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WAVERLEY RESEARCHES IN THE PATHOLOGY OF THE FEEBLE-MINDED.

(Research Series. Cases I to X.)

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PREFACE.

We present herein the first-fruits of an orienting study of some of the more fundamental problems of feeble-mindedness. Rarely has the problem of feeble-mindedness been attacked fundamentally for its own sake. Yet in recent years and in the immediate present the representatives of numerous and varied interests clamor for light upon what appear the limiting problems of their special fields. But what can the workers in feeble-mindedness as such and for its own sake say to

a) the *educators*, responsible for "laggards," truants, "terrors," "exceptionals," "ungraded," specially graded, retarded, and backward children;

b) the *court authorities*, to whom the educators' problems in part drift and figure as psychopathic cases, defective delinquents, prostitutes, and the like;

c) the *social workers*, sometimes over-ready to tar all these cases with the same brush as "mental";

d) the *eugenists*, who either as Galtonians or as Mendelians, often tend to treat as units and thought-counters all sorts of feeble-mindedness and perhaps even epilepsy under a single caption;

e) the generalizing *biologists*, with their counsel of perfection in the guise of sterilization of the potential parents of more feeble-minded;

f) the *syphilographers* and *antialcoholists*, who need facts for their propagandas and at any rate must not universalize what facts are obtainable;

g) the *legislators* and alert guardians of the public purse, who are promised, in lieu of impractical sterilization, a program of segregation — What can the workers in feeble-mindedness offer to all these inquirers?

Satisfied for the moment with an astonishing and unfamiliar publicity, the special workers in feeble-mindedness shortly turn to fundamentals. Assured in our own minds that not all school "problems" are feeble-minded, that not all delinquents are defective, that not all feeble-minded are institutional subjects, that Pearson and Davenport should not lump and unify so medically obvious a *pot-pourri* as feeble-mindedness, that sterilization is a program whose first physiological lines have not been laid down, that syphilis and alcohol have not had their shares demarcated in the matter of feeble-mindedness,

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and that legislators rush in where specialists find no footway, we run to our books. Mierzejewski, Bourneville, Hammarberg, Wilmarth, Tredgold, Bolton, these and others have done their part, perhaps most notably Bourneville. Even Germany has little to offer in the fundamentals, save perhaps the suggestions of the lamented Alzheimer. The great foundations have neglected feeble-mindedness (there is grim humor in the fact that their proposals will often come to naught through the existence of these very feeble-minded whom the great foundations neglect!).

Yet it is not merely the present-day neglect of fundamentals in feeble-mindedness that justifies our undertaking. Other sciences have advanced in the meantime. What would not the modern ideas of glandular dysfunction have meant to Bourneville? How Hammarberg would have rejoiced had the modern work of the cortex-topographers enabled him to omit a large part of his monograph and rush on to deeper studies in feeble-mindedness? How Wilmarth would have enjoyed the resources of a modern laboratory!

These considerations have been set forth more extensively, though still too briefly, in Article I. Here we wish to say that we are minded to present a series of studies of the anatomy, and especially of the brain anatomy, of the feeble-minded (including idiots, imbeciles, and a variety of subnormals under that general term) to which we set the provisional terminus of fifty cases. Until we have presented at least fifty cases from an identical point of view, we feel that we shall hardly be warranted in drawing conclusions. Still, epicritical remarks will be in order with each group of ten cases presented. We plan to present perhaps ten cases a year. The great foundations aforesaid, which have so neglected the topic, might of course speed up the work appreciably; yet it must be granted that every case of feeble-mindedness is so much a unique, not to say Argus-eyed, a problem that speeding-up by funds alone is hardly a practical policy. More living brains are needed to work out the problems of the brains of feeble-minded, whether living or dead — but *cadit quaestio, that* is the urgent lack of mental hygiene as a whole.

We must accord due credit and extend our thanks to the various clinical and post mortem observers who have rendered the work possible, especially to the Waverley School workers, to Dr. Myrtelle M. Canavan of the Pathological Service of the Massachusetts Commission on Mental Diseases, who has performed most of the recent autopsies, and to Dr. Annie E. Taft, Custodian of the Neuropathological Collection, Harvard Medical School, who has been charged with much of the brain analysis. We should not omit to call attention to the excellence of the photographic work of Mr. Herbert W. Taylor, Of great importance also is the work of the technicians, among whom we may mention Miss Ellen R. Scott and Miss Mae Cameron.

Perhaps the most interesting endeavor in the present volume is the attempt to match

brain complexity with mental capacity. To say nothing of the mind-and-brain problem (the less mind, the less brain, and *vice versa*, where "less" is taken not *too* quantitatively), we feel that the whole problem of "mental tests," now *sub judice*, needs anatomical support and anatomical critique. Various authorities glibly discard all "tests"—mind cannot be measured, that is an *à priori* certainty! Other persons, chiefly of the bureaucratizing group, would let fly at the institutions cases "feeble-minded" by a Binet-Simon fraction, particularly if they be antisocial and affected by symptoms never considered by Binet and Simon. The epicritical remarks of the present volume, provisional in character, are therefore levelled at the question of *matching testable mind with measurable brain*. The situation appears on the whole rather favorable. On the basis of the clinical and anatomical details of Article II, Article III goes on to the more doubtful matter of evaluation. The skeptical may confine their attentions to Articles I and II.

A second volume, dealing with Cases XI to XX (including also "Mongolians"), is within hail. Therein we hope to deal still further with the major problems herein touched, such as

a) the correlation of mental and cerebral measurements.

b) the theoretically preventable group, (e. g. syphilitic, post-poliomyelitic).

The grant for the investigation and its publication is from a private endowment of the Massachusetts School for the Feeble-minded. A grant for publication of suitable and numerous plates has been received from Dr. W. N. Bullard, whom we would like to thank in fullest measure.

WALTER E. FERNALD,
E. E. SOUTHARD,
Editors.

I.

GENERAL ASPECTS OF THE BRAIN ANATOMY OF THE FEEBLE-MINDED.

E. E. Southard.

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GENERAL ASPECTS OF THE BRAIN ANATOMY OF THE FEEBLE-MINDED.

E. E. Southard.

Abstract.— Small amount of intensive anatomical work available in the literature (Bourneville, Hammarberg, Tredgold, Bolton). Research emphasis on other aspects (heredity, mental tests, social service, education).

Situation in the biology of feeble-mindedness (Ballantyne). The question whether any feeble-minded persons have normal-looking brains. Relative absence of work on the brains of feeble-minded of higher grades. Modern knowledge of cortex topography.

Tredgold's distinction of primary and secondary amentia ("germinal blight" versus "arrest"). Classification of W. W. Ireland. Exogenous nature of numerous cases (intrauterine lesions). Tredgold's view that germinal variations are pathological and not spontaneous. The suggestion that germ cells may be poisoned. Brain findings in exogenous cases.

Anatomical classification, Bourneville, 1889; Bourneville's researches. Hammarberg's report on 9 cases (psychic defects connected with defective nerve cells in the cerebral cortex). Idiocy thought to be due to developmental check in later foetal life. Marked weak-mindedness thought to be due to arrest of development in a stage normal for the first years of life. A third group with focal factors of inhibition occurring during the first year of life.

Most of Hammarberg's work devoted to the microscopy of the *normal* cortex. Necessity of recognizing certain shrinkage processes as artificial. Hammarberg's claim that disorderly arrangement of cortical cells is also found in normal material (undeveloped cells frequently taken for atrophic ones).

Summary of Hammarberg's work with 4 cases of idiocy, 2 of marked weak-mindedness, 3 of slighter degrees of weak-mindedness.

Tredgold's conclusion that the essential basis of primary amentia is cellular, due to an imperfect or arrested development of cerebral neurones. Tredgold's belief that irregular arrangement of cells is characteristic of feeble-mindedness material.

Situation in the anatomy of feeble-mindedness as presented at the Medical Congress in Paris in 1900.

Work of Mierzejewski (anatomical lesions are developmental deviations). Mierzejewski's special group of cases in which the gray matter is disproportionately large as compared with the white matter. Heterotopia.

Classification of Shuttleworth and Fletcher-Beach; microcephaly probably not caused by premature synostosis of cranial sutures. Congenital hydrocephalus extends the lateral ventricles lengthwise, acquired vertically and transversely (Meynert); scaphocephaly; Mongolian idiocy; other groups.

Syphilis not a common cause of feeble-mindedness; juvenile general paresis as first observed by Clouston; traumatic cases; so-called hypertrophy.

American literature. Wilmarth's study. 25 cases in 100 yielded no brain lesions to gross analysis. Quotations from Wilmarth's conclusions.

Problems suggested by the literature of the brain anatomy of the feeble-minded.

We present herewith the first section of the Waverley Researches in the Anatomy of the Feeble-Minded begun in 1913 under the auspices of the Massachusetts School for the Feeble-Minded. This first section of Researches contains

I, a general introduction to the topic of brain anatomy of feeble-mindedness,

II, a particular description of ten instances of feeble-mindedness of various grades, together with autopsy and microscopic findings, and

III, attempts at correlating the brain anatomy and histology with clinical and especially with psychometric (mental test) findings. Further studies are under contemplation with the object of gradually enlarging the brain series, from which, when it shall have contained say 50 brains, we may be able to draw sound conclusions.

Let us note at the outset our surprise at the extraordinary neglect into which this topic has fallen. Bourneville, Hammarberg, Wilmarth, are the older names of workers contributing carefully sifted cases studied in the interest of feeble-mindedness. Most authors deal with feeble-mindedness as a stalking-horse in some other scientific pursuit, teratological, embryological, anatomical, histological, etc. We claim for the present work that, despite numerous theoretical and ancillary interests, *we have studied feeble-mindedness from the standpoint of feeble-mindedness.* In particular we have tried to place our cases in a tentative order of mental strength, correlating brain appearances with mental tests. Our purpose has been at least as much to evaluate the mental tests by throwing their results against the brain pictures as it might have been to evaluate the brain pictures in the light of mental tests. We may anticipate our conclusions so far as to say that, roughly speaking, the architectonic order in which the brains arranged themselves corresponds to the psychometric order indicated by the mental tests.

Thus, we must emphasize, we are not here studying the brain for the brain's sake, or the mental tests for their sake alone. We have no eugenic, economic or social interests paramount in the present inquiry.

This first series of ten cases is intended as an orienting and introductory series to a more extended study which at present contemplates the examination by modern methods of 40 more cases, to make up a series of 50, from which series it may be safe to draw provisional conclusions. The first series has accordingly been made to include all available material from several sources which was found to comport with certain necessities of modern research in feeble-mindedness.

One might readily get the impression that the literature would be found to contain enough properly examined material from which to draw conclusions. One would have in mind the Bicêtre reports of Bourneville, beginning in 1881 and continued at successive intervals for 20 years. One would have in mind the masterly work done in Sweden by

Hammarberg and published after his death at Upsala in 1895. Another source of information would be the work done by Dr. A. F. Tredgold under the London County Council early in this century and published in Mott's Archives of Neurology in 1903, embodied in Tredgold's book on Mental Deficiency, second edition, 1914.

Then one would find the work of J. S. Bolton "On the Histological Basis of Amentia and Dementia," published in the same volume of Archives of Neurology and later embodied in a more popularized form in Bolton's book "On the Brain in Health and Disease," London, 1914.

In the textbooks on feeble-mindedness, one would find numerous references to Italian work in anatomy. In various textbooks one would find references to considerable work on the anatomy of feeble-mindedness (see for example, the literary references in Ziehen's account of "Diseases of the Brain and Meninges in Childhood," 1912).

In point of fact, however, the anatomy of feeble-mindedness has not been attacked systematically over a period of years having due regard not only to clinical features, but to anatomical and histological features.

On the whole, the remarkable developments in education of the feeble-minded following the initial work of Séguin have developed almost independently of the anatomical, histological and physiological sides of the topic. With the developments of recent years a tremendous emphasis has begun to be laid upon the hereditary and eugenic aspects of feeble-mindedness, and a large effort has been expended upon certain applications of psychology to the mental testing of the feeble-minded. But neither the hereditarians nor the applied psychologists have particularly concerned themselves with the broader biological aspects of the whole topic. We must confess that on the whole, the broader biological aspects of feeble-mindedness have tended to be concealed in the mass of practical applications which the mental testing methods have unfolded and the web of promises spun by the eugenist. Accordingly we face a situation with the feeble-minded in which their education, their heredity, and the methods of their mental testing have absorbed interest. In the spring of 1915, however, the note of research in this field was struck at the National Conference of Charities and Correction, perhaps somewhat to the astonishment of the listeners, who have been far more interested in social service than in the more recondite aspects of research. However, educational research, eugenic research, research in applied psychology, and sociological research, all need effort on the biological basis. It seems as if this basis had been sadly and almost inconceivably neglected in the past.

What is the present situation, biologically speaking? The *biologist*, if asked the nature and measure of his interest in feeble-mindedness, would be apt to answer some-

what as follows: The institutions might supply us with a vast deal of material of the greatest interest from such a standpoint, for example, as that of Ballantyne, whose fundamental collection of material on "Antenatal Pathology and Hygiene" was published in 1902 and 1904 respectively. There should be much, the biologist might continue, of great interest from the standpoint of monstrosities, and the teratologist might be able to unravel any number of intricate problems of the mechanics of development from the anatomical material of institutions for the feeble-minded. He would have to consider not merely the immediate pathology of the antenatal period, but also the postponed effects which Ballantyne has emphasized, and not only the postponed effects in the neonatal period of life, but also its postponed effects upon still later life.

Ballantyne was able to determine important relations of antenatal pathology to no less than seventeen departments of science (see his figure 3, page 18). Of the considerations in Taruffi's *Storia della teratologia* would come an application, and the further development in studies of the idiot types with the methods available to modern research. For example, the microscopy of all types of terata, described in chapters 14–30 of book 3 of Ballantyne's volume on the Embryo would get a new importance if proper histological studies were carried out. But withal, the teratologist would be studying this material from the standpoint of teratology and tissue mechanisms, and the fate of the feeble-minded as such would not be visibly altered for the better in a long period of years.

The *pathologist* might see not only the importance of antenatal pathology in the direction of monstrosities, but he might be greatly interested in what Ballantyne has termed the intrusion of the antenatal factor in neonatal disease as shown by instances of intracranial traumatism and intranatal infections (ophthalmia neonatorum, etc.). The pathologist would also be interested in the neonatal infections, such as sepsis neonatorum, as also very particularly in what Ballantyne has called the disturbed antenatal readjustments, such as icterus neonatorum, and the like. Here again the interest in the anatomy of the feeble-minded would be an interest derived *ab extra*. The pathologist must go to the specialist in feeble-mindedness for his material and carry his interest to that material. Hence from the pathological side it can hardly be hoped that there will be a tremendous flow of new work on this topic.

Again, the *embryologist* must be interested in the subject, as is clear throughout both volumes of Ballantyne's work; but the embryologist has so far shown little interest in what has sometimes been termed "late" embryology, confining his attention to the plentiful and unsolved problems of the earlier months. Hence what happens to the hypoplastic organ in the *viable* victim of feeble-mindedness is of less immediate interest to the embryologist by reason of the embryologist's comparative ignorance of late embryology

in general and hence of the appearances in organs sufficiently developed to permit their bearers to live.

It might be thought that the *physiologist* would take a tremendous interest in a number of aspects of feeble-mindedness since he would perhaps be able to find the differential effects of imperfect glands of internal secretion and a variety of simplified phenomena of nervous and circulatory reactions. On the whole, however, the physiologist probably feels that his knowledge of these matters in laboratory animals remains too tenuous to permit carrying the standard far afield in feeble-mindedness.

The *zoölogist* and even the botanist of course have the greatest interest in the germ cells, their development and interferences with their development. In various writers one discovers tendencies to the thought that there is a pathology of the germ plasm which at first sight spoils the simplicity of the Weismannian doctrines about the immortality and comparative immutability of the germ plasm. The interest in eugenics has fortified this interest, and no doubt all the germ cell biologists as well as the eugenists have fully in mind the value of studies in the ancestry and familial traits of the feeble-minded. Nevertheless these interests have not crystallized into a theory or plan of attack as to feeble-mindedness.

Like the child in Kipling, each science or art above mentioned has mysterious errands of its own; hence it seems likely that the workers in feeble-mindedness themselves must organize their own researches. Where then should research in feeble-mindedness begin? There is no lack of interest in the education of the feeble-minded. There is no lack of interest in various forms of social service, whether it takes shape as help for the individual, aid to the public purse, or an attempt to safeguard society. There is no lack of interest in heredity and eugenics. There is no lack of interest in the general administrative problem which offers one of the most productive regions for the administrator in the whole field of public service. There is no lack of interest in applied psychology as it has culminated in such mental tests as those of Binet, de Sanctis, Healy, Terman, and Yerkes. Little, however, has ever been said in favor of a consistent attack on the anatomy and physiology of the feeble-minded.

The present researches form a comparatively modest introduction to that highly desirable attack upon the physical aspects of feeble-mindedness which have been so inconceivably neglected in the past. We feel that it is wise to concentrate attention upon brain anatomy for the present, despite the fact that the ultimate value of physiological work, notably in the field of the glands of internal secretion may be immeasurably greater. However, in a number of fields of neuropathological and psychiatric research, we have found that much time can be saved if the physiologist and clinician can have the advantage

of knowing whether they deal on the one hand with entirely abnormal brains or on the other hand with relatively normal ones. This dichotomy of the material is rather apt to leave one with three groups of material, — a) material with gross lesions of an obvious and striking nature; b) material with lesions of a doubtful and questionable character, and c) material without obvious evidence of disease. The worker, whether he deal with insanity, epilepsy, criminal anthropology or any other major field touching the nervous system, finds it convenient to utilize various fundamental studies in the two limiting groups in the series, viz., the group of cases with obvious gross lesions and the group of material with no obvious lesions. He must leave the intermediate or non-interpretable group on one side at the beginning of his research.

The topic of feeble-mindedness is rendered difficult by the fact that the observer apparently cannot compare abnormal material with normal looking material just by reason of the absence of any normal looking material. At least an observer like Tredgold remarks that he has never "yet seen the brain of an idiot, a low or even medium grade imbecile which could be regarded as normal upon careful naked-eye examination" (of course it will be noted that Tredgold does not here speak of the imbeciles of higher grade and of the feeble-minded proper). It is safe to say that from the literature one cannot for the present assert whether or not the brains of imbeciles of higher grade, of the feeble-minded proper or so-called morons, or of the dullards or simple-minded persons who grade somewhat higher than the former, have normal looking brains. Beyond question, however, most workers distinguish at least roughly between the idiots and imbeciles of lower grade who are virtually bound to show obvious brain lesions, and the higher grades of feeble-minded, who may or may not show such lesions. In either case there is a tendency to the belief that amongst the higher grades will be found persons whose feeble-mindedness tends toward functionality as a limit. There is a tendency to the belief that the higher grades of feeble-minded are persons whose brains are fairly well developed in the sense of having a full complement of cells like their normal brethren, but whose cells do not for some reason work properly or have not, for some reason, developed their full panoply of processes. That there is a group of feeble-minded subjects that in a sense correspond to idiopathic epileptics or to the so-called functional psychopaths (for example, victims of manic depressive psychosis of modern conception), or like the delinquents whose brains are working, as it were, perversely, without structural basis for the perversity, amounts even to a working hypothesis with many observers. Upon this working hypothesis is founded a number of conceptions as to the educability and comparatively good prognosis of certain feeble-minded subjects "under proper conditions" or with an "improved environment." Whether there is an environmental group

of feeble-minded subjects, whether there is, as it were, an idiopathic or functional group of these cases, is an open question. Just as we suspect that there are frank psychoses which have their bases not in structural brain disorder but in exogenous factors working from outside the brain, so we may theoretically conceive that there are feeble-minded subjects in the same logical group. However, in a general way, the concept of "discords played on a good instrument" is not a concept which appears to have a wide scope in feeble-mindedness.

The literature does not lack work of a systematic and comparative nature upon idiocy. One has only to remember the work of Hammarberg above noted. The literature lacks consistent studies of feeble-mindedness of higher grade from the anatomical and histological points of view. This task should be one of the major ones of the present undertaking. Hammarberg's work was one of the first pieces of work not only in the anatomy and histology of feeble-mindedness but in the topographical histology of the nervous system. The topographical histology of the nervous system has only been thoroughly staked out within the present century. The idea of the brain's differential topography doubtless lay completely in the minds of such workers as Bevan Lewis, Betz, Meynert, Cajal and Nissl in the nineteenth century. Early in the present century, systematic works like those of Campbell, Brodmann and Cajal began to appear and to have currency. These works on topographical histology were supported by such brain anatomists as Elliot Smith and Ariens Kappers, as well as by the comparative anatomist, Edinger. The idea of cerebral cortex mapping, of a kind of cartography of the brain, has now obtained a firm foothold in neurology and neuropathology. It must, however, be accredited to the science of feeble-mindedness that the lamented Hammarberg was one of the first histologists who saw the importance of such cortex mapping and began his work upon the material of idiocy. To be sure it will be many years before a properly equipped institute carries out the inchoate aims of Hammarberg and the aims which we can all now so clearly see when we associate in conception the material of feeble-mindedness with the modern differential work on cortex structure.

One of the most fruitful distinctions in feeble-mindedness of the present day is that of Tredgold, who, seeing the inadequacy of the distinction of the feeble-minded into congenital and acquired cases has replaced this dichotomy with a new and different one, namely, with a division into what he calls primary and secondary amentia.

The primary aments are victims of what is sometimes termed "germinal blight," whereas the secondary aments are victims of some cause working from outside the brain. The primary ament, according to Tredgold, has cells with an intrinsic vital deficiency,—cells which are unable to develop. The secondary ament is the victim of arrest, due to

some external cause. One might say that the cells of the ament, if this distinction of Tredgold can be upheld, have "run down" or ceased to develop at a certain point, whereas the cells of the secondary ament have not run down of themselves, but have run counter to some agent; that is, the cells have not ceased to develop, they have been stopped. The distinction reminds one of the simple nomenclature apparently introduced into neurology by Moebius, namely, the distinction between cases of endogenous and cases of exogenous origin. One deals, as it were, with a plastic impotence, an agenesia, in the case of the primary ament; one deals with a sort of plastic arrest or aplasia in the case of the secondary ament.

The value of this distinction of Tredgold's is that the older heterogeneous anatomical classifications of the feeble-minded, such as were, for example, available to W. W. Ireland in his book on the "Mental Affections of Children" can be supplanted by a simpler classification with a number of new research possibilities. Ireland, for instance, speaks of genetous idiocy, namely, idiocy in which the disease condition which entails deficiency of mind is complete before birth, and in which accordingly the presumption of the hereditary connection is stronger than in other forms. Another chapter deals with microcephalics, a third with hydrocephalics, a fourth with eclampsic idiots, a fifth and sixth with epileptic and paralytic idiots, and further chapters with traumatic, inflammatory, sclerotic, syphilitic and cretinistic idiots, and the last chapter with what Ireland terms idiocy by deprivation (namely, that condition of mind in which a child remains who is deprived of two or more of the principal senses).

According to Tredgold a great number of the intrauterine causes of feeble-mindedness are, as it were, equally exogenous to the feeble-mindedness as would be a cause operating early after birth. The antenatal and neonatal pathology of feeble-mindedness would have to be separated into a congenital and acquired group forthwith, unless we adopt Tredgold's distinction and consider that numerous antenatal causes, such as foetal sepsis, foetal syphilis, and the like are just as secondary in their effects as would be these conditions in the neonatal period. Tredgold accordingly distinguishes an intrinsic from an extrinsic group and would probably subscribe to Moebius' terms "endogenous" and "exogenous" as equivalents of his own. In his original discussion of amentia in Mott's Archives in 1903, he had already practically distinguished the intrinsic forms as due to the condition of the germ plasm and the extrinsic forms as due to the environment, which environment he divided into antenatal, intranatal and postnatal. But in his textbook Tredgold states that the real question is as to the respective influences exerted by heredity and by environment and proposes that the terms "congenital" and "acquired" be given up. Tredgold defines extrinsic causes as any factors of the environment which are cap-

able of prejudicially affecting the brain development of the offspring after conception has taken place, whether these factors be intra or extra-uterine. Amentia due to such causes, Tredgold terms extrinsic or secondary amentia.

Tredgold's employment of the term "hereditary" is possibly somewhat special to him. Primary amentia Tredgold regards as not "due to absence or suppression of some specific germ determinant" such as some Mendelians assert, but "as resulting from a diminished germinal vitality in consequence of which development tends to be incomplete." Inheritance takes the form of the "neuropathic diathesis or innate predisposition to neurotic imperfection." As to the origin of the germinal impairments that lie at the foundation of the intrinsic or primary group of Tredgold, we have a number of possibilities. The Royal Commission on Feeble-mindedness decided that "both on the ground of fact and of theory, there is the highest degree of probability that feeble-mindedness is usually *spontaneous* in origin, that is, not due to influences acting on the parent — and tends strongly to be inherited." Tredgold regards this as a confession of ignorance. He, however, looks upon the hypothesis of Davenport that "feeble-mindedness is not a new variation, but a perpetuation of strains defective from the beginning" as not tenable. Tredgold regards the germinal variations in the parents of the feeble-minded as pathological variations, as a vitiation and not spontaneous. He regards this vitiation as primarily due to the action of the environment, quoting the work of Paul on lead poisoning, the work of Féré on the effect of alcohol on incubating eggs, the more recent work of Stockard upon guinea pigs and other similar work. Not only may the germ cells be poisoned, but they may be altered by a variety of physical conditions, such as temperature and non-toxic chemical agents. Whether or not Tredgold's conception of the causes of what he has termed germinal blight are as he thinks environmental, there can be no doubt that it is extremely valuable to separate out all definitely exogenous cases from a residue whose cause may be left undefined.

What now can we see in the brains of primary as opposed to secondary aments? In the secondary types of feeble-mindedness, we shall often see effects of inflammation, appearances like that of porencephaly, hydrocephalus, and the like, which must appeal to us as flowing from causes operating from without and indicating no essential vital deficiency or vitiation of germ plasm, and we shall be greatly interested to determine the nature of these exogenous processes and the quality and extent of their effects upon the brain map. In the light of these irregularly deficient brains we should, under ideal conditions, be immensely interested to learn whether the mental states of the bearers of these brains also show functional cut-outs and losses to correspond with the irregular defects of the brain lesion. A tremendous number of neurological problems will be settled

and a deep vista of new problems can be predicted. Moreover, we should learn much by extended study of these secondary cases from the standpoint of what is termed in the German "Korrekturbildung." We shall namely be interested in discovering what effects may be wrought by the lack of structure A upon the condition of structure B which, under normal conditions, grows in structural and functional relation to A.

But suppose there be no demonstrable exogenous processes and no basis for supposing an exogenous process in a given case. What now will be found in the brain?

The literary sources of information from which to answer this question are not numerous. We must first resort to Bourneville's volumes on Researches in Epilepsy, Hysteria and Idiocy, published in the form of Reports from the Bicêtre, beginning in the year 1881. These reports contain numerous cases reported as fully as the state of science permitted. The majority of the autopsy reports in the earlier years deal with epileptics. Numerous remarkable cases of tuberous sclerosis (sometimes called Bourneville's Disease) are described and photographed. The majority of these cases of tuberous sclerosis were victims of epilepsy or of convulsions in some form. Numerous reports are scattered through the series dealing with myxedematous idiocy. Through the eighties Bourneville's reports contained few instances of case reports in which convulsions were not prominent. In the nineties began to appear more numerous reports of idiocy and epilepsy without convulsions. Special studies of hydrocephalus, porencephaly and microcephaly appear, and the term "symptomatic idiocy" is frequently used in connection with such cases.

In 1891 Bourneville presents in his 11th volume an anatomical classification of idiocy (communicated at the International Congress of Mental Medicine in 1889) which classification has 8 chief heads, as follows: —

Idiocy symptomatic of hydrocephalus; of microcephalus; of arrest of convolutional development; of congenital malformation of the brain (porencephaly, absence of corpus callosum, etc.); idiocy symptomatic of hypertrophic or tuberous sclerosis; of atrophic sclerosis (A, of one or both hemispheres, B, of one brain lobe, C, of isolated gyri, D, of sclérose chagriné du cerveau); idiocy symptomatic of chronic meningitis or meningo-encephalitis; and finally, idiocy with pachydermic cachexia (myxedematous idiocy connected with absence of the thyroid gland). It can readily be seen how much of this classification, which is based upon fifteen years of work at the Salpêtrière and at the Bicêtre, has relation to those aments whom Tredgold would readily call secondary aments. Bourneville and his collaborators have published excellent photographs of many of the cases in this series of volumes. From time to time, histological descriptions of a relatively elaborate nature accompany the reports and deal with questions of neuroglia prolifera-

tions and the like in quite the modern manner, which is the more remarkable since much of the work was done before the vogue of neuroglia studies begun by the publication of Weigert's book on Neuroglia in 1895.

The reports of the nineties are filled especially with accounts of idiocy symptomatic of atrophic sclerosis and of chronic meningitis. Reports upon congenital idiocy are on the whole infrequent in Bourneville's volumes. Such a case published in volume 13, 1893, by Bourneville and Dauriac was, for example, a congenital epileptic idiot, which was almost completely asphyxiated at birth, began to walk at 8 and completed dentition at 10. This idiot had had convulsions at 8 months followed by incomplete paralysis of the right side. Contractures of all limbs with athetotic movements had supervened. Double craniectomy was performed for therapeutic purposes in the thirteenth year. The autopsy showed, besides the effects of the operation, a cerebral atrophy (weight 855 grams), or as Bourneville says, the autopsy findings may perhaps better be said to have confirmed the diagnosis "arrest of brain development" made during the patient's life. There were no areas of sclerosis or induration and this fact is taken by Bourneville to corroborate the clinical evidence as to the absence of any severe convulsions. There were, however, certain leathery appearances in some gyri, notably in the left parietal and frontal regions and in the right frontal and temporal sphenoidal regions.

From such a description and from Bourneville's own remark as to the possible diagnosis of cerebral atrophy, it can readily be seen that such a case very possibly belongs in the group of secondary aments of Tredgold, although the condition may well have been congenital in the usual acceptance of that term.

In 1895 Bourneville and LeNoir published an example of so-called complete congenital idiocy with paraplegia, contracture and deformity of feet. The brain of this case is described by Bourneville as having been reduced to the greatest simplicity, "virtually down to the elementary convolutions." Although these convolutions are in general voluminous, even strikingly voluminous, and the sulci are of a proper depth, annectant convolutions are almost completely lacking. Plates are presented of this case which died at 18.

In 1896 Bourneville and Ruel present another case of "complete idiocy," probably congenital. The brain showed neither meningitis nor focal lesions.

In 1897 Bourneville and LeNoir again published a case of congenital idiocy due to arrest of development, and state that the brain reveals no macroscopic lesion. Nevertheless its morphology is rudimentary and the arrangement of the convolutions is in many places abnormal. These might serve as examples of the few available reports concerning cases of so-called developmental arrest to be found in Bourneville's series.

Although he terms these cases congenital and speaks of some of them as victims of developmental arrest, they seem on the whole to stand nearest to the cases which Tredgold would be inclined to term primary aments. The number of cases of myxedematous idiocy and of various forms of so-called symptomatic idiocy with atrophic sclerosis and meningitis continue far to outnumber the cases of congenital nature. In passing one can only comment with admiration upon the breadth of Bourneville's general plan of attack on his problem, combining as it does the clinical, therapeutic and anatomical ideals.

We shall now naturally turn to Hammarberg for evidence as to the essential nature of these two groups of cases, whether on the one hand we adhere to the older distinctions of congenital and acquired cases, or adopt the more modern distinction of primary and secondary amentia proposed by Tredgold.

Hammarberg's monograph was published in Upsala in 1895, edited by Henschen, after the death of the author from appendicitis. His work appears to have been done a few years after his graduation in medicine and to represent a comparatively brief period of intensive endeavor. His report deals with 9 cases. He believed that he could prove in all cases that the psychic defect could be connected with defective nerve cells in the cortex. The cortical deficiency he considered to be due either to a standstill in development at some antenatal or early neonatal stage, in consequence of which but a small number of cells were permitted to arrive at high development. He admitted the possibility that cells might be destroyed during the process of mal-development.

In some cases development had been checked in small regions by agents working in a limited and local manner, but in such wise that the total development of the cortex had been unfavorably influenced. If the check in development occurred in the latter stage of foetal life, psychic development in the subject became impossible and these cases are grouped as idiots, "*blödsinnig*", in group A. Since the inhibiting factor acted unequally in different parts of the developing brain, a variety of disorders in the cranial nerves in locomotor capacity and in sensibility, such as were found in many cases, were explicable.

In a second group of cases the cortex had not been permitted to arrive at a degree of development more than one normal for the first years of life. These are cases which Hammarberg groups as markedly weak-minded (*in hohem grade Schwachsinnige*). Such cases also exhibited the same irregular cranial motor and sensory disorders.

A third group of cases is a group in which the inhibitory factor is thought to occur during the first year of life and to affect only a small region of the brain. The rest of the brain is regarded as developing properly in all directions, except that the number of cells seems to remain smaller than normal. The psychic result is that the patient is

inactive and less developed than a normal person of the same age. These are cases classified under B 2, as moderately and slightly weakminded (*mässig und in geringem grade Schwachsinnige*). Similar peripheral disorders are found in these cases also.

The idiots of Hammarberg's series number 4,

The markedly weakminded, 2,

The moderately and slightly weakminded, 3.

Although there are a number of drawings of gross brains in plates 6 and 7 of Hammarberg's monograph, the chief direction of Hammarberg's research is microscopic. Excellent drawings to scale of preparations from various areas of the cortex, stained by methylene blue, are presented in plates 1 to 5. Plates 1, 2 and 3 deal with the normal cortex (since it must be remembered that Hammarberg's work antedated the cerebral topography of late years) and deal with superior and inferior frontal gyri, with the precentral gyrus, the superior temporal, the superior and inferior parietal and superior occipital gyri, the central lobe, hippocampal gyrus, the fascia dentata, cornu ammonis, fascia cinerea, the gyrus cinguli and the gyrus centralis superior.

The pathological plates are 4 and 5 and contain 15 drawings to scale of a number of the above mentioned areas. The whole work is but a torso of what Hammarberg would doubtless have accomplished had he lived.

A number of Hammarberg's observations as to the microscopy of feeble-mindedness exhibit a very modern insight. For example, he dismisses the frequently expressed notion that the pericellular and perivascular spaces are enlarged about the brain cells of idiots, whether in consequence of lymphatic stasis or of cellular atrophy. He regards these features as shrinkage processes, finding them to occur only in material which had at some stage been placed in alcohol of marked concentration, and finding similar changes to occur in normal persons. These findings, then, are dismissed by Hammarberg as artefacts, and it is probable that all modern workers will agree with him.

A second statement to be found in the literature preceding Hammarberg and to some extent since his day is that the brain cells of the cortex in idiocy present a disorderly or imperfect arrangement, so that the apical processes lie at right angles to their normal direction or obliquely. Hammarberg discovered that identical appearances might be found in entirely normal material, noting especially that in the inferior frontal gyrus and the hippocampal gyrus such apparent disorder in the orientation of cells is normal.

A third question of contention in the literature deals with the occurrence of cellular atrophy, vacuole formation and pigmentary degeneration, but such changes as these were found by Hammarberg in but one of his 9 cases, and he notes that undeveloped cells have

frequently been taken for atrophic ones, especially if at the same time an imperfect technique has produced the artefact of dilated pericellular spaces.

Hammarberg accordingly dismisses the idea that the nerve cells of the idiot and imbecile do not fill enough space in the cerebral cortex in which they are embedded, that they are especially subject to irregular orientation therein, and that they are necessarily subject to atrophy, vacuole formation or pigmentary change.

Hammarberg's conclusions then deal with four cases of idiocy, namely, subjects deficient in all the higher psychic functions and incapable of psychic development. In three of these cases the greater part of the cortex was in a stage of development normally found in the last half of intra-uterine life. In a fourth case development had, according to Hammarberg, progressed farther and had reached a stage found normally in the first year of life; but in this case a process had occurred which had reduced development back to the same stage as in the other three cases.

The second group of cases, two in number, is a group of markedly weak-minded subjects, whose mental life corresponds to that of normal individuals of one to five years of age. Their cortex was found, according to Hammarberg, to be in a stage of development found in normal individuals during the same years.

The three cases of the group of moderately or slightly weak-minded are cases in which the mental functions are sluggish and somewhat less developed than in normal individuals of the same age. Such subjects are capable of a certain amount of education. Their degree of development, from a mental standpoint, might readily be compared to any special stage of development in normal individuals. According to Hammarberg, the number of nerve cells in the greater part of the cortex is much less than normal and the remarkable feature of the general brain situation is that in one particular region the cortex will be found to be in a stage of development normally appearing in the very first year of life. Here then a number of claims appear which would be well worth substantiating by extensive studies.

The most elaborate recent statement concerning feeble-mindedness is that by Tredgold in his book on "Mental Deficiency". The ideas are largely based upon special work as published in Mott's Archives of Neurology, Volume 2, 1903, carried out in the Claybury Pathological Laboratory during two years' tenure of the London County Council Research Scholarship in Insanity and Neuropathology. Mention has been made above of Tredgold's conception of primary and secondary aments. He regards the essential basis of primary amentia as cellular. Such gross conditions as porencephaly, hydrocephalus, microgyria, hemiatrophy, and the like, are of course frequently associated with amentia, but they are, according to Tredgold, accidental associations therewith. "The

essential basis of amentia is an imperfect or arrested development of the cerebral neurones." Tredgold calls attention to numerical deficiency in cells (compare Hammarberg's work), to irregular arrangement of cells and to imperfect development of cells, giving rise to the frequent appearance of certain cells first described by Bevan Lewis, or to some still more ill-developed cells which correspond with early neuroblasts. Tredgold states that the amount of change discoverable by the microscope is directly proportionate to the degree of mental deficiency present during life. Tredgold also ascribes importance to pigmentation, believing that it is an indication of defective metabolism in which anabolic processes cannot keep pace with the catabolic.

Hammarberg and Tredgold are accordingly in apparent discord as to the significance of irregular arrangement of nerve cells. Tredgold believes that his own experience as well as that of several others makes it clear that an irregular and haphazard arrangement of cortical cells is very characteristic of the condition. He states that such irregularities are found often where there is no accompanying sclerosis. This problem of the irregular arrangement of nerve cells and its significance accordingly remains a problem in the minds of many workers. Doubtless Tredgold would agree with Hammarberg as to the non-characteristic nature of the pigmentation found.

As to two points, Hammarberg and Tredgold are then in comparative agreement, namely, that numerical deficiency in nerve cells is a major feature in brains of feeble-mindedness and that the cellular appearances indicate imperfect development. If we consider the problem from the point of view of these two authors, accordingly, we must spend a maximum of attention on the number of cells and on the qualitative appearances therein.

We cannot pretend at this time to deal with the literature of the anatomy of feeble-mindedness with any completeness, although we have had the advantage of the relatively complete index of the literature of feeble-mindedness possessed by the Waverley School, from which we have abstracted over eight hundred references which deal pretty specifically with anatomical problems. We present below a selection from these anatomical references, having chosen those that relate more especially to the types of case considered in the first series of ten cases. The outstanding work of Bourneville and of Hammarberg as above mentioned, and the systematic treatment of Tredgold, do not by any means exhaust the anatomical interests of feeble-mindedness as presented in readily accessible literature. A book of strongly anatomical trend is that of W. W. Ireland called "Mental Affections of Children", wherein are given a number of points of view of permanent value. Ireland's classification has been given in brief above in the discussion of Tredgold's point of view.

Below we shall consider some American work in the field, before which it may be thought desirable to consider the status of the anatomy of feeble-mindedness as presented at the thirteenth international medical congress held in Paris in 1900. The eighth volume of the *Comptes Rendus* is devoted to psychiatry. There were three special questions proposed to the psychiatrists at this congress, one of which was the question of the pathological anatomy of idiocy. Reports by Mierzejewski, of St. Petersburg, of Shuttleworth and Fletcher-Beach of England, and of Bourneville, were presented, with a special contribution to histopathology by Philippe and Oberthür.

Mierzejewski considered that Bourneville's classification based upon coarse changes in the brain might well correspond to practical needs, but that a histological classification based upon special studies and upon exact embryological conceptions was sure to develop in the future. Mierzejewski considered that the basis of every anatomical lesion in the idiot's brain is a developmental deviation in the nerve tissue. Its origin must be sought in embryonic life or in disease occurring in earliest infancy. There could be no question of any true arrest of development without its morphological and histological basis in the entire brain. There could be determined, however, a true arrest of development in certain regions as indicated by the presence of neuroblasts. Mierzejewski went on to describe a particular group of cases, namely: those in which the white substance of the cerebral hemispheres is well developed but the gray matter of the cortex is distinguished by appearing in enormous and disproportionate amount. It was true that such brains belonging to microcephalic or demi-microcephalic subjects as a rule showed small convolutions (microgyria), but there were cases of this disproportionate development of the cortex in which there was no microgyria. Mierzejewski had published the first case of this nature in the *Comptes Rendus* of the international congress at Geneva in 1877.

The Mierzejewski type of idiot brain is characterized by a fairly developed centrum semiovale, an enormous distension of the ventricles, with the cerebral hemispheres transformed into a species of thin-walled vessel, 15 or 16 mm. thick. In such extreme examples as this, there is always microcephaly as well as microgyria, but there are other cases in which the white substance of the hemispheres is not so shallow and in which microgyria is absent. In such cases, finer methods will determine a lack or insufficient development of transverse intracortical medullated fibres. In von Monakow's case, there were neither any tangential fibres nor any striations of Vicq-d'Azyr. In other cases these structures were more or less well developed.

The situation was identical with the short and long subcortical association fibres, absent in certain examples, more or less developed in certain others. The radial bundles also varied in their development in these cases.

The gray matter presented at first sight the appearance of making up for the insufficiency of the white, sometimes presenting all its characteristic layers with perfect distinctness; in other cases presenting irregularity of arrangement.

The distinguishing feature of these hemispheres was the interpolation of a layer between the centrum semiovale and the cortex, of an intermediary strip of the so-called neuroblasts; or in certain cases, a layer of cells resembling those of the lower layers of the cortex, but irregularly arranged. In one case there had been interposed between the new layer and the overlying cortex a separate strip of white matter. This condition of affairs is called heterotopia of the gray matter. Although this particular form of heterotopia is diffuse, it does not resemble that more frequent form of heterotopia in which there are islets of ganglion cells scattered through the white matter. The microcephalic cases of microgyria show the interpolated layer to be formed of "neuroblasts." The demi-microcephalics have cells in the interpolated layer which resemble the so-called polymorphous cells of the undermost layer of the normal cortex.

This Mierzejewski type of microcephaly is accordingly distinguished from the ordinary microcephalic brain in that the proportions between gray and white matter, more or less well observed in the usual case, are not preserved in the Mierzejewski types. The demi-microcephalics according to Mierzejewski resemble the normal more closely in that the arrest of development on the part of the white matter is of slighter degree and is accompanied by a diffuse subcortical heterotopia of gray matter. But the principle is identical in both types of case.

Mierzejewski goes on to describe in more detail certain microscopic findings in a fresh case of the disease, and then sums up somewhat as follows: Idiocy may in certain instances accompany a condition in which the gray matter is extremely rich and in which there is an abundance of nerve cells. In these cases the commissural system of the convolutions is arrested in development so that there are not a sufficient number of paths of intercommunication and there is a disharmonious development of the different nervous elements. In the central nervous system, quantity alone does not count, but quality of elements and quality in their reciprocal combinations are the things that count. The white matter is nothing but a mass of prolongations of the nerve cells; accordingly richness of the dendritic branchings and of axis-cylinders of cells is the condition which most favors the plentiful development of white matter; and when these ramifications are few in number, the development of the white matter is insufficient. One of the chief conditions for irregular processes would then appear to be the multiplication and broad expanse of the connections of the pyramidal cells. Mierzejewski attributes to Dejerine the idea that intellectual supremacy seems to be the result, not so much of the number of cells,

as of the multiplicity and extent of their inter-connections. The idiot may have cells enough though these are deprived of their proper branchings. Mierzejewski throws out the suggestion that some of the neuroblasts lying below the normal-looking cortex may now and then proceed to a further development, and thus explain the occasional notable amelioration of mental faculties found in idiots.

The report of Shuttleworth and Fletcher Beach presents a classification of the pathological anatomy of idiocy under three main headings: (a) CONGENITAL; (b) DEVELOPMENTAL; (c) ACQUIRED or ACCIDENTAL. Among the congenital cases Shuttleworth and Beach enumerate eight forms.

The *microcephalic* form contains cases with cerebral hemispheres shortened in such wise as to reveal the cerebellum viewed from above, with such hindrance of development during intrauterine life (probably between the third and fourth months of pregnancy) as to disturb the later growth of the hemispheres as well as the detailed development of those parts which had been already formed. It is perhaps the temporo-sphenoidal and occipital regions which suffer most (as to the occipital region, Cunningham and Telford-Smith are quoted). Microcephaly is probably not caused by the premature synostosis of the cranial sutures. On the other hand, the development of the brain is probably the determining factor as to the form of the cranium (H. A. Humphrey). Microscopically one finds simply rounded or oval cells or cells with but few dendrites.

A second form of congenital disease is *hydrocephalus*, both that due to antenatal and that due to postnatal conditions. According to Meynert, congenital hydrocephalus extends the lateral ventricles lengthwise, while acquired hydrocephalus stretches the ventricles vertically and transversely. Cases of Bourneville, Beach, and Alexander Hill are quoted.

A third form of congenital disease is *scaphocephaly*, a condition possibly due to a premature union of the sagittal suture with an exaggeration in the growth of the coronal and lambdoid sutures in such wise that the head is elongated from before backwards, and may sometimes increase in height. But, on the other hand, this condition may possibly be the result of an original elongation and narrowness of the cranium, such as may be found in the natives of New Caledonia, the New Hebrides, and the Carolines. Sir W. Turner is quoted to the effect that this cranial form is due to intrauterine causes, such as inflammation or injury of the mother during pregnancy. Scaphocephaly does not necessarily produce idiocy, and an approximation of this form of skull may be found in North American tribes due to artificial compression.

A fourth type of congenital disease is the *Mongolian* type, with its special bony, cutaneous, mucosal, and cardiac conditions. Brachycephaly, approximately circular

in circumference, with a tendency to parallelism of the parietal and occipital surfaces, is noted. The skull is thin. There is an anomalously short little finger with lateral incurving displacement of the terminal phalanx. The skin is coarse and scaly, the mucous membrane irritable, the tongue is long, marked with deep folds, due to hypertrophy of papillae. Cardiac lesions are cited from cases of Garrod and of Thompson. Wilmarth's work is quoted as to the brains, which are said to show a very small pons and medulla, as well as very thin vessels. Shuttleworth thinks that these Mongolian idiots are essentially unfinished children, and that their singular appearance is that of a phase of foetal life.

The fifth form of congenital case is the so-called *genetous* or congenital neuropathic form, the offspring of neurotic parents. These cases are said to show very early in life excessive irritability, insomnia, attention disorder, and loss of self control. Such subjects are said to show microgyria at autopsy.

The sixth type is the type of *amaurotic family idiocy* of Tay and Sachs.

The seventh form is that of *sporadic Cretinism.*

The eighth form is a form presenting *partial and local atrophies.* Examples are absence of corpus callosum, porencephaly, cerebellar aplasia, and the like.

In addition to congenital cases Shuttleworth and Beach then consider the developmental group, under which they consider eclamptics, epileptics, syphilitics (including juvenile paresis) and paralytics.

They consider that the lesions found in *eclamptic* cases associated with the severe convulsions of dentition are local hemorrhages and adherent membranes which are inconsistent with the normal nutrition of the gyri. As for the *epileptic* group, the work of Bevan Lewis, of Andriezen, and of Echeverria is quoted.

Syphilis, they consider, is not a common cause of idiocy, but juvenile general paresis they regard as commonly caused by syphilis. They state that since Clouston first described this latter disease, there have been about 100 observations published.

The majority of the *paralytic* group of the developmental cases are of postnatal origin. These cases are attributed to inflammation of the brain or the meninges.

In the third group of accidental or acquired cases, Shuttleworth and Beach consider three forms: traumatic, postfebrile, and sclerotic.

The *traumatic* cases are due to compression of the head during prolonged labor or to the forceps. The lesions are situated as a rule in the Rolandic region. Small hemorrhages occur which are followed by degeneration and sclerosis. In the cases of cerebral diplegia, the most frequent cause is a meningeal hemorrhage especially affecting the veins between the pia mater and dura mater in the longitudinal sinuses, in such wise that the hemorrhage in finding its way into the great fissure compresses the upper borders

of the two hemispheres. The opinion of Osler and of Jacobi is quoted: that certain of these cases are actually due to foetal meningoencephalitis. Intelligence need not be markedly affected despite the severity of the physical disease.

Under the heading of the *postfebrile* cases, Shuttleworth and Beach also take up the cases of so-called hypertrophy. The most frequent acute infections which bring about these postfebrile cases are smallpox, typhoid fever, and scarlet fever. Meningeal, neuroglia, and nerve cell changes are noted in different degrees. The relatively rare affection known as hypertrophy of the brain shows an increase of neuroglia and of the white matter, but the nutrition of the brain seems also to be affected. The brain looks anaemic on section and strikes one as having been under a certain pressure within the skull, showing flattened and compressed gyri with sulci effaced. The most markedly injured part of the brain is the white matter of the hemispheres. The head does not reach the size of the head in hydrocephalus, nor is the increase so marked in the temples as it is in the superciliary region. The hydrocephalic head is rounded; the skull in hypertrophy tends more to squareness. In hydrocephalus the distance between the eyes is increased; not so in hypertrophy.

The third type is that of the sclerotic brains, carefully studied by Bourneville. Wilmarth is quoted as finding in 500 autopsies no less than 96 cases. The tuberous form of sclerosis was found in 15 instances.

Bourneville presented his anatomical point of view under the following ten heads:

1. Meningitic idiocy.
2. Meningoencephalitic.
3. Developmental (simple arrest).
4. Atrophic sclerosis.
5. Hypertrophic or tuberous sclerosis.
6. Porencephalic.
7. Pseudoporencephalic.
8. Myxedematous.
9. Congenitally malformed.
10. Microcephalic.

This account of the general aspects of brain anatomy in the feeble-minded might be concluded by a brief reference to the American literature. Besides the allied but separate topic of amaurotic family idiocy or Tay-Sachs disease, not here considered, and various case reports presented largely from the aspect of being interesting as clinical neurological phenomena by Mills, Spiller, Pearce, and others, the American literature is

devoid of much systematic work except that of Wilmarth about to be considered. From Massachusetts, a few cases have been published by Bullard and one by Southard. An interesting attempt was made by Orton in his "Pathological Study of a Case of Hydrocephalus" to parallel the cortex mapping of the topographers (Campbell, Brodmann, and the others) by a similarly conceived study of a markedly hydrocephalic case. He showed how certain portions of the cortex normally sunken below the surface are, as it were, ballooned out to appear on the surface of the dilated brain. The kind of work there initiated by Orton should be continued and performed in a large series of cases, but such work, of both an intensive and extensive nature, requires the resources of a brain institute to execute.

The largest series of cases of feeble-mindedness as yet studied in America from the anatomical point of view is unquestionably the series reported by A. W. Wilmarth, formerly of Elwyn, Pa. This work is favorably commented upon in many publications on feeble-mindedness.

Wilmarth reported in 1890 on the examination of 100 brains of feeble-minded children. The following table shows the classification employed although it is not now possible to learn which cases were entered twice in the table.

No actual disease or imperfect development of the brain	25
Non-development in various forms	16
Degenerative changes in vessels, ganglionic cells, or medullary substance not constituting true sclerosis	15
Sclerosis or atrophy	12
Diffuse sclerotic change	7
Tuberous sclerosis	6
No actual disease or involved development of brain but hypertrophy of the skull	6
Hydrocephalus	5
Extensive adhesion of membranes from old meningitis	3
General cerebral atrophy	2
Acute softening, recent	2
Demi microcephalus	2
Brain above usual weight but convolutions large and simple	2
Infantile hemorrhage	1
Angiomatous condition of cerebral vessels with degenerative changes	1
Glioma with sclerosis	1
Porencephaly with non-development	1

It will be noted from this list that no less than 31 cases, or at any rate 25 cases, were not observed to show any evidence of actual disease of the brain or of imperfect development therein. These observations are probably the most decisive in the literature upon which to found the idea that the brain of a feeble-minded person may not show in the

gross any evidence of defect. It is well known that in the field of mental disease proper, commonly known as insanity, the opinion long prevailed that there was not necessarily anything grossly abnormal to be found in the brains. Upon the older observations was founded the idea of the functional nature of mental disease so long prevalent. It would have been equally easy to have founded a hypothesis as to the functional nature of feeble-mindedness upon such data as these of Wilmarth. In fact, there are, so far as I know, no more modern data upon which to make a new judgment. In the somewhat allied field of epilepsy, however, Thom and the writer have recently discovered a somewhat similar percentage of normal-looking brains if we confine attention to the gross appearances; namely, about 30 per cent. Our own first series to be presented below (Waverley Research Series Cases I–X) shows at least three cases whose brains might well be considered in the gross as of a normal appearance. Perhaps, then, the generalization which Wilmarth's figures might permit will attain objective value. Wilmarth does not describe further these interesting cases with normal-looking brains. He states that 34 of the 100 cases belong to the school department or were employed as aids in the asylum department, and that 66 of the cases were children belonging properly to the asylum department.

Wilmarth was struck by the comparatively large number of cases of actual cerebral disease in contrast with the relatively small number with imperfect development which seems to be the causative agent. There were, however, 16 cases of so-called non-development in various forms, which put together with two cases of overweight brain with large and simple convolutions, a case of porencephaly with non-development, and possibly two cases of demi microcephalus, make a fairly large total of cases of arrest of development. As against these 21 cases of developmental arrest may be placed 42 cases; namely, 12 cases of sclerosis and atrophy, 7 of diffuse sclerosis, 15 of degenerative changes not regarded as constituting sclerosis, 2 of cerebral atrophy, 6 of tuberous sclerosis. It appears then that, although it is not safe to say that there is a relatively small number of cases with imperfect development, there is a somewhat larger number of cases of actual sclerosis of the brain substance which may be safely regarded as due in some sense to progressive changes, or at least to changes subsequent to birth. How many of these sclerotic cases are merely complications of developmental arrest was of course not possible for Wilmarth to decide and indeed constitutes a problem of the greatest difficulty. Besides the cases of arrest and the cases of sclerosis with or without arrest, there are a few cases of more obviously acquired nature, namely, one of infantile hemorrhage, three of old meningitis, and two of softening (though these latter may well have been cases of complication having nothing to do with feeble-mindedness).

The case of angiomatous condition of cerebral vessels was one of Mongolian idiocy.

Wilmarth had five cases of Mongolian idiocy in his series. He regarded the brains as "of good size for imbecile brains." The pons and medulla were very small, weighing about ½ ounce in each instance, whereas the usual weight according to Wilmarth is nearly twice as much. He found the cerebral vessels inclined to be much thinner than in healthy brains, and was led to suspect from the defective nutrition and circulation of these children that the defective condition of the vessels was a general one. He laid down an interesting hypothesis in the following words: "From the small size of the pons and medulla in every instance, there seems to be a strong probability that the low nutrition, and possibly the other anatomical peculiarities of this group, may be due to the imperfect development or absence of certain cell groups in this region." The following extracts from Wilmarth's paper give further notions as to his conceptions:

"Probably the most frequent morbid condition met with is sclerosis in its various forms. The most destructive of these to the functional activity of the part involved is the so-called *sclérose tubérense*. It is usually seen on removing the membrane, as one or more areas of a white color and considerably harder in consistence than the surrounding tissue, slightly elevated above the level of the neighboring convolutions. It may be single but is usually multiple. It seems to be formed by a finely granular exudate, probably albuminous in character. If it occurs in the motor regions of the brain, the resulting paralysis is very complete, and if a large portion of the cortex is implicated, profound idiocy is liable to be the result."

"Another form of sclerosis, in direct contrast with that just described, is marked by shrunken tissue, the fibrous character of its structure, [*sic*] is more liable to be found in localized mass than in disseminated nodules, and is apparently less destructive to the function of the parts implicated unless it has reached an advanced stage."

"Non-development is found in several forms. A portion of the cortical substance may be thin, and instead of following the typical arrangement of the fully developed brain, form a number of irregular folds, which may be so small and numerous as to resemble a mass of angle worms. Again, the gyri may be of a normal size and appearance, but show a diminution in number, or absence of ganglionic cells in certain layers. Or, again, the convolutions may begin of normal size and development, but soon diminish and sink beneath the surface of the hemisphere, or perhaps entirely disappear, before acquiring near their normal length."

From the above all-too-hasty analysis of the literature of brain anatomy in the feeble-minded, we may suggest the following problems as facing us in the present situation.

1. There should be an attempt all over the world to swell *the number of properly reported cases* of feeble-mindedness *with autopsy*. If we speak of cases with suitable

histological studies alongside the clinical and anatomical observations, we shall shoot far over the mark if we concede that the literature contains 100 comparable cases — certainly a slender material from which to draw conclusions. Tredgold's interesting book, for example, is founded upon something like a dozen cases, among them epileptic and juvenile paretic cases. Hammarberg's work dealt with but nine cases. Many of Bourneville's cases are *curiosa*.

2. *Systematic* external *photography* and photography of the sectional brain should be freely used and reproduced. For in no other way can what is normal in the gross brain be established. The "normal" anatomists have little or no opportunity as a rule to lay down their own standards, using as they often do prison material, asylum material, and the like, and regarding "brain anomalies" with the same equanimity as anomalies in the liver.

3. *Total brain sections* may facilitate the just-mentioned task and are of especial value in marking off "secondary" from "primary" aments, through evidence afforded of special fiber-degenerations.

4. What may be called the "Mierzejewski effect" is worth particular attention, viz. the problem afforded by certain cases of disharmonious development of gray and white matter (gray in excess), in which the commissural system is thought to be deficient. Rich conscious endowment with poor mobilization might result in mild cases, if we carried the Mierzejewski idea to the limit.

5. From feeble-mindedness material we may hope to establish the truth or modified truth of the idea attributed by Mierzejewski to Dejerine, viz. that intellectual capacity depends more on cell interconnections than on cell numbers. The Dejerine argument is probably taken for granted by most workers nowadays but certainly needs proof and extension to details.

6. Hammarberg's idea that he could more or less approximately date the origin of many events of development in his cases requires further work. Such considerations as those of Donaldson *re* Laura Bridgman's brain are decisive here. Who knows what results bearing upon education would be obtained by exact ideas as to the chronology of normal cerebral development? The route to these observations may in part lie through feeble-mindedness material.

7. An exact definition in cerebral terms of "plastic impotence" (agenesia) and "plastic arrest" (aplasia) would help in many fields, especially perhaps in that of dementia praecox. Indeed strong arguments could be brought for the assertion that future psychiatry is to be founded upon a proper knowledge of feeble-mindedness.

8. The problem of the "normal-looking" brain in feeble-mindedness is intriguing.

Is there such a thing as "functional feeble-mindedness"? We speak of manic-depressive psychosis perhaps or of certain psychoneuroses as "discords played upon good instruments." Can the good instrument simply remain in its case, as it were, with resulting amentia?

9. A host of special problems in developmental mechanics, glandular dysfunction, disharmonious development of organs, heterotopia, dislocation of cells, reactive gliosis, satellitosis, premature pigmentation of cells, and the like remain, not to mention the problems of alcoholism and syphilis in parents and the operations of heredity working as it were *per se*.

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NOTE: — This bibliography is partial. It is based upon the entries in the card catalogue of the Massachusetts School for the Feeble-minded and contains those titles which specifically refer to the anatomy of the feeble-minded or articles which are known to contain anatomical material not indicated in their titles. To this fundamental list, which is indicated by Roman numerals and Arabic figures describing the place of articles in the Waverley collection, have been added further references called for by the present work. The bibliography is a working bibliography without pretensions to completeness, and has been arranged by years in such a manner that the history of the development of the subject is clearly shown. Future additions to this bibliography will be made, also by year numbers.

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II.

CLINICAL, ANATOMICAL, AND BRIEF HISTOLOGICAL DESCRIPTION OF TEN CASES OF FEEBLE-MINDEDNESS, WITH EIGHTY-FOUR PLATES (WAVERLEY RESEARCH SERIES, CASES I–X).

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With Eighty-four Plates (Waverley Research Series, Cases I–X).

E. E. Southard and Annie E. Taft.

We proceed to a condensed description of the findings in Cases I–X of the Waverley Research Series. For convenience we offer the following table, showing a number of facts about each case, chiefly of a catalogue nature:

Research Series number	Clinical Source number	Local Autopsy number	Harvard Series number	Photograph number	Sex	Actual Age	Assigned Mental Age	Height last taken	Brain Weight, grams	Brief designation
I	W. 2268	Taylor, 65	14.5	28	M	5	1—app.	82	400?	Microcephaly
II	W. 1437	Taylor, 21	14.6	26	M	20.5	1—app.	subn.	620	"
III	D. 14550	D. 1279	12.20	24	M	45	7 app.	126	610	"
IV	D. 16282	D. 1517	12.21	23	F	62	12±app.	143	1340	Moron or subnormal
V	D. 14779	D. 1413	13.13	1413	M	16	12±app.	168	1435	Subnormal, dementia praecox
VI	W. 1453	Canavan, 3	13.47	75	M	26	2 Binet	158	1130	Idiot
VII	W. 1574, 1618	Canavan, 8	14.13	78	F°	25	5(2—)3 Binet	146	1270	Imbecile
VIII		Magrath, 3917	12.22	1	M	36	12±app.	179	1340	Murderer
IX	W. 808 Burbank 9760	S.B.I. 14.22	14.69	14.69	M	39	7 Binet	178	1620	Imbecile
X	W. 971	S.B.I. 14.69	15.2	15.2	M	37	2.4 Binet	144	1450	Imbecile

W. = Waverley (Massachusetts School for Feeble-minded). Burbank = Burbank Hospital, Fitchburg. Canavan = private autopsy series, M. M. Canavan. D. = Danvers State Hospital. Magrath = Medical Examiner's series, G. B. Magrath. S.B.I. = series since establishment of Massachusetts State Board of Insanity, Pathological Service, July 1, 1914. Taylor = private autopsy series, E. W. Taylor.

The items of our description call for little comment. They are in general:

a. Clinical Findings
b. Autopsy Findings
c. Special Description of Brain
d. Partial Microscopic Description
e. Anatomical and Histological Summary.

As to *a. Clinical Findings:* These have been arranged so far as possible to follow a scheme employed at the Massachusetts School for the Feeble-minded and originated by Dr. W. E. Fernald. The headings are ten in number, as follows:

1. Physical Examination.
2. Family History.
3. Personal and Developmental History.
4. History of School Progress.
5. School Examinations.
6. Practical Knowledge.
7. Economic Efficiency.
8. Social History and Reactions.
9. Moral Reactions.
10. Psychological Tests.

Following the items thus arranged are notes, usually brief, concerning the institutional history, if any, of the patient.

As to *b. Autopsy Findings:* These are limited to the gross anatomical findings of the examiner as made on the autopsy table. Their results are more systematically given in table form in Article III.

As to *c. Special Description of Brain:* The data are founded upon examination, as a rule, subsequent to the autopsy and to the preservation of the brain, which has been in all cases in 10% formaldehyde solution, in many cases for relatively long periods, which are obvious from the dates included in the descriptions. As a rule, the special brain description has had the advantage of comparison of the brain specimen itself with the photographs, systematically taken, of the brain before and after stripping the pia mater. These descriptions are largely the product of Dr. Taft, who has personally stripped each brain of the pia mater, preserving the pia mater in certain cases where there was question of important lesion therein. The nomenclature is, as a rule, self-explanatory and follows largely the nomenclature of Quain's *Anatomy.*

As to *d. Partial Microscopic Examination:* It must be insisted that this is really but partial and has no pretensions to the completeness which the resources of a modern laboratory will eventually command. Nevertheless, we have resorted in each instance to total brain sections stained by the Weigert myelin sheath method in our estimates of the fibre content of the brain. The resulting estimate is, as a rule, entirely qualitative. But it may be questioned whether it is possible at this time to make more than a qualitative estimate of fibre contents. The total brain sections stained by the Weigert method have been of value in the estimates of the depth of the corpus callosum, as well as for the purpose of discovering special periods of degeneration or faulty development of fibre systems.

We have, also, employed staining methods for the study of cortical architecture,

employing as a rule cresyl-violet in place of thionin and methylen blue. This sharp nuclear stain has permitted us in most cases to make a profitable study of the cortical lamination, as well as to bring out the exudate about vessels where exudate is present. As will be emphasized in Article III, the existence of exudate in more cases than had been at first suspected is probably of importance from the genetic standpoint, since syphilis or some kindred chronic inflammatory condition may be suspected in cases with such exudate. Again, we have been able to observe rod cells in certain cases, which cells again tend to indicate a more progressive condition than has been considered typical in cases of feeble-mindedness. In suitable cases, we intend to make special topographical studies of cortical laminations. This we have in hand in chosen instances, and the resulting descriptions are intended to appear in later articles in this series. For the present, as will be evident from Article III, we have limited our microscopy to the question of exudate, rod cells, and the like, on the one hand, and to qualitative estimates of cortex lamination, fibre content, on the other.

As to *e. Anatomical and Histological Summary:* We have attempted to bring out the main structural points in each case, with, however, little or no attention to the total genetic picture presented by the case. This total genetic picture is reserved for Article III.

Further light upon the anatomical situation presented by each brain is offered in the description of the plates. The most inclusive view of each case from all available aspects: clinical, anatomical, histological, and genetic, together with points especially mentioned in the description of the plates, is given in Article III.

Synopsis of Findings of Case I.

a) Clinical.

1. Physical Examination.— P. Cap. was born June 19, 1897, and upon admission, at five years of age, was 32 inches tall and weighed 16 pounds. A photograph shows typical microcephaly, with a head roughly diamond-shaped when viewed from the side, with large ears; spinal caries of the dorsal region, with anterolateral curvature; thighs sharply flexed on abdomen, and legs flexed on thighs; arms slightly flexed and in position of contracture at elbows; strabismus. The eyes are described as "rather intelligent looking" despite the patient's inability to understand things said. Patient would turn head when spoken to, and follow persons or bright objects with his eyes. Spastic crural paraplegia; slight ability to move hands and arms, and ability to grasp small objects with the hands. Although unable to walk, the patient was able to push himself along the floor, and would come at command. Would stop crying when he heard food being brought in the customary way. Absence of control over bladder or rectum; daily vomiting. Patient is described as having had measles, and is said to have been able to kick up his legs at some time prior to his entrance to the school; but it is possible that this reaction, described by

the parents, was merely the effect of action of the muscles of the back and gluteal region. No history of epilepsy.

2. Family History.— Sister (born, Nov. 6, 1894) is an inmate of the Massachusetts School at the present time, having been admitted at the same time, at the age of 7. There are two other children, not microcephalic. The father and mother were born in Italy, and were about 33 and 23 years of age respectively at time of birth of patient. There are two other children, one of which was older than the patient's sister.

Parents are said to have been normal at time of the birth; birth was normal; and, although it was stated that the labor in the case of patient's microcephalic elder sister was a tedious labor with slow dilatation, the delivery was not instrumental in the case of either of the two microcephalic children.

3. Personal and Developmental History.— Patient seemed to the parents never to have developed mentally. He is described as having been fed but once a day, on bananas only.

4. History of School Progress.— Nil.

5. School Examinations.— Nil.

6 and 7. Practical Knowledge and Economic Efficiency.— Nil except for statements made under *Physical Examination* (see above).

8. Social History and Reactions.— Limited to interest in food on the instinctive level; would continue eating indefinitely if food was supplied. Patient wanted to be let alone.

9. Moral Reactions.— Occasional crying spells, possibly related to appetite.

10. Psychological Tests.— Quantitative tests not made. The microcephalic elder sister, obviously a little superior to her brother, measures 1.3 years of mental age by Binet scale. It is clear that the present case would have measured one year or less.

Waverley School History.— Patient's condition was unchanged throughout his stay. Patient was admitted April 12, 1902, when five years old, and died May 21, 1902.

b) Autopsy.

Notes not available; findings are limited to Gross anatomical examination: Microcephaly, dorso-spinal caries, with antero-lateral curvature; thighs sharply flexed on abdomen, legs flexed on thighs, arms slightly flexed and contractured at elbows.

c) Special Anatomical Description of Brain.

The measurements of the microcephalic brain, after preservation in formaldehyde for 11 years, were as follows: —

Length of left hemicerebrum LLH, 9 cm.
right LRH, 8.25 cm.
Greatest width W 8.75 cm.
Greatest height Heit 5 cm.
Cerebral index $\frac{W \times 100}{\text{Length}} = 97.22$ cm.
Greatest width left hemicerebrum WLH, 4 cm.
right WRH, 4.75 cm.
Arc frontal pole to Rolando left LArc FRo 3.08 cm.
right RArc FRo 3.07 cm.
Arc left Rolando to occipital pole LArc RoOc, 5.2 cm.
right RArc RoOc, 4.7 cm.

The brain gives the impression of being very simple and slightly developed. There seems to be a moderate degree of generalized thickening of the pia mater over the entire brain. The vessels are prominent but fail to show gross changes. It is noteworthy that the left hemisphere is longer than the right. The olfactory sulcus of the left orbital surface is short and possesses distal extremity deflected to the left. The olfactory sulcus of the right orbital surface is straight and extends almost to the anterior pole. The sulci on each orbital surface external to the olfactory sulci are small triradiate sulci whose extremities extend forward and sidewise well toward the margins of the orbital surfaces. Posteriorly these sulci approach the olfactory trigone.

The markings of the convexity are atypical and more markedly atypical on the right side. The frontal lobes consist of but two convolutions, the superior of which are broader than the inferior and marked by a number of short fissures. The inferior frontal convolutions are but slightly indented and fail to cover in the island of Reil. This deficiency of the frontal operculum is more marked on the left side than on the right.

The central area of the left convexity approaches normal in structure, although the frontal sulci run back to cover the precentral convolution. Upon the right side, the post-central gyrus is folded over the precentral gyrus, forming an atypical operculum therefor. The right postcentral gyrus is situated nearly a centimeter anterior to the left. Like the left central area, the left parietal lobe has an abnormal construction; this is divided atypically by the interparietal fissure; the postcentral and horizontal portions of the left interparietal fissure are connected with each other. The anterior third of the left interparietal fissure gives off a short descending branch, which branch separates the submarginal from the angular gyrus.

The occipital lobes upon both sides are exceedingly small and scarcely marked by fissures or depressions. On the left side, the demarcation of the occipital from the parietal lobe is effected by means of the transverse operculum. The general appearance of this area suggests the "Affenspalte"; on both sides the posterior extremity of the calcarine fissure is found to extend to the external surface of the hemisphere. Upon the left side this extremity terminates in a single cleft, pointing laterally. On the right side, the extremity of the calcarine fissure is a cleft running more frontalwards and finally terminating in a T-shape.

The comparatively large temporal lobes are almost entirely without sulcal markings, if we except the left second temporal gyrus, which is crossed by two transverse fissures in its middle and posterior portions. The pyriform lobule is well developed on both sides, especially on the right. Each pyriform lobule is marked off by a distinct *fissura rhinica* (Turner).

The lateral situation of the hippocampal lobes yields a notable degree of exposure of the crura cerebri and this appearance of an exposure of the crura is increased by the comparatively small number of transverse pontine fibers. Upon removal of the pia mater, the transverse pontine fibers are found arranged in bundles which are almost distinct from one another. The optic tracts seem comparatively very large and the olivary and pyramidal eminences upon the ventral surface of the medulla also stand out prominently. The cerebellum appears to be not remarkable on gross inspection.

d) and e) Microscopic Examination and Anatomical and Histological Summary.

Upon section the lateral ventricles show a moderate degree of dilatation. There is a slight subpial gliosis, involving both an increase of cells and an increase of neuroglia fibrils. Further points so far as important are placed in the general summary below.

Summary of Anatomical Examination.— The brain as a whole is very small. The cortex averages roughly 3 mm. in thickness. There are only two convolutional divisions in the frontal lobes. The right post-central gyrus forms an operculum which overlies the precentral nearly throughout its entire

length. The sulci of the precentral area are shallow; the individual gyri (especially on the left) are very narrow — some are not separated by sulci — and form thin fingers of white surrounded by grey cortex. The lateral ventricles are moderately large. In the postcentral (parietal) area, the sulci are deeper than precentral (frontal) but the medullary portion of the gyri is narrow. The descending ventricular horns are widened laterally. The structures at the base of the brain, though small, are not otherwise abnormal. The tapetum, optic radiations, and anteroposterior bundles are present. The striae of Lancisi (Indusium) are very noticeable in the anterior plane. The left is much larger than the right but decreases in size posteriorly until in the region of the Splenium, they are scarcely distinguishable.

The inferior olives stand out prominently from the periphery of the medulla; a distinct cleft exists on their antero-lateral surface, separating them from the surrounding portions, and the anterior arcuate fibers curve in deeply around the depth of this cleft.

The *cortex* is of average thickness (3 mm.). The cell supply is scant in all layers, but notably so in the medium sized and large pyramid layers, especially in the frontal and parietal areas. Many of the Betz cells are thin and fusiform. This seems particularly true of the left side.

The leaflets of the cerebellum are small, but do not lack in normal elements, nor present any anomaly.

The blood vessels are not remarkable.

Synopsis of Findings of Case II.

a) Clinical.

1. Physical Examination.— Born September 3, 1876; admitted to the school, April 1, 1893; died March 11, 1897. Characteristic microcephalic with receding forehead; knees stiff and flexed; feet extended; ankles rigid; thighs crossed; right knee crossed left; legs in contracture; hips can be slightly flexed; spastic paralysis of right arm; locomotion by rolling; patient could feed himself by use of left arm and hand; patient unable to walk or sit; head is described as asymmetrical. The right hemisphere of the brain is described at autopsy as having been one third larger than the left, and the skull flattening is described as having been especially marked in the left frontal region. No evidence of epilepsy.

2. Family History.— Born in Provincetown, of American parents; father 25 years of age, mother 22 years of age at birth of child. The microcephalic child was the first, seven other children following, none of whom is described as showing any deformity. The mother's health during pregnancy is described as having been good, and no extraordinary circumstances attended the delivery.

3. Personal Developmental History.— Physician (Dr. Newton) described the patient as having had a paralytic shock, at what time before the age at which first peculiarity was noticed (3 months) cannot now be stated. Another version of the cause is "infantile paralysis," but whether this diagnosis, made in 1876, has reference to epidemic poliomyelitis cannot now be stated. The patient is described as having passed through measles, whooping cough, and chicken pox.

4 and 5. School Progress and Examinations.— Nil.

6. Practical Knowledge.— Able to feed himself with left arm and hand.

7. Economic Efficiency.— Nil.

8. Social History and Reactions.— Patient appears to understand certain things said to him. Unable to talk. (Is described by family as able to say "Mamma.") Patient could make cackling sounds of a loud and shrill nature. By means of these sounds and by gesticulating with the left hand, patient was able to express clearly: pain, pleasure, displeasure, hunger, and thirst; and was apparently able to make the other boys in the ward able to understand these expressions. Patient took interest in events about him, watching the boys at play, liking to look at pictures and bright colors. Patient's hearing was good. He enjoyed music and was able to hum several tunes correctly.

9. Moral Reactions.— Patient is described as gluttonous, swallowing food without chewing. He is described by the family as passionate.

10. Psychological Tests.— No quantitative tests made. Patient would probably measure less than a year.

Waverley School History.— Is sufficiently described above. Death was due to septicaemia following gangrene of extremities.

b) Autopsy.

The description of the autopsy is taken from the report of Dr. Taylor:

The autopsy was made about twenty-four hours after death, the head only being examined. The body was undersized and markedly deformed. The head was markedly microcephalic in type, the circumference 17½ inches; from root of the nose to the occipital protuberance measured 11 inches. The skull was thick, particularly over the parietal regions. Corresponding to two marked depressions in the frontal portion of the brain and easily seen before the removal of the dura were two bony prominences on the inner table of the skull. Dura and pia normal. The brain completely filled the cranial cavity, which was asymmetrical. The left hemisphere was markedly smaller than the right, with abnormal convolutions in both. Very striking at the autopsy was the posterior portion of the brain, including the upper parietal and occipital lobes. The cortex here was so greatly reduced in thickness that fluctuation from the distended ventricle was easily obtained on the left side. The right hemisphere appeared approximately thirty-three and one third percent. larger than the left.

The brain was hardened in formalin, and after hardening weighed 620 grams.

Further examination of the brain 16 years after removal yielded the following measurements: —

Length of left hemicerebrum, LLH, 14.5 cm.
right LRH, 14.5 cm.
Greatest width W 11 cm.
Cerebral index, $\frac{W \times 100}{\text{Length}} = 76$ cm.
Greatest height Heit 5.75 cm.
Greatest width left hemicerebrum WLH, 5. cm.
right WRH, 6. cm.
Arc frontal pole to Rolando LArc FRo 8. Left.
right RArc FRo 9.5 cm.
Arc left Rolando to occipital pole, LArc RoOc, 6.5 cm.
right RArc RoOc, 5. cm.
Frontal pole to corpus callosum, FCorp Cal. 3.2 cm.
Right optic commissure 6 cm.

There is considerable asymmetry in this brain. The hemispheres do not differ materially in length, but the right is considerably broader than the left. This is particularly noticeable at the frontal pole. On the orbital surface the left gyrus rectus is narrower than its opposite, and the convolution which forms the margin is shrunken and shortened. This shortens the distance from the frontal tip to the fronto-temporal angle, and deflects the longitudinal fissure toward the left.

There is a circumscribed area of microgyria about midway the second frontal gyrus on either side, that on the left is more extensive. The third left frontal is complex and the convolutions are well rounded.

The central region marks the beginning of an extremely atypical cortex. The precentral gyrus on both sides is of fair width, and well rounded. The postcentral on both sides is much shrunken and narrowed in its upper half. The fissure of Rolando very nearly cuts the margin of the superior longitudinal fissure on the left, while on the right, its upper extremity is one centimeter from the margin.

The entire parietal cortex over the vertex is much shrunken, the convolutions are very narrow and flat, and on the left a small focus has sunken below the surrounding surface. The normal markings are not made out, on account of the alterations in the cortex. At the parieto-temporal juncture, the convolutions assume a more nearly normal outline. This does not include the upper part of both the supramarginal and angular gyri on both sides, both of which are severely affected; more particularly the former and the cortical change is more extensive and extreme on the left than the right.

The temporal lobes are asymmetrical temporo-frontal distance L. 4. cm. Rt. 4.5 cm.

Both first temporals are very narrow; the right particularly so. The second temporals are broad with transverse markings. The left hippocampal gyrus is completely hidden by the folding over of the third temporal, and only the extreme anterior portion of the pyriform lobule is seen. On the right, the hippocampus and pyriform lobule are not remarkable.

The occipital lobes are very small and show the same changes on the superior surface as the parietal upper regions; the lateral surfaces are more nearly normal. The posterior extremity of the calcarine fissure extends well on to the outer surface of the occipital tip. On the left, the distortion of the parietal cortex has brought the calcarine fissure for a considerable distance nearly to the upper margin of the hemisphere.

The pons, medulla, and cerebellum are not notable on gross examination.

d) Microscopic Examination.

Nerve fibers have been replaced in many instances by patches of rather dense neuroglia tissue. In the prefrontal sections this condition is marked, especially on the right side; farther back the areas of gliosis show cavitation with numerous glia cells in rather thin encapsulating walls, and a number of large pigment-bearing phagocytes. The major amount of cell loss is in this portion of the frontal region confined to the mesial surface. Farther back the frontal cortex shows thinning of all its layers, except the external layer of small pyramids. More posteriorly the total brain sections demonstrate a special loss in size of the two second frontal convolutions; but this loss is more marked on the left than upon the right side. The mesial gyri in this region are also characterized by extreme fiber loss, and the cells which persist are arranged patchily. The sections here indicate that there are more nerve cells than the number of persistent nerve fibers would lead one to expect. More posterior sections exhibit the same higher degree of degeneration upon the left side and this degeneration is shared by both the second and the first frontal gyri. Sections through the plane of the anterior extremity of the lateral ventricles show a marked dilatation upon the left side, with a complete degeneration of the fibers surrounding the ventricle superiorly, externally and upon the external half of the inferior border, whereas the fibers on the mesial border and upon the internal half of the inferior border of the ventricle are preserved. The fiber communications of the first and second frontal gyri that underlie portions of the brain are represented by only a narrow band of fibers.

When the sections begin to cut the corpus callosum, it is found that this body is made up of a very thin bundle with a small number of stained fibers. The white bundle and the third left frontal gyrus are thinned out and in this region there are many large pigmented phagocytes. The first and second frontal gyri in this plane are almost completely degenerated as is also the tissue of the superior mesial surface. The knee of the corpus callosum is also very thin. The lower portions of the hemisphere appear more nearly normal than the upper portions, although the gyri in the lower portions of the hemispheres are too narrow and long as compared with normal. Some sections exhibit a multiple cavitation in the degenerated areas with the cavities surrounded by a dense gliosis. In a plane still farther back behind the temporal lobes, a considerable degree of satellitosis is found in both superior frontal gyri. The lateral ventricles are markedly dilated, although the left more so than the right. Small areas of

Betz cells are found in the precentral gyri on each side, although the tissue for the most part above the temporal lobes is markedly degenerated. In view of this marked degeneration, the internal capsule does not appear to be so shrunken a structure as one might expect. The superior temporal gyrus is itself moderately involved in the degeneration, more particularly on the left side. The transverse temporal gyri are markedly shrunken. Planes posterior to the above continue to show extreme degeneration of tissues superior to the Sylvian fissure and even including the mesial surface on the left side. Of these gyri the callosal gyri are the least affected. The superior temporal gyri and insulae of both sides show equal degrees of degeneration. On the right side, the inferior parietal lobule shows a certain degree of change, but the alteration of the superior parietal lobule is extreme, if we except the tissue along the longitudinal fissure and upon the mesial surface. Large amyloid granulations are found at the base of the pineal body.

The thinning of the cortex in sections posterior to the above is extreme except in the temporal lobes. Appearances suggest that the right superior colliculus is smaller than the left. The pyramidal bundles are smaller on the left side than on the right, their magnitude being reduced possibly one third. The tapetum and optic radiations are markedly thinned out on both sides, although more extremely on the left. A section from the extreme tips of the right lobe exhibits a curious appearance of cells in islands separated by fibers.

e) Anatomical and Histological Summary.

The interest of this case naturally lodges in the combination of microcephaly, hydrocephalus and posterior-lying focal lesion of the cerebral cortex. It is supposed that this latter lesion may have been due to an old cortical lesion, possibly of the nature of cerebral lesions sometimes occurring in infantile paralysis. The brain is somewhat asymmetrical; in particular the right hemisphere is considerably broader than the left. It is an interesting inquiry what relation may be held between the focal parietal, occipital lesion and the shrunken left frontal region with its circumscribed area of microgyria in the second frontal gyrus.

When we find that the nerve fiber loss has been replaced by patches of rather dense neuroglia tissue, we are bound to inquire whether this latter condition of gliosis is to be regarded as an acquired reaction following the destruction of tissue which had been properly laid down, or whether this gliosis is to be regarded as gliosis relative to fundamental absence of nerve fibers. The patchy nature of the gliosis rather inclines the examiner to the belief that we are dealing with a lesion in some sense acquired. The fact that large pigment-bearing phagocytes are found in the gliotic walls of the cavities found in some areas of gliosis is perhaps not decisive; but phagocytosis is consistent with the hypothesis of acquired lesion.

Again the irregular preservation of nerve fibers about the ventricle (mesial and internal fibers of the inferior border preserved) rather suggests acquired lesion.

What the significance of the multiple cavitation in degenerated areas surrounded by dense gliosis may be is doubtful. We may think of the resemblance of these areas to cavitation in the spinal cord in syringomyelia. Patchy nerve cell and fiber loss, cavitation in areas of gliosis, the occurrence of pigment-bearing phagocytes, to say nothing of the somewhat suggestive history of early brain lesion, may well lead us to regard this case as one of secondary amentia in the sense of Tredgold. Whether we may add to this list of factors the presence of satellitosis in certain areas is questionable. On the whole, however, in the present stage of our knowledge concerning satellitosis, it would appear that the finding is rather indicative of an active or acquired lesion. (One here bears in mind the satellitosis about nerve cells in poliomyelitis and other conditions, which may be repeated experimentally; but one also has to bear in mind the satellitosis in certain cases of atrophy of the brain in the senile period where the satellite cells may be regarded possibly as reactive to the condition of atrophy.) It might be that hypoplastic

nerve cells could be affected by satellitosis in ways identical with those of the condition just mentioned. The presence of large amyloid granulations at the base of the pineal body may have significance in these directions but we must at present omit consideration thereof.

The greater degree of disease on the left side of the brain is supported by the smaller size of the pyramidal bundles on the left side above the decussation.

In our judgment the case then is pretty clearly one of secondary amentia.

Synopsis of Findings in Case III.

a) Clinical.

Little is known as to Case III although the patient, D. C., born in 1863, was a somewhat well-known figure in Boston and vicinity all his life, which was passed for the most part outside of institutions. When outside of institutions, patient was regarded as a harmless microcephalic, alternately the pet and the butt of the community. He was of a vagrant tendency, unable to support himself but able to curry favor with various kindhearted persons, who would keep him in their houses for a few days or weeks until the vagrant spirit set in once more. He was known as "Peanut head" by the children, and became later known as "Zip," being a namesake of a subject exhibited at circuses and variety shows. In fact, the patient is said to have been exhibited for a time in a nickel theatre in Boston under the name of "Zip, the wild man of the Philippines." The patient entered the hoax with intelligence, wore a buffalo robe, had his hair cut in a peak behind, and rattled a tin cup, saying "*Education! Education!*"

The patient, born of Irish-American parents (father alcoholic), is said to have been a seven-months child, born in a fracas between his father and a policeman. The patient's father is said to have been occupied with beating the mother when the policeman interposed. The policeman's club is said to have glanced off onto the head of the mother, who died within twelve hours of giving birth to the patient.

The patient is said to have learned to walk and talk at the usual time, was unable to get through the primary school, but is said to have picked up reading and writing. He was at first in a Catholic home; later in a home for destitute children; still later in the State Infirmary in Tewksbury, where he was operated upon for rupture; and he was at the Bridgewater State Farm in 1903. The patient had no abnormal sexual tendencies. He was easily susceptible to alcohol. It is said that at the age of 30 he had a fit, probably epileptic in nature.

The history of the patient just before his death makes a somewhat moving story. Upon good authority, it is related that he was in perfect health until September 15, 1908, being six days before he died. Upon September 15, the patient endeavored to steal a quarter from a kind hostess who had kept him in her house for a number of days, as she had done at previous times. He was found out in his design and was upbraided roundly by his hostess for being a liar. The patient became furious, went to his room in his hostess' house, began to mutter to himself and to tear his hair, and then refused to eat or drink. He is said not to have eaten anything until his death. He was admitted to the Danvers State Hospital *in extremis*, September 20, 1908, and died a few hours later.

b) Autopsy.

Head and trunk were examined by E. E. Southard and the cord by M. M. Canavan.

White male, age 45, 126 cm. long; weight, 88 lbs. Trunk symmetrical; thorax narrow; costal angle acute. Shoulders and hips of equal width. Spinal column straight. Extremities symmetrical and of appropriate length with respect to trunk. Head microcephalic. Skull symmetrical.

Measurements:		
	Glabello-occipital length,	14.5
	Basi-bregmatic height,	12.
	Vertical index,	81.
	Minimum frontal diameter,	9.5
	Greatest parietosquamous breadth,	11.5
	Cephalic index,	79.
	Horizontal circumference,	43.

The glabella is prominent. The general outlines are shown in the accompanying sketch. Extremities rigid. Slight greenish discoloration of abdomen. Pupils: left 5 mm., right 4 mm.

Peritoneal cavity.— Serosa and contents normal. *Appendix* infracecal, free, 7 cm. long. *Mesenteric lymph nodes* not enlarged. Omentum thinly clad with fat.

Pleural cavities.— Slightly moister than normal. Adhesions absent. Roughening of pleura at both apices, in areas less than 3 cm. broad.

Pericardial Cavity.— Not remarkable.

Mediastinum.— No sign of thymus.

Heart.— Weight, 210 grams.

Measurements: T. V. 11. cm. P. V. 5 cm. L. V. 1.5 cm.
M. V. 9. cm. A. V. 6 cm. R. V. 0.4 cm.

Muscle normal. Endocardium shows a slight thickening adjacent to aortic valve. Valves normal. Cavities contain cruor clot. Coronary arteries normal.

Lungs.— Left 355 gr.; right 535 gr. Both edematous and congested behind and below. The dependent portions of both lungs, especially right, are coarsely mottled and non-retractile, and contain bronchial plugs of semifluid pus. There is no palpable deposit of fibrin on the pleurae. The larger *bronchi* are free from pus. The *vessels* are normal. *Bronchial lymph nodes* not remarkable.

Spleen.— Wt. 105 gr. Fir; dark red, without thickening of trabeculae or enlargement of malpighian bodies. Splenculus near hilus below.

Gastro intestinal tract.— Stomach: Contains little or no fluid. Walls covered with brownish mucus which is tenacious. Duodenum negative, also jejunum. In various parts of the ileum greenish black semi-solid pasty material seen. Walls covered with mucoid exudate. No ulceration or hemorrhage seen. The colon also for some distance after passing ileocecal valve shows black content, but at the sigmoid the content is only cheesy mucoid material. Rectum negative.

Liver: Wt. 990 gr. Border sharp. Substance of normal consistence and nut-brown in color. *Gall bladder* not remarkable.

Pancreas.— Small.

Adrenals.— Show no lesion.

Kidneys.— *Left* weighs 170 gr. Measures 13 × 5 cm., is very red and drips blood on section. The capsule strips with fair ease leaving a smooth surface. The cortex is 1 cm. thick, the pyramids are a large distance from pelvis to capsular surface, being 2.5 cm. The pyramids are uniformly diffusely red, while the cortex, mahogany in color, is markedly injected. Kidney substance bulges on section. Two ureters of usual size connect the kidney with the bladder. The *right* kidney measures 9 × 2.5 cm.

Two yellow-white pouches 2.5 × 3 cm. in diameter, spring from either pole of the kidney and descend to bladder as independent tubes 1.4 cm.–1 cm. in diameter. They are 14–18 cm. in length and above the fundus of the bladder appear to unite and descend on right side of bladder. The bladder is deeply congested and in the upper and right side numerous cysts, closely set, appear in wall. Dissection of the ureters shows fine openings into the bladder, two on the left side corresponding with the two ureters and situated appropriately with relation to the trigonum, two on the right side symmetrically placed with respect to those of the left side but draining only the external of the two right ureters. The internal of the right ureters opens into the bladder by a relatively large orifice situated on the lateral surface near the summit.

Organs of neck.— Thyroid weighs 15 gr. Normal on section. Under and along upper border of common carotids a glandular bit of tissue held together by adipose and areolar tissue seen. It is yellow in color and contains numerous glistening red bodies.

Aorta.— Smooth and elastic throughout.

Genito urinary.— Testes yellow-white, small, edematous, do not thread well.

Head.— *Hair* stiff, black, closely cropped. *Sealp* thick and may be readily gathered into loose folds. Calvarium moderately dense. *Dura mater* not adherent to calvarium. *Sinuses* not remarkable. *Middle ears normal.* *Pituitary body normal.* Brain wt. 610 gr. Placed in formalin for later description. Brain reweighed after formalin containing 8% NaCl, 615 grams.

Measurements:	Cerebrum, antero-posterior,	12.5 cm.
	" dorso-ventrally,	9. cm.
	" laterally,	10.75 cm.
	Cerebellum, antero-posterior left,	4.5 cm.
	" " " right,	4.5 cm.
	" greatest dorso-ventrally,	3.5 cm.
	" laterally,	8.5 cm.

Width of pons between origins of 5th nerves, 3 cm.

Antero-posterior basal measurement of pons, 2.5 cm.

Pia mater, everywhere clear. Vessels show moderate injection; small pacchionian granulations alongside longitudinal fissure from middle of superior frontal gyri as far back as superior parietal lobules. Sylvian and Rolandic fissures well marked, almost symmetrical. Gyri of fairly even width. Gyri and sulci so disposed as to yield the impression of a normal though small brain. No sign of Affenspalte. Upon superficial examination, besides Rolandic and Sylvian fissures are made out (before stripping pia), ascending ramus of Sylvian, second and third frontal sulci, first frontal sulcus (interrupted), intraparietal sulcus with branches, first and second temporal sulci, parieto-occipital, calcarine, collateral and calloso-marginal sulci. Orbital surfaces normal. Slightly asymmetrical markings. Cerebellae laminae not remarkable possibly in places wider than usual, notably in Amygdala. The inferior vermis is either absent or obscured by close proximity of inner surfaces of the amygdalae.

Anatomical Diagnosis.

Unequal pupils.
Chronic pleuritis.
Slight chronic endocarditis.
Pulmonary congestion.
Purulent bronchitis.
Acute nephritis.
Cystitis, acute.
Cystic formation in bladder wall.
Abnormality of ureters.

Scalp thick.
Calvarium dense.
Microcephalic brain.
Brain weight, 610 grams.

c) Special Description of Brain.

Subsequent to examination on the autopsy table, the following notes were made concerning the brain:

Length left hemicerebrum, LLH, 16.9 cm.
right LRH, 16.2 cm.
Width W 12, 10.1.
Cerebral index 10.1 × 100, over 16.9 cm.
Greatest height Heit, 7 cm.
Arc frontal pole to Rolando, LArc FRo, 9.3 cm.
right, RArc FRo, 9.4 cm.
Arc left Rolando to occipital pole, LArc RoOc, 8.4 cm.
right, RArc RoOc, 8.2 cm.
Angle of Rolando with long, fissure Right = 53°
" " " " " " Left = 53°
Temporal tip — frontal pole = 3.1 cm.
" " occipital pole = 10.3 cm.
Cerebellum.— Bilateral diam. = 8.7 cm.
Ant. post. at brachial exit = 5.2 cm.

Flocculus fairly developed. Lobes uniform in size and outline.

Fifth nerve leaves the bulb somewhat higher on the right than on left. Cranial nerves otherwise not remarkable.

Basilar vessels show no alterations except slight diffuse thickening of the vertebrals, especially seen on the right about 2 cm. below the basilar.

Frontal segment mesial from corpus callosum = 2.5 cm.

Occipital segment, splenium — occipital pole = 5.7 cm.

Right Hemisphere.— The right Sylvian fissure (6.4 cm.) is very simple, its borders closely approximated. The insula is visible only at the base of the ascending ramus; there is no anterior branch.

Central Rolandic Fissure.— The Fissure of Rolando is 7.6 cm. in length, has no branches and does not cut through the margin of the hemisphere. The genua are represented by two flat curves in the course of the fissure. The superior genu is 1.7 cm. from upper extremity; the inferior, 5.3 cm.

Occipito-parietal.— Rises where the internal border of the cortex cerebri is in apposition with the ant. corp. quad. It approaches the occipital pole in a comparatively horizontal direction. At the margin it bifurcates, forming a broad Y. The occipito-calcarine stem is short, 1.2 cm. The *calcarine fissure* forms an acute angle with the parieto-occipital and continues posteriorly in an S outline extending downward and backward, then outward and upward.

The *precentral fissure-complex.* In the frontal cortex, including and lying in front of the fissure of Rolando, there is one principal fissure, triradiate in form corresponding apparently to the precentral sulcus, frontal from which extends the inferior frontal sulcus. Above the dorsal extremity of the vertical ramus and separated from it by a narrow, plump gyrus, is a short, deep fissure 2.3 cm. in length which cuts the superior margin at right angles. (Sulcus cruciatus?). This sulcal arrangement divides the frontal cortex into two gyri, both of which anastomose with the precentral and anteriorly with each other. The superior frontal convolution is broad and long, and is marked by four short fissurettes which generally

follow an oblique line. The inferior frontal is short and broad, and is marked by three shallow fissurettes, and a deep ascending Sylvian fissure. Very far frontal is a T-shaped sulcus, whose stem runs from a point very near the fronto-Sylvian angle and 0.6 cm. from the orbito-frontal margin to a point 0.8 cm. from the superior margin where it opens into a longitudinal sulcus 2.1 cm. in length, slightly concave mesad.

Temporal and Occipital Fissures.— The parietal lobe presents an anomalous fissure-complex — the postcentral sulcus (1) cuts the margin of the superior longitudinal fissure and extends upon the mesial surface for a short distance. The horizontal interparietal is represented by a very short ramus (2) (1.5 cm.) which springs from the postcentral at the junction of the middle and lower thirds, mesad to this horizontal ramus and 1.4 cm. from the meso-dorsal margin is a deep, curved, horizontal fissure, slightly curved with the convexity mesad. This fissure does not descend vertically into the cortex, but obliquely outward, thus forming a thin operculum which overlies the tips of the horizontal convolutions which form the superior parietal lobule. At its occipital extremity this sulcus opens into a horizontal one 4.3 cm. in length which lies 1.5 on the meso-occipital surface and ends laterad in a forked extremity. The lower lip of this fissure is opercular in character and suggests an *Affenspalte*. The corresponding sulcus on the left side is quite simple, not at all resembling the one just described.

The superior parietal lobule is narrow and its gyri vary in width from 1.2 cm. to 0.4 cm. The supramarginal gyrus is fairly broad — 0.9 cm. in width — but the convolution is short. It anastomoses anteriorly with the postcentral and posteriorly by means of a bridging arm, with the angular gyrus. The latter is made up of three parts which extend horizontally and assume a fan-shaped outline. The gyri are comparatively broad and almost free from ramifications. This gyrus encroaches upon the occipital lobe which is very small. The tips of the occipital lobes of both sides point away from the median line thus exposing a portion of the cortex which is ordinarily found on the approximated mesial surface.

The Temporal Lobe.— The temporal lobe is divided by two very simple fissures. The gyri are relatively broad, smooth and well-rounded, with short shallow fissures at their bases. The sulci on the basal surface are very simple. A deep fissura rhinica separates the uncus definitely from the tip of the temporal lobe. The *inferior temporal sulcus:* It is deep with sloping walls, and is interrupted about midway by an arm from the fusiform gyrus which anastomoses with the third temporal convolution.

The Left hemicerebrum.— The configurations of the area anterior to the Rolandic fissure on the left hemisphere are almost identical with those on the right, in every detail.

The temporal lobe also is uniform with the right.

The parietal lobe.— The ascending ramus of the interparietal sulcus is separated from the horizontal and descending portions by an anastomosing arm between the postcentral and superior parietal gyri. The posterior extremity of the horizontal limb enters into a roughly star-shaped formation. The posterior portion of the second temporal gyrus, representing the area of the angular gyrus, is a separate fragment; below this a continuation of the second temporal fissure extends backward to within 3.2 cm. of the longitudinal margin.

The occipital lobe is noticeably larger than that on the right. Its markings are somewhat complex and may be best studied on the figures and photographs.

The configuration of the basal surface is uniform with the opposite side.

The meninges are not particularly remarkable. The pia is slightly thickened and the vessels injected.

There is noticeable occipital asymmetry; the right side is shorter than the left, and points away from the median line.

The orbital surfaces incline upward to a marked degree both postero-anteriorly and meso-laterally, thus partly exposing them to lateral view. The sulcus pattern here is a very simple tri-radiate one, very similar on both sides. The olfactory bulbs are 3.5 cm. in length, and not notable in development.

Rt. precentral are 9.5 cm.
" postcentral " 8. "
L. precentral " 10. "
" postcentral " 8. "

The markings on the hemispheres are very nearly symmetrical. There is lack of development of both frontal and parietal cortex; possibly involving the former to a greater degree.

The frontal lobes are very pointed at the extreme pole and are made up of only two convolutions. The superior is somewhat narrower than the inferior and is marked with short, oblique fissures, and triangular depressions. The Island of Reil is exposed on both sides: somewhat more on the left than right. At the base of the frontal gyri, and separating them from the precentral convolutions on either side are short, deep, transverse sulci, which resemble the sulcus cruciatus of the dog's brain. In fact this specimen in general contour bears a rather noticeable similarity to the brain of a dog. The convolutional pattern of the central region is very simple. The pre- and post-central gyri on the right are curved only at the genua. The Rolandic fissure on this side points occipitalward at its upper extremity, and nearly cuts the margin of the hemisphere. On the left side the precentral gyrus is interrupted at the point corresponding to the inferior genu; the upper knee is lacking. The Rolandic fissure on this side is formed at its upper extremity by a formation with a convolution of good width between it and the margin of the hemisphere.

The horizontal portion of the right interparietal sulcus is separated from the postcentral portion by an annectant gyrus, between the superior and inferior parietal lobules. It curves around the supramarginal and angular gyri, and seems to form an offset to a portion of the superior parietal lobule. The latter is narrow and marked with transverse fissures. On the left side, the inferior postcentral (descending interparietal) fissure is continuous with the horizontal interparietal.

The superior post central is separated from the former by an annectant gyrus connecting the post central with the superior convolution. A similar opercular formation exists on the left as on the right, but it is made more complex by a reduplication caused by the presence of an additional sulcus which extends with a single interruption, in a curved outline from the posterior extremity of the parieto-occipital fissure to the base of the second temporal from the angular gyrus.

The occipital lobes are very small, and are marked by a few short fissures. The calcarine fissure extends upon the outer surface, ending in a single upward curve on the right, and in a T formation on the left.

The temporal lobes are remarkable only for their simplicity. The gyri are nearly straight, and almost entirely without markings. The first and second convolutions are of good width and well rounded. The third is somewhat narrower and flattened.

The pyriform lobules are well developed, the right apparently more so than the left and are limited anteriorly by a well marked fissura rhinica (Turner) (?).

The markings on the base of the brain are as singularly symmetrical as they are on the upper and lateral surfaces.

The mesial surfaces are not described because the hemispheres were not separated.

d) Microscopic Examination.

The total brain sections stained by the Weigert myelin sheath method permit the observation of a marked gliosis in the white matter in the frontal region, especially about the vessels. In places the cellular gliosis is general and marked and in particular there is an abundance of spider cells in the subpial region. Satellitosis is found in addition to the above mentioned cellular gliosis and in certain areas farther back in the frontal region (sections # 146–151) there appears to be a particular degree of satellitosis about the cells in the lenticular nucleus. Also in this posterior portion of the frontal region, the gyri

begin to show a more marked thinning of their cell layers than is found farther forward. The mesial surface of the uncus of both sides and the basal tissues lying between show a marked subpial gliosis. Large cells were demonstrated in the appropriate region about the ventricle and there was a marked sub-ependymal gliosis with pigmented glia cells and certain large round cells. These findings were especially noted from the mesial angle of the thalamus.

Practically no areas fail to show general cellular gliosis but thinning of the cell layers is shown in addition to that mentioned above for the frontal areas (sections № 156–186) also in the parietal areas (sections № 331-356). The tapetum, optic radiations and longitudinal bundles are intact. The tissue permitted the employment of Van Gieson's stain which demonstrated the subpial gliosis especially well, as also a perivascular gliosis in many places, with lymph spaces apparently containing glia cells (no exudate is present in any area studied). The stem of the olfactory bulb is found infiltrated with amyloid bodies. There was a slight focal thickening of the intima in the basilar artery.

The blood vessels of the white matter appear numerous and there is a good deal of perivascular gliosis. No abnormality of vessel walls is anywhere shown.

e) Anatomical and Histological Summary.

The gyri are everywhere narrow — about 0.8 cm. at their summits — and simple in arrangement. The primary and secondary sulci are of ordinary depth, not complicated by collaterals, and their walls, as a rule, are not approximated at the summits, though the sulcal floor is not exposed.

The frontal lobes are small, and pointed at the anterior poles; the orbital lobes lie in a more or less vertical plane. The markings are notably uniform on both sides.

The temporal lobes are comparatively large and are very simply convoluted.

The parietal lobes present a greater diversity of sulcation than any other part of the brain. The pattern of the two sides varies considerably. This is described in detail in another place.

The occipital lobes are quite at variance in size, the right being considerably shorter than the left. A deep transverse sulcus about midway between the parieto-occipital fissure and the tip of the occipital lobe forms a relatively large operculum and seems to account for the occipital shortening. This lack of uniformity in the size of the occipital lobes is plainly seen on the basal surface. (See illustration).

The brain was not divided longitudinally between the hemispheres and consequently a detailed examination of the mesial surfaces was not made.

Angle of Rolando with long fissure, right = 53°
" " " " " " left = 53°

Temporal tip — frontal pole = 3.1 cm.
" " occipital " = 10.3 cm.

There are varying degrees of satellitosis nowhere marked in the cortex, but rather more extreme in degree in the basal gray matter.

The cortex averages about 3 cm. in thickness.

The cortical cell-supply seems somewhat reduced in amount, especially in the frontal and parietal lobes. Betz cells prominent. The lack seems to be mainly in the outer layers of medium sized and large pyramids — possibly at times the inner layer of large pyramids.

There is considerable subpial gliosis throughout. This is particularly true on the base of the brain between the crura cerebri and the base of the olfactory trigone, especially in the tempero-frontal angle, where the glia network is infiltrated with amyloid granules.

There is also considerable subependymal gliosis — both cellular and fibrillar with pigment in the glia cells. This is confined mainly to the opposed surfaces of the optic thalamus.

Synopsis of Findings in Case IV.

a) Clinical.

Case IV, like Case III, is a Danvers State Hospital case. The patient, M. S., stated to be 62 years of age at death, September 27, 1911, was under hospital observation only from July 26th to her death. The patient was an old almshouse case (Salem and Beverly).

Physically on admission the patient was 4′ 8¼″ tall, and weighed 111 pounds, with arteriosclerosis, slightly enlarged liver, teeth absent, albuminuria, impairment of vision and hearing, thick speech, and a tic of the right side involving head and shoulder.

The meagre history of the patient indicates that she was of old American stock. She had been regarded for the twenty-eight years during which she had been under almshouse observation, as feeble-minded. She was not selfsupporting although she would work, receiving a dollar or a dollar and a half, for a week or more, ranging up to two or three months. At the end of her employment, she would leave with her clothes tied up in a bundle covered by a cloth. Whenever the patient appeared upon the street, she attracted much attention and had a crowd of small boys following her. The tic observed at the hospital was apparently of long-standing and it is said that the twitching was more noticeable when the patient was pleased.

Shortly before admission to Danvers the patient had become untidy. After admission, the patient complained of headaches, was at times restless, was markedly amnesic; did not know the date or her whereabouts, and her attention was hard to obtain. Patient apparently reacted to auditory and visual hallucinations. She said she had seen little women sit on her knees and that they had blue eyes and red hair.

b) Autopsy.

Body of a well developed, well nourished, white female 141 cm. in length. Rigor mortis present everywhere. Skin smooth, hair brown, rather thin. Pupils, right 6 mm., left 5 mm. No teeth on either jaw. No glandular enlargements. No decubitus. Tibial crests smooth. Face asymmetrical.

Ventral Section.— Panniculus over thorax .8 cm., over abdomen near umbilicus 2 cm. Muscle good color. Omentum extends to umbilicus, moderately filled with fat. Lower border of liver extends 8 cm. below ensiform, lower border of stomach extends 6 cm. below ensiform. Few adhesions between omentum and right parietal peritoneum. Adhesions about spleen. Appendix retrocedent, lies over brim of pelvis 5 cm. in length, mesentery throughout. Peritoneal mesentery lymph nodes slightly enlarged, no fluid in abdominal or pelvic cavities. Uterus in right side of pelvis. Diaphragm arches to 4th interspace left, to 4th interspace right. Sternal bone marrow well colored. Internal mammary arteries are not sclerosed.

Thorax.— Left lung adherent along entire posterior region. Adhesions in right lung lie between middle and lower lobes and pericardium, and between the upper, middle and lower lobes. Apex free. No fluid in the pleuric cavity. Anterior edges separated by 2.5 cm. Precordia exposed. Pericardium contains 2.5 cc. of clear straw colored fluid.

Heart.— Greatest width 10 cm., greatest thickness 8 cm. Epicardium contains a small amount of fat. Over the right ventricle is a milk patch slightly irregular in outline, approximately 2 cm. in diameter. Superficial vessels slightly tortuous. Heart contains fluid blood. Weight 225 grams.

Measurements: T.V. 12 cm. P.V. 7 cm. M.V. 8.5 cm.
A.V. 7 cm. L.V. 1.3 cm. R.V. .5 cm.

Endocardium grey, thickened, aortic valve shows slight thickening at line of attachment of cusps,

mitral valve thickened along border. Tricuspid slightly thickened, pulmonary lies flat against wall and shows no thickening. Interauriculus septum not intact. Foramen ovale is patent but is functionally closed. Coronaries are free from sclerosis. Arch of the aorta smooth.

Lungs.— Left lung weight 345, adhesions between upper and lower lobes. Upper lobe air containing throughout, a small fibrous scar at the apex. On section posterior portion darker but crepitant. Lower lobe deep red mottled in lower portion, floats in water. Bronchi in lower lobe congested, upper lobe nothing of note.

Right lung weight 325, adhesions between upper and middle lobes, air containing throughout. Small scar on anterior surface near apex. Upper lobe pale, lower lobe slightly congested; lungs float in water. Bronchi slightly congested. Bronchial lymph nodes not enlarged.

Abdomen: Spleen.— Weight 50 grams. Measures $9.5 \times 5 \times 1.5$ cm. Capsule grey, slightly wrinkled. On section soft, mottled. Trabeculae and Malpighian bodies distinct.

Gastro-intestinal Tract: Stomach.— Lesser curvature 20 cm., greater curvature 26 cm. Stomach filled with about 100 cc. of thick yellow mucus. Mucous membrane slightly congested, rugae distinct, no hemorrhages of ulcers.

Intestines filled with a pale yellow fluid, fecal matter and mucus. Intestines thin walled throughout. Slight congestion of duodenum and jejunum. Peyer's patches not enlarged, no hemorrhages or ulceration. Mucus membrane of colon slightly congested, no hemorrhages or ulcers.

Liver.— 975 grams, 20×20 in quadrate lobe $\times$ 13 in left lobe, 5 cm. in thickest portion. Border well rounded, on anterior surface of quadrate lobe near upper border are three furrows 3.5 cm. in length. On section brownish, lobulation distinct not friable.

Gall Bladder.— Filled with dark, green brown bile.

Pancreas.— 13 cm. long by 2.5 cm. in width.

Adrenals.— Right adrenal weighs 4.8 grams, left adrenal weighs 5 grams. On section firm.

Kidneys.— Right kidney weighs 110 grams, measures $10.5 \times 5 \times 3$ cm. Capsule strips readily, cortex pale, .7 cm. Vessels slightly injected. Pyramids slightly more congested than cortex. On section substance bulges slightly beyond capsule. Left kidney weighs 135 grams, measures $11 \times 5.5 \times 3.5$ cm. Pale, otherwise like the right.

Bladder.— Not unusual. Retroperitoneal lymph glands slightly enlarged and darker than usual.

Neck Organs: Oesophagus.— Nothing of note.

Thyroid.— Fairly symmetrical. Weight 12 grams. Left lobe a little more compact than right lobe.

Larynx, Tongue, and Tonsils.— Nothing of note.

Pelvic Organs.— External os of uterus was closed, canal filled with mucilaginous secretion. Uterus atrophied, shortening of right broad ligament and is adherent to ovary on this side.

Ovary is atrophied, left tube apparently normal.

Rectum, nothing of note.

Head.— Calvarium dense, measures frontal 1 cm., temporal .3 cm., occipital .8 cm.

Large amount of diploe. Scalp not unusual.

Dura mater not adherent to calvarium, slight thickening in frontal portion.

Few adhesions dura and pia over vertex and along longitudinal sinus. Few adhesions between tip of temporal lobes and dura. Lateral sinus of left side contains liquid blood, right side is filled with a firmly adherent clot. Pituitary soft.

Right ear drum thickened with one small perforation of the drum. Left slightly thickened.

Pia Mater slightly thickened notably over vessels over vertex.

Pons and cerebellum weigh 125.

Brain firm, with no marked atrophy of convolutions. Slight flattening in parietal region of right side, left hemisphere appears well rounded. Ependyma free and smooth, no sclerosis in basal vessels.

Brain weight 1215 grams.

Anatomical Diagnosis.

Well nourished.
Rigor mortis.
Unequal pupils.
Asymmetry.
Chronic peritonitis.
Chronic perisplenitis.
Enlarged mesenteric lymph nodes.
Uterus in dextraposition.
Chronic adhesive pleuritis of left lung.
Chronic epicarditis.
Chronic endocarditis.
Chronic aortic and mitral endocarditis.
Fibrous scars at apices of both lungs.
Acute bronchitis left.
Small spleen.
Acute gastritis.
Acute nephritis.
Enlarged retroperitoneal lymph nodes.
Uterus and ovaries atrophic.
Calvarium dense.
Perforation of right ear drum.
Slight chronic pachymeningitis.
Slight chronic leptomeningitis.
Brain weight 1215 grams.

c) Special Anatomical Description of Brain.

After preservation in formaldehyde, the following further description of brain was made: —

The brain is dolichocephalic in type. There is slight asymmetry particularly at the poles, the left in each case being more pointed, and the right occipital tip points away from the median line in a slight degree.

The pia is moderately thickened, and its vessels injected. There are many arachnoid villi along the superior longitudinal fissure. The convolutions throughout are rather narrow, but fairly well rounded, and moderately complex. There is slight opening of the sulci throughout.

The primary sulci of the frontal lobe are not clearly defined. The first can be traced with some difficulty, the line being broken and irregular. On the left it is interrupted by three annectants; on the right the walls interdigitate in a way to make it unclear. The remainder of both lobes is marked by generally transverse convolutions.

The precentral gyrus is continuous on the right; on the left it is cut across by the first frontal sulcus. The postcentral sulcus is very narrow. At about midway its length an annectant connects it with the inferior parietal lobule on both sides. The fissure of Rolando on both sides forms little less than a right angle with the superior longitudinal fissure. Their upper extremities cut the superior margin, and extend upon the mesial surface. The lower extremity on the left is separated from the fissure of Sylvius by the convolution forming the operculum; on the left it cuts through the operculum and extends into the Sylvian fissure.

The convolutions of the parietal lobes are narrow, and complex with very slight separation of their summits. On the right the parts of the interparietal sulcus are continuous; on the left they are interrupted by annectants and all three are separate.

The occipital lobes are not remarkable except for the slight asymmetry already noted.

(Mesial surfaces are not photographed.)

The only feature of the base of the brain worthy of note is the asymmetry of the pyriform lobules. The right is larger and considerably more prominent than the left.

The frontal sections show both lateral ventricles much dilated, except in the descending horns; the optic thalamus and the horizontal portion of the caudate nucleus are considerably flattened. The subiculum gyrus hippocampi also is much flattened and shrunken.

The cerebellum is notable only for the apparent lack of uniformity between the two dentate nuclei.

d) Microscopic Examination.

From the notes of a partial microscopic examination, the following is abstracted: —

The left and right prefrontal areas exhibit a marked gliosis with the appearance of a great numerical increase of cells in all layers. Many of these cells are of a type best described as shadow cells. The plexiform layer shows coarse glia fibers. The remainder of the frontal region exhibits identical changes.

In the white matter underlying both the left and right precentral gyri, there were many ganglion cells largely of the fusiform type ordinarily found in the undermost layer of the cortex. Although there were numerous corpora amylacea in the white matter, there was no gliosis and there was no alteration in blood vessels. The cortex of this region showed a slight degree of satellitosis with a slight subpial gliosis. There appeared to be a focal loss of nerve cells effected in various places of the laminae, except the layer of small pyramids. It is estimated that many Betz cells are smaller than normal. Their protoplasm is granular and there is marked increase of yellow pigment. There are frequent shrunken cells and shadow cells in all layers. The postcentral regions of the two sides are slightly better off as to cell shrinkage than the precentral gyri.

The intermediate postcentral regions also show a considerable degree of subpial gliosis and numerous corpora amylacea. Upon the right side there appears to be a thinning in the layer of large pyramidal cells upon the summit of the gyrus examined. There is no such finding upon the left side. There is a suggestion of increase in glia cells throughout the white substance underlying the intermediate post central gyri.

There is a slight gliosis in the white matter of the calcarine areas. Under the transverse temporal gyri are numerous dislocated ganglion cells in the white substance, resembling those mentioned under the precentral gyri.

The area of Broca shows cellular gliosis with corpora amylacea, a moderate degree of general nerve cell atrophy and a thinning out of the layer of internal large pyramids from the summits of the gyri examined. In the subpial layer are many coarse neuroglia fibers.

The hippocampal gyri, cornu ammonis and pyriform lobules show an unusual degree of subpial gliosis and infiltration of corpora amylacea. There is also a gliosis under the ependyma of the ventricles sectioned at this point. There were a number of cells staining palely with colorless spaces about the nucleus, including some shrunken cells with non-staining nuclei and elsewhere were some examples of the shadow cells above mentioned. There is occasional satellitosis but not confined to special laminae.

The spinal cord and medulla show a marked fibrillar gliosis of the periphery and about the central canal. The posterior columns show a considerable infiltration of corpora amylacea. There appears to be a degree of perivascular infiltration in the posterior fissure.

e) Anatomical and Histological Summary.

The brain of this case shows an atrophy or aplasia of the type with flaring sulci suggestive of a condition in which the white matter is relatively better preserved than the grey. The left side appears slightly more hypoplastic or atrophic than the right. The parietal lobes exhibit particularly narrow convolutions and may indicate a certain tendency to microgyria. The left pyriform lobule is smaller than the right. (This appears accordingly not to be a case of cruciate asymmetry in the sense of case V, for example.)

Employing the microscopic examination for the purpose of determining whether this case is one of acquired lesion, we find a marked gliosis with apparent great numerical increase of cells in many layers. We find a coarse fibrillar gliosis of the plexiform layer in places. We find a considerable variation in degree of gliosis in different places but these findings are consistent enough with the hypothesis of relatively recent changes in a subject of 62 years.

Likewise we may logically dispose of the satellitosis found in certain places as well as the over-pigmentation of certain nerve cells. The shrunken nerve cells, so-called shadow cells, are consistent enough with the hypothesis of relatively recent chronic change. In fact, we should probably be at a loss to determine whether there was any evidence of fundamental change suggestive of long-standing lesion if it were not for the dislocated ganglion cells in the white matter in certain regions. It is important to raise the question whether the slight degree of perivascular infiltration in the posterior fissure of spinal cord in places is to be regarded as a sign of old syphilis. The suspicion must at least be raised in the case, although the data of Wassermann reaction are not available. There are in the body at large no special signs indicative of old syphilis (enlargement of mesenteric and retroperitoneal lymph nodes, valvular and parietal endocarditis, etc.).

The case is of peculiar value in emphasizing the difficulties which attend a decision as to inborn and acquired lesions in cases of elderly subjects. Of course the majority of the feeble-minded fail to reach the sixties, possibly for reasons inherent in their general make-up. This case then must be used as a limiting instance in our total larger series.

Synopsis of Findings in Case V.

a) Clinical.

Case V has been already published in Southard's monograph on dementia praecox, from which the following condensed history is taken:

C. B. was a boy of 15 when he was committed to D. S. H., and died there a year and four months later of *typhoid fever, ulceration, and peritonitis*. The mental diagnosis was eventually considered to be *dementia praecox*.

Mother nervous and melancholy during her pregnancy with this boy; hard usage and neglect by husband complicated the pregnancy. From an accident to the boy at seven, is said to date a change of character, with refusal to go to school, fretfulness, and nervousness. At 14 another accident to back and head. An inmate of a truant school for two periods of three and two years respectively. Uncle a patient at state farm.

Auditory hallucinations, refusal to eat, nervousness, and certain delusions antedated commitment by only a week. (Masturbation had begun four or five months before, but had probably ceased.) Insomnia is said to have lasted four nights after onset. *Food poisoned. Passers-by or Indians were to kill him* (given to blood-and-thunder stories). *"Had never done anything to gladden America," "Traitor," "Going to be shot."*

Cyanosis of hands; gait staggering (no Romberg); right cremasteric reflex not active.

After eight months' observation it was thought that the patient really belonged in the moron or subnormal group of defectives, and in the quasi-criminal class. A little over 11 months after the delusional episode upon which he was committed, patient developed a spell of disturbance which lasted 10 days. In this spell, patient was noisy, threatening, profane, denudative, given to disconnected and incoherent talking, lost weight and grew pale. Almost three weeks later typhoid fever developed, and death followed from peritonitis.

b) Autopsy.

Summary.— Aside from the typhoid fever lesions, there were bronzing of skin, unusually small adrenals (questionable glycosuria had appeared on entrance but later disappeared), a small thyroid, slight sclerosis of aorta and right auricular endocarditis; cardiac hypertrophy, chronic myocarditis, pleuritis, and pericarditis.

The brain weighed 1435 grams (over weight 90 grams by Tigges' formula). There is nevertheless a tendency to *frontal atrophy*, perhaps more marked on *left* side. Striking anomalous folding of convolutions in both calloso-marginal regions above the splenium; but on the left side the fissuration suggests acquired lesion rather than congenital anomaly (region above and behind the rostrum).

c) Special Anatomical Description of Brain.

After hardening in formaldehyde, the following further description of the brain was made:

Measurements:	Length left hemicerebrum, LLH,	18.	cm.
	right LRH,	18.	cm.
	Greatest width W–L.,	6.5	cm.
	Rt.,	7.	cm.
	Height, Heit	10.75	cm.
	Arc frontal pole to Rolando, left,	10.	cm.
	right,	10.	cm.
	Distance temporal to frontal tip, left,	4.5	cm.
	right,	5.	cm.

The specimen shows the not unusual frontal and occipital cruciate asymmetry, in which the frontal lobe, particularly the most anterior portion, and the contralateral occipital lobe, are narrower than their fellows. In this case it applies to the left frontal and right occipital lobes.

There is slight general clouding of the pia over the vertex, somewhat more extreme near the margin of the longitudinal fissure. The pial vessels are moderately injected.

With the pia removed the convolutions appear in general fairly well rounded, but rather narrow; their summits are not closely approximated. There is no remarkable lack of complexity.

The frontal convolutions are distinctly separated from each other by longitudinal sulci, though an annectant connects the middle of the first with the base of the second on both sides. The third is noticeably prominent on the left.

In the central area the Rolandic fissures leave the superior margin of the hemisphere at a right angle. At the superior knee the right points forward more than the left, but the lower extremities both end at a point 7 cm. from the frontal tip. The upper extremity of Rolando extends onto the mesial surface 1.5 cm. in a backward direction on the left, 0.5 cm. perpendicularly on the right. The post central convolution is slightly narrowed in its upper extremity. In the parietal lobes the post central sulcus forms a second or double Rolandic fissure. Posterior to this one the usual interparietal sulci, with the horizontal and descending branch.

The occipital lobes are small; the left side is not otherwise notable, but on the right side the second gyrus is narrowed and shrunken, causing a drawing in of the surface, a part of which is consequently depressed below the surrounding area. On lateral view this forms a slight concavity in the surface of the occipital lobe.

The basal surface of the brain shows no marked anomalies. The collateral sulcus on the left is interrupted about midway by an annectant between it and the fourth temporal convolution. The typical H-shaped sulcation on the orbital surface is absent; the fissures are mainly sagittal in direction, and near the lateral margin, leaving the middle orbital gyrus unusually wide. The lateral view of the region shows the so-called "keel-formation" of the *gyri recti.*

The mesial surfaces of the hemispheres are striking particularly on account of the unusual similarity in their configuration. The cuneate lobule on the left is somewhat smaller than that on the right.

The corpus callosum is slightly thinned in its posterior half.

There is no middle commissure.

Gross frontal sections show the cortex of fair and uniform width throughout.

There is some apparent atrophy at the anterior temporo-insular junction, which leaves the adjacent parts unapproximated.

The cavity of the right lateral ventricle is larger in all its diameters than that on the left, although the difference is only moderate in degree.

There is nothing remarkable about the basal nuclei or the subthalamic structures.

d) Microscopic Examination.

The Weigert-Myelin sheath preparations show good fiber connections in all areas examined. Comparisons were made with the drawings in Campbell's "Localization in Cerebral Function." Evidences of gliosis were as follows: —

The prefrontal tissue shows a moderate increase in the plexiform layer, involving not only an increase in a number of cells but in the number of fibrils. There is no gliosis elsewhere. Although there are frequent ganglion cells in the white matter, there is no notable neuroglia increase in this locus. The prefrontal region showed occasional bi-nucleate neuroglia cells supplied with fibrils in the plexiform layer; but there is no marked gliosis elsewhere.

The same description fits the postcentral tissue, except that there are groups of satellite cells in various layers (right postcentral) as a rule surrounding the small nerve cells.

The superior and inferior parietal regions show similar glia pictures in the plexiform layer. From the standpoint of gliosis, accordingly, the cerebral cortex of this case exhibits in all areas, so far as examined, a moderate degree of cellular and fibrillar gliosis of the plexiform or subpial layer, but fails to show gliosis in other layers, except in the specimens examined from the right postcentral region.

As against the neuroglia picture, we may consider the nerve cell pictures. Of course the excellent preservation of the fiber connections as above noted would indicate that there should be no extensive degree of cell loss. What has so far been found may be summed up as follows:

The prefrontal region (for example, on the right side) shows a suggestion of thinning out of the nerve cells in the supragranular layers; at the summit of the gyrus the cells here are often small and deeply stained.

The large pyramidal cells of the infragranular region seem also thinned out. Similar slight degrees of thinning out of the cells are found in the supragranular pyramidal layers of the precentral region (upper portion right side); identical findings in lesser degree in the postcentral region and in larger degree of the superior parietal and inferior parietal regions. On the whole, the evidence of nerve cell loss or absence is distinct but slight. On the whole the supragranular layers appear to be more involved in this cell loss or absence than the infragranular layers. It is to be noted also that no important degree of satellitosis accompanies this cell loss, if we except the area examined from the right postcentral region.

A common finding in numerous areas is the presence of dislocated ganglion cells in the white matter. These were noted as frequent in right upper precentral, right postcentral, right inferior parietal, right superior frontal, and right prefrontal areas.

As for acute changes there was apparently a degree of chromatolysis in the larger cells in the frontal region, but not elsewhere.

e) Anatomical and Histological Summary.

Frontal and occipital cruciate asymmetry suggest inborn or very early acquired lesion. This case may well have been one of Dementia Praecox grafted upon a subnormal or moron condition and the microscopy is accordingly of interest. As in the previous case, (IV), so here there are frequent dislocated ganglion cells in the white matter. The gliosis and satellitosis of irregular distribution are con-

sistent enough with the hypothesis of acquired lesion. Nerve cell loss might be interpreted in either direction as due to fundamental absence or to early or late degeneration. In various places, the supra granular layers are subject to thinning out without any important degree of satellitosis (exception right post central region). Our best argument therefore for inborn lesion is the dislocated ganglion cells in the white matter, with the thinning out of cells in the supragranular layers as a not very strong secondary argument. Of course it has sometimes been held that satellitosis should be found alongside the cell loss in Dementia Praecox. Special studies in another series of cases, to be published in the Transactions of the Association of American Physicians for 1916, indicate, however, that cell loss and satellitosis do not necessarily proceed *pari passu* and that, in point of fact, in not infrequent instances, the cell loss proceeds independently of reaction. In some instances, it might be argued that the tissues passed through a phase of satellitosis followed by a phase in which nothing but cell loss can be demonstrated. An argument of this nature has been submitted in a paper published in the above mentioned Transactions for 1915. At any rate, it would probably be agreed by all that in Senile Dementia there are cases in which nerve cells slowly disappear without neuroglia reaction. The present case must therefore be used as another instance of the fundamental difficulty which attends a decision as to whether a case is one of primary or secondary amentia, even when a comparatively full anatomical and microscopic analysis is available.

Synopsis in Findings of Case VI.

a) Clinical.

1. **Physical Examination.**— Born in 1887; died Oct. 19, 1913. Slightly microcephalic; tall and rather slight; death from pulmonary tuberculosis. Further data are given in the description of the autopsy, below, in which the following features are of interest from the point of view of the pathological anatomist. The number of scars of "tissue paper" appearance over extremities, back, and head, suggested syphilis. There were also numerous superficially palpable lymphnodes; mal-formed ears; prominent mastoid processes; high palatine arch; asymmetry of face, especially of nose and jaw; under developed hands and genitalia; redundant prepuce; absence of right vocal cord; hypoplastic aorta, and accessory spleens. No history of epilepsy.

2. **Family History.**— Unknown. There is some suspicion of syphilis in the father, but the suspicion has not been well established. Patient had been three years in almshouse at Tewksbury before admission to the Massachusetts School, May 27, 1893.

3. **Personal Developmental History.**— Is said to have had measles. At time of application, at 6 years of age, he is said to have just begun to talk, and as understanding a good deal of what was said to him. Patient is described as very restless and nervous; is usually rushing around; inclined to be destructive, especially of cloth; soils and wets himself several times a day and every night. He would spend his time destroying shirtsleeves and coatsleeves, unravelling his stockings, and the like.

4 and 5 and 6. **School Progress and Examinations. Practical Knowledge.**— Progress at the Massachusetts School was slight. Patient was placed in the training classes but did not advance much. He was able to do simple outdoor manual work. (He could help to clear land, use grubhoe, dig up stones, and the like, and was able to polish floors and carry bundles.) Was able to feed himself with a spoon although he spilled much of the food. Needed to be helped in dressing and undressing.

7. **Economic Efficiency.**— Nil.

8. **Social History and Reactions.**— Unable to speak words. Made curious squeaking attempts at speech if people that he liked were about him.

9. **Moral Reactions.**— If something patient did happened to annoy the caretakers, patient seemed to take pleasure in their annoyance. Patient had the typical idiotic grin when spoken to.

10. Psychological Tests.— Approximately 2. This case, by reason of his aphasia (compare absence of one vocal cord), yielded a poorer impression of his capacity than was perhaps justified. In some respects, patient appears to have been above the rank of idiocy and to fit into the lower grade of imbecility.

Waverley School History.— Patient's tuberculosis resisted outoor treatment.

b) Autopsy.

Body.— That of a slenderly built emaciated white male 158 cm. in length. The *skin* is bluish gray, and abrasions occur over elbows, hands and shins, irregularly. There are also quantities of "tissue paper" *scars* over lower legs and back and scalp varying in size from 0.8–1.5 cm. in diameter. *Lymph nodes* palpable in neck and groins. *Rigor mortis* absent, no body heat. Decomposition bands in flanks.

The head is markedly out of proportion, being long and narrow (occipitomental 56 cm.) the *ears* flare and the measurement from superior portion of helix to lobule inclusive is 5.5 cm. × 4.0 cm. from tragus to lateral side of helix, with a wide and flat antitragus and a minute tragus. Mastoid processes unduly prominent. *Palatine arch* high, teeth in fair condition. *Nose* is deflected to right and the angle of the lower jaw on this side is markedly prominent, the line of the left being longer and the angle less noticeable. The *hands* appear larger than would be proportionate with the arms, and the finger nails are minute, measuring 0.6 cm. in height showing evidences of much nibbling and the eminences below the thumb are absent — giving the palmar surfaces a remarkably undeveloped appearance. Legs not notable. *Genitals:* The scrotum short and broad, extending in direction of but not reaching anal surface. Penis short and thick. Prepuce long, glands very red. No edema; a decubitus measuring 0.4 cm. over sacrum is fairly superficial.

Ventral section.— Panniculus nil. Muscles red, peritoneum shows post mortem change. Omeutnm contains no fat, transverse colon extends in a V-shape with apex downward into pelvic cavity. Stomach 14 cm., *liver* 8.0 cm. below costal margin. *Spleen* free; *mesenteric lymph nodes* remarkable in size and number, some measuring 3 cm. in length. Appendix 8.0 cm. in length — has a mesentery of its own. Prostate not enlarged, no free fluid in the cavity. Diaphragm arches to the 3d interspace on the right, the 4th on the left.

Thorax.— Mammary vessels not sclerosed, sternum measures 3.0 cm. on frontal aspect, 5.5 cm. on visceral. Bone marrow of sternum rich red, smears stained show excess of eosinophiles and few giant cells. No nucleated red cells.

Pleuric cavities obliterated at apices, anterior edges of lungs overlap in median line: pericardium not thickened.

Heart.— (No weights taken), estimated 250 grams. No fat in epicardium. Myocardium greenish red (p. m.). Coronaries show no change. Section shows right auricle and ventricle filled with cruor clot. Myocardium *clastic*, not friable.

Measurements: T. V., 13.0 cm. P. V., 6.0 cm. L. V., 1.0 cm.
M. V., 10.0 cm. A. V., 6.5 cm. R. V., 0.4 cm.

Endocardium not remarkable. Some slight thickening in aortic cusps below corpus aurantii.

Lungs.— Not weighed. Over surface of left there are areas which show as slightly umbilicated centrally, red edges measuring 0.4 cm. Cut section shows a spongy parenchyma, with thickened bronchi in the lower portion and a gush of yellowish puriform material follows knife cut at apex. Stained smear shows tubercle bacilli in countless numbers.

Same process in right lung, possibly more extensive. Peribronchial lymph nodes not remarkable and showed no areas of infiltration on section.

Organs of the neck.— The tongue is heavily coated. The epiglottis is thick and yellowish and bathed in pus. The aryteno-epiglottidean folds are ragged on their free edges. From above superior and

inferior vocal cords appear unequal, and on section there are none on the right, being replaced by small white nodes which extend over the whole cavity of the larynx above the cord district. On the left a ventricle is the only mark by which a superior and inferior vocal cord may be inferred. *Thyroid* not notable. No thymus nor parathyroids seen. *Aorta* is remarkably narrow (3.5 cm. in circumference) and very elastic.

Abdomen.— *Spleen* not weighed. Estimated weight 100 grams. Somewhat autolysed, capsule not thickened. Pulp soft, trabeculae and malpighian bodies not notable. On the inferior edge two small accessory spleens hang, one is 0.2 cm., another 0.8 cm. in diameter.

Adrenals.— Remarkably small, not over 1.5 cm. at the widest part on cut section.

Kidneys.— Not weighed — estimated 200 grams. No perirenal fat; capsule stringy and non-adherent, cortex swells slightly over capsule, color of medulla and cortex homogeneous (from p. m. change). Pelves not notable.

Liver.— Not weighed — estimated 1500 grams. Capsule not thickened. Streaks of yellow occur on surface, organ friable but holds its own weight, inferior edges sharp. No stone in gall-bladder.

Pancreas.— Not examined.

Gastro-intestinal tract.— Not examined. Were deep greenish red from post-mortem change.

Genito-urinary tract.— Prostrate not enlarged. Bladder contains some heavy purulent material. Testis threads well.

Head.— Hair black and short; scalp adherent. Calvarium is well supplied with diploe but has curious inequalities in thickness; frontal measures 0.8–1.0 cm., temporal 0.2–0.8 cm. (in strikingly irregular alternations), occipital 0.9 cm. The dura is not adherent except along the superior longitudinal sinus, and is thickened only along the middle meningeal distribution and the convolutions are plainly seen through it. *Pia mater* is exceedingly thin and delicate, without edema; the twigs of the arterial capillaries are very brilliantly outlined. There are but two large veins on the vertex, these correspond very nearly to fissure of Rolando and are distended. The hemispheres are apparently equal in size with the exception of a bilobed occipital lobe on the right. There seems to be no cerebrospinal fluid. The *brain sags* on a board and tends to assume a nearly round mass. The *convolutional* pattern is simple and the sulci *shallow*, particularly is this so on the *right*, involving the motor and parietal lobule, and seeming to limit the frontal pole, the left parietal lobule having a more complex configuration and the frontal pole a greater extension. Smear from this portion shows striking loss of nerve cells; and infiltration of vessel walls with lymphocytes and polynuclear cells.

At the base.— The 2nd left temporal convolution at the tip has a yellow and denuded look under the pia and is somewhat firmer to the touch. Question of tubercle. Smear from this portion shows cellular detritus. No organisms.

The denuded area on the right temporal tip is artefact. The *basal vessels* are slightly hazy but no buds nor abnormal branching of arteries seen. The *pia* at base binds the 3rd and 4th nerves to the anterior perforated space. *Cerebellum* slightly softened, the olivary bodies are fairly firm as are the occipital tips. Remainder of brain (with exception noted) fairly normal in consistence. Pituitary pigmented on section. Ganglions negative. Petrous portion temporal bone thick. Left ear drum has a hole 0.3 cm. in diameter in its center with white and indurated edges, and smear shows tubercle bacilli. Right negative. Optic nerves show brilliant injection of retinal arteries with question of blurred outline of nerve head. There are irregularities in bone in the middle meningeal distribution. Brain weight, 1130 grams.

Cord.— Not notable.

Anatomical Diagnosis.

Emaciation.
Abrasions over extremities.
?Syphilitic scars over extremities and back.
Scars over head.
Palpable lymphnodes (superficial).
Dolichocephalic head.
Malformed ears.
Prominence of mastoid.
Palatine arch high.
Asymmetry face (nose and jaw).
Nails nibbled below the mid line.
Hands undeveloped.
Redundant prepuce.
Genitals undeveloped.
Decubitus superficial (sacral).
Gastroptosis.
Splanchnoptosis.
Lymphnodes large and prominent (reversion?).
Chronic fibrous pleuritis.
Pulmonary tuberculosis.
Slight chronic fibrous endocarditis (aortic valve).
Tuberculous epiglottis.
Tuberculous aryteno-epiglotidean folds.
?Aplasia vocal cords.
?Tuberculosis of vocal cords.
Small aorta.
Autolysis of spleen.
Accessory spleens.
Small adrenals.
Slight acute parenchymatous nephritis.
Cystitis.
Calvarium unequal in thickness.
Calvarium brittle.
Focal chronic pachymeningitis.
Slight anomaly right occipital tip.
?Hydrocephalus.
Inequality of convolutional pattern, particularly right side.
Slight basal leptomeningitis.
Tuberculous ulcer left ear drum.
Pigmentation of pituitary.
?Choked discs.
Brain weight: 1130 grams.

c) Special Anatomical Description of Brain.

The brain is symmetrical; the frontal lobes small and pointed; the occipital lobes short, and their tips point away from the median line.

The pia is generally slightly thickened, with numerous arachnoid villi along the superior longitudinal fissure on both sides. The pial vessels are moderately injected, including the capillaries.

The convolutions throughout are broad, well-rounded, and there is a noticeable lack of complexity.

In the right frontal lobe, the first convolution is much narrower than the second or third; the two latter are not clearly defined, and are very scantily fissured. On the left there are apparently four longitudinal divisions, and each is somewhat more complex than on the right. The third left frontal is not remarkable.

The central convolutions are of good width, and well-rounded. The precentral is connected about midway its length with the base of the second frontal by a recurved annectant. The postcentral convolution is narrow at its upper extremity on both sides, and again at the junction of the middle and lower thirds on the right. The fissure of Rolando forms little less than a right angle with the superior longitudinal fissure. Its upper extremity cuts the superior margin on the left and on the right extends well over on the mesial surface. The lower extremity cuts the operculum on both sides and is lost in the Sylvian fissure.

The parietal lobes are marked by a very irregular pattern. The sulci are generally arranged in a transverse direction. The superior parietal lobules are broad; the right one somewhat more so than the left, and both present very few secondary fissures.

The sulcal pattern of the temporal lobes is rather irregular; on the right the superior gyrus is narrow

and loosely convoluted. The superior sulcus is distinct and complete. The second convolution is broad with very few fissures, and these are confined mainly to the posterior part of the gyrus. On the left the limiting sulci are indistinct, but on the whole the fissure-complex is less simple.

Of the mesial surfaces, the right is more complex; the cuneate lobule is larger and presents more fissures. The right calloso-marginal fissure is complete from its subrostral origin to the marginal extremity, while on the left it is interrupted several times by annectants. The corpus callosum is thin throughout its entire length.

The most evident peculiarity of the orbital surfaces is the difference in width of the gyri recti. The right is nearly twice as broad as the left, and extends beyond the median line to the extent that its midpoint corresponds in position to that of the optic chiasm.

The pyriform lobules are nearly uniform in size; the collateral fissure is continuous with the fissura rhinica on the right, but fails to join on the left. Altogether the left temporal lobe presents the appearance of better development than the right.

Frontal sections show no focal lesions. The cortex is of fair width, and the white matter comparatively small in amount. All the sulci in the temporal lobe are deep, and the gyri narrow, finger-like processes.

There is no enlargement of the ventricles, nor other anomaly apparent.

d) Microscopic Examination.

From the partial microscopic examination, the following is abstracted:

The findings in numerous areas yield a somewhat general picture. There is, as a rule, an increase of neuroglia cells in the white substance. Even the vessels are lined by an accumulation of neuroglia cells. There is a considerable satellitosis in all layers, including the plexiform layer, but there is no tendency to an excess of gliosis in the plexiform layer. There is a tendency to decrease of nerve cells so far as qualitative estimate can suffice in the diagnosis in most areas. The frontal areas show a smaller degree of scarcity of cells than numerous other areas. On the whole, the qualitative estimate of neuroglia cell increase rather indicates that the left side is more affected than the right (for example, left insula, left superior parietal, left pyriform lobule, left transverse temporal).

The findings of particular interest are as follows: — First, rod cells in the cortex of the right upper post central region, a region which also shows a moderate degree of cellular gliosis in the white matter; but this latter gliosis was characterized by the presence of a great many glia nuclei of unusual shape. The area showed very general thinning out of the cells in all layers, particularly at the summit of the gyrus. Frequently cells are found with an appearance of degeneration. Rod cells are also found in the right pyriform lobule.

Secondly; the finding of one large vessel in the right precentral region surrounded by a relatively heavy collection of lymphocytes; other vessels show numbers of green pigment-laden phagocytes. This area shows considerable satellitosis in all layers; the satellite cells are particularly noteworthy about the large pyramidal cells. There is an increase in neuroglia cells in the plexiform layer with a focal tendency. Of course in connection with a suspicion of brain syphilis, which we may consider proper to raise in connection with the lymphocytosis above noted, especial attention was given to the observation of other foci of lymphocytosis. Several other examples of infiltrated blood vessels were found in the frontal region and with a tendency to accretions in the vertical region.

e) Anatomical and Histological Summary.

This brain of simple construction (1130 grams) is comparatively symmetrical. Its corpus callosum is thinned throughout. The white matter of the centrum semi ovale is comparatively small in amount. It does not appear that the ventricles are enlarged. Microscopically once more we discover a marked

gliosis, with considerable satellitosis corresponding with a certain decrease of nerve cells, if we may judge qualitatively of such loss. On the whole the left side is slightly more affected than the right. We raised the hypothesis in Case IV as to old syphilis, basing our suggestion upon the presence of the effects of perivascular infiltration in posterior fissure of the spinal cord. If we follow the same line of argument in Case VI, we should call attention to the effects of lymphocytosis about a large vessel in the right precentral region. The presence of rod cells in the cortex of the right upper post central region as well as in the right pyriform lobule may perhaps be regarded as evidence running somewhat in the same direction. These rod cells are characteristic of certain cases of General Paresis, although neuropathologists do not customarily make a diagnosis of brain syphilis upon the presence of rod cells. It may be that both the focal lymphocytosis and the occurrence of rod cells are based upon some other condition than syphilis, but at all events, the lesions are of an acquired nature and go to indicate the presence of at least a slight degree of active chronic, inflammatory processes. It is possible to think of tuberculosis as responsible for the lymphocytosis and it may be for the rod cell deposits, since this case showed widely distributed tuberculosis, pulmonary, epiglottis, vocal cords, left ear drum, etc. A review of the anatomical diagnoses in the case will show the presence of numerous stigmata.

Synopsis of Findings in Case VII.

a) Clinical.

1. Physical Examination.— At 15 years of age, patient was five feet tall and weighed 97½ pounds. She was born May 5, 1888, and died March 17, 1914. Patient is described as undersized, poorly nourished, anaemic, slightly deaf; as having a small cranium, with circumference of 19¾ inches, and cephalic index of 84. The face was asymmetrical, the eyes closely set, the teeth irregular, the palate high, and narrow arch. From the autopsy report, given in full below, the following features are culled: Absence of axillary hair, anomalous distribution of pubic hair, rudimentary left nipple, anomaly of toes, deflection of nose to left, dental ridges (mal-nutrition?), ovaries very large, anomaly of kidney; anomaly of sympathetic gangliated cord.

2. Family History.— Patient's father was born in New York City, of French extraction; the mother was born in England. The father was 37 years old at the birth of the child; the mother was 42, and had had four children before the birth of the present case. Three of the children had died before 1893: two girls, described as dying of marasmus, and one boy as dying of croup at the age of 5 years. It is stated that there was no deformity of body or mental deficiency in the other children. The mother is described as never having been very strong, and as subject to fainting fits. She is stated to have been in excellent health until the sudden death of the boy above mentioned, which death took place when she was 7 months pregnant with the present patient. The mother states that she fell off a small box 24 hours before delivery, and that the patient was born at 8 months. An aunt of the patient, the mother's oldest sister, is stated to have become paralyzed at the age of 24 years, and remained in this paralyzed condition until her death, at 42, of consumption.

3. Personal Developmental History.— The patient was carried on a pillow for a year, as she could not hold her head up. Patient began to walk at 3½ years, and became very active. At 5 years, a few words, such as "ma," "pa," "money," "goodbye," "bad boy," were said. The patient is described as having tried very hard to say more words but as being unable to shape them. When desiring to attract attention to something, she would say "Bookit." At the age of 5, she had learned to use a spoon and fork, and kept her person clean. She was restless in sleep. There was a history of middle ear disease on both sides in early childhood. Patient was a mouth-breather, with large tonsils.

4 and 5. School Progress and Examinations.— Patient upon admission was unable to take the regular school work and was placed in the training classes; although kept in these classes for several

years, she made very little advance. She learned to march in step and could play very simple games. She could not be taught to match color and form.

6. **Practical Knowledge.**— When admitted to the school, the patient was untidy, but became thoroughly tidy after training. She was able to feed herself, and to dress and undress herself, although careless with the clothing and inclined to be destructive thereof. Patient learned to do simple work with the hands, such as cutting paper and cloth, but could not cut to a line.

7. **Economic Efficiency.**— Nil. Patient was able to do simple domestic work, such as dusting and rubbing the floor, and could make a bed with help.

8. **Social History and Reactions.**— Patient would often walk towards a person as if about to speak, and then draw back, repeating this action several times in succession. When excited, patient would jump up and down and scream, whether pleased or displeased, and would laugh and cry at the same time.

9. **Moral Reactions.**— Patient is described as having been of a good temper during her Waverley School life. She was fond of music and liked to look at picturebooks. Was apparently perfectly happy if given a doll or other toy. She was particular about food, liking goodies. She was always rather excitable and noisy, and interpretation of her emotion was difficult on account of the identity of reactions when pleased or displeased. Patient gave the impression of laughing and crying at the same time. Patient masturbated.

10. **Psychological Tests.**— Patient's mental age was 5 years.

Waverley School History.— Patient was admitted to the school, June 20, 1895, and after remaining a few months, was taken home by her mother. She was re-admitted in 1896, at the age of 8 years. She was very seldom sick at the school, dragged her feet, and stood and sat in a roundshouldered attitude. She would march in step if some one put hands on her shoulders in the proper way. Patient is described as running about in a lackadaisical manner, and as having a habit of skipping with one foot. She played relay races well, but was unable to see the point of some of the simple circle games. Patient was addicted to talking, and was more or less troublesome. She showed a number of peculiar movements; for example, she would roll and pick the front of her dress with her hands, and finish the process by holding a bunch of clothing in her left hand, passing her right hand in circles over her head. Another characteristic action was pointing her finger downward with arm extended, and repetition of "Yah, mamma, yah." Now and then she would shake her head and squint her eyes. Toward the close of life she had lost much weight and apparently also in height. Tuberculosis was made out in March, 1914, just before her death, involving the entire left lung and right apex.

b) Autopsy.

Autopsy.— Head, trunk and cord, Dr. M. M. Canavan.

Body.— That of a somewhat *emaciated*, slender white female, 146 cm. in length. Skin *waxy yellow white* in color. There is an *abrasion* over the right cheek, punctate in character, 2 cm. in diameter. No decubitus scars, edema, nor palpable lymph nodes. *Axillary hair* absent, *pubic hair*, yellow, grows in an eliptoid manner 4 cm. above the symphysis, and cylindrically over the labia majora. *Left nipple* appears rudimentary. 3d and 4th *toes* on both feet much shorter than 1st or 2nd.

Abdomen distended and gives hollow note on percussion.

Pupils equal, 0.3 cm. Nose deflected slightly to left. *Upper teeth* slightly uneven, (suggestion of early nutrition disease) ridged, but no trace of uneven cutting edge. Orbital arches flattened, forehead measures 8 cm. in height above them and is a straight, not a curved line.

Ventral section.— *Panniculus* nil. *Muscles* in remarkable rigor. *Peritoneum* shining and grey. *Stomach* 20 cm., *Liver* 11 cm. below ensiform. *Spleen* large and free from adhesions. *Appendix* 5.5 cm. in length, somewhat injected, has a mesentery of its own. *Mesenteric lymph nodes* not enlarged. *Ad-*

hesions exist between lower inferior edge of liver and the transverse and ascending colon. Some free fluid — estimated 150 cc. thin and pale in color exists in peritoneal cavity.

Pelvis.— The uterus, 5 cm. in length, is in an anterior position; the right ovary measures 5 × 2.5 × 1.0 cm.; the left ovary measures 3 × 1.5 × 1.0 cm.

Diaphragm.— Reaches to the 5th interspace on right, to the 6th rib on left. Ribs are remarkably close together — hardly an intercostal space that was more than 0.5 cm. in width.

Thorax.— Lungs adhere to chest wall closely showing on the right a yellow puriform open space, and the left is evenly consolidated on palpation with recent fibrin at apex. Lungs not removed but cut in situ with the cavitation of right apex seen: it is extensive.

Heart.— Assumes circular form — epicardial fat scant — coronaries small — not sclerosed. Myocardium grey white. Endocardium not notable.

Valve measurements: T V, 10.0 cm. P V, 6.0 cm. L V, 1.0 cm.
M V, 9.0 " A V, 5.0 " R V, 0.5 "

The aortic valve is miniature in size of leaflets, and there is some sclerosis of the origins of the coronaries. The arch of the aorta is smooth — diameter of arch 2.0 cm.

Organs of neck.— Not removed.

Abdomen: Spleen.— 13 × 9 × 3 cm. Markedly large; trabeculae distinct — pulp slightly watery and pale. No Malpighian bodies.

Adrenals.— None seen.

Kidneys.— Right much smaller than left; contains only three pyramids. Capsule thick, cortex pale and does not show injection. Pelvis very large — somewhat injected.

Liver.— Somewhat large. Capsule of Glisson is thickened, inferior edges fairly blunt. Color red-brown. Nothing of note on section. Gall-bladder wall white and edematous. No stones.

Pancreas.— Not examined.

Gastro-intestinal tract.— Stomach large and distended. Rugae not present. Intestines negative throughout.

Genito-urinary tract.— Ovaries contain pigmented scars — (has not menstruated for a year).

Retroperitoneal tissues.— Peripheral nerves negative. Sympathetic chain seems vastly broken up into fine threads.

Head.— *Hair* fine and abundant if short. Scalp pale, scanty in fat. *Temporal muscles* fairly well developed. Calvarium 0.3 cm. frontal, 0.1–0.3 cm. temporal, 0.4 cm. occipital; it is very dense and eburnated; no diploe. At the junction of the coronal and sagittal sutures the *dura* is markedly adherent (the brain herniating through small points of cut dura on forcible removal) and the dura is thickened from this central point to the area of middle meningeal distribution. Vertex is ball shaped and small. *Pia mater* is thin and delicate except over sulci and vertex where faint hazing appears.

Hemispheres equal but appear very shortened in the frontal portion as though the whole prefrontal area were missing. *Convolutions* are simple and shallow, running with no complexity to the longitudinal sinus. *Post rolandic* gyri simple in pattern. No inequalities on palpation. At the *base* the temporal lobes are exceedingly *plump*, and make the distance between their free edges and the frontal pole appear very short.

Basal arteries clear.

Optic and 3d nerves negative.

Gasserian ganglions negative.

Pituitary softened. Middle ears — left drum ruptured, right not seen.

Cerebellum, pons and cord negative.

Brain weight, 1270 gms.

Anatomical Diagnosis.

Emaciation.
Abrasion of right cheek.
Absence of axillary hair.
Anomaly pubic hair.
Rudimentary left nipple.
Anomaly of toes.
Distention of abdomen.
Nose deflected to left.
Malnutrition ridges on teeth.
Forehead high and flat.
Splanchnoptosis.
Slight injection of appendix.
Chronic peri-colitis.
Slight ascites.
Ovaries very large.
Ribs closely set.
Chronic adhesive pleuritis.
Pulmonary tuberculosis, (advanced).
Cloudy swelling of heart muscles.
Right heart hypertrophy.
Aortic stenosis.
Anomalous kidney.
Chronic perihepatitis.
Chronic cholecystitis.
Anomalous sympathetic ganglionated cord.
Calvarium dense.
Chronic external adhesive pachymeningitis (focal).
Chronic fibrous pachymeningitis — (middle meningeal).
Slight chronic leptomeningitis sulcal and vertex.
Anomalous frontal poles (shortening).
Simplicity of convolutions.

c) Special Anatomical Description of Brain.

A more detailed description of the brain is as follows: — The brain is of fair size and suits the dolichocephalic type of skull. The dura mater was attached rather more firmly than usual to the pia mater and the brain by means of numerous pial vessels and their investments. There is no evidence of arteriosclerosis. The pia mater is everywhere slightly thickened and of a dull, cloudy appearance.

There is slight asymmetry of the two hemispheres; the right is shorter than the left, and the right occipital lobe points away from the median line. The convolutions are everywhere of fair width and usually well rounded and approximated.

In the frontal lobe, the dividing sulci are distinct. On both sides the first sulcus cuts across the precentral gyrus. The second frontal gyrus is broader than the first on both sides.

The central convolutions are quite narrow, the postcentral notably so.

The pattern of the parietal lobes is in general transverse in direction; the interparietal sulci are interrupted by an annectant between the postcentral gyrus and the superior parietal lobule on both sides. This leaves the ascending postcentral sulcus separate from the descending portion and the horizontal branch. On the right, at the junction of the descending and horizontal interparietal sulci, there is a distinct rosette formation. The size of the two parietal lobules, superior and inferior, is nearly the same on both sides.

The occipital surfaces are not notable except for the slight asymmetry already remarked upon.

On the base of the brain the orbital surface presents the usual triradiate sulcal pattern.

The collateral fissure on both sides is continuous with the fissura rhinica (Turner), thus separating the pyriform lobule entirely from the temporal lobe. The right pyriform lobule appears somewhat larger on the right than on the left.

The mesial surfaces present no remarkably anomalous cortical pattern. The corpus callosum is narrow in its horizontal portion as compared with the rostrum and splenium. There is no middle commissure.

The frontal sections show no pathological lesions. The cortex is everywhere of good width. There appears to be a rather smaller corona radiata than one usually finds. The basal gray matter is present in the usual amount.

The callosal angles of the lateral ventricles are rounded, but there is no definite enlargement of the ventricle cavity.

The cerebellum, pons and medulla are negative to gross examination.

d) Microscopic Examination.

The results of partial microscopic examination may be stated as follows:—

The most striking and general characteristic of the tissues is the generalized gliosis involving both an increase of cells and increase of fibrils. This gliosis affects the spinal cord as well as encephalic tissues. There is a suggestion of narrowing of the cortex cerebri throughout and a crowding together of the laminae. The blood vessels are everywhere prominent; this appearance is partly due to thickening but largely due to the frequency of perivascular gliosis.

One of the most important findings in the case is that of rod cells such as were found in case VI (although in case VI there are a few foci of perivascular infiltrations). The variations in the different areas examined are quantitative rather than qualitative. The rod cells are found most frequently in the precentral, post central, calcarine and temporal areas.

One area, the right transverse temporal, shows a canalized thrombus in a small vessel, with a vacuole or approach to a small glia-lined cyst nearby. There is a small acute hemorrhage under the pia mater of the right angular gyrus. The right superior frontal region appears to show a larger degree of cell atrophy (with numerous shadow cells) than is found elsewhere.

e) Anatomical and Histological Summary.

This brain of an imbecile weighed 1270 grams, is fairly complex in construction, of fair size, with slight asymmetry and a narrow corpus callosum as to its horizontal part. There is apparently no definite enlargement of the ventricular cavity. Microscopically there is a generalized gliosis which involves not only the encephalic tissues but the spinal cord. The most important finding in the case is that of rod cells, as in Case VI, but the perivascular infiltrations of Case VI are not present in Case VII. The rod cells are more widely distributed in the present case.

A canalized thrombus in one region may be regarded as another sign of acquired lesion; but as to the date of this lesion nothing can be said. Note is made below of the fact that the mental age of this patient, set in a general way at 5 years, was regarded in some respects as over-estimated since in a number of directions the patient appeared to verge upon idiocy.

Synopsis of Findings of Case VIII.

a) Clinical.

This case will probably be reported *in extenso* elsewhere in collaboration with Dr. G. B. Magrath, Medical Examiner of Suffolk County, who performed the autopsy in pursuance of official duty. We have acted as his deputies in executing our brain studies.

The case belongs in Dr. Magrath's series of "judicial homicides" having been electrocuted for murder. A number of more or less competent persons regarded the subject as "defective." Further details are reserved for the present. The brain is presented as a foil to the others of the series. If "feeble-mindedness" is a term roughly equivalent to inability of self-support, then this subject should perhaps not be regarded as feeble-minded. If "defect" may be used to include lack of "normal inhibition," then probably this case might classify as defective.

The subject was physically well developed, had been a "nervous" and "bad" boy, could read and write, had marked practical ability in certain ways, earned a sort of living, was criminalistic, had an

ungovernable temper and was easily overcome by alcohol. There is no means of knowing what his mental "grade" would have been; we have placed it at 12 plus.

b) Autopsy.

Gross findings reserved.

c) Special Anatomical Description of Brain.

There is slight asymmetry of the occipital lobes; the right is thin and points away from the median line, while the left does not.

The pia is thickened and white over the vertex, and the vessels are considerably injected; particularly the larger ones.

The convolutional pattern is noticeable in that the direction of the gyri is a transverse rather than an antero-posterior one. (This has been noted as a developmental anomaly in mental defectives.) The gyri are well rounded and of fair width, but their summits are not approximated. The markings are of medium complexity in the anterior region, but less so in the part adjacent to the central lobule.

In the central region the precentral gyrus is unusually wide in its upper third; the postcentral is very narrow in its upper third, but becomes of average width below. The upper extremity of the fissure of Rolando extends barely to the border of the superior longitudinal fissure and approaches it at an acute angle. The lower extremity does not cut the operculum.

The parietal lobes are well developed with distinct markings. On the right the interparietal sulcus is typical; the postcentral portion is continuous and is intercepted at its middle point by the horizontal limb. On the left the ascending postcentral is separated by a narrow annectant from the descending and horizontal branches, which are continuous. The convolutions of the inferior parietal lobule are broader and less complex than those of the superior parietal. This lobule is marked both by longitudinal and transverse fissures. The entire lobe is characterized by the lack of approximation of the walls of the interparietal fissure and of the convolutions of the superior lobule.

The occipital lobe is not remarkable; the convolutions are of good width, fairly well rounded, their walls well approximated, and of complex arrangement.

On the orbital surface the convolutional pattern is not markedly different from that ordinarily seen.

The pyriform lobules are noticeably asymmetrical; the right is considerably wider than the left, and this is true of the entire hippocampal lobule. The rhinal fissure (Turner) is well marked. The convolutions of the extreme tip of the right occipital lobe are narrow and irregularly arranged. The sulcal pattern over the entire base of the brain is unusually symmetrical.

The main differences of configuration on the mesial surface are the presence on the left of two sulci parallel with the callosum, while on the right there is but one. On the left, the parieto-occipital fissure is wide open, disclosing an otherwise hidden convolution which dips down from the superior surface of the superior parietal lobule and turning back upon itself, joins posteriorly with the superior occipital gyrus. The sulcal complex on the right precuneus is triradiate, while that on the left is made up of four branches. The former has one fissure extending upward toward the superior margin, and two downward and outward; on the left two point upward and outward, and two downward and outward.

The callosum is of good extent and thickness except in the posterior third of the horizontal portion where there is moderate narrowing. The middle commissure is present.

The frontal sections illustrate well the lack of complete longitudinal sulci in the frontal lobes; these can be easily identified only in the most anterior portion.

There is nothing remarkable in the appearance of the cortex, nor of the basal nuclei.

The ventricles are not dilated.

d) Microscopic Examination.

The results of a partial microscopic examination may be abstracted as follows: —

Gliosis is noted in numerous areas but was rarely of marked degree and often showed a tendency to focalization. Subpial gliosis is noted in the paracentral, postcentral (more marked left), temporal pyriform, cornu ammonis, left prefrontal, right insula, and Broca areas. This subpial gliosis is, as a rule, more marked at the bottoms of the sulci or along their sides than at the tops of the gyri. It is not often of marked degree.

Perivascular gliosis is especially noted in the right precentral, right middle temporal, superior frontal, middle frontal prefrontal and Broca areas.

Satellitosis is especially noted in the paracentral, right postcentral, left cornu ammonis, superior frontal, middle frontal, prefrontal and insula regions, and to a lesser extent in several other regions. Gliosis of the white matter is especially noted in the right middle temporal, middle frontal, prefrontal, insula and Broca areas, as well as in areas noted above, showing perivascular gliosis in the white matter.

Dislocated ganglion cells are noted especially in the white matter of the right precentral area and appear in general to be rare in this case.

The only instance of exudate consists in infiltration of the space about the small blood vessels in the left prefrontal region, where cells are found highly suggestive of plasma cells. Evidence of recent cell destruction, completed or in process, are few but distinct. The notes show cells with poorly staining nuclei and surrounded at their bases by satellite cells in both paracentral regions. Satellitosis was also found in the postcentral region in considerable degree.

In the right transverse temporal gyrus, cells are occasionally found of the small pyramidal type, having a clear ring outside the nucleus, suggesting that the chromatic substance had undergone a solvent process in the cell interior. Shadow cells are found in the pyriform lobules and are here associated with cells showing the clear perinuclear rings just mentioned as found in the transverse temporal region. Other areas show occasional cell changes of the same general nature.

The total brain sections in this case show deep sulci, possibly more so on the left side; the cortex roughly averages 3 mm. in depth at the summits of the gyri and is thinner elsewhere. The white matter appears to preserve a fair proportion to the cortex. There seems to be notably less white matter on the left side of the brain. The larger vessels in the white matter are surrounded by large pigment-laden cells.

Especially interesting was a slight degree of heterotopia of the gray matter consisting in four small areas below the cortex on the superior surface of the temporal lobe. The blood vessels of the temporal lobe show dilatation of their perivascular spaces with a few large pigmented cells. The ventricles are not dilated; the corpus callosum is of proper thickness, approximately 8 mm.

It does not appear that there is any evidence of thinning out of the nerve fibers. The suggestion that the left hemisphere has comparatively less white matter than the right is based upon observations both in the anterior and posterior part of the brain.

e) Anatomical and Histological Summary.

This brain (1340 grams) in a physically healthy, executed subject, is found to show a number of minor asymmetries, such as the transverse direction of convolutional pattern in many regions. The summits of the gyri are not always approximated. The corpus callosum is of proper size except for a moderate narrowing in the posterior third of the horizontal portion, in which latter connection may be mentioned that the parietal lobules are especially characterized by the lack of approximation of the walls of the gyri.

Microscopically there is a certain tendency to slight gliosis of a focalized nature. This gliosis is more, as a rule, at the bottoms of the sulci where there is normally more fiber-producing tissue. Both the fibrillar gliosis and satellitosis are fairly widespread in cortex and white matter and there is a certain amount of perivascular gliosis. The argument for fundamental hypoplastic changes may perhaps best be based upon dislocated ganglion cells in the white matter of the right precentral area, but the rarity of such cells is conspicuous and it would not seem well to make a diagnosis of hypoplasia or heterotopia upon this single finding. Again, the single focus of perivascular infiltration by cells resembling plasma cells in the left prefrontal region is perhaps but slight evidence on which to ground the hypothesis of brain changes of an important nature in this executed criminal. A vast deal of comparative work in "normal" cases must yet be done before a decision can be rendered on such a point.

That the case had inborn peculiarities may be argued from the stigma of heterotopia in the temporal lobe and perhaps somewhat upon the deep sulci, possibly deeper on the left side, which the total brain section demonstrated. The suggestion that there is somewhat less white matter on the left side of the brain than on the right may also be entertained as indicative of inborn peculiarity. There is in the case apparently no evidence of thinning out of the nerve fibers.

An analysis of this case in comparison with others in a series of executed homicides will be profitable.

Synopsis of Findings of Case IX.

a) Clinical.

1. Physical Examination.— Patient was born August 3, 1875; admitted May 17, 1883; died August 30, 1914. The patient at autopsy showed an enlarged thyroid and cystic kidneys, but had no other anomalies. The stomach was dilated. He is described as having been a strong and healthy subject.

Voice hoarse, words pronounced in a staccato manner. Patient a little deaf; at all events, later in life.

2. Family History.— Father and mother are said to have been strong and healthy; the father 30, and the mother 32 years old at the time of the birth of this child. There had been four children before the birth of the patient, and one thereafter. One of the children had died of "summer complaint." It is stated that there are no evidences of deformity or mental deficiency in the other children. The mother died in childbirth, at 37 years. The family was American.

3. Personal Developmental History.— Patient began to walk at 2 years, and to say a few words at 5 years. His mental deficiency was noticed at 3 years of age, when it appeared that he was not trying to talk. At 8 years, when patient was admitted to the school at South Boston, he could not talk plainly and had untidy habits. He had not learned to read or write.

4 and 5. School Progress and Examinations.— Patient upon admission was placed in the training classes, and learned a good deal. He learned to do simple outdoor work on the farm, such as grub-hoeing, picking up stones, and the like. In his 25th year, he was transferred to the Colony, where he developed markedly and was regarded as fairly intelligent and industrious. His strength and abundance of health permitted him to help the blacksmith; he could measure and saw off wood, and use a plane and sandpaper. He would keep the blacksmith's tools lined up for him, and assist in shoeing oxen.

6. Practical Knowledge.— His practical knowledge is indicated by the remarks under *School Progress* (see above). Although he could use the plane, saw, and chisel well, his sense of measurement was so deficient that, for instance, he could not tell the difference between 5 and 9 inches. However, if given a piece of board 4 inches wide, patient could go and get another of the same width. If the car-

penter marked with pencil a line to which patient was to plane on a piece of board, he could plane to that mark. Practically speaking, he seemed to be better than his Binet age would indicate.

7. **Economic Efficiency.**— See above.

8. **Social History and Reactions.**— Patient was much beloved by the caretakers. He was kind-hearted and anxious to please, and after his fashion had a very keen sense of humor, apparently understanding much more than he was able to express; used nouns, active verbs, and a few descriptive words, but never learning to put sentences together.

9. **Moral Reactions.**— Patient belonged in the amiable and " altruistic " group.

10. **Psychological Tests.**— Binet tests performed in 1914 show the patient to be of a mental age of 7.

Waverley School History.— The advance of this patient under supervision and the conditions of the school, and later of the colony, was considerable, as above indicated. As the patient grew older, he was given more difficult work, which he was able to perform with fair satisfaction; this progress was of a degree rather unusual in the feeble-minded. Patient was fond of music and of humming melodies, although he did not pronounce the words.

The patient was killed by being run down by an automobile while being taken off the grounds with a group of boys on an outing. He had been in general regarded as able to take care of himself and keep out of the way of traffic, although the conditions of state roads may have been more complex than ordinary for him. There was a certain error of judgment on the part of the automobile driver.

b) Autopsy.

Body of a well built and nourished white male 5 ft., 10 in. in length (177.8 cm.). *Skin* rosy grey but shows marked number of recent abrasions as follow:

Right parietal boss is prominent and exhibits a superficial cut 5 cm. in length by 0.5 cm. in width. Hair shaved on this side. *Right mid scapular* line shows abrasions 3 × 0.5 cm. *Right elbow* 3 small ones 3 × 2 cm. in extent. *Right lumbar region* just above crest of ilium 5.0 cm. × 1 cm. *Right popliteal* space 5 × 1 cm. *Right knee* inner aspect 3 × 0.8 cm. *Right mid shin* anterior aspect 3 × 2 cm. *Left, below crest of ilium* (gleutial side) an abrasion 12 cm. in length × 2 cm. wide. *Left thigh* lower third outer aspect is another 3 × 1 cm. *Left knee* inner aspect 2 × 2 cm. *Bruises, right knee, right shin, right ankle* (inner and outer aspects), *left ankle* slight bruising 7 × 7 cm.

Rigor mortis present, in legs — not in arms. No palpable lymph nodes. *Pupils* 0.5 cm. equal. Slight amount regurgitated material left face and neck.

Ventral Section.— Fat over abdomen measures 3½ cm. Over thorax ½ cm. Muscles red. Peritoneum is bluish red in lower anterior portion and there is blood between the fascia of the rectii muscles and the peritoneum as high as 10 cm. above the symphysis. There is *free blood* in the peritoneal cavity, estimated quantity 25–40 cc. The *intestines* are dilated, the *liver* is above costal margin and lightly stained with blood. *Stomach* dilated and 10 cm. in length. *Spleen* free. *Mesenteric lymph nodes* not palpable. *Hemorrhages* exist in the layers of the mesentery. *Diaphragm* arches to the 4th rib in right to 4th on left.

Thorax.— Muscles removed from anterior thoracic wall shows much hemorrhage into the muscles between the *left clavicle* and sternum between 1st and 2nd rib. On the right between clavicle and first and between 1st and 2nd ribs, there is a separation of the clavicle and the sternum *on the left* by hemorrhagic and edematous muscle and the first rib is broken approximately 1 cm. from the junction of it to sternum. *On the right* the upper end of the chest wall sinks in a little more than the left and a fracture is present on right first rib 8 cm. from mid sternal line and the 2nd is also fractured somewhat latterly to the first. Anterior portion right chest presents subperiosteal fractures of the 7th, 8th, 9th and 10th ribs with marked hemorrhage into parietal pleura. None on the left.

Upon removal of the lungs, there is also seen marked hemorrhage in posterior surface of right pleura and underneath it the 3rd, 4th, 5th, 6th, 7th, 8th ribs are fractured about 1–3 cm. from vertebrae and 10 cm. from vertebrae the 10th and 11th ribs are also. Marked hemorrhage into posterior mediastinal tissue, on either side, also retroperitoneally in abdomen. *Heart* weight 12½ oz. (354 grams). Epicardial fat scant, muscle somewhat pale but firm. Valves negative. Endocardium smooth.

Measurements: T.V. 5¾ in. (14 cm.) P.V. 3¼ in. (8.25 cm.) L.V. ½ in. (1.2 cm.).
M.V. 4¾ in. (12.0 cm.) A.V. 3½ in. (8.8 cm.) R.V. ⅛ in. (0.3 cm.).

Lungs.— Right 22½ oz. (638 grams). Left 20½ oz. (567 grams.). Nothing of note except old adhesions left apex and recent congestion of both lungs.

Organs of the Neck.— Thyroid markedly enlarged and protuberant; weighs 3½ oz. (99 gms.). Normal on section except right lobe which is very much congested. *Tongue, larynx, trachea, esophagus* negative.

Abdomen.— *Spleen* 6½ oz. (184 gms.). Nothing of note. Capsule not thick, pulp fairly soft, trabeculae prominent. *Adrenals* small; otherwise negative.

Kidneys 9½ oz. (269 gms.). One kidney fairly normal; the other, the left, is studded with smooth, firm glistening bodies 0.2 cm. to 0.3 cm. in diameters. Capsule peels with difficulty. Cortex reduced and replaced by these cysts which appear colloidal; the cortex is also injected. Pelves negative.

Liver 58½ oz. (1610 gms.). Nothing of note. No gall stones.

Pancreas. Very white instead of yellow and a curious softness unlike the usual gland. No hemorrhages.

Gastro-Intestinal Tract.— Negative throughout. Remains of hastily eaten vegetables in stomach. Well distended, few rugae.

Genito-Urinary Tract.— Bladder external wall very hemorrhagic; inner wall clear. Clear fluid in bladder. *Prostate* not examined. *Testes* thread well.

Retroperitoneal Tissues.— Aorta and branches smooth and elastic. No lymph nodes seen. Marked hemorrhages into the retroperitoneal tissues.

Head.— Hair brown, cropped on right. No marks on inner side of scalp. Calvarium thick; frontal ¼ in. (0.6 cm.); temporal ⅛ in. (0.3 cm.); occipital ⅜ in. (0.9 cm.). Not much diplöe. Dura thickened but not adherent. Pia thin and delicate. Convolutions simple and wide, in every location. General feeling of "normalness" to fingers, slightly putty-like at base. No atrophy nor gliosis.

At the base, a slight amount of meningitis bends the optics, the circle of Willis and the 3rd nerve together. Tips of olfactory bulbs adherent to skull. Optic nerves slightly edematous but smaller than normal. Ganglions and pituitary negative. Ears and discs not examined.

Brain wt. 54 oz. (1622 grams.). Cord not removed.

Anatomical Diagnosis.

Well nourished.
Abrasions, head, trunk, extremities.
Bruises, extremities.
Subperitoneal hemorrhages.
Free blood in peritoneal cavity.
Mesenteric hemorrhages.
Intercostal hemorrhage.
Fractures ribs, right anterior 2nd, 7th, 8th, 9th, 10th.
Fractures ribs, right posterior 3rd, 4th, 5th, 6th, 7th, 8th, 10th, and 11th.
Fractures ribs, left, 1st rib.
Slight adhesive pleuritis.
Pulmonary congestion.
Enlarged thyroid.
Cystic kidneys.
Hemorrhagic cystitis (bladder wall).
Retroperitoneal hemorrhages.
Normal brain.
Slight edema optic nerves.
Brain weight 1622 gms.
(Tigges' formula 1424.)

c) Special Anatomical Description of Brain.

The brain of this case is large and suits the dolichocephalic skull. The orbital frontal angle made between the basal and lateral aspects of the frontal lobe is unusually prominent throughout. The occipital lobes exhibit a slight degree of asymmetry; the inferior parietal lobules are slightly more prominent than the surrounding tissues.

The intermediate precentral area has the "coronal" type of construction. The gyri are in general of good width and of fairly simple pattern. The summits of the gyri do not, as a rule, well approximate; in many places the summits do not lie in the same plane; this gives the brain surface a motley, irregular configuration, somewhat suggestive of foothills.

The corpus callosum is of a good depth except in the posterior third where it is comparatively narrow. There appears to be no middle commissure.

The pia mater is slightly thickened and hazy throughout and the arachnoidal villi are moderately well developed along the vertex of the superior longitudinal fissure. There are small pinpoint white nodules in the pia mater, especially over the sulci, chiefly in the region of the superior parietal lobule.

d) Microscopic Examination.

An abstract of the most important findings yet made is as follows: —

Subpial gliosis is nowhere in evidence. Gliosis of the white matter is found in a number of areas, precentral, postcentral, temporal, occipital, frontal, but is nowhere marked. There is no perivascular gliosis except for a slight amount in the section over the right prefrontal region. Satellitosis is nowhere noted.

The fiber stains show no notable variation from the normal (as judged by drawings in Campbell's "Localization of Cerebral Function"), except that a section from the right superior temporal gyrus shows some diffuse thinning of radiary fibers; but even this appearance may possibly be regarded as artificial. We have not regarded the presence of knoblike enlargements along the course of the fibers as of pathological significance.

As might be expected from the nerve fiber and glia appearances, the nerve cell pictures are not far from normal upon qualitative examination. There are, however, a number of stratigraphical appearances consistent with the hypothesis of arrest of development. Thus the supragranular layers of the precentral, postcentral and frontal regions are not definitely laminated.

The evidences of cell loss or numerical hypoplasia in the supragranular layers is, as a rule, confined to the lower portions of these layers. The granular layers are, as a rule, distinct, where they are normally distinct and in general of a normal appearance, unless we regard the cells as slightly thinned out in some places (right upper postcentral, right superior parietal).

The infragranular layers show apparent scarcity of cells in a number of regions, for example, precentral, postcentral, occipital, superior parietal.

The above description suggests a certain irregularity in the distribution of the hypoplasia. The area which more than any other shows architectural disturbance appears to be illustrated by a section from the right superior frontal region. The cortex here shows a narrow plexiform layer but underneath a complete absence of distinct lamination. The granular layer, for example, is nowhere distinct. The scarcity of cells seems to be a definite one and of a convincing degree. The cells that persist show a variety of staining reaction — many of the cells have a pale and finely granular protoplasm. On the other hand a good many cells contain perfectly staining Nissl granulations; there is a moderate degree of phagocytosis in the perivascular spaces in this area and, as above noted, a slight cellular gliosis in the white matter.

The sections stained for the demonstration of neuroglia fibrils fail to show any convincing increase thereof. The Weigert myelin sheath stains are negative as to nerve fiber change. The prefrontal cortex on the right side shows an even more marked scarcity of cells.

A type of problem is brought up, for example, by a section from the right upper postcentral region. Here there is a number of giant pyramidal cells suggesting that there might have been an error in the labelling of the section and that it had been mistaken for a section of prccentral cortex. On the other hand, the supragranular layer in the cortex here sectioned is much more like the postcentral cortex and there seems to be no reason for supposing that in this case there was a mislabelling of sections.

A section from the right intermcdiate postcentral gyrus shows an unusual tendency to the grouping or bunching of nerve cells in compact nests containing 2–3–4 cells; sometimes the nerve cells are found overlying each other slightly.

In a section from the right superior occipital area, a number of dislocated ganglion cells are found a good way into the white matter. One of these dislocated cells was found with two nuclei. On the whole, however, in this case there are not many instances of dislocated ganglion cells in the white matter.

e) Anatomical and Histological Summary.

The brain of this imbecile of 39 years weighed 1622 grams, as is perhaps consistent with his belonging to what is sometimes termed, for practical purposes, the able-bodied or colonial type of feeble-minded. The corpus callosum is of good depth except for a narrow place in the posterior third. Microscopically we failed to secure evidence of gliosis in the subpial region nor marked evidence of gliosis in any locus. In particular, there was no evidence of satellitosis. Accordingly we may regard this case as without evidence of active phenomena, whether inflammatory, sclerotic, neuronophagic. There was one region in which there was a moderate degree of phagocytosis in the perivascular spaces. This region, the right superior frontal, was interestingly enough the region in which occurred the most marked architectural disturbance with a complete absence of distinct lamination.

This brain, accordingly, shows one of the purest examples yet seen of uncomplicated irregular arrest of development with stratigraphical, architectural alterations (right superior frontal, right upper postcentral, dislocated ganglion cells, right superior occipital) without complications of acquired disease.

Synopsis of Findings of Case X.

a) Clinical.

1. Physical Examination.— Patient was born Oct. 3, 1876; admitted Feb. 17, 1887; died of pellagra, Nov. 17, 1914. Patient was slenderly built, and well nourished up to two years before his death, when pellagra set in. From the autopsy findings, some points concerning the physique of the patient may be taken. The nose was saddleshaped and the head narrow, dolichocephalic, eyes small, ears irregular with adherent lobules, prominent abdomen. The patient was never very strong and had a poor circulation so that he could be kept out of doors only in the warm weather.

2. Family History.— The father was a native American, 25 years of age at the birth of the child. The mother was born in Canada, and was 20 years of age at the birth of the child. She had had one child before the birth of the patient, and has had two since without evidence of physical deformity or mental deficiency. There is no history of mental disorder in the family. There was nothing unusual about the pregnancy or delivery.

3. Personal Developmental History.— The patient began to walk at two years; was not able to

talk, and is described as having been of a passionate temper. Late in talking; could say very few words at time of admission.

4 and 5. **School Progress** and **Examinations.**— There was improvement physically when the patient was placed in the outdoor classes.

6 and 7. **Practical Knowledge** and **Efficiency.**— Patient could carry stones, use a grubhoe, carry bundles, and rub floors.

8. **Social History and Reactions.**— Patient had a quick temper and would attack people who got in his way. On account of his tendency to cruelty to weaker subjects, he was kept with the older boys. He could say a few words, could not talk in sentences, but used profane and obscene phrases.

9. **Moral Reactions.**— Patient was more or less irritable, violent, inclined to be noisy, and as above stated would attack persons. Masturbation.

10. **Psychological Tests.**— Binet test made in 1914 showed the patient to be 2.4 years of mental age.

Waverley School History.— The patient grew more and more troublesome as he grew older, becoming noisy and more untidy. In his thirty-seventh year, the patient began to lose weight and became emaciated, and developed a peculiar skin eruption on the back of the neck and extensor surfaces of the hands. The eruption was symmetrical and was regarded as pellagra. This eruption healed with stimulating ointments, and the patient grew physically somewhat better. Later the eruption returned and, despite outdoors treatment, a decubitus developed, finally covering a portion as large as a dinner plate, with great destruction of tissue and offensive odor.

b) Autopsy.

Body of a slenderly built and poorly developed white male, 144 cm. in length. *Skin* is yellowish grey except over the face where it is diffusely pigmented (freckles) and over the exposed portions of the head where the hair sparsely grows (frontotemporal region): here it is a deep brown color with crackled lines alternating with patches of white. This condition is especially marked in the back of the neck extending as far backward as the 7th cervical vertebra where it is abruptly replaced by normal appearing skin. The *hands* are brown, especially over the knuckles where the skin is scaling, so much that it may be lifted from the pale true skin. This pigment is apparent on the hands up to the wrists and extending in a gauntlet shape 4 cm. above the wrist where it also fades from this diffuse and dense color into isolated pigmentation (freckles) that gradually become less until the axillary region is reached. There are some scattered freckles over the chest and abdomen and upper thighs.

Over the mid-shin region of both legs there are brawny brown areas measuring 8 × 4 cm. and 12 × 6 cm. There is a bruise measuring 3 × 2 cm. over the anterior surface of the right mid-thigh; another 4 cm. above left patella.

There are *gangrenous areas* on the skin over the posterior portion of the right shoulder, measuring 4 × 3 cm., over the right trochanter major measuring 12 × 10 cm. This is markedly sloughing and extends deeply into the muscular substance. Probing this muscular substance, a soft, mushy, non-resisting tissue appears in the place of the normal firm resisting muscular tissue.

A similar area over the left trochanter major measures 8 × 6 cm. over the left shoulder, another measures 4 × 5. Also in the inner surface of each knee similar areas vary in size from 3 × 4 cm. — 1 × 1 cm. and 0.8 × 1 cm. in diameter.

A few scattered hairs over upper sternal region. Pubic hair more or less triangular in shape, grows in a straight line to the ensiform; hair also grows on inner thighs, also legs and forearms. There is scanty evidence of hair over face.

Nose slightly saddle shape with broad bridge and small alae.

The *pupils* are dilated and unequal; the right measures 0.7 cm., the left 0.6 cm. The lips show

sordes. The *teeth* are poor and there is a bloody fluid between them. (Some report of sore mouth previous to death).

Ventral section.— Fat over abdomen 1.0 cm., over thorax, 0.5 cm. Muscles pinkish. Peritoneum is grey.

Liver.— 6 cm. *Stomach* 8 cm. *Transverse colon* 15 cm. below the ensiform.

The *omentum* is very rich in fat and covers intestines completely. *Intestines* are injected and slippery to the touch. The appendix measures approximately 4 cm. and is large at its distal end. The *bladder* is not distended. There is a little free fluid in the abdominal cavity. The *mesenteric lymph nodes* not enlarged. Spleen exceedingly large. *Diaphragm* arches to the third rib on the right and to the 5th on the left.

Thorax.— Lungs adherent to the chest wall in the mid-anterior portion. There is no free fluid in the *pleuric cavities.* Pericardial sac contains slight traces of fluid.

Heart.— Assumes a circular shape on being removed. It is very flabby and it would be difficult to tell which were the left side on inspection. The descending branch of the left coronary is smooth and delicate, as is the right coronary. *Myocardium* is pale and flabby and not friable. Valves are negative except the aortic which shows a sessile red adherent body clinging to the middle of the posterior cusp measuring approximately 0.8 × 0.6 cm. No other vegetations.

Approximate measurements: TV 8.0 cm. PV 5.0 cm. LV 0.9 cm.
MV 9.0 cm. AV 8.0 cm. RV 0.3 cm.

The intima of the *aorta* is very red and firm.

Lungs.— No weights taken. Right lung has but two lobes. The upper is normal; the lower is injected on lateral surface and on section there is reddening of the lung tissue superficially. *Bronchi* are covered by reddish thin fluid. Left lung is markedly congested in the posterior portion. Pleura has been torn on removal of lung but there is no raised area around the bronchi at that point. There is a small area not more than 3 cm. in diameter where the bronchi have induration about them in the central portion of the lower lobe. *Bronchi* reddened throughout.

Organs of the Neck.— Not removed.

Abdomen.— *Spleen:* Markedly large; measures approximately 14 × 10 × 4 cm. *Capsule* not thickened but dark areas (?). Hemorrhages show through the capsule. On section the pulp is very soft, scrapes heavily and the malpighian bodies faintly descernible. The *pulp* swells above the cortex on section and there are greyish pink wedge shaped areas with their bases toward the capsule that show red external borders. *Trabeculae* indistinct.

Adrenals.— Small. The cortices are both pale yellow edematous and fade into a deeper brown at irregular intervals. The central portion is brown and there is no softening.

Kidneys.— Fairly well supplied with fat. Capsule strips easily but is opaque. The cortex measures 0.6 cm., is brilliantly and uniformly injected and red points dot the intervascular injections. At one point there is a bright yellow-grey wedge shaped body occupying the cortex for a space of 1.5 × 0.6 cm. The *pyramids* are poorly differentiated, look yellow in their centers. *Pelves* markedly congested. The other kidney not so injected but swells over capsule on section and on stripping the capsule the kidney surface is pulled away at two points. No *cysts* seen.

Liver.— Measures approximately 20 × 18 cm. The surface is mottled yellow and red. Cut surface swells slightly over the capsule. Color of the liver on section is not remarkable; shows lobules well marked but no congestion. The *gall bladder* very small; contains a few cc. of turbid yellow fluid.

Pancreas.— Not examined.

Gastro-Intestinal Tract.— Stomach dilated. Swells somewhat; edematous; no rugae. Mucous wall drips grey fluid and the wall has a granular and somewhat injected appearance. The same is true of the lower tract but no ulcers of hemorrhagic state of the intestines seen, with the exception of focal areas of injection in the upper third of the descending colon. The content of the entire intestinal tract and particularly the colon is fluid brown yellow.

Genito-Urinary Tract.— *Bladder* negative. *Prostate* not enlarged. *Testes* not examined.

Retro-Peritoneal Tissues.— The lymph nodes are not present. There are (?) hemo-lymph nodes along the aorta. The sympathetic ganglia small. The *aorta* is small in calibre and thin walled. The peripheral nerves negative.

Head.— The cranium is narrow, measures 52 cm. in circumference and the hair grows scantily over the entire scalp. The scalp is adherent and inelastic. *Calvarium* is dense and measures, frontal 0.4 cm., temporal, 0.2 cm., 0.4 cm. and occipital, 0.6 cm. Grooving for the middle meningeal arteries very narrow and shallow on the right. The *dura* is not adherent and in the frontal region one sees the convolutions through it. There is some thickening of the dura in the middle meningeal distribution. The longitudinal sinus free. The dura seems dense and fills the cranium cavity except in the frontal portion where there is a space between the frontal portion and the inner table sufficient to insert three fingers. The pia mater is somewhat injected over the vertex.

The *brain* is exceedingly compact and firm.

At the base of the brain a striking appearance is presented by the depression of the orbital portion of the frontal lobes in their central portions and the marked foreshortening or (?) absence of the prefrontal regions. The olfactory bulbs somewhat softened. The *optic nerves* slightly thinned but show no atrophy. The pia mater over the cerebellum is markedly thickened and opaque. The floor of the *4th ventricle* is clear. *Pons* and *medulla* are firm. There is marked pressure pointing over each lobus pyriformis. The *3rd ventricle* contains a quantity of clear fluid (specimen taken for gold reaction).

The *superior surface* of the brain is uniformly firmer than normal, there being no points of softening or change in consistency with the exception of the posterior portion of the left angular gyrus. The convolutions of the entire surface have a tendency to be less plump on the crowns than is normal; this is seen particularly on the right side.

The *occipital lobes* somewhat foreshortened but are flush with the cerebellum. The first temporal on the right is slightly wider than its fellow and there is a difference in the winding of the second convolution.

The *Gasserian ganglia*, the *pituitary*, the *optic discs*, *middle ears*, and *cord* negative on section.

The brain weight is 1450 grams.

Anatomical Diagnosis.

Poorly developed.
Pellagra.
Bruises, extremities.
Decubitus (Shoulder and trochanter major, knees.)
Saddle shaped nose.
Unequal pupils.
Sordes on lips.
Teeth poor.
Induration appendix.
Chronic fibrous pleuritis.
Acute vegetative endocarditis (aortic valve).
Pulmonary congestion; beginning bronchial pneumonia.
Acute bronchitis.
Acute splenitis.
Infarct spleen.
(?) Post mortem change adrenals.
Infarct of Kidney.
Acute parenchymatous nephritis.
Acute hepatitis.
Chronic gastritis.
Head narrow.
Scalp adherent.
Calvarium dense.
" thin.
Focal thickening of dura.
Injection pia.
Thickening of pia cerebellum.
Evidences of brain swelling.
General cerebral gliosis.
Superficial atrophy convolutions (flattening of crowns).
Inequalities first temporal convolutions.
Brain weight 1450 gms.
(Tigges' formula 1152 gms.)
Cord not remarkable.

c) Special Anatomical Description of Brain.

The brain in general suits the dolichocephalic skull. There is a slight occipital asymmetry such that the right lobe is shorter than the left, deviates somewhat more from the median line and has a groove upon the posterior extremity, whereas the left occipital lobe deviates slightly to the right and even somewhat overreaches the median sagittal plane (the posterior extremity of the left occipital lobe, however, deviates again leftwards).

The gyri of this brain are in general rather narrow, closely approximated except at the borders of the summits. The corpus callosum is of good width except for a somewhat narrow middle third. There is no evidence of the middle commissure. The cerebellum projects farther backward on the right than on the left (compare contralateral asymmetry of occipital lobes). The cerebellum suggests in general a reduction in size and the inferior surface of the cerebellum shows a considerable thickening of the pia mater, especially over the incisure. The pia mater of the brain in general is slightly thickened and hazy.

d) Microscopic Examination.

An abstract of important findings is as follows: —

Subpial gliosis is found in a number of areas, for example, focally in the precentral and postcentral angular gyrus; but in other areas the plexiform layer seems to be normal. The white matter is not, as a rule, affected by gliosis, although the angular gyrus of superior parietel area and frontal regions show a general increase.

Satellitosis is found in a number of areas (precentral, occipital, temporal angular, superior parietal, prefrontal.

The Weigert myelin sheath stains show a certain indistinctness in the supraradiary layers, precentral, postcentral, temporal, angular, superior frontal (here patchy). A good deal of this fiber change might be within the limit of artefact.

The nerve cells show numerical hypoplasia in a number of areas; the right upper precentral area, for example, shows very few cells in the supragranular region, aside from the outer small pyramids. This condition is less marked in the postcentral area where the distinction of supra and infragranular layers is more evident. The right superior temporal region shows marked cell scarcity and the arrangement of the cells is markedly irregular. The supragranular layer of the right angular gyrus is narrow and of a somewhat abnormal development. The infragranular layer here shows marked thinning out of cells. The right superior parietal area shows a more marked architectural disturbance in the infragranular region. The cells of the second supragranular layer are also much thinned.

The right superior frontal region shows marked lack of cells in the second and third layers. The prefrontal region, on the other hand, shows fairly well developed supra granular layers with a few foci here and there of thinning out. These foci affect all the layers. The infragranular layer also shows a tendency to patchy cell loss. This case being one of Pellagra shows a rather rich display of acute nerve cell changes, with excellent examples of the axonal reaction in the Betz cells. There was in one region (right, upper postcentral) a focus in which there were a few round cells in the perivascular spaces; it could not be made out that these were definitely lymphocytes and they may perhaps be regarded as neuroglia cells.

e) Anatomical and Histological Summary.

The brain of this case in an idiot or low imbecile of 2–4.5 years, Binet age, weighed 1450 grams, and may be regarded as fairly complex in construction. The grey matter, white matter and corpus callosum stand in fair proportion to one another, except that the gyri of the brain fail to approximate along the borders of their summits and that the middle third of the corpus callosum is narrow.

Subpial gliosis and satellitosis are in fairly frequent evidence, but there is little or no affection of the white matter by gliosis. The nerve cell pictures show, to qualitative judgment, frequent instances of cell scarcity affecting both the supra granular and infra granular layers.

The following is a brief characterization of each of the ten cases in the Waverley Research Series, which may be helpful for quick reference.

Case I. The brain of this microcephalic idiot, of a mental age estimated less than one year, has somewhat the suggestion of the brain of an ape. A good portion of the cerebellum is visible from the dorsal aspect of the nervous system. Whatever the cause of microcephaly, the fact that an elder sister of this patient was almost equally deficient and almost identically microcephalic, must be taken into account, as well as the fact that other children before and after the microcephalic pair seem to have been normal, or at all events not microcephalic. The mentality of this case was undoubtedly the lowest of our series.

Case II. Also microcephalic, this case presents a combination of microcephaly with a high degree of internal hydrocephalus. This hydrocephalus is one doubtless *ex vacuo*, being very possibly due to an early non-syphilitic (poliomyelitic?) infection at three months of age. The detailed description will show how well preserved was the vision of this case, functionally speaking, despite the remarkable cystic condition and microgyria of the posterior portion of the cerebrum. In point of fact, the visuo-sensory portion of the cortex appears, however, to have been preserved, and the tremendous lesion to be noted on the upper aspect of the parieto-occipital regions did not take effect upon the elementary visual functions of the cortex. The case is evidently one of focal brain lesion, and very probably belongs in what Tredgold would call the group of "secondary amentia." Of course it is possible that the microcephaly was in some sense an example of primary amentia and that the focal posteriorlying lesions with compensatory hydrocephalus were due to intercurrent disorder. It is possible, accordingly, that this case may be one of a combination of primary and secondary amentia.

Case III. The brain of this microcephalic imbecile was so small (610 grams) that any coarse arrangement of the brains of the present series (Cases I–X) must naturally show "Zip's" brain lower than his actual capacity would have warranted. It is stated that this patient knew how to read and write a little. Although unable to support himself he had lived most of his years outside of institutions, being a harmless, amiable vagrant. With some hesitation, he may perhaps be placed among the imbeciles. Microscopic study indicates a special thinning of cells in the frontal and parietal regions as well as a degree of gliosis in the outermost layer of the cortex and below the appendema of the ventricles. The corpus callosum was well preserved and of a good size in proportion to the

brain. Microscopically the case will form an excellent foil to Case I, a case of far less intelligence, but the microscopy is complicated by gliosis, whose age is difficult to determine. (In this connection, it must be remembered that the patient is said to have had an epileptic attack at one time.) Although the brain at first sight seems a good instance of a brain in miniature, and although the frontal and temporal regions are fairly uniform, the parietal and occipital lobes are quite asymmetric, especially in that the right occipital lobe is considerably shorter than the left; there is a suggestion of an *Affenspalte* on the right side.

Case IV. This case is the oldest of the series (62 years), being an old almshouse case, apparently of feeble-mindedness, of unknown grade but apparently far higher than that of any other member of the series (except V, with the question of dementia praecox, and VIII, the executed murderer). It is of course possible that this case is an old dementia praecox (compare hallucinations observed at Danvers Hospital). The corpus callosum was rather thinner than normal; the brain weighed 1340 grams at death. Throughout the brain, in different places, there were various degrees of gliosis and satellitosis. This was especially marked in the left and right prefrontal regions. The whole brain suggested a slight degree of atrophy (slight sulcal flaring). There was a slight asymmetry, particularly of the poles of the brain; the parietal region shows a tendency to microgyria, and the right pyriform lobule is larger and more prominent than the left. There is a marked dilatation of the lateral ventricles except the descending horns. The gross appearance of the brain must be admitted to be entirely consistent with the hypothesis of dementia praecox; yet the case is a good instance of the so-called old demented imbecile found in almshouses and is of value in this series for comparison with others.

Case V. Case V falls in a group with Cases IV and VIII as of higher intelligence than any of the others in the present series (Cases I–X). It is possible that the original intelligence of Case V was higher than that in Case VIII, and highly probable that it was higher than that in Case IV. The case is interesting as showing the outcome of a truant high school boy existence. It is probable that the patient was a moron, or at all events a subnormal subject and perhaps belongs in the delinquent group. The unilateral moderate hydrocephalus is not infrequent in dementia praecox. The left frontal and right occipital lobes show contralateral asymmetry, being narrower than their fellows. The right occipital atrophy or aplasia appears to centre in the second gyrus. The corpus callosum in this case is of a good size. The brain weighed 1335 grams.

Case VI. This case is that of an idiot of two years mental age, who seemed to combine the typical features of idiocy in such a way as to be a case *par excellence* for clinical demonstration. The brain, weighing 1130 grams, shows the smallest brain weight of our series outside the group of pronounced microcephalic cases. The brain is of very simple construc-

tion. The histological examination showed in this case, as in Case VII, the occurrence of the so-called "rod cells" in the cortex. These cells, characteristic of general paresis when associated with the exudative and destructive changes found therein, are not to be interpreted as necessarily indicating syphilis when they occur alone. Perhaps they indicate merely proliferating vascular changes or proliferations of unknown nature of mesenchymal tissue. There is, however, a suspicion of syphilis in the antecedents of this almshouse transfer case, and certain "tissue paper" scars found on the skin at autopsy may add to the suspicion that this may be a case of congenital syphilis of a non-exudative type. It is also possible that good fortune, or a still more minute microscopic examination than we have been able to perform, would reveal exudative changes at some point. Another feature of the greatest interest in Case VI is the virtual absence of one vocal cord as demonstrated at the autopsy. This fact may serve to illustrate the difficulties of proper interpretation in routine cases. In this case, for instance, the aphasia could not be taken to be necessarily of nervous origin.

Case VII. Like the brain of Case VI, the brain of Case VII showed rod cells. With respect to the possibilities of syphilis in the family, it is important to consider that of five children, three of the children were dead (two of marasmus), and that the mother was subject to fainting fits. A great variety of anomalies in the body is probably entirely consistent with the hypothesis of syphilis. There was a certain irregularity in the mental capacities of the case, which should also be regarded as thoroughly consistent with this hypothesis. Thus the case, classified as an imbecile with a mental age of five, seemed in a number of respects not to reach above the highest grade of idiocy, or a lower grade of imbecility than the mental age would superficially indicate. There was some sensory disorder in the case as well. Accordingly, both VI and VII are cases which bring up the question of congenital syphilis of a character not so destructive or progressive as cases of exudative syphilis (of the group, for example, of juvenile paresis) would be. To be sure, the rod cells indicate a certain activity and progressiveness on the part of the disease. Theoretically and academically, at any rate, these cells may indicate that conditions are not so entirely stationary in the nervous system as we are led theoretically to suppose should be the case in the brains of the feeble-minded.

Case VIII. This belongs with Cases IV and V, amongst those of highest intelligence in the present series (Cases I–X). The case was that of an executed murderer. The brain was viewed on anatomical grounds to be that of a defective before there was any clinical confirmation or knowledge of the fact. The gyri were noted to be of a generally transverse direction. There was a lack of complete longitudinal sulci in the frontal lobes. A blunting of the frontal lobes was possibly the most convincing suggestion of the probable

clinical deficiency on the part of the subject. The parietal region was relatively and absolutely well developed. There was an asymmetry of the pyriform lobules, to some extent of the entire hippocampal lobule. The corpus callosum was of good size though there was a question whether it might not be regarded as somewhat thinned out posteriorly. It seems safe to regard this case as either in the moron group or as at any rate to some degree subnormal.

Case IX. Case IX illustrates the "ablebodied" type of imbecile (mental age, 7). The weight of the brain in this case was even in excess of what it should have been by Tigges' formula; namely: 1622 grams, as against 1424 by Tigges' formula. (It is true that the violent death of this patient may have led to a certain congestion of the brain which artificially increased its weight.) The brain of Case IX, quantitatively adequate as it would seem, was a brain which must strike the anatomist as of exceedingly poor mould.

Case X. The brain weight in Case X was also high, being 1450 grams, and in point of fact 298 grams in excess if we should trust Tigges' formula. He is described as of a very excitable nature. The brain shows a very considerable degree of hydrocephalus. Attention may be called to some theoretical points concerning hydrocephalus in these cases in a special section devoted to this topic. Case X, like Case VII, was of the excitable group, and the brain of Case VII also showed hydrocephalus, which likewise does not seem to be explicable on the basis of a compensatory condition (*ex vacuo*, as illustrated by Case IV), or an anomaly of the dementia praecox group (as illustrated by Case V).

III.

NEUROPATHOLOGICAL CORRELATIONS WITH CLINICAL AND PSYCHOMETRIC FINDINGS IN FEEBLE-MINDEDNESS (WAVERLEY RESEARCH SERIES, CASES I–X).

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III.

NEUROPATHOLOGICAL CORRELATIONS WITH CLINICAL AND PSYCHOMETRIC FINDINGS IN FEEBLE-MINDEDNESS (WAVERLEY RESEARCH SERIES, CASES I–X.)

E. E. Southard and Annie E. Taft.

The province of the present article is limited. We intend to make a provisional epicritical review of the problems presented by the first ten cases (I–X) of the Waverley Research Series of cases of feeble-mindedness. This term we use to include, not only feeble-mindedness proper (i. e., the so-called morons of recent American nomenclature), but also the subnormal persons above the grade of morons, which modern research is bringing into the field of feeble-mindedness, and also the imbeciles and idiots which lie below the grade of moron or of the feeble-minded proper. It is the plan of the present series of anatomical examinations, therefore, to work up successively a series of all types and grades of feeble-mindedness and subnormality without undue regard to their clinical classification or intelligence grading. Already, however, the data of our first ten cases have suggested certain lines of correlation which it seems worth while to set down here in the guise of provisional hypotheses.

Among all the problems sketched above (problems of the schools, the courts, the social agencies, the eugenics record offices, the biological laboratories, and the like), perhaps no problem is more acute than that of the significance and possible future of mental tests after the manner of Binet. We must leave to the psychologists the evaluation of the details of these tests and the construction and choice of new variants. We must leave to those versed in the statistics of mental measurement the decision how far the age level of the Binet tests and the percentage level of the Yerkes tests apply to actual conditions. We must concede forthwith that Binet left but a torso of what the future has in store in the shape of mental tests, and we may concede that these tests, for the most part, yield little information as to those emotional and volitional impulses, instincts, and sentiments which are so important in social life. On the whole, however, it is generally admitted that progress is being made to a more and more accurate measurement of mental capacities of certain sorts. This bit of progress is enough to brighten the path of the anatomist. The anatomist feels that he must follow far in the rear of the psychologist and physiologist in his analysis; particularly in his analysis of deviations and perversions which are of a qualitative nature or cannot readily be reduced to quantitative values. When, however, the anatomist learns

that psychometry is not merely an idle dream, but that distinct losses,— when, at any rate, these losses are of large degree,— are demonstrable and to a certain extent measurable, his hope is kindled that the brain itself may unexpectedly at least yield up a set of useful correlations.

What is the relation of mind to brain? Speculation halts shortly before the intricacies of this problem when it is taken as a problem of the relation of the full-blown, healthy mind with the entirely normal, richly active brain. Important light is thrown upon the topic, of course, by the anatomo-clinical method which yielded such rich data in the last half of the nineteenth century; for by this method definite losses of brain substance were often found to be associated with equally definite losses of mental power. The limitations of this method are numerous and need not detain us. The method has not been exhausted; but its future fruits depend upon elaborate tissue analyses which the ordinary laboratory and the ordinary scientific leisure available under our present institutional system do not permit. Almost equally rich results have accrued from work in comparative anatomy, latterly supplemented by behavioristic studies.

What we at present need is to supplement the methods of normal neurology and psychology, the methods of anatomo-clinical research, and the methods of comparative anatomy with a method which has alliances with each of these but seems to us to contain certain elements of novelty. It would be strange if the field of anatomy in feeblemindedness were not full of novelty since it had been so poorly tilled heretofore. The novelty in the anatomic situation with respect to feeble-mindedness seems to us to lie in the fact that we deal with all grades of defect, from an equality with brutes up to subnormality, hardly removed from ourselves, and thereby gain a number of steps upon what the comparative anatomist can ever take. It is conceded that the higher mental operations have much to do with speech,— its storage and its expression. Now, among animals few traces of speech exist. But among the feeble-minded all degrees of speech loss may in the course of years be studied. The missing links and intergrading transitions between the primates in general and man in particular may be found in feeble-mindedness. By proper sifting of cases, future investigation will discover by an accumulation of instances the minimum mechanism with which speech can be effected.

It is this principle of the minimum apparatus, the minimum machinery with which a function may be performed, that we regard as the most important and promising aspect of feeble-mindedness from the standpoint of research into mental function. As we have elsewhere sketched, fundamental researches in the psychology of efficiency may well be made with the feeble-minded, since among them we shall be able to discover what can be done with the least apparatus. For the purpose of the present argument we need not

develop the collateral fact, that in brain disease of the normal adult and in brain disease of epileptics we often deal with far more complicated problems since the destruction of a few bits of brain apparatus in one region may lead to functional disuse of great masses of mechanisms that are structurally quite intact. Diaschisis and shock are instances in point. There is no method of determining, at least in the majority of nerve cells, whether they are performing vegetative or conductive functions, or no functions whatever. Consequently the principle of discovering the minimum machinery with which a given act may be performed cannot be applied as a rule in the field of the neuropathology of destructive lesions.

Mill's method of study by means of concomitant variations is a method much more readily applied to material in feeble-mindedness than to the ordinary anatomo-clinical material of the neurological clinic.

Now, it must be conceded forthwith that, if the authorities in mental tests are in doubt as to their ultimacy, the brain anatomists are equally modest in their claims. As we looked over the claims of the workers in mental tests, we concluded that these mental tests could at least lay claim to a certain orderliness, such that a patient stated to have a mental age of six, was at all events of less mental capacity than one having a mental age of seven or eight. In short, the dicta of the mental tests might have an ordinal value if they did not possess cardinal value. Very possibly, also, this point might apply to the point-scale data of the Yerkes tests, although this perhaps was less likely on account of the fact that a given percentage in the Yerkes tests might be made up of exceedingly heterogeneous successes in puzzle-solving, the doing of sums, and the answering of questions. Conceding for the moment that the mental tests are now so good as to have approximately an ordinal value, such that a patient of eight years mental age has higher intelligence than a patient having a mental age of seven years, we can see that it might be well to compare our brains of feeble-minded subjects together so that we might learn whether here also there was an ordinal value to attach to the brains. We are, doubtless, far from the achievement of *cardinal* values, indicating definite increment of mental capacity to correspond with definite increment of brain mass, even should our studies be so minute as to permit us to evaluate the dendrite supply and synaptic surface area of the brain cells instead of their mere proportionate numbers. But might we not be able to arrange our brains in an ordinal series which would be convincing because of the derivation of the principles of arrangement from a number of sources: say, brain weight, proportionate brain weight to body length, complication of sulci, size of corpus callosum, etc., etc.?

And suppose this order of brains to be obtained, would it fit at all with the order of brains obtainable by mental tests? If the two series were largely identical, then might

not some progress be thought to have been made in the problem of the relation of intelligence to brain? We are, of course, entirely aware that this principle is applicable to a large series only, and the present tables are offered chiefly to lay down a few principles as to the proper lines of future correlation on the basis of more cases. We have placed at the head of Article II a statistical table of the ten cases in which some data for identification are given. In the following table, we again arrange the brains in their order of acquisition by the laboratory. In separate columns we place the age, mental age, brain weight (whether less or more than average according to our view), together with certain anatomical and histological data.

TABLE II.

Table Displaying Mainly Histopathological Findings, Cases I–X.

Number	Age	Mental Age	Brain Weight probably	Focal Destructive Lesion	Hydrocephalus	Exudative Lesion of Rod Cells	Nerve Cell Aplasie	Fiber Loss	Gliosis	Satellitosis	Dislocated Ganglion Cells
I	5	1–, est.	minus	0	moderate	0	marked	marked	slight	0	0
II	20	1–, est.	minus	+	marked	0	marked	marked	marked, patchy	+	0
III	45	7, est.	minus	0	0	0	+	?	+	+	0
IV	62	moron or subnormal	plus	0	+	+	+	+	+	focal	+
V	16	subnormal	normal	0	slight	0	+	0	+	0 (sl) focal	+
VI	26	2, est.	minus	0	0	+ rod cells	slight	?	+	+	0
VII	25	5, Binet	normal	thrombus (Hist.)	0	rod cells	slight	?	+	0	0
VIII	36	subnormal	normal	0	0	+	+	0	+	+	+
IX	39	7, Binet	plus	0	0	0	+	0	0	0	+, slight
X	37	24, Binet	plus	0	0	?	+	?	+	+	0

It will be seen that one case (II) is apparently the result of a focal destructive brain lesion of great size. This case was probably one of encephalitis,— possibly due to poliomyelitis,— and yielded a marked degree of hydrocephalus. This fact, however, did not complicate the estimation of its proper place in the anatomical or psychological series, since the brain was clearly upon the score of weight and simplicity not of so low grade as Case I, but lower than any other brain. Cases I and II, then, appear to be the lowest brains in point of anatomy and the lowest cases in point of intelligence. The comparison may be made even finer, since beyond question the brain of Case I was the least complex of the entire series, and the mentality of this case, whose actual age was but five years, was also minimal.

There are, however, three cases of pronounced microcephaly, namely; cases I, II, and III. The instance of Case III is the most disquieting in the analysis so far from the

standpoint of aligning the brains with estimated mental capacity. Case III, it may be remembered, was that of Little Zip, a microcephalic, who lived for forty-five years a vagrant life, which was almost self-supporting in the hobo sense of the term. With the brains superficially suggestive of a dog, the patient himself exhibited a number of doglike features in his vagrant, happy-go-lucky life, journeying from the barn or house of one friend to that of another. Arranging the brains in the order of their complexity, the brain of Little Zip must be placed third, after the microcephalic case I and the microhydrocephalic case (post-encephalitic) II, and yet the mental age of Little Zip may be safely estimated as about seven years. The mental age is, at all events, much higher than that of the other microcephalics. Thus, if the brains are arranged in order of their estimated age, the brain of Little Zip comes in sixth or seventh place, instead of in the third place.

Another case (IX) may be chosen as well illustrating the possibility of wrongly estimating the mental capacity of a patient from his brain appearances; for this brain was placed upon the score of its complexity, largely in the gross, in the fifth place. Its mental age was seven. Yet two brains that are regarded as still more complex, namely; those of Cases X and VII, yielded Binet ages of $2\frac{1}{5}$ and 5 respectively. The microscopy of Case IX, however, should that be taken into account, would insure a somewhat lower place for the brain of this able-bodied imbecile, of great stature and large, poorly-molded brain (1620 grams; more than 200 grams in excess of an estimated norm). The examples of Little Zip (Case III) and of the able-bodied imbecile (Case IX) suffice to indicate the difficulties of these analyses.

The following table (Table III) shows the brains arranged in the order of their estimated complexity, and the figures in the intelligence column will readily demonstrate the facts just mentioned, concerning the dislocation of cases III and IX from their proper places in the series if we estimated the brains on their gross complexity alone.

TABLE III.

Comparison of Brain Complexity, Brain Weight, and Order of Intelligence in Cases I–X.

Number	Age	Brain Complexity	Brain Weight	Intelligence
I	5	a)	400–, est.	1–, est.
II	20	b)	620	1–, est.
III	45	c)	610	7, est.
VI	26	d)	1130	2, est.
IX	39	e)	1620	7, Binet
X	37	f) or g)	1450	2.4, Binet
VII	25	f) or g)	1270	5, Binet
IV	62	h)	1340	Moron or subnormal
V	16	i) or j)	1435	Subnormal
VIII	36	i) or j)	1340	Subnormal

Table IV arranges the brains in the order of actual brain weight, but gives also the brain weights estimated by Tigges's formula (eight times the body length in centimeters = brain weight in grams). The brain weight for the normal age and sex of the individual taken from Vierordt's tables is also given.

TABLE IV.

Comparison of Brain Weight, Corrected Brain Weights, and Order of Intelligence in Cases I–X. (Arranged in order of actual brain weight).

Number, Age, Height (cm.)	Brain Weight Tigges	Brain Weight Age normal (Sex)	Brain Weight Actual	Intelligence
I, 5, 81	648	1282, M	400–, est.	1–, est.
III, 45, 126	1008	1348–66, M	610	7, est.
II, 20, subn.	?	1358–96, M	620	1–, est.
VI, 26, 158	1264	1358–96, M	1130	2, est.
VII, 25, 146	1168	1234–39, F	1270	5, Binet
IV, 62, 141	1128	1178–1210, F	1340	Moron or subnormal
VIII, 36, 179	1432	1365–6, M	1340	Subnormal
V, 16, 168	1344	1358–96, M	1435	Subnormal
X, 37, 144	1152	1365–6, M	1450	2.4, Binet
IX, 39, 178	1424	1365–6, M	1620	7, Binet

Table V presents the cases in the order of their estimated intelligence.

TABLE V.

Comparison of Order of Intelligence with Complexity of Brain in Cases I–X.

Number	Actual Age	Intelligence	Brain Complexity	Remarks
I	5	1–, est.	a)	
II	20	1–, est.	b)	
VI	26	2, est.	d)	Vocal cord absent
X	37	2.4, Binet	f) or g)	
VII	25	5, Binet	f) or g)	
IX	39	7, Binet	e)	*Out of place*
III	45	7, est.	c)	"Zip," out of place!
IV	62	Moron or subnormal	h)	Almshouse transfer
V	16	Subnormal	i) or j)	Dementia praecox
VIII	36	Subnormal	i) or j)	Murderer

It seems safe to conclude from these tables that at least the brains of least complexity are correlated with the minds of least range, and that the brains of greater complexity are in a general way correlated with minds of greater range. By microscopic correction of the anatomical decisions, we may even cause our figures to look still more satisfactory from the standpoint of identical ordering of anatomical and psychological data. Whether

the future may show how to account for the apparent too-great simplicity of the brain of Little Zip, remains to be seen.

The second point in our epicritical review may be presented briefly, partly on the basis of Table II, in which the findings of exudative lesion or rod cells are entered, and also by means of Table VI, in which are displayed the majority of the anatomical findings in the series. How many of our cases may be regarded as essentially preventable by the means theoretically available to the mental or psychiatric hygiene of the individual, leaving out, that is to say, the eugenic line of attack? Let us, perhaps, grant that Case II, with its focal destructive lesion,— possibly of post-encephalitic origin,— its marked hydrocephalus, marked nerve cell and fibre loss, and marked patchy gliosis, with its focal microgyria in other regions than the most marked post-encephalitic lesion, is a case that is preventable,— that is by social and scientific devices which shall exclude this presumably infectious disease from occurring.

If we take at their face value the slight mononucleosis of cases IV, VI, VIII, and X, we may perhaps consider them as either syphilitic or as suffering from progressive exudative disease of a mild nature, also theoretically preventable. Now, in point of fact, we are far from proving that these four cases are cases of syphilis; or that Case VII, a case which fails to demonstrate lymphocytosis but does demonstrate rod cells, is a case of syphilis. It is, however, of importance to consider that in a series of ten cases of numerous grades of feeble-mindedness, no less than six show processes of a suspiciously progressive nature or suggestive of infection. Here is a problem that is well worth working out in the utmost detail, whether we lay down the problem as a problem of the frequency and distribution of syphilis in the feeble-minded, or whether we seek to establish the part played in feeble-mindedness by acquired disease.

Let us sum up these considerations by saying that the number of cases in which the hypothesis of infection must be more or less firmly entertained is much larger than has been supposed. By consequence, the scales tip much farther for the group of the so-called secondary amentias than was the supposition with most workers.

In addition to these main points in our epicritical review, namely; the point concerning the relation of the complexity of brain to capacity of mind, and the point concerning the theoretical preventability of certain cases, we should not dismiss our review without slightly indicating the richness of the field in points of interest to neurology. The teratologist and embryologist are sufficiently aware of these values, so that we need not make special point of the *Affenspalte* question (for instance, Case I and Case III); the question of cruciate asymmetry (Case V), the question of the absence of the middle commissure (Cases V, VII, IX, and X), the question of presence of dislocated ganglion cells in the white matter

TABLE VI.

Case Number	Calvarium	Dura Mater	Pia Mater	Vessels	Brain Weight Grams	Gyri	Diffuse Atrophy or Aplasia	Focal Atrophy or Aplasia	Corpus Callosum
I	———	———	slight general thickening	negative	400, est.	Microcephaly	marked	variations; "affenspalte" left	proportionate
II	thick frontal; depressions	normal	normal	negative	620	Microcephaly	marked	microgyria, 2nd frontal, etc.	thin
III	moderately	normal	normal	negative; except vertebrals	610	Microcephaly	marked	minor anomalies	proportionate
IV	dense	slight thick, frontal	slight thick, vertex	negative	1340	Focal microgyria and asymmetry	no	minor anomalies	rather thin
V	negative	negative	slight general clouding vertex	negative	1435	Cruciate asymmetry	absent	frontal; opercular	slightly thin, posterior third
VI	inequalities of thickness, brittle	thick along middle meningeals	slight general thickening	negative	1130	simple, symmetrical	absent	Frontal	thin throughout
VII	dense	adherent at corono-sagittal suture line	faintly hazy, vertex and sulci	negative	1270	slight asymmetry; simple construction	absent	Frontal	thin, horizontal portion
VIII	———	———	thickened	negative	1340	slight asymmetry; transverse gyri	absent	left parietal	moderate thining, posterior third
IX	thick, somewhat dense	thick	focal basal leptomeningitis	negative	1620	slight asymmetry; "poorly moulded"	absent	absent	narrow, posterior third
X	dense	focal thickening	cerebellar pia thickened; pia in general thick	negative	1450	slight asymmetry	general superficial	Frontal	slight thinning, posterior third

TABLE VI (*continued*).

Middle Commissure	Consistence	Sclerotic Foci	Other Foci Destructive	Ventricles Hydro-Cephalus	Cerebellum	Spinal Cord	Remarks
———	———	absent	———	moderate	negative	———	
———	not remarkable	———	left parietal	marked	negative	negative	early focal encephalitis, (poliomyelitic?)
———	not remarkable	absent	absent	0	inferior vermis absent?	negative	
———	not remarkable	absent	absent	present	dentate nuclei unequal		mononucleosis, posterior septum, spinal cord
absent	normal	absent	acquired? slight lesion, left callo-so-marginal	slight	negative	negative	
———	normal in general	left 2nd temporal firm, yellow	absent	0	negative	negative	rod cells; lymphocytes in smear, frontal
absent	normal	absent	canalized thrombus (histologically)	0?	negative	negative	rod cells
present	———	absent	absent	0	negative	———	plasmocytosis, focal, left prefrontal
absent	normal; putty-like at base	absent	absent	0	negative	———	
absent	unusually firm; "brain swelling"	absent	absent	0	small	negative	?mononucleosis, focal, right postcentral (dead of pellagra; tuberculous)

(Cases IV and V, and to a slight degree, Case IX), the problem of the time relations of satellitosis (as shown in cases II, III, VI, VIII, and X; and focally, in IV and V), and the question of defective cortex lamina (as shown characteristically in Case IX). We choose for exposition a somewhat more recondite point, namely; the significance of hydrocephalus in some of these cases.

The significance of hydrocephalus in Cases I and II with microcephalic brains is hard to judge. Whether the microcephaly is due to hypoplasia or to agenesia, it would naturally not be unlikely that there should be an absence of neurones, both on the exterior and in the interior, or both in the outer and inner portions of the brain substance. Accordingly the hydrocephalus exhibited in the brains of Cases I and II is not necessarily to be associated with increase of intracranial pressure at any time in the lives of the patients. The hydrocephalus in Case II is very possibly to be related with an early focal acquired lesion of the nervous system (paralytic shock stated to have occurred at three months of age by attending physician). The hydrocephalus in Case III is possibly to be explained similarly, and does not require the hypothesis of heightened intracranial pressure at any time in the life of the patient.

Aside from hydrocephalus associated with microcephaly, and possibly due to the operation of identical causes, we must consider on another basis the hydrocephalus of four other cases: IV, V, VII, and X.

The hydrocephalus of Case IV is voluminous and is associated with a corpus callosum rather thinner than normal, and in fact very thin posteriorly opposite the region of greatest ventricular dilatation. The motor restlessness of this case may or may not be correlated with the ventricular dilatation, as has been contended by Southard in his work on dementia praecox in 1915, and in a later analysis of hydrocephalus in the so-called functional psychoses, as yet unpublished. However, it may well seem to the anatomist that the hydrocephalus would best be regarded as one due to tissue atrophy of a gradual nature, not in any wise necessarily related to alterations of intracranial pressure. The external features of the brain do not show recent effects of ventricular swelling. Accordingly, we should not lay particular stress on the occurrence of ventricular dilatation associated with motor restlessness in Case IV.

Case V shows dilatation of the right lateral ventricle without dilatation of the left (in fact, there may have been a slight degree of internal brain swelling which has caused the surfaces of the left ventricle to come into apposition). This finding of unilateral hydrocephalus has not been infrequent in Southard's dementia praecox series of 1915; has been there referred to at length, and also in the later, as yet unpublished, analysis. In cases VII and X, we are dealing neither with pronounced microcephaly (the brain weights

respectively are 1270 and 1450 grams, accordingly over weight according to Tigges' formula of brain weight in grams equal to eight times the body length in centimeters), nor with the special conditions of a possibly atrophic brain in an imbecile of 62 years, Case IV, and the special features of the case of dementia praecox, Case V. Cases VII and X, curiously enough from our present standpoint, are examples of such an imbecile as is rather apt to be termed by the laymen insane, or "crazy", imbecile. The point of the attendant's or layman's remark is not that such an imbecile belongs in the group of the psychoses as the physician views them, but that the patient is insane or "crazy" from the nonmedical standpoint; showing spells of marked irritability. Thus, Case VII is described as having had tantrums, as being "hysterical" on examination, laughing one minute and crying the next, rushing about, jumping up and down, and screaming on occasion, and was regarded as a very troublesome case.

Case X is described as having been somewhat feeble on admission to the school at the age of 10 years, but as later becoming very troublesome, with quick temper and noisiness.

This problem of the relation of hydrocephalus to hyperkinesis, as first developed from Southard's dementia praecox work of 1915, can hardly be settled in its relation to feeble-mindedness from this series, for if we exclude the three markedly microcephalic cases (Cases I, II, and III), the old demented imbecile (Case IV), the case of dementia praecox (CaseV), and the executed murderer (Case VIII), we are left with but four cases: VI, VII, IX, and X, of which VII and X were hydrocephalic and had been as sketched above as to their tendencies to over-activity. If we consider in contrast to these VI and IX, (the cases we have termed "typical idiot" and "peculiar imbecile" with some capacities above the imbecile grade), we find ourselves dealing with brains of simple construction and of poor moulding respectively, in which brains there is no evidence of hydrocephalus despite the fact that the brains show the corpus callosum in both instances to be thinned out posteriorly. (In fact, the corpus callosum of Case VI is everywhere rather thinner than usual.) Case VI was a very restless and nervous idiot, described as "always looking around," and as "inclined to be destructive". It appears that these tendencies were constant and not more marked at one time than another. The case seemed to be one of slight microcephaly (brain weight, 1130, is 134 grams under Tigges' formula for the patient's height, 158 cm.). "Constant nervousness" or hyperkinesis can hardly be supposed to be associated with hydrocephalus except accidentally and independently of any casual interrelation. The point of the coexistence of overactivity and hydrocephalus in the microcephalic would lodge rather in the simplicity of the neuronic system in the microcephalic case. It is clear that, if the microcephalic brain is *also* hydrocephalic, the neuronic systems of the brain in question must be still simpler in make-up. If the brain in question is

taking in anything like the normal number of ingoing stimuli, and the capacity of the brain to dampen or inhibit the stimuli and divert them from taking effect in motion, is a diminishing capacity, then over-activity becomes the most natural result of the simplicity of the brain and one quite to be expected.

Accordingly, although the typical idiot(Case VI) yielded a brain without hydrocephalus and yet was over-active, it is clear that the over-activity is not of the type of the occasional hysterical outburst; thus, whereas Cases VII and X are described as excitable, Case VI would best be described as restless or "nervous".

Case IX showed no restlessness or excitability at any time, having a good disposition and in general a rather coöperative nature.

Our problem, accordingly, as based on the analysis of this group of cases from the standpoint of hydrocephalus, may be summed up as follows:

The association of internal hydrocephalus with microcephaly may well be incident to the mal-development, that is to say, merely a part of the result of the factors which have retarded or stopped the growth of the brain. Accordingly, under these circumstances, alterations of intracranial pressure are neither necessary nor in fact very likely to occur. Moreover, dilatation of the ventricles in old age and in dementia praecox must be considered separately. If there is any relation between occasional outbursts of excitability and alterations of intracranial pressure which have to do with the production of hydrocephalus, many more cases will be necessary to make the point certain. We have one good instance to show that *constant* restlessness is not necessarily associated with hydrocephalus. A further physiological and psychological analysis of the difference between excitability and restlessness would be very desirable, both for its intrinsic interest and for the light it may throw upon these structural conditions, which are difficult, if not impossible to interpret when considered entirely by themselves.

Summary.

The entirely provisional conclusions of the epicritical review may be briefly stated as follows:

First, it is not impossible that the problem of matching brain complexity with mental capacity may be solved by a much larger series of instances than is here available; but the instances of such matching as has been undertaken are somewhat convincing as to the correlations of low orders of intelligence with simple brains and of higher orders of intelligence with more complex brains. Occasional exceptions to the rule may be explained by

the finer anatomy of certain cases (Case IX); others remain less easy to explain away (Case III).

Secondly, the partial orienting and microscopic examination yielded more instances of slight exudative lesions (including in some instances rod cells) than might have been *à priori* expected from a relatively stable institutional material like that here largely drawn upon. What the share of syphilis in this group of cases may really be is doubtful. There was one instance of feeble-mindedness very possibly due to an early focal encephalitis entailing mal-development of brain.

Thirdly, as an example of special neurological interest attaching to this study, some considerations about hydrocephalus offered bring up the question of the relation between occasional bursts of excitability and alterations of intracranial pressure with the production of hydrocephalus.

Description of Photographs.

Views of Case I.

The gross photographs of the stripped brain alone are presented since, although there was a slight general thickening of the pia mater, the microscopy of the tissue has so far shown no evidence of exudate.

Note in photographs I–a, I–c, and I–d the striking projection of the cerebellum behind the occipital poles. Note the suggestion of *Affenspalte* (I–a).

Note in I–b the large exposure of the crura cerebri, as well as the comparatively small number of transverse pontine fibres arranged in almost distinct bundles. Note, also, in I–b the comparative size of the optic tracts and olivary eminences.

Note in I–a, I–c, and I–d the bi-convolute construction of the frontal lobe; the superior frontal convolution, broader than the inferior, may represent superior and middle frontal convolutions of more highly developed brains. Note the excellent development of the pyriform lobule on each side, especially the right (I–b).

We present in I–e, I–f, and I–g, three views of the brain in total section as stained by the Weigert myelin sheath method. (Note that the stains are relatively successful despite the preservation of the brain for eleven years in formaldehyde solution.) A moderate degree of hydrocephalus is notable in I–f. The small size of the leaflets of the cerebellum is to be noted in I–g. The comparatively greater depth of the sulci than those found in more normal brains is obvious in each of the total brain section views.

This case is thought to have possessed the lowest mental age in the entire series. An older sister of the patient was almost equally defective and almost identically microcephalic; other children, before and after, were not microcephalic.

Views of Case II.

The gross photographs of the stripped brain alone are presented since the membranes were normal and there were no microscopic evidences of exudative disease.

Photograph II–a demonstrates the focal posterior-lying areas, especially marked on the left side with microgyria. This case of microcephaly exhibits a higher degree of hydrocephalus than did Case I, as is demonstrable from the total brain sections II–f, II–g, and II–h. II–a also shows frontal depressions corresponding with depressions in the skull. Note in II–a, II–c, and II–d the distinct projection of the cerebellum behind the occipital poles; a projection less marked than in Case I.

Note in II–c an area of microgyria in the second frontal gyrus; a similar area is less marked in II–d. II–a demonstrates that the area of microgyria begins almost abruptly with the postcentral gyrus; whereas the precentral gyrus on each side is of fair width and well rounded. Photographs II–c and II–d demonstrate that the microgyria is far less marked on the flanks and inferiorly than superiorly; and this contrast is strongly brought out in the total brain section view, II–g and II–h.

The mental age of this case was, also, one year or less, but the intelligence of the patient was greater than that of Case I. Clinical observation of the patient's general behavior indicated that he was in the habit of attending to bright colors, looking at pictures, watching boys at play, and taking interest in events about him. The question arises, whether these functions are largely related so far as they are of visual origin with the inferior rather than the superior tissues of the occipital lobe. It may be noted that the Weigert myelin sheath stains are relatively successful with this brain although it was preserved in formaldehyde for 13 years.

Views of Case III.

The views of this case are, also, limited to the stripped brain for the same reasons as in Cases I and II. Note the general symmetry of this brain. The views do not include the cerebellum, which, however, did not project beyond the plane of the occipital poles. Note in III–a the suggestion of *Affenspalte* on the right side.

Note that the gyri are everywhere narrow and simple in arrangement, and that the secondary sulci are rarely complicated by collaterals. A special description in the text is made of the parietal regions, which in III–a are quite unlike.

III–c and III–d show at the base of the frontal gyri, separating them from the precentral convolutions, short, deep, transverse sulci, somewhat suggesting the sulcus cruciatus of the brain of a dog. Note, in fact, the somewhat general superficial resemblance of the brain of this case to that of a dog.

Views of Case IV.

Views IV–a and IV–b are presented of the unstripped brain viewed from above and below, partly because there is a slight thickening of the pia mater at the vertex, and partly because microscopic examination showed a mononucleosis of the posterior septum of the spinal cord, which naturally gives rise to the suspicion of former syphilis, although it

fails to prove the existence of syphilis. IV–c and IV–d show the same aspects with the pia mater stripped.

IV–c, IV–e, IV–f show a lack of division of the primary sulci of the frontal lobe on account of the interruption of annectants. The parietal convolutions are also narrow: IV–c, IV–e, IV–f.

IV–d shows a right pyriform lobule larger and more prominent than the left. Now, the left side of the brain in general appears slightly more hypoplastic (or atrophic?) than the right, so that Case IV exhibits a diminution of the crossed and uncrossed systems on the same side of the brain. (We here proceed upon the basis that the connections of the left pyriform lobule are largely with the left olfactory zone, whereas the connections of the neopalium are largely crossed.) Compare, accordingly, the cruciate asymmetry which appears in Case V. We might argue that the conditions in Case IV are of more recent origin than those of Case V. At least, if we desire to attribute the cruciate asymmetry of Case V to the operation of a single agent at or before the beginning of decussation, of course this case,— the oldest in the series,— was microscopically found subject to gliosis, and the whole brain shows a tendency to atrophy with slightly flaring sulci. It may be that the condition is one of atrophy and not of aplasia, and that the atrophy has little or nothing to do with the feeble-mindedness of the case.

The gross sections are presented with some completeness (four views, IV–g to IV–j) for the reason of the somewhat surprising degree of hydrocephalus which the external views would hardly give one cause for suspecting.

The best argument for feeble-mindedness in this case is microscopic, namely; the presence of dislocated ganglion cells in the white matter.

Views of Case V.

The unstripped views of Case V are omitted, as in several previous cases, because of the lack of acute or progressive exudative process.

V–a presents the not unusual frontal and occipital cruciate asymmetry. In this instance, the left frontal and left occipital lobes are smaller than their fellows. The mesial views V–g and V–h show a slight amount of thinning out in the posterior third of the corpus callosum.

As in Case IV, the best argument for feeble-mindedness in this case is microscopic, namely; the presence of dislocated ganglion cells in the white matter. Naturally, this argument may not be convincing. In general, the gross findings in the case resemble

those of dementia praecox in their mildness and unilaterality. These appearances are borne out in a cross section (V–d).

Views of Case VI.

Unstripped appearances from above and below are presented in VI–a and VI–b, because there was not only a general thickening of slight degree of the pia mater, but lymphocytes were found in a smear from the frontal region as well as a few rod cells in certain areas. These appearances may well suggest syphilis. But one focus of perivascular lymphocytosis has been found in the microscopic study, which was in this instance chiefly limited to the stripped brain. Despite the indication of syphilis, this case was a rather typical instance of a "grinning idiot", who was perhaps classed too low under the Binet test because of his aphasia, which aphasia may have been in part due to the absence of one vocal cord. There were certain other suspicions of syphilis in the case. The corpus callosum was thin throughout (see especially VI–g and VI–h).

Views of Case VII.

The unstripped appearances in Case VII, viewed from above and from below, are likewise shown, although the pia mater was but faintly hazy throughout. The microscopic examination, however, demonstrated rod cells, from which may perhaps be suspected a more progressive condition than is assumed to be typical of feeble-mindedness. The mental age of this case, though set at five, seemed too high in a number of respects. The case also showed some sensory disorder. As in Case VI, there was apparently a suspicion of syphilis.

The views of the stripped brain exhibit a fairly complex construction. The cross sections, VII–g and VII–h, show a narrow corpus callosum.

Views of Case VIII.

On account of the focal plasmocytosis of the left prefrontal area, the unstripped views of the vertex (VIII–a), the left flank (VIII–b), and the left mesial surface (VIII–c) are presented. There was no suspicion of syphilis in this case clinically, and it may be that the condition found is due to some other intercurrent condition, which may possibly have set in even after the crime for which the patient was executed. The stripped appearances show a number of transversely directed gyri and a failure to approximate on the part of the summits of numerous gyri. This is especially shown in the view of the left flank (VIII–b).

Perhaps the strongest argument for feeble-mindedness in the case is the dislocated ganglion cells in the white matter of certain areas (very few). The narrowing of the posterior third of the corpus callosum is best shown in the mesial views VIII–c, VIII–e; less well in VIII–f, VIII–g, VIII–h, and VIII–i.

Views of Case IX.

The unstripped appearances in Case IX are presented from above and below (IX–a and IX–b) and mesially (IX–c and IX–d) to exhibit a focal leptomeningitis. especially at the base. But there was no evidence of acute or chronic inflammation microscopically.

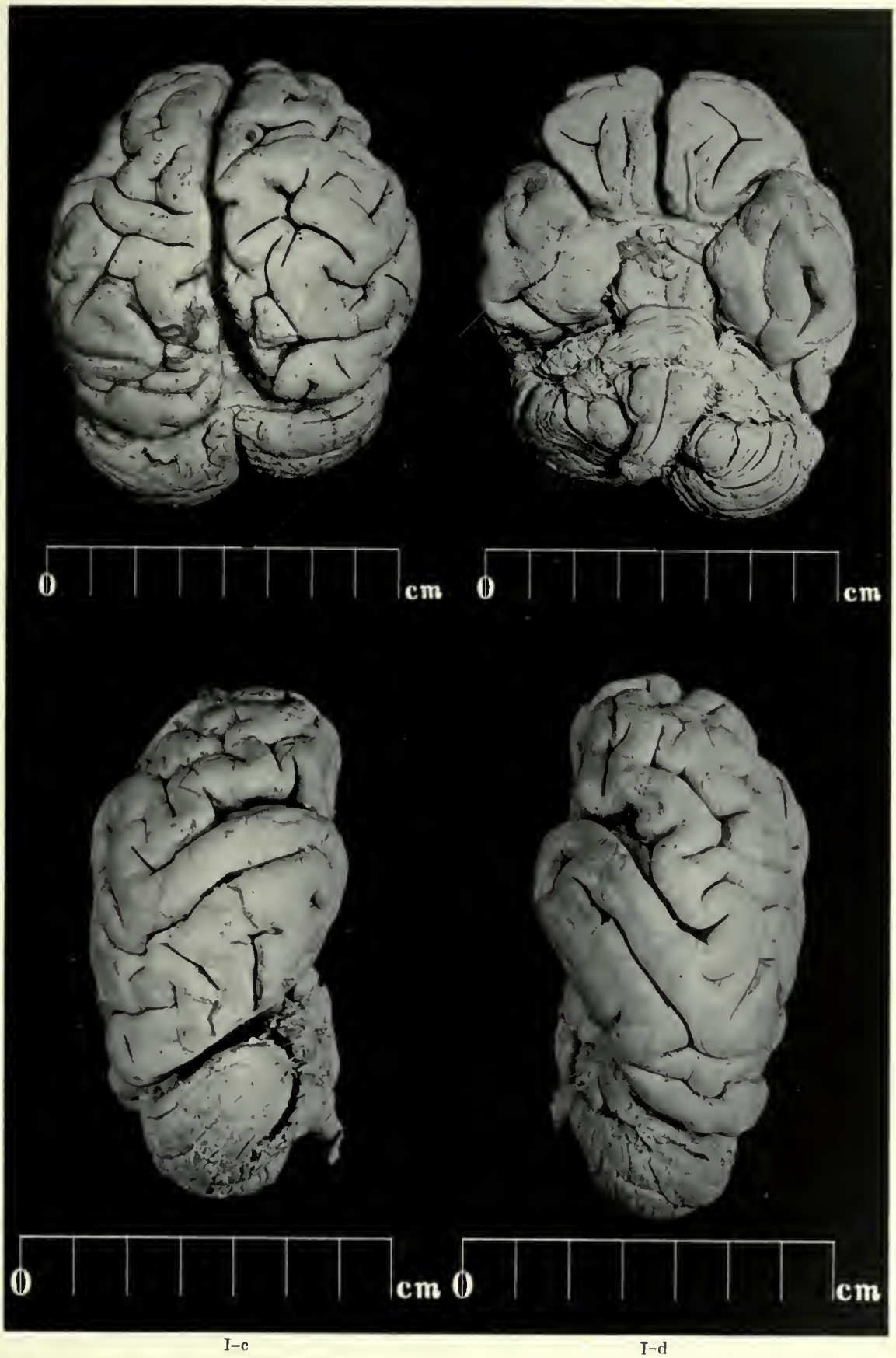
The stripped appearances (IX–e to IX–j) show slight asymmetry and a brain in general of great size but poor molding. The mesial views (IX–i and IX–j) show a narrowing of the posterior third of the corpus callosum.

Views of the brain in cross section are shown with comparative fulness on account of the irregularity in the distribution of hypoplasia as suggested by the microscopic examination. Especially the right superior frontal region (IX–k) exhibited the most marked architectural disturbance microscopically. A cyst-like area in the right hemisphere of IX–l is due to artifact.

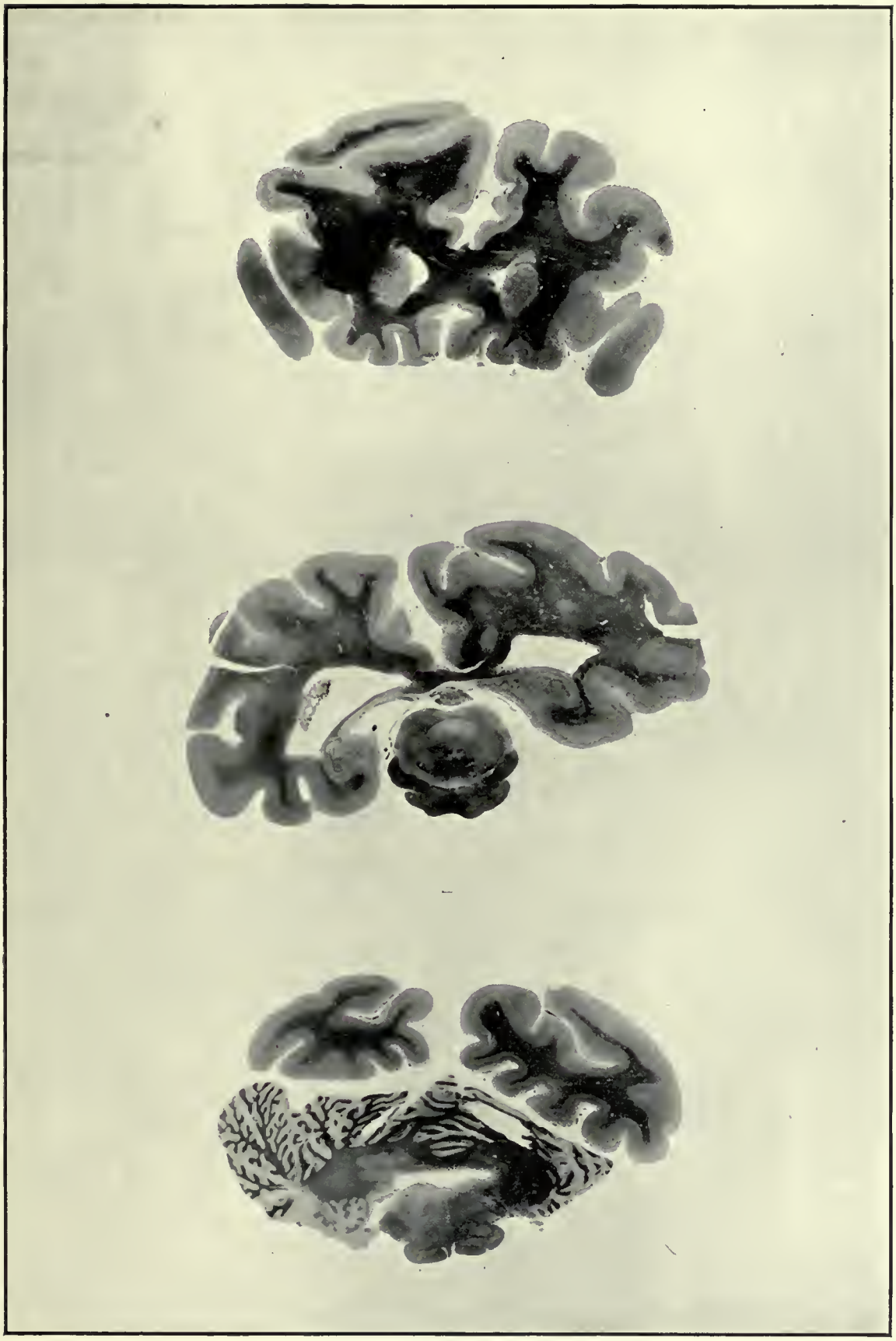
Views of Case X.

Photograph X–a illustrates the unstripped appearances, to which are added X–b and X–c to illustrate the thickening of the cerebellar pia mater. The case exhibited a somewhat questionable focal mononucleosis about the vessels of one region.

Microscopic examination showed numerical hyperplasia of cells in a number of places. The brain is fairly complex in construction, but the corpus callosum (X–h and X–i) shows a slight thinning in the posterior third.

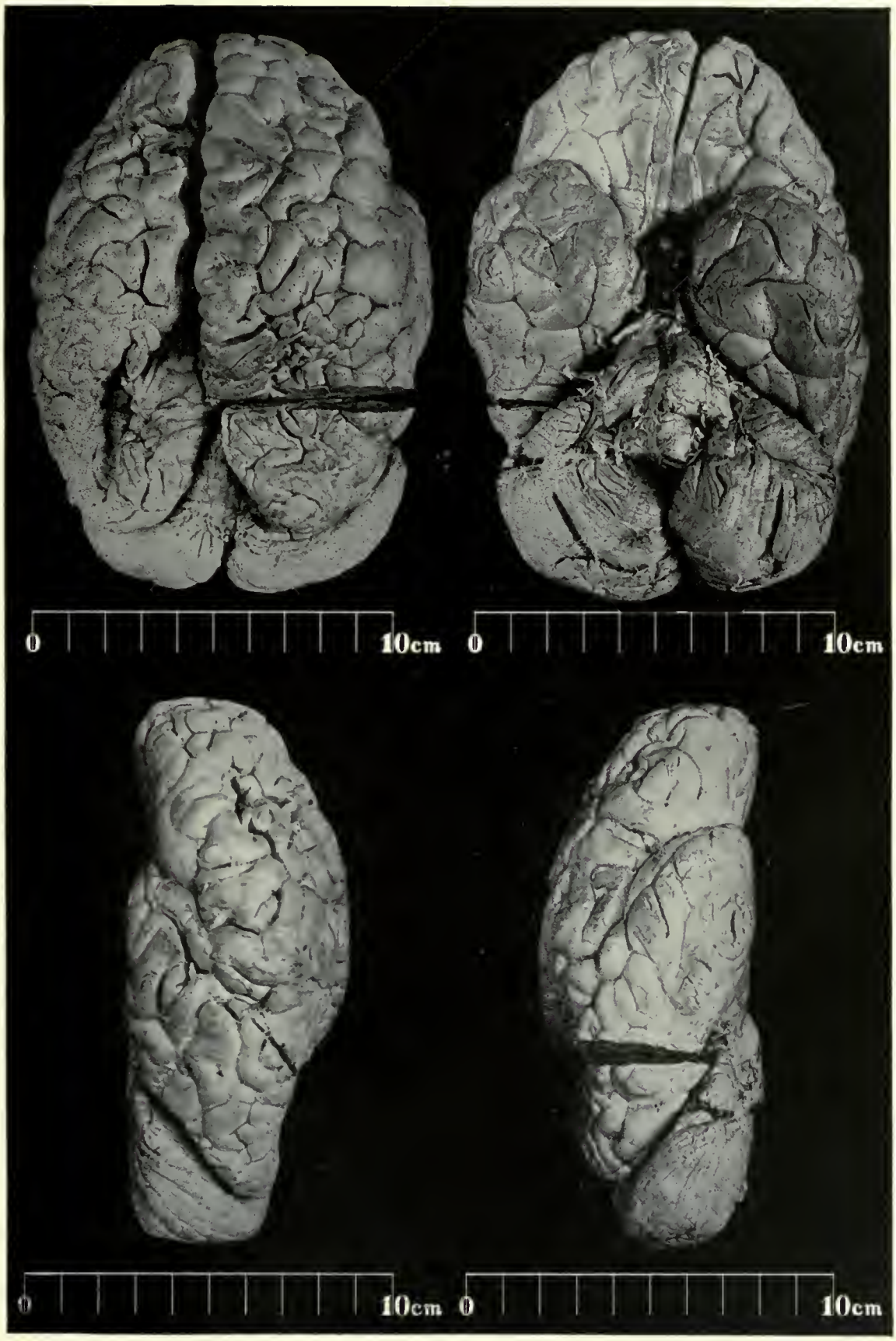


CASE I — MICROCEPHALY. LOW IDIOT



1–e, 1–f, 1–g

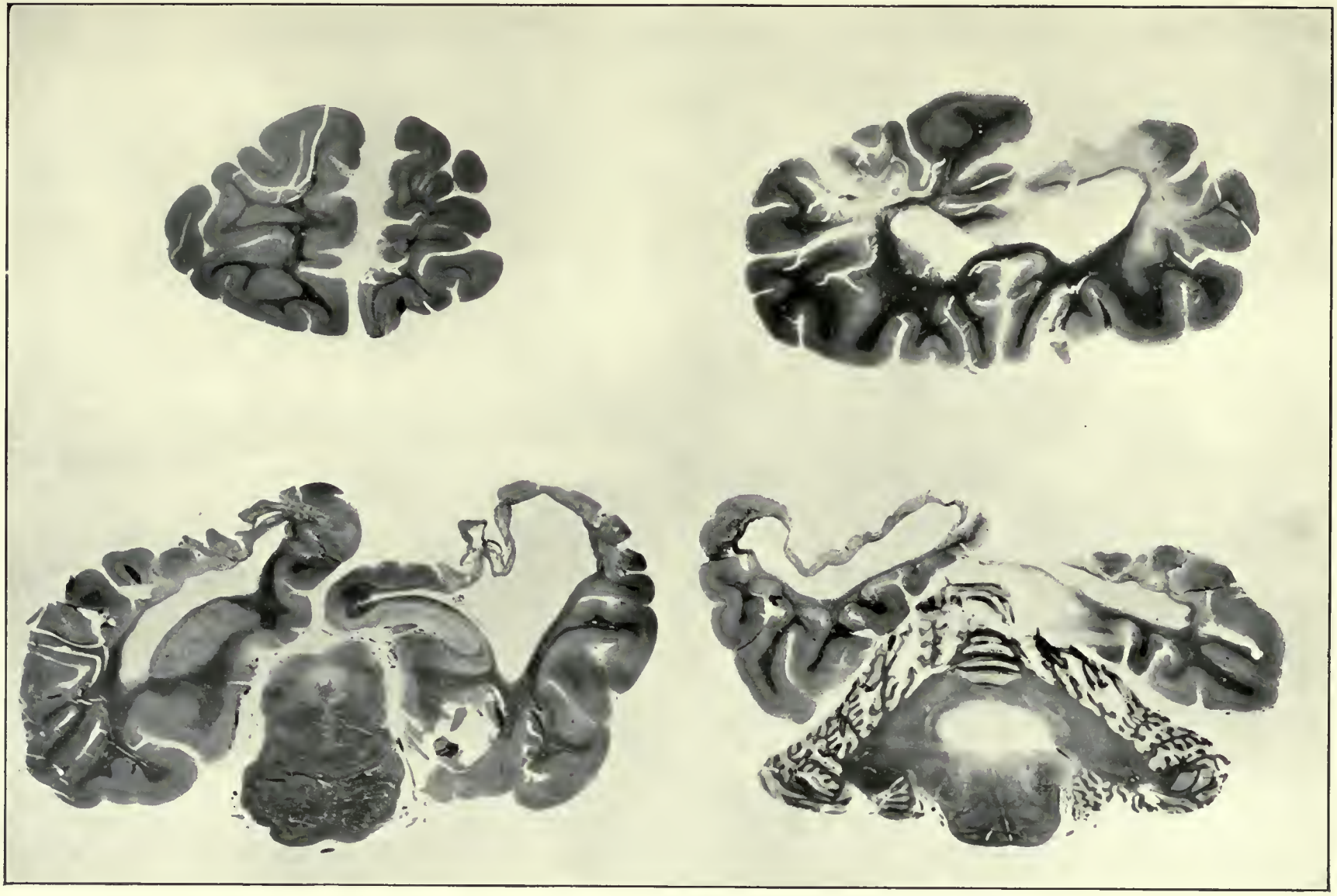
CASE I — MICROCEPHALY. LOW IDIOT



CASE II — MICROCEPHALY. ENCEPHALITIC. LOW IDIOT

II–e

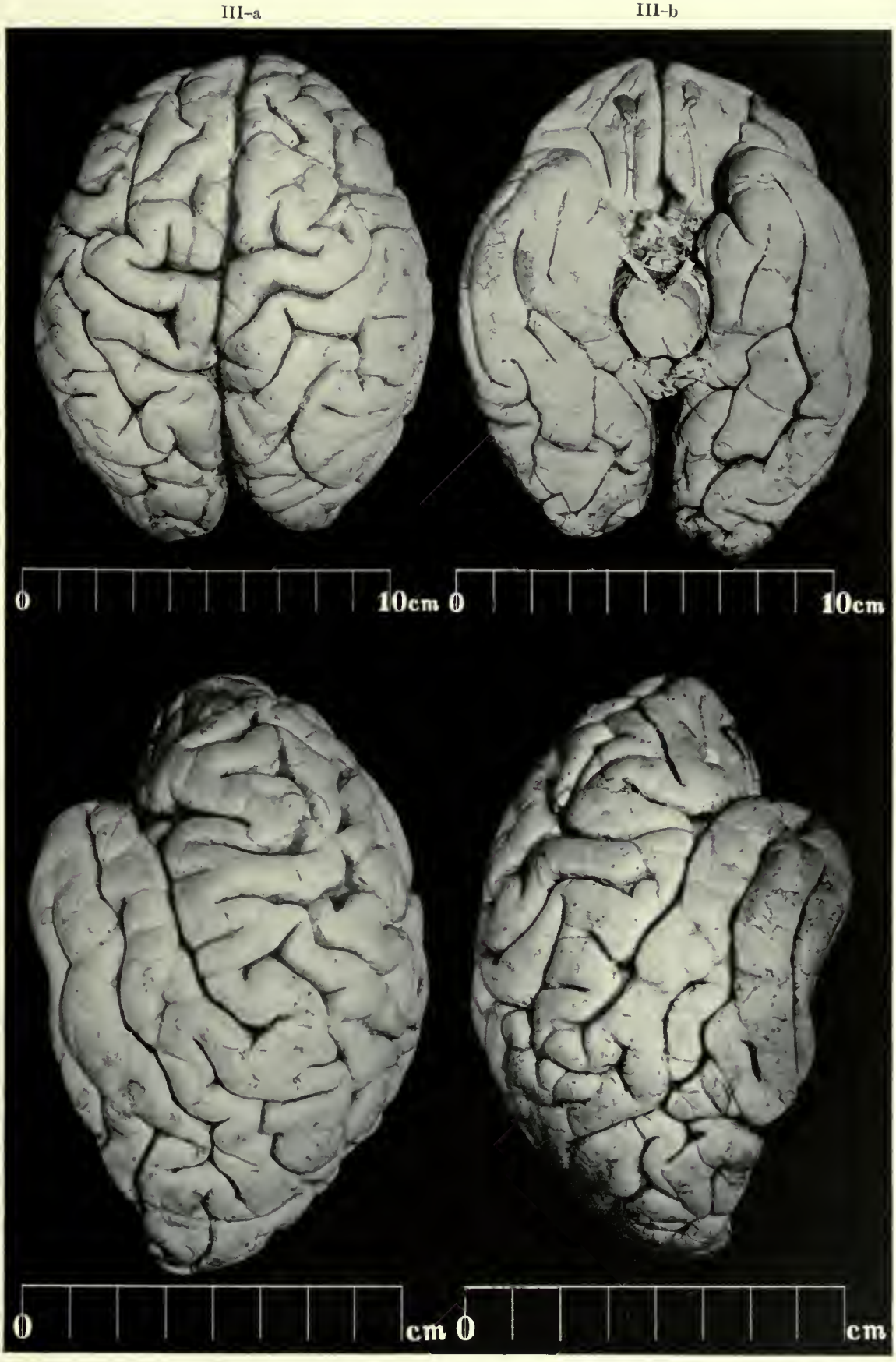
II–f



II–g

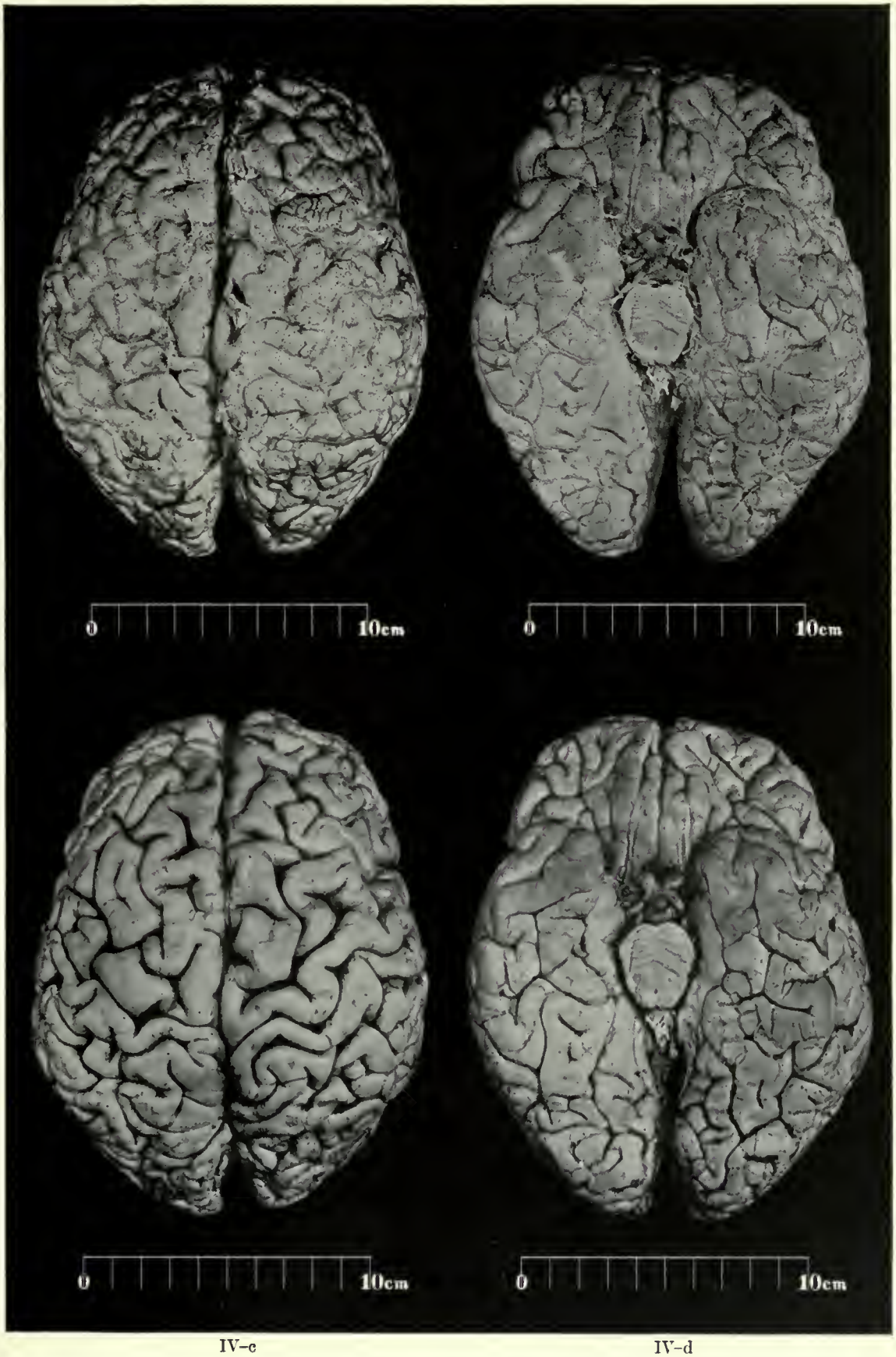
II–h

CASE II — MICROCEPHALY, ENCEPHALITIC. LOW IDIOT



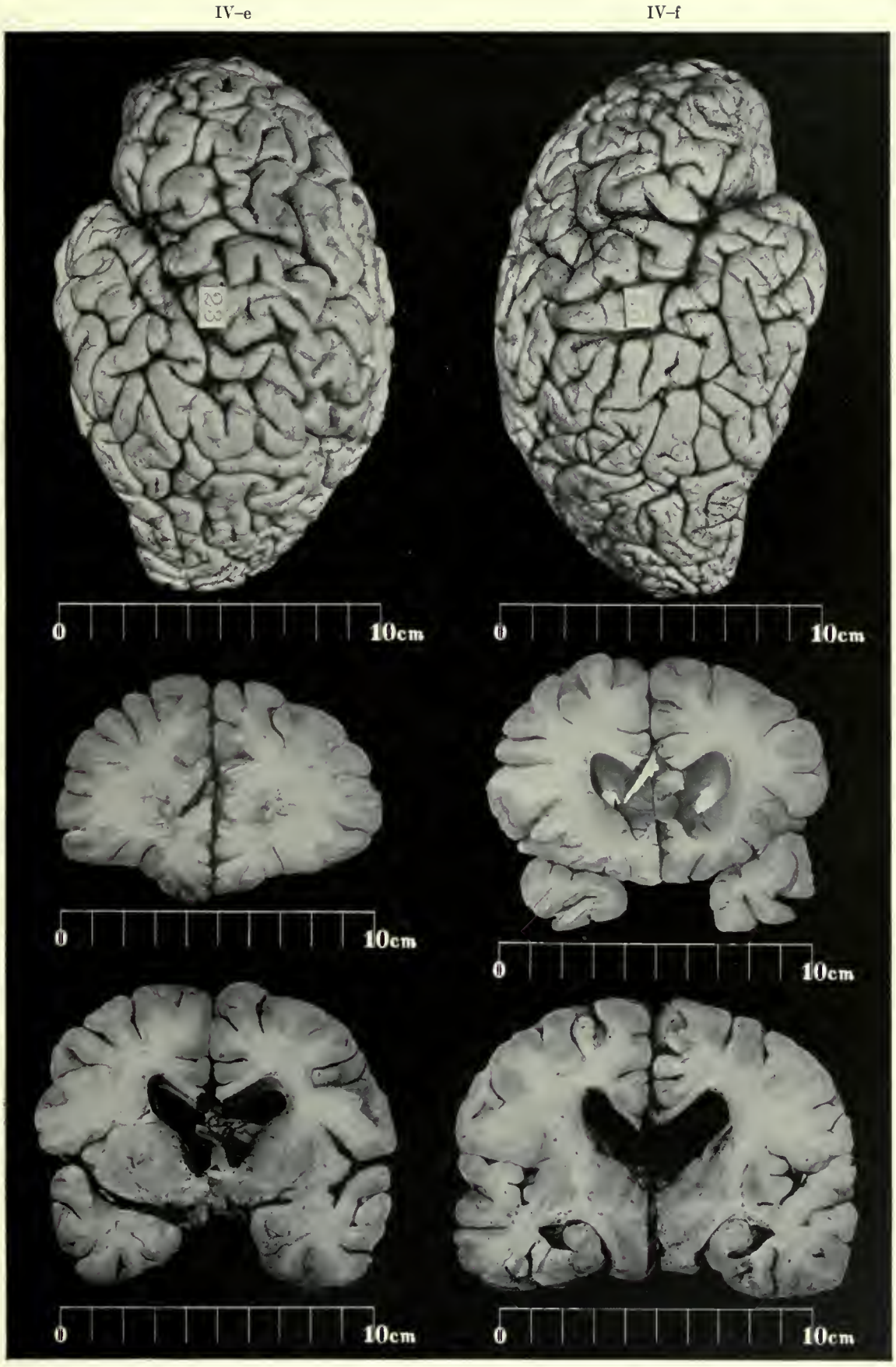
CASE III — MICROCEPHALY. IMBECILE "LITTLE ZIP"

IV–a IV–b



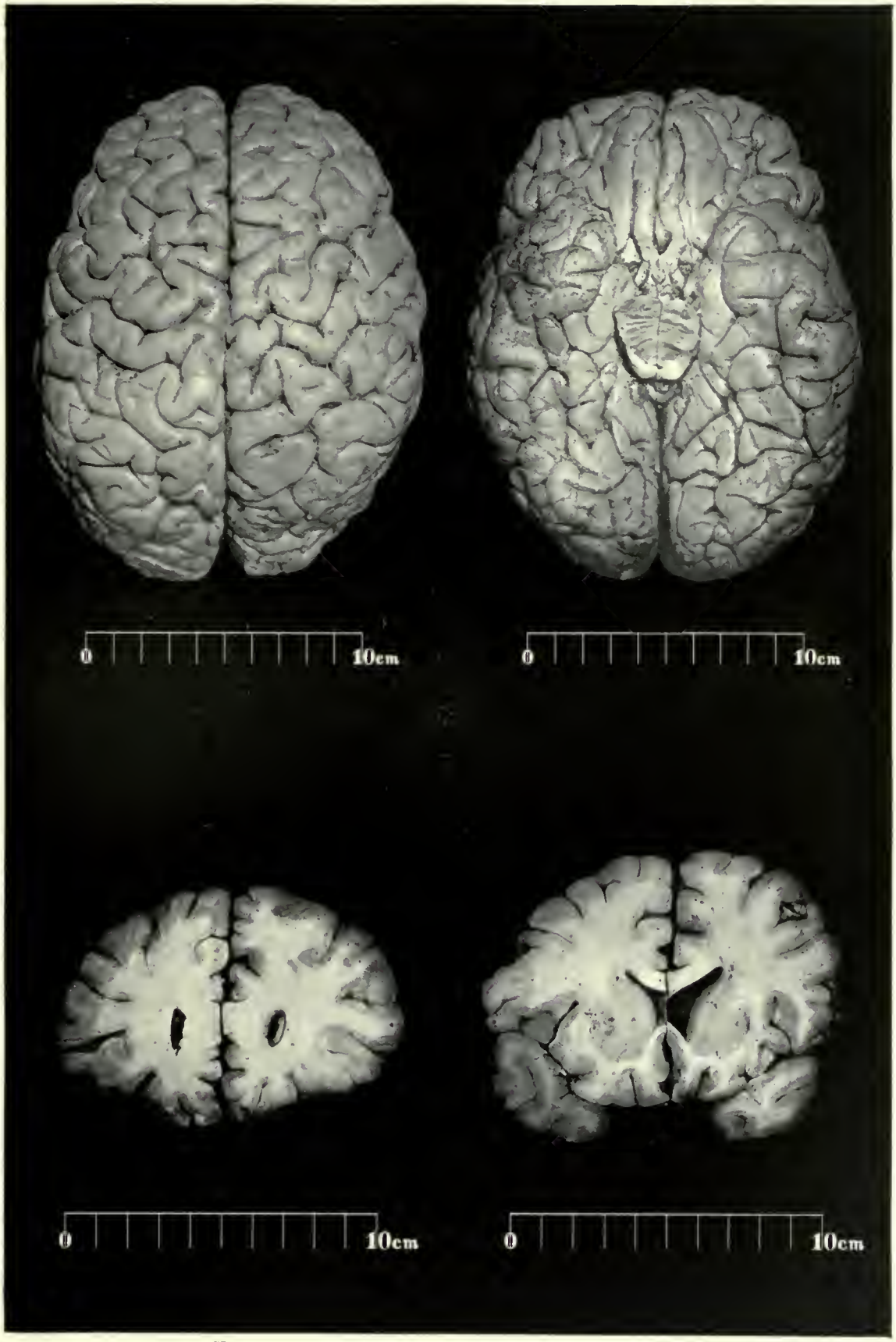
IV–c IV–d

CASE IV — OLD ALMSHOUSE MORON



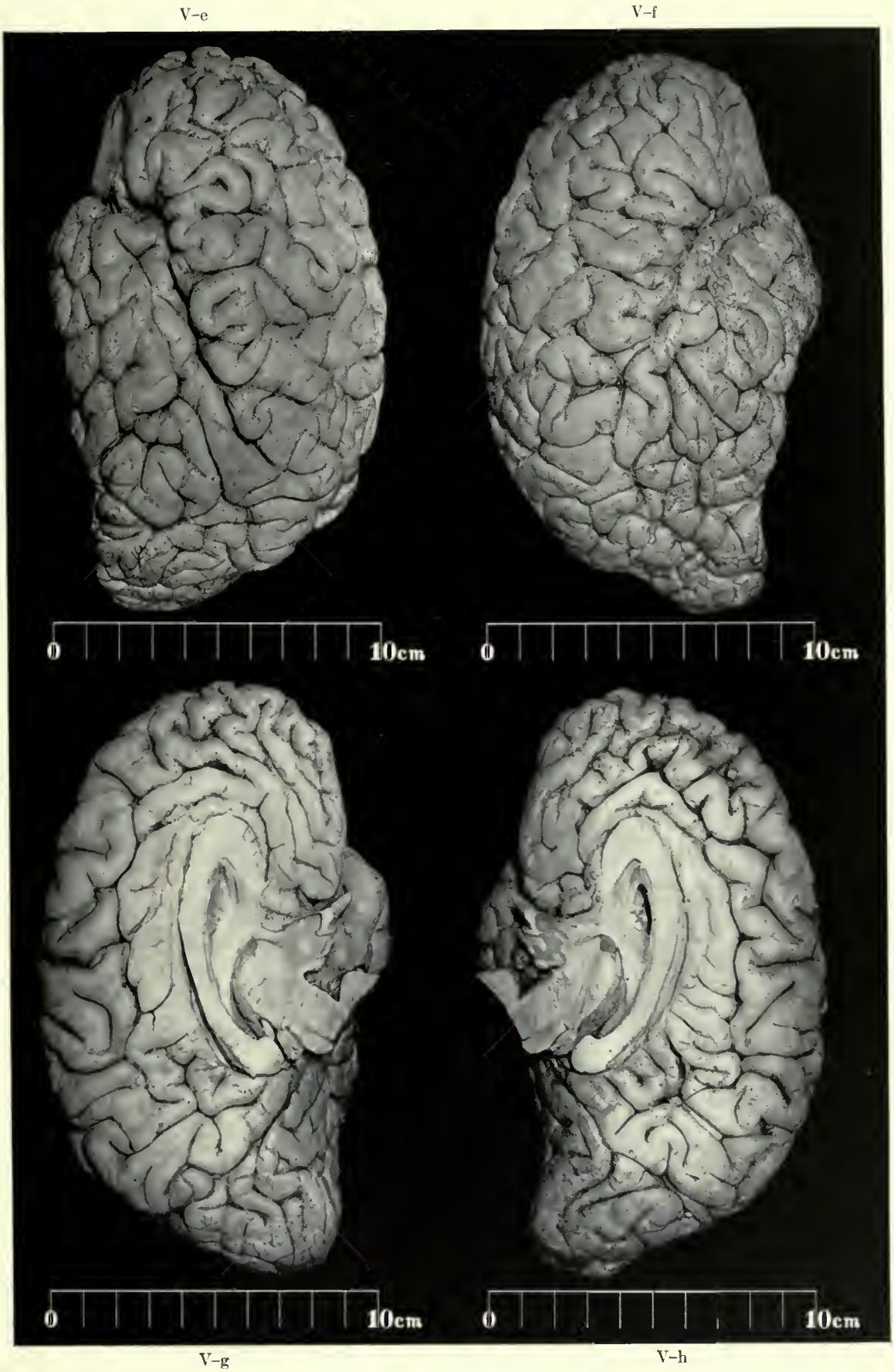
CASE IV — ALMSHOUSE MORON. NOTE HYDROCEPHALUS

V–a V–b



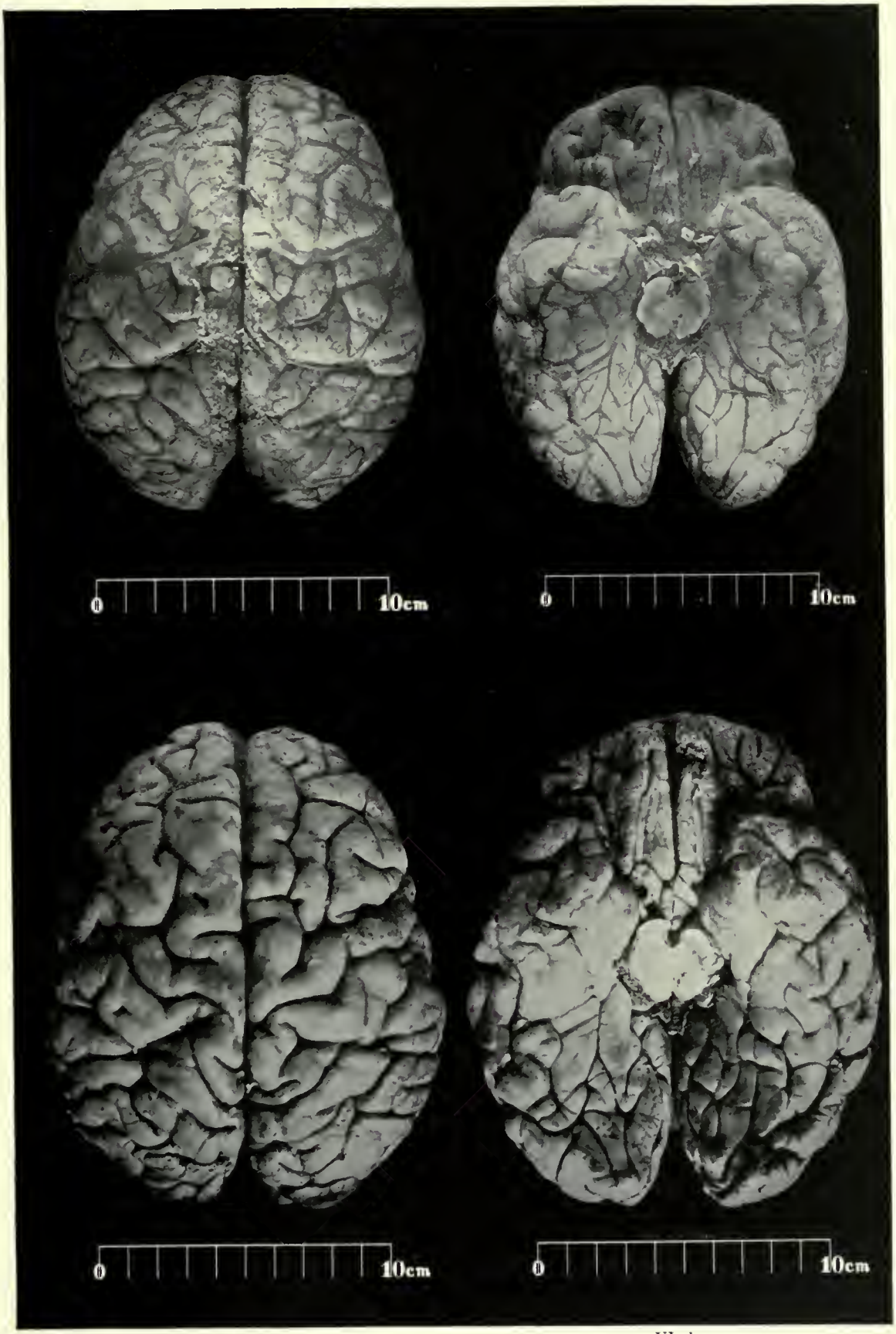
V–c V–d

CASE V — MORON, TRUANT, DEMENTIA PRAECOX (?)



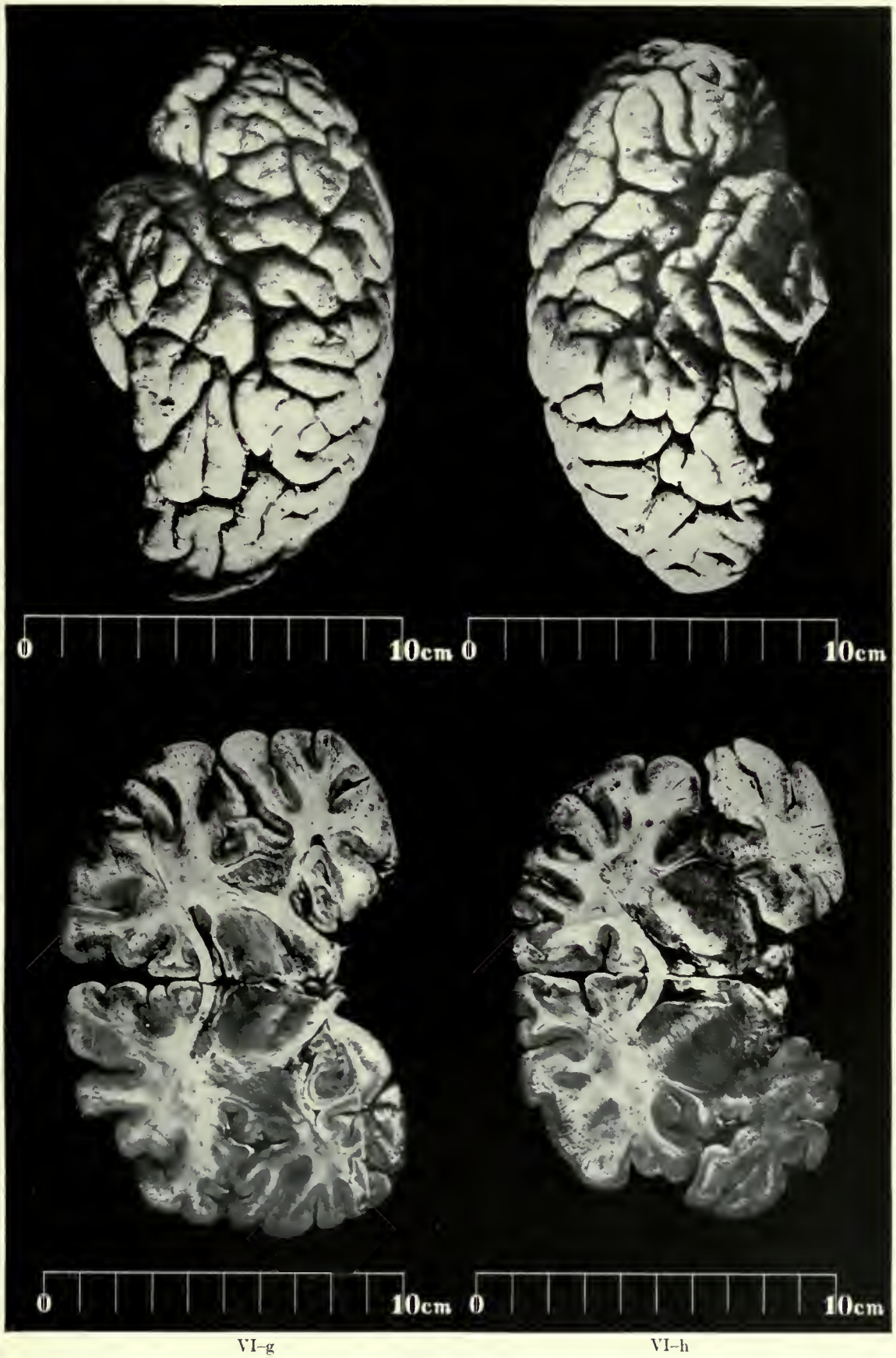
CASE V — MORON, TRUANT, DEMENTIA PRAECOX (?)

VI–a VI–b



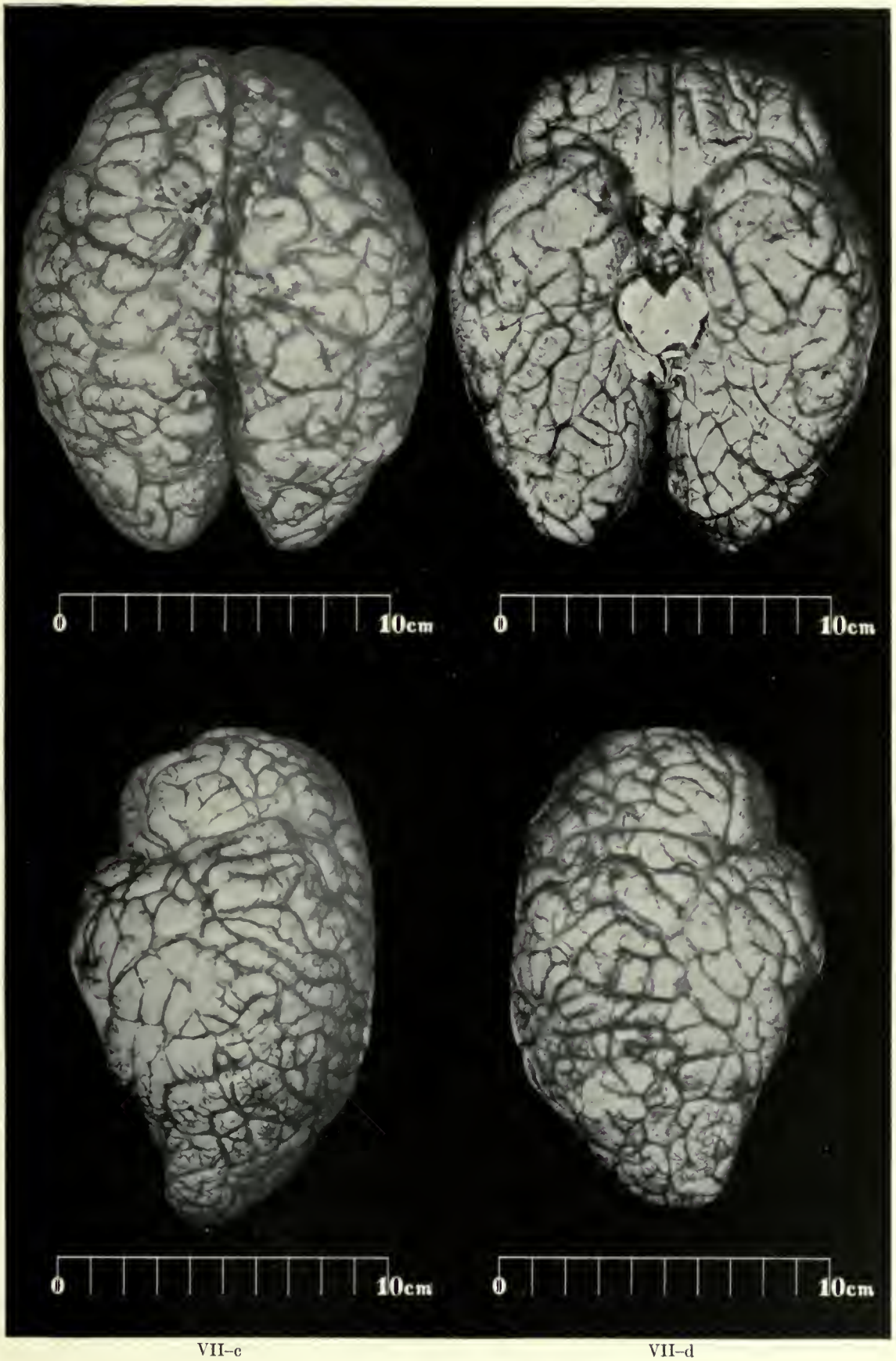
VI–c VI–d

CASE VI — IDIOT (APHASIA. VOCAL CORD ABSENT). SYPHILITIC (?)



CASE VI — IDIOT. SYPHILITIC(?) CORPUS CALLOSUM THIN

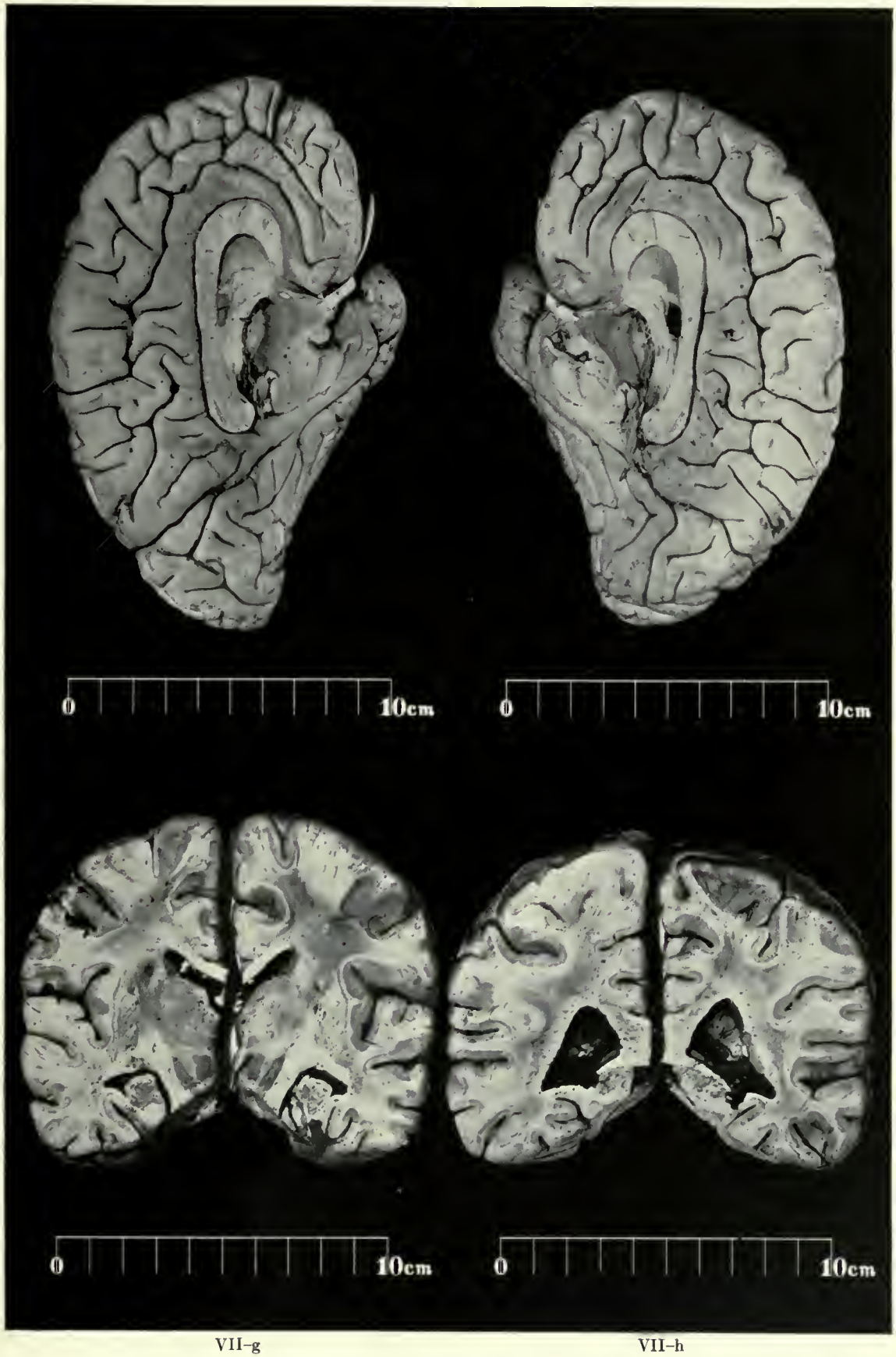
VII–a VII–b



VII–c VII–d

CASE VII — IMBECILE. SYPHILITIC (??)

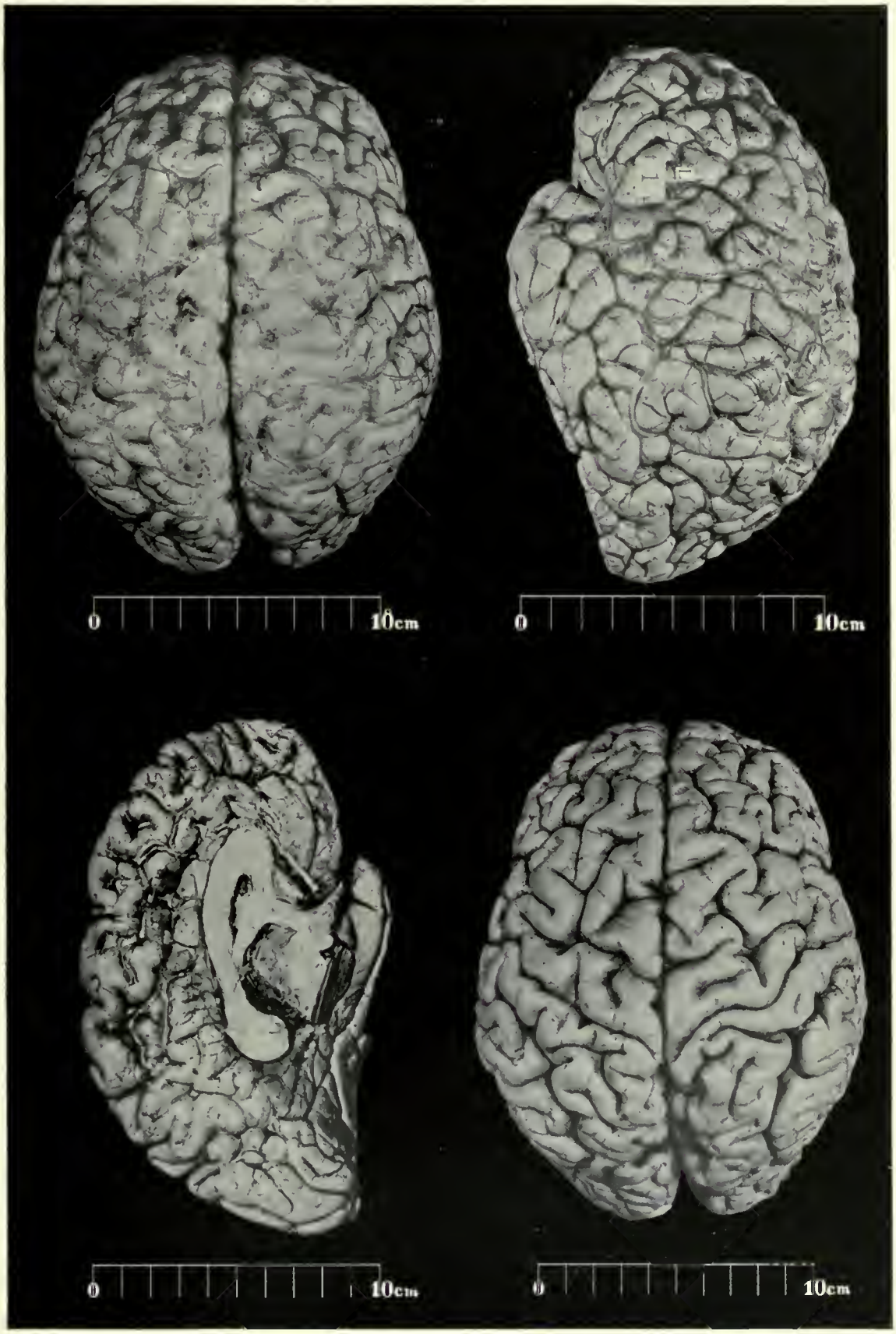
VII–e VII–f



VII–g VII–h

CASE VII — IMBECILE. SYPHILITIC (?)

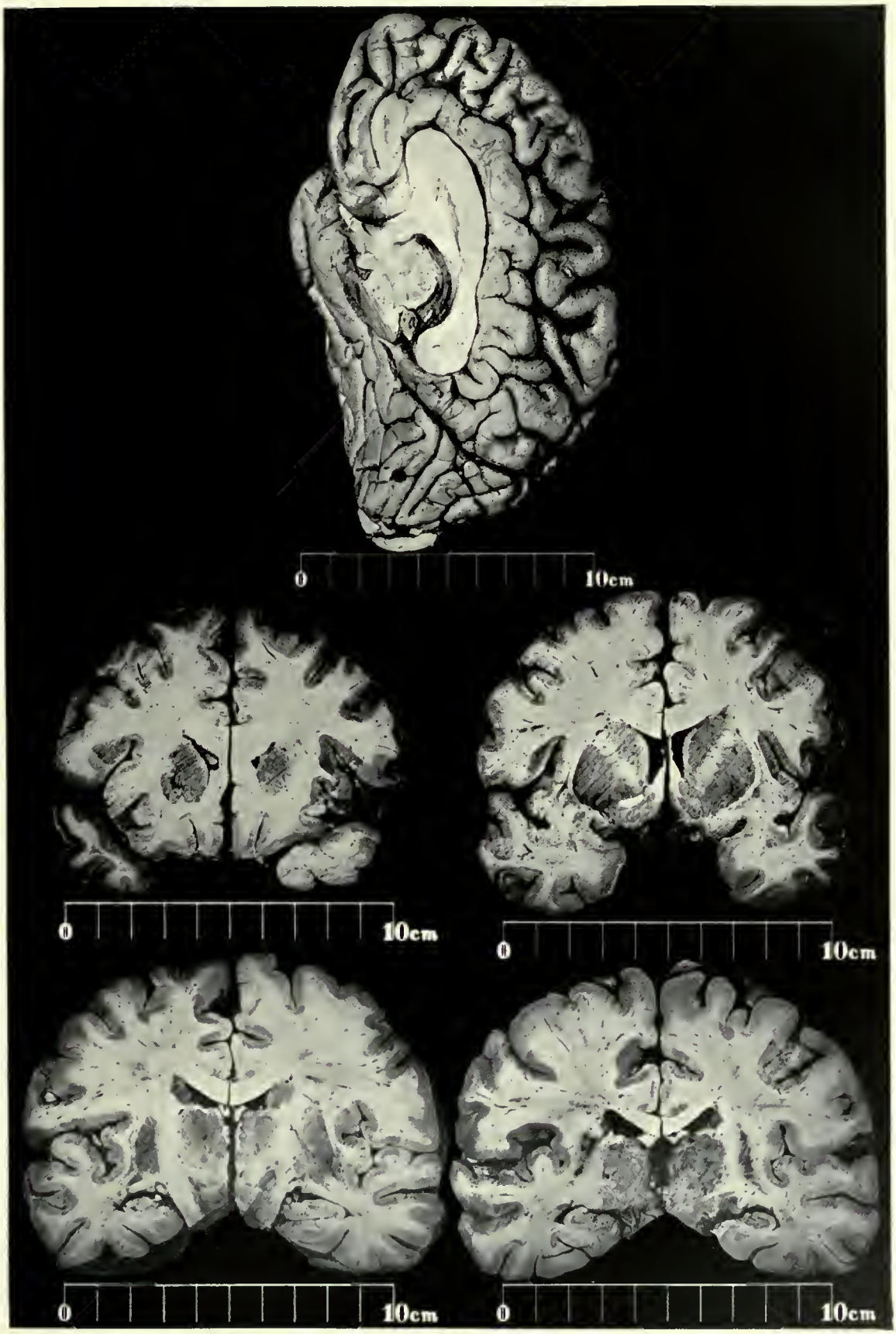
VIII–a VIII–b



VIII–c VIII–d

CASE VIII — MORON OR SUBNORMAL. EXECUTED MURDERER

VIII–e



VIII–f

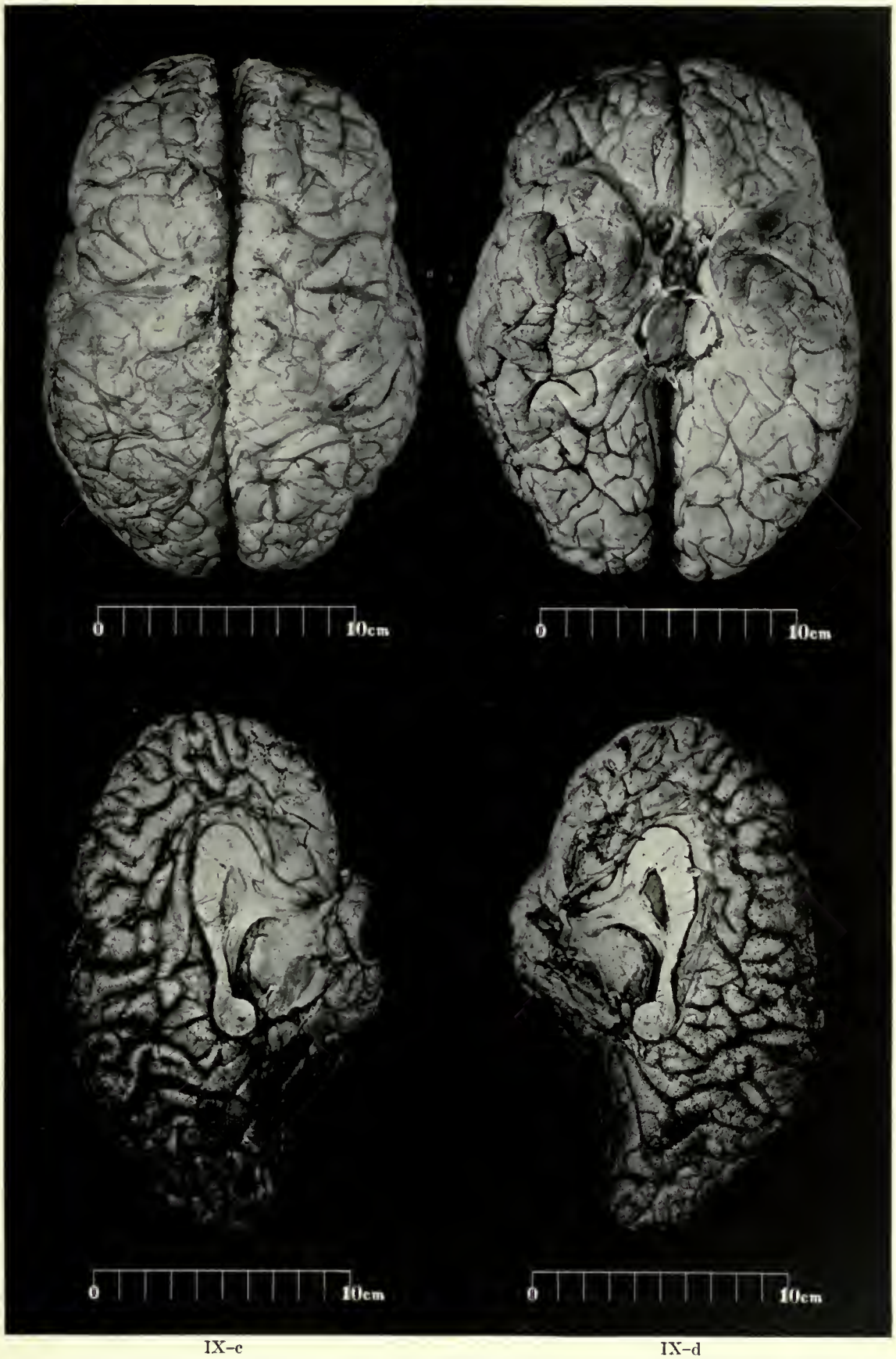
VIII–g

VIII–h

VIII–i

CASE VIII — MORON OR SUBNORMAL. EXECUTED MURDERER

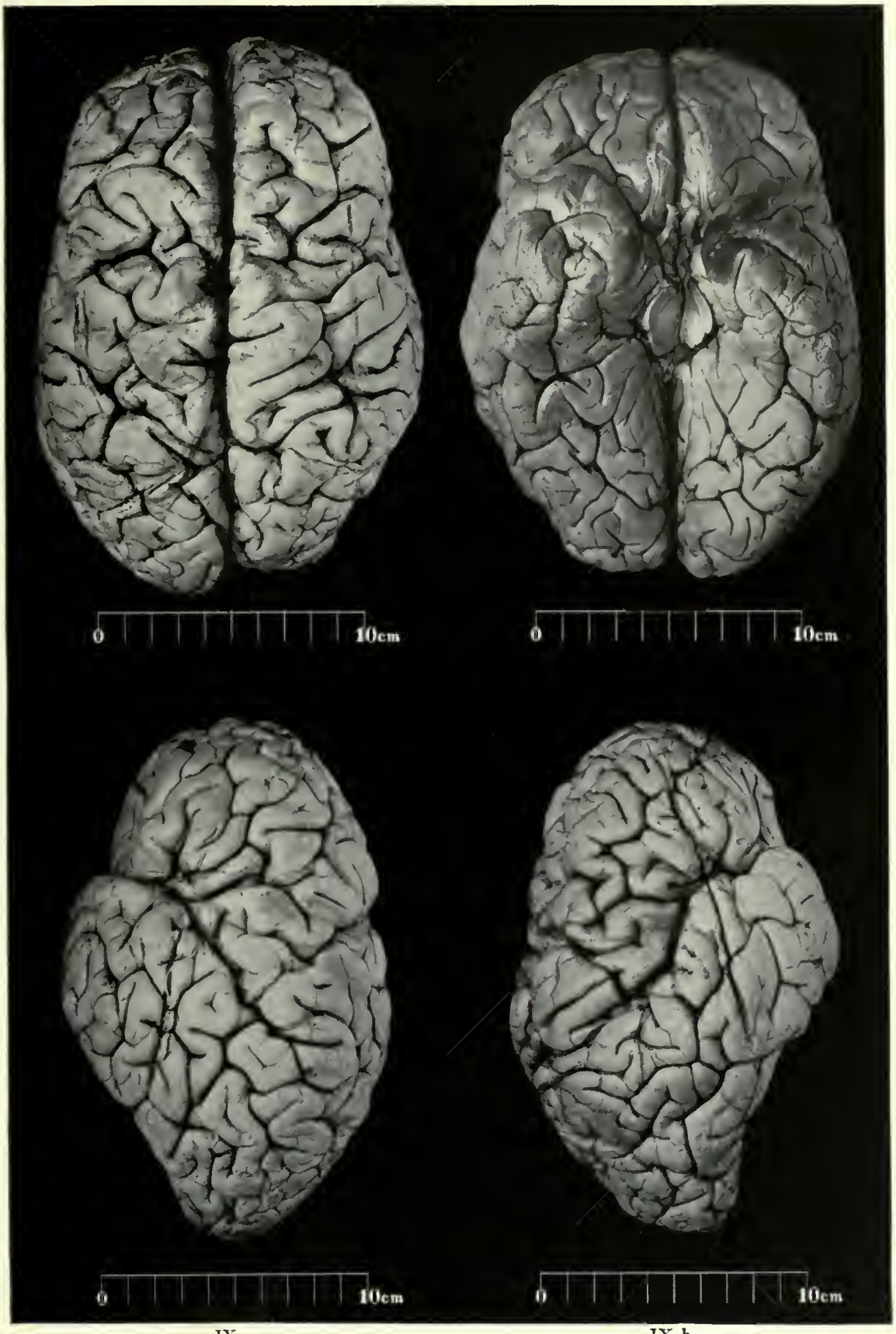
IX–a IX–b



IX–c IX–d

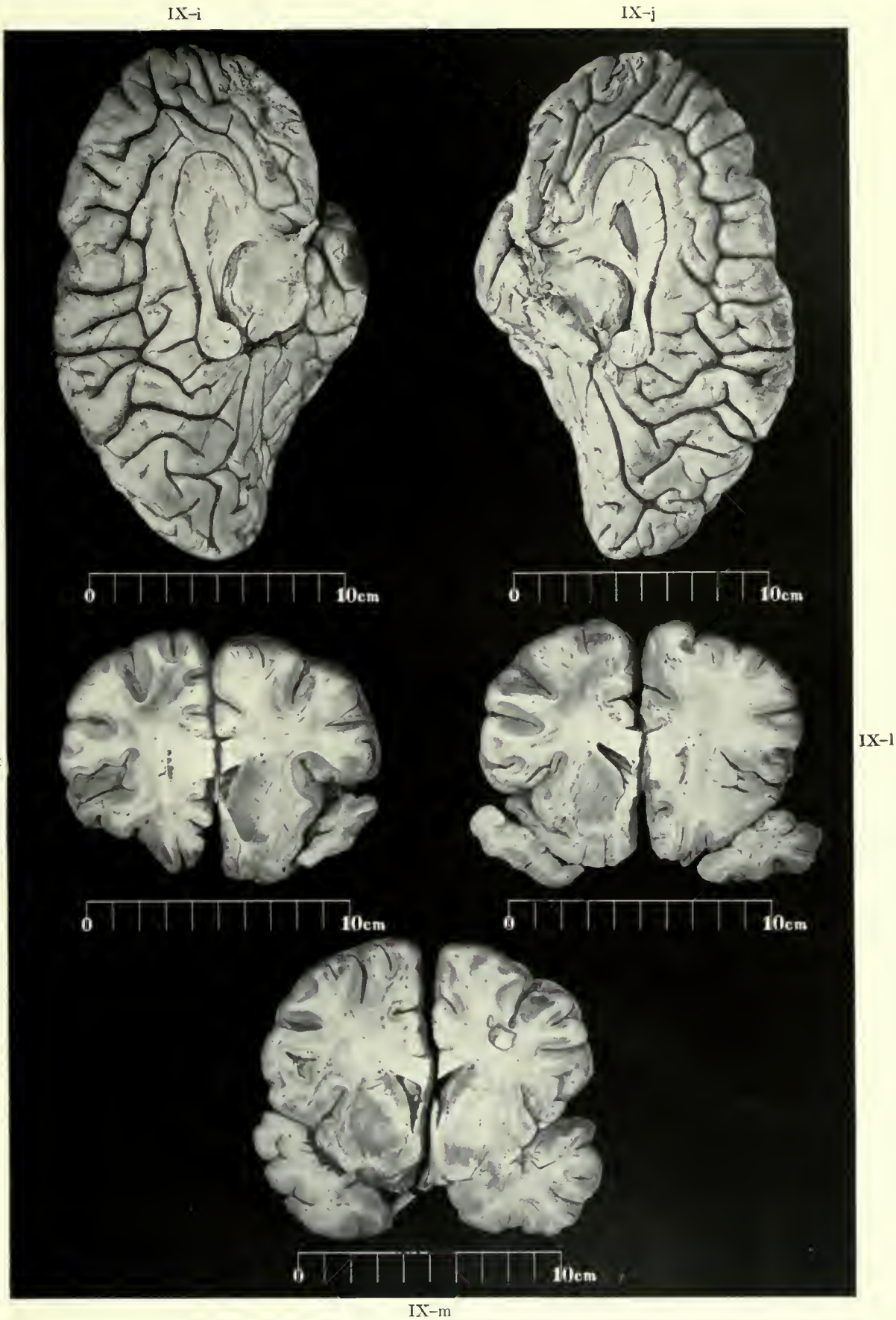
CASE IX — "ABLE BODIED" IMBECILE. BRAIN WEIGHT 1622 GRAMS

IX–e IX–f

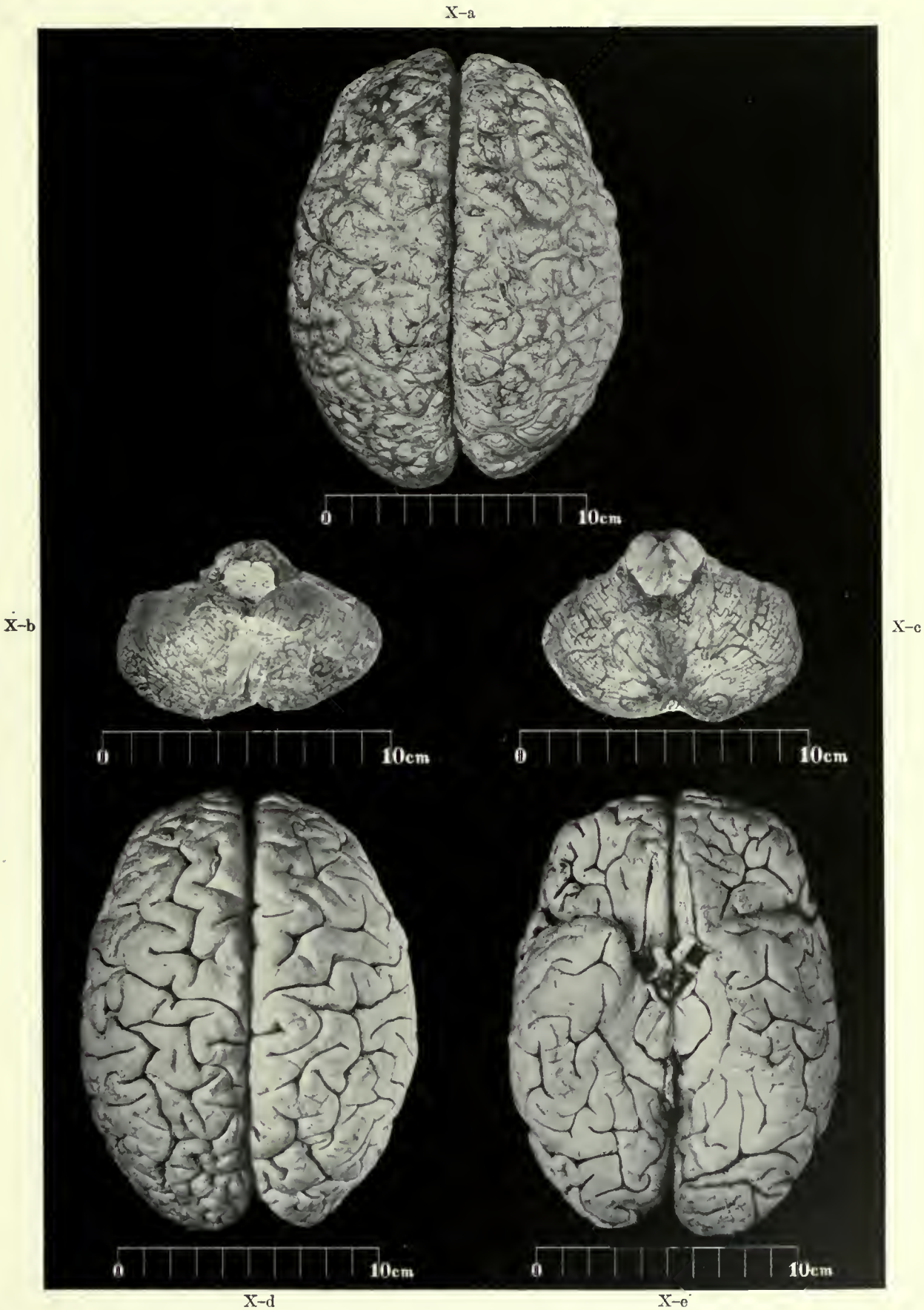


IX–g IX–h

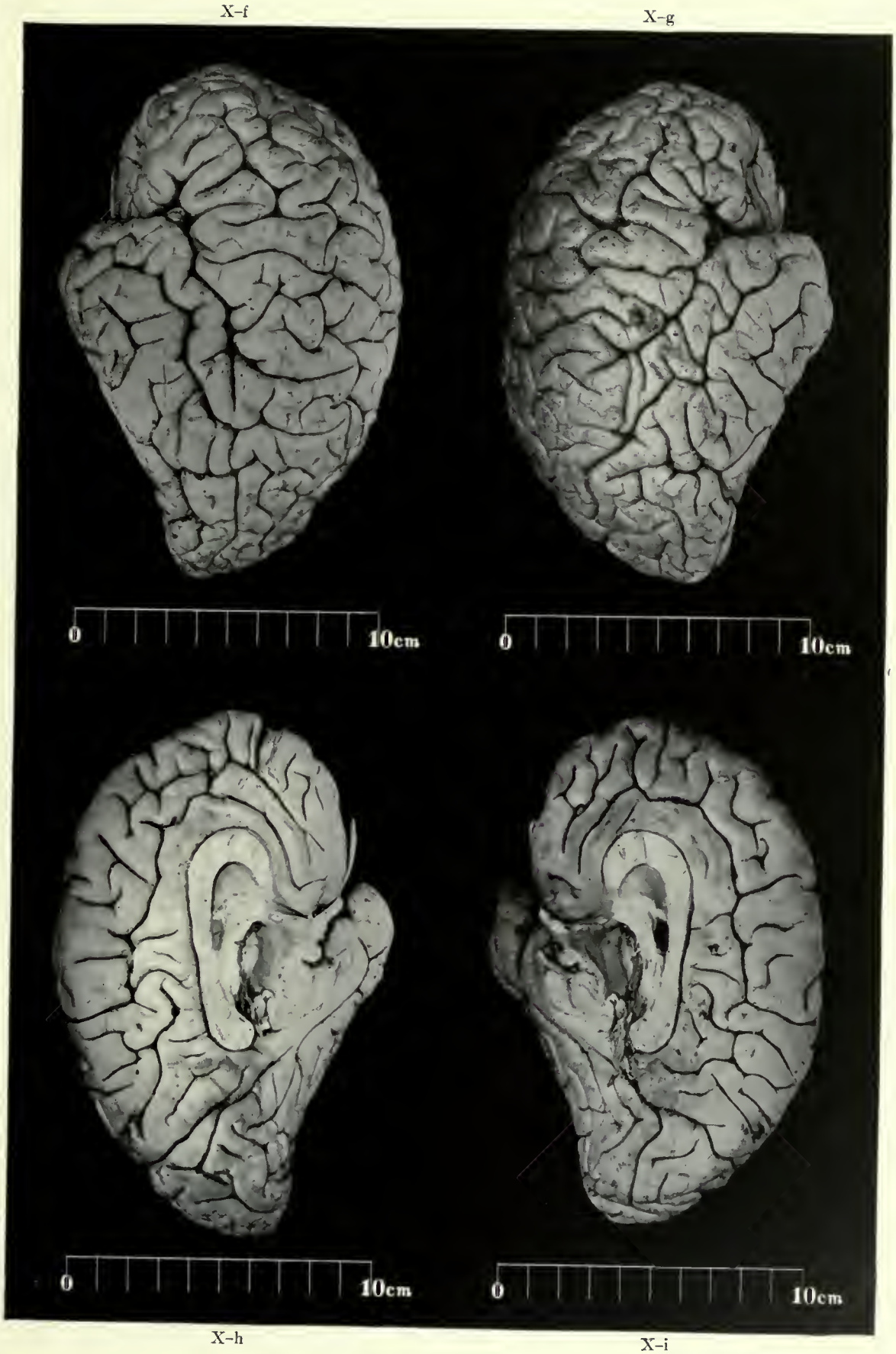
CASE IX — "ABLE BODIED" IMBECILE BRAIN WEIGHT 1622 GRAMS



CASE IX — "ABLE BODIED" IMBECILE. BRAIN WEIGHT 1622 GRAMS



CASE X — LOW IMBECILE (BINET 2.4)



CASE X — LOW IMBECILE (BINET 2.4)