen Krankheiten*, Heft I.
2 *American Journal of Insanity*, July, 1877.
3 *Allgemeine Zeitschrift für Psych.*, 1877.
4 *Samml. Klin. Vorträge*, 1877.
5 Handbuch.
6 Lehrbuch.
7 *New York Medical Journal*, May 5, 1883.
8 Insanity.
9 *Amer. Jour. of Neur. and Psych.*, vol. iii.
10 *Chicago Medical Journal and Examiner*, 1884.
11 *Alienist and Neurologist*, October, 1882.
12 *Detroit Lancet*, February, 1884.
13 JOURNAL OF NERVOUS AND MENTAL DISEASE, 1881,
14 *Chicago Medical Journal*, August, 1884.
15 JOURNAL OF NERVOUS AND MENTAL DISEASE, Oct., 1885.
16 *Alienist and Neurologist*, October, 1882.
17 Pepper's System of Medicine, vol. v.

Clinical Cases.

REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.*

Prepared under the direction of PHILIP LEIDY, M.D., Physician-in-Chief, and CHARLES K. MILLS, M.D., Consulting Physician.

We design from time to time to furnish notes of the more interesting cases of insanity occurring in the Insane Department of the Philadelphia Hospital. It is our purpose not only to select the striking cases, but also to study some cases of all types in detail; for while the reporting of clinical cases has been carried almost to an extreme in some departments of medicine, comparatively little work of this kind has been done in the field of psychiatry.

CASE I.—*Paretic Dementia.*

Unsystematized delusions of grandeur of extraordinary character, with inequality of the pupils, and general physical depression, stamp the following case as one of paretic dementia, although there is a curious absence of some of the most common and decided symptoms of this affection, such as staggering speech, tremor, paresis, and apoplectiform or epileptiform attacks.

B———, admitted March 30, 1886; white, married, age fifty-seven, hotel keeper, common-school education.

His height is 5 feet 9 inches; weight, 138 lbs. He is slender and poorly nourished. His head is well shaped and symmetrical. His expression varies somewhat with the mood he is in, but one of sadness predominates, even when he is talking excitedly of his great wealth, etc. His pupils are variable at times; the left pupil is noticeably contracted, and will not respond to light. His sight

* Notes of several of these cases were carefully prepared by Dr. Geo. M. Wells, formerly resident physician to the Insane Department of the Philadelphia Hospital.

is somewhat impaired, but he is presbyopic, having used spectacles several years. Hearing appears to be normal. The respiratory murmur is good. No lesion of the heart can be discovered ; his pulse is rapid but strong, 90 per minute. His blood-vessels are atheromatous. His tongue is pale ; appetite capricious.

The urine is amber-colored, acid in reaction, sp. pr. 1,026, slight turbidity, but no sediment after standing twenty-four hours. Microscopical examination reveals amorphous urates, a few uric-acid crystals, some oxalate-of-lime crystals, and epithelial cells. No albumen can be detected by heat or nitric acid.

So far as is known there is no insanity in the family.

For several weeks, perhaps months, prior to March 27, 1886, the patient had been noticed to act strangely. He was naturally high-tempered, irritable at times, and easily excited, but at the same time was exceedingly kind-hearted. During the past year he had several times threatened to kill himself, but had never made any attempt to do so. For several weeks prior to admission he would have spells of great depression, would worry greatly about business matters, and became exceedingly irritable. He would frequently hire a coupé and, after being driven from place to place, would be unable to pay, and would then insist that the carriage was his own and that he need not pay for it. This conduct caused his arrest and confinement in a station-house, where he was examined, pronounced to be insane, and sent to the hospital.

He was a moderate drinker. For about six years past it was reported he had been especially interested in a certain young woman, and since his admission she has been constantly uppermost in his mind. He is constantly calling her to come to him, wanting to send for her, imagining she is in the asylum, in the next room, etc. He seldom speaks of his wife, although she visited him soon after admission. He has delusions of grandeur —of wealth and power. He has also hallucinations of sight and hearing, and at times of taste.

May 3d (five weeks after admission).—His mental condition is very much the same as when admitted. He has lost flesh noticeably. Several days ago, when attempting to scale the fence, he fell and severely sprained his left ankle, and probably also produced a slight fracture of the external malleolus. A plaster dressing was applied, to which he objected vigorously, saying he could cure himself, as he was the Holy Ghost and knew all about such things.

May 10th.—The swelling of the ankle is greatly reduced. Restraint is necessary to keep him in bed, and to keep the dressing on. He has delusions of great wealth ; says he owns thousands of diamond mines, and that he has paved the streets with them seventeen feet thick. He has been telephoning to the young woman before referred to to come to him. He will call aloud making a request, answer himself *sotto voce*, and then repeat the answer aloud.

May 12th.—When asked about his wealth this morning, his re-

ply and further talk was as follows : "Yes, I own millions, billions, trillions, a million thousand billions of them ; why, the streets are paved with them, diamonds seventeen feet thick. * * * I 'll give you a set of them thirteen pair of rings. Oh, I 've got them all, made them ; why, G—d d—n it, I made every thing, I am Jesus Christ." On being told that he had said he was the Holy Ghost, he replied : "So I am the Holy Ghost, G—d d—n you, there are seven of them and I made them all ! Call S—— ; telephone to her to send my clothes. Yes, I own the whole world. It is full of giants seventeen feet high. Now grow forty feet ; if you don't I 'll kill you."

May 16th.—"Oh, I own them all, I own every thing. I have billions, millions, trillions, billions. I can go everywhere. Telegraph to Jesus Christ to come here ; I want him ; I am married to his sister. I have two of the prettiest daughters ever you saw, but you can't have them. I want them myself. I know you. I am going to marry them." * * * "I made every thing,—sun, moon, and stars. I am going to make a new sun, so there wont be any more night." On being told to make two, he said. "No, G—d d—n it, one 's enough. If you don't shut up I wont make any. * * * Now grow forty feet, d—n you. Begin at once."

May 18th.—He has been telegraphing to Wanamaker for new suits of clothes, and also to Jesus Christ, each time saying that his orders would be carried out, or answering *sotto voce*, and then repeating the words aloud. He says he owns a million billion of elephants, all eighteen feet high, and that they can move faster than the devil. He says that he is President, but that Cleveland is acting for him. Mayor Smith is also acting in his stead.

May 24th.—He has been ballooning and sailing in ships all made of gold, making a trip with Jesus Christ.

June 3d.—He is very quiet, not inclined to talk. He says it is all a damned lie ; he does not own any thing ; even his clothes have been stolen from him, but that he will fix the thief. Physically he has grown much weaker and thinner. His appetite is very poor. Milk and eggs are about the only kind of food he will take. He is also receiving whiskey.

June 18th.—He has been comparatively quiet for two or three days, and is inclined to sleep. He takes his food better, however. He is still destructive at times ; he will tear his bedclothes into shreds, also his clothing.

June 28th.—He says that he went through the North and the South pole. He wrote the first Bible that was ever printed, but it was the worst G—d d—ned job he ever did. He has the finest stock in the world. He makes the first Mexican horse in the world. He never told a lie in the world, and never would. The three best dictionaries in the world are Webster's, Worcester's, and another one whose name he does not remember,—but that he made them all.

His condition changed but little from day to day.

August 10th.—Since the last record he has had two attacks of almost complete collapse, lasting some hours, from both of which

he emerged under the use of revulsives to the chest and extremities, and stimulants internally and hypodermically. After the attacks his mental condition was perceptibly improved for at least twenty-four hours, being able to remember things and people he could not before, and speaking more rationally. His delusions are now those of grandeur interspersed with others of poisoning. He says that he is worth all the money in the world, and owns all the ships; that he made the first man; and that he wrote the first Bible when he was only three years of age. Has attacks of wrath, usually from being thwarted in some purpose, or from the presence of a man he dislikes. His delusions of being poisoned are always present, but he thinks that milk counteracts the poison. When he is in a happy frame of mind he thinks the poison is for the time counteracted. He is excitable, but not as aggressive in the expression of his delusions, which, however, are still exalted.

Both pupils are somewhat contracted, and the left is distinctly smaller than the right. He eats and drinks excessively.

CASE II.—*Paranoia.*

This case of *paranoia*, or *monomania with delusions*, furnishes a good contrast to the case just presented in the character of the delusions, which, although varying in their expression, are of a distinctly systematized character. Some secondary mental deterioration has evidently taken place.

P., admitted June 12, 1885; æt. forty-two, born in England, white, single, seamstress, Protestant, good education.

Her height is 4 feet 10 inches; weight, 86 lbs. She is slender and delicate-looking, but erect and well formed. Hair and eyes are dark. Her expression is pleasant, but rather sad.

Her general health is fairly good, and physical examination fails to reveal any organic disease of heart or lungs. Heart action, however, is irregular; pulse, 84. The respiratory murmur is rather feeble. Examination of urine reveals no renal disease. Appetite and digestion are poor, and bowels habitually costive.

Her father was insane and died with phthisis; her mother also died of phthisis. She has no relatives living.

No satisfactory previous history has been obtained. About thirteen years ago she began to have delusions of persecution. Upon admission to the hospital, and for a short time previous, the subject of spiritualism was her all-absorbing talk.

She is extremely clean in her habits, not destructive, and has never attempted suicide; is quiet and lady-like. She is said to have premeditatively attacked her sister at one time.

The following is one account given by the patient of herself:

"I was born in England, but brought to this country when too young to recollect any thing about the trip. I am antagonized by the house of Orange, and they are trying to set aside my birthright and to break the link in the chain of my house. As to the house to which I belong, I decline to state; that is something for

others to find out. Since I was born I have been gifted with second sight—I mean the power to see and recognize the spirits of the dead. They do not speak to me audibly, but their thoughts are communicated instantly, so that I know their conversation.

"I was poisoned with belladonna seven years ago at the Pennsylvania Hospital; it was first put into my soup. Several others were poisoned at the same time in order to get at me without suspicion. The poisoning was a Nihilistic plot of the house of Orange, who were my enemies. The poison was administered for two years in very small doses."

July 4th, she handed in a letter containing an account of the plot laid for her ruin at the Pennsylvania Hospital. This was given in the form of a dying confession from one of the parties interested in the plot. She also gave some account of the plans that were laid against her, and said that "some have reported me dead, and are following it up by scandal, coercion, and finally imprisonment in the Hospital for the Insane in Blockley, fearing that the truth should come out as to their doings."

July 7th.—A note is found signed "Emilie," and stating: "I am clairvoyant, so-called, in medicine, and will state some facts which ought to be known at the present time. It is the foundation of medical science. People are being made victims of vile practices in a way not recognized in the past."

"As Belladonna is used in the practice. I wish to give some points that are not generally understood. It is a very cold poison and affects the eye-sight; it injures permanently the eye-sight on the lower natural plane, introducing the eye more keenly into the internal nature, a plane we have no right to invade. This is a plane opened to society in distinction to a higher one, which is a gift of God. During its course it produces the symptoms, all its corresponding diseases, affecting corresponding parts of the body, with this difference that the symptoms are apparently real, but pass more quickly, and each may be followed by another one so quickly as to baffle the skill of the physicians under the present remedies. The parties may be very near death one hour and the next apparently well, so much so as to at last lose the entire sympathy of people; for they are apt to attribute it to a growing disposition to insanity, or to regard it as hysterical. Owing to the constant fluctuations of the animal spirits, they are one moment feeling well and cheerful, then suddenly almost prostrated without any apparent reason for it."

July 14, 1885.—Her mental and physical condition are unchanged. She still continues to write notes about herself in the same rambling style.

August 14th.—She is wilful and obstinate, thinks herself superior to the people about her. She is usually quiet and likes to be alone.

February 14, 1886.—She thinks an attempt is being made to poison her in the hospital. She says the hot air of the register is full of a poisonous dust, and that the hot water is full of poison.

She also believes that the room which she occupies was formerly occupied by a small-pox patient, and still contains the germs of the disease.

CASE III.—*Sexual Perversion.*

J. M., admitted March 17, 1886, is twenty-two years old, born in Philadelphia, white, single, comber of wool, common-school education.

His height 5 feet 7½ inches, weight 139 lbs., is of medium size rather poorly nourished, and has a simple, meaningless, and, at the same time, anxious expression.

Head 22 inches in circumference; hair light-brown, eyes blue, and lips very pale. The respiratory murmur is normal; the chest somewhat flattened. The heart is normal; no valvular lesion; pulse 90.

The urine is amber-colored, faint acid reaction. It shows no sediment after standing twenty-four hours, sp.gr. 1,021. No albumen is found by heat or nitric-acid tests. The microscope shows a few triple phosphates and oxalate-of-calcium crystals. The tongue is pale but clean; the bowels inclined towards constipation; appetite fair. There is no bodily injury excepting a few slight scars. Sight and hearing both appear to be normal. The genitalia are normal in every respect.

No insanity can be traced in the family. Father and paternal uncle were both hard drinkers, and most probably died from the effects of alcohol. A sister died at nine years of age of hydrocephalus.

He was effeminate from childhood, but this never attracted much attention. Prior to 1875 he would leave home and keep his family in ignorance of his whereabouts. He obtained employment, but lost it several times on account of inattentive and foolish actions. A week prior to his admission to the hospital, he returned home from one of his wanderings, and acted in a more than usually foolish manner. He talked strangely and excitedly, was arrested at his mother's request, and, after being examined, was pronounced insane and sent to the hospital.

For some time past he has talked constantly upon religious subjects, saying he was going to study for the priesthood, and finally imagined that he was a priest. He also became very much excited over the Knights of Labor question.

He has hallucinations of sight and hearing; he says he sees people strangely dressed, and that they are talking to him. He hears most beautiful music which is played for him. He has not shown any disposition to injure himself or others. On one occasion, however, he secured a knife and said "he was going to kill a nigger." He is clean in his habits, but very destructive to clothing. He will endeavor to take pieces of glass, nails, etc., into the ward, to deface the furniture, etc., by concealing them in his mouth.

He practises masturbation, but his great propensity is to fondle men, both with his hands and mouth. He has been detected[1] in this loathsome practice a number of times, both in the airing court and at night. This, it appears, was a common practice with him before his admission to the hospital. He is very bold with it, and has ventured to try to make engagements with visitors for such purposes.

He has repeatedly pierced his ears with pins, and twice with an eighth of an inch wire. When asked why he does such acts as defacing the walls, tearing his clothes, etc., he has said, "to let my upper ten out!" at the same time laughing and acting in a most silly manner. He says his name is Jane, and that he is a girl. He is fond of looking in the mirror. He talks in a squeaking, effeminate voice.

[1] His overtures appear to be most gladly received by some epileptics who are known to be masturbators.

Periscope.

ANATOMY (INCLUDING PATHOLOGICAL ANATOMY) OF THE NERVOUS SYSTEM.

The Course of Fibres in the Posterior Horn of the Human Spinal Cord, and the Relation of the Lesion of Tabes to it. HEINRICH LISSAUER. *Arch. für Psych.*, xvii., 377.

1. The normal posterior horn receives two classes of fibres, large and small, which are easily distinguished from each other, though the actual diameter of the fibres varies considerably in each class. The posterior horn can be separated into a number of regions : first, the region of the entrance of the posterior root ; second, the region of the substantia gelatinosa Rolandi ; and third, the region of the substantia spongiosa. (1) The region of entrance of the post. root is seen to contain bundles of nerve fibres running vertically through the cord, parallel to bundles in the posterior and lateral columns, and forming a sort of annectant column between these two, since it is not separated from them by any special septa. It is distinguishable from these columns, however, by the fact that it contains fine fibres only. It is traversed by the post. nerve roots. This column is named the peripheral zone of the post. horn. If any large fibres are found in this zone, they are on its edges, and belong really to other columns. The bundles of fibres in this zone are often separated from one another by septa of basis-substance, which is present in large amount in this region. They are also separated by the post. nerve roots, though in some specimens the bundles of fine fibres are seen to cross or wind around the nerve roots in their upward course. In the usual description of the post. nerve root, two divisions of fibres are mentioned, one of which enters directly the post. horn, the other turns toward the median line, and enters the post. lateral column. There is really a third division, which is made up exclusively of fine fibres. These turn upward immediately after entering the cord, and pass into this peripheral zone, which is thus seen to have its origin in the posterior nerve root. The fibres which make up this peripheral zone do not, however, pursue a long course upward. This is proven by the fact that the zone does not become larger as it advances toward the brain. The fibres which make it up terminate in the gelatinous substance,

but always at a level higher than that of their entrance. The peripheral zone is, therefore, formed by small fibres of the posterior nerve root, on their way up to the gelatinous substance.

2. The region of the gelatinous substance shows a peculiar spongy structure on its posterior edge, which must be distinguished from the other part. This consists of a confused mass of fine fibres passing in all directions, and mingled together so as to form a thick mesh. Into this mesh, three sets of fibres are seen to enter: (*a*) from the peripheral zone, (*b*) from the posterior nerve roots, (*c*) from the posterior column. From this mesh many fibres pass forward into the deeper gelatinous substance. What the relation is between the fibres entering and leaving this region is doubtful. It is possible that here the fine fibres terminate; if so, this region equals the central gray matter in importance. It is possible that the fibres simply traverse this region to end elsewhere. It is also possible that an independent set of fibres exists here, not related directly to the roots; a hypothesis which seems to be supported by the fact that in tabes there is here a region unaffected by the degeneration of the posterior root zone. The typical gelatinous substance lies in front of this spongy zone. It is made up of a coarse mesh of fibres, both large and small. The large fibres pass in bundles from the post. roots, either directly forward or in large curves to the deep region of the posterior horn. The small fibres, on the contrary, come chiefly from the spongy zone; a few, however, curve through the posterior root zone, and thus reach the gelatinous substance. The majority of the fibres in the gelatinous substance pass directly forward horizontally, but some pass in other directions, so that the mesh is very thick. Fibres may often be seen passing from the gelatinous substance into the lateral column; but the fact that these never degenerate in tabes proves that they are not continuous with the post. nerve roots. Compact longitudinal bundles in the gelatinous substance, when found, are simply aberrant bundles of the lateral or posterior columns.

3. The region of the substantia spongiosa lies between the gelatinous substance and the posterior gray horn. Its basis-substance is a mesh of very fine fibres. It is to be noticed that in this can be distinguished a posterior and an anterior division, separated by fine fibres, which pass longitudinally through the region. Into this region pass many fibres from the posterior roots, which turn upward in their course, and are known as the longitudinal bundles of the posterior horn. Bundles of fibres are also seen to pass forward and into the anterior horn of the cord, and into the anterior commissure. Lastly, in the basal part of the posterior horn, in a fine mesh of fibres, are seen the bundles entering from the posterior lateral column and from the lateral column, which pass toward the ant. and post. commissures.

These details of structure have been discovered by the aid of Weigert's new method of staining. The same method was applied to the study of tabetic cords. It was found that the peripheral zone

of fine fibres was always sclerotic in tabes, though not always among the first portions affected. The zone was shrunken, and its outline was very definitely distinguished from the lateral column. In some cases in an early stage it was distinguishable from the posterior column, being the part chiefly affected. This seems to warrant the conclusion of the author that it is to be regarded as a distinct column. Any constant relation between its sclerosis and the appearance of any definite symptom was not determined.

Attention is also called to the changes in the substantia spongiosa in tabes. It was found that in tabes many of the finer fibres in the meshwork of the posterior part of this region were degenerated, so as not to be visible by Weigert's stain. This result, however, is not new, since it is well known that this part of the posterior horn and its plexus of fibres are affected in this disease. The large root fibres, which pass directly into the horn, are affected late in tabes, and the progress of their sclerosis is slow. The root fibres, which curve through the posterior lateral column, degenerate at the same time as this column, and they are affected earlier than the mesh work of the substantia spongiosa, into which they pass.

Changes in the vesicular column of Clarke are constant, and appear early in the disease. The cells are not affected, but the plexus of fine fibres in which they lie show marked evidence of degeneration, and finally disappear. The fibres entering the column from the posterior roots also degenerate.

The author emphasizes the fact that the lesion in tabes is not simply a sclerosis of the posterior root zone, but that it is a complex lesion, involving in different degrees the large and the small fibres of the posterior roots, and also the plexus in the posterior horn and in the Clarke column. It is too early to assign to each of these its rôle in the production of the symptoms of tabes, but such a careful study of pathological facts as is here presented cannot but lead in the end to this result. M. A. S.

The Origin of the Corpus Callosum: A Contribution upon the Cerebral Commissures of the Vertebrata. By HENRY F. OSBORN, Ph. D., Professor of Comparative Anatomy, Princeton College. *Morphologische Jahrbuch*, July, 1886.

The value of this very complete article is attested by the fact of its publication in English in the chief German periodical of comparative anatomy. The author has had at his disposal the extensive material in the laboratories of Gudden and Kupffer in Munich, and his conclusions are drawn from a wide range of observations. After a critical review of existing views as to the origin of the cerebral commissures, and a clear statement of the facts observed in the large number of brains investigated, he reaches the following conclusions:—(1) In the brain of fishes, the olfactory lobes are united with each other by an olfactory commissure, which probably is homologous with that part of the pars olfactoria in the mammalia which supplies the olfactory lobes. Within this

are the inner olfactory tracts, which arise from nuclei of uncertain position, and crossing each other in front of the olfactory commissure, enter the olfactory nerves, forming an internal and posterior root. The cerebral lobes are united by one or more bundles of fibres, which constitute the commissura inter lobularis. (2) In the amphibians, the cerebral commissures lie behind the foramen of Monro. They consist of an upper and a lower bundle. The former is much the larger. It passes upwards and forwards behind and above the foramen, and gives the commissural supply of the dorso-medial portion of the hemispheres. It is the corpus callosum. The lower bundle is the anterior commissure. Its main division is the pars olfactoria, which supplies a part of the brain stem, the ventro-lateral portion of the hemispheres, and probably the olfactory lobes. Its lesser division, supplying the lower posterior portion of the hemispheres, is the pars temporalis. The inner olfactory tracts arise near the anterior commissure, cross each other, and pass into the olfactory lobes. (3) In the reptiles, the cerebral commissures lie below and slightly in front of the foramina of Monro. The larger upper bundle ascends in front of the foramen, and divides. The anterior division, or corpus callosum, supplies the whole of the inner mantle. The small posterior division supplies the inner fold of the hemispheres, and represents a portion of the fornix. The lower bundle is the anterior commissure. It consists of an upper tract, the pars olfactoria, the distribution of which is doubtful, and of a lower tract, the pars temporalis, which is distributed as in the mammalia. In connection with the corpus callosum is a bundle representing a portion of the fornix. (4) The position of the cerebral commissures in the birds is similar to that in the reptiles. The upper bundle is very small. It divides and is distributed as in the reptiles, but the division representing the corpus callosum is much smaller than that representing the fornix. The anterior commissure consists principally of the pars temporalis, the pars olfactoria being reduced or wanting. The corpus callosum is larger than the anterior commissure in the amphibia and reptiles, but in birds it is much smaller. These proportions are in relation to the thickness of the inner mantle. In the birds, accordingly, where the inner mantle has suffered the greatest reduction, the corpus callosum is a mere rudiment. In the other groups described, it is far from rudimentary. In comparison with what we observe even in the lowest mammals, the size of the cerebral commissures is diminutive in proportion to that of the hemispheres. M. A. S.

Descending Degeneration in the Brain and Cord Experimentally Produced, and its Relation to the Doctrine of Localization of Functions. L. BIANCHI. *Neurol. Centralbl.*, No. 17.

Following the method of Gudden, Bianchi has extirpated the motor zone of the cortex in new-born dogs, and has observed the

results after a lapse of one to two years. The symptoms produced in all cases were permanent changes in power in the limbs of the side opposite to the hemisphere operated on; temporary disturbance of sight; little or no alteration of tactile sensibility; and no particular change in the psychical functions. The disturbance of motion did not consist in ataxia, but in a true paresis, a loss of muscular power, with some contracture. The loss of voluntary motion was total, and automatic movements were also impaired. The disturbance of sight was of the character of hemianopsia, and was very much more marked just after the operation than later. It could not be determined whether it was a total or a psychical blindness. Microscopic examination of the brains showed the orginal lesion to have been limited to the gray cortical layers. A descending degenerative atrophy was traceable through the centrum semiovale, the internal capsule, the crus, the pons, and the medulla in the well-known course of the motor tract; and in the opposite lateral column of the cord in the pyramidal tract, and in one case in the column of Türck as well. The special interest of the experiments lies in the fact that a noticeable atrophy was found in the corpus callosum, where it reached the middle line; and also in the caudate and lenticular nuclei. These latter showed an increase of nuclei, and of neuroglia. The optic thalamus was not changed in size, but was rounder, probably because of the dilatation of the lateral ventricle caused by the atrophy of the caudate nucleus.

The conclusions reached from the experiments are as follows:

1. Since the fibres beneath the cortex, were not injured by the operation, the degeneration of the tract must have been due to the cortical lesion.

2. Since the degeneration started in the cortex, the cortex and the affected fibres must be considered as a functional unit.

3. The corpus striatum is intimately connected by a system of fibres with the motor zone, a fact urged by Meynert, but denied by Wernicke.

4. If the pramidal tract is motor, its centre of origin must also be motor. The results of the experiments opposed the theories of Munk and Schiff, that the motor cortex is an area of muscular sense, since there was no true ataxia.

5. The motor area of the cortex is not only a motor centre for the highest evolved movements, but is also the source whence all motions issue, since walking, running, jumping, are all imperfectly performed after its extirpation.

6. Dogs whose motor cortex only is removed, preserve the same instincts, the same spirit as normai dogs; are intelligent, faithful, playful; are well nourished; and can propagate their species, but their puppies are epileptic.

7. The recovery of automâtic movements in some degree is not to be ascribed to the action of the corpus striatum, since this is atrophic. It is rather to be considered as a function of other portions of the cortex, which thus take up and carry out the action

of the deficient part, but in an imperfect manner. The portion to which the author asigns this vicarious work, is the uninjured motor cortex of the opposite hemisphere. M. A. S.

The Degeneration Following the Partial and Total Extirpation of the Cerebellum. *Gaz. degli Ospitali*, Aug. 22, 1886, No. 67.—In this paper, which was presented to the *Societa Medico-fisica* of Florence by Dr. VITTORIA MARCHI, the following results were announced :

1st. After total extirpation of the cerebellum, there occurs a diffuse sclerosis, especially of the gray substance of the pons Varolii which surrounds the pyramid, and of the inferior olive ; of all the cerebellar pedunculi, including the direct cerebellar tract of Flechsig, without any lesions of the nuclei of origin of the cranial nerves.

2d. After extirpation of half of the cerebellum, there is degeneration of the elements mentioned above, especially in the part corresponding to the lesion, but also in parts of the other side. It was interesting to note that many of the degenerated fibres of the superior cerebellar peduncle, instead of crossing entirely over to the opposite side, took a direct course to the nucleus of Stilling of the same side without decussating.

3d. Following the extirpation of the middle lobe there took place a degeneration of the greater part of those fibres which constitute the direct cerebellar tract of Flechsig, and the small ventral fasciculus of the pons described by Löwenthal, which, in the restiform body, is associated with fasciculus of Flechsig in extending itself along the external portion of the lateral columns of the medulla. A few of the arciform fibres were degenerated, and many of those pertaining to the inferior convolutions of Reil, and many fibres of the pyramidal tracts.

4th. It was observed, after total or partial extirpation of the cerebellum, that the cranial nerves were subject to the degenerative process. More especially the third, fifth, sixth, seventh, eighth, tenth, and twelfth. The nuclei of origin and the fibres emanating from them seemed exempt from degeneration, the fibres not showing degeneration till their emergence in the vicinity of the pons.

The experiments from which these conclusions were deduced were performed upon six dogs and two monkeys. Of the three dogs in which the cerebellum was totally extirpated, one lived eight months after the operation, the second a year, and the third about two years. In the three cases of partial extirpation, the two dogs lived two months, and the monkey eleven months.

G. P.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

On Localization of Function in the Brain. By Dr. Von Gudden.—*Biolog. Centralbl.*, Nos. 18 and 19, 1886.

Dr. Gudden approached the problem from the anatomical side. His object was to trace from the periphery towards the centre the distribution of the nerve fibres. If the physiologist, taking away different parts of the cortex to observe the result, does not know the distribution of the fibre systems in the cerebrum, he is certain to fall into error. Phenomena, due to secondary injury of the underlying fibres, will be ascribed to the cortex. In short, physiology must have its verdict corroborated by anatomy before the final judgment can be given. Any result drawn from the physiological study of the brain must be abandoned if it is contradictory to any well-established anatomical fact.

If you destroy the eye of a young rabbit, the opposite optic tract, as far as it is developed, will atrophy, with the exception of the uncrossed bundle of fibres. This, in the rabbit, is so insignificant, that an animal possessing it alone in one eye is blind in that eye. Following the atrophy of the optic tract will be that of the primary subcortical centres—the external geniculate body (the centre for the pupillary reactions), and the upper layer of the anterior corpus quadrigeminum (the visual centre). From here, by means of the connecting fibres, the secondary cortical centre of vision should degenerate; but no difference, except that due to the shifting caused by the atrophy of the anterior corpus quadrigeminum, can be noticed in the cortex. Microscopic examination reveals no difference in the size or number of the cells, and simply shows that the part above the atrophied anterior corpus quadrigeminum, having more room to develop, has developed more. The effect of extirpating both eyes is practically to duplicate these appearances on the other side. Sections of the other sensory nerves promise equally definite results.

Special attention is given to the atrophy of the olfactory nerve. The glomerulus layer of the olfactory bulb is the primary smell centre, and the rest of the bulb is cerebral. Proof of this will be given elsewhere. Part of the anterior commissure serves exclusively to connect the olfactory lobes. The importance of the olfactory nerve consists in the fact that the manner of the connection of its centre with the cortex is clear. If you destroy the olfactory membrane of one nostril of a young rabbit, you will cause the atrophy of the glomerulus layer of the bulb. Or better, if you separate, by the insertion of a fine knife within the cranium, the olfactory bulb from the lobe and allow the animal to grow up, you will find (1) a line of loose connective tissue at the point of section (reunion of nervous tissue does not occur within the brain); (2) the olfactory tract gone; (3) the olfactory lobe to all appearances normal.[1] This fact of the preservation of the lobe when separ-

[1] A microscopic examination showed a slight alteration of the cells in one out of three specimens. It is suggested that this may have been due to a meningitis.

ated from its nerve, Dr. Gudden, remembering that comparative anatomy showed that the development of the lobe was proportional to the size of the nerve, explained as due to the connection with the opposite olfactory lobe through the anterior commissure. This view had to be abandoned when he succeeded in separating both bulbs from their lobes and yet found practically no alteration in the lobes. Upon the interdependence of the development of the olfactory nerve, bulb, and lobe on the one hand, and on the (nearly) normal development of the lobe in separation from its tract and atrophy of the bulb on the other hand, Dr. Gudden bases his view of the localization of function in the brain.

The same is true of motor nerves. The facialis, hypoglossus, etc., can be cut without causing a degeneration in the cortex.

If the entire hemisphere of the brain of a young rabbit, including the corpus striatum but not the optic thalamus, be removed and the animal allowed to grow up, no difference in the sight, hearing, or feeling of such animal is noticed. They behave perfeetly normally, and show no difference between the sensibility of the two sides. No anatomical basis for the unilateral control of the brain is as yet found. The main point to be noticed is that the sight centre (upper layer of anterior corpus quadrigeminum) was in no way altered, and the optic tracts and nerves were alike on both sides.[1]

Dr. Gudden succeeded in destroying the left parietal and occipital lobe of a four-weeks-old kitten without producing hemiopia (though the so-called visual centre in the occipital lobe was destroyed), and moreover both optic tracts were equally developed. More remarkable is the following case. The entire occipital and parietal lobes of *both hemispheres* were removed from four young rabbits. They developed apparently like normal animals, and as regards vision, they fled when a hand was waved at them from a great distance, jumped from heights, avoided all obstacles; in short, though without the slightest trace of either visual area, they saw and interpreted what they saw. In another series of rabbits the frontal lobes on both sides were removed and yet the animals saw, heard, felt, and moved as normal rabbits. If, however one makes a deep incision into the brain and removes much of the matter, many of the rabbits die; and the survivors, though they see, hear, and feel in a rudimentary way, yet show peculiarities, are sleepy, idiotic, puny, weak, etc.

As the primary sight centre does not atrophy when the occipital lobe is destroyed, so the subcortical centres of all sensory and motor nerves remain intact when they are separated from the cortex. If you cut off the olfactory lobe, the glomerulus layer remains intact, only the small part of the tract belonging to the lobe remains, and the commissural fibres of this part are gone.

[1] Dr. Gudden enters into a controversy as to how far this relation is true in the higher animals, in which he considers that the one negative case is conclusive, and that in those cases in which some atrophy does occur, it is secondarily caused.

Passing from these facts, gained with much labor and difficulty, to their interpretation, one must be still more careful. They certainly do not favor the view of exactly localized, circumscribed, functional, distinct areas ; nor does the histological character of the cortex favor such a view. The objection that results derived from the study of the brains of young animals are not entirely applicable to mature brains, is not without weight. But similar facts can be shown in fully developed animals, although half-grown ones, owing to their greater vitality and smaller liability to secondary atrophies, are preferable.[1] Dr. Gudden has the brains of two full-grown dogs, which show no atrophy of the optic tract, though the visual area was destroyed ; and no such operation can be used as evidence for making out a certain area to be the sight centre, unless the atrophy of the optic tract (if it occurs) can be shown not to be due to secondary causes. In the motor area, the difference between operating on a young and on a mature animal is marked ; the former can bear the removal of the frontal area without motor disturbance, while the latter suffers considerably, but even then the effects are not permanent. The anatomical effect is in rabbits a complete, in cats an incomplete, atrophy of the pyramidal tracts. A dog, both of whose motor areas were removed by Prof. Golty, was very awkward in its motions, but not paralyzed. A series of young rabbits similarly operated showed very serious motor disturbances, but even here, as the animals grew up, the effects gradually grew less and less. But all these experiments are only in their infancy, and it was the expressed intention of Dr. Gudden not to draw any but provisional conclusions until more evidence was collected.

Dr. Gudden does not believe in exact localization, but does not oppose all localization whatever. The anatomical relations on which this position is based are, by way of summary : (1) in the whole mammalian series there is a constant relation between the development of the olfactory lobe and that of the bulb and nerve. One must remember that the rabbit, both of whose olfactory lobes were separated from the bulbs, and yet the lobes developed normally, argues against the olfactory lobes as the exclusive centres of smell. At any rate this suggests a modification of the theory. (2) Removal of the frontal lobes, and of these alone, causes atrophy of the pyramidal tracts ; hence the frontal lobes have a special function. (3) The dependence of the lemniscus upon the parieto-occipital lobes, first shown by Gudden. (4) Some of the nuclei of the thalamus are independent of the cortex. (5) The medial posterior ganglion of the mammillary body is also dependent on the parieto-occipital region, but not on the frontal.

All that these facts show is that the division into motor and sensory functions of the cortex is justified. To go on with this study one must cut the subcortical fibres and trace the degeneration in the cortex. For this purpose, Dr. Gudden cut (through

[1] Dr. Gudden mentions some of the cautions to be observed in operating, etc., for which reference is made to the original paper.

the foramen opticum) the middle third (pyramidal tract) of the internal capsule. Subsequent examination showed that the attempt was almost entirely successful. The result was a degeneration of *large* motor cells only in the cortex. This suggests that the different cell layers of the cortex may have different functions.

In fine, enough has been said to show that much remains to be done to complete the doctrine of the localization of function in the brain. *First* anatomy and *then* physiology, was Dr. Gudden's watchword ; but if physiology first, then not without anatomy.

J. J.

THERAPEUTICS OF THE NERVOUS SYSTEM.

The Result of Nerve-Stretching for Facial Spasms Five Years after the Operation.—Mr. F. A. SOUTHAM, in a "note" in the *Lancet*, April 10, 1886, states that there has been no return of the spasm, though five years have elapsed. The case was previously published in the *Lancet* five months after the operation. The spasm had been of two years' duration. The operation was followed by complete paralysis of all the muscles supplied by the facial nerve. At the end of five months the paralysis had completely disappeared. S. refers to nineteen cases of this operation, collected by Tesas (*Wien. med. Wochenschr.*, Nos. 27 and 28, 1885, and this JOURNAL, Oct., 1885). In only two of these—viz., in one case, operated upon by Tesas himself, and in another by Navratil—in addition to the one above referred to, has the cure been permanent. In four cases there was considerable improvement, while in ten the operation failed to give more than a temporary relief ; in the remaining two cases the result was doubtful, as their subsequent history was not followed out. Inasmuch, however, as benefit has followed in seven out of seventeen cases, Tesas is of opinion that the operation should still be performed whenever the spasm is not due to intracranial lesions.

Antipyrin in Headache.—According to Dr. JOHN R. WHITE (*Med. Rec.*, Sept. 11, 1886) antipyrin "not only promptly relieves the symptoms of headache, whenever present, whether resulting from disordered digestion, disturbance of the menstrual functions, loss of sleep, undue mental effort, or even that associated with dreaded anæmia, but also possesses reliable prophylactic virtues against recurrent attacks of cranial neuralgia." Relief often follows a single dose of fifteen grains within half an hour. "A sense of drowsiness ordinarily supervenes, followed by a brief but sufficient slumber, and the patient awaking quite relieved from the distressing symptoms."

Urethane in the Treatment of Traumatic Tetanus.—Mr. W. T. JACKMAN publishes in the *Lancet*, June 12, 1886, a case

of traumatic tetanus, in which recovery was thought to be due to the use of urethane in conjunction with chloral.

Urethane in Insanity.—Drs. OTTO and KOENIG (*Therap. Gaz.*, July 15, 1886), as a result of a trial in a number of cases in the hospital for lunatics and epileptics in Dalldorf, did not find urethane as valuable as chloral or paraldehyde. Dr. Rottenbiller, on the other hand, thinks, as a result of his experience in the insane asylum at Budapest, that thirty to sixty grains will produce several hours' quiet sleep, without disagreeable after-effects. He also made a number of experiments with the subcutaneous employment of the drug, hoping to avoid the gastric disturbances apt to be produced by such large doses. In nine cases, two injections of four grains each produced six to eight hours' sleep, or one fourth the quantity needed by the mouth. The injections caused no local inflammation. He used a thirty-per-cent. watery solution.

Sciatica Cured by Puncture of the Sheath.—Sir JOSEPH FAYRES reports (*Practitioner*, April, 1886) a case of long-standing sciatica, in which tenderness and fulness existed over the sciatic, near its origin. On puncturing the swelling, about two drachms of clear serous fluid were drawn off. Instant relief and rapid recovery followed.

Nerve-Stretching for Anæsthesia of Leprosy.—Dr. E. DOWNER records (*Lancet*, June 5, 1886) the effect of nerve-stretching in thirty-two well-marked cases of leprosy, in which anæsthesia of the legs was an early and well-marked symptom. All the cases were benefited. In some the ulcers, which were present, healed completely. In one case, the longest under observation, sensation continued good for two years.

Another Local Anæsthetic.—The *Therapeutic Gazette* (July, 1886) directs attention to Dr. MAYS' experiments with theine. Dr. Mays had already shown experimentally the anæsthetic effect of brucine when applied locally, and afterwards that theine paralyzes the nerves of sensation in frogs; and that this impairment of sensibility from theine proceeds from the centre to the periphery, and not, like that of brucine, from the periphery to the centre. Subsequent investigations by the same author (*Med. News*, Dec. 12, 1885, and April 17, 1886) prove that it exerts the same influence on man. The clinical results obtained in thirty-nine cases, including neuralgia, lumbago, myalgia, rheumatic pains, etc., in which from one half to two and one half grains of the alkaloid were given subcutaneously at or near the central origin of the pain, were most favorable. Not only was immediate

relief given from pain, but a cure was effected in many cases. Localized anæsthesia below the seat of injection was produced, but it does not seem to produce any systemic disturbance even in maximum doses. The writer, in the *Therapeutic Gazette*, has also, on many occasions, witnessed patients suffering from the most excruciating neuralgic pains, who were entirely relieved in less than five minutes. MORTON PRINCE.

Hysterical Mania Cured with Hypnotism. CASTELLI E. SUMBROSO. *Lo Sperimentale*, 1885, fasc. ii. Quoted in *Revista Sperimentale*, vol. xii., 1886.

Young woman of twenty-six, with hereditary taint, had symptoms of melancholia, distaste for food, and tendency to suicide. She had hemianæsthesia of the left side, some hyperæsthetic zones, with neuro-muscular hyper-excitability. After some attempts they succeeded in hypnotizing her with the three states of lethargy, catalepsy, and somnambulism. The suggestions which the writers were able to provoke in this last state were numerous. It was found that, contrary to the observation of others, the paralysis provoked had not a constant character, and was not like that of central origin.

It was also found that those suggestions which Bernheim calls negative are not alone those of sight and hearing, but also of touch and pain. The case described is most interesting from a therapeutic standpoint, because with hypnotism by means of suggestion, the cure of the patient was obtained, including all her psychical trouble.

Cure of Mental Maladies by Hypodermic Injections of Hydrochlorate of Morphine in Large Doses. By VENTURI E. CANGER. *Il Manicomio*, fasc. i., ii., iii.

The authors have experimented in several forms of mental disease with the injection of hydrochlorate of morphine in large doses. The experiments were also made in those states of insanity which are accompanied with a tendency to congestion of the head, which, according to Voisin, who was the first to adopt the methodical injection of morphine in the insane, contraindicated this cure. The dose of morphine used was twenty centigr., which was gradually increased up to thirty-four centigr. in the day.

In twenty cases of simple mania twelve were healed and seven improved. In a single case there was no result.

Cure was obtained in eight cases of acute mania, in three of simple lipemania, and in one of lipemania with stupor. Only an amelioration of symptoms was obtained in four cases of acute mania, in two of lipemania with stupor, and in one of chronic mania. A single unsuccessful case of lipemania with stupor.

GRACE PECKHAM.

Basedow's Disease Cured by Operation.—HACK reports (*Deutsch. med. Wochenschr.*, June 24, 1886) a case of this disease which was unexpectedly, and to his astonishment, cured by cauterization of the inferior turbinated bones. The patient came for relief of the nasal symptoms from which she suffered. After cauterizing the right side of the nose the exophthalmus of that side disappeared, and similarly with the left side, though not at once or to the same degree. The other symptoms, including increased action and enlargement of the heart, enlargement of the thyroid gland, and even the impaired eyesight, improved. Hack explains the case on the theory of its being a reflex neurosis of the sympathetic.

Trephining for Meningeal Hemorrhage.—As the diagnosis of the seat of meningeal hemorrhage is very difficult, if not impossible, when there are no signs of external injury, and inasmuch as the extravasation occurs not only in the middle fossa of the skull, where it is most common, but also in the posterior and anterior fossæ, Krönlein (*Deut. Zeitschr. f. Chirurgie*, Bd. xxiii., Heft iii.) advises that the trephining be first performed—in doubtful cases—in the temporal region, at the point advised by Vogt, Beck, and others. If the hemorrhage be not found here, a second opening is to be made under the parietal eminence, the first for a hæmatoma anterior and media, and the latter for a hæmatoma posterior. This opinion is based on four cases of his own. The first case was a hæmatoma fronto-temporalis, right side, cured by trephining at the usual point. The second case was due to rupture of the left middle meningeal artery. There was a left-sided supra-dural hæmatoma, with right-sided hemiplegia. On account of enormous extension of the hemorrhage into the occipital fossa, it was necessary to trephine in both places. Recovery ensued, excepting that during convalescence paralysis of the fourth nerve came on and persisted. The third case was also trephined in two places, first in the usual spot, then three *ctm.* above and one *ctm.* behind. The hæmatoma was not found, because it was parieto-occipital. Death followed. In the fourth case, a diagnosis of probable intracranial hæmatoma of the right middle meningeal artery. It ended fatally, because it turned out to be a parieto-occipital hæmatoma, and was not reached by the operation, which was performed in the temporal region.—*Centralbl. f. Nervenheilk.*, Nov. 14, 1886. MORTON PRINCE.

VOL. XIII. DECEMBER, 1886. No. 12.

THE

Journal

OF

Nervous and Mental Disease.

Original Articles.

PRELIMINARY COMMUNICATION CONCERNING THE DECUSSATION OF THE PYRAMIDS.

By E. C. SPITZKA, M.D.

IN a cat, which was kept alive three months after the destruction of an entire cerebral hemisphere [the left] as well as the corresponding thalamus, and in whose brain, as the microscopical examination showed, there was complete destruction of the pyramid tract in the crus, pons, and oblongata, I was surprised to find that an indication of it apparently existed in the region of the decussation. While the right pyramid crosses over in the characteristic manner, breaking through the gray substance of the opposite side so as to decapitate the anterior horn, there is a slim but perfectly symmetrical bundle which intersects it and crosses in the same manner. Being less compact, however, it has not yet been possible for me to decide whether it enters the lateral column of the spinal cord or the gray matter which lies in the debatable ground between the nuclei of the posterior columns and the reticular formation. For a brief distance in its cephalad course it occupies the ventral face of the right half of the oblongata.

The bundle in question sends off a few detachments into the fibre bundles, which, running into the anterior horn, constitute a transition from the type of the massive decus-

sation of the true pyramid to the fascicular decussation of the anterior commissure. A portion, however, as above stated, exactly repeats the course of the true pyramid decussation. Individual fibres can be traced uninterruptedly from the neighborhood of the right "debatable" field across the median line, along the mesal side of the left half of the oblongata facing the interpyramidal sulcus to the ventral face of the oblongata. From the fact that absolutely no trace of the left pyramid can be found in the level of the trapezium or pons, it is to be inferred that cephalad it becomes a part of the lemniscus. This tract, although greatly atrophied, is represented on the left side.

In some measure this observation is supplementary to one made some years ago [1] in a case of secondary degeneration of the interolivary layer. I there found that the atrophic field of the interolivary tract was connected with degenerated fasciculi which it derived from the area of the anterior pyramid, whose veritable fibres were healthy, although the detachments from the interolivary layer were absolutely destroyed. Such detachments could be traced the entire length of the oblongata, even to the border of the pons.[2] It appears, then, that destruction of the pyramid tract does not destroy certain fibres participating in the true pyramidal decussation [cat], while destruction of the interolivary layer results in their death. The inference is obvious: that there is a system of fibres, intermediate in position, between the pyramid and interolivary tract, decussating with the former—apparently derived from the nuclei of the posterior columns,[3]—and running with the latter in its cephalic course. It was possibly the discovery of similar fibres that misled Meynert into the formation of his well-known but now abandoned view regarding the "sensory" and "motor" decussation of the pyramids.

Should it be determined that the preserved fasciculus crossing in the pyramidal decussation from the left oblon-

[1] "A Contribution to the Morbid Anatomy and Symptomatology of Pons Lesions." *American Journal of Neurology and Psychiatry*, vol. ii., No. 4.

[2] *Loc. cit.*, plate v.

[3] This remains doubtful, both in the negative observation in the human case, and the positive one made on the cat.

gata to the right cord became an ingredient of the crossed pyramid tract in the latter, it would prove a confirmation of Steinlechner-Gretschischnikoff's [1] opinion, that this tract contains fibres from centres situated caudad of the cerebral hemispheres. These observations would also tend to support the view first expressed by Rohon, and criticised by Starr, that the pyramids do not exclusively develop from the cerebrum downward. It may not justify the contrary statement, that a part of them develops from below upward, but they conclusively prove that the pyramid tracts contain fibre admixtures from other sources than those from which Flechsig exclusively derived them.

[1] "Ueber den Bau des Rückenmarkes bei Mikrocephalen." *Archiv. für Psychiatrie*, xvii., p. 689.

ATHETOSIS, ITS TREATMENT AND PATHOLOGY.*

By GRÆME M. HAMMOND, M.D.,

ASSOCIATE PROFESSOR OF DISEASES OF NERVOUS SYSTEM IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL.

SINCE the time when athetosis was first described by Dr. W. A. Hammond, in 1871, a large number of cases have been reported both in this country and in Europe.

The subject of spasm, both fixed and mobile, has been studied very thoroughly within the last few years, and the result has been to classify all localized spasmodic diseases due to cerebral lesions, as exemplified by athetosis, chorea, ataxia, tremor, and tonic spasm, as allied affections, all depending upon lesions occurring in the same projection-system. Dr. Hammond, in his original article, located the disease in the optic thalamus or corpus striatum. Oulmont, in his monograph on athetosis, attributes the disease to what he calls "athetoic fibres," which he supposes to exist in the posterior part of the internal capsule in front and outside of the sensory tract. Denange states that all forms of spasm have a common origin, and that they may be produced by a lesion in any part of the motor tract. In this view, Sharkey fully concurs, and I believe that opinion is generally held at the present time. That lesions of a certain part of the motor-tract will produce mobile spasms, is undoubtedly true, but that lesions of any part of the motor tract will produce tremor, ataxia, or athetosis, is not, to

* Read before the section on Neurology of the New York Academy of Medicine.

my mind, supported by fact. I shall endeavor to show in this paper that athetosis and its allied disorders are seldom the result of lesions in the motor tract, and that when the lesion does occur in the motor tract it is confined to a particular part of it.

Athetosis is usually, not invariably, preceded by hemiplegia or epileptic or epileptiform siezures. Cases have been reported by J. Coates, George Ross, Fletcher Beach, Landouzey, and others, in which the patients had neither suffered from paralysis nor epilepsy in any form. When there has been hemiplegia, the athetosis does not usually make its appearance for a considerable length of time. Sometimes several years elapse between the advent of the hemiplegia and the appearance of the athetosis.

In a large proportion of cases in which the athetosis has been preceded by hemiplegia, it is a noticeable fact, and one well worthy .of attention, that either the paralysis disappeared before the athetosis became apparent, or else the athetoic movements increased in direct proportion as the hemiplegia decreased. I think that this fact is of pathological interest, as I shall attempt to show later.

On the treatment of athetosis there is very little to be said. The very nature of the lesions which have been found to produce athetosis, precludes the possibility of their ever being removed by any remedial measures that we are able to resort to at the present time. But if we are unable to remove the cause of the disease, I think the following cases tend to show that the manifestation of the disease can be arrested for a longer or shorter period of time, and in some cases, in which the symptoms are slight, perhaps a permanent cessation of the movements can be obtained. In nerve-stretching, which at one time cured almost every disease known to neurologists, but which at the present time has rather fallen into disrepute, we have the means of completely arresting athetosis by producing permanent paralysis of the extremity by severe stretching; or we can produce temporary cessation of the movements, unaccompanied by paralysis, by employing a lesser degree of force. In four cases in which there was marked improvement, the result

was obtained by nerve-stretching in three of them; in the fourth case galvanism is credited with producing the cure.

The first case in which any permanent improvement was obtained, was in a case reported by Dr. Gowers,[1] in 1876. In this case the athetosis followed an attack of hemiplegia; the paralysis was slight, as was also the resulting athetosis, but movements could plainly be observed in both the hand and foot. The patient had never had any convulsions. He was treated with iodide of potash and bromide of potash, and sent on a sea voyage. He returned without showing any improvement. He was then treated with the constant galvanic current. The positive pole was applied to the nape of the neck, and the negative pole rubbed over the overacting muscles. This treatment was continued for two months, with the result that the athetoic movements gradually diminished and finally ceased, in the arm, but no improvement was observed in the leg.

The second case was one reported by Dr. W. J. Morton.[2] The patient was of special interest, as he was the second case on record, and the first one observed in Great Britain. A report of the case was made by Dr. W. T. Gairdner, in the *Journal of Mental Science* for July, 1873. The history of the patient is as follows: When he was three years old he had an attack of hemiplegia, which was not attended by loss of consciousness, nor by convulsions. Three months after the attack, athetoic movements began to show themselves in the hand. The paralysis gradually disappeared, till at the age of eleven he had entirely recovered. The athetosis, in the meanwhile, steadily grew worse. He came to this country and presented himself for treatment at Dr. Hammond's clinic. Dr. Morton took him in charge, and after all medicinal and electrical treatment had failed to give any relief, he decided to stretch the median and ulnar nerves. The operation was performed on November 16, 1882, and was, I believe, the first time that nerve-stretching had been resorted to for the relief of athetosis. While the patient was under the influence of ether all athetoic move-

[1] *Med. Chirurg. Trans.*, London, 1876, vol. lix.

[2] JOURNAL NERV. AND MENTAL DISEASES, 1882, N. S., vii.

ments ceased. The nerves were very forcibly stretched. Quoting from Dr. Morton's report, he says: "Using the nerve as a loop, and the index finger as a crook, the extended arm was lifted again and again from the table, and pulled strongly downwards, so as to pull upon the spinal cord. The ulnar nerve was treated in the same manner. After the patient recovered from the anæsthesia, it was found that the hand and forearm were motionless. The last time Dr. Morton saw the case, between two and three months after the operation, the hand was still paralyzed.

The third case, which I am fortunately able to present to you to-night, is one of great interest, both from a clinical and historical standpoint. He is the original case whose symptoms were first described by Dr. Hammond[1] in 1871. His history is briefly as follows: In 1860 he had an epileptic attack, and has continued having them ever since, sometimes as often as four or five times a week. His habits were bad. He was a gin-drinker, and he states that he has often taken as many as sixty glasses of gin in one day. In 1865 he had an attack of delirium tremens, and remained in a more or less unconscious state for six weeks. After recovering his intelligence he noticed a sensation of numbness in the right arm and leg; severe pain also appeared in these parts, which was shortly followed by the complex movements described under the name of athetosis. At first these movements could be controlled by the will, and ceased entirely during sleep, but gradually, as the athetosis advanced, the will lost its power over the muscles, and eventually the motions were present both day and night. The pain in the arm and leg was intense and frequently kept him awake all night.

I saw him in the spring of 1882. Various medicinal remedies and the different forms of electricity had been used without any beneficial result. I decided to stretch the median nerve, and on May 27, 1882, I performed the operation at the Metropolitan Throat Hospital. While the patient was under the influence of ether no athetoic movements were observed. The median nerve was exposed

[1] "Diseases of Nervous System."

at the inner edge of the biceps, and was apparently in a healthy condition. Passing the finger underneath the nerve, strong traction was brought to bear on it, principally in a downward direction. On the patient's recovery from the ether it was found that the athetosis had entirely ceased both in the arm and in the foot, and that the muscles in both extremities were completely under the control of the will. It was subsequently ascertained that his epileptic paroxysms also ceased, and the pains in both the arm and leg completely disappeared. At the time of the operation he was having from four to six attacks weekly. I exhibited the patient that summer before the American Neurological Association as a case of athetosis cured by nerve-stretching. But in that I was a little premature, as the case will show. About three months after the operation the patient had an epileptic fit. As time went on he had more of them, the pains returned in the arm and foot, and the athetoic movements began slowly to make their appearance, so that in about five months after the operation he was as bad as ever. In 1884 I operated on him again. I exposed the nerve in about the same place. It still appeared healthy. The stretching was performed in the same way as in the first operation, and was attended with the same results. That is, the athetosis disappeared in both hand and foot, the pain ceased, and the convulsions were arrested. He remained well for about four months, then his symptoms reappeared again, and in about eight months' time they were as bad as ever. On February 17, 1885, I operated in the same manner as on the two previous occasions. This time the nerve did not appear to be in such good condition. It had lost its white glistening color and seemed grayish ; it was soft and had lost its elasticity, so that when I stretched it a loop about three inches in length remained outside the wound. The results obtained were the same as in the other two operations. The only difference is that this time they have been more lasting. It is now sixteen months since the operation was performed, and so far as the athetosis goes he is almost recovered. He is able to use his hand in writing, dressing himself, eating, and in fact for

almost any purpose. The muscles of the arm, hand, and foot are perfectly under the control of the will. He can perform extension or flexion, or retain his fingers and toes in any desired position as long as he pleases. When his attention is not directed towards his hand, it is seen to resume its old position, though the motions are absent. This may be the result of habit, as he has been forced to carry his arm in that position for over twenty years. He has an occasional epileptic paroxysm about once or twice a month. He also suffers from that interesting epileptic condition known as double consciousness. He has been for as long a period as four days at a time in that state, apparently perfectly rational, but not being conscious of a single act.

How long his present condition may last, it is of course impossible to state. In view of my past experience in this case, I do not venture for a moment to call this man cured. I would state, however, that I think more improvement has been obtained from nerve-stretching than has ever been secured from the use of electricity or any internal remedies. It will at once occur to you to ask how it is possible, by stretching the median nerve in the arm, to arrest athetoid movements in the foot, and to control epileptic seizures. The only answer I can make to this question is to say that I don't know. That he had the symptoms previously referred to, is a matter of history about which there can be no doubt, and that he has not them now is self-evident. No one would pretend for a moment that such organic lesions as are known to produce athetosis could be removed by stretching a nerve in the arm. If the man had only had athetoic movements in the arm, or if only the movements in the arm had ceased, the improvement could very readily be attributed to a local effect upon the nerve at the point where it was stretched. But the complete cessation of the movements in the hand and foot, the relief of the epileptic seizures, and the intense pain, and the return of the muscles to the control of the will, would seem to indicate that stretching of the nerve has produced such an effect upon the diseased organs that the generation of athetoic impulses becomes impossible. This hypothesis is not so improbable

as it would at first seem to be. Dr. Dana showed conclusively that stretching the sciatic nerve also stretched the spinal cord as high up as the cervical region, and perhaps higher. It therefore does not require that the imagination should be stretched in order to comprehend how stretching the median nerve, which is so much higher up than the sciatic, could influence the corpus striatum or even the cortex. That nerve-stretching holds out the only hope of relief for athetosis, I am firmly convinced. An interesting case is reported by Griedenberg.[1] One of his cases of athetosis was operated on by Dr. Fricke, on August 24, 1882. "The median nerve was stretched, using the finger as a hook. Immediately after the operation, and on the following day, no movements were noticed. On the second day after the operation the athetosis re-appeared, and on the fourth day the movements had regained their former intensity. The disease then remained *in statu quo ante*." It is quite likely that subsequent operations would have been attended with longer intervals of rest.

There is undoubtedly a close connection between all forms of muscular spasm and incoördination due to cerebral disease, as exhibited variously in tremor, clonic spasm, chorea, ataxia, and athetosis; and some authors claim that a lesion in any part of the motor tract, from the cortex cerebri to the spinal cord, may give rise to any of these forms of motor spasms. That this is true of tonic spasm, is proved beyond a doubt by the enormous number of cases with which you are all familiar, and I think it may be shown by the same cases that rigidity and tonic contractions are due to irritation of nerve fibres and not of nerve cells. On the other hand, mobile spasms, as exhibited by chorea, ataxia, and athetosis, depend upon irritation of motor or coördinating cells. There is no evidence to show that a lesion only affecting the motor fibres in the brain has ever been followed by mobile spasms of any kind. On the contrary, the large number of autopsies held on persons who have died while suffering from cerebral chorea, ataxia, and athetosis, invariably present lesions of the cortex or basal

[1] "Vier Falle von Athetose." *St. Petersburg Med. Wochenschr.*, 1882, vii.

ganglia, and when rigidity and tonic spasms are also present the motor fibres are found to be involved in the lesion. In an interesting paper on this subject, Dr. Emil Denange[1] reports eleven cases of cerebral ataxia, chorea, and athetosis, accompanied in many cases by hemiplegia and hemianæsthesia. In some of the cases the disease appeared subsequently on the other side of the body. In nine of these cases autopsies were obtained, and in every one, with the exception of the case of athetosis, a lesion was found either in the corpus striatum, and particularly in the lenticular nucleus, or else the optic thalami were involved. In those cases in which the disease became bilateral, a similar lesion, usually a patch of softening, was discovered in the lenticular nucleus or optic thalamus upon the opposite side. When there was rigidity and hemiplegia, the lesion was observed to affect the motor nerve fibres in the internal capsule.

In the case of athetosis the lesion was found to consist of extensive softening of the cortex-cerebri in the region occupied by the motor centres. Many other similar cases are reported by Charcot, Weir Mitchell, and others.

Dr. S. J. Sharkey,[2] in his exhaustive lecture on muscular spasms, details a large number of cases of tonic spasms and two cases of athetosis. In those cases in which the cortex was involved, tonic spasms followed the paralysis and the post-mortem disclosed degeneration of the motor fibres. In none of the other cases of tonic spasm were the basal ganglia involved, except in one case where there was a tubercular mass in both optic thalami, but in this case there was also tremor. In the two cases of athetosis no post-mortem was held, as the patients did not die. Dr. Sharkey also reports one case of almost complete destruction of one corpus striatum, in which no mobile spasm existed, the patient merely suffering from a temporary hemiplegia, and quotes this case in support of the theory that athetosis and the allied disorders do not depend upon disease of this organ for their existence. But I do not think this one case proves that his views are correct. If mobile

[1] *Revue de Médicine*, May, 1883.

[2] Gulstonian Lectures, *Brit. Med. Journ.*, March 27, 1886.

spasm depends upon irritation of the cells in the corpus striatum, the cortex, and of the optic thalamus as well, the destruction of these organs would undoubtedly prevent the conception of spasmodic impulses. It is only when the destruction is partial, when the cells of these organs are not destroyed, but only irritated, that mobile spasms become possible.

Nothnagel cites several cases of lesion of the lenticular nucleus, followed by hemiplegia, but unaccompanied by mobile spasms, but his cases all died within ten days after the lesion occurred, and before they had time to get athetosis or any thing else.

I think that a careful study of these cases will show pretty conclusively that cerebral mobile spasms, such as tremors, ataxia, chorea, and athetosis, depend upon lesions of the corpus striatum, the optic thalamus, and the cortex cerebri, and not upon disease of the motor nerve fibres, nor any other part of the motor tract.

A lesion simply involving the corpus striatum or optic thalamus does not necessarily produce hemiplegia nor hemianæsthesia. The motor fibres do not pass through the corpus striatum at all, but if a hemorrhage occurring in the corpus striatum is extensive enough, it will exert pressure upon the motor fibres as they pass through the internal capsule, and thus produce a hemiplegia which will gradually disappear as the absorption of the hemorrhage takes place. This is what occurs in a large proportion of the cases of post-hemiplegic athetosis. If the lesion in the corpus striatum does not interfere with the internal capsule, athetosis may result without hemiplegia. In those cases in which the hemiplegia becomes permanent there is undoubted rupture of the motor fibres, but the corpus striatum or the cortex must be included in the lesion as well. I do not think there is a single case on record of post-hemiplegic athetosis in which the lesion was found to be confined entirely to the motor nerve fibres. I have been able to secure the histories of nine cases on whom post-mortem examinations were held. The first case, according to Brissaud,[1] was reported by Lauenstein. A detailed history of the

[1] *Gaz. Hebd.*, 1880, p. 803.

case is not given, but the autopsy disclosed a lesion involving the posterior part of one optic thalamus. The corpora striata and cortex were healthy.

The second case was reported by Pick.[1] Here also the lesion was found to exist in the posterior portion of the optic thalamus.

Grasset[2] details the result of an autopsy held on a case of athetosis. In this case there were three spots of softening, one on the inferior portion of the optic thalamus, one in a portion of the caudate nucleus, and one in the lenticular nucleus.

The fourth case was one of Richets, but was reported by Oulmont. In this case there were several spots of softening in different parts of the hemispheres. There was also an area of softening which destroyed almost the entire posterior portion of the caudate nucleus, and another area which had made a deep cavity in the lenticular nucleus, which further on crossed the internal capsule and joined the lesion in the caudate nucleus.

The fifth case came under the observation of Dr. Fletcher Beach.[3] There had never been any hemiplegia, but the patient had suffered for some years with epileptic seizures. The necropsy was held thirty-six hours after death. The corpus striatum and optic thalamus appeared to be healthy. A microscopical examination was made of the rest of the brain substance, but a similar examination of the corpus striatum and optic thalamus was, for some reason, not deemed necessary. The microscope revealed an increase in number of the vessels, distension of many of them, extensive infiltration of the tissue with leucocytes, especially in the perivascular sheaths of the vessels, and many of the vessels contained clots. These changes were principally in the cortex of the inferior parietal lobule, and first temporo-sphenoidal convolution. It is very likely that the microscope might have demonstrated similar changes in the corpus striatum and optic thalamus.

---

[1] *Prager Vierteljahrschrift*, 1879, p. 141.

[2] *Prog. Méd.*, Paris, Nov. 13, 1880.

[3] "An Account of the Microscopical Appearances in a Case of Athetosis." *Brit. Med. Journal*, 1880, i., 967.

In the sixth case, reported by Ringer,[1] the athetosis had been preceded by hemiplegia, hemianæsthesia, and aphasia. Before the athetosis appeared he had regained his speech, the anæsthesia disappeared and the hemiplegia was partially recovered from. The autopsy showed the left optic thalamus to be smaller and flatter than the right one. There was a cyst occupying the posterior part of the lenticular nucleus, and involving the white matter outside and beneath the thalamus and a small part of the thalamus itself. About one fifth of the lenticular nucleus was destroyed, together with a few fibres of the internal capsule.

The seventh case was reported by Landouzey.[2] There was nothing apparently in this case to account for the disease. The patient was about thirty-two years of age, and had had athetosis from childhood. She had never suffered from epilepsy, nor had she ever had hemiplegia. The autopsy revealed a focus of softening in the extra-ventricular nucleus of the corpus striatum, on the left side. In the centre of this patch of softening, a calculus about the size of a bean, was found. The athetoid movements were in both the hand and foot, on the right side.

The eighth case was reported by Dr. Murrell.[3] The patient at the time of death was thirty-three years of age. When he was three years old he had an attack of whooping cough, and soon afterwards two epileptic attacks, which left him paralyzed down the left side. The paralysis gradually disappeared, so that when he was ten years of age he could run and play like other boys. As the paralysis left him, athetoid movements began to show themselves in both arm and leg, but were always very slight in the leg. The patient died from phthisis. The necropsy was held thirty-six hours after death. The whole right hemisphere was smaller and about three quarters of an inch shorter than the left one. Almost the entire lenticular nucleus was destroyed. The posterior part of the caudate nucleus was unaffected.

[1] "Notes of a Post-Mortem Examination in a Case of Athetosis." *Practitioner*, London, Sept., 1879.

[2] *Progrès Méd.*, 1878, Nos. 5 and 6.

[3] "Case of Athetosis. Death from Phthisis. Post-Mortem." *Lancet*, Lond., 1879, i., 369.

The ninth case was reported by Emil Denange.[1] The patient had suffered from hemiplegia, which was followed by athetosis in the hand. The autopsy showed that the corpus striatum and optic thalamus were perfectly healthy. There was a large patch of softening on the cortex, which involved all that portion of the posterior ascending convolution in which Ferrier locates centres for the complex movements of the finger and hand.

It is therefore evident, from a study of these cases, that in three out of the nine the lesion was found to be in the corpus striatum; in two others in the optic thalamus; in three others both the corpus striatum and optic thalamus were involved; while in the other two cases a diseased condition of the cortex existed.

In regard to the situation of the lesions in chorea, ataxia, and tremor, they may be said to be identical with those of athetosis.

Gowers [2] reports a case of a patient who had an attack of hemiplegia, which came on somewhat deliberately in the course of an hour, was never quite absolute, and soon lessened. The ataxy developed with the recovery of power. It was present in the arm only, and was not accompanied by spontaneous mobile spasms. The incoördination was at one time so violent that if the patient raised an object from the table the arm would fly up over the head. The autopsy revealed a mass of cicatricial tissue through the middle of the optic thalamus, and was evidently the remains of an old focus of softening. The corpus striatum was not affected.

Denange [3] reports nine cases embracing tremor, chorea, and ataxia, in which one or both of the basal ganglia were involved. In one case of ataxia the disease was found to be due to a cortical lesion. In every case where the internal capsule was included in the lesion rigid spasm was also present.

Weir Mitchell reports two cases of post-hemiplegic ataxia. In both cases the lesion was found in the optic thalamus.

[1] *Revue de Médecine*, Paris, May, 1883.

[2] *Med.-Chirurg. Review*, London, 1876, vol. lix.

[3] *Op. cit.*

Sharkey [1] reports two cases, one of tremor accompanied by paralysis and rigidity, in which both thalami were invaded by tubercular deposits, which also pressed upon the internal capsule; the other was a case of ataxia, in which the lesion was found to exist in the cortex.

It is mainly from a study of autopsies that we are able to derive any definite knowledge of diseases of this character, and if autopsies prove any thing, I think these show that athetosis and its allied disorders depend upon lesions of the corpus striatum, optic thalamus, and cortex cerebri, and of these parts only.

[1] *Op. cit.*

Clinical Cases.

VESICO-GENITO-POST-FEMORAL NEURALGIA AND NEURITIS.

By LANDON CARTER GRAY, M.D.

It has been my fortune during the last year to have met with two cases of a peculiar variety of neuralgia and neuritis that has not, so far as I have been able to ascertain, been hitherto described. The first case I was disposed to regard as a merely accidental localization, but the occurrence of a second precisely similar and far graver case demonstrated that this could not be so.

Case 1.—Male, aged forty, general health excellent, of markedly lithæmic temperament. Has for years been subject every summer to quasi-malarial attacks, characterized by sudden neuralgia, which is followed within a few hours, unless checked by large doses of quinine and opium, by a remitting fever of moderate type. This fever will usually last three to four days. Hitherto the neuralgia has always been gastric or intestinal. This summer the patient had been unusually well, and had congratulated himself that he would escape his usual trouble. In the latter part of July, however, there were several days of intensely hot weather, upon the last of which the patient felt much exhausted. This high temperature was suddenly transformed, in a night, into weather that was autumnal, the thermometer falling from 98° F. to 60°, an enormous difference, for summer-clad individuals, of 48°. Upon the first day of this change no untoward symptoms were experienced ; indeed, the patient went to bed feeling unusually well. But upon the second day, on getting out of bed in the morning, a sharp, tingling pain was felt through both buttocks, the perineum, the scrotum, the tip of the penis, and down the back of both thighs to somewhat above the knee. Some slight smarting was felt in urination. Towards the afternoon the pain began to lessen. The patient then, unfortunately, took a Turkish bath, in which, curious to say, he found it impossible to perspire, even at a temperature of 180°, although the heat was sufficient to nauseate him. The Russian or vapor bath, however, caused free

action of the skin. Immediately upon his return home from the bath he became much worse, for not only did the cutaneous pains become violent, but urination was scalding, and over and above this particular difficulty in voiding urine, the bladder became paretic, so that there was a visible effort in forcing the urine into the urethra. The next day there was a febrile movement to 103° F., and for three days after there was a temperature of 100° in the morning, rising to 102° or 103° in the afternoon, the neuralgic symptoms still existing, but not causing much inconvenience. The patient was then sent to the country, and upon his arrival there had a violent attack of supra-orbital neuralgia, after which he quickly recovered. For several weeks, nevertheless, there was some pain and difficulty in micturition, and considerable pain and stiffness at times down the back of the thighs. During the first four or five days of the attack, the sexual desire was entirely lost, and for several weeks afterward the orgasm was wanting; whilst the seminal emissions produced a burning sensation at the meatus. For the first day or two there was slight impairment of tactile sense, but none of the other senses of pain, temperature, or muscle. Skin reflexes were lost; Cremaster reflex could not be evoked during the first week.

Case 2.—Female, aged thirty-five, unmarried. Seen in consultation with Dr. J. Hobart Burge, on Sept. 6, 1886.

In the last two years patient seems to have had a good deal of sciatica. Otherwise she has enjoyed good health; is a large, robust, healthy-looking woman. In December last was suddenly attacked with sharp pain in buttocks, perineum, labia, and down back part of thighs to the knee. There was simultaneous retention of urine, so that catheter had to be used. The trouble was regarded as rheumatic by the attending physician, and it was not until several weeks later, when she passed into Dr. Burge's care, that the true nature of the case was recognized. Dr. Burge then found a tactile anæsthesia of the buttock, perineum, labia, and the back part of the thighs to just above the knee. When I saw the patient, nine months after the onset of the trouble, she had a vesical anæsthesia, evidenced by the fact that the urine would be voided without her knowledge upon the street or wherever she might be. Over the area of pain above described, with the exception of the labia, which the patient's modesty would not permit me to examine, I found marked impairment of the tactile sense, of the sense of temperature, of the sense of pain, but slightly less near the knee than above. There had never been any motor impairment, not even reflex stiffness.

I can find no cases like these in the books of Rosenthal, Ross, Erb, Anstie, Eulenburg, Strümpell, or Hammond. Perineal and anal neuralgias have been described, especially by Dr. Weir Mitchell,[1] but they have not been accompanied by the other symptoms of my cases. Dardel[2] has written upon ano-vesical neuralgia,

[1] "Anal and Perineal Neuralgia." *Phil. Med. Times*, 1873.

[2] "Deux observ. de névrose ano-vesicale opiniâtre, sans cause matér. appréciable." *Gaz. Méd. de Lyon*, 1867.

but I have not been able to obtain access to his article, and I am therefore unable to say as to whether his cases are similar to mine. Certainly the title of the paper would seem to indicate that they were not. Neuralgia of the post-femoral region has also been mentioned, but only in connection with sciatica.

These cases have a clinical interest, I think, because the knowledge that such a neuralgia or neuritis may occur, will make us chary about diagnosticating a central affection, as we might well be inclined to do in the early stage, especially when there was vesical, motor or sensory paralysis.

REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.*

Prepared under the direction of PHILIP LEIDY, M.D., Physician-in-Chief, and CHARLES K. MILLS, M.D., Consulting Physician.

Chronic Mania.

M. Q. was admitted March 22, 1872, to the hospital. This was his second admission; age twenty (1872), born in Philadelphia, white, single. He had no occupation nor religion; he could read and write.

When he was first admitted he remained only a few weeks. He was six months at the Pennsylvania Institution for Feeble-Minded Children, at Elwyn, Pa.

There was no insanity known to exist in his family.

When he was seven years of age the patient had a fall of about four feet, striking upon his head and inflicting a deep scalp wound. This wound, however, healed up quickly and kindly, and did not appear to leave any bad effects. At thirteen years of age he began to show signs of great depression of spirits; previous to this time he seemed to have a good disposition, and was obedient, moral, and cheerful. He now would not play at games with his friends, etc., but preferred to be alone and remain quiet; he would sneak off in a corner by himself. He was addicted to the habit of masturbation at this time, and it is believed that he continued to practise this habit.

When he was fourteen years of age, chorea developed, and a few months later symptoms of insanity began to appear, and gradually increased until his nineteenth year, when the disease assumed a chronic form.

He has hallucinations of sight and hearing. He will frequently address people as "brother Billy," and also call some person by an unknown name. He will point towards a certain spot, and talk, scold, and swear at imaginary people by the hour. He will talk to himself, asking and answering questions, etc. He has

* Carefully prepared notes were furnished by Dr. Da Costa, one of the assistant physicians to the hospital.

shown at times a disposition to injure others when he becomes enraged, but he has never threatened or attempted suicide. He is filthy in his habits, and destructive to clothing, furniture, etc.

He never used stimulants in any form, and neither smoked nor chewed tobacco. His physical condition is good.

The following is a sample of the conversation of this patient, which was, as is always the case, accompanied by striking attitudes, exhibiting every phase of emotion, and passing from tears to laughter with great rapidity :

"I shot you, brother Willie. It is grandfather. Do you love Mike ? I do, Mr. Quinn. Say, Mr. Quinn, what do you see ? I see a corpse in behind, it is brother Willie. Brother Willie is dead." (This with tears.) "No, it is the devil, the devil after Mike !" (This with a look of abject terror.) "He is gone, Doctor. Did you see grandfather up there ? He is going to the almshouse." (This with laughter.) "Go to hell, damn you, you dirty dog, you louse," etc. (This with an appearance of frightful rage.) And so he continues for hours every day.

Stuporous Melancholia with Cataleptoid Symptoms—Effect of Inhalation of Nitrite of Amyl.

S. E. was admitted to the hospital Oct. 18, 1885, aged twenty-eight years. He was born in Poland, and does not speak English ; was married, and was a peddler. About two weeks before admission he showed signs of great fear and suspicion, raving occasionally. A physician said he had typhoid fever, and he was carried to the hospital in an ambulance, fighting violently when he was taken out of it. On admission he was thought to be suffering from acute melancholia. In a day or two he passed into a condition of stupor, refused food, and had to be fed with a tube ; soon after this cataleptic symptoms developed. He remained in this state for ten months, and then he began to eat of his own accord, if he was put at the table, and a fork placed in his hand. His urine and fæces apparently passed involuntarily, and saliva dribbled constantly from his mouth.

His tongue was pale and coated. He had no apparent defect of sight or hearing, and his lungs were in a normal condition. His heart action was normal but weak, and his pulse 92 and small.

Oct. 18, 1885.—Harsh respiration and impaired percussion-resonance were present at the left apex. He was in a markedly cataleptic state, with limbs, head, and even the lower jaw remaining for a long time in any position in which they were placed. After a time tremor began in the part placed in any fixed position, and became more marked until it returned to its place from exhaustion. When in the usual position he showed considerable contraction of the flexors, which took some force to overcome ; but when a limb was extended it took more force to put it back, the resistance being apparently volitional, and ceasing when the part became tired. He sat with an expression of pro-

found gloom, alternating with one of abject terror, probably caused by frightful hallucinations. His hands and feet were blue and cold, and his appetite poor. The bowels were constipated. His pupils were dilated and equal, and reacted to light. The reflexes were exaggerated. There was excess of phosphates, but no albumen in the urine.

He was placed on tri-weekly inhalations of six drops of amyl nitrite, the amyl being used to the point of marked flushing of the face. After the first inhalation he got up voluntarily and walked for twenty minutes, a thing he had not done for months.

Since these administrations he has developed a new symptom: if a motion is started, such as moving his head backwards and forwards, or clapping his hands together, he will continue it until he is exhausted.

FURTHER NOTE ON A CASE OF TREPHINING FOR TRAUMATIC EPILEPSY.

In the January number of this JOURNAL a case of trephining for traumatic epilepsy was reported by Charles K. Mills, M.D., and J. William White, M.D. The patient was a young man twenty-six years old, who had suffered from fracture of the skull from a pistol-shot. He had severe convulsions, which were presumably due to inflammation of the dura mater. Fragments of bone were removed from the dura mater by the operation. The operation was performed January 27, 1886, and the history of the case published in the JOURNAL extended only to Feb. 11th.

The patient made a perfect recovery from the operation; and since that date has had two convulsions and a slight attack of partial unconsciousness. He is still very slightly paretic on the left side.

Comparing his condition during the ten months since the operation with that during the four months preceding, a very great improvement has resulted. During the four months preceding the operation he had eight severe convulsions altogether, including one just prior to the operation. In the ten months since he has had only two convulsions, and his general condition is good.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, October 5, 1886.

C. L. DANA, M.D., President, in the chair.

Case of Congenital Absence of the Faculty of Co-ordination.

Dr. G. M. HAMMOND presented a boy four years of age, brought to his clinic on account of inability to walk. He was born at full term; labor was natural; he appeared to be perfectly healthy at birth. But shortly afterward he became sick, and continued more or less ill for six months. The attending physician diagnosticated colic. Since recovery from this attack the patient had had no sickness. The special senses were normal; the patient understood as well as other children of his age. Perhaps he did not speak as plainly as he should, but two other children in the family, perfectly healthy, talked in the same manner. There was no history of syphilis. The patient was well formed; the muscles of the limbs were well developed for a child who did not walk; muscular reaction, to both electric currents, was normal; the reflexes were normal. The only apparent reason for his inability to walk was want of power to retain his equilibrium. He could crawl on his hand and knees perfectly well unless he attempted to go very fast, when he would fall, and he always fell toward the right. He could stand, holding to a chair, and walk pretty well if held upright. There was also inco-ordination in the upper extremities. He widened his base in standing. Dr. Hammond had not decided whether there was congenital absence of sensory tract in the cord or cerebellar disease.

Dr. JULIUS RUDISCH had seen two similar cases, both in girls,

one about eight years old and the other about thirteen. The first was seen some years ago, was under observation but a short time, and his recollection of the case was indistinct. But he was impressed with what he took to be muscular weakness ; not simply ataxia, but weakness in the back. The child, if sustained, could walk well ; if not sustained, it would fall like the boy presented by Dr. Hammond. The older patient could walk, but in a peculiar, ataxic way and in the position of marked lordosis. The legs were well developed, and for that reason he thought the trouble was in the muscles of the back. The cases were not, in his opinion, congenital locomotor ataxia.

Dr. N. E. BRILL thought such cases were not uncommon, especially among idiots. The gait of the boy presented reminded him of the swaying motion of a cat, sent Dr. Spitzka by Prof. Wilder. Other actions than walking were natural. Rumpf reported a similar case and found a rudimentary cerebellum. Dr. Spitzka removed the cerebellum of this cat and found it natural. Dr. Brill thought he had to distinguish in these cases between locomotor ataxia and static ataxia. Dr. Hammond's case appeared to be one of static ataxia due to rudimentary cerebellum.

The PRESIDENT had found in his case-books one in which a provisional diagnosis of infantile ataxia was recorded. The girl, about two years old, was well nourished, large, could not coordinate the hands or feet. In addition there were some forced movements ; the head would suddenly plunge forward.

Vesico-genito-post-femoral Neuralgia and Neuritis.

Dr. LANDON CARTER GRAY read a paper in which he described two cases, seen during the year, of a peculiar variety of neuralgia and neuritis that had not, so far as he has been able to ascertain, been hitherto described. See p. 743.

Dr. RUDISCH asked whether an examination had been made for prostatitis which sometimes caused symptoms in a degree like those described.

Dr. GRAY said the area of distribution of the pain was not like that in prostatitis ; besides, the second case occurred in a female.

Dr. W. H. THOMSON referred to the case of a woman from the country, a locality said by her physician to be free from malaria. After a prolonged convalescence from an attack of pleurisy she began to suffer severe pain in the anterior part of the left thigh, and from slight trouble with the bladder, the pains coming on

certain days of the week, lasting one day and two nights. This continued five months, when she was free until the following fall. The medicine prescribed by Dr. Thomson had not prevented a return of the singular symptoms again the present fall. There was no indication of sciatica.

Discussion on the Uses of Hyoscyamine.

The PRESIDENT stated that there were two preparations of the drug, the crystalline and the amorphous. The former seemed to be similar in property to the opiates, while the latter seemed to have neurotic properties. He had heard that hyoscyamine was employed in the asylums for the insane in New York, but not very successfully, whereas in the asylums of Pennsylvania its success had been marked. He had employed hyoscyamine in paralysis agitans, in chorea, and in a few cases as an hypnotic, and it had been employed as an hypnotic to a considerable extent in his service at Bellevue Hospital. The number of cases of chorea in which he had used it was six ; in three it was noted to have been of benefit, or caused very rapid or very marked improvement. One of the cases was marked, and had not yielded to other treatment. In three cases the results were very doubtful. He had employed it in four cases of paralysis agitans. In two, he thought it an unquestionable benefit. In two it seemed to produce no benefit at all. On the whole he thought that unless given at rather an early stage of paralysis agitans it did no good. The form employed in chorea and paralysis agitans was the crystalline, but he was not sure that the amorphous form would not be the better preparation in such cases. He thought he could get along perhaps as well without as with hyoscyamine.

Dr. B. SACHS' experience with hyoscyamine had not been very extensive, but he had employed it in a few cases of paralysis agitans, acute mania, and the insomnia accompanying the neurasthenic condition. He had employed only the crystalline form. In contradistinction to what the President had said, that to be effective it must be given in the early stage of paralysis agitans, he remembered one case in which every other therapeutic agent had been tried without success, when hyoscyamine was administered in about one-hundredth-of-a-grain doses twice a day, with the effect of making the patient very much more comfortable, and of diminishing somewhat the annoying movements of the hand. In another chronic case it had been of no benefit. He had obtained

excellent effects from the drug administered to allay the excitement of acute mania. It had been disappointing, however, in insomnia accompanying neurasthenia. It seemed to be more valuable against insomnia from mental restlessness.

Dr. W. H. Leszynsky said that about eight years ago it was quite fashionable to use hyoscyamine in asylum practice, and he had employed it in chronic mania, acute mania, and epileptic forms of insanity. First he used the amorphous, and later, sulphate of hyoscyamine. It was claimed that the latter form was easier absorbed, and produced its effects in smaller doses. The sulphate was also preferred for hypodermic use, in which manner he had employed it in one-sixtieth-of-a-grain doses. To patients with recurring attacks of maniacal symptoms, the drug was given a few days before an expected attack, and continued until the attack was aborted.

In a state of exhaustion he would regard hyoscyamine as a dangerous drug to administer. But where there was no objection to its use on that ground, he had known it to produce sleep where chloral and morphine had failed. Given to patients subject to epileptiform convulsions before menstruation, it seemed to avert the attack. He had given it in small doses in two or three cases of chorea, and thought it produced some benefit.

Dr. L. C. Gray had been using hyoscyamine ever since it had been introduced to the profession, and he must say that for certain purposes there was no drug in the pharmacopœia that he could not better afford to dispense with. The most convenient form was in tablets, one one-hundredth of a grain. In some people hyoscyamine would produce seemingly serious retention of urine. It might also produce disastrous results if given to persons whose general strength was below par. In an old gentleman, with atheromatous arteries, hypertrophied and feeble heart, one one-hundredth of a grain of hyoscyamine caused a condition of collapse. He knew of one patient suffering from melancholia who was sent to Greenwood by hyoscyamine. He had given it in two cases of chorea, one being an exceedingly violent case, the child finally dying in a convulsion. To that patient he could never give a second dose of hyoscyamine, because of the alarming prostration which a first dose would cause. In another case, in which the child had to be held in bed, the drug proved an effective means of restraint, but the child was always found prostrated to a marked degree the next day. In paralysis agitans it had been very useful,

and came to be with him a routine treatment. He thought the reason why it had been of more benefit in his practice was that he combined with it some stimulant or tonic to prevent its depressing effect. He gave with it good food, one or two grains of quinine a day, sometimes alcoholic stimulants. He had satisfied himself that it was the hyoscyamine in this treatment which had a restraining effect upon the movements in paralysis agitans.

But it was especially in cases of mental trouble where hyoscyamine was of great benefit. In insanity with hallucinatory symptoms, especially in the early stage, before the patient could be taken to an asylum, hyoscyamine would do much toward restraining the patient, and it would seem aided in cutting short the disease. He was very careful to give no larger dose of the drug than was absolutely necessary, and he combined it with bromide of potassium, which increased its effect. He had never seen hypnotic effects from hyoscyamine.

Dr. W. H. THOMSON said that his experience with hyoscyamine, almost from the beginning, rather prejudiced him against it. One of the first cases in which he employed it was that of a judge troubled with insomnia. The next day he was unable to hold court, had bladder symptoms, etc. He found it useful in asthma with considerable dilatation of the right side of the heart, without bronchitis, but a congested state of the lungs. He had employed it in facial neuralgia, headaches, and various neurasthænic conditions, but had nothing definite to say about its effects. One patient with paralysis agitans was benefited by it among many with whom it was a failure.

Dr. KELLOGG had used hyoscyamine in cases of mental excitement, but it had not proven the sedative he had supposed it would, but it controlled muscular excitement. He had failed to get any hypnotic effect from it. He had not been favorably impressed with its after-effect in acute mania.

Stated Meeting, November 2, 1886.

The President, C. L. DANA, M.D., in the chair.

A Case of Bi-temporal Hemianopsia.

Dr. EDWARD WAITZFELDER presented the patient, a lad whose general history was negative until five months ago. He then noticed a "blur" over the right eye. He consulted Dr. P. A. Callan, at the New York Eye and Ear Dispensary, who diagnosti-

cated right temporal hemianopsia. The condition grew worse ; the left optic nerve became involved, and left temporal hemianopsia developed. Two months ago the patient had choked disk in the right eye. His condition November 1st was as follows : No other basal nerve affected ; he has complete bi-temporal hemianopsia, the whole of fixation point being included in the seeing field. Theoretically the fixation should be bisected, but practically it never is. There is atrophy of both optic nerves, most marked in the right. Vision—R. E., $\frac{8}{200}$; L. E., $\frac{20}{20}$. Diagnosis—Tumor of the chiasm in the anterior portion ; pressure upon the fasciculi cruciati of both optic nerves, the fasciculi laterales being as yet unaffected. There is no tubercular or syphilitic history. The rapid growth of the lesion points strongly in the direction of sarcoma.

So far as Dr. Waitzfelder could ascertain this was the first recorded case in the English language. The point of special interest in the case was that the fixation point was " dodged " by the line of the hemianopsia. Why this was so he could not say, but it would seem to indicate that there was a special set of nerves intended only for extreme central vision.

Remarks were made on the case by Drs. Pooley, Webster, Starr, Leszinsky, and Bullard.

Thomsen's Disease.

Dr. GEORGE W. JACOBY presented a young man suffering from Thomsen's disease. The history will be published hereafter.

Remarks on Cocaine and the So-called Cocaine Habit.

Dr. W. A. HAMMOND made some remarks upon his personal experience with some of the preparations of cocaine. He had used only the fluid extract, various wines, and hydrochlorate of cocaine. The fluid extract had been discarded by him since two or three years, mainly because it had been badly borne by the stomach ; it excited nausea, and was disagreeable to the taste. He then began the use of the wines, but finding that they differed so much in their effects, he gave them up, until he suggested to Thurber & Co. to try to make a wine of coca free from tannin and extractive matters, and they had, he believed, entirely succeeded in doing so. There were two grains of the hydrochlorate of cocaine to the pint of wine. With this preparation he had had an extensive experience, not only upon others, but upon himself. He had used it in spinal irritation with excellent results—results

which could not be attributed alone to the wine, but in part to the cocaine. He had used it also as a general tonic and for fatigue. For some time past he had been in the habit of taking a wineglassful at the close of his day's duties, and with benefit; it certainly had a decidedly restorative effect, without being followed by a feeling of depression. He had also used it in some cases of dyspepsia with a very irritable state of the stomach. He supposed its action was by lessening the sensibility of the stomach, as it lessened sensibility when applied to other parts. It was remarkable to what an extent the irritability of the stomach was overcome by doses of two or three teaspoonfuls of the wine of coca repeated at intervals of fifteen or twenty minutes, until half a dozen doses had been taken. If the first doses were vomited, the succeeding ones would be retained longer, until finally they were retained altogether. Cases of irritability of the stomach, due apparently to spinal irritation, had been relieved within a few hours by this treatment. Generally when he wished in any case to produce a powerful therapeutical effect, he employed the salt.

Dr. Hammond here spoke briefly of the physiological effects of coca, and said that the first writer on this drug, who had described its effect upon the native Indians of South America, gave an exaggerated account of its baneful influence, and his ideas had been copied over and over again, without the authority being given, until our minds had become thoroughly indoctrinated by them That author said, among other things, that the coca rendered the teeth black, produced ulceration of the tongue, caused the breath to be fetid, the jaws to become ulcerated, the bones to soften, and rendered the patient an idiot. But subsequent observers said that such results must be entirely exceptional, as they had never seen them. If there was discoloration of the teeth Dr. Hammond thought it might be accounted for by the lime which the Indians mixed with the leaves, or by the presence of tannin. There had recently been some very striking stories in the newspapers regarding the injurious effects of the drug upon persons who had become addicted to its use. In order to determine whether there was any truth in these statements, Dr. Hammond made some experiments upon himself. He first injected hypodermically one grain of the hydrochlorate of cocaine, which caused an exhilaration of spirits and a happier state of mind than he had enjoyed during that day. He was unable to sleep that night until four or five o'clock in the morning, and when he got up he

had a severe headache. He also had a large evacuation of urine. The effect of the drug was to produce an exhilaration, such as would be produced by two or three glasses of champagne. The next night he injected two grains, which produced the same pleasant feeling, and in addition he felt an inordinate desire to write. He wrote eight or ten pages of foolscap, and thought it was the best that he had ever written, but the next morning he found that it was the most extreme nonsense. Each sentence was complete in itself, but no two sentences had any relation to each other. The first part was more incoherent than the latter. The next night he injected three grains, and although he again felt the disposition to write, he did not indulge it, but he talked a great deal, and made speeches. He knew what he was about, and was able to restrain himself, but it was pleasant to speak. He went to sleep late and again awoke with a severe headache. It was a peculiar fact in his case that, at the point of injection, there always developed redness, swelling, stopping short only of an abscess. He now had several hard spots on his arm, and waited four or five days, when he injected six grains of hydrochlorate of cocaine, three grains at two different places. He then felt decidedly "upset," yet he did not lose consciousness nor his relation to things. He gave instructions to the servants correctly. But he did not feel a strong disposition to write or to talk. He was unable to sleep at all that night. The injections were always followed by large evacuation of urine, and by headache next day, but without debility. Three nights later he injected eight grains with about the same effects. The next night he injected eighteen grains, making six different punctures, all inside of twenty minutes. He became intensely exhilarated, and was unable some hours afterward to recall what he did. He was in his office, but in some way got to bed, and the next day he found things in more or less disorder in his office. His headache remained for two days, and there was great action of the heart, palpitation ; he could hear it beating on raising the arm to the head. Exaggerated action of the heart had also attended the smaller doses. But he experienced none of the horrible effects which were said to attend the use of the drug in large or continued doses—no disposition to murder, or commit acts of violence. He acquired no habit ; he was able to quit its use at once. And regarding the cocaine habit he would say that he had given the drug in doses of from one to five grains for three months to a lady suffering

from exophthalmic goitre, and she was then able to discontinue its use without any difficulty. At no time did she manifest any loss of moral principle. She took two doses a day. From a theoretical standpoint, perhaps cocaine should not be administered in this disease, but it proved beneficial in this case, for the heart's action, which had been increased, diminished, became steadier, and the patient felt much better. He also gave it for some months to a lady addicted to the opium habit, carrying the dose up to five grains injected once a day. It overcame the opium habit, and the patient failed to acquire the so-called cocaine habit. In this, and other patients to whom he had administered cocaine, it produced, as in his own case, extraordinary action of the heart, increased temperature and blood pressure, perspiration, and indisposition to sleep.

He had used a ten-per-cent. solution of cocaine, soaked in lint and applied to the vulva, for the relief of masturbation. But it had failed in one case, that of a girl four or five years of age. It had been ineffectual in boys, applied to the glans penis.

In three cases of melancholia in women who refused to speak, injections of hydrate of cocaine had overcome the prolonged silence. The first was a marked case of melancholia with stupor, and the patient had not spoken for nine months. At the first sitting he injected one grain of hydrochlorate of cocaine. The patient then nodded or shook her head in reply to questions, but would not speak. At the next sitting three grains of the drug were injected, and within four minutes the patient replied to questions by yes and no, and within ten minutes she began to talk, and kept on talking, although incoherently. She did not sleep that night, and seemed to have pain in the head the next morning. The next injection of three grains caused the patient to talk, but less incoherently. This was a year ago, and the patient continued as melancholic as before, but she talked, if that was any advantage. Dr. Hammond had failed occasionally to induce patients to speak by injections of cocaine.

As to the cocaine habit, Dr. Hammond regarded it as similar to the tea or coffee habit, and unlike the opium habit. He did not believe there was a single instance of well-pronounced cocaine habit, the patient being unable to stop it at any time, if he chose to do so. If a person were to continue its use for a long time, he should be inclined to look for trouble with the heart rather than with other organs.

Dr. J. B. MATTISON, of Brooklyn, could not agree with Dr. Hammond that there was not a cocaine habit. Within a few months Dr. Mattison had had seven cases of the cocaine habit under his care, five in physicians, two in druggists. He certainly believed there was such a thing as cocaine addiction. He regarded the drug as most dangerous and destructive of the tissues. In certain cases its action was more unfavorable even than morphine. The cases reported in the newspapers he thought were founded on facts. In one instance he wrote to a physician asking whether the report was true that a certain doctor had been arrested in the street under the influence of cocaine. The physician replied that it was true ; that the doctor was a victim to cocaine. He could cite other similar cases. In one instance a physician attempted to write a prescription for a patient, but instead wrote for the sheriff to come and take him to jail. The effects of cocaine, as far as he had observed, were similar to those described by Dr. Hammond, but besides the action upon the heart, the great volubility and the unrest, he had noticed hallucinations aud delusions, but no homicidal or suicidal tendency. In some cases there was marked emaciation. He thought the effects of the continued use of cocaine were more decided than those of the continued use of morphine. The patients whom he had treated had acquired the cocaine habit gradually, making comparatively small injections several times a day. Dr. Hammond seemed to think that no dose was toxic, but Dr. Mattison regarded Dr. Hammond's case as exceptional, and he would not advise any physician to repeat the experiment.

Dr. J. LEONARD CORNING thought there was a morbid fear of cocaine spreading through the country, and he thought the remarks of Dr. Hammond were timely, as they would tend to allay the prejudice against a most useful remedy.

Dr. L. C. GRAY remarked that between Dr. Hammond on the one side, and Dr. Mattison on the other there was considerable distance, and he did not know how the question could be solved, except by further experience. Dr. Hammond's statement that no cases had been reported by medical men was a mistake. Cases had been reported in Europe, but they were not numerous.

The PRESIDENT read a communication from Dr. C. H. HUGHES, of St. Louis, in which he said : Most of the cases of cocaine habit seen by me have been mixed cases of opium, cocaine, and alcohol or ether inebriety, combined or alternating ; though I think I

know of cases where cocaine is the chief if not the exclusive reliance. But these patients are not reliable in their statements. I have not seen a physician addicted to cocaine who stuck to cocaine exclusively. The finale has generally been cocaine and opium and whiskey and ether and all the other narcotic stimulants. Opium is a much more agreeable stimulant, and most patients evidently try to get back to the fatal bliss of opium. I have never relied on cocaine alone in breaking up the opium habit. I never use cocaine to intoxication, and never regularly. My rule with cocaine cases, as I usually see them, is to get them back to plain opium, and then break them of that if advisable.

Dr. Hughes referred to the fact that in some cases cocaine produced poisonous effects.

The PRESIDENT referred to thirteen cases of cocaine habit reported by Erlenmeyer, and to a case reported by Bornemann. The subject, he said, had recently been discussed at the meeting of the German Congress of Physicians and Naturalists. Dr. SMIDT reported some cases of cocaine-morphine habit. The general opinion was that pure cocaine addiction was rare, but that the cocaine-morphine habit was not so, and was a very destructive and pernicious habit.

Dr. HAMMOND, in closing the discussion, said he did not deny the existence of a cocaine habit; he only claimed that it was unlike the opium habit, for the patient could break it off at will. He was aware that patients addicted to the use of opium, sometimes added cocaine, greatly to their detriment. As to cocaine being a poison, twenty and even thirty-two grains had been taken without serious results. He differed from Dr. Mattison, who thought it was more injurious employed hypodermically; but the patient came under its influence more slowly when it was taken into the stomach.

The PRESIDENT reported for Dr. HERMAN M. BIGGS

A Case of Subacute Spinal Paralysis,

and exhibited specimens of the cord and sciatic nerve. The case was one characterized by gradual paralysis of the lower and then of the upper extremities, moderate atrophy, later a slight anæsthesia of the lower extremities, no pain, loss of tendon reflexes, and no bladder troubles. The course was progressive. Death took place in five months. History: The patient was a male, aged fifty-three, not syphilitic. The interest in the case lay

in the rarity of the affection, and especially of cases in which post-mortem observations had been made. Clinically it resembled mostly the subacute spinal paralysis of Duchenne, although that disease is very rarely fatal. It still more strongly resembled a chronic form of Landry's acute ascending paralysis, and gave support to Ross' classification, of (1) Landry's paralysis ; (2) the subacute paralysis of Duchenne ; (3) periependymal myelitis ; and (4) progressive muscular atrophy, as inflammatory processes, attacking the central gray matter of the cord, and distinguished by the greater or less acuteness of the process. The case was interesting also as showing that these paralyses are not always, at least, due to neuritis. Dr. Dana showed sections of the lumbar and upper dorsal cord, which, he thought, showed evidences of a low grade of central myelitis. The anterior roots and the sciatic nerve were apparently normal.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 25, 1886.

The Vice-President, CHARLES K. MILLS, in the chair.

Dr. MORRIS J. LEWIS read a paper on

A Partial Study of the Seasonal Relations of Chorea and Rheumatism.

The paper is based upon a study of the seasonal relations of chorea and rheumatism, and for this purpose the weather at Philadelphia, for the years 1876–1885 inclusive, has been investigated, together with the number of storm centres, or centres of low barometer, passing across or near the United States. These data were obtained from the monthly weather reports, and from the records kept at the signal station in that city, which have been kindly placed at his disposal.

Recent investigations, undertaken by him in 1881, in regard to the relation existing between the number of cases of chorea and the varying states of the weather, showed such a curious resemblance between the chorea and storm tracings that he deemed the subject worthy of further study

The former work is embodied in Dr. S. Weir Mitchell's book of "Lectures on Diseases of the Nervous System, Especially in Women," and the statistics there mentioned were based upon the study of the months of onset of one hundred and seventy separate attacks of chorea occurring in the years 1876–1880 inclusive, but at that time the storm records of the years 1878–1880 inclusive only were available, during which time eighty-seven of the above-mentioned one hundred and seventy attacks occurred. For the present study he had collected all the separate attacks of chorea which had been noted at the clinics of the Orthopædic Hospital and Infirmary for Nervous Diseases during the years

1876–1885 inclusive. These number in all four hundred and thirty-seven, and necessarily include those previously studied.

The reader continued : As it is a well-recognized fact that a relationship exists between chorea and rheumatism, which, it is needless to say, is as yet imperfectly understood, I thought that it would be of much interest to compare with the cases of chorea the months of onset of attacks of acute inflammatory rheumatism occurring during the same period, and for this purpose have carefully studied the notes taken at the Pennsylvania Hospital, which have been placed at my disposal ; from these I have collected four hundred and sixty-seven separate attacks of rheumatism, having excluded those that are at all doubtful, and those which occurred among sailors at sea, who were afterward brought to this port.

It must be borne in mind that while the records of the weather are complete as far as they go, the cases of disease here reported are but a small portion of those that must have occurred in this city during that decade ; but, despite this fact, there is every probability that the percentage of chorea cases per month is practically correct, as may be seen by the almost exact resemblance between the two tracings of one hundred and seventy and four hundred and thirty-seven attacks, respectively—Nos. 3 and 5.

To study properly the effect of the weather upon disease, there should be a *daily* record of both weather and disease taken, as has been so carefully done in the relation of pain to weather by Captain Catlin, recently reported by Dr. Mitchell before the College of Physicians ("Transactions of the College of Physicians of Philadelphia," vol. vi.). In the diseases in question, however, the *day of onset* could only be obtained with certainty in a few cases, and, therefore, it was necessary to be satisfied with the *month of onset ;* this, of necessity, destroys, to some extent, the value of the observations, but sufficient of interest remains to justify the present study. By taking the average of too long a period of weather the effect of sudden changes and all irregularities are lost sight of, and in this way some facts of importance may be overlooked.

In the present study the monthly average of the weather for ten years has been taken and compared with the monthly average of disease, but the peculiarities about to be spoken of are best seen when a table is made which shows the mean of the ten years in question, month by month.

The accompanying table is so arranged by repeating the first half of the year, that any portion of the tracings may be studied in unbroken sequence.

Tracing No. 5 (shown at the meeting) represents the *months of onset* of four hundred and thirty-seven separate attacks of chorea, and shows the actual number for each set of months, or the average number for each set, according to the manner of reading the figures; thus, January shows thirty-six attacks, which means thirty-six attacks for the ten Januarys, or 3.6 cases as an average for each January.

This tracing will be seen to be lowest in October and November, eighteen and nineteen attacks, respectively (4.1 and 4.3 per cent.); it then rises rapidly, until thirty-six attacks in January have been reached (8.2 per cent.), falls slightly in February, and again rises, reaching its highest point in March, which shows sixty-two attacks (15.3 per cent.); the line then falls in April to thirty-eight attacks (8.6 per cent.), and after two more rises—one in May, forty-seven attacks (10.7 per cent.), and the other in July, forty-six attacks (10.5 per cent.)—falls almost perfectly regularly to its lowest point in October. Before attempting to find an explanation for the high percentage in March, I will again draw attention to tracing No. 3, which represents the one hundred and seventy separate attacks previously reported; this will be seen to resemble, in every essential particular, the tracing we have just studied. These cases I have included in the present paper. Nothing especial is seen in comparing, with the chorea tracing, that of the *mean relative humidity* (No. 9), or that of the *mean barometer* (No. 2), except that there appears to be an increase in the number of attacks of chorea with a fall of the mean relative humidity and barometer tracings; neither is much seen in comparing with it the mean daily range of the thermometer (No. 1), which shows the variableness in temperature of the different months; this is greatest in May, and least in December.

A resemblance begins to be apparent between the chorea tracing and the tracing of cloudy days (No. 7). The mean temperature tracing (No. 4) does not throw much light on the subject.

The tracings of the number of days on which rain or snow fell, and that showing the amount of rain or melted snow in inches, also show a slight resemblance to the chorea tracing. These lines are not shown on the table.

The probable cause of the resemblances noted becomes more apparent when the storm tracing is studied, because these meteorological factors may be considered as component parts of a storm.

In studying the storms, circles of varying radii were thus drawn around Philadelphia and the number of storm centres, as marked on the weather-bureau maps, counted in each, but the closest resemblance appeared to be with the number of storms passing within the four-hundred-mile circle, and for this reason it is the only tracing that I have placed on the table. In the former study the great resemblance between the chorea tracing and the storms within the seven-hundred-and-fifty mile circle was pointed out, but the result of the present study being more exact and more extended is the one to be most relied upon.

The small number of storms passing within the four-hundred-mile circle occurs in August, a rapid rise of the tracing then takes place until December and January are reached, then there is a slight fall in February, immediately followed by a rise to the highest point in March, after which there is an irregular fall until the low point in August is reached.

Any one looking at these two tracings, as shown in the table, will have to acknowledge that the marked resemblance which exists is more than accidental, but to decide which factor in this complex disturbance, which we call a storm, is the baneful one, or in what manner it acts, is difficult.

Let us now turn to another disease, and study its tracing, and for this purpose acute articular rheumatism has been selected on account of its well-recognized relation to chorea.

November shows the fewest attacks, viz.: twenty-four, or 5.1 per cent.; from this point the tracing rapidly rises until January, with fifty attacks (10.7 per cent.), is reached, then falls slightly in February and March, to rise to its greatest height in April, eighty-one attacks (17.3 per cent.); from this point it rapidly falls to thirty-two attacks in June (6.8 per cent.), and then gradually and slightly irregularly falls to its lowest point in November.

While the chorea tracing shows a strong tendency to keep pace, month by month, with the irregularities of the storm tracing, that of rheumatism, while also strongly resembling the latter, may be seen to be exactly *one month later*, looking as if the effect of the meteorological changes was immediate in the case of chorea (as it is now acknowledged to be in some varieties of neuralgia), and preparatory only in the case of rheumatism.

From the close relationship known to exist between these two diseases, one might be led to suppose that the rheumatism tracing would precede, or at least accompany, that of chorea, instead of following it, as this study seems to show.

In Zurich, according to Lebert, as quoted by Senator in Ziemssen's Cyclopædia, out of two hundred and twenty cases of rheumatism during the years 1853 to 1859, the highest percentage occurred in January, 9.5 per cent., and in April, 13.3 per cent., agreeing exactly with the months of highest percentage in this city. A study of the storm records abroad, and their relations to rheumatism and corea, would be interesting, and probably explain the seeming diversity of opinion.

Many of the statistics of chorea are of but little value in this connection, as the months of application for treatment, and not the months of onset, are given.

The reports of observers in different countries do not agree as to the season of the year in which chorea is most prevalent.

Angel Money (*Brain*, 1882, p. 512) reports the *months of application for treatment* of the two hundred and twenty four cases to University College Hospital and to the Hospital for Sick Children. The greatest number occurred in January, viz., thirty cases, and the smallest number in July, August, and September, viz., eleven cases each. Arranged according to seasons, spring shows seventy, summer forty-seven, autumn forty-four, and winter sixty-three.

Germain Sée (*De la Chorée*, Paris, 1850, p. 460) is the authority for the statement that, in France, chorea is principally observed in the autumn and winter. My cases, arranged according to seasons, show the following order: Spring one hundred and fifty-two, summer one hundred and twenty, autumn sixty-four, winter one hundred and one.

A comparison of the rheumatism tracing of this table with the varying states of the weather, leaving out the storm records, does not explain the reason of the highest point in April. That month shows the lowest mean relative humidity, and also the lowest barometer; but, in opposition to this, the next highest month, viz., January, gives the highest mean relative humidity and the highest barometer. The temperature in April averages a little over 50°, and January about 32°. The most changeable month is May, so that this cannot be the explanation.

It may be well to explain that the mean relative humidity does

not show the *mean actual amount* of moisture continued in the air, but the *mean per cent.* of the moisture that could be held in suspension at the *mean temperature* of each month ; the amount representing complete saturation being indicated as one hundred.

Thus a given bulk of air at a low temperature, if saturated, and ready to drop its moisture, will become dry, if the temperature is raised, although it still holds the same amount of moisture.

This study may be considered as forging one more link in the obscure chain which binds together these two dissimilar affections.

Dr. WHARTON SINKLER said that this paper was valuable and interesting because it showed the close relation between chorea and rheumatism, which has been denied by some authors. For instance, Sturgis had two hundred and nineteen cases of chorea in the Hospital for Sick Children in London, of which number twenty per cent. also had rheumatism ; but he did not attach much importance to this connection, because, as he stated, fifteen per cent. of all children have rheumatism. Other writers have expressed the same views. This paper seemed to prove the relationship between the storm centres and attacks of rheumatism and chorea.

Dr. G. E. DE SCHWEINITZ reported

Eight Cases of Tobacco Amblyopia ; One Complicated with Disease of the Spinal Cord.

Since 1854, when Mackenzie showed that defective vision might be produced by tobacco-smoking, and since, ten years later, when the subject was renewed and enlarged by the observations of Hutchinson, tobacco amblyopia has become one of the well-recognized facts of ophthalmological science. As is well known, progressive and usually equal failure of vision, unimproved by optical therapeutics ; a feeling of wretchedness on the part of the patient ; an ill-defined ophthalmoscopic picture ; and a symmetrical, oval scotoma in the fields of vision, lying between the fixing point and the blind spot, and often including both, in which the perception of green and red is especially defective, constitute the marked symptoms of this affection.

CASE I.—A. G., æt. forty-nine, a book-keeper, presented himself in the University Hospital, because his vision was failing and because of sleepless nights. He was a large, dark-haired man, with a restless, irritable disposition. Vision in the R. E. $\frac{20}{C}$,

L. E. $\frac{20}{CL}$. Pupils about normal in size and reaction. Vision best in the early morning. Both optic nerves slightly hazy and gray in their deeper layers. The refraction H., but vision unimproved by the correcting glass. The fields of vision for white and red about normal. Between fixation—*i. e.*, the macula and the blind point, *i. e.*, the optic-nerve entrance—there was an oval, slightly sloping, relative scotoma, including both In this area green appeared dirty-white, and red could not be distinguished as such. For twenty years he had smoked excessively, averaging two pounds of Cavendish a month. He drank one pint of spirits a day. He was ordered to stop tobacco absolutely and to diminish his daily quantity of spirits. In a month his vision had improved to $\frac{20}{XXX}$ in each eye, and with his correcting glass he could read with fair ability.

CASE 2.—B. E., æt. fifty-three, laborer, came to the University Hospital for failing vision. He was a small, dark-skinned man, with fairly steady nerves. Vision, R. E., $_{L}\frac{20}{XXX}$; L. E., $\frac{20}{L}$. Pupils normal in size and reaction. Vision worse in a bright light. Both optic nerves were gray-red, and the veins full and dark. The fields of vision showed no marked peripheral change, but perfectly characteristic oval scotomata were present. This man had smoked two pounds of "hard tobacco" a month for many years, and was accustomed to drink one quart of spirits a week. The usual order to stop tobacco was given. Sufficient time has not yet elapsed to prophesy how successful the case will be.

CASE 3.—J. M., æt. thirty-five, a mechanic, came to the University Hospital because of defective vision, headache, and great nervousness. He was a short, thick-set man, tremulous, and despondent. Pupils exhibited no abnormalities. Vision, R. E., $\frac{20}{LXXX}$; L. E., $\frac{20}{C}$. The nasal edges of the optic nerves were hazy, and the temporal sides gray; arteries rather small. The fields for white and red not far from normal, those for red being slightly contracted. In the right eye, from fixation, and including it and passing outward, there was an oval scotoma for red and green, which reached to the blind spot, but did not embrace it. In the left eye, a similar, rather more irregular scotoma. Up and out from the fixing point a small area of the scotome was absolute. This man for fifteen years had smoked about six ounces of coarse tobacco a week, and occasionally cigars and cigarettes. He denied taking any thing to drink, except a little beer or ale. In eight days his vision in right eye had become $\frac{20}{XXX}$, and in the left eye $\frac{20}{LX}$. He then passed from observation.

CASE 4.—Ida J., æt. twenty-seven, came to the University Hospital because of failing vision and great "nervousness." She was a finely formed brunette, of rather pallid countenance and excitable temperament. Vision in R. E. all of $\frac{20}{XL}$, some of $\frac{20}{XXX}$, The L. E. had been removed years ago for disease. Pupil responded freely to the change of light and shade, and was about medium size. The optic nerve was pallid, edges clear, arteries rather small. The field of vision both for white and red was somewhat contracted, and surrounding fixation in a band-like manner, but not extending to either side, there was a relative scotoma, in which this color appeared "brownish" or rather "dull-colored." The girl used no tobacco, but had worked for some years in a tobacco factory. On this account she was advised to get other employment, and also to take one twenty-fourth of a grain of sulphate of strychnia three times a day. In two weeks the scotoma was no longer demonstrable, and in about one month the vision had returned to normal, and is now above normal.

CASE 5.—H. H., æt. fifty-eight, a shoemaker, came to the dispensary of the University Hospital, because his eyesight was so poor he could no longer see to do his work. He was a medium-sized man, with dark hair, beginning to turn gray, tremulous, and easily startled. Vision in the R. E. $\frac{20}{CC}$; the same in the L. E. The pupils were small, and failed to respond to the changes of light and shade, although they contracted in the efforts of accommodation. The optic nerves showed a large, shallow excavation, widening of the scleral ring all around, and were of a distinctly gray color. There were no changes in the central circulation. The fields of vision showed slight contraction for white, distinct limitation for colors, and oval scotomata from fixation to blind spot, including both. The man smoked three ounces of tobacco weekly and drank whiskey, but not to excess. Further examination developed the fact that the patellar reflexes were absent, that with closed eyes the upright position was maintained with difficulty, and on attempting to walk, the gait became staggering, if not positively ataxic, and that preceding the time of failing vision there was a history of diplopia. These symptoms, together with the state of the pupils, rendered the presence of sclerosis in the posterior columns of the cord a fair inference. There was no history of syphilis. Examination of the urine revealed it normal. The patient was ordered to stop the use of

tobacco, and take iodide of potassium. Although two months have elapsed since he applied for treatment, no improvement in his vision has taken place, nor have the scotomata changed or lessened.

CASE 6.—W. H , æt. fifty-eight, a farmer, applied for treatment in the University Hospital, because of a "cloud" before his vision and inability to do even ordinary coarse eye-work. He was a well-preserved, large-framed man, with iron-gray hair, and presented no noteworthy unsteadiness of the nervous system. Vision in the R. E. counts fingers at two feet, in the L. E. $\frac{10}{cc}$. The pupils were small ; they responded to the changes of light and shade, and also to the efforts of accommodation. Both optic nerves were small, surrounded by a halo, and their edges and the surrounding retina hazy. The disks were gray-red in color. In the neighborhood of each macula were slight patches of choroiditis. In the R. E. the field of vision for white was contracted, but not markedly so, and the fields for blue and red showed similar slight limitations. Between fixation and the blind spot, a large irregular scotoma for all colors. In the area surrounding the fixation, and for a small distance toward the blind spot, the loss of color was absolute ; from there to blind spot relative. In the L. E., the field for white was limited above, and the fields for red and blue irregular and constricted. A similar scotoma, absolute near its centre, extended between fixation and blind spot, and included both. This man smoked four to six cigars daily, and drank "an occasional glass of whiskey." The patella reflexes were prompt, and greater on the right than the left side. His urine has as yet not been examined.

CASES 7 and 8.—These cases occurred in private practice, and were both men over forty years of age, of active business habits, who complained of indistinct vision. In both the vision was not far from normal, viz., $\frac{15}{xx}$ and in both the eye-grounds presented no abnormal features. There were no true scotomata, only slight dimness of the perception of green and red between the macula and the optic nerve. These men were inveterate smokers. The difficulty of vision disappeared in one patient when he ceased using tobacco, the other declined to abandon his habit. While tobacco was evidently producing deleterious effects, these cases can scarcely be classified as true instances of tobacco amblyopia.

In three of the cases reported, the ophthalmoscopic examina-

tion revealed "hazy disks" either with or without full, dark retinal veins (Cases 1, 2, and 3); in two, if the last record be included in the list, there were no fundus changes (Cases 7 and 8). In one the optic nerve was pallid, the arteries rather small, perhaps the appearance of beginning atrophy (Case 4), and in one the nerve showed evidences of gray atrophy (Case 5), while in another the appearances were those of a subsiding neuritis (Case 6). Three of these cases are perfectly typical instances of tobacco amblyopia (Cases 1, 2, and 3); and two of them, if not characteristic cases, at least showed the result of tobacco impression (Cases 7 and 8). Case 4 is an atypical one, inasmuch as the scotoma surrounded the fixation but did not include it or pass to either side, and, furthermore, is noteworthy because of the fact that the tobacco found its entrance into the system by absorption through the skin. This woman worked in tobacco, but did not use it. Case 5 may be one of those rare instances in which a central scotoma appears with a spinal atrophy of the optic nerve, but inasmuch as the man used tobacco, although moderately, its influence cannot be excluded. An almost precisely similar case is recorded by Gowers (*Med. Ophthalmoscopy*, p. 111). In Case 6 it is doubtful if the use of tobacco produced the scotoma, although, as in the last instance, the patient was accustomed to its use, and hence its influence cannot be excluded. The whole subject of tobacco amblyopia has so often and so thoroughly been discussed, notably by Mr. Nettleship ("St. Thomas's Hospital Reports," 1878), that it is useless to do more than report these cases and point out the features that are interesting.

Dr. B. ALEX. RANDALL remarked that some ophthalmologists disbelieve in the existence of tobacco amblyopia. Very few cases have been put on record in which the influence of alcohol could be wholly excluded; but the combination of alcohol and tobacco is certainly a serious disturbing element in a number of cases. This affection is constantly coming under notice—occurring some eight or ten times in a thousand cases. While the scotoma is usually of an oval form, he had seen it distinctly annular (as in Case 4) in a case typical in other respects.

It has recently been claimed that the absolute quantity of nicotine used does not bear that relation to amblyopia that we should infer, and that those who smoke the finest cigars are more liable to this affection than those who smoke the poorest and strongest.

Dr. JAS. HENDRIE LLOYD asked if this affection were due to neuritis or to some disturbance of the intra-ocular circulation? The cases reported suggested an interesting analogy between tobacco amblyopia and peripheral neuritis, especially that form due to toxic agents. Is this a form of toxic neuritis of a special nerve?

Dr. J. MADISON TAYLOR, referring to one of the cases reported, asked if it were not possible to have a powerful effect from the absorption of tobacco by the skin and lungs? In children working or living in tobacco shops, he had, in several instances, seen profound disturbance of the heart as the result of absorption.

Dr. LOUIS J. LAUTENBACH remarked that there had always been an element of doubt about these cases, and that of late this had become stronger. There are cases reported in which alcohol has been stopped, while the tobacco has been continued, and the patients have gotten well. Dr. Minor, of Memphis, reports eight such cases. In addition to the discontinuance of alcohol, he administered in these cases either iodide of potassium or strychnia. Nettleship, Foerster, and a few others, have succeeded in curing some of these amblyopia cases by the discontinuance of tobacco alone. About two years ago, at the Philadelphia Eye and Ear Dispensary, he had experimented in this matter, and had cured a number of cases by removing alcohol, allowing them to use tobacco, without the use of any medicines. The same treatment had resulted satisfactorily in two cases treated in private practice. One case was that of a professional base-ball player, who was in the habit of drinking thirty-five or more glasses of whiskey, and smoking from eighteen to twenty-five cigars a day. When examined, both eyes gave V.$= \frac{3}{LX}$(?). The patient was directed to stop the use of strong liquors, but to keep on with his tobacco. In four days later V.$= \frac{3}{XXXV}$. Vision continued to improve, although no medicines were used.

The condition which causes this amblyopia may be a slight grade of retro-ocular neuritis, involving only the macula lutea fibres, which may be induced not only by tobacco but also by alcohol; lead occasioning it in some few cases. Depressed conditions of the system may probably cause it. This amblyopia with central scotoma, is coming to be looked upon not as a special symptom of tobacco abuse, but rather as an indication of the depression of the general system which may be brought about by various poisons.

Dr. WHARTON SINKLER referred to a paper published in one of the New York journals by Drs. Roosa and Ely, giving the results of the examination of a large number of workers in cigar factories, many of those examined smoking a great deal. Only a very small proportion of these cases was found to present any defect of vision or any disorder of the nerve He asked if in the experience of other members they had found that a large proportion of nervous diseases were attributed to tobacco. He saw but few patients at his clinic at the Infirmary for Nervous Diseases with nervous disorders which could be directly attributed to the use of tobacco.

A point to be remembered in the consideration of this subject is that a person who takes alcohol will use more tobacco than one who does not. If he stops his alcohol he will take less tobacco.

Dr. MORRIS J. LEWIS stated that in studying the subject of writer's cramp, he had found that in the majority of cases the use of tobacco increased the difficulty. A few out of forty-three cases said that it did no harm, and three said that it was of service.

Dr. FRANCIS X. DERCUM asked if those who used tea or coffee to excess, especially tea-testers, presented symptoms analogous to those presumably caused by tobacco and alcohol.

Dr. B. ALEX. RANDALL said that there were some cases in which alcohol *could* be entirely excluded in the causation of this affection. As to the pathology of the trouble, it is interesting to note that he knew of no case on record of true neuritis in the sense of any thing like a choked disk, which had any apparent relation to tobacco abuse ; although such cases are not rare in lead poisoning and other toxæmic conditions. In all cases of actual neuritis of the optic nerve, as in inflammation of other nerves, strychnia has to be carefully avoided in the acute stage. In most cases reported, the use of strychnia had no injurious effect, and in fact caused, or was followed by, improvement. In some cases where strychnia has been given and the alcohol and tobacco both continued, recovery has taken place. The amount of poison required to produce a certain effect probably depends upon the constitution and condition of the patient. If the general condition is brought up, a given amount of the poison no longer has the same effect.

Dr. CHARLES K. MILLS, referring to the question of Dr. Sinkler, stated that his experience agreed with that of Dr. Sinkler, that

nervous disorders are rarely attributable to the use of tobacco, although some cases of tremor and general nervousness, and a few other affections, undoubtedly originate in this way.

Dr. GEORGE E. DE SCHWEINITZ, in closing the discussion, said that he believed that in these cases there was an axial neuritis. He has had no experience with the poisonous effects of tea and coffee. Some of the cases reported had recovered, although the free use of spirits was continued. One of the patients continues to drink a pint of whiskey a day, and yet his vision has remained normal since the discontinuance of tobacco. The only way to reach a satisfactory explanation of this subject is to continue the experiments.

One object in presenting this series of cases was to call attention to the importance of a careful study of the field in nervous cases. If this were done more frequently, many interesting discoveries would probably be made.

Referring to the question of Dr. Taylor, he said that tobacco is freely absorbed through the skin, but inasmuch as it is rare to find scotomata under such circumstances, he had reported the case.

Dr. FRANCIS DERCUM reported

A Case of Unusual Paresis of the Forearms.

Dr. CHARLES K. MILLS referred to three cases which had been reported a few years ago, by Dr. Kerlin and himself. These cases were studied at the School for Feeble-minded Children at Elwyn. The patients were brothers, and in them was a marked combination of pseudo-hypertrophy and muscular atrophy. One of the boys, who has since died, presented in the calves the typical appearance of pseudo-hypertrophy, and also in one or two other isolated portions of the body. In the back, however, were all the distinctive signs of progressive muscular atrophy. In the second case the changes were principally those of progressive muscular atrophy. In the third there was a combination of the two conditions. Why in one case we get the pseudo-hypertrophic form, and in another the atrophic form, he was not prepared to say. He believed that in all cases there are cord and nerve lesions, but he did not know which of these was primary. In the treatment of such cases, that pursued in the present instance would seem to offer the most hope. He was in the habit of putting these cases on strychnia, iron, cod-liver oil, and similar tonic and nutrient

remedies. He had never seen much benefit from local treatment, but if myositis was present, he thought it worthy of a trial.

Dr. JAMES HENDRIE LLOYD said, with reference to the age at which this affection may develop, that he had recently seen a case in which a married woman, thirty years of age, who had been under treatment on several occasions without a positive diagnosis having been made, returned after an interval of four months with hypertrophy of the muscles of the calf, lordosis, and other characteristics of pseudo-muscular hypertrophy. This was the first case which he had seen in which the disease had apparently occurred as a primary disease in an adult with no hereditary tendency.

Dr. WILLIAM OSLER remarked that some observations had been made by an English physician with reference to the atrophy which follows great use of the muscles. After examining the microscopic specimens, he agreed with Dr. Dercum in regard to the condition present in this case. There was no essential difference between the atrophic and the pseudo-hypertrophic form of the disease, and both may be present in the same case.

Dr. WILLIAM OSLER exhibited the following specimens :

Pachymeningitis.

J. K., was admitted to the drunkards' ward of the Philadelphia Hospital, on August 26th, with delirium tremens, and died September 4th. I did not see him during life, but from the account given by the resident physician he appears to have had an ordinary attack of delirium tremens without any paralysis, local or general. He was not in a condition to give a satisfactory account of himself. Death took place from exhaustion. The post-mortem showed an extensive pachymeningitis of the right half of the dura mater. The specimen presents layers of exudation two lines in thickness over the central portion of the dura, gradually becoming thinner toward the tentorium or the orbital surface. In these regions there was a delicate uniform sheeting on the dura mater, and no hemorrhage had as yet taken place ; but on examination with a low-power lens there could readily be seen a plexus of wide vessels. In the central part there were several layers of altered blood, alternating with colorless firmer exudation. The surface of the pia mater of this hemisphere was a little stained, but there had evidently not been any pressure upon the convolutions. There were no other lesions.

Phthisis; Tubercular Meningitis; Aphasia.

J. McD., æt. thirty-six, laborer, was admitted to the Philadelphial Hospital, June 5th, with phthisis. There was nothing special in his family history; five of his brothers and sisters had died from causes unknown to him. He had been ill for over eighteen months with cough, fever, chills, and sweats. Had brought up blood and had had severe diarrhœa. When I saw him first, on September 4th, there were signs of extensive disease in both lungs. Ten days before he had two convulsions, after which he had some difficulty in talking. On the 4th he looked bright and intelligent, the pupils were dilated and there was no paralysis. Expressed himself with difficulty; when asked his name, said *Joseph Mac*, but could not say the *Donald*. After several attempts and a good deal of worry, he did so, and seemed much pleased. Some words he said clearly, others he could not. Thus he could read the letters of the word *shaken*, but could not pronounce it. He was delirious at times during the next two weeks, and one night he got from the ward on the fire-escape. The aphasic condition improved somewhat. The eye-grounds were examined twice, with negative results. At the time of my visit on September 10th he had an epileptic seizure, strong spasm of respiratory muscles, and great lividity; tonic spasms of arms, gradually relaxing and becoming choreic; facial muscles in violent action; face drawn to the left, eyes to the right (strongly) and upward. He became unconscious the next day and died on the 14th. The autopsy showed extensive disease of the lungs. The brain was large; at the base the membrane was thickened and infiltrated and tubercles existed along the vessels. On exposing the Sylvian fissures, the pia on the left side covering the insula was very thick and studded with tubercles the size of a pin's head. The process was most advanced upon the three or four posterior gyri of the insula. The gray matter was reddened and soft. The third left frontal convolution was not specially involved; there were a few isolated tubercles on the pia, but the membrane was not thickened. In the right Sylvian fissure there were tubercles scattered along the vessels, and the arteries of the anterior perforated spaces presented many bead-like and oval swellings. The ventricles were dilated and their walls softened.

Embolism of Right Middle Cerebral Artery; Chronic Nephritis.

J. W., laborer, æt. forty-nine, was admitted to the Philadelphia Hospital Oct. 7, 1886. No special history could be obtained, as

he was very dull and heavy. When I saw him on the 9th he was apathetic, answered questions with difficulty. The face was anæmic, and he had a decidedly renal look. The pulse was slow and hard, sixty to the minute. No increased heart dulness, possibly a murmur at apex, but it was not at all distinct, and at base very ringing. There were albumen and casts—hyaline. The case was regarded as one of chronic interstitial nephritis.

On the morning of the 12th he had a hemiplegic attack. I found him at 1 o'clock P. M., comatose, with puffing respiration and complete left hemiplegia, including face. Right pupil larger than the left. He died during the night.

The autopsy showed cirrhotic kidneys, slight atheroma in large vessels, moderate hypertrophy of left ventricle. The mitral valve presented recent vegetations, three in number, on the auricular face of segments, freely movable, and on the posterior flap, a flattened area, looking as if eroded. The right hemisphere of the brain looked fuller than the left, and the vessels were not so full, particularly in the central region. The vessels of the circle of Willis were denuded *in situ*, and the right middle cerebral just beyond the first two branches (temporal) was found plugged with the firm embolism here shown. It was very evident before opening the vessel, as it showed grayish-white throughout the coat, while on either side of it were dark clots, and the branches beyond it were very small. The embolus is as firmly adherent, and had a small pale clot on its proximal, and a firm dark one on its distal side. Examination showed it to be an endocardial vegetation, and it doubtless came from the rough spot on the posterior segment. The convolutions of the central portions of the hemisphere were swollen, and œdematous, particularly the gray matter, and softer. There was a light gray-yellow tint, but there had not been time for much change. The right anterior cerebral was greatly enlarged, three times as big as the left, doubtless a compensatory process.

Dr. CHARLES K. MILLS remarked that cases of pachymeningitis hemorrhagica were somewhat common in the Philadelphia Hospital. He had met with a comparatively large number of cases in making autopsies in that institution. A form of hemiplegia met with in the wards of this hospital is undoubtedly due to pachymeningitis hemorrhagica. These cases make partial recoveries, and then again break down, and at the autopsy is found the condition exhibited to-night. He had reported one

case five years ago, with a number of other cases, in the English journal *Brain*. This condition is sometimes mistaken for embolism, hemorrhage, or some other form of disease producing hemiplegia.

In some of the cases he had seen there had been distinct naked-eye flattening of the convolutions of the motor region, which he supposed accounted for this peculiar, variable form of hemiplegia.

Editorial Notes and Miscellany.

WITH this December issue, the thirteenth volume of the JOURNAL (the first under the exclusive management of the present editor) is brought to a close. It was with some misgivings that the editor undertook at the beginning of the year the task, not only of editing a journal which had fallen into arrears, but of changing the form of that journal from a quarterly to a monthly. The event has proved that the difficulties of the task were overrated; for the JOURNAL has now been brought up to date, and instead of there being a lack of material, the editor has in every instance been puzzled to know how to keep the monthly issue within the prescribed limit of sixty-four pages. An American neurological MONTHLY is, therefore, no longer a bold experiment, but a marked success!

This success is due in a small measure only to the personal efforts of the editor; the chief credit should be given to the many physicians and other scientific men who have so generously responded to his call for aid in making this JOURNAL a credit to American research. It is certainly a matter of congratulation that the "List of Contributors" appended to the present volume contains so many familiar names, and that there are but very few prominent specialists in the country who have not, either contributed to this volume of the JOURNAL, or consented to become active collaborators in the future. The general excellence of the articles published in the volume for 1886 has unquestionably helped to make this the representative journal of American neurology. Apart from the original articles, we claim that the Periscope abstracts and the book reviews have been most carefully written, and that the work done in these two departments of the JOURNAL reflects the chief advances made in neurological science during the past year.

While thanking the friends of the JOURNAL for their kind assistance, the editor begs all of them to seek to extend the influence of the JOURNAL in and beyond their own immediate circles. The JOURNAL is not intended for neurologists only; the requirements of other specialists and of the general practitioner are thoroughly considered and to the fullest extent that a high scientific standard will allow.

In order not unduly to increase the size of the volume (which should not exceed 768 pages), the December issue contains a lesser number of pages than any of its predecessors. Much material that was intended for this number has been kept over for the first number of the new year, which will appear on or before the first of January next.

The announcement is herewith made that THE JOURNAL OF NERVOUS AND MENTAL DISEASE will hereafter be issued under the imprint of J. H. Vail & Co. (21 Astor Place, New York City). Subscriptions for the coming volume, all exchanges, books for review, etc., should be forwarded to the new publishers. Contributions to the JOURNAL, reprints of articles to be reviewed, and all editorial communications should be addressed to the editor, Dr. B. Sachs, at 30 West 59th Street, New York City.