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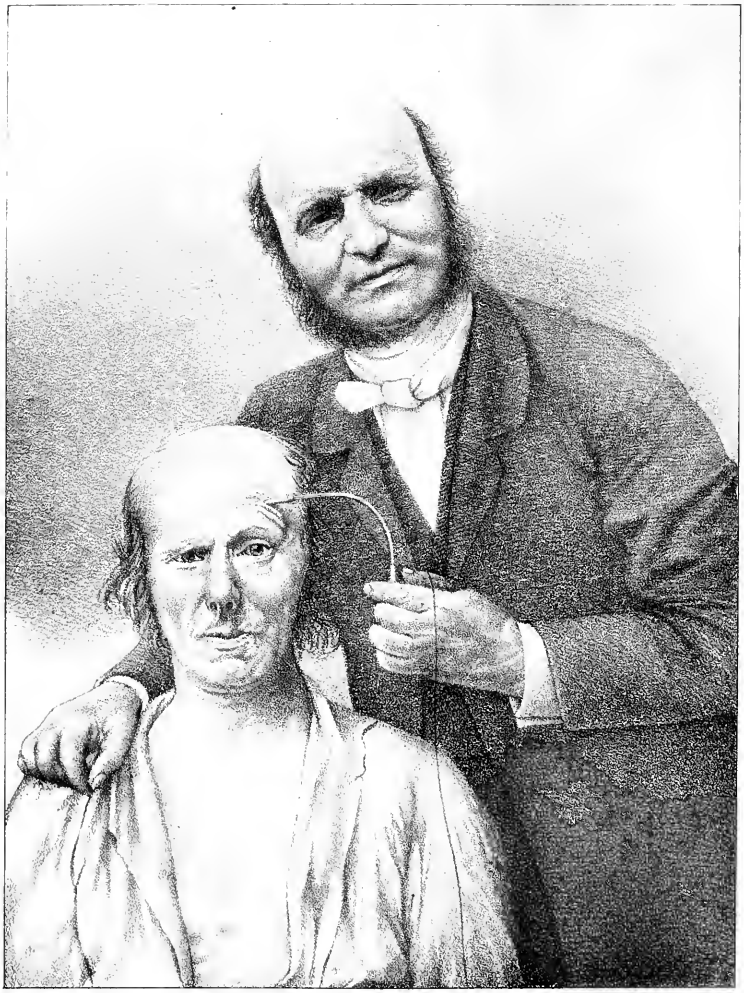

DUCHENNE FARADISING THE FRONTAL MUSCLE

From a photograph in the Album de Photographies Pathologiques (1862).

SELECTIONS

FROM THE

CLINICAL WORKS

OF

DR. DUCHENNE (DE BOULOGNE).

TRANSLATED, EDITED, AND CONDENSED

BY

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THE NEW SYDENHAM SOCIETY.

MDCCCLXXXIII.

PREFACE.

But few words are necessary in explanation of the method I have followed in editing this volume of selections from the clinical works of Duchenne de Boulogne.

Dr. Tibbits' well-known translation of the first part of *L'Electrisation Localisée* has made it unnecessary for the New Sydenham Society to undertake the translation of any of Duchenne's writings which treat of purely electrical subjects. The ensuing chapters, therefore, will be found to be almost exclusively devoted to those clinical observations by which the author's great professional reputation was mainly established.

The reader cannot fail to notice that, with very few exceptions, the selections are taken from the last edition of *L'Electrisation Localisée*. The reason of this is obvious. Duchenne's observations were originally published separately, or in one or other of the French medical journals, but almost all of them became ultimately incorporated with the book which to the last retained its inadequate and rather misleading title of *L'Electrisation Localisée*. Each edition of this work was bigger than the one which preceded it, its growth being mainly due to the addition of those papers on clinical, physiological, and pathological subjects which the author had written in the interval which elapsed between the editions.

It will be observed that there are no selections from the *Physiologie des Mouvements*, which by many would be considered the greater of Duchenne's two great works. Most of the propositions, however, which are put forward in the *Physiologie*

des Mouvements in a physiological form appear also, in clinical form, in *L'Electrisation Localisée*, and it will, in fact, be found that the action of most of the muscles of the body is considered in the ensuing pages. Again, the *Physiologie des Mouvements* is so great a work that one cannot but hope that it may be translated in its entirety for the benefit of the English-speaking races.

Some explanation is necessary as to the somewhat unusual method I have pursued in editing this volume. My commission from the New Sydenham Society was accompanied by an intimation that the selections must be comprised within the compass of a single volume of moderate dimensions. Had I followed the ordinary course of selecting a few of Duchenne's monographs and giving them entire I should have been able to give but a slight idea of the indebtedness of medicine to this great observer. Nobody has said of Duchenne that he was a concise writer, and all his critics seem agreed that prolixity of style was by him carried to an extreme degree. I was especially anxious to put the English reader in possession, if possible, of all the clinical *facts* which we owe to Duchenne, and in order to do so within the compass of this volume it became necessary to deal rather ruthlessly with mere words. My editing has therefore been, as it were, interstitial. I have weighed the value of every sentence and every paragraph, with the result that I have omitted much and compressed a great deal. I am well aware that in taking this course I have incurred no little responsibility. With regard to much that has been omitted there can, I think, be no doubt that its omission is a gain to the book. Polemical writing, of which there is fair amount in the original, I have thought wholly unsuitable for a modern scientific work; I have accordingly omitted all polemical passages, as well as many long discussions on questions which are now no longer matters of controversy. A great part of Duchenne's original papers are devoted to cases which differ from each other only on minor points. When an author puts forth

new views, it is, of course, necessary to substantiate such views by as many illustrations as possible; but when once these views are acknowledged, by common experience, to be correct, any redundancy of illustration becomes a hindrance rather than a help to argument and exposition. While, therefore, I have been careful to retain and give at length the various cases which serve as types of the morbid condition which may be under consideration, I have not scrupled to omit or condense a great many cases which are little more than repetitions of the original type.

In this way I have been able to give nearly all the clinical facts which Duchenne was the first to discover or satisfactorily expound in a space not much more than a third of that occupied by the original. I have been particularly careful to omit no argument which the author advances in support of his views, and I have endeavoured to make the necessary curtailments without interrupting the practical continuity of the text.

Wherever omissions or condensations have been made the fact has been indicated; in the former case by a few dots, or asterisks, and in the latter case by enclosing those passages of which an abstract only is given between square brackets, thus [ ].

It may be interesting to mention that in making this translation I have found at times no little difficulty in converting French anatomical terms into the Latin equivalents which are used in this country. The sole object of using Latin, for anatomical terms is to obtain some community of scientific expression among different nations. A reference to some of the lists of muscles which are scattered through this volume, and in which I have placed in parallel columns the names given to the same object in French and English anatomical text-books, will show that the French and English differ nearly as much in their anatomical as they do in their vernacular expressions.

To some of the chapters I have appended an Editorial Note, in which I have endeavoured to give some of the more recent facts which seem to throw light upon the subject treated of.

My best thanks are due to my friend Mr. William Pasteur, M.B., who kindly undertook to revise the proof sheets, and by so doing he has rendered most valuable service both to the editor and reader.

G. V. Poore.

30, Wimpole Street, W.,
July 25th, 1883.

A SHORT SKETCH

OF THE

LIFE AND WORK OF DUCHENNE

(DE BOULOGNE).*

Guillaume Benjamin Amand Duchenne was born at Boulogne-sur-Mer, on September 17th, 1806. His family had lived at Boulogne since about the middle of the eighteenth century, and he ever retained for his native town an affection which was truly filial. His mind, his character, and his features, all marked his Boulognese descent. Below the middle height, thickset, active in movement, slow of speech, and retaining to the last some of his provincial accent, his appearance recalled his father, Jean Duchenne, a sea-captain of Boulogne. He was educated at the local college, whence he went to pursue his studies at Douai, and at the age of 19 he was admitted Baccalauréat-ès-lettres on the same day as his friend and schoolfellow Dr. C. Gros, to whose lot it fell, half a century later, to deliver his funeral oration. His youth was passed without incidents which call for any remark. From Douai he went to Paris to study medicine, and in 1831 he returned to Boulogne to pursue the practice of his profession. His graduation thesis was an "Essai sur la brûlure" (35 pp. 4to. Paris, 1831). About the year 1833 he seems to have tried the effect of electro-puncture of the muscles on a patient under his care, and this operation (in which he was assisted by his friend

* For most of the facts in this brief memoir the Editor is indebted to a notice by Lasègue and Straus which appeared in the *Archives Générales de Médecine* for December, 1875. Certain other facts have been found scattered through the writings of Duchenne, and in various short biographical notices of him which appeared at the time of his death in French and English journals.

Gros) appears to have been the first means of directing Duchenne's mind to those obscure questions on which he was destined to throw so much light. He devoted himself more and more to the study of the curative value of electricity, and as his native town did not offer a sufficient field for observation he removed to Paris in 1842. Here he worked without ceasing for over thirty years. While the last edition of *L'Electrisation Localisée* was in course of preparation the Franco-German war occurred, and the siege of Paris put a stop to his labours for a time. In the preface to this (third) edition Duchenne says (July 31st, 1872): "The printing of this third edition took place during our unhappy war with Germany, and at a time when my health is seriously affected by a long and painful illness. These unfortunate circumstances have been the cause of the printing errors which have escaped my notice, but which the intelligence of the reader will easily correct." What was the exact nature of the illness from which Duchenne suffered during his later years does not appear, but he died from the effects of cerebral hæmorrhage on September 17th, 1875, the 69th anniversary of his birth.

On his arrival in Paris in 1842, Duchenne set himself to work with an ardour which has rarely been equalled, and never surpassed. He sought no official appointment, and he was never attached to any hospital. It seems extraordinary that, under such circumstances, he should have succeeded in collecting such a mass of clinical facts.

In a footnote on page 601 of the *Electrisation Localisée*, Duchenne, in replying to the reproach which had been levelled at him of neglecting the study of pathological anatomy, says: "Those who have made these reproaches were doubtless ignorant of the fact that I have merely been permitted to *glean*, as it were, in our hospitals, and that if I have been able to exist in a scientific sense, it is only because I have been fortunate enough to collect, by searching over the numerous clinics of our hospitals, certain clinical facts which have been, I will not say *despised*, but, rather, which have escaped notice. Those who have criticised

me should know also that when I have happened to discover a morbid *species* not hitherto described, I have had no control over the subjects upon which, when living, my clinical observations were made. I should have had, it is true, the right of completing my researches by anatomical investigation if I had been a hospital physician. But then, *riveted* as it were to my *own* wards, I should not have been able to do the work of a *searcher* which I set myself to do, and I am sure that my chief works (among others those on progressive muscular atrophy, atrophic paralysis of childhood, progressive locomotor ataxy, glosso-labio laryngeal paralysis, and pseudo-hypertrophic paralysis) would never have seen the light. To accomplish these, in short, a field of observation more vast, and extending to all the hospitals of Paris, was necessary. If then I have renounced, and still renounce, the honour of an official position, it is because I wish to give myself up freely and entirely to my irresistible taste for physiological and pathological researches. Ought it to be a subject of reproach to me that I have disinterestedly followed science solely for the love of it?"

Tied by no hospital appointment, nor expending his energies in lecturing, Duchenne was free to follow his own bias in the field of work which he had set himself to explore. The large amount of work which he accomplished seems to be due mainly to three circumstances: 1. The great natural energy of the man; 2. The fact that he followed that line of work for which he was by nature fitted; 3. The absence of distractions in the form of teaching and routine duties.

How often do we see men pursuing a line of study with admirable energy, but without obtaining results at all proportionate to the energy expended, and mainly because they have chosen a field of work for which they have no special talent. Duchenne seems to have been blamed because he devoted his attention more to bed-side observation than to microscopical anatomy. The fact was that he had no special talent for the pursuit of morbid histology, and although latterly he devoted some attention to

the subject, and was one of the first who attempted to fix microscopic results by the aid of photography, he did not succeed in adding to our pathological knowledge, and one cannot but feel that anything which took a man of his habit of mind away from the bed-side, where he was unequalled, did not, to say the least, help the advance of medical science. A wise division of labour has been, and will be, a powerful cause of the advance of knowledge, and a great clinical observer should rather be encouraged than otherwise to leave the delicate work of histology to those whose natural talents lead them to devote their best energies to the pursuit of it.

Every morning with rare exceptions, we are told, Duchenne visited one or two hospitals, selecting for study those cases which most suited his purpose, reading the notes of the case and showing his honest willingness to profit alike by the positive facts and the more doubtful hints. In return, he freely placed his well-stored mind and his technical experience at the disposal of any and every one. In the course of friendly discussions he made no secret of his ideas and notions, or of the work which he had in hand, and with him one could not be reserved or reticent. There were but few hospital wards in Paris where he was not welcome, and those in which he was really "at home" were amply sufficient as a field for his activity. By this mutual arrangement of give and take, he attained the end which is sought after by scientific societies, without the risk of reading papers or the formalities of debate.

It is easy to comprehend how unwilling he was to fetter his productive liberty by official duties, and one understands also how the desire of being a fellow of learned societies had little influence with him. Many learned societies, from that of Boulogne-sur-Mer to that of Moscow, had made him an honorary or corresponding member; but such honours, though they might flatter him, could not turn him from his work.

To believe that this position was won without difficulty would show but a poor knowledge of human nature. He had had his

struggles, and had suffered not a few humiliations; for he had found it necessary, in addition to the permission of the physician, to win the good-will of the more sensitive students. Duchenne possessed a perseverance which overcame the greatest obstacles, but he wanted that pliability of character which avoids conflict. One can fancy the annoyance which manifest or latent opposition must have caused him, and how long it must have taken him to win the freedom of the hospitals.

Scientific courage, of which Duchenne possessed so much, does not lead to great adventures, but shows itself in a passive resistance which none the less imposes painful sacrifices. Few men have resigned their full susceptibilities to an equal extent, or have opposed ill-will by such a passive determination. Success rewarded his efforts in the end, and when it was attained his task was easy. How many young physicians would have the resolution to do as he did? What time is there for original work while life is being spent in competitions? The unsuccessful retire exhausted and half ashamed, and they dislike re-entering the hospital to take a position subordinate to their more successful rivals. Conventionality and the exigences of practice work together in restricting the field of their observations, and instead of collecting valuable material they merely accumulate prejudices. Duchenne had the great merit, and he stands almost alone in the possession of it, of never being satisfied with his wealth of facts. Like men of business he was never satisfied with the sum of his (scientific) riches; and thus we find him, even when worn by suffering, working still with all the ardour of youth.

If it is instructive to contemplate what one may call his way of getting, it is not less so to inquire by what succession of ideas he turned to account the material which he unceasingly stored up. Beginning with treatment, and after lingering long over clinical work he finished with pathological anatomy. By diligently following his hard-working *confrères* every novelty was brought to his notice, and he had no reason to go against

the stream. His great powers of observation were of service to him when with physicians as when with patients.

It was not possible to cut his work in two—one part original, and all his own and the other borrowed, inspired, unconsciously it may be, by conversations, or bred of the scientific atmosphere in which he lived. Hence arose disputes as to priority, from which, however, his best works have escaped, and quarrels now forgotten which, far from discouraging him, served but to whet his zeal. The jealous disputes of savants are but "a spur to prick the side of the intent," and many a man of eminence has reason to bless rather than complain of the bitterness of criticism. It is so ill-mannered to praise oneself that anything which gives an excuse for asserting originality and accuracy does one a friendly service. By pleading for your own the world is taught that you are the real proprietor.

Influenced or not by his surroundings, Duchenne restricted himself to discovery. His course in passing from therapeutics to clinical observation, and thence to pathological anatomy, was not the result of chance. He was aware when he tacked, and knew his reason for doing so better than any one.

Whoever has worked up the action of one or two drugs which may have taken his fancy, and feels no necessity for reconsidering his pathology, must be a short-sighted investigator; our classifications alter whenever the object in view of which they were made alters.

Already the nosology of the pathological anatomist is not that of the clinician; but therapeutics, in the limited sphere in which Duchenne worked, has very special needs. With little knowledge of agents other than electricity he might be compared to the mineral water physicians, but with this difference, that he could choose his patients.

The therapeutic problem resolved itself to the formula, "In what cases will the remedy succeed?"

For the practitioner who roams at large over the almost indefinite range of medicine the question assumes a different form.

If the mode of treatment selected proves of no use, another must be tried; the failure of to-day may be turned into a success to-morrow by employing other means. Pathology takes higher rank than treatment, and one is more ready to blame the insufficiency of the treatment than to find fault with the notions of the pathologist, and it is the former which is ever in need of reform.

With one remedy only, and a firm resolution to stick to it, therapeutics become fixed and dominate pathology; diseases divide into two classes—those which do and those which do not yield to the remedy. The remedy becomes in fact a touchstone.

In this respect electricity is better than hydropathy or mineral waters. Producing immediate effects, easy of management, and not requiring the removal of patients, it is even more easy to use than drugs; and its sphere of action is restricted, like that of other local remedies which produce no vague impressions on the organism as a whole. In the hands of specialists more versed in the intricacies of their machines than in the study of disease electricity had a doubtful position, and the good attributed to it barely compensated for its evil repute.

Duchenne saw quickly enough why it was that electricity held this doubtful position, and his ingenuity devised what might almost be called an electric pathology. By this door, which he was one of the first to open, he penetrated into the very heart of nervous pathology. By considering the state of nervous pathology when he began to work we shall appreciate the progress which it has made, and to which he contributed so powerfully.

When electricity, from being merely a means of treatment, began to be employed for investigation and classification it was used entirely for motor troubles, and almost entirely for motor deficiencies. Spasms, contractions, and contractures had escaped its action, and it was in this vast domain, among others, that the activity of Duchenne found work.

Convinced of the necessity of grounding his work on the muscular system, he began his magnificent labours on the physi-

ology and anatomy of the muscles. Later he investigated the nervous system with similar success. His chief glory will be that he produced order out of chaos, and physiology no less than medicine will remain indebted to him. Before his investigations the action of individual muscles, the co-ordination and co-operation of their contractions, the part which each played in the movements of each region (limbs, trunk, or face), was but vaguely known. He classified and defined and demonstrated the justness of every dogma he uttered. The task was heavy, and of this the best proof is that he has but few followers. Usually when a leader shows the way others press upon his heels, but here the way is too steep for the crowd to follow him.

In the last period of his scientific life Duchenne became less interested in clinical work. For the study of the muscles he substituted that of the nervous system, and took to pathological histology with an ardour which was really juvenile. His conversion was late, and in spite of his powers of observation and description, and in spite of his talents as a photographer, he merely succeeded in confirming ideas which were not his own.

Without doubt Duchenne will take a firm position at the head of that progress which we have seen born, and which now is still surely advancing. His numerous monographs and papers will remain a lasting monument, an inexhaustible mine, where those of to-day and those who will come after will always find a new lode to explore.

It is the sovereign and decisive quality of master minds that their works may be read and re-read without exhausting our curiosity, and to comment upon or develop the propositions they contain is accounted a merit. The teachings of a master may be modified or reversed, but one can never be indifferent to their first creation.

In a work devoted to the clinical writings of Duchenne it would be out of place to criticise them. The reader of the following pages will do that for himself, and the list of his works

given at page xxi. will give an adequate idea of the range of his labours, and of his untiring industry.

Great as is the debt we owe to Duchenne for his work in the field of clinical medicine it is only just to say that by many his great book on the "Physiology of Movement" is considered his masterpiece. Its originality is absolute, and the assertions which he made in it, many of them startlingly new, with regard to the action of muscles, have for the most part stood the test of time.

The following notes culled from various sources may serve to bring Duchenne more forcibly to the mind's eye of the reader.

A writer in the *Gazette Hebdomadaire*, quoted in the *Medical Times and Gazette* for October 2nd, 1875, says: "The priority of Duchenne's discoveries has often been disputed, and especially with regard to locomotor ataxy. It is well known that he was not what is called an erudite; and at the time when his first works appeared he read but little. No one was more astonished than himself when it was proved to him that certain facts —which he as well as a great many others believed to be absolutely new—had been described before, if not in France, at least among our neighbours. The merit of the invention was not, however, the less for him, and what cannot be denied is that he knew how to place it in a light and bring it out in relief which were unknown before.

"Who does not recollect the astonishment exhibited in the clinic by that experiment of Duchenne of drawing from his bed a patient regarded as absolutely paraplegic, and loading him with the weight of a man of ordinary size without his ever flinching under it? It was one of his merits to always have at his disposal some demonstrative procedure capable of striking the attention and fixing the facts on the memory.

"Until his last days he was ever working, investigating and perfecting. The clinic, at the bedside of his patients, was his place of predilection. He expounded his ideas badly, his diffuse explanations contrasting with the certainty of his investigations and the clearness of his conclusions, which he was fond of

summing up in formulæ easily retained. It was during the examination of the patients that he recovered all his superiority, no one being better able than he to analyse a clinical fact. His patience was extreme; nothing was neglected. How many patients have been watched by him for several years, and at the same time aided, succoured, and encouraged with a benevolence that never was wanting! This patience in investigation, joined to his remarkable faculty of invention, constituted Duchenne truly an exceptional observer. His ardour never abandoned him. He was a true artist in medicine. His reputation, slowly acquired, increased day by day, and his superiority was rarely met by incredulity."

On September 25th, 1875, the *Lancet* says: "Duchenne had a stroke of paralysis about three weeks ago, from which he partially recovered, but which has ended in destroying his life."

A writer in the *Lancet*, for October 23rd, 1875, who apparently had some personal acquaintance with Duchenne, makes the following among other remarks on the character of the then lately-deceased physician:—

"When the day comes in which medical workers of the present generation can be regarded in the perspective of the past, perhaps no figure will stand up so conspicuously among the rest as that of him whose body was lately laid in his native town, towards which he had turned his life through with such warm affection. Duchenne 'de Boulogne' has written his name among the clinical facts of medical science in indelible characters, and in his own special qualities his form will always tower distinct among his contemporaries. No field of work was ever seized upon with more eagerness, ever cultivated with more earnestness, or perhaps ever made to yield a better harvest, than that which the discovery of induced electricity placed at the disposal of the man whose genius should first recognise and talents secure the opportunity which it afforded. The new means of investigation which faradisation presented were prosecuted by Duchenne in every direction. Dark places in physiology and

pathology were made plain, and the advance which each science received was real and considerable. Almost the whole of the muscular system of the body was brought by him within the range of experimental observation, and both the mode and form of its function were investigated and illustrated by a method of unrivalled clearness and unsurpassed accuracy. . . . . The credit of discovery can hardly be denied to Duchenne, even in the cases in which he had been, unknown to him, anticipated, as was undoubtedly the case with regard to locomotor ataxy. . . . . His mind, indeed, was hardly less precise in its action, less comprehensive in its grasp, less secure in its results than the agency which he employed. His energy was inexhaustible. Fed by an unfailing spring of enthusiastic love for his work, it carried him successfully over obstacles which would have arrested many workers of inferior force. No pains were too great for the full investigation of a case in the most laborious manner; no time or trouble too much to secure accuracy of detail and result. No one who had witnessed his demonstrations of pathological or physiological facts could fail to be struck with the remarkable care and quickness, but, withal, painstaking precision with which he worked. Somewhat profuse in description, his utterances had that sharp emphasis which, as it were, italicised the salient points of his discourse. A rather quaint mannerism marked his delivery, which was accompanied by the demonstrativeness of style so common to his nation. Few will forget the quiet explosive '*Bon!*' with which he used to announce the satisfactory result of his experiments. . . . His style of description was graphic, but to an English reader diffuse and a little tedious, especially when drawn on by the attraction of past polemics to 'fight his battles o'er again.' . . .

"Taking his work at its lowest estimate, he was a man to whom medical science owes a large debt of gratitude, and whose memory deserves a warm tribute of regard."

The Paris correspondent of the *Lancet*, in a letter which appears in the number for October 30th, 1875, states that

Duchenne was born in 1806. After enumerating his works the writer goes on to say: "His features were familiar to all who visited habitually the wards of the Paris hospitals. Every morning Duchenne was to be seen in one or other of the hospitals, studying cases, examining specimens, drawing his photographs of microscopical appearances, in which he was extraordinarily skilful. For a long time Duchenne's invariable presence in the wards, his incessant moving about, his ardent interrogation of patients, caused him to be looked upon with a somewhat suspicious and anxious eye by many of the hospital physicians. But his consummate experience of disease, his wonderful keenness and ability in making out a diagnosis in cases of paralysis, the sincerity and earnestness of his manner, the honesty of his proceedings, the authority which he gained by the publication of his original researches, the services which he rendered daily in the wards of the hospitals, brought him the esteem and appreciation of all, and made him a welcome guest everywhere.

"His patience was extraordinary. He would pursue the investigation of a case for years, never losing sight of it, and following the patient in his peregrinations from hospital to hospital, and from house to house, often affording help and means of subsistence.

"It may be said of Duchenne that under many adverse circumstances—the suspicions of *confrères*, the disputes as to priority, the difficulty of finding a field of study and experiment, as he had no hospital appointment—his reputation has come out clear and bright as an honest, hard-working, acute, and ingenious observer, an original discoverer, a skilful professional man, and a kind-hearted, benevolent gentleman."

LIST OF DUCHENNE'S WORKS.

THE following list of Duchenne's works, in the order of their publication, is taken from the third edition of *L'Electrisation Localisée*:—

1. On the Art of Limiting Electrical Stimulation in a given Organ without Pricking or Cutting the Skin: called Localised Electrisation (compte rendu de l'Academie des Sciences, 1847, and *Arch. Gén. de Méd.*, 1850—51).
2. Description of a Double-current Volta-Faradic Machine. 1848.
3. Investigations in Electro-Physiology, Pathology, and Therapeutics. A series of Memoirs presented to the Academy of Science in May, 1849 (*Couronnés par l'Institut*). One of these papers was on *Wasting of Muscles with Fatty Change*, and on Wasting Palsy from Injury or Lead Poisoning.
4. Notes on "*Cauterisation Auriculaire*" as a Treatment for Sciatica, and on Stimulation of the Skin by Electricity as a Treatment for this form of Neuralgia. 1850.
5. A Critical Investigation, by the help of Electricity, on the State of Contractility and Sensibility of the Muscles in Paralysis of the Upper Limbs. 1850.
6. On the Functions of the Facial Muscles. 1850.
7. On the Choice of Induction Apparatus, &c. 1850.
8. Investigation of the Physiology and Pathology of the *Chorda Tympani*. 1850.
9. The Application of Localised Galvanisation to the Study of Muscular Function. 1850—51.
10. On Muscular Irritability in Paralysis (a reply to Marshall Hall). 1850.
11. Investigations on the Physiological and Therapeutic Power of Frictional Electricity; Electricity of Contact and Induction Currents. 1851.
12. Electrical Investigations on the Physiology and Pathology of the Muscles which Move the Shoulder, Trunk, and Arm. 1852.
13. Electrical Investigations on the Special Action of the Muscles of the Thumb and Fingers. 1851—52.
14. Comparative Study of the Anatomical Lesions in Progressive Muscular Fatty Atrophy and General Paralysis. 1852.
15. On Electrisation of the Skin in Angina Pectoris. 1853.
16. On the Use of Localised Electrisation in Progressive Muscular Atrophy. 1853.
17. On the Special Action of Induction Currents on Muscular Tone. 1853.
18. Electrical Investigations (Physiological, Pathological, and Therapeutical) of the Diaphragm. 1853.
19. Investigations of a New Property shown by Disease, the *Power of Movement Independent of Sight*. Called by the author *Muscular Sense* (*Conscience Musculaire*). 1853.
20. Electro-Pathological Investigation of the Uses of Muscular Sensibility. 1853—54.
21. On the Therapeutic Action of Localised Electrisation in the Treatment of Palsies due to Cerebral Hæmorrhage. 1854.
22. On the Influence of Localised Electrisation on Facial Palsy, and on the frequent Termination of this Disease by Contracture. 1854.
23. Infantile Wasting Fatty Palsy, its Diagnosis, Prognosis, and Treatment by Localised Electrisation. 1855.
24. On the use, in Chloroform Poisoning, of Artificial Respiration produced by Faradising the Phrenic Nerves. Addressed to the Société Médicale d'émulation. 1855.
25. Investigation of the Second Stage of Walking. 1855.
26. Irritability is not necessary for Motility, or the Integrity of Electro-Muscular Contractility is not necessary for Voluntary Movement. 1856.
27. Note on Certain Differences in the Properties of Induction Currents of the First and Second Order. 1856.

28. Investigations of the Muscles which Move the Foot on the Leg. 1856—57.
29. Orthopedy of the Hand. 1856—57.
30. Physiological Orthopedy, or Practical Deductions from my Electro-Physiological and Pathological Researches on the Movements of the Hand. 1857.
31. Faradisation of the Chorda Tympani and Muscles of the Ossicles applied to the Treatment of Nervous Deafness. 1858.
32. On Functional Spasm and Paralysis. 1859—60.
33. Investigation of Locomotor Ataxy; a Disease specially Marked by General Troubles of Motor Co-ordination. (Compte rendu de l'Académ. des Sciences; Bullet. de l'Academ. de Médec., 1858—59., t. xxiv., p. 210; and *Arch. Gén. de Méd.*, December, 1858, and Jan., Feb., and April, 1859.)
34. Of the Genesis of Valgus Flat Foot by Palsy of the Peroneus Longus, and of Valgus Hollow Foot by Contracture of the Peroneus Longus. 1860.
35. Progressive Muscular Paralysis of the Tongue, Soft Palate, and Lips. A disease not before described. (*Archiv. Gén. de Méd.*, September and October, 1860.)
36. Physiological Muscular Prothesis of the Lower Limbs. 1861.
37. On the Curability and Diagnosis of Nervous Deaf-muteness by Faradisation of the Chorda Tympani and the Muscles of the Ossicles. 1861.
38. The Mechanism of the Human Countenance; or an Electro-Physiological Analysis of the Expression of the Passions. Paris, 1862. With an Atlas of 74 Electro-Physiological Photographic Figures.
39. Album of Pathological Photographs. Complementary to the book called "Localised Electrisation." Paris, 1862.
40. Medical Dynamometer. 1862—63.
41. Clinical Investigation of the Condition of the Great Sympathetic in Locomotor Ataxy. 1864.
42. Photo-Autography, or Autography on Metal and Stone of Photo-Microscopic Figures of the Nervous System. 1864.
43. Differential Diagnosis of Cerebellar Affections from Progressive Locomotor Ataxy. 1864.
44. Researches in the Microscopic Photography of the Nervous System. 1864.
45. Photo-Autographic Microscopic Study of the Ganglia of the Great Sympathetic of the Neck. 1864—65.
46. The Tissue Punch. 1864—65.
47. Microscopic Anatomy of the Nervous System. Researches with the help of Photo-Autography on Stone and Zinc. 1865.
48. The Movements of Respiration. 1866.
49. The Physiology of Movements, Demonstrated by the Help of Electrical Experiment and Clinical Observation, Applicable to the Study of Paralysis and Deformities. Paris, 8vo., pp. 872, and 101 figures. 1867.
50. On Cramp of the Foot, or Functional Impotence of the Peroneus Longus and Functional Contracture of the Peronens Longus. Paper read at the Surgical Section of the Medical Congress at Oxford in August, 1868.
51. On Pseudo-Hypertrophic or Myo-Sclerotic Paralysis (*Archives Gén. de Méd.*, 1868).
52. Photographic Iconography as an Aid to the Study of the Intimate Structure of the Nervous System. 1869.
53. Icono-Photographic Researches on the Morphology and Structure of the Human Medulla; their Application to the Anatomo-Pathological Study of Glosso-labio Laryngeal Paralysis. 1870.
54. Functional Impotence and Functional Spasm of the Peroneus Longus (*Arch. Gén. de Méd.*, 1872).
55. Note on the Pathological Anatomy of Five Recent Cases of Pseudo-Hypertrophic Paralysis. 1872.
56. Duchenne's great work, "De l'Electrisation Localisée," passed through three editions. The first edition was published in 1855; the second edition, consisting of 1046 pages and 179 figures, with one lithographic plate, was published in 1861; and the third edition, consisting of 1120 pages, with 255 figures and 3 lithographic plates, was published in 1872, the preface bearing the date of July 31st.
57. Graduation and Dosage of the Continuous Current, chiefly by the Rheostat-Voltameter. 1873.

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NOTE FOR THE READER.

In all cases where omissions have been made from the original text, the fact has been indicated by a line of dots, thus . . . Passages of which a condensed abstract alone is given are included between square brackets, thus [ ].

SELECTIONS

FROM THE

CLINICAL WORKS OF DUCHENNE

(DE BOULOGNE).

CHAPTER I.

PROGRESSIVE LOCOMOTOR ATAXY.*

Researches on a disease specially characterised by general troubles in the co-ordination of movements. [Duchenne's first description of this disease was published in the *Archives Gén. de Méd.*, 1858—59.]

PROGRESSIVE loss of co-ordination of movements, and apparent paralysis, contrasting with an undiminished muscular power, are the fundamental characteristics (but not the only ones) of the disease which I propose to describe.

Its symptoms and progress constitute a perfectly distinct morbid entity. I call it "*Progressive Locomotor Ataxy*," a name which I shall justify further on. If I were asked to give a more complete definition I would suggest the following, although (necessarily) a long one. Progressive locomotor ataxy is characterised :—

1. By increase of the connective tissue (sclerosis) of the posterior columns of the spinal cord affecting more or less of its length ;

2. In the first stage by squint due to paralysis of one or more of the motor nerves of the eye ; by wasting of the optic papilla, and by irregularities of the pupil ; by peculiar pains which are specially characteristic—deep, shaking, stabbing, and like lightning—and accompanied by over-sensitiveness of the skin, of momentary duration and limited to the painful spot.

3. In the second stage by difficulties of co-ordination and

* From the last edition (1872) of *L'Electrisation Localisée*, pp. 616—671.

balancing, which are equally characteristic. These are worse in the dark, but are also present when the patient can make use of his eyes.

4. By a sense of weakness while standing or walking, which shows itself at the same time as the difficulties of co-ordination and balancing, and which is in strong contrast with the perfect power for partial movements.

5. By loss of sensibility in varying degrees (sense of pain and touch being lost in the skin and parts beneath, *i.e.*, nerves, muscles, bones, and joints), which increases the difficulty of co-ordination and balancing; but these latter troubles are, nevertheless, independent of the former.

6. By functional troubles affecting the organs of generation, bladder, and intestine (impotence or satyriasis, with difficulties in micturition and defecation).

7. Lastly, by the slow and fatal progress of the symptoms, and by their appearance, in general, in the order above given.

. . . . . .

Once on the track of this disease, I soon recognised its frequency. In a little time I had collected twenty cases of it without hunting after them, and all from my private practice or my own clinic. In 1857 I made a communication to the Société de Médecine of Paris on the existence of this disease as a distinct morbid entity (*espèce morbide*). Only a few weeks before the publication of my memoir on this disease, I pointed out in the different wards of the Charité and the Hôtel Dieu several typical cases of progressive locomotor ataxy in stages more or less advanced.

. . . . . .

Before going deeply into the study of this disease, I will relate a case reported by the patient himself, an intelligent and educated man. My own report would undoubtedly be more scientific, but it would, I think, have less real value; for no matter how honest we may be, we are always, without doubt, under the influence of preconceived notions while we are questioning a patient. Observations have been too often systematically collected for the purpose of proving a theory, and it has too often happened that many pathologists have given very different accounts of identical facts.

Here is the history of a case sent to me in 1856 by the

patient, M. X., a merchant, 48 years of age, living at Marsigny-sur-Loire.

Case No. 1.—*Typical Case of Progressive Locomotor Ataxy in its Third Stage.*

The patient is 48 years old; he for a long time lived in a damp house where the sun never penetrated, leading a very active life and much worried by business. In 1835 he woke in the night with violent pains in the calves, which were relieved by walking about, and a few months later he was troubled again in a similar way. Little by little these pains became more frequent, and in 1840 and 1841 the waters of Aix were prescribed for them. At the end of the second season he returned home very weak, and he then noticed when waltzing a difficulty in turning round. Very gradually his power diminished, and at the end of 1842 his walking had become unsteady like that of a drunken man, and particularly so in the dark. At the end of February, 1843, he went to Lyons for advice, and the physicians there declared him to be affected with a disease of the spinal cord. He then underwent very violent treatment (the administration of strychnine, and the application of caustics, actual cauteries, moxas, and frictions of Venice turpentine to the spinal column). In July he took the waters of Bourbon-Lancy for fifty days, which appeared to do him good and brought back some sensibility to his legs; but during the winter the disease became aggravated in spite of the continuation of violent remedies. Electricity by means of the electric machine—the "Crown of Cups" (*pile à godets*), acupuncture, the waters of Balarue, de Bourbon, were successively used without any result. The disease continued its slow progress. In the winter of 1844—45 six months' hydropathic treatment at Ternes (in the neighbourhood of Paris) stopped the progress of the disease and gave some strength to the patient, who continued to carry on the treatment at home for another year. In 1856 he passed another season of thirty-eight days at Bourbon. His physician advised him to take much exercise, and applied to him the "Machine de Breton." While he was taking the waters his legs got warmer and stronger.

State of the Patient in 1856.—All his senses were affected by the disease, the cause of which he could not determine. He had

never had any sudden attack, nor had he been guilty of any excesses, having always lived moderately as regards both eating and drinking. *Hearing:* quite lost in left ear, in which also there is a continuous buzzing. *Vision:* at the beginning of the disease the two eyes were not parallel, and there was distinct squinting. Since then the squint has gradually disappeared, but the sight, which was perfect, has become weak. The left eye is *presbyopic* and the right *myopic;* with the right he reads easily without glasses, the left not acting at a certain distance. *Smell and Taste* are apparently unaltered. *Touch:* the left arm and hand are slightly numb, and touch is obtuse; this is less marked on the right side, and the patient writes easily. His movements must be guided by the sight. With his eyes shut the left hand cannot find the tip of the nose, but the right does so easily. When in bed he cannot tell, even with his right hand, when the temperature is the same, whether he is touching his mattress or his thigh. When his skin is slightly scratched he recognises the fact, but he cannot tell whether it is the right or left thigh which is being touched. When he walks with crutches (*entre des perches*), managing to get some 1,200 paces in three attempts, resting every fifty steps, he is obliged to look constantly at his feet to direct them, for his soles do not appreciate the contact of the ground. Although the patient can stand upright for five minutes when clinging to the furniture, the least forward movement causes him to fall. The digestion has always been good; speech is free; intellectual faculties unchanged. Although somewhat sluggish, the bowels act regularly, but when he has diarrhœa he is unable to retain his fæces. Incontinence of urine, present at first, no longer exists. All his limbs are well nourished, and, from their appearance, one would not suspect any disease, but the hips are a little wasted. He sleeps very lightly and for short periods, and it is this which most fatigues the patient. The want of sleep must be attributed to the pain which will be mentioned hereafter. When the weather is changeable, the patient suffers from painful twitchings running with lightning speed over the whole body, with a resounding in the left ear (*retentissement*). With the exception of the vertebral column and back of the trunk these twitchings affect every part of the body from the crown of the head to the tips of the toes. Usually these pains are only felt

over a very small area at a time, and last for 12, 24, 36, or even 72 hours. They begin indistinctly with intervals which get shorter by degrees, until at last he can scarcely breathe four times without having a twinge. In the knee, for example, it feels as if a needle were slowly perforating the joint; in the foot, as though a horse were trampling upon it; in the thigh and calves, as if an iron rake were tearing them; while the wrists, arms, and chest feel as if they were being squeezed in a vice. In the head, above all, he suffers unheard-of agony. Sometimes it is as though the skull were being struck by a hammer, and at another time the shocks in the nerves of the neck are so violent that the whole head vibrates like a bell that is violently rung, and he is obliged to hold it to prevent its twitching. His sufferings have been lessened at certain times by different purgative and sudorific remedies, and during the last season by the waters of Bourbon.

In this case are found the chief phenomena of the disease of which I have sketched the main features above; it is only to be regretted that the patient has not classified them according to the order of their manifestation. In 1858, however, two years after sending the above history, M. X. sought my advice, and I was able to remedy this defect by my own inquiries and observations. On asking M. X. to tax his memory I elicited the following facts. 1. The double vision existed from the beginning of the disease (1835); the left eye was turned in without falling of the upper lid; in short, the sixth nerve was paralysed. The double vision lasted only a few months, but his sight, previously excellent, was weakened. At this time the pains, gradually increasing in severity, were present, as at all periods of the disease. These phenomena constituted the *first stage of the disease*, which lasted for several years. 2. A second stage was ushered in (towards the end of 1841) by troubles of movement and touch limited to the legs. M. X. asserts in fact that from the moment when he began to lose his balance (as in waltzing or turning short) the sense of touch had already become blunted in the soles of the feet; he felt the ground imperfectly, and he always seemed to be walking on a carpet. Later, when the disease grew worse, he felt as if he had elastic cushions under his feet which compelled him, as it were, to prance when he walked; then his balancing power became so bad that he could not stand without

help. In walking he threw his legs in front of him in a most irregular manner. It was at this time (towards the end of 1843) that a paraplegia from disease of the cord was diagnosed and treated, and of this treatment he bears unmistakable marks on either side of his spinal column. A few months later (in 1844) the sense of touch in the upper limbs became affected; at first a numbness of the ring and little fingers of the left hand and then of the right. This was the passage of the disease into its third stage, *i.e.*, the generalisation of the troubles of co-ordination. Already there was a difficulty in using the hand, not only because of the impairment of touch but also by reason of a certain difficulty in making the fingers work together.

This state of things got rapidly worse, and soon all the muscles of the arms were affected, till M. X. became unable to dress himself, and could only feed himself with difficulty. When I first saw M. X. in 1858 he could not rise from his chair, nor remain standing nor walk without the help of several persons, and even then he only managed to throw his limbs about in a most disorderly manner; the arms were as much affected as the legs. I should mention that sight was useless to control the disorderly movements. He attributed all his clumsiness to a lessening of the muscular power, or, as he had always been told, to paralysis; and he was not a little surprised when I showed him by means of the dynamometer that his muscular force in partial movements was at least as good as mine. I must not forget to mention that the paralysis of the *sixth*, which, according to M. X., had almost disappeared, completely returned in 1857, and from that time remained. I observed that the pain, which had always been intense, preserved the character so well described by M. X., and I noticed a peculiarity which is not without interest. The pains felt by M. X. were always deeply seated; nevertheless, when one made pressure on the painful spot at the moment of onset he felt great relief, but if this same spot were lightly rubbed the pain became terrible and referred to the surface as if the skin were being torn.

I noted also that the muscles had preserved their "electric contractility," but that the sensibility to faradism had very notably diminished; their nutrition was intact. M. X. became gradually impotent from the onset of the second period.

Lastly, the urine, analysed by M. Mialhe and myself, was normal.*

Symptoms. — The physiognomy or "facies" of progressive locomotor ataxy is known by those principal characteristics which I have just described, and which I need not recapitulate.

Neither shall I, in this section, classify those symptoms with which I propose to deal minutely, according to the order of their appearance, but rather according to their symptomatic importance. . . .

1. Troubles of Co-ordination.—A. The first sign of derangement in the lower limbs is a difficulty in standing without swaying to and fro, or without support, or in performing certain movements in walking, such as circular or lateral movements. The patient feels that he is threatened with a loss of balance; sometimes he feels a kind of weakness in the lower limbs. Usually the sense of touch in the soles of the feet is soon dulled or altered in some way. Sometimes, when patients walk on a hard floor (pavement or parquet) it seems to them as if their feet were resting on something soft (straw or carpet); at other times when they place their feet on the ground they fancy they are standing on elastic bodies or springs, and this gives them a prancing gait when walking; sometimes they feel as if they were pushed forward by some invisible force; they are also less sure of foot, and are afraid of falling when they go a little quick or go down-stairs. From this time the instinctive movements of the legs become more and more disorderly, especially does it become more and more difficult to keep time when walking. They cannot in fact walk without throwing the feet before them in a foolish fashion, and without striking the ground forcibly with the heels. These movements are sometimes so strong and sudden that the whole body vibrates and they lose their balance at each step; this is why they fear to walk without the support of an arm. These troubles continue to get worse till walking and standing become almost impossible. They are obliged to be carried, and if they attempt to walk a few steps their limbs are

* I am sorry to have neglected to mention in my first paper the fits of pain in the belly from which the patient suffered in 1857, which lasted two or three days, and recurred at considerable intervals in a remittent fashion. The patient had mentioned them in the communication made to me in 1856, and given above. I then considered them an intercurrent complication. I shall return to this "epiphenomenon" of locomotor ataxy.

violently moved in a strange and uncontrollable way, and they are soon exhausted by their efforts and ask to be led to a chair. They pass their lives either in a chair or in bed. These movements cannot be controlled by the eye, no matter whether or no the patient has double vision. The attitude of an ataxic patient in the second stage, with the head held forward and the eyes fixed on the ground or his feet, while he feels for some support, no matter how slight, with his hands, is very characteristic, and enables the disease to be recognised at a distance. When the muscles of the trunk are attacked the patients cannot remain sitting without being agitated by shocks sometimes violent enough to throw them on the floor. These disappear if the trunk is supported and the vertebral muscles are in a state of repose. Then comes a time, when the patient is sinking, when he can neither stand nor even remain sitting, but it is still possible to demonstrate the want of co-ordination in the legs by making him raise them alternately while he is lying on his back. One then sees that he cannot prevent them from swaying to one side.

B. The spreading of these troubles to the arms is almost always ushered in by numbness and creeping in the little and ring fingers. Only once have I seen numbness in the fingers supplied by the median nerve. The numbness of the fingers is soon followed by a weakening of their sensibility. These sensory troubles, which at first only exist on one side, soon invade the opposite side and spread eventually to all the fingers, always remaining most marked in the ring and little fingers, or in the "median" fingers if the numbness began originally in these latter.

With the enfeebled sense of touch there appears an inco-ordination of the movements of the fingers and phalanges. They are most peculiar, and I will try to explain them. There is a clumsiness in using the hand, and soon its functions are lost. All movements of the upper limbs are affected. I have seen one, for example, who had a difficulty in raising his glass to his mouth. His movements were very curious. He held his glass firmly, but he could not raise it to his lips without performing a series of zig-zags, without shocks or tremor; but to do this he was obliged to watch it constantly and throw his whole will into the effort. The least inattention caused him to upset the glass, or

perhaps by a sudden movement of the arm to throw the liquid in his face.

2. Apparent Weakness, contrasting with the latent integrity of Muscular Power. — The patient, whom the slightest blow would upset; who can neither stand nor walk without support or the assistance of an arm; who lets fall things which he holds in his hands, and who can scarcely use his upper limbs; who during locomotion struggles wearily and in vain against his powerlessness, such a patient, one can readily conceive, imagines himself to be smitten with paralysis. Such has been the opinion of physicians consulted by my patients; such was equally my own opinion before making my last researches. It will be understood also how surprised one must be when, by measuring the force of partial movements, the fact is established that the strength is considerable.

To examine the force of partial movements, the patient must be sitting or lying down; and for testing the lower limbs it is of advantage to let the patient remain horizontal. I cause those movements which I wish to study to be made and then, when the muscles producing them are shortened and held strongly contracted by the patient, I pull on the extremity of the limb in an opposite direction. Thus, for example, if I want to measure the power of the extensors of the leg, the patient lying on the opposite side and the thigh being held steady, I exert force on the lower end of the leg so as to cause *flexion* of the knee while the patient is attempting to *extend* it.

Many years ago I devised a medical strength-gauge (dynamometer) on the principle of the steelyard. It is of small size, and has enabled me to measure force from 1 to 100 kilogrammes. This instrument gives also the power of extension put forth by a patient during a trial. During such experiments the patient exerts in general a good deal of force without showing fatigue, although while standing or walking he soon becomes exhausted. In the latter case he is using great and vain efforts to perform functions with regularity, and the nerve-centres doubtless give out an amount of force which they cannot, in a short space, repair, and the patient evinces a frequent need of repose.

[To test the force of flexion of the hand, the dynamometer must be held in the palm and the two handles (B, fig. 1) forcibly squeezed together; this causes the two limbs (C) to separate, and

the consequent movement of the index from left to right shows the power exerted in kilogrammes (from 1 to 100). If the handles (B) be not crossed as in fig. 1, but be arranged as in fig. 2 (which can be effected by slipping them down to the round ends of the limbs and simply turning them round as in fig. 3), the dynamometer may be used for testing other partial movements. Thus to test the force of flexion of the elbow, attach a

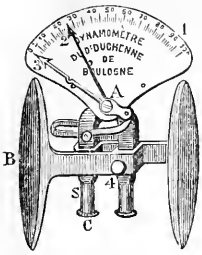

Fig. 1.

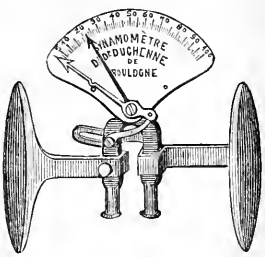

Fig. 2.

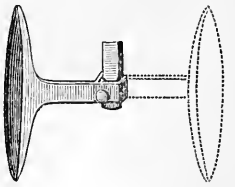
Fig. 3.

band, such as a folded handkerchief or a strap, above the wrist and fasten it to the handle of the dynamometer, then, while the patient strives his utmost to *flex* the elbow, seize the free handle of the dynamometer and pull upon it until extension of the elbow is produced. The needle will indicate the force exerted.

A more sensitive instrument with longer limbs (fig. 4) is used for testing (in grammes) the power of paretic patients. In using these dynamometers care must be taken not to jerk them lest their delicacy become impaired.]

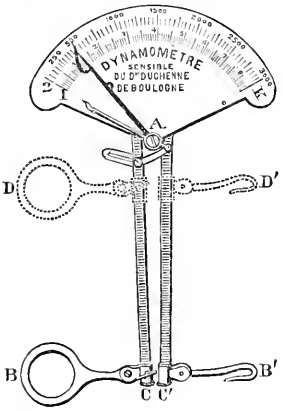

Fig. 4.

The index of my dynamometer is intended also to show the amount of nervous incitability (*incitabilité nerveuse*). Some persons having attained their maximum in a partial movement, can maintain it by continuing their effort for a greater or less time. Others again can only maintain their effort for a very short time after reaching their maximum. I call this *the exhaustion of nervous incitability*. Thus, of ten persons measuring their power of grasp and using my instrument to mark the maximum, some

can keep the index at their maximum for many minutes and others for a few seconds only. Some patients who can register as much as 40 kilos allow the needle to return instantaneously to zero notwithstanding all their efforts to prevent it, and they need many minutes (as much as ten) of rest in order to recover their normal power. These patients become, in a sense, paralysed, for in walking they are obliged (so to speak) to rest between every step; and sometimes even they become exhausted when standing. Might one not compare this state of the system to a battery which rapidly polarises when the circuit is closed?

3. Loss of the Sense of Touch and Pain.—I have often established the fact of the existence of troubles of co-ordination before the sensibility of the skin or muscles becomes affected. The sensibility of the feet and hands, especially on the soles and palms as I have before remarked, is in general more or less diminished as soon as the trouble appears. It is of importance to carefully study this anæsthesia and to determine as accurately as possible what part it plays in the disorders of locomotion. [I shall deal with the subject under the head of Diagnosis.]

Sensibility (both for touch and pain) is lost or weakened in different degrees in the skin, muscles, and joints. The senses of touch and pain are sometimes lost simultaneously in the skin, but usually the sense of pain remains intact or is little impaired. Appreciation of temperature is affected last. I have met persons in whom this was normal, although the sense of touch and pain was absolutely lost.

I must not forget to say that often enough it happens that sensations, artificially produced, travel slowly from the lower extremities to the *sensorium commune*. I have counted two or three seconds between the excitation and the perception; I have even seen one patient who did not perceive the pain of a pinch or of faradisation till nine or ten seconds after its infliction.

The loss of the sense of touch and pain of the plantar and palmar surfaces usually spreads from the foot to the thigh, and from the hand to the arm, and sometimes to other regions of the body, but in a less degree.

4. Pains.—[The pains of locomotor ataxy are characteristic, and may be an aid to diagnosis in the early stages of the disease.] They

are usually sudden and alarming, like the sensation of a tolerably big stabbing instrument driven into the tissues with a screw movement. Nearly all patients give this description. Sometimes it is a far-spreading shooting pain. These pains are limited to definite spots. The skin is usually too sensitive at this point during the continuance of the pain only, over an extent of from 1 to 4 centimetres. Light friction is strongly felt, but firm pressure relieves the suffering of the patient.

These pains usually come on in fits which last from a few minutes to 12, 24, or 48 hours or longer. Each painful sensation is very short, sometimes quick as lightning, or an electric discharge, or the blow of a hammer; at others lasting for one, two or three seconds, and returning at intervals of a few seconds, or minutes. They usually wander, travelling from one region to another; but for half-an-hour or some hours at a time, and sometimes during the whole period of each fit, they remain fixed to one spot. These fits are terrible, making the patient cry out and contract his features, when, after a short period of quiet, they take him by surprise, as always is the case. To these pains others are added, which continue during the whole time of the fit, either cramps in the calves, or pains affecting a large area of the trunk, or the temples, or nape of the neck. The patients sometimes say that a part of their body is as if held in a vice or squeezed in a ring, or the chest feels as if compressed in a sort of cuirass. One can easily understand how, under the excitement of these terrible and often obstinate pains, they express themselves in an exaggerated manner, and depict their sufferings in colours which I need not employ. I have, I believe, described truthfully enough those principal pains of which patients rarely fail to complain and almost in the same words.

These painful fits are subject to weather, and are brought on by bad weather, and tell like a weather-glass the change from dry weather to rain or wind, or *vice versâ*. Sometimes they come on without any known cause, and sometimes also they get worse during the evening or night.

These pains almost always appear at the onset of the disease, alone, or accompanied or preceded by other important first signs, with which we shall deal anon; weak at first, they grow worse, and torture the patients. [Duchenne has never known these pains to be entirely absent in cases of locomotor ataxy. They

may be only present at the beginning of the disease, and the patient may fail to connect them with his locomotor troubles, or they may be much less intense than usually is the case; but, if inquired for, a history of pain, more or less characteristic, will always be elicited.]

5. Paralysis of the Eye. Amaurosis.—Paralysis of one of the motor nerves of the eye is usually one of the first signs of progressive locomotor ataxy. It was only wanting in three out of the first twenty cases which I collected. I noted in these cases that the sixth was more often affected than the third, though now, having seen some hundreds of cases, I should say that the third is more often attacked at the onset than the sixth. Three times I noticed a double paralysis of the sixth, but in all the other cases the paralyses, whether of the sixth or third, were on one side only. These paralyses may improve or get well without any arrest or improvement of the disease in other respects. [See Case 1, p. 3.]

Seeing then that these paralyses are often merely temporary, careful inquiries must be made for them while getting the history of the patient, who often does not trouble to allude to them. One must bear in mind that double vision may be wanting even when the outward movement of the eye is incomplete, and neither of the eyes is blind. I have noticed this when the ocular paralysis has been double. I have now under my care two ataxic patients suffering from double paralysis of the sixth, neither of whom have ever seen double. In one of these, the paralysis is slightly marked, and I only recognised its existence by noting that when she turned her eyes outwards alternately on both sides, the edge of the cornea remained separated by 1 or 2 millimetres from the external angle of the eye.

Eye-palsy, as a first sign in conjunction with other symptoms of locomotor ataxy, has often thrown light upon obscure cases. I shall return to this symptom when discussing diagnosis and prognosis.

Amblyopia, Amaurosis.—Weak sight, and later complete blindness through wasting of the optic papilla, often complicate in different degrees the eye-palsy of locomotor ataxy; such at least is my experience.

The examination of the fundus oculi with the ophthalmoscope

in cases where the sight is much weakened or completely lost, has taught me that the optic papilla is wasted, showing, as one knows, a lesion (sclerosis) of the optic nerve at its origin or in its length. I reported a remarkable case of this in my last edition, and gave a picture of the fundus oculi. I did not conclude from it, however, that the blindness of ataxic patients was always caused by the same lesion, for up to that time I had not made any ophthalmoscopic observations on the greater part of my ataxic patients suffering from dim sight. I foresaw, however, that this wasting of the papilla must be frequent, if not constant. It became necessary then to examine the fundus oculi of ataxic patients as soon as a slight dimness of sight began to show itself, in order to study the manner of development of this lesion, and the possible congestive or irritative appearances which precede it. My surmises have since proved correct. Further, having never neglected since then to examine the fundus oculi in ataxic patients with dim sight, I have always noted varying degrees of whiteness of the papilla, with a wasting of the retinal vessels, directly proportionate to the dimness of sight. In ordinary squint, as one knows, dim sight does not exist, and by making the patient use one eye only, the sight becomes perfectly clear. I have met with cases in which while the squint diminished, the sight got gradually more dim. Nevertheless, the squint and the dimness usually progress *pari passu*. Complete blindness may, however, exist without squint at the very outset of the disease. Dim sight affects usually one eye only, and when there is squint this affects also the same eye ; but too often, indeed, both eyes are affected, and too often also, in spite of all treatment, sight is completely lost before the locomotor troubles have appeared, or have become generalised. Nothing can be more pitiable than the condition of these poor blind patients when the ataxy has reached its last stage. With their legs already deprived of the power of orderly movement ; unable to appreciate the resistance of the ground ; without the power of correcting their movements by the eye ; standing or walking become to them more and more difficult and painful, and accompanied by most irregular actions. Finally, when the sense of touch in the feet is completely lost, they are unable to walk at all, although still possessed of considerable muscular power.

I have often noticed in the first stage of locomotor ataxy an

appearance of the pupil which is mentioned in some of my cases, but the importance of which I have not brought into sufficient relief. It will be understood that I do not allude to dilatation of the pupil, which is only a consequence of the wasting of the optic papilla, or significant of paralysis of the third nerve.

I allude to *contraction* of the pupil, which in these cases is significant, as I have shown, of paralysis of the sympathetic. At the beginning of my investigations I had noted contraction of the pupil, usually on one side, sometimes on both. I have seen it in the first stage, and in fact, when the patients have been able to give me any information on the point, they have assured me that they noticed it before their motor power was affected. This contraction does not seem to cause any trouble of the sight. I have met with extreme degrees of it in ataxic patients, whose optic papilla was entirely wasted, in whom the pupil ought to have been on the contrary widely dilated, but in whom dilatation was only brought about with extreme difficulty and by the aid of strong doses of belladonna.

What was the cause and the mechanism of this contraction of the pupil? The following case, of which I give only a *résumé*, has afforded me some idea of it.

Case No. 2.—In 1860 I noticed in a patient, who had been ataxic for four years, a contraction of the pupils, accompanied on the right side by redness, heat, and pain of the conjunctiva, the skin of the eyelids, and of the temple. The patient was blind, and I had much trouble to dilate the pupils sufficiently, by means of atropine drops, to examine the optic papilla, which I found almost absolutely white, especially the right. Having been present during one of his fits of pain, which were very severe, I was astounded to see during it that the pupils dilated considerably, and that at the same time the redness of the conjunctiva and eyelids disappeared. When the pain passed off, the contraction of the pupils and the redness of the skin and conjunctiva reappeared. I observed these same phenomena many times in this patient. I was struck in this case by the resemblance which existed between these phenomena and those obtained by dividing the sympathetic nerve in the neck, and exciting the proximal end of it. (It will be remembered that division of the nerve causes a contraction of the pupil, and the electric excitation of the cut end causes it to dilate.) I concluded from this that the contraction

of the pupil seen in locomotor ataxy was caused by a slight degree of paralysis of the cervical sympathetic or of the superior cervical ganglion, a pathological condition which, in the case recorded, had probably caused that complete series of phenomena which characterise the maximum degree of this paralysis. (Pupillary atresia, neuro-paralytic hyperæmia of the conjunctiva and the skin of the eyelids and temple.)

This idea has been confirmed, as will be shown further on, by anatomical investigations.*

6. Other Paralyses or Local Troubles. (Epiphenomena of Progressive Locomotor Ataxy.)—I have, till now, been describing the main features of progressive locomotor ataxy, such as by their individual and collective characteristics make of it a distinct morbid species.

Those of which I have now to speak are, by comparison, less important, and although it would be of great interest to discuss in detail the cases in which I have met with them, space compels me to mention them merely.

Paralyses of the fifth, sixth, seventh, and eighth Nerves.—Other cranial nerves, in addition to those of the eye, may be affected in progressive locomotor ataxy, but such instances have been rare in my experience. Twice have I seen paralysis of the fifth co-existing with paralysis of the third. Once these paralyses were on the same side, and in the other case the paralysis of the *fifth* was bi-lateral, while that of the *third* was limited to the left side only. In this latter case also the paralysis spread to the soft palate and larynx. Once I have seen a paralysis of the seventh on the same side as the paralysis of the third.

In all my ataxic patients the sense of *smell* has been unaffected. Once only was *hearing* affected on the same side as the squint.

Generative Function.—The sexual power in man sooner or later has manifested considerable change: *once* it was increased; in all the others it was weakened or abolished. I saw one case with M. Trousseau in which it remained normal, although the ataxy had become general.

I have sufficiently often seen impotence (anaphrodisia) occur

* This case was reported, with details, in a paper communicated in February, 1864, to the Société de Médecine of Paris—"*Recherches Cliniques sur l'état du grand sympathique dans l'Ataxie locomotrice* (Gazette Hebdomadaire de Médecine, 1864, 2e Série, p. 116).

before the loss of co-ordination, to regard it now as one of the symptoms of the first period. Many times it has been the first sign of the disease; most often it has appeared after the lightning pains, and sometimes long before the loss of co-ordination. I have met many ataxic patients who, having suffered from lightning and boring pains before marriage, were scarcely able to get through their honeymoon, for sooner or later ataxic patients are usually stricken with impotence, and I have always advised those who have suffered from the special pains of ataxy not to marry. Sometimes complete impotence comes on suddenly. Occasionally it is preceded by sexual excitement and painful Priapism occurring during sleep.

Difficulties in Defecation and Micturition.—The expulsion of fæces and urine is sometimes difficult or impossible, from varying degrees of paralysis of the rectum or painful spasm of the sphincters of the rectum and bladder; sometimes, on the contrary, the patients cannot retain their fæces or urine from paralysis of the sphincters. In one case, nevertheless, the patient only suffered from slight constipation, although in an advanced stage of the disease.

With the assistance of M. Mialhe I have in most cases analysed the urine, but in no case have we found either albumen or sugar.

Sometimes the patients have had, at the outset, a paralysis of the bladder, accompanied, as a rule, by vesical catarrh, and I might, under the influence of preconceived notions, have considered the troubles of locomotion as secondary to the urinary troubles (by reflex action), and especially when, as in many cases, the ordinary signs of the first stage were wanting or slow to appear. Sooner or later, however, the disease became general and declared itself, and it became impossible not to regard it as a case of locomotor ataxy. The functional troubles of the urinary organs were only symptomatic of the central lesion.

Other Gastric and Visceral Troubles.—Vomiting has never marked the onset of locomotor ataxy, as is the case in cerebellar apoplexy, and cases of cerebellar tumour. Stomach troubles, or pain in the stomach or bowels, have occurred at times in the course of the disease without recognisable cause.

From the first I have noticed giddiness and dizziness, which

is caused or aggravated by throwing the head backwards. But I have never noticed in uncomplicated cases either hesitation of speech, trembling of the lips or tongue, or exalted delirium. On the contrary, I have been struck by the integrity of the intellectual powers, which, like that of the muscular force, is preserved to the end.

The electro-muscular contractility is always unchanged, and the texture of the muscles is not altered.

Lastly, there is no fever.

*Additional Remarks on some of the Epiphenomena of Locomotor Ataxy.**

When I undertook to write a description of progressive locomotor ataxy, I was confronted with such numerous symptoms and functional troubles, so varied in their intensity, and some of them so often irregular in their appearance, that I lost all hope of performing my task with clearness. Therefore, in order to be as concise as possible, I united into a primary group, as has been seen, those symptoms which, either singly or by their order of succession or concurrence, appeared to me to be the characteristic signs of this disease. The other functional troubles, which can scarcely be considered characteristic, I formed into a second group under the name of *Epiphenomena* of Locomotor Ataxy.

I feel satisfied thus to have separated the symptoms from the functional troubles, for the epiphenomena which form the second group could not indicate locomotor ataxy, although they increase its gravity and add to the sufferings endured by the patient. As the number of them has very greatly increased, owing to the great quantity of collected cases, they might possibly turn the attention of the observer away from the symptoms of the first group, which should form the true ground of his diagnosis, and of which he should never lose sight.

I must now mention those epiphenomena which I have myself observed, many of which have been discovered by other pathologists. These are pains in the bowels, neuralgia, neuroses of the respiratory tract, lesions of nutrition in the joints (arthropathies), muscles, and skin (zona).

* These remarks form a footnote in the original.

Pains in the Bowels (visceralgiæ).—I have already said that digestion is normal. The only trouble is an obstinate constipation, which comes on at a certain period of the disease, accompanied by difficulty in micturition, and which is soon followed by paralysis of the rectum and bladder. I have, however, observed in one case peculiar attacks in the belly which came on suddenly without obvious cause, and disappeared in the same way, after having lasted from a few hours to two or three days. They recurred at irregular and considerable intervals (of many weeks or months) and consisted of belly-ache and swelling of the epigastrium, accompanied sometimes by vomiting. The pains were terrible and continuous, but with paroxysms. Having only seen this in one case at the time of my first paper, I hesitated to rank it with the nervous troubles caused by locomotor ataxy. I nevertheless took note of it, and said that "there sometimes occur in the course of this disease certain troubles of pain or otherwise in the belly" (*certains troubles gastralgiques ou gastriques*) without, however, describing them. But having met them since then in many cases, I must now enter them definitely among the symptoms of locomotor ataxy, and place them among the epiphenomena.

I have known these pains seated in the bowels, and sometimes accompanied by true dysentery and bloody stools; I have elsewhere recorded a case of this. When the fits of pain in the bowels are accompanied by constipation they are distinguished from lead colic by the distention of the belly. Finally, I have known these pains to spread from the stomach to the belly; they are continuous, as I have said, and become at times terribly aggravated, making the patient cry out. Over-sensitiveness of the skin of the belly makes them more intolerable.

During the fits of belly-ache the patients appear to be in a critical state; but suddenly they vanish, and all functions are normally performed as before. The sudden disappearance and the intermittent return of these fits must be looked upon as probable signs of ataxy.

Neuralgiæ.—It is not unusual to meet with neuralgic pains, also remittent, and lasting from a few hours to a few days, continuous while they last, with sharper fits, and causing horrible agony. I have noticed them in many regions, *e.g.*, the intercostal nerves, sciatic nerves, and spinal nerves. I have often

met among my patients with a transient intermittent neuralgia of the spermatic cord and of the bladder, with spasm of its neck.

Neuroses of the Air Passages.—I have met with cases of spasm of the glottis among my patients causing hoarseness, lasting for a few days only, and returning at greater or less intervals. I once saw a spasm of the glottis so complete and enduring as to threaten life. I have also seen nervous coughs, obstinate and without fever, and sometimes accompanied by a sort of bronchial flow of short duration, and returning at intervals.

Arthropathies.—The discovery of this unexpected lesion is due to M. Charcot, who made it the subject of several interesting articles (*Archiv. de Phys.*, Jan., Feb., 1868, p. 162). This rare affection is neither preceded nor accompanied by pain, heat, redness, or fever. It shows itself only by a swelling of the joint and its neighbourhood, and it produces a serious change in the surfaces and ligaments of the joints. M. Charcot, after a microscopic investigation, considers it to be caused by an atrophy of the cells of the anterior cornua which are in connection with the damaged joint.

Muscular Wasting by the Spreading of the Irritation to the Anterior Cornua of the Cord.—This is a kind of muscular atrophy which sometimes develops in the course of locomotor ataxy, and which must not be confounded with progressive muscular atrophy. The discovery of this complication and its cause is due to M. Charcot. I shall return to this subject when I have shown what is the state of our knowledge of the pathological anatomy of locomotor ataxy.

The Progress of the Disease. — While studying the symptoms of locomotor ataxy we saw how some of them, individually, were characteristic of the disease; but that which makes of it a distinct *morbid species* is the regularity with which the symptoms in general appear and develop—their relationship, their course and progress. This is a capital proposition, depending upon numerous clinical observations, and the truth of which I can prove in an incontestable manner.

In spite of the care which I have taken to separate the symptoms of locomotor ataxy, in order the better to analyse them and learn their individual value, there can be no one who, on reading them, would not notice that the disease, which is always localised

at first, is essentially an encroaching disease, and one which tends to become general.

The division of locomotor ataxy into three distinct stages naturally follows from a study of the symptoms. *The first stage* is characterised by eye-palsy, dim sight, and wandering, lightning, boring pains; *the second stage* by a staggering gait and occasional ataxy of the upper limbs, soon followed by failure of sensibility in the muscles, joints, bones, and skin; and *the third stage* by the generalisation of the disease. The functional troubles of the bladder, rectum, and generative organs are only epiphenomena. The first stage may develop irregularly, or be wholly or partly wanting. Nevertheless the disease pursues its course, and the symptoms of the first stage appear sooner or later in the following stages.

I have said that this change of order of the stages is exceptional, for at the time of publishing my first memoir I had only two examples of it. Clinical observation compels me now to add that these exceptions are less rare than I then thought. I had only once known the eye-palsy and dim sight to be absent, the invasion of the disease being marked by the characteristic pains. Since then, however, I have observed four similar cases with Professor Trousseau.

I will add that, of all the signs of the first stage, the special pains of the limbs is that most rarely wanting. When I published my memoir I had only once known locomotor ataxy to start with want of co-ordination in the legs.

Duration.—It is difficult to determine the exact duration of locomotor ataxy, because, before the complete development of the disease, the patients fall rapidly into a state of nervous exhaustion, which prevents them from resisting any morbid influences which may surround them. They usually succumb to intercurrent diseases. The facts already given show that locomotor ataxy is of long duration, for the first stage alone, that of squint and pains, may last, as we have seen, for a dozen years.

[A case is given of a lady who was seized with the pains of ataxy in 1836. Vision became impaired in 1851, the staggering gait began in 1856, and after becoming completely blind and losing all power of movement, she died in 1861 from the exhaustion caused by her incessant agony.]

I know other patients, who, after twenty years, continue to eat and digest well and seem likely to live a long time yet. From these facts it results that locomotor ataxy is in general of long duration, although I have seen many cases of a rare, almost acute form which reaches its third stage in six months *or a year.*

Prognosis.—Can locomotor ataxy get better? I hasten to state that on this point I have no doubt, and I have not used the word "*progressive*" in the false and fatal sense attached to it by Requin. The word "*progressive*" is used, usually, in pathology to convey the idea that the disease has a tendency to become general and to invade *seriatim* a greater or less number of organs. No matter how hopeful one may be, it is not possible to be deceived with regard to the *prognosis* of locomotor ataxy, which is always of the utmost gravity. The truth of this is, alas! too evident in the sad histories I have given.

Diagnosis.—It is needless to say that not every case of squint occurring in an adult is the precursor of locomotor ataxy, but it would be, says Duchenne, of interest to seek out those patients who have suffered from transient squint which has apparently yielded to treatment, and determine how many of them have become later on definitely ataxic. When to squint is added dim vision, and when to these are joined erratic pains, our suspicions of ataxy should be strongly aroused, but even this conjunction of symptoms does not constitute a certain sign. The diagnosis becomes certain directly we detect the inharmonious action of the lower limbs. A case is recorded (Obs. cix. p. 642) of a young man aged 28 who sought Duchenne's advice on account of impotence. On making further investigation he was found to suffer from double vision, and within a very little time he began to stagger as he walked, and this was followed by the characteristic pains of ataxy. *Apropos* of this case Duchenne insists on the importance of making a diagnosis of ataxy as early as possible in the course of the disease, and thus summarises his remarks on this head.

1. The paralyses of the muscles of the eye which appear in the first stage of locomotor ataxy are either permanent or temporary, and even intermittent or remittent, and last several months. I have not met with these kinds of *repeated paralyses*

of the motor nerves of the eye except in locomotor ataxy, and they must therefore be considered as signs of great value in this disease.

2. The pains of locomotor ataxy (like lightning, stabbing, boring, with over-sensitiveness of the skin of short duration and limited to the painful spot) are characteristic, and have no resemblance to the pains of gout, rheumatism, neuralgia, or nocturnal bone-pains.

3. I have often seen anaphrodisia appear at the commencement of locomotor ataxy. If then it comes on, without appreciable cause, in an individual in whom formerly the sexual power was normal it ought to be considered as a probable sign of the onset of this disease.

Locomotor ataxy may be confounded with *paralysis of the muscular (and articular) sense*, but we shall defer making remarks on this head until we come to deal with the latter disease.

It is possible that progressive locomotor ataxy may be confounded with *general paralysis of the insane*, but only in those cases in which the muscular troubles and the peculiarity of gait take an exceptional precedence over the concomitant symptoms.

In spite of the similarity Duchenne says it is easy to distinguish between the movements of the two diseases. "In *general paralysis*, contrary to what is seen in *locomotor ataxy*, the patients always retain the instinctive knowledge of muscular combinations, by virtue of which they execute perfectly the special movements of the different periods of walking, although they in some measure exaggerate them; they have no fears for their balance when standing or walking, and do not constantly look at their feet or search for a support. They only cease walking when they become paralysed and completely helpless."

Squint and dim sight are not characteristic of general paralysis of the insane, although inequality of the pupils is common to general paralysis and locomotor ataxy.

It is scarcely possible, with ordinary care, to confound locomotor ataxy with ordinary paraplegia or with acute spinal paralysis.

Progressive muscular atrophy also is easily distinguished from locomotor ataxy, for in the latter disease muscular wasting and deformity of the limbs do not occur.

It is not necessary seriously to discuss the diagnosis of

locomotor ataxy from tremor due to alcohol, mercury, paralysis agitans, or ordinary chorea.

With regard to the diagnosis from the sclerosis *en plaques* of M. Charcot (a disease which Duchenne speaks of as choreiform paralysis) he says, "Whoever has compared two patients suffering from these diseases will not be able to confound the one with the other." The sufferer from sclerosis *en plaques* does not when standing sway to and fro like the ataxic patient, but is shaken by sharp shocks. When he walks he seems to reel as if he were jerked out of the straight line by clonic contractions, and does not constantly look at his feet like ataxic patients. Further, in sclerosis *en plaques* there is a peculiar "scanning" manner of speech and a certain amount of muscular paresis which are not found in locomotor ataxy, while the symptoms peculiar to the first period of ataxy are absent.

"In an advanced stage of locomotor ataxy contractures of the muscles which move the foot on the leg sometimes occur. This is caused by an extension of the pathological lesions from the posterior to the lateral columns of the spinal cord. In the same way the disease which has been lately described as a fasciculated sclerosis of the lateral columns may reach the posterior columns, and then to the symptoms of this disease are added the pains which are characteristic of locomotor ataxy."

"An error in diagnosis which I one day made in a patient apparently the subject of 'writer's cramp' may prove instructive. The slightest attention would have prevented the error made at first in this case. In fact, 1. In writer's cramp the difficulty in writing is the only trouble and the other muscular functions remain intact. 2. There is no resemblance between the morbid muscular phenomena of writer's cramp and those which characterise locomotor ataxy. In the former case there is a localised spasm (nearly always the same) in such or such muscles of the fingers, hand, forearm, or even shoulder. In the second case there is disordered movement without contractions during every kind of voluntary movement of the hand.

"In this patient I was so preoccupied with the writing difficulty, of which alone he complained, that it was only, as it were, by accident that I noticed his ocular paralysis, and elicited a history of double vision and occasional difficulty in walking."

Progressive locomotor ataxy may be complicated by other

muscular troubles. Duchenne gives two interesting cases in which well-marked ataxy of the legs was complicated with equally well-marked progressive muscular atrophy in the upper limbs. In one of these (Obs. cxi.) it is mentioned that a few weeks after marriage the patient was suddenly smitten with impotence.

Another case is given in which general paralysis of the insane was combined with locomotor ataxy, and mention is also made of the combination of locomotor ataxy with hemiplegia (of cerebral origin) and with paraplegia (of "hysterical" origin). "These cases of exceptional coincidences give rise from time to time to bad diagnoses and to strange descriptions, and by complicating the clearest and best understood points in pathology serve to arrest its progress."]

Causes.—Ataxy has generally commenced in my cases between the ages of 18 and 40. In three cases the patients were women. I will not conclude from this that locomotor ataxy is a disease of adults, and especially attacks men, because I know that these facts must be confirmed by time and by numerous observations. Nevertheless, I may add, in order to give greater value to the conclusions which we may logically deduce from my observations, that the cases which I have observed, but which I have not put upon record, go to strengthen the conclusions drawn from the first series. (Have I not elsewhere already said that I have collected only the clinical facts of the last few years, because I knew better how to diagnose them? But now I can recall to mind how the previous cases of locomotor ataxy which I have seen were exactly similar to those which I have been describing.)

Nothing seems to me more difficult than to determine the causes of locomotor ataxy. Certainty on such a point is impossible.

The following are the principal causes to which one might attribute locomotor ataxy.

In a young girl, aged 18, unrestrained masturbation appeared to be the cause; but this is the only case of the kind which I have seen, unless one regards as an ordinary cause of locomotor ataxy those sexual excesses to which the majority of mankind are addicted. If it were so, how common the disease would be! Some patients make mention of sudden or prolonged chills, and the checking of perspiration. A sportsman, *e.g.*, has waded too

long in a marsh, another has sat too long in a cold hip-bath, a third (an ice-maker) has checked his perspiration while making ices. Can one conclude from this that ataxy is caused by chills or rheumatic influences? Other alleged causes are equally doubtful. Some of my patients had suffered from syphilis, but in the absence of any special symptoms pointing to syphilis one could not feel certain as to the relationship of the two diseases. Sometimes, it is true, patients have suffered from increase of pain at night, or suffered chiefly at night, but this is observed often enough when no syphilitic cause exists. The result of treatment would, ordinarily, in such cases, serve as a touchstone; but, alas! in my cases specific treatment has had no effect upon the course of the disease.

In short, the different causes which, in my cases, appear to have exercised some influence over locomotor ataxy have so little in common that they appear to me to throw no light on the causation of the disease.

Trousseau has with reason attributed an important part in the causation of ataxy to certain *diseased nervous conditions.* Thus he has often remarked among the antecedents of ataxics certain singular neuroses. There has been a history of bed-wetting in their own infancy, or among their children, or there has been a history of epilepsy, or many members of their family were also ataxic (I have seen, in consultation with Trousseau, three ataxic brothers). Researches made since 1861 show that moral causes have a considerable influence on the development of locomotor ataxy. In a great number of patients this disease has appeared some months or a year after the death of some dearly loved friend, or after money losses, or after a social or political revolution. Those who have just suffered (1871) so cruelly in our unhappy country afford me every day numerous and fresh proofs of this.

Pathological Anatomy.—Do the symptoms of the different stages of locomotor ataxy indicate damage to some point of the nervous centres? I am not yet in a position to deal completely with this subject. . . . . I may say that when I was making my earliest investigations I was present at the post-mortem examination of a patient who had suffered from this disease for two years. The following is a *résumé* of the facts.

Case 3.—Demay Victor, artist, aged 28, in the third stage

of locomotor ataxy (ocular paralysis, characteristic pain, almost total inability to walk). He had suffered for two years, had had syphilis, and had been treated with iodide of mercury. *The brain and spinal cord, examined with the greatest care by the naked eye, did not show any appreciable anatomical lesion.*

At that time (1858) the loss of co-ordinating power in locomotor ataxy was attributed to cerebellar disease. . . . The pathological anatomy of locomotor ataxy is well understood now. It consists of a lesion of the posterior columns of the cord, localised at first in the tract nearest the posterior edge of the commissure and situated in the connective tissue, whence the mischief spreads in the very advanced stages of the disease either to the antero-lateral columns or to the cells of the anterior cornua (called *motor*, and which one ought also to call *trophic*). This change is recognisable to the naked eye by a greyish gelatinous appearance of the damaged spot. Microscopic examination shows (what is not visible to the naked eye) an increase of the connective tissue.

The structure of the altered points presents very different appearances according to the amount of increase of the nuclei of the network of connective tissue (neuroglia). The appearances differ also according to the degree of separation of the tubules of the nerves by the increased connective tissue. Photographs taken from sections show distinctly three degrees of this increase in posterior sclerosis, and the changes which are the consequences of it.

1. If one examines the circumference of a hardened patch, one sees that the net-work is made more evident by the growth of one or two rows of nuclei in the threads of the meshes.

2. This condition increases towards the centre of the lesion, forming a second zone, in which the nuclei surround the nervous tubules and separate them from each other. The resulting compression causes wasting and deformity of the tubules, or presses them into a mass of myeline, isolated and irregularly dotted throughout by one or more axis cylinders, which are often wasted but rarely overgrown. The new nuclei are ten times as big as at the circumference.

3. There are seen also in these points smooth slabs which have not the fleecy look of the mass of myeline. These are numerous and of variable size. This kind of change has been

well described by a Swedish pathologist, Axel Jäderholm, of Stockholm.

In a more advanced stage the new connective tissue (*tissu fibroïde*) is replaced by a true fibrillar tissue (*fibrillaire*). This is the fibrillous change of Formann and Charcot. Many amyloid bodies are found mixed with the nuclei. These have been looked upon as characteristic of sclerosis, but they are often completely wanting. This is due to the mode of preparation. Besides, they are met with in considerable numbers in conditions in which they cannot be attributed to any pathological state.

Enlarged capillaries are seen mingled with the foregoing changes.

The usual seat of these changes in the posterior columns is confined to the posterior fissure, to the points called the wedge-shaped columns of Goll. Sometimes the lesion is exactly limited to the course of the nervous tubules of the posterior roots, which, after entering the cord, traverse for a certain length the posterior columns under the name of internal root-bundles (the internal fibrous masses of the posterior roots of Köllicker). Fig. 5 represents a transverse section of the spinal cord in the lumbar region from a case of locomotor ataxy combined with muscular wasting, contractures, arthropathy, and tremor during voluntary movement. The patient was a woman, aged 60, under the care of M. Charcot at the Salpêtrière, and the case was published by M. Pierret in 1870 (*Archives de Phys. et de Pathol.*). It shows that not only the posterior columns, but also the lateral columns, and the outer part of the right anterior cornu has been invaded by the indurating process.

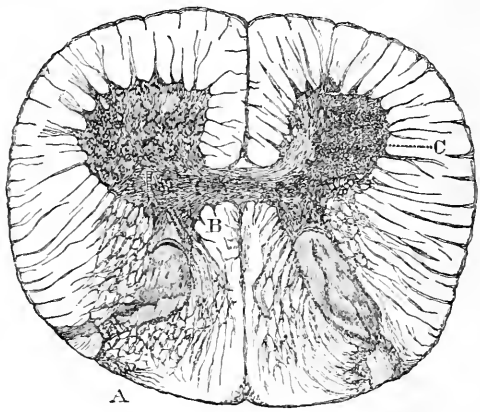

Fig. 5.

Pathological Physiology.

What is the relationship of the posterior sclerosis to the disorders of locomotion seen in this disease?

A. *Is the loss of co-ordinative power a consequence of the loss of sensibility?*

It is very necessary to analyse with care the facts of motor co-ordination. I did so in 1859, when I proposed a theory of co-ordination of voluntary movements. I tried to show: 1. That a co-ordinative faculty puts in action, during walking, muscular associations, impulsive, moderating and collateral (harmony of antagonists), and during standing the association of spinal muscles. 2. That its existence is proved by clinical facts. 3. That absence of touch and sight causes a certain trouble in the exercise of this faculty, but that nevertheless it is independent of them. My *résumé* of this subject was in the following words:—

. . . . . .

"I could not, like most physiologists, conceive that so complicated a mechanism could be put in action with such marvellous facility and precision without the help of a co-ordinating faculty; this hypothesis seems to me now to be a fact, because I have shown that a disease can destroy this faculty in part or in whole."

A talented physiologist, M. Jaccoud, has, however, formulated the following series of propositions as a "*Physiological Equation*":—

1. Motor co-ordination as a voluntary act is subordinate to the integrity of muscular and tactile sense.

2. Disorder of movement signifies loss of muscular sense.

3. If the disorder is complete all these conditions will be present, if incomplete some will be wanting.

This "physiological equation," so cleverly built up, is destined, alas! to be stillborn. I have had no hand in its premature end. This task belongs to daily clinical observation, which has certainly proved that the inco-ordination of movement peculiar to locomotor ataxy can exist equally in patients *who enjoy every kind of sensibility.* It will be shown further on that "muscular sense," to which M. Jaccoud attributes the chief part in motor co-ordination, is only a myth, notwithstanding its discovery by *Charles Bell.*

B. *The power of co-operating in motor co-ordination possibly belongs to certain nervous fibres in the posterior columns.*

If the facts exposed in this chapter have already proved that sensibility only *helps* in motor co-ordination, and that its abolition merely hinders the exercise of motion, it is none the less proved that the sclerosis of the posterior columns produces the dissociation of movements. It has indeed resulted from the clinical facts collected during several years by M. Charcot that the localisation of the irritative lesion (*travail irritatif*) in those points of the posterior columns corresponding to the internal root bundles (*faisceaux radiculaires internes*) is necessary for the manifestation of motor troubles in locomotor ataxy. I do not hesitate to say with M. Pierret, "these facts tend to show that the co-ordinating faculty is not indiscriminately assigned to every part of the posterior columns."

When this hypothesis shall be established, or shall rest on a greater number of facts; we shall still need to know the real cellular origin of the posterior internal root bundles.

One sees indeed how much obscurity still remains with regard to the pathological physiology of locomotor ataxy.

Pathogeny.—The pathogeny of this morbid species is as obscure as the pathological physiology. One knows that subacute congestion is shown by the presence of big vessels in the hardened patches. But what is the cause or mechanism of this congestion? Is it a diseased state of the local circulation? It follows from the study of the pathology of other diseases, that from similar causes different morbid species result indicative of damage to different points in the nervous system. To quote an example, why should conditions which destroy or exhaust nervous power (such as great trouble, or social and political disturbances) produce in one case *sclerosis of the posterior columns* (locomotor ataxy), in another *atrophy of the nuclei of the bulb* (glosso-labio-laryngeal paralysis), or a *disseminated sclerosis in patches* (chorei-form paralysis), or a *primary fasciculated sclerosis of the lateral columns* (paralytic contractures)? I could call to my aid in these cases, it is true, constitutional causes; but would the pathogeny of locomotor ataxy become more clear thereby?

Prognosis.—[Duchenne expresses a hope that when (as has rarely been the case) the diagnosis of locomotor ataxy is made

in its very earliest stage, the disease may not prove so utterly intractable to a rational treatment as has been the case hitherto.]

Treatment.—[Duchenne has known cases in which ocular paralyses have improved under faradisation, and others in which the pains have been lessened by its application.

Continuous currents have proved of no service in his hands.

Baths and douches seem always to ameliorate the patient's condition.

Iodide of potassium or mercury should be given whenever there is a distinct history of syphilis, and the former should be used even when there is no question of syphilis. Iodide of potassium is of no use in the later stages, but should be given in the very early conditions (of diplopia, strabismus, &c.) Duchenne himself gave iodides internally, and applied localised faradisation externally to relieve symptoms. "When indeed the local treatment triumphs over a symptom I do not slumber in a false security, and do not continue the internal treatment of the disease less persistently."

The motto of all who treat this disease should be "*Principiis obsta*," a precept which I think myself happy to have made easy of application by laying the foundations for a sure diagnosis of the early stages of this formidable disease.]

Some Historical Considerations.—Contrary to ordinary custom I have not prefaced my work on locomotor ataxy with any historical considerations, and I owe some explanation of it to my readers. When one wishes to devote oneself to the observation of the signs of disease and their nature independently of all external influence it is necessary, I think, to guard against filling one's mind with the work of others who have dealt with the matter in hand. This is a principle from which I have never deviated, always postponing till the end my bibliographical researches, and never mentioning the work of others until I have given my own. Had I done otherwise I should always, as it were, have been looking at my facts with another man's spectacles, and this must have hindered my own observations. In order to see facts *with my own eyes* I have often forced myself to forget the knowledge of medical literature which I possessed. It is by following this method of observation that I have been able to lay bare the facts of locomotor ataxy, and to arrange the

ideas which have come into my mind after much reflection and many years of research.

I am eager to acknowledge that the symptomatology of locomotor ataxy is not entirely new, although previous descriptions of it left much to be desired. In other authors, in fact, observations can be found which resemble those which form the basis of my own work. It could not be otherwise, for without doubt the disease is common enough and has always existed; but these observations are either incomplete; or resemble mine only in a few points; or are detailed in such a way that it is neither possible to recognise the different aspects of the disease of which I have made a morbid species, nor to diagnose them with certainty. Further, the clinical facts have sometimes been confounded with other troubles which are symptomatic of essentially different diseases.

Some authors have considered themselves justified in establishing different diseases on clinical facts similar to the preceding which they have collected. I will recall the most important of these.

Professor Romberg, of Berlin (*Lehrbuch der Nervenkrankheiten*, 1851), has described an affection, under the name of *tabes dorsalis*, which very nearly approaches locomotor ataxy. . . The patients observed by Romberg suffered from a disturbance of balance, but after reading the symptoms it would be difficult to say whether their trouble arose from a loss of sensibility or from a loss of the co-ordinative faculty. In addition he speaks of a loss of muscular power such as does not exist in true locomotor ataxy. Romberg reports among his cases that of a country doctor who, after exposure to weather, was struck with a *paralysis* of his lower limbs, and with dimness of vision in both eyes, ending in blindness. Sensibility was normal, and up to the time of his death the patient could distinguish different temperatures. At the lower end of the cord, which was diminished by a third, the medullary substance of the posterior roots had almost completely disappeared and had a greyish yellow tint.

Romberg relates other cases in which he proved that the grey matter of the cord was *softened*, so that *a part of the lumbar and dorsal regions were almost in a fluid state.*

These extensive lesions of the cord must have caused, during

life, proportionate troubles in muscular movements, weakness and loss of voluntary and electric contraction, wasting and loss of sensation; symptoms which characterise subacute spinal paralysis, and which can never be confounded with locomotor ataxy. Apart from this unfortunate confusion I am happy to admit that M. Romberg has well described and observed most of the symptoms which are met with in progressive locomotor ataxy.

[The "*dorsal consumption*" described by *Lallemand* is especially characterised by muscular weakness, and is clearly not to be confounded with ataxy. *Bouillaud* (*Traité de nosographie médicale*, Paris, 1846) alludes, in the class of diseases which he calls *ataxy of the nervous centres*, to certain motor troubles, different from convulsions and paralyses, and which consist of a *disorder*, an *inco-ordination*, an *ataxy of movement*. To this head he refers chorea and certain tremors and troubles in walking and standing seen in general paralysis. In many passages he speaks of *locomotor ataxy* as a symptom, which he was inclined to attribute to disease of the cerebellum. He had made no pathological observations, and did not attempt to describe the disease which I have called locomotor ataxy.

Sandras (*Traité pratique des maladies nerveuses*) has described a case which one would be inclined to call locomotor ataxy, because the patient suffered from the first and during the whole course of the disease from wandering pains, which he himself compared to lightning, and because "he afterwards lost his power of balance, and his movements, sudden, jerking, and disorderly, were not under the control of his will. Sense of touch was also diminished, so that the patient did not know whether he was in bed, between the sheets, or outside of them, or whether his trousers were on or off." The description is very confused, so that it is impossible to determine whether or no the muscular power was intact, and whether the troubles were due to loss of sensibility or to a failure of the co-ordinative faculty.

It is known that Charles Bell discovered the existence of a paralysis of muscular sense in 1822, and that therefrom he concluded the existence of a sixth sense (muscular sense), which was nothing but a condition of the general sensibility. In 1850 I described pathological facts analogous to those of Charles Bell.

In 1852 Landry, in ignorance of Bell's previous work,

described similar cases of "paralysis of muscular sensibility." "Unhappily this author," says Duchenne, "did not know how to distinguish functional troubles, arising from a want of feeling in the contracting muscle, from those which are caused by a lesion of the *psychic faculty* of motor co-ordination," and he has consequently mixed with his cases undoubted instances of locomotor ataxy. It is evident that up to the time of my own description locomotor ataxy had not been described as a distinct morbid species.]

It was necessary to give the disease a name, although I should have been happy to dispense with one. Not being able at this time to name it from its anatomical lesion, which, if it existed, had yet to be discovered, it was necessary to give a name which recalled the groups of leading symptoms. If, in accordance with modern usage, I had chosen a Greek name, it must have been composed of a number of words offensive alike to the tongue and the ear. Convinced that a name derived from symptoms is always bad or insufficient, since one cannot embody in it a complete definition, I was obliged to call the disease after one of its chief symptoms, viz., the progressive loss of the co-ordination of movement. The name "*progressive locomotor ataxy*" seemed to me to give the most exact idea of this kind of motor trouble.

In September, 1863, having already given my description of progressive locomotor ataxy in the *Archives générales de médecine*, without troubling myself about its history, my friend Professor Sée, well versed in German medical literature —which I admit I have too much neglected—told me that the symptoms of the disease, which I had just described, were to be found, in part at least, in one of the chapters of Romberg's book on diseases of the nervous system, under the name of *tabes dorsalis*.

I had just returned from a visit, made for scientific purposes, to Berlin, Leipsig, and Zurich, and there my attention had been directed in some of the hospitals to patients suffering, as I was informed, from *tabes dorsalis*, but of whom the greater part were in reality *paraplegic*. In 1856, when I was at the Congress of Naturalists at Vienna, I saw the same mistakes in diagnosis made by Ludwig Turck, who showed me in his hospital cases of *paraplegia* as typical instances of *tabes dorsalis*. I knew then

that the disease called *tabes dorsalis* by most Germans was not the same which I have described under the name of locomotor ataxy. The perusal of Romberg's short chapter on *tabes dorsalis* has confirmed me in this opinion. And this I am bound to say, while rendering full justice to the eminent pathologist of Berlin.

Finally, these are the facts which seem to me to be perfectly established: 1. That prior to my investigations locomotor ataxy was completely ignored in France; 2. My description of the symptoms differs essentially from those muscular conditions to which it has certain points of resemblance; *e.g.*, the *tabes dorsalis* of the Germans, in which the symptoms of paraplegia and ataxy are mixed and confounded together; the *dorsal consumption* of Lallemand, described in his treatise on seminal losses; the *cerebellar paralyses* of Bouillaud, which cause only a little staggering giddiness perfectly distinct from inco-ordination; *paralysis of the sense of muscular activity* (Gerdy), or of the *muscular sense* (Bell), which is nothing but a complete loss of ordinary sensibility, and which has been confounded by Landry with locomotor inco-ordination.

NOTE BY THE EDITOR.

The description of locomotor ataxy given by Duchenne must be received as still almost complete, comparatively few touches having been added to the picture since it first left the master's hand.

The coarser features of this disease are sufficiently often associated to constitute a morbid entity, an *espèce morbide* as Duchenne calls it, and the name which he gave to this new species has been of great clinical value, and has enabled the pathologist to determine those minute changes in the spinal cord upon which the symptoms depend.

It must be remembered that it is no more possible to draw rigid lines of demarcation in pathology than in other departments of natural science. "Morbid species" fuse imperceptibly together, the divisions are artificial, and the separation of the symptoms into *phenomena* and *epiphenomena* is artificial also.

When a pathological process has once started, who shall assign a limit to its operations? It may be true, nay, is true,

that the hardening process, which we call sclerosis, very often limits its operations to one or other of the columns of the spinal cord, and travels apparently along the path of least resistance, but it is true also that sclerosis having once started may wander from one column to another, may go upwards or downwards, and may even involve the brain itself. Thus it is that clinicians tell us that locomotor ataxy is sometimes complicated in its later stages with progressive muscular atrophy; that it often appears in conjunction with general paralysis of the insane; that occasionally, while one leg presents the symptoms of ataxy, the other is more suggestive of *spastic paraplegia*, and so forth. The time will come, perhaps, when these clinical designations will be abolished, and we shall not use so often the somewhat clumsy expression that one disease complicates another. The only satisfactory form of nomenclature is that which designates the disease-process and its seat; and when we state that a hardening process is involving such and such columns and sections of the spinal cord, a complete picture of the symptoms will arise in the mind of the physician whose clinical knowledge is based, as it should be, on physiology and pathology.

There is one symptom of posterior sclerosis which has been worked out since Duchenne's time. This is the loss of the knee-jerk, knee-reflex, patellar-tendon-reflex, patellar-reflex, tendon-reflex, knee phenomenon, patellar-tendon phenomenon, or myotatic phenomenon, as it has been variously called by different writers. When one knee is crossed over the other and the leg hangs effortless, the physiological jerk forward of the foot which should occur when the patellar tendon is struck with the side of the hand or the end of a stethoscope, is generally absent in posterior sclerosis, and absent, moreover, in the early stages of the disease before the coarser phenomena are seen. When knee-jerk is absent in a patient, whose quadriceps extensor cruris muscles are sound, the fact should arouse the suspicions of the physician as to the possibility of a degenerative process having commenced in the posterior columns of the cord.

Knee-jerk is so easily tested, and its absence or excess is so very readily determined, that it has come to hold a routine position among the symptoms for which the physician seeks. Knee-jerk is of course absent if the muscles which extend the knee be paralysed or wasted and unable to perform their function.

Knee-jerk is increased whenever the influence of the will is removed from a healthy spinal cord. It has been found slightly excessive on the paralysed side in cases of recent cerebral hemiplegia, and in cases of localised damage to the cord in its upper part. Excessive knee-jerk reaches its maximum, however, in such cases, gradually, and it is not till the will-path (the pyramidal tracts in the lateral columns of the cord) has undergone complete degeneration that it is seen in its highest degree.

When the path for the will through the spinal cord has degenerated, and the path for reflex movement remains intact, knee-jerk and other reflex phenomena are excessive.

When the path for reflex movement is damaged in the lumbar region, knee-jerk is lost or lessened. This is necessarily the case if the quadriceps extensor cruris be wasted, in consequence of damage to the motor cells in the front horns of the cord. If the posterior roots or root-zone be damaged, knee-jerk equally disappears, although the quadriceps extensor cruris may be, for voluntary acts, as strong as ever. It is in this connection that knee-jerk is capable of affording us very valuable information of commencing trouble.

Clinical and pathological considerations seem to point undoubtedly to the fact that, for the occurrence of knee-jerk, the reflex-loop must be sound. Nevertheless physiologists hesitate to say that knee-jerk is a true reflex phenomenon, because the contraction of the muscle follows the tap on the tendon in less than one-twenty-fifth of a second, whereas if it were a true sensori-motor phenomenon, at least one-fifteenth of a second would be required for its manifestation. It is clearly not a reflex phenomenon in any way comparable to the skin reflexes which occur on stimulating the sensory nerves without subjecting them to coarse mechanical disturbance. For the production of knee-jerk the extensor muscle must be first stretched (hence Gowers suggests the adjective *myotatic* as applicable to the group of phenomena to which knee-jerk belongs), and when it is in this stretched condition, a further shock is given to it by the tap on the tendon, so that there is probably a mechanical disturbance of the whole length of the nerves of the muscle, and the phenomenon may very possibly be a true reflex phenomenon, notwithstanding the two-seventy-fifths of a second which is needed to bring the disputants together. In the loss of knee-jerk we have,

probably, a clue to the cause of the staggering gait, the ataxy, which accompanies posterior sclerosis. Every muscular act must be looked upon from two points of view: from the point of view of *permission* as well as *performance*. When the knee is bent, the gradual relaxation of the extensors is probably as important for the regularity of the act as is the contraction of the flexors, and the physiological knee-jerk is possibly a coarse manifestation of the mutual relationship existing between antagonistic groups of muscles, whereby movement is produced with regularity and smoothness, the stretching of one group of muscles by the action of their antagonists leading to an echoing contraction in the muscles stretched.

In posterior sclerosis this mutual reflex communication between antagonising groups of muscles is lost or lessened, and in consequence the movements of the limbs become jerky and irregular, and balance cannot be maintained without a maximum amount of assistance from the eye.

The diminution of reflex action in muscles is noticeable along the whole length of the spinal axis. The so-called "Argyll-Robertson" pupil, which is capable of pseudo-voluntary contraction during the accommodation of the eye, but is incapable of reflex contraction when light falls upon the retina, is often seen in posterior sclerosis, and is a phenomenon which is distinctly analogous to the loss of knee-jerk. This analogy is strongly insisted on by Buzzard and Erb. I have myself seen more than once (and once certainly in connection with posterior sclerosis) a failure of the soft palate to contract when locally stimulated, notwithstanding that perfect movement of it occurred when the patient took a deep inspiration.

Again, those cases in which the vocal cords fail to move during inspiration, notwithstanding that during phonation their movements are perfect, probably belong to the same category, and it is probable, as pointed out by Dr. Semon, that the nerve fibres supplying the posterior crico-arytænoid muscles have a central origin different from those other fibres of the recurrent laryngeal nerve, which supply the rest of the laryngeal muscles. Seeing how great must be the mutual dependence of the muscles of the eye in bringing about regularity of movement, it is, I think, probable that some of the cases of squint which occur in the early stages of posterior sclerosis are due more to the loss of

power of communication, by means of the reflex arc, between one set of muscles and their antagonists, than to the destruction of the motor cells from which the third, fourth, and sixth nerves take their origin. The often transient nature of this early symptom of posterior sclerosis is not easy to explain by any theory.

Another set of reflexes which are often implicated in posterior sclerosis are the vascular reflexes, and chief among these is that concerned in the erection of the penis. There seems no reason to doubt that impotence is common in cases of posterior sclerosis. It is proverbially difficult to learn the truth concerning the sexual functions. Nearly all patients will certainly lie rather than confess to a loss of virility. So that when the loss of virile power is acknowledged, we may feel tolerably certain that it is so. It seems consistent with the other symptoms of posterior sclerosis that the sexual reflex upon which erection depends should be early lost, and such undoubtedly is the case. The question of virile power in connection with posterior sclerosis was a matter in dispute in the case of Bagot *v.* Bagot, tried at Dublin in 1878. The testator had by his will denied the legitimacy of his reputed child, and the object of the trial was to prove that the will, which partially disinherited the child, was unreasonable. In 1869 Bagot was paraplegic, the result of an injury to the spine. From this he fairly recovered, but in 1872 he began to manifest symptoms of posterior sclerosis, which were strongly marked a year later, and from which he died in 1877, aged 52. In August, 1875, he married a lady who was in the seventh month of pregnancy, and the son and heir was born two months after marriage. At the time when this son was presumably procreated (February, 1875) Bagot was three years advanced in posterior sclerosis, and since he subsequently conceived the idea that the child was not his, it was sought to prove that the posterior sclerosis from which he suffered would, among other things, give support to such a supposition. Only one, among some dozen medical witnesses examined, was of opinion that Bagot's condition was compatible with full virile power, all the others asserted, more or less positively, that impotence was an ordinary concomitant of advanced locomotor ataxy.

There seems reason to suppose that sexual irritability with inordinate desire is occasionally a premonitory symptom of posterior sclerosis, and it has been disputed whether the excessive

exercise of the sexual function is to be regarded as the cause of the posterior sclerosis or merely a symptom of its commencement. It is well established, however, that when the disease is fairly advanced the sexual power is either lost or lessened. This is the experience of Duchenne, Trousseau, and nearly all English writers. It is certainly the experience of the editor of this book, an experience which is quite in harmony with theoretical considerations.

With regard to the trophic changes (herpes zoster, bullæ, arthropathies, &c.) which occasionally complicate posterior sclerosis, it seems to the writer probable that their explanation will be found in derangement of the vascular reflex arc upon the integrity of which nutrition changes very largely depend. Irritation of a sensory nerve causes first a contraction and then a dilatation of the vessels in the area of that nerve. This is a reflex phenomenon, and one point of reflection is probably the ganglion on the posterior nerve roots in which sensory and sympathetic branches are brought into close connection.

Charcot's explanation of his so-called "arthropathies," that they are due to destruction of the motor cells of the front horn, is inadmissible, because in anterior poliomyelitis, where an extensive destruction of these cells occurs, arthropathies are not found.

The connection of posterior sclerosis with syphilis has lately occupied the attention of many observers. According to Buzzard (*Disease of Nervous System*, p. 210) there was a history of syphilis in 45 cases out of 100 seen by himself, and the statistics of Fournier, Erb, and Buzzard taken together show that there was a history of syphilis in 76 out of 127 cases, or 59·8 per cent.

It is not always easy to be sure that a "history of syphilis" is really so, especially after a lapse of some time.

It must be remembered that those who have had venereal troubles have not seldom been "free livers" in many other respects, free drinkers perhaps, and given to sexual excess or exciting dissipation which has taxed the powers of the nervous system.

Posterior sclerosis is rare in women, and it does not occur more frequently among syphilitic prostitutes than among other classes of women.

Mercury and iodide of potassium have no curative effect upon posterior sclerosis.

The above considerations cannot but make one hesitate before enrolling posterior sclerosis among the syphilitic diseases of the nervous system. Before statistics can be of any use in determining the relationship between the constitutional and the local malady it would be necessary to know the percentage of adult males (especially among the class from which our hospitals draw their patients) whose personal experience includes a "history of syphilis" real or fancied.

Although Duchenne's description of locomotor ataxy must be regarded as really and truly "original" as far as he was concerned, there is no doubt whatever that the disease had been described by Romberg with a completeness which leaves little to be desired. The cases from which Romberg's description was written were, for the most part, in an advanced stage of the disease, and hence he has included among the symptoms loss of motor power, girdling pains, wasting of the muscles, and loss of control over the bladder, symptoms which we now accept as indicating that the degenerative change is no longer limited to the posterior columns. On the other hand, Romberg includes in his description the characteristic sensory troubles, the necessity of guiding the movements by the eye, the characteristic gait, the staggering, especially in turning round or in the dark, the squint, the condition of the pupil, the dimness of sight going on to blindness, the shooting pains in the limbs, the pains in the belly, and the sexual impotence. Romberg, writing in 1851, states that he had directed attention to the disease ten years previously. In his description of the morbid anatomy as seen by the naked eye, Romberg also points out that it is common to find the posterior nerve roots and posterior columns of the cord shrunken and hardened, while the anterior nerve roots and anterior columns of the cord retain their normal appearance. The claims of Romberg to be considered as at least an earlier describer of the disease than Duchenne are thus incontestable. It was Romberg's misfortune, however, to describe the disease under a name (tabes dorsalis—wasting of the back) which conveys no accurate idea to the mind of the physician or pathologist, while the name which Duchenne invented, so admirably suggestive of the leading symptom, was well calculated to bring the disease home to the minds of the great mass of the profession.

CHAPTER II.

PROGRESSIVE MUSCULAR ATROPHY IN THE ADULT AND INFANT.*

[Duchenne claims that the description of this disease published by Aran in 1850 was founded upon facts collected by himself and communicated to Aran in 1849. Duchenne's first memoir on the subject was published in 1853, and in 1861 he wrote an entirely new description founded on 159 cases. The description which follows (written in 1870) is founded on "a great number of cases."]

Symptoms of Muscular Atrophy in the Adult.—The first symptom complained of by patients smitten with progressive muscular atrophy is always a difficulty, a weakness, or an impossibility in executing movements, and which they never fail to attribute to a muscular paralysis. This also is the first idea of the physician. Nevertheless if the state of the muscle producing the imperfect movement be attentively examined, it is seen to be wasted almost in direct proportion to its weakness. It is not possible to prove the existence of this atrophy by the eye when the muscle is situated in one of the deep layers. For example, an atrophy of the deltoid is easily seen, but this is by no means the case with the serratus magnus, which lies deeply. Abundance of subcutaneous fatty tissue also masks the atrophy. I will make it evident in due course that "electro-muscular exploration" is alone able in these cases to demonstrate the existence of muscular atrophy.

This loss of power keeps pace with the diminution of muscular fibres. It is in the last stage, when the fibres are changed in texture, and have given place to a granular or fatty-granular tissue, and perhaps at the time when the nuclei of the sarcolemma are proliferating, that voluntary contraction completely disappears in the damaged muscle. (I shall return in due time to the discussion of this question.) Such is the clinical fact which, with some rare exceptions, recurs constantly in the cases which I have collected, and which I have demonstrated a hundred

* From *L'Electrisation Localisée*, 3rd edition, pp. 486—563.

times in hospitals and in my "polyclinic" by analysing the voluntary movements, and by "electro-muscular exploration."

I am bound to admit the possibility that muscular atrophy may have behaved differently in the cases reported by authors who have studied this disease.

Clinical observation shows then perfectly that the feebleness or loss of contractility in a muscle stricken with this condition is chiefly the *consequence* of the wasting of the muscle, or of its change in texture, and not the result of paralysis, *i.e.*, of a failure of the motor nerve-action.

Can it be said that the muscle thus affected in its nutrition preserves all the power of contractility which it could possess in the normal state with the same amount of fibres? I would not say this. I could, on the contrary, hardly comprehend that such a peripheral disease-state could leave the muscular force quite intact. I have often indeed seen muscles possessing a fair amount of force after having lost a third of their volume; but I do not think that this force was quite normal, and indeed I have sometimes found with my strength gauge that it was notably lessened, and not in proportion to the size of the muscle. This weakness was always the result of the local disease, and the muscle contracted until the last of its fibres was, so to say, stricken with death, which I think may happen either at a certain stage of proliferation of the nuclei of the sarcolemma, or when their tissue is destroyed.

It must be borne in mind that there are muscles (the diaphragm and the intercostals for example) which cease to be able to perform their functions as soon as they have lost a certain amount of power. Under such circumstances these muscles seem to be completely wasted and degenerated, although the texture of some of their fibres is still intact, and they still retain their contractility; but they are not able to contract under the influence of the voluntary or electric stimulus, because the levers which they have to move offer too great a resistance. It is possible to prove the contractility of other muscles as long as they preserve a few sound fibres, *e.g.*, the muscles of the fingers, because their levers only offer the trifling resistance of their own weight. I have proved with the strength gauge that their power was then only equal to

one or two kilogrammes. These considerations explain why, in a tolerably recent case, to which I shall refer hereafter, the diaphragm when a certain degree of atrophy or proliferation of the nuclei of the sarcolemma had been reached was not able to contract, or in other words, appeared paralysed, while the muscles of the upper limbs, *more atrophied than it*, could move their levers and stretch their tendons. I shall have occasion to apply these ideas to certain clinical facts.

The muscles whose nutrition is threatened, or actually damaged, are often jerked by little fibrillary or partial contractions. When this takes place the skin is seen to be raised and depressed as if by the tightening and loosening of delicate cords acting in the direction of the muscles. These contractions are of short duration, but they succeed each other with tolerable rapidity (at intervals of from one to four seconds), and occur in many points of the surface of the muscle. At other times the muscles are agitated with little wormlike movements. In some patients these contractions are almost continuous, and occupy a whole limb or a great part of the surface of the body, while in others great attention is necessary to prove their existence, owing to their rarity and weakness, and often they need to be provoked by voluntary or electric stimulation, or by compressing or pinching the muscles. After such stimulation the reflex fibrillary contractions are stronger and more numerous. Partial or fascicular spasmodic contractions sometimes cause little movements of the limbs, especially of the fingers or thumb. Like the foregoing they are very short and intermittent, but more rare. These fibrillary and partial muscular contractions are noticed by the patients, who compare the former to a slight shiver, and the latter to very weak and limited shocks.

Too much importance should not be attached to these little spasms, nor are they inseparable from progressive muscular atrophy, as many have said. In point of fact they are not always present, and in at least a *fifth* of the cases which I have observed I could not ascertain their existence. I have even seen patients come to the end of their troubles without having once experienced this symptom. On the other hand, fibrillary contractions show themselves in many other muscular affections; sometimes they constitute the only sign of disorder. I was consulted by a provincial *confrère* in whom for four years these

contractions had been seen in all parts of his body without any attendant emaciation or loss of muscular force. This sign, taken by itself, has not then a very great diagnostic value.

Fall of temperature in the affected limb is a constant symptom of progressive muscular atrophy, but I have never seen it present in a marked degree until the patient has reached a very advanced stage. The patients experience, at first, in the atrophied limb a sense of chill, and are very sensitive to cold, and later on the fall of temperature is appreciable to the touch. Then also the capillary circulation becomes less active, the veins of the skin are smaller, and the skin easily gets blue with the cold.

Electro-muscular contractility is normal in progressive muscular atrophy. This fact is established beyond doubt by long experience. Nevertheless at an advanced stage of the muscular atrophy one must take care not to be deceived by appearances. Thus faradisation of a very wasted muscle in the last stage of atrophy causes no movement, or only a feeble one, of the limb or part of the limb to which it belongs, especially when the healthy antagonising muscles oppose a tonic resistance to its action. We must not conclude that the contractility of such a muscle is weakened, the true meaning of such a fact being merely that the fibres are insufficient for performing the normal work of the muscle. In such a case we must take into account not the movement of the limb, but only the manner in which the muscular fibres contract while they are kept shortened; then we can prove that their contractility is normal. We shall see further on that when a muscle is partly changed in texture, those fasciculi which are still sound may be surrounded by fat or be divided transversely by one or more degenerated sections. These sound fasciculi still have their normal electric contractility, but it is conceivable how in such conditions there may be a difficulty in seeing their contractions through the skin, and we must not, therefore, conclude that their irritability is weakened. Certain muscles, the diaphragm for instance, cannot manifest this electric contractility when they have reached a certain stage of atrophy because of the great resistance of the lever which they have to move. There is also another kind of error against which I must warn the observer. When the contraction of a muscle whose antagonists are atrophied and fatty is provoked, the resulting

movement is so brusque and energetic that at first we might consider the electro-muscular contractility to be above normal, whereas the excess of movement is only due to the absence of tonic resistance on the part of the antagonists. We must take care in such cases not to conclude that the electro-muscular contractility is increased.

The importance of testing the muscles by faradisation must be clear to all. At the outset of my researches, the kind of "living autopsy" made daily in the wards of the Charité, in patients with muscular troubles, of necessity revealed the existence of progressive muscular atrophy even before the post-mortem examination and the microscope could show us the change of texture in these cases (a proliferation of the nuclei of the sarcolemma, and a waxy, glazy, fatty, or granulo-fatty degeneration).

The matter was demonstrated to me in this way. I found, in a certain case, a deltoid muscle in the last stage of atrophy, which, though reduced to an extreme thinness, responded perfectly at every point of its surface to electric stimulation, and caused slight movements of the upper limb. The will caused similar movements. Later, however, the contractility disappeared gradually, until at length electric stimulation only provoked the movement of a few fasciculi here and there. I attributed to a purely dynamic condition this preservation or disappearance of contractility in different parts of the same muscle. I felt (and told it to those who were watching my experiments) that where the contractility was lost the fibre had become altered or destroyed, because I had observed in all other wasting muscular affections (lead palsy, and paralysis from nerve-injury) that the loss of electro-muscular contractility affected the whole mass of the stricken muscle. Further, it was no surprise to me when later the first autopsy made at the Charité by Cruveilhier showed us that in all those wasted spots which had lost their contractility the muscular fibre was profoundly changed. I merely learnt then from the microscopical examination that the transverse markings and the longitudinal striæ had more or less disappeared, and had been replaced by granular matter and fat vesicles. And, indeed, without this electro-muscular exploration how would one distinguish progressive muscular atrophy from those atrophic muscular conditions

with preservation of muscular contractility which are unattended by change of tissue? Thus localised faradisation applied to muscular physiology and pathology showed me how frequent was progressive muscular atrophy. Patients suffering from this disease served me, as is well known, for my electrical experiments on muscles in my studies on the functions of the muscles of the hand, shoulder, foot, &c.

All authors who have written on progressive muscular atrophy have said that sensibility has always been normal in this condition. Their experience was too limited to formulate so general a proposition. Nor is it correct, for I have ascertained in a good third of the cases that the electro-muscular sensibility, as well as cutaneous sensibility, was more or less weakened. The want of sensation is sometimes so great that the patients do not notice the strongest faradisation nor the application of fire. have seen some who have been badly burnt on their anæsthetic parts, without being conscious of it at the time. This anæsthesia is noticed usually in the upper limb, and diminishes from the hand to the shoulder. Sometimes indeed it is irregular in its position, and is not always proportional to the atrophy. Thus I have seen it limited to a part of the trunk or to the shoulder; at other times it was absolute in the right upper limb, and feebly developed in the opposite limb, which, moreover, was much the less atrophied of the two.

This muscular and cutaneous anæsthesia only affects as a rule those patients who have suffered pains which have been attributed to a rheumatic influence. We shall see later that it is but a complication, or at most an extension of the anatomical central lesion (to the posterior horns of the cord).

The Situation and Progress of Muscular Atrophy.—*Commencement in the upper limbs.*—As a rule, before becoming general, progressive muscular atrophy attacks the upper limb, and destroys its muscles in an irregular fashion. It begins in such cases by attacking one after another the muscles of the thenar eminence, spreading from the superficial to the deep layer. As soon as the abductor pollicis is wasted, its absence is marked by a depression, and by the attitude, during repose, of the first metacarpal bone, which lies too close to the second. (See fig. 6.) When the deep muscles are affected the thenar eminence gets quite flat, and the first

metacarpal bone always lies in the same plane as the second. Depressions of the hypothenar eminence and interosseal spaces next announce the atrophy of the muscles of those regions.

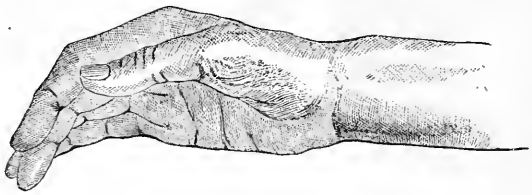

Fig. 6.

The loss of the interossei muscles is shown by the claw-like attitude of the fingers during the extension of the hand. (See figs. 7 and 8.) The functional troubles caused by these

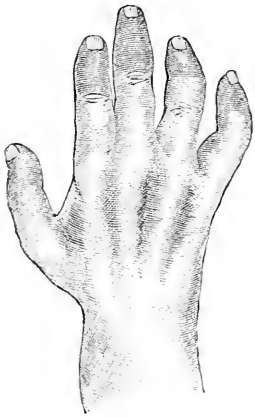

Fig. 7.

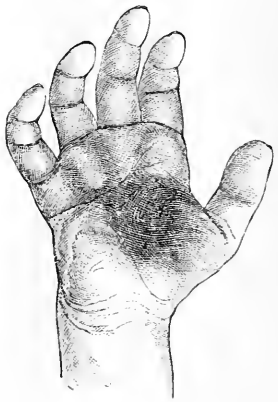

Fig. 8.

atrophies are considerable, and are described in my work, *Physiologie des Mouvements* (1861).

. . . . . .

If the flexors and extensors of the fingers become in their turn atrophied, the hand (which was "clawed" while the atrophy was limited to the interossei) assumes the death-attitude, and the forearm is literally dissected.

Occasionally, but not often, the muscles on the back of the forearm are first attacked, but even in these cases the preceding muscles are soon involved. The atrophy may remain thus

localised for many years, as was the case with the patient whose hand is represented in figs. 7 and 8.

. . . . . .

I have always been careful to examine the state of the muscles of all parts of the body, and have ascertained that generally the atrophy was limited in the first instance to the hand and forearm. But this limit once passed the muscles of the arm and trunk waste irregularly and partially.

The flexors of the elbow and the deltoid are the first to atrophy, sometimes the one and sometimes the other taking precedence. The triceps extensor cubiti is the last of the muscles of the upper limb to become affected. Once, indeed, I have seen it completely destroyed, while the rest of the muscles of the shoulder-joint were but little affected.

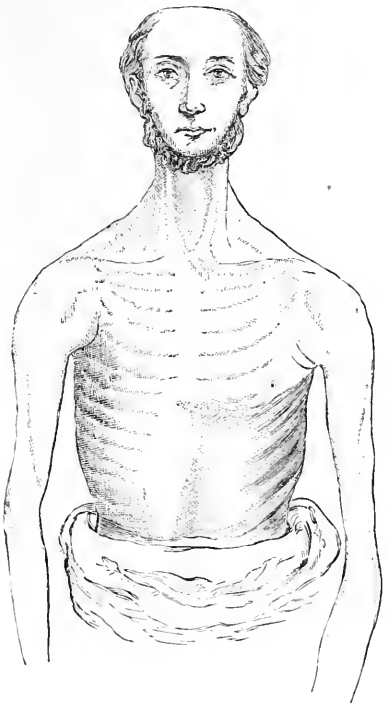

Fig. 9.

Whenever all the muscles of the arm have been atrophied, I have found a greater or less number of the muscles of the trunk in the same condition. The patients are nearly always in ignorance of the time when these muscles began to waste, because, not being much hindered in their movements, their attention was not forcibly drawn to them, unless the serratus magnus, one of the most necessary muscles for the movement of the upper limb, has been affected early, as was the case in the patient represented in fig. 9.

The following is the order in which the trunk muscles usually waste. First, the lower half of the trapezius disappears, and then the spinal border of the scapula is further removed from

the median line than on the sound side. The clavicular portion of this muscle is generally the *ultimum moriens* of all the muscles of the trunk and neck. Next in succession atrophy attacks the pectorales, the latissimi dorsi, the rhomboidei, the levatores anguli scapulæ, the flexors and extensors of the head, the erectores spinæ (*les sacro-spinaux*), and the muscles of the abdomen. At this time I have usually seen the muscles of breathing and swallowing become affected. The atrophy equally invades the lower limbs, but only when the muscles of the upper limbs and trunk are in great part destroyed. It is most marked in the flexors of the ankle and hip. The other muscles of the lower limbs may become atrophied in the long run.

I have not seen atrophy attack both sides at once, but when one muscle or group of muscles is affected the corresponding muscles are usually attacked at no distant time, and before the disease spreads to other regions. Thus I have seen a certain number of cases in which the same muscles were successively attacked in the two hands. The same has also occurred with the deltoids and the serrati magni. Such is the usual mode of development of progressive muscular atrophy.

It must be borne in mind, however, that the course of development may be different. Of this the following is an example.

Case No. 4.—In a patient from Barcelona, æt. 32, whom I treated in consultation with Professor Trousseau, and in whom the atrophy became general in two years, the muscles were attacked in the following order. The muscles of the right hand were first attacked, then the flexors of the left ankle; next the left hand was attacked, and this was followed by atrophy of the flexors of the right ankle and right hip; then, in different degrees, the atrophy affected the biceps, deltoids, and muscles of the trunk, neck, and face. When I first saw him the diaphragm, tongue, and muscles of swallowing were so seriously affected as to endanger the patient's life by asphyxia or starvation.

. . . . . .

Commencement in the Trunk Muscles.—Muscular atrophy does not always begin in the upper limbs, and I have collected a dozen cases of its attacking the muscles of the trunk in the first instance. Once I saw it begin in the erectores spinæ, which progressively atrophied in a poulterer, who then began to carry

his great flat baskets filled with chickens on his head instead of on his back as formerly. . . .

In a patient named Bonnard the atrophy attacked nearly all the superficial muscles of the trunk before reaching the upper limbs. (See figs. 9 and 10.) . . .

In 1859 I saw a labourer (Thais, æt. 46, who had suffered for two years), in whom the atrophy had taken the same course, and had destroyed the same muscles of the trunk and upper limbs. . . .

In another patient—an athlete—the pectoral muscles, the latissimi dorsi, and the right serratus magnus were alone attacked. The thinness of the trunk was in striking contrast with the high muscular development of the upper limbs, which were still intact.

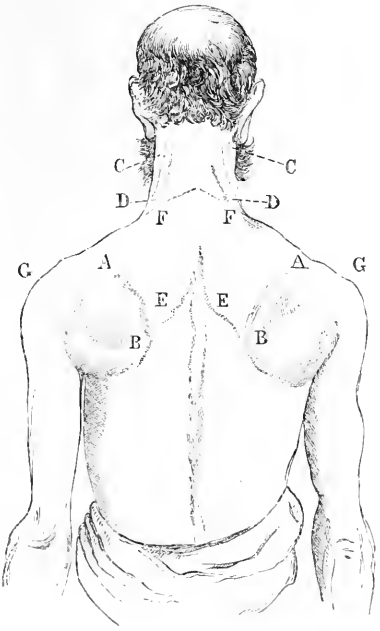

Fig. 10.

Cases Nos. 5, 6, 7.—I saw simultaneously three women who had lost the serrati magni, the trapezii, and the rhomboidei on each side after having carried heavy weights on the arm for a long time. Two of these women, who had been nurses for many years, had carried children in their arms for whole days when they were quite young. One of them tells me that when she was eleven years old the village schoolmaster used to punish her by making her kneel in sabots and hold a heavy stone in each hand while the arms were extended. This barbarous punishment was continued during many hours every day for nearly a year. At the end of this time her shoulders began to project and the curvature, observed during standing, made its appearance. (See fig. 13.) The muscles going from the trunk to the scapula were the first to atrophy; the muscles of her abdomen were the next

to go, for she said that her back was bent backwards after the deformity of the shoulders. I diagnosed the paralysis of the abdominal muscles by electrical exploration and by the movements which I made her execute. In fact these muscles did not contract under the influence of electric excitation, and when she lay on her back she could not raise herself, although when bent forward she could straighten herself forcibly, and one could then feel the spinal muscles swelling and contracting forcibly. The disease became general in the unhappy woman. She had besides lost in infancy the orbicularis oris and some other muscles of the face. Her lower limbs were beginning to suffer. There was in this patient a peculiarity seen only in this disease, viz., the presence of muscles perfectly intact alongside others which were as perfectly wasted. In particular the pectorales and muscles of the upper limbs were strikingly developed. This patient had never suffered the least pain, and there was a complete absence of fibrillary contraction.

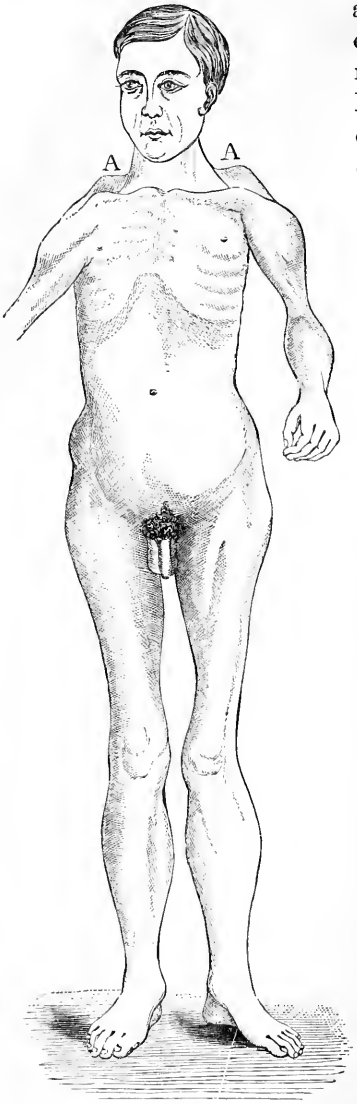

Fig. 11.

In the patient represented in fig. 11 most of the trunk muscles are destroyed (pectorales, trapezii, rhomboidei, latissimi dorsi, serrati magni, flexors of the lip, and other muscles of the lower limbs), but in the upper limbs this patient has only lost the long supinators which give a peculiar (fusiform) shape to the

forearm. The flexors of the elbow are now only beginning to atrophy. Lastly, I have only seen progressive muscular atrophy begin in the lower limbs (attacking the flexors of the ankle) twice out of 159 cases.

Changes of Form and Attitude—Locomotor Troubles.—. . . . The local signs of atrophy of individual muscles, putting aside the evidence obtainable by electro-muscular exploration, are—

1. Superficial deformities, *i.e.*, irregular depressions corresponding to the wasted muscles.

2. Alterations in the attitude of limbs during repose, caused by the loss of muscular equilibrium.

3. Anomalies during voluntary movement by the failure of a muscle either to produce its own movement or to co-operate effectually with others.

We will consider these signs in detail.

Superficial deformities. . . . A muscle may be wasted alongside of others which are perfectly intact notwithstanding a community of nervous supply. Normal muscular projections become depressions, which contrast strongly with the development of the neighbouring parts. [This peculiarity may be said to be the "facies" of this disease, and it is well shown in the numerous figures I have inserted in the text.] The atrophy of a great number of muscles is sometimes masked by obesity, as was the case in one patient, whose atrophied pectorales were concealed by a thick layer of fat. At an advanced stage the aspect of the disease is no longer the same. Abnormal depressions no longer stand in contrast with reliefs and bosses; the muscular layer has all vanished, especially on the trunk and upper limbs, and the skin seems literally to be fastened on the bones. The patients have then a peculiar appearance, which could not be confounded with the wasting of other diseases.

. . . . In these cases the well-nourished face contrasts with the wasted body, whereas in ordinary wasting diseases the thin furrowed face is in harmony with the thin body and limbs. In mere emaciation one may find the remains of muscular reliefs, but in advanced muscular atrophy they have all disappeared.

Faulty attitudes of the limbs during repose.—The position of limbs at rest is due to the tonic force of their muscles. There

is no muscle which has not its antagonist, and directly one of the opposing muscles is weakened the tonic equilibrium is destroyed, and the limbs are drawn in the direction of the stronger force. Such is the explanation of the faulty attitudes which are seen in progressive muscular atrophy, in the hand, shoulder, or trunk. . . .

Functional troubles during voluntary action.—These troubles are of two kinds. They affect the proper movement of the muscle itself, or the resultant movement of co-operative muscular efforts. All voluntary movements are made up of the movement of the chief muscle and the co-operation of others which help, regulate and moderate this movement. The proper individual actions of muscles are not all of the same importance in voluntary acts. Some are indispensable to the right use of a limb, while others are of secondary utility. I have seen patients who had lost the whole of their superficial trunk muscles (pectorales, latissimi, trapezii, rhomboidei, muscles of secondary utility), and who nevertheless knew nothing about it, so little were they troubled in their movements. They had not sought any treatment until muscles absolutely necessary for certain movements had become affected. One (for example) consulted me about a difficulty of raising the arm (his deltoid was atrophied); another about some difficulty in flexing the elbow or using the hand; but none of them complained of the loss of the trunk muscles, whose atrophy I ascertained by electro-muscular explorations, by faulty positions of the shoulders, or by deformities of the trunk. Thus we should never neglect to look at all the muscles of the body although the patient may only complain of partial wasting of one limb.

Failures of co-operation are not less important as signs of partial wasting. I will content myself with one example. Sometimes while the arm is being raised one sees the scapula project like a wing from the thorax. The co-operation of the serratus magnus is here wanting, and this pathological movement is the evidence of it.

Troubles of muscular co-operation may afford evidence of the wasting of a muscle which is still apparently sound. Thus when the flexors of the fingers are weakened or lost the patient cannot shut the hand without extending the wrist forcibly, or when the extensor of the fingers (*i.e.*, of the near phalanges) no

longer acts, the patient is obliged to flex the wrist when he wishes to place the near phalanges in the same plane as the metacarpal bones. When I first saw this I thought that in the first case the flexors and in the second case the extensors of the wrist were destroyed, although there was nothing of the sort. (See *Physiologie des Mouvements.*) . . . . To complete the study of this subject I will allude to the position of the

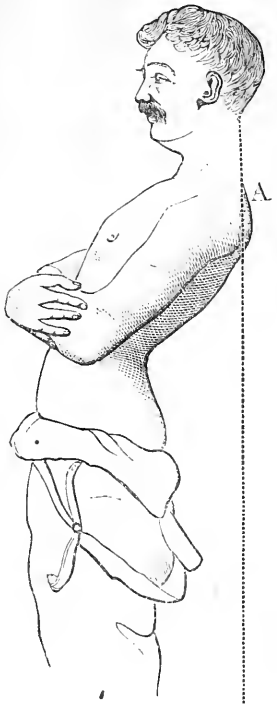

Fig. 12.

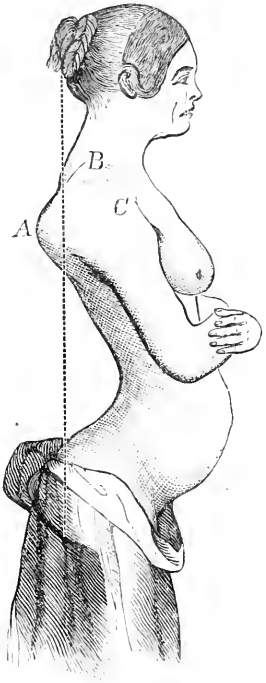

Fig. 13.

trunk during standing when the extensors or flexors of the spine are atrophied.

Figs. 12 and 13 represent two patients affected with lordosis through muscular atrophy. At first sight they look alike, but yet it appears that the curvature is caused by the atrophy of muscles which act on the trunk in opposite directions. It is easy to show that they differ essentially from each other. By looking at the position of the vertebral column one sees that an imaginary

line falling vertically from the first dorsal vertebra passes, in fig. 12, well behind the sacrum, while in fig. 13 this line passes in front of it. I have elsewhere explained the mechanism of the different attitudes of the vertebral column in these two kinds of lordosis. (*Physiologie des Mouvements*, p. 716.) [The lordosis in fig. 12 indicates a failure of the extensors of the trunk, while the lordosis of fig. 13 is indicative of failure of the flexors of the trunk (the abdominal muscles).] According to M. Bouvier a wasting of the extensors of the hip may cause a lordosis like fig. 13. I am sorry I cannot agree with my learned friend, because the extension of the trunk on the thigh is no longer possible when, owing to paralysis of the muscles producing this movement, the pelvis is bent forwards on the thigh.

Lastly, the lordosis from wasting of the abdominal muscles offers some resemblance to that which is caused by contraction of the flexors of the legs, or by congenital double dislocation of the hip-joint; but the proper signs of this last named affection render any confusion impossible.

General Symptoms.—Progressive muscular atrophy may reach its last stage (that of generalisation) without occasioning any other trouble but faulty muscular nutrition. It has never caused the least degree of fever in any of the cases I have seen. Fever is a mere accident, or due to the intercurrent diseases which carry off the patient.

Digestion is always good. Feeding becomes difficult when the muscles of chewing and swallowing begin to waste. The depressors of the lower jaw are among the first of the masticatory muscles to be affected. In such a case the depression of the lower jaw is only made with effort, and becomes more and more limited, until the depressor muscles are destroyed, when voluntary separation of the jaws becomes impossible, and feeding, even with liquids, is very difficult. The patient then carries his lower jaw protruded (doubtless by the help of the pterygoids), so as to leave an interval between the teeth of the upper and lower jaw. It is through this little slit, while the lips are forcibly separated, that he manages to introduce soup or liquid into the mouth. In one patient this state of things lasted for more than a year. At first he could not open his mouth without effort, and at this time I ascertained the existence of considerable wasting of the depressors of the lower jaw, which

were scarcely strong enough to overcome the tonic resistance (very great, as one knows) of the elevators. The power of separating the jaws then gradually diminished, and some months before his death, when he wished to open the mouth he began by protruding the lower jaw, and then managed to depress it for 1 or 2 centimetres. Finally, this power of depression was lost, and he could only manage to protrude the jaw. It will be seen from this case how serious a matter is atrophy of the depressors of the jaw. The difficulty in seizing and chewing food is usually accompanied by laborious swallowing. It is well to bear in mind that an apparent excess of saliva, due to the difficulty of swallowing it, is one of the first signs of impaired deglutition. Before long it becomes very difficult to swallow food, and this trouble gets progressively worse, till the patient loses the little strength he has, and is sometimes in danger of dying of starvation.

There is no paralysis of the bladder or rectum in this disease, although defecation and micturition become less easy when the abdominal muscles are wasted.

Respiration remains normal as long as the muscles concerned in its mechanism are sound . . . Wasting of the diaphragm, while it proves a great hindrance to breathing, and still more to phonation, does not produce any immediately serious accident; but in such a case a slight bronchitis may prove fatal, as was the case with a patient whom I saw with M. Cruveilhier. When the diaphragm is attacked the patient's life is in danger.

The intercostal muscles waste before, after, or at the same time as the diaphragm. Since they are inspiratory muscles, it follows that the instant they, as well as the diaphragm, cease to act, asphyxia takes place. Three times I have had an opportunity of seeing atrophy of the intercostals, while the diaphragm remained sound or nearly so, and I was then able to observe the degree of utility of these muscles and the functional troubles caused by their atrophy alone . . . When the intercostals were atrophied, so that the ribs could be no longer raised, it was with difficulty that the patients could cry or sing, but they were not completely noiseless, because the diaphragm was sound. Their voices were weak, however, and their phrases frequently interrupted by the necessities of respiration. They could not take a long breath, and when they tried to do so the epigastrium and base of the thorax heaved strongly with the action of the

diaphragm, while the upper two-thirds of the thorax remained motionless. When they tried to blow out a candle the feeble puff scarcely stirred the flame, and in spite of all their efforts they could not extinguish it. The weak voice is thus accounted for. Although with the help of the diaphragm they might draw a long breath, expiration was always very short, so that they could only utter two or three consecutive words without taking a fresh breath. It is always easy to recognise atrophy of the intercostals if attention be directed to the point, especially when, as is usually the case at this stage of the disease, the pectorales are almost entirely destroyed and allow the intercostal spaces to be seen. What strikes the observer is the deepening of these intercostal spaces, and absence of expansion in the upper part of the thorax during inspiration contrasting with the integrity of diaphragmatic respiration. I admit that for a long time I misunderstood the signs of partial atrophy of the intercostals, and the cause of this error of diagnosis is easily shown. For when these patients after a long inspiration only make a short expiration, it is only natural to attribute this to paralysis of the abdominal muscles. Yet very little care is needed to recognise that these muscles are sound, for they are seen to contract vigorously. Their action, however, is to compress the base of the thorax, and then to shorten its vertical diameter by pushing up the diaphragm indirectly through pressure on the abdominal viscera. The expiratory force is diminished at least one-half by the atrophy of the intercostals. This is explained by the want of the elastic recoil of the ribs, which in these cases remain depressed. This elastic recoil plays an important part, as is well known, in expiration. The elastic power of the ribs which tends to contract the framework of the thorax is so great that it has to be moderated by the tonic antagonism of the intercostal muscles. In fact the chest is as much contracted in patients whose intercostals no longer act (see fig. 14), as it is enlarged in those whose diaphragm no longer works (see fig. 15). Atrophy of the intercostals seems to me to be as serious as that of the diaphragm. In either case a simple bronchitis may cause asphyxia.

The thoracic perimeters shown in figs. 14 and 15 are from two patients suffering from progressive muscular atrophy. They were taken during full inspiration by means of the cyrtometer of

Woillez, applied at the level of the xiphoid cartilage. In the case represented by fig. 15 the diaphragm, as well as most of the muscles covering the thorax (pectorales, trapezii, rhomboidei, latissimi), had lost its power. During inspiration, consequently, the epigastrium was depressed, while it was raised during expiration. The direct electrisation of the intercostals in each space caused them to swell, and the lower rib to be raised towards the upper. From this I concluded that the intercostals were elevators of the ribs, and therefore inspiratory muscles, and indeed we see that an exaggeration of this action, combined with the supplementary thoracic movement (costo-superior), had actually produced an increase of the antero-posterior diameter of the thorax, such as is shown in fig. 15. The perimeter in fig. 14 was taken from a patient in whom

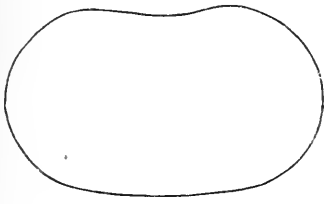

Fig. 14.

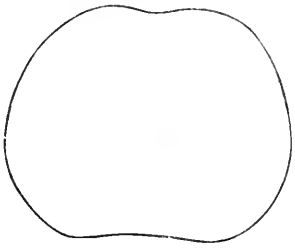

Fig. 15.

inspiration was entirely diaphragmatic, so that during inspiration the epigastrium bulged enormously, and the upper two-thirds of the thorax remained fixed in spite of every effort at costal respiration.

. . . . . .

From these two cases, and from others analogous to them, which I communicated to the Paris Medical Congress in 1867, I concluded—

1. That the intercostals (both external and internal) are inspiratory muscles. This fact has been shown by M. Duval, the late Director of the Naval Medical School at Brest, who in 1866 made many electrical experiments on the sailors of the *Fœderis-Arca*, who were executed, and among others on the intercostals, which were laid bare immediately after death.

2. That these muscles are necessary for maintaining the normal capacity of the thorax.

I have already said that in progressive muscular atrophy there is no nervous trouble beyond a diminution of muscular sensibility, keeping pace with the atrophy, and that there is occasionally met with a want of sensibility of the skin in the atrophied regions. I have also stated that any neuralgic or rheumatic pains which may have preceded or accompanied the atrophy should be considered as a mere complication, because in the majority of cases they are wanting.

There are, finally, no mind-troubles in this disease, but, on the contrary, the intellect remains clear and the memory perfect.

. . . . The usual course of the disease (as given above) is rarely departed from. The muscles of swallowing, articulating, and breathing, are usually the last to be attacked.

Duration.—This is very variable. The disease may reach its last stage in less than two years. I have indeed seen a case where, in this time, it had more or less affected a great many of the muscles of the upper limbs and trunk, as well as some of the muscles of the lower limbs, and in the last two months had involved the facial muscles and the muscles of swallowing and breathing. This is the quickest course that I have ever seen this disease take.

It is not unusual for muscular atrophy to remain stationary for a greater or less time, or even to stop altogether after having invaded one or more regions. I have seen it in this way remain localised for some eight or nine years in the muscles of the hand or trunk. I have notes of a case in which, after remaining localised in the muscles of the thenar eminence for fifteen years, the disease again took on its progressive course.

Happily patients may live a long time, even when the atrophy has become general, provided that the muscles essential to life be not attacked. I am reminded, indeed, of several members of one family who lived to a very advanced age, notwithstanding that muscular atrophy had in them become generalised ever since the age of 18. They were not carried off by their muscular troubles; but, be it remembered, the disease had respected the muscles essential for swallowing and breathing.

Symptoms of Progressive Muscular Atrophy in Infancy.—Long ago (1855) I published clinical facts which proved that this disease may occur in infancy, and that it then presents some peculiar characters. As I formerly thought these facts were

exceptional, I have not till now drawn special attention to them, and hence it happens that they are usually ignored or misunderstood. For this reason, perhaps, progressive muscular atrophy of childhood has been confounded with other muscular affections of that age. One of the chief reasons for trying to dispel this confusion is the fact that it has been created by observers of great merit. In order that such errors of diagnosis may not occur in the future, I wish to seize this opportunity to recall, or even to describe afresh from new facts, the principal characters of this variety of progressive muscular atrophy occurring in childhood or youth.

This form of the disease, of which I have collected more than twenty cases, has this peculiarity, which I have never met with in the disease as it affects the adult, viz., that it begins in certain muscles of the face, giving to it a peculiar expression, before attacking the upper limbs or trunk. This commencement in the face may be considered as a characteristic of the progressive muscular atrophy of childhood, and is further a premonitory sign of its extension, sooner or later, to the muscles of the limbs and trunk.

Case No. 8.—M. X., æt. 64, whose father died of atrophy, deprived of nearly all movement, had seven children, of which six are still living. Four of these enjoy good health, except one who has *big lips with little mobility*. The two youngest, a boy and a girl, began towards the age of 5 to manifest a peculiarity in the expression of the face. At 6 years of age the lips *had become big and had lost their mobility*. Their expression, when at rest, was dull, although their intelligence was not affected. The face was thin, and during laughter, which was of a sardonic character, the cheeks were flattened. There was a peculiarity in the articulation of labials. Between 11 and 12 there was a progressive wasting of the shoulder and arm on one side, and weakness in raising the arm, the scapula of the affected side projecting during the attempt, owing to atrophy of the serratus magnus, and there was further a difficulty in flexing the elbow. The atrophy next extended to the same muscles on the opposite side, and then to those of the thorax, most of which disappeared in turn and almost entirely. The muscles of the forearm and hand remained, nevertheless, intact. Some of the muscles of the lower limbs, flexors of the hip chiefly, were the last attacked,

and this rendered walking difficult and fatiguing. They are now respectively 31 and 41 years of age.

M. X. transmitted to two of his children (whose history we have just given) the germ of progressive muscular atrophy which he had himself inherited from his father. M. X. himself remained free from all muscular trouble till he was 48 years old, when, without any pain, his shoulders began to grow thin, and raising the arm became more and more difficult, and ere long his shoulder blades projected like wings. Then the muscles of his forearm and chest successively and slowly atrophied, and finally the muscles of his lower limbs were, in their turn, attacked. In fact the progressive muscular atrophy invaded the same muscles as in his children, without, however, affecting the muscles of the face.

Seeing himself threatened with immediate loss of all motor power, M. X. came to consult me as to the means of arresting the progress of his disease. It was then that he related this sad story of his family disease, and I had also an opportunity of ascertaining with my own eyes and by electrical testing the different degrees of atrophy of many muscles.

The two first of the three cases given above afford a faithful picture of the progressive muscular atrophy of childhood. Indeed out of fifteen cases which I have collected, bearing a strong analogy to these two, the onset of the disease, between the ages of 5 and 7 years, was marked by the atrophy of the same muscles of the face, and primarily by atrophy of the orbicularis oris, the want of contractile power in which caused the characteristic thickness of the lips. The facial expression then assumed the character above described, especially during laughter, which was effected solely by the buccinators or the *risorius santorini*. After a period of arrest the atrophy invaded, between the ages of 9 and 14, the upper limbs, trunk, and lastly, the lower limbs, the muscles of which it destroyed progressively, irregularly, and capriciously, leaving some of them intact, just as in progressive muscular atrophy in the adult.

The distinguishing and assimilating characters of these two diseases are both brought into prominence by the story of the father, who, after having transmitted to his two children the germ which he had himself inherited, suffered no attack himself till the age of 48. We have seen how in him the family disease,

leaving the muscles of the face intact, began with the upper limbs, spreading thence to the trunk, and finally to the lower limbs, destroying successively almost the same muscles as in his children.

In order to complete this sketch of the progressive muscular atrophy of children, I have tried to give its objective signs by means of photographs which were published in the *Revue Photographique des Hôpitaux de Paris* in March, 1869.

In the great majority of cases of progressive muscular atrophy of childhood the disease has been hereditary.

. . . . . .

Case No. 9 (summary).—A boy, æt. 13. In childhood his lips became thick and pendulous. There was want of action (as established electrically) of the orbicularis oris, levatores labii superioris, zygomatici, and an inability to pout the lips or bring them firmly together. When he wished to close the mouth he stretched the lips laterally by means of the buccinator. He raised the lower lips by means of the chin muscles, and depressed the upper lips by means of the depressor alie nasi (myrtiform) muscles. He had the same movements for laughing and crying, which gave an odd expression to the face. At 12 he had wasting of the thoracic muscles, separation of the scapulæ from the thorax like wings, when the arms were raised, &c. Since then the wasting has increased. The disease attacked in youth three of his maternal relatives. His grandmother, aged 60, began to suffer at 19; a maternal uncle, aged 33, was attacked when 13, and in his mother, aged 30, the disease was congenital. In the two first most of the muscles of the upper limb were progressively destroyed, whilst it was limited to the face in the mother, who, like her child, had thick lips.

Case No. 10.—L. Hottmann, æt. 9, was sent to me in September, 1868, suffering from progressive muscular atrophy. His lips were thick, and remained separated when at rest, the lower one being pendulous. The obicularis oris remained inert and he could not pout. The other lip muscles had also lost their power. There were no naso-labial grooves, and when he laughed his mouth stretched sideways (by the action of the buccinators) and his lips turned a little outwards, giving his laugh so odd a look that his comrades said he laughed

"*en cul de poule.*" The trouble began when he was 3 years old. At 5 the trunk and lower limbs began to grow thin, and between 6 and 7 wasting of the upper limbs began. This wasting was not preceded by fever or wide-spread palsy. The mother had begun to suffer from weakness of the arms at the age of 13, and had wasting of many muscles and thick lips, which were a family characteristic.

Her eldest son at 14 had suffered for some months from a weakness of the arm, which prevented his using the hammer. He had thick lips, and I found wasting of the deltoid, serratus magnus, and lower two-thirds of the pectoralis major on the right side. A sister, who resembled the father rather than the mother, had escaped the disease.

. . . . . .

I have, nevertheless, seen infantile progressive muscular atrophy develop without hereditary cause.

In short it results, from my cases—

1. That this disease sometimes begins in late childhood (towards 6 or 7) in the facial muscles, which gives an odd expression. The lips are thick and motionless, and the cheeks hollow when laughing.

2. Later it invades the muscles of the trunk and limbs.

3. As in the adult, it attacks the upper limbs before the lower.

4. In its course it destroys the muscles partially, successively, and in a peculiar manner.

5. It abolishes isolated movements, and causes deformities after the destruction of the muscles.

These chief symptoms of the progressive muscular atrophy of childhood form the elements for diagnosis from the same disease in the adult.

Pathological Anatomy.—[Duchenne's earliest investigations of the pathological anatomy of this disease were made on the body of a patient named Lecomte, who was in the Charité under M. Andral in 1850, and there attracted the attention of Duchenne. By Duchenne's desire Andral kept this man under his care for two years, but he died under the care of M. Cruveilhier, who published the case in the *Bulletin de l'Acad. de Méd.*, 1852—53, t. xviii., p. 490—546. The muscles were examined by Duchenne, Mandl, Aran, and Galliet. The account given by Edward Meryon of a case of muscular

degeneration in the Medico-Chirurgical Transactions for 1854 and 1866 refers in reality, according to Duchenne, to pseudo-hypertrophic paralysis.]

Duchenne gives a coloured figure (fig. 113, plate 1, of the third edition of *L'Electrisation Localisée*) of the dissected arm of Lecomte. In this figure all the muscles of the arm, although in the last stage of atrophy, are seen to have preserved an almost normal colour. There was no microscopic change in these muscles, and they retained their electric contractility up to the time of Lecomte's death. The brachialis anticus was alone much changed and had a pale grey colour. Its electric irritability was extinct. On the front of the forearm the flexor carpi ulnaris (*cubital anterieur*), the flexor carpi radialis (*grand palmaire*), and the palmaris longus (*petit palmaire*) were reduced to mere tendons with a few muscular bundles attached to them. *These bundles, which were still mostly of a pale red colour and had a normal appearance, were no longer contractile.* Their transverse striæ were irregular or absent. There was no trace left of the pronator radii teres. In the deeper layers were found the remains of muscular bundles belonging to the superficial and deep flexors and to the pronator quadratus, and showing different degrees of discoloration from yellowish red to pale grey. All the muscles of the palm of the hand had reached the last stage of tissue change, except some muscular bundles of the thenar eminence which were still coloured and contractile. And thus, he says, "were I to describe each region of the body, it would be seen that alongside of changed and fatty muscles there were others whose fasciculi were still unchanged though much wasted. . . . I am anxious to state that the discovery of this fact in pathological anatomy belongs entirely to M. Cruveilhier. . . Another patient, named Legrand, died of this disease under Cruveilhier's care, and here again to the naked eye *many muscles were found entirely fatty.*"

In the case of Lecomte we found that after the wasting had reached a certain stage there appeared amongst the muscular bundles a greater or less number of granulations, which gradually obscured the striation and acquired the characters of fatty elements. It was the knowledge of this fact which decided me in giving this disease, which was the subject of my memoir at the Institute, the name of *muscular atrophy with fatty change.*

This change seemed to me to be characteristic of the most common form of progressive muscular atrophy. It is moreover incontestable, having been confirmed by pathologists whose names are an authority. The following passage shows that it was reported by Virchow himself.

"One of the objects submitted to us exhibits an interesting pathological change, viz., a piece of muscle of which one fasciculus shows progressive muscular atrophy. The degenerated bundle is smaller and narrower than the rest, and fatty globules are interposed with the longitudinal striæ. The wasting causes a diminution of the diameter of the primitive fasciculus. In proportion as the fat increases the contractile substance diminishes in volume. The contractile power of the muscle becomes less intense in proportion as the contents of the primitive bundles becomes less abundant, and in fatty atrophy *the fat replaces by degrees the contents of the muscular fibre.* The more fat, the less contractile substance. In a word, the contractile power of a muscle gets weaker in proportion as the normal contents of the primitive fasciculi diminish." (*Cellular Pathology*, 1861.)

M. Virchow was the first to observe and describe in progressive muscular atrophy an entirely different order of pathological phenomena. It is no longer a question of fatty changes in the contents of the primitive fibre, but of a series of transformations of the connective tissue between the fibres. Thus, M. Virchow admits two forms of this disease, the *parenchymatous* and the *interstitial.* In the former the primitive fibres, and in the latter the connective tissue between them undergoes a fatty degeneration. M. Virchow met with the two forms simultaneously in the case reported by him in 1855. For my own part I have noticed one or other of these two forms, and have often seen them associated.

[Sometimes the primitive fasciculi become *pitted* and granular without the granules assuming the evident characters of fat. This form of degeneration has been particularly insisted upon by M. Ch. Robin*; and M. Hayem† has described a fourth form of degeneration observed in this disease in which there is a considerable increase of the nuclei of the sarcolemma.]

* Ch. Robin, *Notes sur l'atrophie des éléments anatomiques* (Comptes Rendus des séances de la Société de Biologie, 1834 (?), L. 1, de la 2e série, pp. 5 et 6).

† Hayem, Note sur un cas d'atrophie musculaire progressive avec lésions de la moelle (*Arch. de Phys. Norm. et Path*, t. ii., 1869, p. 262).

Atrophy of the Anterior Roots of the Spinal Cord.—[In 1849 I formulated the following proposition: "Muscular atrophy with progressive fatty change may develop itself although the influx of nerve force be not hindered either by a lesion of the spinal marrow or by a destruction of the nerve roots." This was said on the strength of Cruveilhier's first autopsy, in which he declared that with the naked eye he had detected no appreciable lesion either of the nerve centre or its roots. In 1853, however, Cruveilhier announced that in the autopsy of Lecomte he had found *the anterior roots of the spinal cord considerably atrophied*. This fact discovered by Cruveilhier has been confirmed by a great number of observers.]

It is nevertheless proved that progressive muscular atrophy may exist without atrophy of the anterior roots. In support of this assertion I will recall the two first autopsies made by Cruveilhier, and a case which I found in 1860 under the care of Andral, and in which the autopsy was made by his then assistant, M. Axenfeld. This assertion rests also on the observations of German pathologists whose authority cannot be contested, among others of MM. Virchow, Oppenheim, and Hassé.

Atrophy of the Anterior Cells of the Spinal Cord.—The atrophy of the anterior spinal roots in progressive muscular atrophy is a secondary lesion. This idea must have occurred to every pathologist who has studied the pathogeny of this disease. But is this atrophy of the roots consecutive to a lesion of the cord, or to a primitive lesion of the muscles?

Although M. Cruveilhier had concluded, mainly from his researches, that the anatomical character of this disease was atrophy of the anterior nerve roots, he had nevertheless made a reserve, by a fetch of imagination, that one should look in the grey matter of the cord for the true origin of the atrophy of the roots.

For my own part, seeing that his researches had not established any appreciable change in the cord, and that the atrophy of the nerve roots was far from being constant, I had concluded that the muscular change was probably of peripheral origin, and that this constituted the essential point of the disease.

This opinion has been shared by other pathologists, first by Aran and then by Virchow, who has written, that "the disease is first fixed in the muscles, and then proceeds step by step to the centres."

I formerly held that the lesion was peripheral, although M. Luys believed that he had demonstrated, by means of microscopical examination of transverse sections of the spinal cord of a patient who had died of progressive muscular atrophy, that *the atrophy of the cells of the anterior cornua of the spinal cord constitutes the chief anatomical lesion of this disease.* [Luys recorded this case in the *Gazette Médicale* for 1860. It was that of a man aged 57 who had atrophy of the left hand and forearm, and who died of pneumonia. There was found in the spinal cord, at the level of the brachial plexus, atrophy of the left anterior nerve roots, great capillary engorgement of the corresponding parts of the cord, complete disappearance of the cells of the anterior cornua, and also of the cells in *the corresponding posterior regions.* Duchenne very properly remarks that in the absence of any details with regard to the exact symptoms of this patient he should hesitate to regard the case as one of progressive muscular atrophy, but should look upon it rather as one of "subacute spinal paralysis." The same remarks apply also to a similar case published by Valentiner, of Kiel, in 1855.

Later on Lockhart Clarke also described the existence of atrophy of the cells of the anterior cornua in this disease; but Duchenne says that of the three cases recorded by Clarke one was certainly a case of infantile atrophic paralysis, and another a case of subacute spinal paralysis, and he makes the same objection to two cases recorded by Charcot and Joffroy, in which atrophy of the cells of the anterior cornua was detected.]

The only *true* case of progressive muscular atrophy in which the spinal cord, after hardening and preparation, has been examined by most competent micrographers and pathologists (MM. Vulpian and Hayem), the only one for which I can vouch —having carefully observed it for more than a year, and photographed the transverse sections of the cord—is that which was published by Hayem, and to which I have already alluded when speaking of the pathological condition of the muscular fibres in this disease. I delayed giving the results of the microscopic examination of the spinal cord. The following is a *résumé* of it:—

"Atrophy or disappearance of a great number of the cells of the anterior cornua and normal vascularisation (*sic*) with

dilatation and sclerosis of the arterioles and chief capillaries are the main facts which, in this case, result from the histological examination of the cord.

"So far," says M. Hayem, "everything favours the belief that we must attribute the nutritive troubles of the muscular fibres to damage of the grey matter, and particularly to the concomitant atrophy of the nerve cells."

(I would here remark that, some time before, M. Charcot had already set forth in a demonstration at the Salpêtrière in June, 1868, identical views as to the part played by the change in the so-called motor nerve cells in progressive muscular atrophy, infantile paralysis, and central myelitis.)

Hayem also found the anterior roots of the second, third, fourth, and fifth cervical nerves excessively damaged, a large number of their nerve tubules being completely empty. This case proves, he adds with reason, that the muscular lesion in progressive muscular atrophy is not always absolutely the same, and that it has no pathognomonic signification.

In this case M. Hayem established the almost complete integrity of the grey and white matter in the dorsal region (Hayem, *Arch. de Phys.*, &c., No. 3, p. 264, 1869).

M. Hayem has described in three cases a more or less abundant exudation round the vessels. "Is this lesion," he asks, "the same as that found by Lockhart Clarke? Do the empty (*lacuneux*) spaces round the vessels represent what that author speaks of as *granular disintegration?* As yet we know nothing of it, but we are thus led to suppose that there exists in the cord a change which is peculiar to progressive muscular atrophy."

I would urge, in opposition to M. Hayem, that this same granular disintegration has been met with in diseases other than progressive muscular atrophy, *e.g.*, in tetanus and atrophic infantile paralysis; and it cannot therefore be looked upon as peculiar to progressive muscular atrophy.

Finally, in the preceding case, an increase in the connective-tissue elements accompanied the atrophy of the nerve-cells. "It is," says M. Hayem, "a condition comparable to the lesion of the nerve-tubules and interstitial tissue seen in sclerosis of the white substance of the cord."

This comparison does not seem to me warrantable. For, as a fact, the proliferation of the connective tissue is so great in

sclerosis that it seems to smother the nerve elements without, however, destroying them, whereas in *progressive muscular atrophy* it is the cells which are primarily atrophied and destroyed, and the increase of the interstitial tissue is infinitely less than in sclerosis.

Pathogeny.—*Is the disease peripheral in the first instance?* I was the first to maintain this hypothesis in 1849. It rested on the inconstancy of the atrophy of the anterior roots of the spinal cord, the only central lesion which had been found at the commencement of the study of the pathological anatomy of this disease. This opinion was shared by Aran and other pathologists, and by Virchow amongst others, who held that, the muscular atrophy being most marked in the extremities, and thence propagated to the other muscles, it might be inferred that the disease is situated at first in the muscles, whence it spreads gradually through the nerves, and finishes by reaching the cord itself (Virchow, *Arch. für Pathologische Anatomie*, t. vii. 537, 1855). The microscope, however, now shows us that the anatomical change is *primarily* spinal, and that the peripheral lesion is situated in the muscles which derive their nerves from the damaged region (the cells of the anterior cornua).

Is the disease consecutive to a lesion of the great sympathetic? This notion was first maintained by Schneevoogt in 1854, who reported a case of progressive muscular atrophy in which he found a fatty degeneration of the cervical and dorsal portions of the great sympathetic. This pathologist, whom I met at the Congress of Naturalists in Vienna in 1856, discussed this theory with me, and I then admitted the possibility of this pathogenic cause. In fact in preceding editions I have written, "this disease is probably peripheral, unless it depend upon 'damage to the ganglionic system.'" Since then four new cases of similar damage to the great sympathetic have been met with in progressive muscular atrophy, of which two were recorded by Jaccoud and two by Dumesnil, but in other cases the sympathetic, when examined by pathologists very familiar with this kind of microscopic research (Charcot, Vulpian, and Hayem, among others), has been found perfectly normal. Consequently the pathogeny of progressive muscular atrophy cannot be explained by a lesion of the great sympathetic. [Duchenne throws considerable doubts on the accuracy of Jaccoud's obser-

vation, and says that he was unable to detect any pathological conditions in the specimens of the sympathetic submitted by that pathologist.]

What is the nature of the atrophy of the cells?—The irritation which accompanies or produces the atrophy of the cells of the spinal cord in progressive muscular atrophy is shown by the hypertrophy of the vessels of the anterior cornua, by the thickening of their walls, and the increase of their nuclei. The production of granular bodies in the vascular lymphatic sheath is a consequence of this. In default of this proof of the inflammatory nature of the lesion I would point to the change in texture of the muscular fibres, the increase of the nuclei of the sarcolemma terminating in granulo-fatty degeneration. It is indeed established by experiment and clinical observation that the irritation which accompanies the atrophy of the anterior cells of the cord is necessary for the production of these changes in the muscles. It is this fact which accounts for those paralyses without complicating atrophy and muscular change, *e.g.*, glosso-labio-laryngeal paralysis (which forms the subject of another chapter), in which the atrophy of the cells is the primary change.

The cells of the anterior cornua of the cord are at once motor and trophic. The trophic function is alone affected in progressive muscular atrophy.

It was long since established by experiment and clinical observation that the anterior cells of the cord preside over motility. But in progressive muscular atrophy, the atrophy of these cells, of which the wasting of the anterior roots is a consequence, is intimately connected with wasting and alteration of the muscular tissue without accompanying paralysis; therefore these cells preside also over the nutrition of the muscles, and as a corollary from what goes before I conclude that they are at once motor and trophic.

It is not, indeed, for the sake of establishing this physiological proposition that I have formulated this argument. I would only state that without this physiological fact the pathogeny of progressive muscular atrophy is inexplicable, and that it would be necessary to have recourse to my first hypothesis of the peripheral origin of the disease.

The trophic faculty alone of these cells is affected in progressive muscular atrophy, while in atrophic spinal paralysis, whose

diagnosis from progressive muscular atrophy is discussed elsewhere, the motor and trophic faculties are damaged simultaneously. It has not as yet indeed been possible to discover any appreciable special change which might account for these pathological differences.

In short, there exists a class of muscular affections which are characterised by atrophy of the anterior spinal cells. These are easily distinguished one from another by their clinical signs, but afford no characteristic central change to aid in their differential diagnosis.

After this study of the pathogeny, the considerations which I have already been obliged to offer on the name of progressive muscular atrophy become superfluous.

Diagnosis.—[The diseases with which progressive muscular atrophy may be confounded are: 1. General paralysis of the insane. 2. "General spinal paralysis." 3. Atrophic paralysis of childhood. 4. A kind of muscular atrophy of the hand or upper limb from damage to the ulnar nerve, or occurring in chronic articular rheumatisms, and also in one form of elephantiasis. 5. Certain coincident muscular troubles.

Progressive muscular atrophy could be mistaken for general paralysis of the insane in those cases only where the articulation is affected. The great wasting of the tongue in such cases, and attention to the concomitant symptoms, ought to prevent mistakes.

A reference to the symptoms of *acute spinal paralysis in the child and the adult* will show that they are quite distinct from those of progressive muscular atrophy. But when these diseases have reached the stage of complete disappearance of muscle, and permanent consequent deformity, it may only be by a careful attention to the history of the case that a diagnosis is to be made.

Injury to the ulnar nerve will cause a deformity of the hand very like that which is seen in progressive muscular atrophy. It is known that the interossei and lumbricales receive their innervation from the ulnar nerve, except the *two first lumbricales*, which are supplied by the median. The action of the lumbricales and interossei being identical, it follows that, in injury to the ulnar nerve, the third and fourth fingers are more clawed than the first and second.

Rheumatism of the shoulder and consequent atrophy of the

deltoid may be mistaken for progressive muscular atrophy, and of this Duchenne recites an instance.

The clawing and wasting of the hand which is met with in certain cases of *chronic articular rheumatism* has certainly a great resemblance to the clawed hand of progressive muscular atrophy (see fig. 7). The pain and the swelling of the finger joints serve, however, to distinguish the rheumatic affection. "This chronic rheumatic atrophic clawing is caused by the change in the small joints and ligaments of this region."]

Is muscular atrophy of the hand peculiar to one form of elephantiasis?

I had the opportunity of seeing three cases of elephantiasis contracted in different parts of America. They presented the symptoms of that form of the disease called in Norway anæsthetic *Spedalskheld* by Danielssen and Boeck, as well as a muscular atrophy of the hand having some resemblance to that seen in progressive muscular atrophy, but differing from it in the contraction of the flexors of the fingers. This atrophy of the hand with curvature of the fingers and distortion of the joints co-existed with elephantiasis. It was no mere coincidence, but, indeed, one of the symptoms of the latter disease.

Since the time of *Rhases*, who called attention to the deformity of the hand seen in leprosy, many authors have described a special atrophy of the hand with deformity in elephantiasis. The following is the description of this deformity given by Danielssen and Boeck: "The fingers become successively inactive and bent; the back of the hand is flattened, the first phalanges are extended, and the others flexed, so that the hand becomes convex on the palmar and concave on the dorsal surface."

But do the muscles completely disappear in this atrophy of the hand? I have investigated this point during life in three cases by means of electrical testing, and will now show how this kind of atrophy differs from progressive muscular atrophy.

[The first case recorded by Duchenne is that of a young man who had a clawing and wasting of the hand of five years' duration, and *in whom he made a diagnosis of progressive muscular atrophy*. Six or eight months after this diagnosis unmistakable symptoms of leprosy showed themselves, and it then appeared that the initial symptoms of leprosy had been present before

the deformity of the hand, but had been concealed by the patient.

The second case was that of M. M., who suffered from articular rheumatism after bathing at Venezuela in 1851. At the end of three months he began to suffer from cutaneous eruptions characterised by red spots passing to a colour like *café au lait*. These recurred two or three times a year between 1851 and 1855. These spots became anæsthetic, and before long the backs of the hands and forearms had also lost their sensibility. Deep muscular pains, with contraction of the flexors of the fingers, accompanied by some fever usually preceded the appearance of the spots. The deformity of the hands began in 1852, and when seen by Duchenne the muscles of the thenar eminence and interosseal spaces had almost disappeared, and the hand was clawed and almost useless. The temperature of the hand was lowered, the skin thick and of an earthy tint, and the subcutaneous veins very thick. There was also a slight degree of retraction of the flexor profundus digitorum. The sensibility of the skin of the hand, of the deep parts, and of the pulp of the fingers was entirely abolished. The left hand was in a less advanced stage than the right. The third case, that of a woman aged 60, is in all essentials exactly like the preceding; there were red spots becoming pale and anæsthetic, and the atrophy and deformity of the hands was very marked indeed. One of these hands is shown in fig. 16.]

Fig. 16.

The three preceding cases seem to me to show that atrophy of the muscles of the hand is one of the symptoms observed in certain forms of elephantiasis or dry leprosy. In some cases, then, one might, as I have done, confound it with progressive muscular atrophy, which often begins in the muscles of the hand. Since, then, the signs furnished by the functional troubles of the hand are the same, and that muscular atrophy of the hand may be the precursor of leprosy, or may occur in the very first stage of that disease, it is of the first importance, when this condition of the hand is met with, to ascertain if the patient has

been in conditions favourable for leprosy, or if he has already presented those symptoms which I have recorded in the three cases mentioned above.

We should not, however, attach too much importance to any *one* of these symptoms taken singly. This cutaneous anæsthesia of the ends of the limbs, which is one of the characters of leprosy, is sometimes also met with in progressive muscular atrophy. I long ago saw a patient at the Charité, named Gervais, whose interossei muscles and those of the thenar eminence were in great part destroyed by progressive muscular atrophy. He had lost, like the patients I have just discussed, the sense of touch and pain. The strongest electric irritation of his skin caused no sensation in his upper limbs. I need scarcely add that this patient, a Parisian, was not leprous.

I would now direct attention to an appearance which seems to me to be one of the distinctive signs between leprosy and progressive muscular atrophy—I mean the contraction of the flexors of the fingers. This was noticed in the three preceding cases, especially in the last. Fig. 16 is from a photograph of the atrophied hand of a leprous patient in the St. Vincent ward of the Charité, while fig. 8 is from a photograph of the hand of Gervais mentioned above. By comparing these figures one sees that in the *leprous hand* the two last phalanges are in a state of extreme flexion and turned towards the thumb, while in figs. 7 and 8 the terminal phalanges are only semi-flexed. In the first (the leprous) case I found retraction of the superficial and deep flexors, which I have never met with in progressive muscular atrophy.

Dry or moist (with ulceration and caries) atrophy of the end phalanges, appearing sooner or later in leprous atrophy, is a sign which distinguishes the clawed hand of leprosy from that of progressive muscular atrophy.

Concomitant Muscular Troubles.—Diseases, as one knows, may co-exist and proceed in couples or in threes. I have noticed this in progressive muscular atrophy. Unfortunately, these complex and exceptional conditions but too frequently supply to opponents and cavillers the weapons wherewith to attack work which rests on long and numerous observations. And again, distinguished and serious observers, from want of a sufficient clinical experience of these same conditions, have

from time to time thrown everything into confusion by their inability to separate clear and well established facts from rare and complex conditions.

Those who wish to guard against similar errors must bear in mind that progressive muscular atrophy may be complicated by any one of the following diseases, or may itself complicate them.

These are, in their order of frequency: 1. Progressive locomotor ataxy; 2. Glosso-labio-laryngeal paralysis; 3. Sclerosis of the antero-lateral columns; 4. General paralysis of the insane. These coincident muscular troubles are now-a-days perfectly well known. The elements of their differential diagnosis cannot be confounded with those of progressive muscular atrophy.

NOTE.—*On the confusion of progressive muscular atrophy of children with infantile spinal paralysis.*

When in 1868 I wrote on the pathological physiology of pseudo-hypertrophic paralysis I showed that neither analogy nor any physiological fact could lead us to attribute this disease to any primitive lesion of the cord.

In infantile wasting paralysis, on the other hand, reasoning by analogy necessarily leads the observer to a primitive spinal lesion, which alone can account for the collective symptoms (paralysis of voluntary contractility, diminution or loss of electro-muscular contractility, and consecutive granular and fatty degeneration of the muscles).

That I have long held this opinion is shown by what I wrote in 1854: "Reasoning by analogy, I have been led to think that the starting point of these infantile paralyses must be in the spinal cord. In fact, in almost all the traumatic lesions of the cord which I have met with in the adult the symptoms are exactly those seen in infantile atrophic paralysis. In both paralysis marks the onset of the trouble; then, after a variable interval those muscles, that are in relation with the least affected areas of the cord recover their voluntary power and their nutrition, while those depending for their innervation on the more damaged parts atrophy or become fatty. It is difficult not to recognise in symptoms so similar the expression of an analogous lesion of the cord. The discussion of this point was rendered necessary by an opinion expressed by M. Bouchut (*Union Méd.*, 1867, Nos. 130, 131, and 134).

Resting on a case of infantile paralysis seen in his wards, in which no recognisable lesion of the cord was found post-mortem, and upon three borrowed cases (two from MM. Rilliet and Barthez, and the other from Edward Meryon), this pathologist holds, contrary to general opinion, that in this disease it is the *muscle trouble which causes the paralysis and the consecutive fatty atrophy*. He has consequently proposed to call it *myogenic paralysis*. The disease he likens to progressive muscular atrophy of infants and adults. Finally, he asserts that his view of the case is supported by certain facts, hitherto little known, in comparative pathology. In the horse, after long fatigue, it sometimes happens that the hind limbs cease to move freely; or, again, that a sudden paralysis may show itself in a horse that has worked the whole day long. In such cases the autopsy, made two or three days after the accident, has shown that *all the muscles of the paralysed hind quarters are yellowish, granular, and infiltrated with fat*, while those of the fore-quarters retain their natural healthy red colour; that the spinal cord is healthy, *and that muscular atrophy has not had time to be developed*. "It is incontestable," says he, "that these facts bear a strong analogy to fatty paralysis."

This question was discussed by the Société de Médecine de la Seine, on the 7th of March, 1868, and the following were the heads of my argument against this opinion of M. Bouchut:—

1. That in symptoms, course, and consecutive muscle-changes, infantile paralysis (*paralysie atrophique graisseuse de l'enfance*) and spinal paralysis of the adult bear a close resemblance to each other.

2. That, reasoning by analogy, it is rational to infer that there is the same primitive spinal lesion in both diseases.

3. That this hypothesis has been confirmed by the microscopic examination of the cords from patients with infantile paralysis (two reported in 1863 by MM. Bouvier and Roger, and the third by M. Cornil).

4. That of M. Bouchut's four cases which he brings in opposition to the three above mentioned, two (those of MM. Rilliet and Barthez, who, themselves, disown them) have no kind of bearing on the question at issue, while the third (Meryon's case) belongs to another "morbid species" (pseudo-hypertrophic paralysis), as I show elsewhere.

5. That the single pathological fact observed by M. Bouchut in his own wards, and which alone deserves to be considered, does not refute the three cases observed by MM. Roger, Cornil, Duchenne, and Laborde, because in this single and exceptional instance the primitive inflammatory lesion of the cord may well have disappeared after a lapse of years, as indeed sometimes occurs in the adult and in inflammatory primary lesions of the cord.

6. That comparative pathology, invoked by M. Bouchut in support of his opinion, is contrary to his doctrine, because in the particular case upon which he rests, the sudden paralysis (in the draught-horse after excessive work) which he attributes to a muscular lesion is, according to the opinion of the best observers, generally caused by a primary lesion of the cord or nerves. This was the result of a learned discussion of the subject by M. Henri Bouley at the Imperial and Central Society of Veterinary Medicine in 1865 (*Arch. gén. de Méd., Revue Vétérinaire de* 1864 *et* 1865, vol. xi. p. 78).

Prognosis.—The facts and arguments which I have brought forward, while dealing with the symptoms and course of progressive muscular atrophy, show how grave is the prognosis of this disease. In fact, when once the diagnosis is established one has always to fear, from the very beginning, either the loss of muscles essential for using the limbs, or the generalisation of the disease, and death by hunger or slow asphyxia, within a period which is sometimes not very long. Nevertheless the physician must not always look to this very gloomy side of the picture which I have given of this terrible disease.

It should be remembered that it does not always march relentlessly onward to a fatal termination, as might be inferred of any muscular affection to which the word *progressive* is applied. On the contrary, it may be arrested in its course. It sometimes remains localised in a certain number of muscles, without affecting those which are essential to life. Doubtless, if the muscles of breathing or swallowing are involved at the beginning, the disease must prove rapidly mortal, but happily in the numerous cases I have seen these muscles have usually been the last to suffer.

I can show that there is another hope to which one may cling in certain cases. It is known that in its destructive course this

disease abolishes, in succession, the use of the hand, and then of the upper limb, and that, finally, standing and walking become difficult. Such a prospect is a terrible one for the patients. I know many who, for years, have been condemned to live in this pitiable condition. However, I have noticed that, when the muscles of the trunk, many of which are of secondary utility, are attacked first, the muscles of the limbs, and especially those of the hands, suffer late, or escape altogether. For example, I have reported the case of a mechanic, a living skeleton (see figs. 9 and 10), who, although he had for years past lost most of the muscles of the trunk, had nevertheless retained intact the muscles of his upper limbs, which enabled him to follow his trade. When, therefore, the atrophy begins in the trunk-muscles, the prognosis appears to be less grave than when it begins in the hand.

I would like to add a few remarks which may assist the prognosis of this disease. I said, while treating of the causes of progressive muscular atrophy, that, often enough, the disease declares itself without any appreciable cause, and that consequently there exists for it, as for many other diseases, a "diathesis." The gravity of the prognosis evidently depends on the intensity of the diathesis. I think it is possible to tell under what circumstances to expect a slight or intense predisposition. I have noticed that when the disease develops without known cause, it usually becomes rapidly generalised and ends fatally; but if the atrophy has been provoked in workmen by over-work, and appears first in the most fatigued muscles, then it either remains localised in these muscles, or it progresses less rapidly, or it offers less resistance to the curative effect of localised faradisation. Does it not follow from these facts that in the latter case the intensity of the predisposition is less severe than in the former, and that without the determining cause—without, for example, the over-work—the atrophy would never have shown itself? I conclude therefore that the prognosis must be less grave when the atrophy first becomes localised in muscles which have been over-fatigued by excessive and too continuous work.

Treatment.—If electro-therapy cannot cure progressive muscular atrophy, can it not, at least, stop the invading course of the disease and improve the condition of the muscles? I will

answer this question by the therapeutic experience of twenty years. The indications which electro-therapy has to fulfil are to act, (1) peripherally on the muscular nutrition by localised electrisation; and (2) on the spinal cord, the starting point of the disease, by the aid of the reflex effects of continuous currents.

Treatment by localised faradisation.—As I originally thought that the damage to the muscular nutrition was the starting point of the disease, my efforts were for a long time limited to exciting the local circulation and the nutrition of the muscles by means of localised electrisation with intermitting currents. As I have not in practice found any appreciable difference in the results thus obtained with faradisation and intermittent galvanic currents I have given the preference to the former.

When we see the muscles of so many patients suffering from this disease, die, so to speak, one by one, in spite of all medicinal treatment are we not bound to accept in all its rigour the fatal name of *progressive* muscular atrophy? That at least was my opinion in 1848. I had formed this desperate prognosis by relying mainly on the history of a patient (*capitaine au long cours*) who was discussed in the first edition of this book. This man had the conviction that his disease would send him to his grave, and he spoke these sad words, which have remained engraved on my memory:—

"The very day I noticed," said he, "the commencing wasting of a part of my body I knew that I was lost, for three members of my family (a brother and two maternal uncles) had succumbed to a disease which began and progressed like mine. I have also two brothers who await a similar fate. It is a family complaint." These fears were but too well founded, for a few days later he died asphyxiated by the failure of his breathing muscles. By the side of this case I could range many others equally desperate which I have seen since then, and which almost discouraged me from making any attempts at electrical treatment. But happily the numerous cases I have collected since 1849 allow me to affirm, as I have stated above, that localised faradisation may sometimes call back the nutrition of muscles far advanced in atrophy, provided they be not as yet changed in texture.

I would here allude to the case of Bonnard, which has been quoted many times. (See figs. 9 and 10.) This case alone would be sufficient to prove the power of and the necessity for

the use of localised faradisation in the treatment of progressive muscular atrophy. When Bonnard came to consult me, his disease had invaded a great number of muscles. Some were either wasted or destroyed in varying degrees, while others which were still well developed were the seat of incessant fibrillary contractions. It certainly cannot be denied that the disease was on the high-road to become general. I confess that when I first saw him I thought him doomed to a speedy death, the more so that his diaphragm was affected. After this muscle, I said, the turn of the intercostals will come (most of the muscles of the trunk had already vanished), and then asphyxia will be inevitable. All this seemed to me certain. I then commenced treatment by faradism without the least hope, but nevertheless the results of it were most satisfactory. These were the stoppage of the progress of the disease, the re-establishment of the functions of the diaphragm and of the nutrition and power of one muscle (the biceps) which was essential for the use of the upper limb, the disappearance of the fibrillary contraction in the muscles already attacked by the disease, and finally the persistence of the cure after two years in spite of his resuming manual work. These were the incontestable results of treatment by faradisation.

It must not be concluded that patients whose progressive atrophy has been arrested in its course, and whose atrophied muscles have again developed under the influence of localised faradisation, can always expose themselves with impunity to those determining causes which have favoured the development of their atrophy, especially to muscular fatigue. I have shown, while treating of the causes, that exaggerated muscular action and especially prolonged contraction are the chief determining causes of progressive muscular atrophy.

The most important deduction to be drawn from this, in a therapeutic point of view, is the advisability of counselling the patient, whose condition one has had the happiness to relieve, the necessity of renouncing the exercise of his profession when once its influence has clearly been established to be harmful. But the workman who has no other resources but his trade is not always able to follow this good advice, although he may well understand its importance. Driven by necessity, he abandons his treatment and resumes his occupation as soon

as he feels strong enough to do so. This is the chief cause of the numerous relapses from which the unhappy patients suffer, and of the difficulty of getting a complete cure. It happened to a man named Goulard, whose case I reported in the last edition as a remarkable instance of cure by localised faradisation, and of relapse caused by the muscular fatigues inseparable from the occupation of a market porter. The faulty attitude of his body while standing after the atrophy of his erectores spinæ muscles is shown in fig. 12.

I have shown, it will be remembered, that progressive muscular atrophy is not a paralysis; and that when treating of the name of this "morbid species" it followed from the facts and considerations given, that to call it "atrophic paralysis" would not only give a completely inexact idea of its nature and real symptoms, but might give the physician an idea of false security as regards the muscles whose existence was threatened, and would only attract his attention to this disease at a time when every chance of therapeutic success had vanished. In fact the belief that paralysis is the primary symptom, a belief which would be fostered by the false name of atrophic paralysis, would lead him naturally to direct his attention only to those muscles whose functions are weakened, or in other words to those muscles which have already reached the last limits of atrophy and tissue change. If, on the other hand, the physician is forewarned that, in the patient who comes to him merely in the hope of recovering the use of certain muscles which are powerless or enfeebled, the other muscles also whose movement and force appear intact are threatened with extinction as soon as they begin to atrophy and become the seat of fibrillary contraction, he surely will not delay the treatment of these muscles until their function is abolished or their texture altered. It will then be possible for him, by timely intervention, to stop the invasion of the disease.

I have long held that localised faradisation, properly applied, re-forms—if I may be allowed the expression—the fibre in the atrophied muscle. Nothing is more clear. Does it not follow, in fact, from microscopic examination that in this disease the quantity of fibres in the atrophied muscle is diminished, *i.e.*, that many of them have completely disappeared? If, then, an atrophied muscle enlarges under localised faradisation, one may strictly say that faradisation increases the number of its fibres,

or in other words, that it causes actual repair. Indeed, this happens often enough when localised faradisation is used in the treatment of progressive muscular atrophy.

Up to the present time I have never seen any muscle-growth occur in places where absence of electro-muscular contraction showed the tissue to be destroyed; but wherever a few contracting fibres have remained they have often become, as it were, nuclei or centres for other fibres whose size has increased, and whose power has proportionally grown in response to localised faradisation.

The question of treatment is intimately connected with that of prognosis, to which I may be allowed to return. The cases reported (which establish incontestably that the invasion of progressive muscular atrophy may be stopped, and that muscular fibres in great part destroyed may recover) serve to modify, I hope, in a favourable manner, the prognosis of this disease which there has been too great a tendency to place in the same category with "general progressive paralysis," from the point of view of incurability. Nevertheless in these cases the physician must give a most guarded prognosis, and must bear in mind those sad cases quoted at the beginning of this article, from which it would seem that progressive muscular atrophy, in spite of all treatment, marches ever towards a fatal termination.

Central anatomical conditions which allow localised faradisation to exercise a favourable influence on muscular atrophy.—Microscopic examination of transverse sections of cords from patients who suffered from muscular atrophy secondary to lesions of the anterior cornua (acute and subacute anterior spinal paralysis and progressive muscular atrophy) show that the anterior cells are damaged in different degrees. The muscle change then is in direct proportion to the degree of damage to these cells. It is plain that in those cases of progressive atrophy in which all the anterior cells are completely destroyed, and in which the muscular tissue must sooner or later become deeply altered, faradisation of the muscles can do no good. But it is not so in the cases, frequent enough, where hard work, or such as has demanded prolonged muscular contraction for months or years, has been the determining cause of the atrophy. My observations have led me to believe that in such cases the muscular atrophy would not have appeared without the determining

cause. These are the cases in which localised faradisation, favoured by rest, has been able to restore the nutrition of atrophied muscles, or to stop the progress and extension of the disease. These are the cases in which probably but few cells are deeply altered, and the change, for the most part, but slight. As long as I believed in the peripheral origin of progressive muscular atrophy, the therapeutic use of localised faradisation seemed to me most rational, because it rouses the local circulation and restores the nutrition of the muscle when the tissue change is not too far advanced. I thought that it could check the wasting in its course from without inwards. But knowing now that wasting of the anterior cells of the cord is the primary lesion in progressive muscular atrophy, it is evident that localised faradisation of the muscles merely copes with the chief symptom of this damage, the lesion of muscular nutrition.

Modes of procedure.—The chief indication to be fulfilled by localised electrisation in progressive muscular atrophy is, as I have said, to stimulate the local circulation and the nutrition of the muscle. The reader must recall those electro-physiological considerations which I have elsewhere dwelt upon, to show the power of localised faradisation on the vasomotor nerves and on trophic nerves. He will apply to the peripheral treatment of progressive muscular atrophy the therapeutic deductions which I have drawn from them. I would remark that the electro-therapeutic precepts which I have formulated have been established by numerous researches and clinical experiments made empirically at first, and that the electro-physiological considerations which I refer to are merely intended to support these precepts as much as possible by scientific facts, or in other words to make the application of localised faradisation more rational. The following is a summary of the modes of procedure which I advise:—

(*a*) *To move the moist rheophores, held as closely to each other as possible, over the surface of each of the affected muscles, with an induction current of greater or less intensity, so as to stimulate all the anatomical elements which enter into the composition of the muscle.*—In order to restrict the stimulation to the upper layers I generally use the current of the primary coil (the "*extra current*," the tension of which is weak); while the current of the secondary coil (the *induced* current, the tension

of which is relatively greater) serves to stimulate deeper layers.

(*b*) *To stimulate the muscles moderately, and to use a current with long intermissions.*—This precept rests on this observation which I have often made in progressive muscular atrophy, that faradisation used for too long a time, or too strongly, or with too rapid intermissions, produces fatigue or cramp, and often notably increases the wasting. Further, it results, it will be remembered, from my experiments, that strong induction currents paralyse the vasomotor nerves.

(*c*) *To faradise those muscles only which still respond to electric stimulation, and by preference those which are most necessary for the use of the limbs; and finally to faradise lightly the most important among those muscles which are threatened by the invasion of the atrophy.*—The following are the reasons for these rules: 1. As I have never seen, in this disease, an atrophied muscle recover its nutrition or motility after response to electric excitation has ceased, I have concluded from this fact that it is, so to say, dead, and have given up trying to excite it. 2. It is important to bear in mind which are the muscles most necessary for the functions of the limbs. This I have tried to demonstrate in my electro-physiological researches (*Physiologie des mouvements*, 1866). As an example I will allude to the case of Bonnard, mentioned above. I devoted most attention to his diaphragm (the most useful of the respiratory muscles which was already beginning to fail); then to the deltoid, biceps, and brachialis anticus on the right side (muscles essential for the use of the arm); while I neglected other affected muscles of the trunk, because they are of secondary importance (pectorales, latissimi, trapezii.)

The "atrophic" patients under treatment in my private practice generally serve me for demonstrating the action of those muscles which are not yet changed, or but little affected by the disease. For example, Bonnard has served me for many years to show the proper action of the diaphragm, intercostals, serratus magnus, rhomboidei, &c. Having noticed that in a certain number of those who had most often submitted to this kind of experiment, the progress of the muscular atrophy was arrested, I was careful to faradise those muscles whose nutrition was menaced, devoting my chief attention to the most indispensable

muscles. This practice has succeeded perfectly in my hands, and I advise others to follow it.

Treatment by the Continuous Current.—In presence of the cases given above, none can doubt the value and necessity of localised faradisation in this disease; but it must be admitted that this method of peripheral electrisation treats merely the chief symptom of the disease, and that we must discover a means of attacking the point of origin of the disease, *i.e.*, the anterior spinal lesion.

Electrisation by reflex action is, as I have shown, a sure method of making electric stimulation reach the spinal cord. Since the commencement of my investigations I have, often enough, in the treatment of progressive muscular atrophy, tried this reflex method of electrisation by *intermittent currents* (galvanic or faradic), but I was obliged to renounce it, because the sittings were usually followed by cramp, and the atrophy, so far from being improved, was aggravated by it.

The use of continuous currents in the treatment of progressive muscular atrophy has been extolled of late years by Remak and his followers. I have already said that, since 1860, I have used, without appreciable results up to the present time, the different procedures (galvanisation by stabile and labile currents) indicated by them. It has indeed happened that, after applying the continuous current to cases of progressive muscular atrophy for twenty sittings without the least result, I have been able to restore the utility of certain muscles essential for breathing or the movement of the limbs by means of localised faradisation.

However, since it has been proved that continuous currents have the advantage of causing neither cramp nor fatigue, and that their electrolytic action has merely the inconvenience of causing burning and blisters, I usually associate it with localised faradisation and employ them alternately. This mixed treatment of progressive muscular atrophy is rational, because it seems to me possible that in certain cases it may produce in the long run more complete results than the use of localised muscular faradisation alone.

I pass by in silence the pretended cures of progressive muscular atrophy by continuous currents, because in the histories given by the authors of the cases I recognise errors in diagnosis. These cases of cure belonged to that kind of progressive pseudo-atrophy

which I have described under the name of *subacute general spinal paralysis*, which, as I shall show, usually gets well of itself, and which, in consequence, could get well while continuous currents were being applied, as happened in the cases recorded.

I will say nothing of the different internal medicines which I have used (nitrate of silver, preparations of arsenic and of phosphorus), the therapeutic action of which has not been very appreciable.

[The ætiology of progressive muscular atrophy is made the subject of a short appendix to the last edition of the *Electrisation Localisée*.]

[It is most common in males. It is common to all climates and countries, but Duchenne's cases were mostly Parisian.]

I have sometimes known cold and damp assigned as causes; but over-work is the most common cause among the working-classes.

These causes (to which others, such as mental worry, social reverses, masturbation, venery, &c., may be added) are only occasional.

[In a great number of cases no cause is discoverable.]

Heredity is a cause in some cases.

CHAPTER III.

INFANTILE ATROPHIC PARALYSIS.*

THE disease forming the subject of this chapter is chiefly characterised by—

1. Muscular paralysis occurring suddenly in infancy, usually without known cause, with fever, but sometimes without appreciable fever, with more or less diminution of electric contractility proportionate to the extent of the spinal lesion.

2. Simple atrophy of the paralysed muscles, and change in texture (granular and granulo-fatty degeneration) of those which are completely deprived of central nervous influx.

3. Slow secondary tonic retraction of those muscles whose antagonists are atrophied and degenerated, and consequent deformities from changes in the position of limbs (club-feet, &c.).

4. Atrophy of those parts of the skeleton whose innervation is defective.

5. Destruction of the cells of the anterior horns of the cord.

I call this disease "*infantile atrophic paralysis*" (*paralysie atrophique de l'enfance*). If in the first memoir which I devoted to this disease in 1855, I gave it this name (to which I added the epithet "fatty," which I now renounce, because it makes too long a title) it was because it had been described under vague names which did not establish any well-marked distinctions between it and other muscular affections of childhood.

During the last twenty years or more, I have seen many hundreds of cases of this disease. Now that its characteristics are known, it has become more common than when I began my researches; not a week passes in fact without meeting with fresh cases of it. All these cases conform to the summary description which I originally gave, and justify the title which I propose to retain.

This name is practical, because it recalls the chief clinical features of the disease, paralysis, atrophy, muscle-change (this last can, if necessary, be demonstrated in the living by the aid

* From *L'Electrisation Localisée*, 3rd ed., pp. 381—437.

of my tissue-punch, which is described elsewhere). It is more accurate than the name *infantile spinal paralysis*, because I have discovered two other infantile muscular affections, *infantile progressive muscular fatty atrophy*, the central lesion of which is also spinal and affects the anterior cells of the cord, and *pseudo-hypertrophic paralysis*, the central lesion of which has not yet been discovered, but which possibly is also spinal. If I were to give this disease an anatomical name I should call it *acute paralysis of childhood from atrophy of the anterior spinal cells*.

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[According to Duchenne, Underwood (1784) merely foreshadowed this disease. Rilliet (1851) gave it the name of *essential paralysis of childhood.*]

Typical case. Case No. 11.—Maurice X., living at No. 5, Rue Ventadour. Æt. 9, of delicate constitution, though usually enjoying good health; suffered from whooping cough when 4 years old, but without any grave sequelæ. He was not liable to ephemeral febrile attacks, had never suffered from worms nor had he experienced any teething troubles. When thirteen months old, in February, 1846, he had a severe fever, lasting three days, without eruption, convulsions, cramps, or any derangement of the chest or belly. He was comatose during the whole course of the fever, so that it was not perceived until the end of three days that he was completely paralysed, being unable to raise his head or to sit up. It was doubtful at the time whether this was due to weakness or to paralysis. Six weeks before his illness this child could walk alone, but a month or two afterwards, notwithstanding the recovery of his strength by good feeding, he not only could neither stand nor sit, but could not move his arms. He was evidently stricken with a general paralysis. As movement did not return, flying blisters, sprinkled with strychnine, were applied along the spine, and then friction with an ammoniacal oil was resorted to. In other respects his condition was satisfactory, and he had begun to eat as soon as the fever left him. After five or six months of treatment the child began to move his upper limbs, held up his head, and could sit up. In 1847 M. Trousseau prescribed strychnine, which caused general shocks. After five or six months of this treatment the movement of the upper limbs and trunk had completely returned, but the paralysis was persistent in the lower limbs. Two or three months after the onset of this general paralysis the mother noticed a considerable wasting of the lower limbs, especially the right, although the upper limbs preserved their normal volume. This wasting steadily progressed. Before the illness the feet were not deformed, neither splayed nor turned in, but a year after the onset the right foot was twisted outwards and the left inwards. At the same time the feet got shorter, as the mother said, the dorsum projecting and the sole being too much hollowed. This deformity was allowed to increase for a year without restraint. It was only in 1850 (four years after the onset) that

an orthopedic apparatus was employed to stop the bending of the feet. This was used for seven months, but appeared only to act slightly on the left foot. This local improvement had no effect on the paralysis of the lower limbs. Nevertheless under the influence of general means employed uninterruptedly (baths of Barèges, rubbing with oil and stimulating ointments) the left thigh improved a little, and the child could flex the left hip slightly. The intelligence was normal, and after the cessation of the fever the little patient was as merry as ever. There was never any trouble with the bladder or rectum after the initial fever.

In 1852, when the child was sent to me, its condition was as follows:—The left thigh much stronger than the right. The legs equally shrunken. Talipes varus (hollow foot twisted inwards) on the left side, Talipes valgus (direct hollow foot) on the right. On both sides the heel is depressed, and cannot be raised by any extension of the ankle; considerable arching of the right foot; the near phalanges are bent backwards on the metatarsal bones, and the two end ones flexed. On the left side there is the same "clawed" position of the toes. There is no power of extending the knee or of flexing the hips, except to a slight extent on the left side. Electro-muscular contraction present in the muscles of the left thigh, and feeble in the flexors of the right knee and adductors of the right thigh. All the muscles of the leg contract, although much atrophied, except the right gastrocnemius, tibialis anticus and tibialis posticus (*jambier ant et post*) and the left peronei.

In the case just related we have the series of symptoms which characterise this kind of paralysis in its most usual form, and the course which it follows at different periods. From the very beginning this child lost all movements. This was attributed to the intense fever, and it was only when the fever subsided that the paralysis was noticed, so that the exact time of the onset of the paralysis is not known. Towards the end of the sixth month movement began to return in the upper limbs, neck, and trunk, but the recovery was not complete till after five months of treatment with strychnine. The paralysis persists in many of the muscles of the lower limbs.

In spite of their prolonged paralysis the upper limbs are not wasted, their muscles are intact, and their bones (unlike those

of the lower limbs) have suffered no arrest of development. This fact proves that something more than want of movement is necessary to cause a great nutrition-trouble, and that to produce it a profound nervous lesion is necessary, such as one sees the effects of in the lower limbs. By electro-muscular exploration I found some of these muscles still irritable, although much wasted, others retaining only a few fibres, and others giving no signs of life. The deformity of the feet and toes is due to the unopposed action of certain muscles (those which are least atrophied).

In fact the lesion of nutrition is plainly shown by an arrest of development in the bones of the leg and foot, the innervation of which is most seriously damaged.

These signs of disease, which have successively developed in this child (paralysis, wasting, probably change of muscle-texture, deformity in consequence of the unequal affection of the muscles, or because of return of movement in the muscles least affected), have presented themselves in a similar way in the other cases of this disease which I have seen.

The preceding case does not, of course, suffice to show all the varied forms which this disease assumes after becoming localised, when the paralysis and muscle-wasting cause a great variety of functional motor troubles and deformities, especially club-feet.

[Cases occur in which the paralysis affects all four limbs, or it may be limited to the upper limbs or to the lower. The extent to which the limbs are affected varies very much.]

Case No. 12.—Eugène Sivry, æt. 11. When 3 years old without known cause he was convulsed and insensible for three hours. The parents did not notice any fever. After the convulsions he recovered his spirits and appetite, but could not move his legs nor sit up. He complained of no pain. A month later he could sit up, and straighten himself when he bent forward, but his lower limbs remained paralysed, and then rapidly wasted. Active treatment was pursued from the outset—blisters to the spine, strychnine, sulphurous baths—but nothing was of any use. The bladder and rectum were unaffected.

In 1858, eight years after the onset, he was brought to me, and his condition was then as follows:—The skin of the legs was literally lying upon the bones, and no flesh was recognisable by pinching. Electric irritability was absent, except in the

right (*sic*) tensor vaginæ femoris. No movement of legs, feet, or toes. He could flex his left hip slightly when he lay on the right side, but when sitting this power of movement was unable to overcome the weight of the leg. With the hip flexed there was no power of extension. On the right side the movements of the thigh are nil. A band formed by the tensor vaginæ of the left (*sic*) side opposes the complete extension of the thigh, and when an attempt is made to overcome this resistance, the pelvis rotates backwards on the spinal column so as to produce a marked "saddle-back." When the child is held upright the three segments of the lower limbs are flexed upon each other as in fig. 17, in which he is represented as clinging to his mother's neck to prevent himself from falling. Nevertheless a few months after the onset of his attack, this child managed to move about by the aid of his hands, dragging his hind-quarters after him like a cripple without legs. But he soon devised a more convenient and quicker method of using his lower limbs. Bending himself, as shown in fig. 18, he seized his feet with his hands and carried them forward one after the other, thus imitating a walking pace. He soon became so clever at this exercise that his locomotion was quicker with it than by dragging himself. He could even go upstairs. The first time I saw him he had walked in this way

Fig. 17.

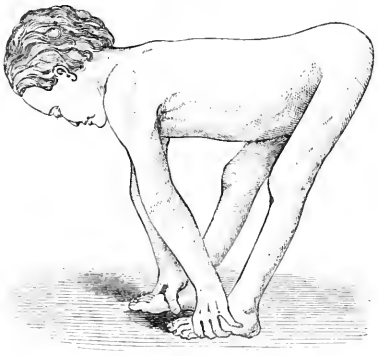

Fig. 18.

for more than six years. The ligaments of the elbow, too weak at that age to support the whole weight of the body all at once, had become stretched on the aspect of flexion, so that the forearm and arm formed an obtuse angle backwards during extension when he assumed the attitude which I have described (see fig. 18).

To complete the relation of the chief facts of this disease in all its varied forms, allusion must be made to cases in which the paralysis affects a single limb, or perhaps a single muscle in a limb. The tibialis anticus, or the triceps muscle of the calf, are often affected in this way, and their degeneration causes special varieties of club-foot, which necessitate the wearing of orthopedic apparatus night and day. I could mention cases also in which the paralysis has affected the arm or the leg either in a "hemiplegic" or a "crossed" fashion.

My description of the disease is drawn exclusively from my own experience, now extending over more than twenty years, and from my own naked eye and microscopic examinations.

Symptoms.—1. *Phenomena of onset.*

(*a*) *The initial fever is usually but not invariably present.*—M. H. Roger and M. Laborde have stated that the initial fever is invariably present.

As this form of paralysis appears to every attentive observer to consist primarily of an acute myelitis, situated, as I shall show hereafter, in the front horns, it is hard to conceive it starting without febrile reaction. Nevertheless I have opposed to the rational opinion of M. Roger certain clinical facts which have shown me incontestably that this paralysis sometimes occurs in infants without any appreciable fever, making its appearance in the daytime, and while they are in their mothers' arms. This statement is corroborated, amongst others, by the following case:—

Case No. 13.—In 1867 I saw, in consultation with MM. Nelaton and Oliff, a young English girl, æt. 8, who was suffering from extensive atrophic paralysis of some years' duration. She was of good constitution, and had never ailed except with infantile febrile attacks, occasioned by dentition. One morning when she was about 2 years old, without known cause, and about an hour after breakfast, she suffered from lassitude followed by an inability to

stand. The mother finding no sign of fever about her called in Mr. Paget, the celebrated London surgeon, who, finding the child free from fever, pain, or convulsions, reassured the family. Nevertheless the weakness of the legs went on increasing, and towards evening the child lay in bed without power of movement of any kind. There had been neither fever nor any kind of complaint, and the intelligence and sensibility of the skin remained unaffected. This condition lasted for a couple of months, and then, in spite of active treatment by many celebrated London doctors who met in consultation (blisters to the spine, strychnine internally, frictions, and shampooing), the lower limbs and the right upper limb began to waste rapidly. Such was the story told me by the mother of this patient when I first saw her, four years after the onset of the disease. At this time I found destruction of the muscles of the lower limbs, except parts of those moving the thigh on the pelvis and the foot on the leg. Two kinds of club-foot had been produced (right equino-varus and left valgus). The muscles of the left upper limb and of the trunk and neck had recovered. The bowel and bladder were acting healthily.

I have seen other children who, according to their parents, have been stricken with this kind of paralysis in the day-time, when in robust health, without the least sign of fever or increase of temperature, without any pain, or even an interruption in the act of suckling. Sometimes it comes on in the middle of their games. In short, it must be admitted that the fever may be entirely absent.

(*b*) *Duration of the fever.*—This is most variable, as shown by the following table made by my son from cases collected in my practice. Out of 70 cases

The fever was absent in	7 cases	lasted 5 days	in 5 cases
lasted 1 hour	,, 1 case	8 ,,	,, 3 ,,
2 hours	,, 1 ,,	10 ,,	,, 1 case
"a night"	,, 11 cases	11 ,,	,, 2 cases
"2 or 3 days"	,, 6 ,,	15 ,,	,, 1 case
4 days	,, 7 ,,	(?) ,,	,, 25 cases

I have noticed that the duration and intensity of the fever both diminish in proportion to the youth of the patient. Finally, I

have seen this paralysis follow an eruptive fever, typhoid fever and an intermittent fever.

This paralysis usually occurs at an age when the patients are too young to give an account of their symptoms. Patients who have been attacked at the age of 3 or 4 have sometimes complained during the acute stage of severe pains in the limbs, usually increased by movement.

Convulsions not unfrequently precede or accompany the fever. My son records 13 instances out of 70 cases. Once they preceded the paralysis without any fever. They have no special characters.

2. *Paralysis and Atrophy.*—No matter whether the paralysis be or be not preceded by the usual initial symptoms, it generally attains its maximum from the first. More rarely it begins with a weakness and reaches its maximum gradually. Thus a child after the initial fever executed all its movements naturally; then there was a difficulty in moving one or other of its limbs, and in two or three weeks they became absolutely inert.

Sometimes also, but still more rarely, I have seen the paralysis relapse after recovery. M. Duchenne fils has reported two such cases from my practice. In three cases I have seen a paralysis of the tibialis anticus follow, at an interval of several years, a similar paralysis of the corresponding muscle.

The following table shows the different forms which this paralysis may assume :—

In 62 cases there were—
5 of general paralysis
9 ,, paraplegia
1 ,, hemiplegia
2 ,, crossed paralysis
25 ,, paralysis of right leg
7 ,, paralysis of left leg
10 ,, paralysis of right or left arm
2 ,, lateral paralysis of the upper limb (*paralysies latérales du membre supérieur*)
1 ,, paralysis of trunk and abdomen (Duchenne fils).

(*a*) *Loss of electro-muscular contractility.* — The earliest electrical examination I have made was on the third day.

Case No. 14.—A child 10 months old was brought to me in 1863, three days after the onset of the disease. There had been two days of fever and some vomiting. The mother noticed on the night of the third day that the right arm was useless. On the following day I found the arm quite deprived of movement, but it was only on the fifth day that I found the electro-contractility of the deltoid weakened as compared with the corresponding muscle, notwithstanding that it remained normal in the other muscles of the limb, and that the muscles of the hand had already begun to recover. Blisters were applied to the cervical spine, and faradisation of the deltoid was immediately commenced. On the seventh day the electro-contractility of the deltoid was lost, and in a month its form had disappeared, the skin seeming to be in immediate contact with the shoulder joint. After ten applications of faradisation (twice weekly) the arm and forearm, which had wasted, were nearly their normal size, and the movements of the forearm and hand seemed to be as forcible as those on the opposite side, but the shoulder remained wasted and the elevation of the arm was completely lost.

In this case, then, where the central lesion had completely deprived the deltoid of innervation, the electro-muscular contractility of that muscle was not completely abolished until the seventh day.

I have often established this same fact in other muscles; and when the paralysed muscles retain their electro-muscular contractility after the seventh or eighth day I have always seen them recover their motility, the rapidity of recovery being proportional to the slight impairment of irritability.

(*b*) *Atrophy of the paralysed muscles.*—The wasting in this disease progresses very rapidly, more rapidly than that produced by traumatic lesions of mixed nerves in the adult. It is proportionate to the loss of electro-muscular contractility, or in other words to the nerve-lesion. When the parents have noticed wasting specially pronounced in certain regions a few days after the onset of the disease, I have established the fact, later, that in these regions electro-muscular contractility was lost or lessened.

[Duchenne believes that the degeneration of the muscle follows close upon the abolition of its electro-muscular contractility, but does not precede it.]

(*c*) *Temperature of the paralysed limbs.*—Atrophy so rapid and severe, affecting as we shall see even the osseous system, indicates a severe damage to the vaso-motor nervous system of the local trophic circulations. The temperature of the paralysed limbs is a sure sign of this. I have often asked those who were present at the invasion of the disease if the paralysed limbs were warmer than the others, and all have replied negatively. This question must be reserved, and must be studied by physicians themselves. As for the lowering of the temperature in the atrophied limbs, I have myself established its existence on the fourth or fifth day when the nervous lesion was severe.

The second stage of the disease is marked by return of voluntary movement, electric contractility and tonic power in certain muscles, by granular, fatty granular, or fatty degeneration of others, by atrophy of the bones, by the retraction of muscles whose tonic power is not balanced by antagonists, and by consequent deformities and varieties of club-foot.

The moment when the unchanged muscular tissue recovers its voluntary power marks the termination of the paralytic period. The duration of this period is very variable, for in the same limb may be seen muscles which have recovered in a few days or weeks, along side others which have, on the contrary, remained paralysed for many months, or even more than a year.

I have shown that in the paralyses from injury of mixed nerves, voluntary movement returns before electro-muscular contractility (as is also the case in lead palsies). I have not seen this occur once in more than 300 cases of infantile atrophic paralysis. I have not once seen a muscle, the contractility of which had been more or less weakened, fail to recover its electric as quickly as its voluntary contractility. The nutrition of muscles which thus recover improves, and they increase in size. The muscles, on the other hand, which remain paralysed waste more and more, and the degenerative process which had attacked some of them continues to spread. The degree of wasting and degeneration of these muscles can be estimated by the thickness of the cellulo-fatty tissue.

The nutrition of the bones of the paralysed limbs suffers in various degrees. The bones waste in all directions. I have seen, for example, long bones shortened to the extent of 2, 6, or 8 centimetres in a few years. The bone-wasting and the paralysis are not always proportionate to each other. Thus a limb may lose the greater part of its muscles and be shortened only to the extent of 2 or 3 centimetres, while another limb may be shortened to the extent of 5 or 6 centimetres, although the muscular lesion may be limited to two or three muscles. . . .

The functions of the intestine and bladder always remain intact in this disease during all its stages, no matter how extensive the paralysis may be.

Pathological Anatomy.—*Spinal cord.*—[Reasoning from analogy, and from the similarity in the symptoms of this disease with those often seen in wounds of the spinal cord, Duchenne stated his opinion as early as 1855 that the spinal cord was the seat of the lesion in infantile atrophic paralysis. MM. Barthez and Rilliet were unable to find any *naked eye* change in the spinal cord of a little girl who died of pneumonia, a month after the onset of "essential paralysis." In 1864 M. Cornil examined the body of a woman æt. 49 who had been stricken with infantile wasting paralysis (of the lower limbs) when two years old, and found—1. Complete fatty degeneration of the muscles; 2. Fatty degeneration of the nerves with wasting of the nerve tubes; 3. Wasting of the antero-posterior (*sic*) fasciculi of the cord with formation of amyloid bodies in all its length.

In 1867 L. Clarke (*Med. Chi. Trans.*, 1867) published under the name of "muscular atrophy" a case, presumably of this disease, in which wasting of the cells of the front horns of the cord was found together with granular degeneration of the grey matter.

MM. Charcot and Joffroy published the case of a boy aged 19 who had suffered from infantile wasting paralysis, attacking all four limbs, but persistent in the lower since the age of 7, and who died of consumption in 1866. After giving minute details of the changes seen in different regions of the spinal cord they say:—

"These lesions began to be appreciable at the upper end of the cervical enlargement, where there was merely a little

diminution in the thickness of the left front horn. Between this and the dorsal region the change was well marked, as also in the dorsal region itself, and throughout the length of the lumbar enlargement where it was more marked than in the cervical region."

The front horns of grey matter were chiefly affected, although the back horns were sometimes affected also.

Attentive observation showed that groups of nerve-cells had disappeared and had been replaced by a granular, fibrillated transparent substance, or by a thick opaque network of interlacing fibres. . . . The anterior nerve roots were also wasted, especially those corresponding to the damaged front horns. The white matter was much less changed than the grey. The posterior columns offered no marked change. The antero-lateral columns were obviously wasted, especially in the cervical and lumbar enlargements, and that on the side where the grey matter was most changed. They were not only wasted, but the neuroglia was much thickened, forming coarse fibres and trabeculæ especially near the wasted front horns.

The most damaged parts of this cord were precisely those which innervated the most wasted limb.

"The changes just described are evidently merely the last traces of a pathological process, the activity of which has been long established," say MM. Charcot and Joffroy. "What has been the beginning and the nature of the disease-process which has brought about these changes? This can hardly be determined from a single specimen." From the suddenness of onset during life one might be led to admit the previous existence of a medullary hæmorrhage or a central softening of the cord. Yet we believe that nothing of the kind has occurred. Nowhere in the cord are found either vacant spaces or any remains of an ochrey focus or a scar.

. . . . "The trouble has systematically implicated from the first the front horns of the grey matter, and it has only spread secondarily to the back horns and the white columns. But which elements in the front grey horns have been first attacked? Was it the neuroglia or the motor nerve cells? The second hypothesis seems to us to be the most likely."

These pathologists had already made the same hypothesis

concerning progressive muscular atrophy. When reproducing it *apropos* of a case of infantile paralysis they say:—

"A fact, moreover, which specially supports this view, is that in certain regions of the cord the disappearance of one or more groups of these cells, with the accompanying consequent atrophy of the front nerve roots, is the only change recognisable with the microscope."

. . . . These facts were confirmed by another case reported by MM. Parrot and Joffroy, in which there was almost complete paralysis of the left leg, and partial paralysis of the right, in a boy aged 3 who died of measles. Transverse sections of the cord, hardened in chromic acid, showed in the lower lumbar region, close to the "filum terminale," an almost complete absence of the cells of the left front grey horn, while those remaining were deformed. The cells were absent in the posterior two-thirds of the right front horn, and present in normal quantities in the anterior third, but partly deformed and without nuclei or prolongations.

At the level of the altered region, the vessels were more numerous, bigger, and notably changed within the front horns. The change in the calibre of the capillaries of a certain size, caused probably by inflammatory action, was mostly limited to the external tunic, but reached in certain points perhaps to the middle tunic without ever passing beyond it. Hæmatoidin crystals and masses of newly formed nuclear elements were seen in the lymphatic sheaths.

In the regions where the nerve-cells and axis cylinders had disappeared the tissue traversed by the vessels was made up of a fine fibrillated transparent network studded with nuclear elements like the nuclei of the neuroglia, but usually smaller. They were scattered at regular intervals, and were not usually more numerous in the neighbourhood of the vessels.

There was atrophy and sclerosis of the antero-lateral columns proportionate to the damage of the front horns, characterised by thickening of the trabeculæ and wasting of the nerve-tubes, which were gradually deprived of their myeline, but which in some instances retained their axis cylinders.

MM. Parrot and Joffroy remark, in the considerations appended to their microscopic observations, that they have seen "the vascular lesions, the inflammatory nature of which is

incontestable, very irregularly distributed; they are found at points where the motor cells are scarcely affected, and they do not necessarily exist at all those points where the cells are most seriously damaged." They finish with the following conclusion: "We think that we may affirm that the motor troubles of infantile paralysis are to be explained *by a primary change of the nerve cells of the front grey horns of the cord.* The nature of this change and its immediate cause have, however, yet to be determined. . . ."

In two other cases of infantile paralysis, both of which died comparatively early, one two and a-half months and the other six and a-half months after the onset of the paralysis, MM. Roger and Damaschino found great enlargement of the vessels of the cord, an abundance of granular bodies in and about the lymphatic sheaths, and atrophy of the cells of the front horns on the side corresponding to the paralysis. The question to be decided is whether the wasting of the cells or the vascular (inflammatory) change be the primary lesion.]

. . . . . .

Muscles. Case No. 15.—M. Bouvier has given me an opportunity of examining the condition of the muscular fibre in a young girl, Alphonsine Quaras, æt. 12, who died of typhoid in M. Blache's wards at the Neckar Hospital. She had suffered from atrophic paralysis, which had caused on the right side a talipes varus (*un talus pied creux varus dans sa moitié antérieure*). The fleshy mass of the gastrocnemius and soleus was whitish yellow, and had undergone almost complete fatty degeneration and substitution. It was hard to say where the muscular tissue began and the surrounding fat ceased; nevertheless in the substance of the gastrocnemius were found a few isolated points (1 or 2 millimetres in size) of a more or less good red colour, and in which were recognisable striated fibres mixed with others, granular, or fatty granular, and not yet completely changed into fat.

The other muscles offered to the naked eye all the degrees of tint seen in progressive muscular atrophy, from bright red or the pale red of simple atrophy to a more or less marked yellow.

I have established by microscopical examination that the change of the muscular fibre was, as in progressive muscular atrophy, directly proportional to the yellow colour of the muscle.

Thus, (1) a red colour even in the most wasted muscles showed a normal state of nutrition of the fibres; (2) slight discoloration corresponded with the disappearance of the transverse striæ; (3) a yellow colour (gradually deepening) corresponded with the disappearance of transverse and longitudinal striæ; and (4) in the last stage with the production of fat vesicles. . . In one case only have I found the muscular fibres presenting an amorphous granulation, which, according to Virchow, precedes the fatty change.

On electro-muscular exploration I have always found the greatest number of healthy fibres in the contracted or retracted muscles. It could hardly be otherwise, for it is the muscles which recover from the paralysis which drag the limb in their direction, and by their continued contraction produce the deformities of the limbs and joints. On the other hand, the muscular fibres are most changed and fatty in the antagonists of the contracted muscles.

Nevertheless it is incontestable that one has sometimes seen the contracted muscle undergo fatty degeneration. Such muscles must have contracted before they wasted. . . .

Pathogeny.—[The only doubtful point in the pathogeny appears to be whether the atrophy of the cells or the hyperæmia is the first change.]

Pathological Physiology.—It is important to bear in mind that atrophic infantile paralysis, whatever may be its extent or intensity, is sometimes neither preceded nor accompanied by any fever at its onset, and that when fever exists it is usually of slight duration. The hypothetical doctrine of general atrophy of the cells of the front horns of the cord would account for this fact.

The primary atrophy of the cells of the front horns would also explain the sudden appearance of the paralysis, since experiment has shown these cells to be motor. On the other hand, the complete or almost complete preservation of the skin-sensibility is explained by the small amount of change in the back horns.

But we were ignorant of the fact that the so-called motor cells presided also over the nutrition of the muscles and the bones which they are destined to move. This information has been supplied to physiology by pathological anatomy.

I have been struck by the absence of sacral slough, or any other serious change in the nutrition of the skin, even in cases so severe as to cause wasting and degeneration of all the muscles of the lower limbs. Would it not therefore appear that the cells of the front horns have little influence on the nutrition of the skin? It is necessary for the production of these severe changes in skin nutrition that all the constituent parts of the cord be destroyed, as in certain hæmorrhages into the cord, or that the spinal ganglia, the organs which reinforce the trophic nerves of the skin (*organes de renforcement des nerfs trophiques de la peau*), be damaged. In short, it results from the preceding facts that physiology only clears up certain points in the pathogeny of this disease, but on the other hand it derives therefrom much information on the troubles which follow wasting of the front horns of the cord. Clinical observation has, in fact, taught that muscular nutrition, independently of the great sympathetic—derives innervation (1) from the cells, or some of the cells, of the front horns; (2) that these motor cells exercise no appreciable influence on the contractility of the bladder or intestine.

Ætiology.—This disease is not hereditary. I have not met with any nervous trouble having any relationship with this disease among the parents or ancestors of the little patients. Once, it is true, I met with locomotor ataxy in the father of a little girl stricken with wasting paralysis of the right lower limb; but in this case the first signs of ataxy—the lancinating pains and the vision troubles—appeared in the father after the birth of the daughter. This isolated fact can only be regarded as a coincidence. I have never even seen two children of the same family suffering from this kind of paralysis.

Conditions of misery and privation in the parents and children do not seem to act as causes, for the disease is equally common among rich and poor. The quality of the nurse's milk appears to be without influence in the development of this disease. Most of the children had excellent nurses, and had not shown, previous to their paralysis, the emaciation and almost continuous diarrhœa which accompany a bad or insufficient milk supply. There is no evidence that the disease is more common at the time of weaning.

Usually the disease begins about the age of 2. The following

table made by my son from fifty-six cases occurring in my practice shows the exact proportion of cases commencing at different ages. It shows that the proportion increases from the twelfth day to the end of the first year, and then decreases to a similar extent till the age of 10.

12 days after birth	1 case	18 to 24 months	11 cases
1 month	1 ,,	2 to 3 years	5 ,,
2 months	2 ,,	3 to 4 ,,	2 ,,
4 to 6 months	6 ,,	7 years	1 ,,
6 to 12 ,,	6 ,,	10 ,,	1 ,,
12 to 18 ,,	20 ,,		

Emotional states, such as fright or anger, do not cause atrophic paralysis; these act more particularly on the brain, causing intellectual troubles, hemiplegias and general paralyses, with contractures, but without change in the muscular tissue. I have observed this last fact twice especially; among other cases, in a little boy aged 4, who after a violent fit of passion was shut up in a dark closet by his father, and came out of it idiotic and paralysed. Electrical examinations five years after the onset established the integrity of electric contractility, and consequently of the texture of each of the paralysed muscles.

The only predisposing cause which seems to be incontestable is dentition. "In fact," says M. Duchenne, junior, "according to the exact statements which we have been able to get with regard to the onset, the paralysis has come on thirty-seven times between the sixth and the twenty-fourth month, the period of dentition, and only seventeen times outside of this period. We have further established that thirteen times the onset occurred when one or several teeth were being cut."

In opposition to the observation of Heine, who makes abnormal or difficult dentition play an important part in the causation, we have observed, according to the statement of the parents, that the paralysis occurring while a tooth was being cut, was not accompanied by more violent or prolonged fever than those which occurred after the teeth had pierced the gums. It would seem, therefore, that there are individual predispositions, and that causes which have no effect upon the spinal cord of one child act violently on that of another.

All the ailments of early infancy have been looked upon as

causes. Diarrhœa, vomiting, stomach troubles, intestinal worms, would produce reflex paralyses. As a fact, infantile paralysis occurs with all of them. M. Duchenne, junior, has seen it follow measles on five occasions, and slight typhoid twice. It may occur without appreciable cause. In a good number of my cases it was impossible to establish the slightest fever or indisposition at the onset. External causes such as chill may, according to Kennedy, produce infantile paralysis. . . .

Diagnosis.—[The following are the points of diagnostic value :—

1. *Sudden onset, usually with fever, sometimes without; with or without convulsions.*

2. *Paralysis, at first complete and extensive, gradually diminishing and settling in a greater or less number of muscles.*

3. *Diminution of electric contractility from the first in direct proportion to the amount of damage done to the innervation of the paralysed muscles; after a time return of electric contractility in those muscles, or parts of muscles, the tissue of which is not changed.*

4. *Various partial deformities of the limbs in the very advanced stage, resulting from the loss of balance in the tonic muscular power; arrest of development of the bones in the regions whose innerration is at fault.*

5. *A primary spinal change, demonstrated by analogy and confirmed by pathological anatomy.*

These points have never failed me when making a diagnosis at any stage of this disease.]

. . . . . .

Prognosis.—I do not know, as I have already said, that atrophic paralysis of children has ever terminated fatally.

The rare autopsies on such cases have been made on patients who have succumbed to accidental diseases, or at a very advanced period of life.

Although the disease does not threaten life, it is nevertheless a grave affection ; because, by causing the destruction or wasting of organs of movement, it compromises the muscular functions, the shape and attitude of the limbs. I may give an idea of the gravity of this disease by saying that it is the main source of supply for orthopedists.

The prognosis at the outset has no relationship to the con-

vulsions of the first stage, the extent of the paralysis, or the intensity and duration of the fever. I have, indeed, seen a considerable number of cases in which at the outset the muscular system was stricken with paralysis, accompanied by long and intense fever, in which, nevertheless, the muscles recovered their motility without wasting (these are the cases called *temporary* by Kennedy). In some cases after the paralytic period very few muscles remain wasted or changed, while in others in which the paralysis has invaded one or several limbs without fever or convulsions, all or nearly all the muscles have been destroyed by fatty or granular change.

The gravity of the prognosis is in direct proportion to the amount of nerve-damage which causes the wasting and fatty change in the paralysed muscles. Now a knowledge of the amount of nervous damage menacing muscular nutrition can only be got at by electro-muscular exploration.

I have pointed out that in this disease, as in paralyses following injuries of nerves or of the cord, the more the contractility and sensibility of paralysed muscles are diminished, the more these waste, and the more rapidly they become fatty and granular.

I must here remind the reader that absence of electrical reaction in a muscle has not the same value at all periods of this disease, for during the paralytic period it merely shows that the muscle is menaced, while in the chronic stage it shows that it is actually changed in texture. It may happen, however, that a few muscular fibres remain intact, but so enveloped in fatty tissue that their contraction is inappreciable.

It is hardly necessary to add that the gravity of the prognosis depends less on the number of muscles affected than on their functional importance.

To form an exact prognosis an exact knowledge of the changes in form and utility of the limbs caused by the loss of this or that muscle is necessary. These are the points which have been cleared up by my electrical investigations. Who would have thought, for example, that the foot is less deformed, and the functions of the lower limb less impeded, by the loss of all the muscles of the foot than by the paralysis of only a few of them?

When a single muscle is wasted very considerable deformities

are gradually produced, which sooner or later impede both standing and walking, rendering them difficult or even impossible. The following is an example of this:—

Case No. 16.—A child of 3 years old was brought to me because of a limping which had come on without appreciable cause. On electrical exploration I found apparent absence of the muscles of the calf. I therefore advised faradisation of the muscles, and the use of a sandal at night for maintaining a continued extension of the foot. This was not done, and six years later the child was brought to me with direct hollow talipes (*talus pied creux direct*), the front view of which is shown in fig. 19. Movement of the ankle joint was abolished, and flexion of the foot was only managed by a supplementary movement between the calcaneum and astragalus, which caused a flexion with extreme abduction, as is shown in fig. 20.

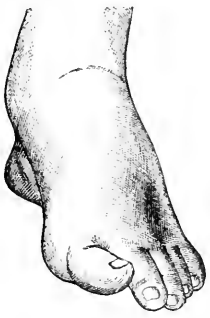

Fig. 19.

This is a serious deformity, and if it exist on both sides, walking and standing become painful and difficult. . . .

If I were to take the other muscles of the foot individually and singly, I could show by clinical observation that these partial atrophic paralyses are usually more serious than the total destruction of the muscles of the foot, although at first they cause so little hindrance to locomotion that very great attention is necessary for their diagnosis.

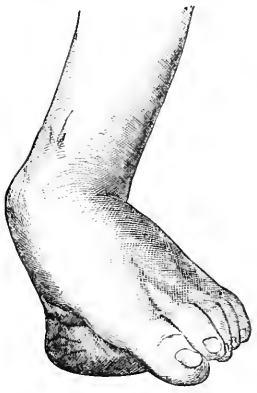

Fig. 20.

Finally, among these partial paralyses of the upper or lower limbs there are some muscles whose loss hinders function more than that of others. If the total destruction of the muscles moving the ankle causes no great hindrance to locomotion, it is far otherwise with the muscles of the hip. . . . . In the upper limbs the loss of the deltoids and flexors of the elbow almost completely annuls the use of the arms. In the hand the muscles of the thenar eminence are of the first necessity. . . . .

THERAPEUTIC ACTION O LOCALISED FARADISATION.—*At a period not far removed fro the onset.*—Localised faradisation applied in time, *i.e.*, at a riod near the commencement, can lessen the duration of the aralysis, diminish, if not prevent, wasting of the muscles, an possibly prevent fatty change.

1. I do not recommend th use of localised faradisation at the onset, especially when fever hows a state of inflammation. At this period we need only hav recourse to general antiphlogistic measures suitable to the ag of the subject. These should be designed to combat a lesio of the spinal cord or part of it. Consequently, in its neighborhood and along its course dry or moist cupping should be apped with blisters, &c.

2. But when the acute stge has passed (usually after three or four weeks) localised excitatio of the muscles becomes necessary, while the general treatmen is continued at the same time —revulsives to the skin or inestinal tract, calomel internally, as advised by Kennedy, and dect excitants of the nerve-centres, such as strychnine, &c.

I have shown that in its symptoms and course this disease resembles paralyses followin injuries of spinal nerves. It is well known that at the outse of this paralysis a great number of muscles are equally paralyed, but that some of them rapidly recover their motility withou wasting or suffering any nutrition change, while there are others in which movement does not return till after a long period, remaing more or less wasted, and that finally some remain permanently paralysed and become fatty and granular. Further, it is posible by electro-muscular exploration to distinguish those muscles in which movement ought quickly to return from those which will remain long paralysed, and which will, in addition, suer in their nutrition.

We may expect as favourabe results from localised faradisation in wasting infantile palysis as in paralysis following injury of the nerves and cor. It happens, in certain cases, that muscles whose nervous influx shows feeble obstruction in its course from the centre to he periphery, in consequence of a slight lesion of that point of the cord whence this influx emanates, slowly recover their motility. I have quoted cases in which movement returned only after many months, although the muscles had suffered very litt in their nutrition, showing that the nervous lesion was not ver severe.

In similar cases of wasting infantile paralysis, when electro-muscular exploration shows that the muscles are but slightly damaged, electric excitation would probably hasten the return of motor power. Need one insist on the importance of this quick re-establishment of motility at a time of life when nutrition is so active, and limbs may suffer an arrest of development as a consequence of their absolute immobility ?

These precepts when first put forward were not supported by sufficient experience ; I foresaw merely that their application must be crowned with success. Now, however, this point is settled, for all the cases of infantile wasting paralysis seen by me, in which electro-muscular contractility was merely diminished, recovered rapidly enough without wasting or deformity, when localised faradisation was applied a short time after the onset.

The infantile paralyses of the same kind, of six months, a years, or two years duration, in which electro-muscular contractility was no longer weakened, also recovered with faradisation ; but the limbs had become weakened by the long duration of the paralysis, and when the paralysis was situated in the feet these were more or less deformed.

3. Muscular faradisation may be applied to very young infants, for a considerable period (one or two months), and in strong doses, provided the intermissions of the current are sufficiently slow. I have excited the muscles of many children from a few weeks to a year old, sufficiently to get strong contractions. These children have not usually shown signs of pain when I have taken the precaution to accustom them gradually to the strange, but not painful, feeling of electric contraction of muscles. They soon begin to be amused by the operation. It is true that some of them, according to their parents, show more gaiety and vivacity towards the end of their treatment (after thirty sittings, for example). It is then advisable to suspend the treatment for a few weeks, and to resume it as occasion demands.

I regret that I did not appreciate earlier the harmlessness of localised faradisation with slow intermissions on the general conditions of young children, for, governed by prejudice, I have often refused to apply it when it would have been most useful.

4. The muscles whose contractility is very feeble remain a long time paralysed, and waste considerably. Although it may

be impossible to prevent wasting, localised faradisation prevents it from becoming extreme, and sufficient fibres may by this means be preserved to enable the muscles to fulfil their functions.

5. The muscles which from the outset fail to respond to localised faradisation waste completely, and these ultimately become altered in texture wholly or in part. But must they infallibly perish? Reasoning by analogy we may hope that this need not always occur. In fact this abolition of electric contractility shows, in paralysis from nerve-injury, a complete stoppage of the nervous influx. But we know how in this last affection localised faradisation has restored movement and nutrition in muscles wasted to the last degree when it has been applied at the opportune moment, *i.e.*, after the cure of the nerve lesion and the regeneration of the nerve. We may hope for a like result in infantile wasting paralysis; for when the spinal lesion is cured, and the nerve force returns to the muscles, localised faradisation sometimes restores their nutrition and prevents the degeneration which sooner or later would have occurred, as it does in cases of paralysis from nerve-injury which have not received a timely electrical treatment.*

6. But when, in infantile paralysis, should faradisation be applied to a muscle which has lost its faculty of contracting with electrical stimulation? In other words, when does the central nervous influx return to the muscles? I have proved by experiment that in paralysis from nerve-injury the application of faradisation made regularly from the beginning does not prevent the muscles, whose irritability is lost, from wasting, almost completely, for six, eight, or ten months, and that it is only after this lapse of time, the nerve lesion being then cured and the nerve force returning to the muscles, that faradisation can not only save them from complete destruction but can even recall their nutrition and their motility. In my first edition I wrote, "It would be imprudent to wait so long before submitting these muscles to electrical treatment continued for a sufficient time, for, after six months of this disease, I have known muscles disappear completely, and in spite of all efforts I have been unable to restore

* Duchenne does not appear to me to attach sufficient importance to the probable fact that the repair of motor cells once lost, is impossible; while the repair of conducting fibres emanating from healthy motor cells need never be despaired of.—G. V. P.

them. It is not surprising if, under the influence of an analogous cause (a pathological state of the cord), fatty change occurs sooner in the child than the adult, and since the time of the return of nervous influx, or, in other words, the cure of the nerve lesion, has not yet been determined in these cases of infantile paralysis I advise the treatment by localised faradisation of the muscles which have lost their contractility to be commenced as soon as possible. By acting thus can muscles be more often saved from complete destruction? We have a right to hope so, but the future can alone decide the question." This is what experience has taught me since I wrote those lines. Children have been sent to me two or three months after the invasion of wasting paralysis. Many of their paralysed muscles no longer responded to the strongest electrical excitation. Some of these have occasionally been saved from complete destruction. Among these muscles some are of very great utility, however feeble they may be, and it is these which we must try particularly to develop.

7. After the loss of a muscle, the limb, obeying the tonic power of its antagonists, whose exaggerated movement is no longer held in check, is ultimately dragged in the direction of these latter. Hence occur faulty positions and deformities which may prove great hindrances to muscular functions. . . .

I enter into these details in order to make it plain that it is important not only to try to recall nutrition in muscles threatened with destruction, but also to prevent, especially in the lower limbs, the deformities which necessarily result from the contraction and retraction of antagonising muscles. . . .

Treatment at a time very distant from the onset.—It is almost always at a very advanced period of infantile paralysis, when every kind of treatment has been exhausted in vain, that electricity is resorted to as a last resource. It is still able to do good service, but it is conceivable that its chance of success is lessened by its tardy application.

1. The paralysed muscles which at the onset of the disease retain intact their electric contractility and sensibility, rapidly recover their power of voluntary contraction, but often remain for a long time weakened and notably wasted.

It is important to faradise these muscles after they have recovered their motility. A short stimulation with a moderate

current suffices in a short time to develop their power and stimulate their growth. I have noticed this in most of the little patients who have been sent to me long after the onset of their disease.

2. The muscles whose electric contractility is more or less weakened in the acute stage ultimately recover this faculty as well as their power of voluntary movement, but this latter is often very slow to return. Localised faradisation will restore movement after all other measures have failed. . . .

3. Those wasted muscles which after a month or a year are still deprived of their electric contractility and sensibility are probably altered in texture. If this nutrition change has completely destroyed the muscular fibre we cannot hope to restore it by any means. But the change of muscular tissue is often irregular, as is shown by electro-muscular exploration; post-mortem examination is still better, or the examination of the living muscle made with the help of the tissue punch. In such a case, the healthy fibres may become the nuclei of new muscles under the influence of localised faradisation. Indeed, new muscular fibres may develop around those which are intact. In a granular or fatty muscle there may still remain many sound fibres; these sound fibres may be so enveloped in fat that their contraction is no longer visible when testing the condition of the muscle. Therefore although the absence of electro-muscular contractility in the advanced stage of this disease may indicate a fatty or granular state of the muscle, we may still hope, even in the absence of this sign, that there are still enough healthy fibres hidden by the fatty tissue to form the nucleus of future fibres or muscles which may be developed by localised faradisation.

. . . . [Duchenne records the case of a boy, aged $7\frac{1}{2}$, who had atrophy of the deltoid and upper limb of four years standing, the result of infantile atrophic paralysis. This patient was treated by localised faradisation, with slow intermissions applied three times a week uninterruptedly for two years. To the electrical treatment were added gymnastic exercises according to Ling's method. There was a steady but slow improvement. The fibres of the deltoid gradually recovered except at the posterior part, and with its restored nutrition the functions equally returned.] "This good result was due, no doubt, to the perse-

verance, admirably seconded by the parents, with which the treatment was carried out. Not only were the parents not discouraged by the length of the treatment, but they persevered with the shampooing and localised gymnastics in a most scrupulous manner."

Since the publication of this case I have obtained a considerable number of equally happy results in analogous cases.

These facts show incontestably that even where the absence of electro-muscular contractility gives cause to fear that the muscular tissue is absolutely degenerated, there may still remain a few sound fibres, while other healthy fibres may be developed even to the extent of the reformation of entire muscles. Such a result, it must be known, can only be got by long treatment, and it is useless to attack such cases without sufficient force of will to continue for one or two years, or even more, as was the case with the patient alluded to above.

If the result were always favourable, the physician would never shrink from the weariness inseparable from such a long course of treatment, and the parents would cheerfully face the cost and annoyance it entails. But unfortunately we can promise nothing. [A case is quoted in which faradisation applied to the deltoid for eighteen months consecutively produced no result. In this case the treatment was commenced eight years after the commencement of the trouble.]

Even when all hope of restoring the muscles is lost, faradisation will be useful, and in certain cases necessary for the development of the bones. I have said, it will be remembered, that this disease does not merely affect the muscles, as its name would imply, but that the bones also undergo an arrest of development proportioned to the damage of the nerve centre. If the trouble lies in one of the lower limbs there results a shortening which may considerably hinder both standing and walking. I have seen a shortening of six centimetres in the lower limb of a girl of 9, the result of a paralysis when she was 2½. In others aged 12 or 15 the shortening amounted to 8 or 9 centimetres. This shortening caused so much limping that it had to be compensated by a thick-soled shoe. Such a shoe is necessarily ungraceful; it can not be concealed, and causes great fatigue to the joints of the foot, the muscles of which are almost completely wasted. It is never too early to begin trying to prevent, if

possible, so great a difference in the length of the limbs. I know no agent equal to faradisation for helping the growth of the bones. I know how necessary it is to exercise caution in all therapeutic questions, but I nevertheless affirm, so far as my long experience will permit, that in this disease under the influence of localised faradisation the bones increase in length and circumference, and that with this treatment, employed for many years on poor children, I have only had one instance of shortening of two or three centimetres in a case in which the shortening would probably have been greater by five to nine centimetres if the wasted limb had been left to itself.

4. One word on treatment by continuous currents. The attempts I have made cause me to fear lest it often prove difficult of application by reason of the pain at the points of contact of the rheophores with the skin. Most of those submitted to continuous currents, even from a small number of elements with slight electrolytic action, have cried terribly. Sometimes the parents have discountenanced the treatment after a few sittings. Nevertheless I associate it as much as possible with localised faradisation.

5. Concurrently with electricity we must substitute the lost muscles by elastic apparatus, and later employ "orthopedy" when the paralysis is in the lower limbs. We should endeavour —1. To favour the nutrition of those muscles which are most wasted and most threatened by atrophy by placing them in a state of continuous shortening. 2. To prevent the retraction of those muscles whose tonic power has become predominating, and the club-feet which will result therefrom, by balancing their tonic power by an antagonistic mechanical power. 3. In a very advanced stage, when recovery of the wasted and degenerated muscles is no longer possible, to help standing and walking by suitable orthopedic apparatus.

CHAPTER IV.

ACUTE ANTERIOR SPINAL PARALYSIS OF THE ADULT (ATROPHY OF THE ANTERIOR CELLS OF THE CORD).*

. . . . . .

The following case establishes the existence of this form of paralysis in the adult.

Case No. 17.—A young lady, æt. 22, habitually healthy, awoke one morning with fever, general aching, and a difficulty in moving, and this without obvious cause. About an hour later there were pains along the course of the spinal column, particularly severe in the cervical region, and radiating to the upper limbs, with formication in the fingers; then followed loss of all movement. *Nevertheless the sensibility of the skin remained intact, and there was no trouble in micturition or defecation.* Four days after the onset the fever disappeared, but the paralysis lasted for two months and a half, when the lower limbs began to recover their motility, with the exception of flexion of the left ankle. Towards the third month some movement returned in the upper limbs; at this time the spinal pains, which had diminished for a month past, completely disappeared; but the upper limbs, the right in particular, were atrophied, especially at the shoulder, arm, and thenar eminence.

Six months after the onset of the disease the young lady was sent to me from the country, and I then established: 1. That motility had returned in the muscles of the lower limbs, except in the right (*sic*) tibialis anticus, which was atrophied, and had produced on this side a commencing equinus. 2. That in the right upper limb the deltoid, infraspinatus, flexors of the elbow, interossei and the muscles of the thenar eminence, contracted neither to the will nor to electricity; that these muscles, especially the posterior third of the deltoid, were atrophied, while on the opposite side the paralysis was limited to the

* From *L'Electrisation Localisée*, 3rd ed., pp. 437—486.

serratus magnus and the flexors of the fingers. The condition of this young lady was much improved by localised faradisation of the wasted muscles. But the deltoid and abductor pollicis on the right side, and the left (*sic*) tibialis anticus gave no signs of life after twenty sittings. Microscopic examination of a small portion of the last muscle, which I removed with my "tissue punch," showed me that it was degenerating. In this group of symptoms (sudden general paralysis localising itself many months later in certain muscles which waste in various degrees, without sensory troubles or loss of function in the bladder or rectum) who can fail to recognise the symptoms of infantile atrophic paralysis?

As I record this case, two others have just come under my notice which will serve to demonstrate the real existence of this form of paralysis occurring spontaneously in the adult. The following is a short summary of one of them, the other will be given when dealing with *prognosis*.

Case No. 18.—On March 21, 1870, I was called in consultation by my *confrère*, M. Martin, to see M. X., aged about 42, living at Passy. He was strong and habitually healthy, having never suffered from any grave disorder; but had contracted the habit for the past twenty years of taking a glass of absinthe every day, and of smoking night and morning tobacco (caporal) containing a large quantity of nicotine. These were the only possible predisposing causes of the nervous disorder with which he was stricken. In 1848, while making a great effort to raise a heavy weight from the ground, he felt, he says, a "crack" in the loins, followed by severe pains radiating to the limbs, and at the same time a numbness of the legs and feet, formication in the toes, and then a weakness in the lower limbs, which gradually increased, and ended in complete paralysis. This paralysis was cured in about two months, but the activity of the legs never completely returned, although his health remained perfect till July, 1869, when, without appreciable cause, he was suddenly attacked one day with a feeling of lassitude, which was soon followed by a general cramp, which compelled him to keep his bed. A fever then set in which lasted several days, and was accompanied by pains in the limbs. A little time after taking to his bed he experienced great difficulty in moving, and the next morning he was completely paralysed

and could not move in his bed in the slightest degree. This general motor paralysis lasted till the month of December, when some movement returned in the upper limbs, first in the flexors of the elbow, then in the flexors of the wrist, and lastly in the extensors of the shoulder, beginning on one side and extending, a week or a fortnight later, to the other. These movements, feeble at first, got stronger by degrees, but the extensors of the thumb and fingers and the muscles of the thenar eminence on the right side remained paralysed. The movements of the lower limbs returned about the same time, but in an irregular manner. Thus the flexors of the ankle, extensors of the knee, and flexors of the hip remained paralysed. Some weeks after the onset of the paralysis the upper limbs wasted rapidly and to a considerable extent, and the thenar eminence on both sides entirely disappeared. The muscles of the lower limbs also wasted, especially the tibialis anticus. The flexors of the knee and extensors of the ankle were a little retracted, especially on the right side. These retractions occurred gradually through the failure of the antagonising muscles. There was never any derangement of bladder or rectum, and the sensibility of the skin was always normal. The general health remained good after the subsidence of the initial fever.

In the two preceding cases the spinal lesion (probably acute atrophy of the anterior cells) occurred without appreciable cause. This was not so in analogous cases which I have collected. In most of them a chill, more or less severe, was the determining cause.

[A case is quoted of a Russian gentleman who suffered from acute spinal paralysis brought on by sleeping in the snow in order to win a bet made in a fit of drunkenness.]

. . . . . .

Symptoms.—The symptoms of the preceding cases are incontestably those of atrophic infantile paralysis, and they were, doubtless, caused by the same anatomical lesion, viz., acute myelitis of the anterior cornua. It is needless to give here the symptomatology of this form of acute anterior spinal paralysis of the adult, which is identical with that of acute infantile paralysis, excepting that in the latter the osseous system undergoes more atrophy than in the former. This difference is explained by the arrest of development which the osseous system

must suffer in atrophic infantile paralysis. The other facts mentioned, while describing the latter affection, are applicable to acute anterior spinal paralysis of the adult.

Diagnosis.—[In the acute stage the anterior spinal paralysis may be mistaken for acute myelitis affecting both the anterior and posterior cornua.]

When treating of electro-muscular contractility as a means of determining the diagnosis and prognosis of spinal paraplegia I shall bring forward clinical facts which show that destruction of the cord in the lumbar or sacral regions causes not only atrophy of the muscles innervated by those regions, but also paralysis of the bladder and rectum, loss of sensibility of the lower limbs, and sacral slough; while acute atrophy of the anterior cells in these regions causes merely atrophic paralysis of the lower limbs.

In short, it results from the facts above stated, 1. That during the acute stage, atrophy of the anterior cells and diffused myelitis have as common symptoms atrophic paralysis and loss of electro-muscular contractility of the muscles receiving their innervation from the affected part of the cord. 2. That diffuse myelitis is distinguished from the former condition by loss of sensibility, by paralysis of the bladder and rectum, frequently by sloughs on the sacrum, and, lastly, by the presence of reflex contractions in the muscles which receive their innervation from those parts of the cord situated below the lesion.

When acute anterior spinal paralysis has become chronic it may be confounded with atrophic infantile paralysis, with progressive muscular atrophy, and with general subacute spinal paralysis. [The ultimate effects of these several diseases may be identical, and atrophy of the cells of the anterior cornua of the cord may be found in them all, so that to make a diagnosis in such cases we must appeal to the history and cause of the attack.]

Pathological Anatomy.—[Reasoning from analogy, Duchenne concludes that the anatomical lesion must be the same as in atrophic infantile paralysis. He has no actual facts to offer.]

Causation.—The disease usually appears in young people, between the age of 18 and 45. It is not usually traceable to any cause, but often a prolonged chill while the body is sweating, or after excessive muscular exertion, such as a long

walk, is assigned as the determining cause. I have not traced it to heredity.

Prognosis.—The disease does not seem to threaten life, however long it may last. . . . The prognosis is grave only in proportion to the degree in which muscles whose functions are of vital importance are affected. The functions of the abdominal viscera and the sensibility remain intact; there is no sacral slough, and the lesion is probably limited to the front horns of the cord. Hence it is that this disease never threatens life.

But if the signs of acute spinal paralysis show that the lesion of the cord is diffuse the prognosis becomes very grave. Of this the following is a remarkable instance:—

Case No. 19.—In 1853 a young woman of fair constitution suffered suddenly from numbness and formication in the lower limbs, and from weakness which prevented her from standing for long together. This weakness increased so that she had to keep her bed on the second day. On the third day she was removed to the Charité under the care of M. Briquet, where I saw her for the first time. I found the movements of the ankle and of the extensors of the knee completely lost. She could still flex slightly the knee and the hip. The sensibility of the feet and legs was diminished, and was soon completely lost in the lower limbs. She was impressionable, but had never had fits of hysteria, and M. Briquet thought that her paralysis might depend on an hysterical condition. Electricity may throw much light upon this kind of paralysis, and, in fact, I found in this case that faradic irritability was much diminished in the muscles of the toes and ankles, and that the patient did not feel the strongest currents. These signs did not admit of a diagnosis of hysterical paralysis, in which disease the electric irritability is always normal. I diagnosed a severe dynamic or organic lesion of the cord, and gave a grave prognosis. A few days later the paralysis reached the upper limbs, being preceded by formication in the fingers, and the electric irritability which had been normal was now diminished. I therefore diagnosed a general diffuse acute paralysis (which it has been wrongly proposed to describe as a new variety under the name of ascending paralysis), and I anticipated a fatal termination. The paralysis, in fact, got worse, and I found the electric irritability (after many trials at intervals of some days) gradually diminish and die out after the

power of movement had begun to be lost. The bladder and rectum were paralysed, the breathing was attacked in its turn, and the patient died on the nineteenth day after her admission to the hospital. At the autopsy no appreciable lesion of the nerve centres was discoverable *with the naked eye*.

In this case the cord seemed to all appearances healthy. It was certainly, therefore, neither diffluent nor even softened, for such a lesion could not have escaped the careful examination to which it was subjected. It is very probable that the microscope would have revealed an alteration in texture extending to all the constituent parts of the cord. What was the nature of the lesion? Which anatomical elements were specially affected by it?

Whatever it may have been, the two preceding cases show the importance of not confounding these two varieties of acute spinal paralysis in the adult, of which one never threatens life, whilst the other is rapidly fatal.

GENERAL SUBACUTE SPINAL PARALYSIS.

[Duchenne makes two subdivisions of this disease, viz., "*anterior general subacute spinal paralysis*," when the lesion is mainly limited to the anterior cornua; and "*diffuse general subacute spinal paralysis*," when the lesion is not so limited. His first observations were made in 1847, and his first account of the disease was published in 1853.] . . . .

Anterior general subacute spinal paralysis in its development sometimes follows an ascending course, beginning in the lower limbs, more commonly in one of them; and sometimes a descending course, attacking one or both upper limbs. I will content myself by giving two selected typical cases, showing this disease in its two forms, ascending and descending. These will serve to give a picture of the malady.

Case No. 20.—Martin, æt. 55, a military pensioner, of fairly good constitution. No history of syphilis, nor of venereal excesses. Has not been exposed to lead poisoning, nor has he ever suffered from colic or constipation, which might give colour to such a supposition. No whitish (*liséré blanchâtre*) line on the gums. No muscular rheumatism nor previous neuralgic pain. On September 28th, 1846, while carrying a load of wood on his left

shoulder, he fell and strained himself, and was admitted to the Beaujon Hospital. A month later "phlegmon," followed by intense fever, showed itself. Towards the end of March, 1847, he left the hospital incompletely cured of his strain, and using a "*crochette.*" . . . .

Some time after his dismissal *he began to notice a loss of power in his lower limbs. Then the muscular weakness gradually increased, so that standing became impossible. In October,* 1847, *he was obliged to lie up, and then felt a loss of power in his upper limbs. The patient then noted a diminution in the size of his legs.* In the course of the paralysis there was no fever, no digestive trouble, no affection of the bladder or rectum.

In December, 1847, when he came into the Charité, I noted the following appearances. Standing and walking were impossible; when in bed the patient could move his lower limbs in all directions, but slowly, and with great effort; he could sit on the bed and turn in all directions. He felt some weakness in his upper limbs. There was no paralysis of bladder or rectum, and no pain in the head, limbs, or spine. No tremor of the limbs nor shocks, but on examining him with care, there were fibrillary contractions (not numerous), raising up the skin in all parts of the body. *Electro-muscular contractility almost abolished in the lower limbs and in the abdominal muscles,* but intact in the muscles of the trunk, upper limbs, and face: the popliteal nerves when stimulated still caused feeble contractions in the muscles which they supply, but these muscles did not contract when stimulated directly. Electro-muscular and cutaneous sensibility were diminished in the lower limbs, but the sensibility of the skin was normal everywhere else. *Six months later,* when a fresh examination was made, there was loss of power in the upper as well as in the lower limbs, and the muscular atrophy had increased and reached the upper limbs. But this atrophy had progressed in a uniform way, invading at once an entire region or a whole limb. . . . . Although the patient preserved his appetite and suffered no pain, he "felt he was dying," to use his own expression. Speech was slow and difficult, mastication required effort. Electro-muscular sensibility was lost almost everywhere. Electro-cutaneous sensibility was intact on the trunk and upper limbs. There was no paralysis of bladder or rectum. Intelligence remained perfect.

The patient lived some months in this condition, preserving his intellectual power and suffering from no digestive trouble, but he was gradually "extinguished," and died without the occurrence of any complication, even in the very last stage of his disease. The autopsy was made by M. Empis, in the presence of M. Pidoux, acting for M. Andral. The brain and its membranes, the spinal cord and its roots, were examined with the greatest care. *They presented to the naked eye no appreciable anatomical change.* The other organs were normal. The muscles of the lower limbs were much wasted, *some more or less decolourised, others partly fatty to the naked eye, and, strangely enough, the greater part of the muscles of the leg, although much wasted and paralysed from the outset, both as regards their voluntary and electric contractility, preserved their normal colour. Examined under the microscope by M. Lebert, the fibres of these last muscles were found to be normal, although some of the muscles of the thigh, which were more or less decolourised and yellowish, were transformed into fat in various degrees.*

The following are some of the reflections which I made upon this case in my memoir of 1849:—

"Muscular weakness has been the most marked symptom in this patient; weakness which prevented him from walking or standing at a time when the muscles were still big enough to perform these functions. The wasting was not sufficient cause for the weakness, for in a case of muscular atrophy with fatty change, then in the wards of the Charité, the patient could still walk, although the muscles of the lower limbs were much more emaciated (this patient was the long-voyage captain alluded to in the chapter on progressive muscular atrophy).

"The loss of movement showed itself first in the lower limbs, and following an ascending course, invaded all regions by degrees, even the tongue and one-half of the face.

"Post-mortem it was shown that certain muscles were much changed, being in great part fatty, but that this change was less general than in 'muscular atrophy with fatty change.' Finally, and this is important to note, the muscles which during life did not contract under electric stimulation were found, though atrophied, perfectly sound as regards colour and texture."

I still hold the same opinions, and believe that the case was

one of subacute paralysis, steadily progressing towards generalisation, affecting successively all the regions of the body, causing atrophy *en masse* of the muscles, weakening and extinguishing their electric contractility in various degrees, but only changing the texture of some of them. This case is a specimen of *ascending* general spinal paralysis.

The following is a case in which, on the contrary, the course was *descending*, beginning in the upper limbs.

Case No. 21.—In September, 1847, Madame D. noticed a weakness of her right upper limb, and a little later of her lower limbs. In October, 1848, the right arm was useless. The paralysis soon reached the left arm, and in November, 1848, she was entirely crippled. Speech and swallowing became difficult in August, 1849. I was asked by my *confrère* M. Charrier to examine this lady in order to form a diagnosis by means of localised faradisation, when I established the following facts:—Electric irritability (even to electro-puncture) was absolutely lost in the front and outer part of the leg, while in the back of the leg electro-puncture caused only a few fibrillary contractions. The electric irritability of the thigh, though still considerable, was less than normal. The rheophores applied to the back of the right forearm only caused a feeble contraction of the extensors of the thumb. The muscles of the front of the forearm only contracted feebly when the median nerve was excited, with very intense currents. The muscles of the arm contracted better, but only to a very strong current. On the left side the muscles of the upper limb were more irritable. The contraction of the abdominal muscles was less than normal, while those of the trunk seemed to have preserved their natural irritability. In the face certain muscles had lost in a slight degree their irritability. Intelligence, sensibility, digestion, micturition, and defecation were unaffected. [Duchenne does not give the date of this examination.] Two months later this lady died suddenly, without pain, in perfect enjoyment of her intellectual faculties.

Since the publication of the two preceding cases in 1848 and 1849, I have collected many other examples of anterior general spinal paralysis in its ascending and descending forms. I shall mention many others, actually under observation, in the course of this chapter.

The two typical cases just related give an exact and complete

picture of anterior general subacute spinal paralysis in its perfect development. They enable us to follow it in its ascending or descending course to its final stage, which is chiefly characterised by troubles of speech and swallowing. (I can perfectly call to mind now that these functional troubles, especially in the second case, were of the kind which characterises glosso-labio-laryngeal paralysis, a disease to which a subsequent chapter is devoted.)

But this picture would give an inexact idea of the disease, and might leave the impression that it always ends fatally, were I not to place alongside the preceding cases another, in which nutrition recovered in most of the muscles after the paralysis had remained stationary and generalised for more than a year, but without having attacked the tongue or the face.

Case No. 22.—Foucault, æt. 51, a saddler, usually healthy. In February, 1865, without known cause, without fever, and without numbness or tingling of the right fingers, he experienced a weakness of the movements of the right hand, chiefly in the index and middle fingers, but which did not prevent his working for eight or ten days. Five or six days later the weakness attacked the whole limb. Six weeks later weakness showed itself in the lower limbs. Finally the paresis became general, and deprived the patient of all movements except those of the head. After two or three months there was a broken and bruised feeling in the wrists and knees, and at the same time a weakening of the sensibility of the limbs. The functions of the bladder and bowels were preserved, and the appetite was normal. This condition lasted for about thirteen months, when there was a progressive return of movement, first of the arm on the shoulder, then of the forearm on the arm, and after a short time the patient was able to execute all normal movements, though with a certain feebleness, excepting those of the hand on the forearm, and of the fingers, which remained quite paralysed. On May 1st, 1870, I found that *on the left side* extension of the hand and of the first phalanges was re-established, but that the interossei were paralysed; that when the wrist was kept straight he could flex the fingers feebly; that the muscles of the thenar and hypothenar eminence were paralysed. *On the right side* that he could extend the wrist and fingers feebly; that the muscles of the thenar and hypothenar eminence were paralysed; that all

movements of flexion could be very feebly performed; that electro-muscular contractility was abolished on the back of the forearm, normal in the interossei and abolished in the thenar eminences; that the muscular masses were uniformly developed in the forearm, and that the thenar eminences were flattened and effaced; that both hands were always shut, or, in other words, that the fingers and thumb were continually flexed, and that the first and second metacarpal bones were in the same plane; there were no troubles in the face, nor were speech, swallowing, hearing, or sight affected. General health fairly good.

Symptoms and course. *Ascending form.*—The motor troubles usually begin in the lower limbs. There is first a weakness in one limb, or in both of them, but more marked in one.

If the motor power of each division be then examined it will be generally found that the flexors of the ankle and hip are affected first and most, and next that the extensors of the knee are successively and progressively attacked. This causes a considerable difficulty in walking (a difficulty in throwing the leg forward, and in raising the body on the point of the supporting foot). In these cases there are no disordered movements such as are seen in locomotor ataxy, choreic paralysis, or sclerosis *en plaques*. The paralysis increasing, walking and standing soon become impossible, and eventually partial movements of the lower limbs are abolished.

Shortly after the onset of the weakness, the electric contractility of the muscles diminishes and is gradually lost. At length their texture is more or less changed. The irritability of the muscles of each region is affected in different degrees. It diminishes first in the muscles of the front and outside of the leg, then in those of the foot and back of the leg, and then the muscles of the thigh and abdomen are affected almost simultaneously.

The paralysis remains confined to the lower limbs for some time before attacking the upper limbs. Sometimes the weakness of the lower limbs has scarcely lasted a week before loss of power begins in the hand, while at other times the paralysis may continue as a paraplegia for many months or even years before spreading. But as soon as the upper limb begins to suffer the paralysis progresses as it did in the lower limbs, attacking first and most the extensors of the fingers. Then the muscles of the

trunk and even of the face are in their turn stricken with weakness. In the end the paralysis is almost always most marked on one side of the body.

The paralysed muscles are not slow to waste. The atrophy attacks *en masse* the limbs, which seem to mummify (*se momifier*) in a manner very different to the irregular and partial disappearance of muscles which is seen in progressive muscular atrophy. I have attentively noted the order in which the muscles of the upper limbs lose their electric irritability, viz., 1. The muscles of the back of the forearm (*la région antibrachiale postérieure*). 2. The muscles of the hand. 3. The muscles of the front of the forearm and arm. 4. The muscles attaching the arm to the chest. During the progress of the paralysis, the muscles in groups or in mass suffer equally in their electric contractility and their nutrition.

The symptoms and course of the *descending form* of anterior general subacute spinal paralysis are like those of the ascending form described above, with the exception that, having started in the upper limbs, it progresses by degrees to the lower limbs and thence to the trunk.

Finally, if the paralysis do not improve or come to a standstill, certain muscles of the face and tongue are attacked, and there results a difficulty in speaking and swallowing. Lastly, breathing is affected, and then death by syncope or asphyxia is not long delayed.

Whatever form the paralysis may take, ascending or descending, the motor trouble may be arrested for a time. It may, for instance, remain limited for a long time to the extensors; or again, and this is more common, after having attacked the muscles of the forearm it attacks those of the leg, or *vice versâ*. In this case it assumes a hemiplegic form for some time and spreads slowly. The paralysis may recover once or twice before it becomes general or definitely established. I have known several cases where it was limited at the outset, get well, and have found them later in other hospitals with the disease localised after one or two relapses.

We must not expect to find in this kind of paralysis complete extinction of electro-muscular irritability (which may happen, however, in the last stage of the disease). Usually electro-muscular irritability is only diminished. In some rare cases the

greatest care is necessary to establish in the early stages of the disease any diminution of the irritability.

Intellect, sensibility, and the functions of the bladder and rectum are not affected in this disease. Its course is slow. It may be prolonged for several years.

Pathological Anatomy and Pathogeny.—[In one case observed in 1847, there was noted with the naked eye an injection and diffluence of the front part of the cord for a length of 6 centimetres at the level of the fourth cervical vertebra. There were no morbid naked eye appearances in the lumbar region in this case, notwithstanding an atrophic paralysis of the lower limbs as well as of the upper. (For details see *Elect. Loc.*, 1861, case xxxvii., and 1855, obs. xcvi.) In another case already quoted there were no naked eye appearances in the cord.

Notwithstanding these negative results Duchenne does not doubt that, reasoning from analogy, the anterior cells of the cord are the seat of the lesion in this disease.]

"In short, there exist three kinds of paralysis due to atrophy of the anterior cells of the cord.

"1. Atrophic infantile paralysis.

"2. Acute spinal paralysis of the adult.

"3. Anterior subacute spinal paralysis (which I have called general because of its progressive course towards generalisation)."

Diffuse general subacute spinal paralysis is the second variety of the muscular trouble which I regard as indicating a sub-inflammatory state of the cord, and of which I have just described the first variety under the name *anterior general subacute spinal paralysis*. The description of this variety as regards symptoms and course is almost absolutely applicable to the other. The picture of this variety will be complete if to the symptoms of the first variety be added: 1. Pains more or less severe either along the spinous processes of the vertebræ (*points du rachis*), or in the course of the nerves, or in the paralysed muscles. 2. Sensory troubles (cutaneous hyperæsthesia and anæsthesia). 3. Contraction or stiffness of the limbs. 4. Paralysis in various degrees of the bladder and rectum and sexual disorders. 5. Sacral slough.

But this list of symptoms is not always complete, the reason being that the presence of all these morbid signs indicates the extension of the lesion to all parts of the cord, which extension

does not always take place. I have never found a perfect resemblance between cases of diffuse general subacute spinal paralysis.

The pains usually occur at the outset. They increase by paroxysms; precede or accompany the paresis and paralysis; and persist for years, lasting often till the very end. They are not fulgurant, but have a certain continuous character which distinguishes them from the pains of locomotor ataxy. They often remain for some time in the nerve tracts before the occurrence of paralysis and atrophy, and simulate neuralgia; or they occur in the muscular masses and even in the joints, and are increased by pressure. They are sometimes then mistaken for simple rheumatic pains, especially if it happen that the patient has been exposed to cold, and it is only when the paralyses and wastings appear that the true diagnosis can be made.

The pains sometimes radiate to the fingers and toes, in which tinglings and numbness have been previously felt. The sensory skin-troubles appear in the course of the disease, sometimes in the form of over-sensitive patches on the trunk or limbs, giving place ultimately to anæsthesia; at other times the extremities—hands and forearms, feet and legs—are smitten at once with anæsthesia, which steadily increases.

When the lower limbs are stricken with paralysis the functions of the bladder are often affected. When the disease is moderately advanced there are usually difficulties of micturition, accompanied at times by spasm and obstinate constipation. Later both micturition and defecation take place involuntarily. At the same time there are sexual troubles, immoderate excitement or impotence, sometimes accompanied by obstinate priapism during sleep. Finally, in the last stage sloughs occur upon the sacrum.

Such are the morbid signs which, added to those of progressive atrophic paralysis, characterise diffuse general subacute spinal paralysis.

As my space is limited, I will not give all the cases from which I have drawn the preceding symptoms. After the description just given, it will be easy to recognise elsewhere good examples of this kind of paralysis reported by authors as cases of progressive muscular atrophy. I will quote amongst others two cases published under this title by MM. Charcot and Joffroy. These pathologists have elsewhere distinguished them from this

last disease by regarding them as cases of *false or hybrid muscular atrophy*. I will merely give an abstract of one of these, which to my eyes is a typical case of diffuse general subacute spinal paralysis. The post-mortem examination, given in some detail, will serve for the study of the pathological anatomy of the disease we are considering.

Case No. 23 (abstract).—Augustine C., æt. 29, after exposure to wet and cold, suffered in August, 1865, from severe pain, without fever, in the right limbs, following the course of the nerves and increased by pressure. In October there was a difficulty in opposing and abducting the thumb, decrease of the thenar eminence, and hollowing of the interosseal spaces on the right side; loss of the power of separating and approximating the fingers, and lessening of the contractility of the muscles of these regions. In April, 1866, the patient had become very infirm. She could still stand, and even walk, or rather shuffle the feet along alternately without raising them. The muscles of the limbs, especially those whose contractility, both voluntary and electric, was lost, were wasted. In September, 1866, sensibility was still normal. A year later there was decided contraction of the flexors of the left hand and legs, which contraction ceased in October, 1867. On December 12th there was complete paralysis of the upper limbs, and the condition of the patient was aggravated.

Towards the end of 1867, M. Dieulafoy, assistant to M. Jaccoud at the Hospital St. Antoine, took me to the bedside of this patient, and the following are the notes which I then made, and which will complete this interesting case.

My attention was first chiefly directed to the difficulty of breathing. The chest was completely motionless, and the respiration diaphragmatic, in contrast to the normal thoracic respiration seen in women. I ascertained that this trouble was not caused by atrophy or wasting of the intercostals, but by the severe pains situated in the cervico-dorsal region, and radiating over the upper half of the thorax. I made her take several fairly deep thoracic inspirations, but these increased her pain, so that she could not take two such inspirations consecutively. For this reason her breathing was abdominal, and the thorax was instinctively kept motionless and drawn together. The spinal pains were also felt in the muscles of the back of the neck, which

caused the movements of the head to be painful, and almost impossible; and also in the left upper limbs, where they were increased by pressure on the nerves and muscles. Finally, there was hyperæsthesia of the skin over many points which were the seat of pain. Questioned as to the history of her motor trouble, she gave me precise details, from which I concluded that, at first, the muscles of the lower limbs were stricken, some with paresis, and others with paralysis, before the limbs began to waste, which made standing and walking difficult and almost impossible. (I shall again refer to this fact when discussing the diagnosis.)

I found her limbs permanently flexed—the thigh on the pelvis, the legs on the thigh—from the stiffness of the joints, caused by the contraction of the muscles. But she stated that previous to the contraction she could not move her legs. Most of the movements of the left upper limb were also hindered by contraction; but on the opposite side, where there was no contraction, movement was completely abolished, although fairly developed muscular masses, whose tissue was probably normal, were still to be found there. In short, I concluded from my examination that the case was one of atrophic spinal paralysis. At the beginning of 1868, this patient was taken to the Salpêtrière. It was then found, in addition to the condition given above (and minutely described by MM. Charcot and Joffroy, who borrowed it in part from M. Jaccoud), that there was cutaneous anæsthesia of the left side of the thorax, right upper limb, left hand and both lower limbs, but least marked in the latter. During the month of April severe paroxysmal pains were felt at the back of the neck, on a level with the lower cervical and first dorsal vertebræ, radiating over the antero-superior part of the thorax and to the arms. Similar pains were felt in the left lower limb. Towards the end of May there was an aggravation of the general state, a slough on the right buttock, and obstinate diarrhœa. The contraction and the fits of rigidity of the right limb disappeared. Death occurred on June 1st, 1868. (The microscopic examination of the cord is given below.)

Pathological Anatomy.—By the light of ideas acquired by modern researches, it may easily be seen to what anatomical lesions each of the morbid signs appearing during the invasion and course of diffuse general subacute spinal paralysis corresponds. In fact, the severe continuous pains following the course

of the nerves, and felt in the joints, muscles, and skin—pains which usually mark the onset of the disease—should indicate a spinal meningitis; the paresis and atrophic paralysis, invading successively and *en masse* the different parts of the limbs and trunk, should indicate that the inflammatory action has reached the front horns of the cord. The other symptoms are also explicable: the contraction of the muscles and stiffness of the joints by sclerotic change in the antero-lateral columns; the marked changes of sensibility by a lesion of the posterior horns; and the functional troubles of the bladder and rectum by a great diffusion of the spinal lesion, especially just above the lumbar enlargement.

This is no mere theory, for MM. Charcot and Joffroy have ascertained the existence of all these anatomical lesions in the post-mortem examination of Case No. 23.

These observers have admirably interpreted the pathogenic significance of this cord. The details, as well as the pathogenic considerations flowing therefrom, are given at length in their memoir, from which I borrow the following extracts:—

1. *Meninges.*—The cervical enlargement of the cord appeared for the greater part of its length to be enveloped, in the posterior two-thirds of its circumference, in a kind of fibrous muff, the texture of which, examined in transverse sections, bore no small resemblance, both in aspect and consistence, to the tissue of the cornea. It was easily seen that it was formed of dura mater and pia mater considerably thickened and intimately blended. This thickening of the meninges was especially marked at the middle of the swelling, and became less towards its extremities, and to this was due the fusiform enlargement of the cord in this region. It may be remarked that the posterior nerve roots traversed, as it were, hollowed canals, formed in the thickness of the fibroid tissue; but at all points of their passage the nerve tubules, although pressed against each other, had preserved all their normal characters. In this patient the disease began, the day after an exposure to wet and cold for several hours, by repeated shiverings, which were quickly followed by paroxysmal pains, often severe enough to disturb sleep, and which occupied mainly the course of the nerves of the limbs. These pains, which in some of their characters were like the lightning pains of ataxy, constituted, so to say, for two or three months the entire

disease, and subsequently often recurred. Is it not evident that they were caused by the posterior meningitis, which at this time did not occupy the cervical region exclusively, and that they were due to irritation of the sensory roots?

2. *The grey matter* in the cervical region had almost lost its normal aspect in all its length. Many nuclei coloured by carmine, often massed and pressed together, could be distinguished in it; the vessels were numerous and bigger than usual; their walls thickened, and the lymphatic sheaths showing much multiplication of their nuclei.

At certain points, chiefly in the front horns, the space separating the nuclei was in great part filled by dense fibrillary tissue.

Elsewhere, especially in the back horns, the nuclei were in places less numerous, and imbedded in an amorphous, finely granular semi-transparent soft material. In other parts of the back horns the nuclei had disappeared, and the amorphous substance there constituted centres more or less voluminous, clearly defined and circumscribed by a kind of resisting membranous zone. We call these "*centres of granular disintegration*," because of the analogy they seem to bear to the lesion described by Lockhart Clarke under the same name. Transverse sections made at different levels, and sufficiently numerous, showed that these "centres," seen in the sections, corresponded to tracts running in the long axis of the cord, usually pursuing a straight course, but deviating at points, so that they, though for the most part entirely in the grey matter, here and there invaded the white substance of the cord. . . . .

3. *Change in the nerve cells.*—It is remarkable that with such marked changes in the neuroglia many of the nerve cells of the front horns preserved most of their normal characters, with a distinct nucleus and nucleolus, which were of normal size and colour. The only change seen in these cells was the absence or extreme shortness of their prolongations. Some cells of normal size were remarkable for a pronounced yellow colour. On the other hand, each section showed a considerable number of cells which had wasted to a marked degree—small, irregular, shrunken, without prolongations, and without distinguishable nucleus or nucleolus. In fact, it seemed to us, taking everything into consideration, that many cells had completely disappeared without leaving a trace.

The nerve cells had the characters just indicated in all the upper part of the cervical region. In the lower part the change was less marked. "As to the change in the grey matter, it seems commonly to be due to an irritative process, which disorganises the nerve cell, and causes finally its complete atrophy." MM. Charcot and Joffroy make the observation that "pathological anatomy seems in the present day to point to the grey matter of the anterior horns as the common seat of spinal changes, and to indicate more precisely the nerve cells of this region as the organs, a lesion of which entails damage to the muscular fibres.

4. *Change in the white columns.*—In the upper part of the cervical enlargement, *sclerosis* in different degrees occupied almost the whole of the white columns, the posterior equally with the antero-lateral, but it predominated remarkably in the hinder part of the antero-lateral, *i.e.*, in the seat of election of symmetrical ribbon-like sclerosis (*la sclérose rubanée symétrique*) when it occupies the lateral columns.

The paralysis and contractions seen in this patient seemed to MM. Charcot and Joffroy to be due to the symmetrical sclerosis of the lateral columns. This is reasonable as far as concerns the contractions, but I believe the paralytic phenomena, and also the atrophy, to have been mainly due to the damage of the anterior cells. Besides, the motor troubles began well before the contractions, and it is without doubt due to carelessness on their part that these observers have written to the effect that the paralysis appeared in the last stage of the disease; for it is mentioned that in April, 1866, a year previous to the contractions, in addition to the motor troubles of the upper limbs, which kept pace with the wasting of their muscles (*amyotrophie*), "the patient can stand and even walk, but more by an alternate shuffling of the feet than by lifting them; at the same time there is a peculiar swaying of the trunk from side to side." These functional troubles certainly indicate a paralysis of the flexors of the hip which, during walking, lift the feet from the ground (which she could not do). They further show a paralysis of the glutei medii, without the action of which the trunk must be inclined laterally at each step. Further, when I examined this patient in 1868, I questioned her specially as to her motor troubles, and I learned that for a long time before her lower limbs were stiff, they were very weak and thin; that she could not bend her feet; and that she not

only walked with difficulty, but when standing, as just mentioned, suffered great fatigue, and could only take a few steps without resting. These are the signs of true paralysis and paresis which characterise general subacute spinal paralysis, and which are not seen in progressive muscular atrophy, even when the patients have lost many of the muscles of the lower limbs.

In short, as MM. Charcot and Joffroy judiciously remark, "in this case meningitis must have been the primary lesion, whence the irritation situated in the dura mater and pia mater invaded the grey matter *viâ* the trabeculæ of the connective tissue. Lastly, it must have invaded the lateral columns, first in the cervical region, and then progressively in the whole length of the cord."

Case No. 24.—In another case of diffuse general spinal paralysis, reported in the same memoir under the name "*progressive muscular atrophy*," the symptoms (paralysis of the legs September 6th, 1864, and weakness and atrophy of the arms, with pain in the hands, September 20th, 1864), show that the spinal lesion must have started in the front horns at different levels, whence it spread to the fourth ventricle, affecting chiefly the hypoglossal nucleus (glosso-labial paralysis with atrophy) towards the end of October; and that it finally reached the antero-lateral columns (contraction of some of the muscles of the lower limbs).

It is enough to give here this summary of a typical case of diffuse general spinal paralysis reaching the highest point in the spinal cord without touching the posterior horns. (I shall return to this fact in the chapter devoted to glosso-labio-laryngeal paralysis.)

The lesion of diffuse general subacute spinal paralysis begins sometimes in the grey matter surrounding the central canal. It is then a "*subacute central peri-ependymous* (*péri-épendymaire*) *myelitis*." In such cases it radiates to the neighbouring parts, destroying them successively, and causes peripheral troubles symptomatic of these central changes.

M. H. Hallopeau, hospital "interne," communicated to the Biological Society on August 15th, 1869 (*Gazette Méd.*, 23rd July, 1870), a case of this kind (met with in M. Vulpian's wards at the Salpêtrière) with a microscopic examination of the cord. He made a very interesting memoir of the case, and collected all

the other known cases of dilatation of the central canal. Most authors have attributed this lesion to a hydro-myelitis, but M. Hallopeau has shown that it is usually caused by an inflammation of the central grey substance (*sclérose péri-épendymaire*). I photographed many transverse sections (prepared by M. Hallopeau) of the cord of this patient. Some show that sclerosis has invaded nearly all the grey substance, and produced enormous enlargement of the central canal. In other sections the canal is very slightly enlarged, but the sclerosis is not the less observable in the front and back commissures, whence it spreads to the other parts of the grey substance. This preparation shows that there had not been in this case a dropsy of the central canal, but that the lesion was a peri-ependymous sclerosis.

Of these cases of enlargement of the central canal, nearly all those in which the symptoms have been carefully noted, have afforded an almost complete collection of the morbid signs of diffuse general spinal paralysis, and shown the peri-ependymous sclerosis spreading more or less to all the constituent parts of the cord. Among these observations I will allude, in addition to those of M. Hallopeau, to those of M. O. Schüppel (*Fall von Hydromyelie, Archiv. für Heilkunde*, 1865).

Diagnosis.—The diagnosis of general subacute spinal paralysis from lesion of the front horns is very simple. In fact its symptoms may be easily summed up in a few characteristic groups.

1. Commencement usually with weakness of one or both of the lower or upper limbs, gradually involving the whole limb, either both together, or both on one side, or crossed, without fever and without pain, formication, or numbness.

2. Gradual increase of paresis progressing to complete paralysis, affecting some of or all the muscles of one part of a limb, or the entire limb, or even the entire body, in the last stage.

3. In the last stage usually troubles of speech and deglutition, like those of glosso-labio-laryngeal paralysis.

4. Usually there is lessening or loss of electro-muscular contractility, proportional to the degree of paralysis.

5. Wasting *en masse* of the limbs shortly after the onset of the paralysis, increasing gradually and in proportion to the degree of paralysis and loss of electro-muscular contractility.

6. Lessening of electro-muscular contractility in proportion

to the degree of paralysis; with integrity or slight loss of skin-sensibility.

7. Micturition and defecation normal.

8. Sometimes arrest of the disease before its last stage, with a return of voluntary power even before the electro-muscular contractility.

. . . . . .

[The disease may be confounded with—1. General paralysis of the insane. 2. Progressive muscular atrophy. 3. Lead palsy. 4. Acute spinal paralysis in its chronic stage.]

The symptoms (pains, sensory troubles, paralysis of the bladder and rectum, sacral slough), which in *diffuse general subacute spinal paralysis* are added to those just given, form a collection of diagnostic elements which renders all risk of confusion of this disease with any other kind of paralysis or atrophy impossible. It is superfluous to discuss its differential diagnosis.

Ætiology.—I have not known this form of paralysis occur before 35 or after 50 years of age. I have encountered no facts which point to heredity as a cause. The causation is most obscure. Every patient, however, is certain as to the cause of his trouble. With one, a fall had caused a strain of the foot, but in this case the paralysis did not occur till six months later! It is necessary, however, to take note of this because no other cause is discoverable. Another patient had dwelt in a damp place, and a third had often been exposed to a draught. In the majority of cases there is no appreciable cause.

I have lately had occasion to see in succession two cases of anterior general subacute spinal paralysis preceded by intense fits of pain in the belly, which might be considered as the chief cause of the progressive, descending, feverless, wasting paralysis which followed close upon it. The following is a *résumé* of the first, sent to me by M. Le Roy de Mericourt.

Case No. 25.—M. W., æt. 35, born in France, living in Brazil since 1861, strong, athletic, usually healthy. In 1865 fits of terrible abdominal pain with a deep sensation of burning, tearing, and contraction, with vomiting and distension, but with no colic nor constipation. During these fits he rolled, crying, on the floor. The fits lasted about half an hour, and continued for nine days, with half an hour's interval (about) between each fit. Six

months later he had a second attack like the first, but was able to follow his occupation in the interval. In 1870 he had a similar fit. About March 15th he noticed a tremor of the hand while writing, or when opening and shutting the hand, together with general weakness. A month later loss of power in the hands, gradually extending to the whole of the upper limbs. From the onset of the paralysis there was rapid general emaciation, with a melting away, as it were, of the paralysed muscles. There was also over sensitiveness of the paralysed muscles, and to a less extent of the skin. No trouble in micturition or defecation.

Present state.—All movements of the lower limbs are preserved, but weakened. Standing and walking are possible. The upper limbs are completely motionless, except the flexors of the fingers and thumb, which, as well as the muscles of the thenar eminence, contract feebly. The limbs are wasted *en masse*, especially the deltoids, and electro-contractility is very feeble even in the muscles which are under voluntary control. There is hyperæsthesia of the muscles increased by pressure, and considerable tremor of the fingers and hand during movement. General health fairly good. All functions normal. Intelligence intact.

The doctors at Bahia considered this case to be one of *Béribéri*, a disease which rages sporadically or epidemically in hot countries, especially amongst the coloured races. But I see nothing in the history resembling the description of "Béribéri" given by M. de Mericourt, who does not share the opinion of the Bahia doctors. Béribéri, according to M. Mericourt, "is characterised by a general sense of weakness and oppression, often accompanied by anasarca and serous effusions in the splanchnic cavities. It often also presents troubles of motility and sensibility with an ascending course." A similar case occurring in Paris shows that this form of paralysis, accompanied by abdominal troubles, is not peculiar to Brazil.

Did the fits of pain in the belly in this case produce the paralysis by a kind of reflex action? The possibility of some such relation existing between the two sets of symptoms must at least be admitted, notwithstanding the long interval between them.

But in the following case the reflex effect on the cord was apparent during the gastric attacks, and seems to give some

colour to the hypothesis of an influence by the gastric attacks on the paralytic symptoms which followed them.

Case No. 26.—M. X., æt. 32, healthy till 1864. From the age of 27 he suffered from megrim, with vomiting, which recurred at first monthly, then fortnightly, and finally every week. The attacks lasted a day, during which he could take nothing. In 1867 he had a violent gastric attack lasting fifteen days—frightful epigastric pain, internal shootings, burnings, and spasmodic contractions lasting an hour, and causing the patient to cry out. The limbs during the attack became tetanically stiff, so that the patient could not bend them. The pain was relieved by vomiting a greenish or yellowish fluid. Habitual diarrhœa; no colic; no fever; loss of appetite. Quite well between the attacks. In 1868 and 1869 he had three or four similar attacks. In 1869 the right upper limb became feeble, and trembled when he wrote. In January, 1870, he had a fresh gastric attack lasting eight days, followed by loss of power in the right arm. The day following another attack, followed by weakness in the left arm. He improved a little under electrisation, but then the left lower limb became weak, and on March 25th the hands and wrists "fall completely." On March 28th he had another gastric attack, followed by weakness of the limbs, so that he could not rise from a chair or go upstairs. This condition lasted some weeks, and was accompanied by quick pulse and sleeplessness.

Present state.—Muscles much atrophied, especially deltoids, flexors of elbow, muscles of the back of forearm, and thenar eminences. The muscles innervated by the brachial nerves (*nerfs brachiaux*) were completely paralysed, while the other muscles of the upper limbs and the muscles of the hip were paretic. When the patient opened or shut his hand, the fingers, and even the hands themselves, were agitated by a sort of tremor. Pinching or percussion of the paralysed muscles caused contractions like transverse cords, which was possibly an evidence of the integrity of their faradic contractility. This, however, was absent in the muscles supplied by the *musculo-spiral* nerve. Sensibility was normal except in the fingers, where the touch was a little obtuse. Finally, the functions of the bladder and rectum were normal.

The tetanic contractions of the limbs, which in this case

accompanied the gastric attacks, show clearly the excitement of the cord which these provoked. It is intelligible that these repeated long attacks might exercise some influence over the general subacute spinal paralysis which followed them.

But it does not follow that they were the cause of the paralysis. On the contrary, if we look upon these gastric attacks as first signs or warnings of other spinal troubles (*e.g.*, sclerosis of posterior horns) we might regard them merely as symptoms of the spinal trouble which caused the paralysis.

Prognosis.—[The prognosis is much more grave than that of acute spinal paralysis of the adult or infant, which it so closely resembles, since it may cause death if the lesion reach the bulb, by affecting the muscles of breathing or swallowing.

The disease, however, is sometimes arrested, and may even undergo improvement. "For this reason I have guarded against the use of the word 'progressive,' an expression of despair because of the fatal sense which Requin has given it in medical language." The prognosis is grave in proportion to the importance of the muscles implicated.

The diffuse variety is more grave than the other because of the greater number of troubles which it causes, troubles which are explained by the greater extent of the spinal lesion.]

Treatment by Faradisation and Continuous Currents.—When we wish to judge of the therapeutic value of any agent we must bear in mind what would have occurred if the disease for which it is used had been allowed to take its natural course. This maxim is specially applicable to subacute general spinal paralysis.

I will, for example, merely call attention to Case No. 22. In this, paralysis and atrophy invaded successively and in a few weeks the muscles of the limbs and trunk, and remained thus generalised for thirteen months. Energetic treatment (blisters to the neck, strychnine, purgatives), pursued from the beginning and continued for several months, having been unable to stop the course of the disease, the patient lost heart when it became general, and would do nothing more. Nevertheless we have seen that after remaining stationary for thirteen months the paralysis of itself gradually improved, and that in two months motility and nutrition had reached their normal state, except in the muscles of the fingers, thumb, and hand, of which

the most paralysed and atrophied were those receiving their innervation from the musculo-spiral (*radial*) nerve and the muscles of the thenar eminence; these muscles had lost their electric contractility and remained atrophied. Here, then, is a case of anterior general subacute spinal paralysis which, after having been left to itself in a stationary condition for twelve months in its generalised stage, spontaneously follows a retrograde course and is gradually cured in a few weeks, with the exception of some muscles of the hand. This is not the only case of this disease which I have seen improve spontaneously.

The lesson to be learnt from this is that we must not be too ready to give the credit of curing this disease to any particular treatment. This lesson has been forgotten by those who assert that they have cured subacute general spinal paralyses by continuous currents, and who (by a too common error in diagnosis) have classed it with progressive muscular atrophy.

The possibility of spontaneous cure diminishes the value of cures obtained by continuous or interrupted currents, but I nevertheless think I may affirm that when applied in time they hasten the cure.

But there is a time when localised faradisation must be applied, if the muscles are not to be allowed to disappear and incurable infirmities to be developed. It is when atrophic paralysis persists, as in Case No. 22, where the muscles, animated by the musculo-spiral (*radial*) and ulnar (*cubital*) nerves and those of the thenar eminence, seemed to me to be menaced the first day I saw the patient.

What kind and what method of electrisation should be used? I gave the preference to faradisation because in one of my patients who was in a similar condition I had already applied continuous currents, for twenty sittings of fifteen to twenty minutes each, without any result. Faradisation has already in ten sittings restored voluntary movement in some of these muscles (the extensores carpi radiales and the extensor carpi ulnaris), which, nevertheless, have not yet recovered their electric contractility (a phenomenon always interesting to those who have not seen it, and to which many pathologists—Charcot, Joffroy, and Hallopeau among others—can bear witness).

The "hyposthenisant" influence of a descending or centrifugal continuous current has nevertheless seemed to me to exert a

good effect upon the cord in these cases. For this reason I advise its use concurrently with localised faradisation, especially during the period of development and generalisation.

I have just used this combined electrisation to Case No. 26 with the best results . . . . I cannot ascribe the improvement entirely to the treatment, because we have seen a spontaneous cure in Case No. 22. Nevertheless I would remark that this spontaneous cure was very tardy (thirteen months), while in Case No. 26 the therapeutic action of mixed electrisation was manifested in a few sittings, and from that moment improvement was progressive.

[In Case No. 24, in consequence of the muscular hyperæsthesia, localised faradisation was not applicable, for indeed the hyperæsthesia was made worse thereby, and therefore continuous currents to the spine were employed. "I am already at the twentieth sitting," says Duchenne, "and have only obtained a feeble forward movement of the arm."]

CHAPTER V.

GLOSSO-LABIO-LARYNGEAL PARALYSIS.*

[Duchenne, in his original Paper in the *Archives Générales de Médecine*, 1868, called this disease "*progressive muscular paralysis of the tongue, soft palate, and lips;*" but he afterwards adopted the name which it now bears, proposed for it by Professor Trousseau.]

Between 1852 and 1861 I had collected thirteen cases, which have now (1870) increased to thirty-nine, of a paralytic affection which invades without known cause the muscles of the tongue and soft palate and the orbicularis oris, and which in consequence produces progressive troubles in articulation and swallowing, which at an advanced period is complicated with respiratory troubles, and in which the patients at last succumb to the impossibility of taking food, or perhaps die during a syncope.

In all my cases the disease has begun, progressed, and ended in the same manner. Its symptoms cannot be confounded with any other disease. Its group of characteristics show it to be distinct from all other muscular affections, and it consequently seems to me to be worthy of a place in our nosology and to merit a description of its own, which will form the subject of this chapter. The patients in whom I have observed this kind of paralysis have all been seen in private practice, and it has not been possible to make any post-mortem examination of those who have succumbed. I am therefore only able to lay before my readers a clinical study, but which I hope to show is sufficient to establish its differential diagnosis.

M. Dumesnil, Director of the Anatomical Studies in the School of Medicine at Rouen, published in 1860 a case which had much analogy with those which form the basis of this chapter. The case was complicated. The paralysis of the tongue, palate, and lips was in fact complicated with progressive muscular atrophy,

* From *L'Electrisation Localisée*, 3rd ed., pp. 564—595.

and the author regards the local paralysis as a variety of the latter disease. This case is narrated by an anatomist and a distinguished observer, but I shall show in the sequel that M. Dumesnil has clearly made a confusion; for the disease of which I shall treat in this chapter is a paralysis without atrophy, while progressive muscular atrophy is a lesion of muscular nutrition without paralysis. The first is seen to be perfectly isolated from beginning to end, although it may, as I shall bring facts to show, be complicated like all *morbid species* by one or several other affections, as, *e.g.*, progressive muscular atrophy. This is demonstrated by the consideration of the cases I am about to give.

Sketch of the Disease.—In 1852 I had an opportunity of seeing, with my lamented master Chomel, for the first time the progressive paralysis of the tongue, soft palate, and orbicularis oris. A patient was sent to him, as suffering from an obscure kind of angina. The history of this patient is identical with that of the similar cases which I have since collected. It presents nearly all the symptoms of the morbid species which I am going to describe, and will therefore serve me for a picture of it.

Case No. 27.—The disease with which we have to deal in the present instance was of nearly seven months duration, when the patient was sent to M. Chomel. It had begun without appreciable cause (being neither preceded nor accompanied by pain), with a difficulty in swallowing and a little trouble in articulating words. During the first two months the patient was so little troubled by it that he paid no attention to it. But it got gradually worse, and soon swallowing began to be so difficult, that he could scarcely gulp down his saliva, which sometimes dribbled from his mouth, and had to be caught in his handkerchief. Then his speech became so entangled and queer that at times he was unable to make himself understood. Active treatment (blisters round the neck, purgatives, astringent gargles) had had no influence on the course of the disease. Chomel recognised that all his troubles were probably caused by damage to the muscles of swallowing and articulation, and asked me to examine, electrically and physiologically, the muscles presiding over these functions. This is the result. The tongue was of normal size, but depressed and fixed, as it were, behind

the arch of the lower teeth; the surface was a little wrinkled, and it had very little motion. The patient could not raise its tip nor apply the back of it to the palate, he could in fact only move it a little forwards and to the side. There was no deformity of the soft palate or uvula, which contracted naturally when tickled. Phonation as regards power was normal, but great efforts were necessary for articulation. I had not observed this kind of speech-trouble in any other disease. It evidently depended on the almost entire loss of movement in the tongue. I do not know how to describe it, but one may get an idea of it by trying to speak while the tongue is kept firmly on the floor of the mouth with the tip fixed behind the teeth of the lower jaw. The voice was a little nasal. The patient could not blow with sufficient force to put out a candle, but if his nose were pinched the blast of air was strong enough to extinguish it. The articulation of the labials *p* and *b* was better when the nostrils were kept closed. These signs showed a weakness of the soft palate which one would not suspect when looking at the forcible reflex contraction produced by tickling the uvula, which did not deviate. The articulation of the labials was nevertheless not normal even when the nose was closed, and it was evident that the orbicularis contracted feebly. This was proved by asking him to say the vowel *o*, which was impossible for him; neither could he pout the lips as for kissing, and for the same reason whistling was impossible. Much thick spittle always filled the mouth; he could not spit, so that he was obliged to remove the dribbling saliva with a handkerchief. When he drank he paused between each gulp of fluid, which passed with difficulty and effort, and then a part of the fluid came back through his nose. Solid food could only be swallowed when cut very fine and mixed with fluid, and even then it was necessary to grind it a long time with the teeth. The taste and general sensibility of the tongue were normal. Finally, the patient suffered at times from great difficulty of breathing, although the respiratory movements appeared quite normal. Since the last two or three months he had become generally weak and had lost his plumpness. I proved, however, that the nutrition and movement of his muscles were intact. Faradisation made the tongue contract almost as in health; the muscles of the face, especially the orbicularis oris, and the muscles of the

soft palate retained electric contractility. Faradisation of the affected muscles for a fortnight seemed at first to improve the condition of the patient; the tongue quickly recovered its normal size;* its surface became smooth; its movements and those of the lips evidently improved, but swallowing remained difficult and saliva continued to dribble. Feeding becoming more and more difficult the patient rapidly lost strength. I then decided to apply a rheophore to the pharynx and œsophagus. This fresh faradisation was as impotent as the former. The patient, tired out, returned home, where he died some months after, exhausted for want of food. During the last months of his life it was necessary to inject beef-tea and milk slops into his stomach with the stomach pump. His hunger was never satisfied; then the attacks of suffocation became more and more frequent, and in one of them the patient died.

The functional troubles in this case were clearly due to a paralysis of the tongue, and to a want of power in the muscles of swallowing and articulation. (We shall see presently that the attacks of suffocation and syncope which occurred at times were evidence of another lesion.)

The case I have just narrated gives a sufficiently good picture of the symptoms which I have seen in all the other patients. It is thus that their disease has progressed and ended. But we must expect to meet with individual differences, as is the case with all morbid species.

Let us now deal with each of the symptoms of the disease.

The paralysis of the tongue usually appears first. It is the chief symptom of the disease, for it is that which threatens life by impeding nutrition. The troubles which it causes in pronunciation are characteristic. . . . The palatal and dental sounds are articulated like *ch*, and this peculiarity is in proportion to the difficulty of raising the tongue. Speech becomes more and more unintelligible by the progressive weakness of the movements of the tongue, and when this can move no more the articulation of the palatal and dental sounds becomes quite impossible, and the patient can only make a sort of grumbling noise. Swallowing is interfered with at the same time as articulation. This causes but little trouble at first, but later on the patient is unable to swallow even liquids, and then

* Duchenne has previously said that the tongue was of normal size.—Ed.

the mouth fills with saliva, which dribbles incessantly and soaks his handkerchief.

The increase in the quantity and stickiness of the saliva is explained as follows:—In health the saliva is swallowed as soon as secreted, and at each effort of swallowing—an effort which is made incessantly and instinctively—the tip and sides of the tongue are strongly applied to the corresponding parts of the roof of the mouth, so as to make a gutter inclining from before back, and from above down; then all parts of the dorsum of the tongue from the tip to the base are pressed in succession against the palate.

The moment these movements are weakened, the saliva is incompletely swallowed, and later this becomes impossible; it then accumulates in the mouth, and gets sticky by its long stay there. Then it dribbles abundantly, and when the patient opens his mouth a number of threads and columns of sticky spittle are seen adhering by their ends to the lips, tongue, and palate. This saliva is sometimes so sticky that the patient can scarcely separate it from the walls of the cheeks, and it bothers him so that he is continually cleaning his mouth with his fingers and handkerchief. There is, however, neither redness nor any change in the mucous membrane of the mouth or pharynx.

Soon solids are no better swallowed than liquids, and semi-liquid foods and soups can alone be taken; and finally, when the tongue is completely paralysed, swallowing is as impossible as when the tongue is depressed and the mouth kept widely open. The patients, in order to swallow, are then obliged to throw their heads back and hold their hands before the mouth, in order to prevent the food from being pushed out again by the efforts to swallow. It is only necessary to recall to mind the great importance of the tongue in the first and second stages of swallowing to understand the gravity of a complete paralysis of all the muscles charged with the performance of this duty.

Paralysis of the soft palate enormously increases the troubles caused by the paralysis of the tongue. Articulation is affected by it. The articulation of those labials which remained normal becomes changed. *p* and *b* are then spoken like *me*, *fe* or *re*, because the posterior nares can no longer be shut off by the sort of sphincter formed by the soft palate and the superior constrictor of the pharynx—a sphincter discovered by Gerdy and still

better studied by Dzondi, and to the mechanism of which I shall have occasion to refer. The expelled column of air, instead of going entirely through the mouth and separating with more or less force the lips held close for the articulation of labials, divides into two columns, one of which travels by the open nasal orifice, and makes a nasal sound, while the other separates the lips softly and feebly. This division of the out-going air makes the articulation of the other consonants, already altered by the paralysis of the tongue, still more confused. In addition to the difficulty of swallowing, this paralysis of the palate causes a part of the food and drink to return by the nose. It is easy, when the paralysis is limited to or is most marked on one side, to recognise it by the swerving of the uvula, or the inequality of the palatal arches. But in most cases I have seen neither, because the paralysis is equal on the two sides. This paralysis of the palate might easily be missed, the more so that its sensibility being usually sound or but slightly impaired, tickling causes reflex contractions of the palate, and of the pharynx also, as is evident during efforts of vomiting. The nasal phonation and the faulty articulation of the labials, as above described, are, in this case, the only symptoms which give warning of its presence. A certain proof of it can be acquired if we compel the volume of air to go with greater force through the mouth by pinching the patient's nose while he is speaking, and thus succeed in making the labial sounds much more clear.

Paralysis of the orbicularis oris and pterygoid muscles.

The *orbicularis oris* is paralysed gradually. The patients have at first a difficulty in speaking the vowels *o*, *u* plainly, as if the lips were semi-paralysed by the cold, and soon they cannot contract the muscles for whistling or kissing. Before long they are unable to hold their lips close enough for speaking labials plainly, and later, when the weakness increases, their articulation becomes impossible. At this time I have sometimes seen the elevator of the lower lip (by the muscle of the tuft of the chin) considerably weakened, or even completely paralysed. Sometimes, also, the depressor labii inferioris and the depressor anguli oris are affected, so that the patients are no longer able to pronounce the vowels *e* and *i*.* I have never seen the orbicularis pal-

* All the vowels alluded to in the text are to be pronounced in the *French* and not in the *English* fashion.

pebrarum nor the zygomatici, levatores anguli oris or levator labii superioris alæque nasi appreciably affected in this disease, and I do not think that the buccinator is affected.

At first sight one might be led to think that this last muscle is paralysed because the patient cannot whistle, but the least amount of attention shows that this is due to the paralysis of the orbicularis oris. In fact if the lips are held together with the fingers while the patient is made to blow, the cheeks can be seen to contract against the alveolar border, instead of swelling out as when the buccinator is paralysed.

The paralysis of the orbicularis oris gives a predominating tonic force to the muscles which move the angles of the mouth and act on the upper lip. From this it results that when the mouth is shut the line between the lips is lengthened transversely, and the naso-labial lines get more hollow and circular, thus giving a look of weeping to the face. I have noticed this peculiar countenance in all my patients. I have seen one case indeed in which the lips separated in all directions during laughing or crying, the patient being unable to bring them to their normal position. All the teeth then remained exposed until he had pulled his lips together with his fingers. Is it not interesting to note that in this disease the paralysis is localised in the muscles which preside over the same functions—those which control articulation of words and swallowing. For this reason, when the disease is completely developed, the patient can only make inarticulate sounds, and when he wishes to swallow, liquids, or even a bolus of food, will come back through the lips and nostrils.

I must mention, besides, the paralysis of the *pterygoids*. This is shown by asking the patients to perform the movement of grinding food ("*diduction*") which they can no longer do in an advanced stage of the disease. I had neglected to investigate this fact when I wrote my first memoir, but have since then noticed it in every case. This is how I was led to discover it. At a certain time the patients show a difficulty in *grinding* solid food, although they can *divide* it with force. I then ascertained that they could move the jaw up and down with force, but that the grinding movement which is performed by the pterygoids was lost. We shall see later, while studying the pathological anatomy of this disease, how this paralysis of the

pterygoids is accounted for by the histological examination of the bulb.

Paralysis of the Bronchial (intrinsic expiratory) Muscles of Reissessen, and of the Pneumogastric.—Respiratory troubles are added to those I have just described; viz., recurring attacks of suffocation, which get more frequent as the disease approaches its end. They are often brought on by movement, especially walking, but also occur without known cause. They happen during the day or night. I have seen many of these attacks, and have ascertained by auscultation that at the first there exists apparently no trouble in the mechanism of respiration; and that besides, there is no paralysis nor spasm of the diaphragm. These fits of suffocation are sometimes complicated by syncopes, which get worse in the final stage and may prove fatal. Three of the last cases which I saw in the course of the year 1859 ended in this way.

[A case is given (Obs. xcix. in original) of death by suffocation, in which, however, there are no new facts except with regard to the paralysis of the tongue. Duchenne noticed that when he raised the tongue with his fingers, the patient could depress it forcibly, thus proving that "the depressors of the tongue were not at all paralysed, and it was doubtless their tonic action predominating over that of their antagonists which kept the tongue fixed to the floor of the mouth."]

. . . . . .

I have had opportunities of observing these attacks in my private practice, and have ascertained that the patients die sometimes of asphyxia and sometimes of syncope. These facts have put me on the track of a number of signs of lung or heart trouble, apparently trivial at their outset, but of extreme gravity because they are inevitably fatal when they reach a certain degree of intensity. (I shall try anon to explain these symptoms by pathological anatomy and physiology.)

1. The respiratory troubles show themselves by decreased power of expiration. The patients begin to show fatigue and rapid exhaustion when they talk, and it is evident that their expiratory power is much weakened, because they cannot blow out a candle Nothing, however, appears abnormal in the extent or rhythm of the respiratory movements. No râles are heard on auscultation, and if the patient be made to breathe deeply the

air is heard to enter the bronchi forcibly during inspiration, which is long, while the noise of the expiratory sound is very short. Although at each inspiration a great quantity of air enters the bronchi unopposed, the patient is incessantly short of breath; he expresses it by saying that his chest is always full, and often makes vain efforts to expire, trying to empty the lungs by contracting the abdominal muscles. The uneasiness he feels is like that experienced when, after a big inspiration, only a very little of the air which has entered the chest is allowed to escape. In a word, it is the prolonged stay in the air vesicles of non-respirable air (deprived of its normal quantity of oxygen and charged with carbonic acid) which causes this uneasiness. I have elsewhere shown (*Physiologie des mouvements, &c.*, p. 688, 1866) that these respiratory troubles must result from a paresis of the bronchial muscles of Reissessen. It is plain that the patient can then neither blow the nose, nor spit, nor cough with ease, and that the least bronchitis may endanger his life by causing asphyxia. This state is permanent, but in different degrees; it gives rise to those attacks of suffocation with cyanosis (doubtless by a more or less complete paralysis of the bronchial muscles), which may end, as I have often seen, in asphyxia. (I would allude to the fact that I have noticed this kind of asphyxia, due to paralysis of the expiratory muscles of Reissessen, in diphtheria.)

2. The cardiac troubles are shown by a sense of swooning, by a kind of cardiac oppression with extreme anxiety and fear of impending death and by a very quick (140), irregular, intermittent, and small pulse. No murmur is heard on auscultation, but the valvular sounds are very confused, as though the heart were working in a fluid; the face is pale and the eyes dull. Syncopal attacks are not uncommon, and in one of these the patient usually dies.

In my first memoir I omitted to mention an accident which is a consequence of the difficulty in swallowing, and especially of the paralysis of those muscles which carry the tongue backwards and move the epiglottis on to the glottis—I mean the passage of saliva, food, or drink, into the trachea during swallowing. All my patients have complained of this introduction of liquids into the air passages at an advanced stage of the affection.

[Duchenne describes the case of a patient who was nearly suffocated in his presence from the passage of saliva into the trachea.]

Is it not possible that some of these patients may have died in this way, especially when during sleep the saliva, accumulated in the mouth, has flowed into the air passages?

Troubles of Phonation.—At an advanced stage, when articulation is no longer possible, troubles of phonation are observed which I will try to analyse and explain. I have already said that the muscles presiding over the mechanism of breathing are not paralysed; of this I have assured myself by getting the patients to inspire and expire slowly and deeply. When they can no longer articulate, speech tires them so that, after uttering a few tolerably loud sounds, they feel exhausted and their voice becomes weak. This might be explained by the efforts which they make to be understood, for, strangely enough, they have a mania for wishing to talk, even when they can only make inarticulate sounds. One of my patients always answered questions in a strong voice, although he could only say the vowel *a*. He knew, however, that he could not be understood. After long efforts which exhausted him his voice failed, and then he used to decide to make himself understood by signs or writing.

I noticed troubles of phonation in other patients who had arrived at the same stage of the disease. Does this weakness of voice depend on a particular nervous condition, similar to that which causes the weak and fatiguing phonation in patients with paralysis of the palate? or does there exist at the same time a certain degree of paralysis of the nerves presiding over phonation, *e.g.*, the recurrent laryngeal? I believe that these two causes act in different degrees. Thus I have seen a lady who, in a very advanced stage of this disease, had a weak voice when she attempted to speak though unable to articulate, but during hysterical fits, to which she was subject, she uttered piercing shrieks. Later the paralysis of the larynx becomes more evident. I have seen, *e.g.*, one lady whose voice was almost completely extinguished as soon as she lost the power of articulating. But this is the only case where aphonia was so marked. Other patients whom I have seen till death preserved the power of phonation, though weakened. (I ascertained

by the laryngoscope that in these cases the vocal cords were relaxed.)

General Symptoms.—There is no fever. Fever ordinarily comes on only during the final stage. I have not once noticed it during the onset or course of the disease.

The digestion is only too good. Although the patients can only take soups their appetite is never satisfied. Their craving for solid food and for drink is all the greater because their sense of taste is usually preserved. They in fact suffer the torture of Tantalus.

They lose their strength gradually, but are not paralysed; the proof of this has been their ability to come to my room for treatment in a very advanced stage of the disease. They went up and down many stairs every day. They begin to grow weak as soon as they cannot swallow solid food or satisfy their appetite. They attribute their weakness to this cause only, and with reason, although I must make some reservations on this head.

Ought we not to take into consideration the loss of saliva as one of the causes of this weakness? I have indeed noticed that all my patients lost strength as soon as they could not swallow their saliva. The influence of saliva on digestion is well known. Besides this, experiments on animals seem to show that saliva is necessary for nutrition. Professor Vella, of Turin, made parotid salivary fistulæ in a horse so that all the saliva flowed away. The animal's digestion in no way suffered, but swallowing merely became difficult through the dryness of the food-bolus, and in order to facilitate it, it was necessary to mix the food with liquids. Although richly fed, especially with oats, it soon became weak and thin. This experiment was made publicly at the Veterinary School of Turin. M. Vella told me of it when I showed him two patients affected with glosso-labio-laryngeal paralysis. He thought that their weakness might in part be attributed to the loss of the saliva which they had not been able to swallow for many months.

But is there no other kind of weakness in our patients but that due to troubles of nutrition? It must be remembered that they not unfrequently have attacks of giddiness, which cause them to fall while standing or walking unsupported. Is it not possible that the central lesion which causes the giddiness is

also answerable for some of the general weakness? To be quite accurate, I ought to mention that in a case which I saw with Trousseau one of the upper limbs was notably weak. It was then the only case of the kind I had seen. Was it due to an exceptional extension of the paralysis, or to a complication? This must be determined hereafter by pathological observation.

Lastly, the intelligence remains unaltered. I must not, however, forget to mention that some patients who are deprived of speech (notably women) become very emotional in an advanced stage of the disease. The least allusion to their sad situation causes tears. This moral state is explained by the despair into which the unhappy patients fall when they see everything fail to combat the disease, and see themselves menaced by death from hunger or thirst. Their desperate state unceasingly occupies the mind, but their highly emotional condition is not due to any intellectual failure, for their intellects, on the contrary, remain intact till the very last hour.

Course of the Disease.—Judging by my reported cases, the tongue muscles are usually first affected. Later the palate muscles suffer, and after these the orbicularis oris. Finally, at a later stage the attacks of suffocation and syncope come on. The functional troubles which precede or accompany the attacks of suffocation occur tolerably early in the disease, but the heart troubles supervene only in the last stage. The breathing troubles are constant, but the heart troubles are sometimes absent. Such is the usual order of the symptoms. In one case, however, the palate and orbicularis were affected before the tongue.

Pathological anatomy teaches that the lesion of this disease is an atrophy of the nerve origins situated in the "bulb." On the other hand pathological physiology compels us to think that this anatomical lesion has a tendency to spread progressively from above down, beginning with the lower cells of the nucleus of the hypoglossal, to the spinal accessory and pneumogastric. But, indeed, I have seen one case of the disease pursue a contrary course. (2nd Edition, Obs. cxlv., p. 635.)

The course of the disease is always chronic. I have never seen it last less than six months nor more than three years.

It has not retrograded in its course, and in general has not remained stationary, no matter what treatment has been em-

ployed. The prognosis is very grave, and the name *progressive paralysis*, which Requin gave to every paralytic affection which once born, marches relentlessly to a fatal end, is certainly applicable to it.

Diagnosis.—[In the early stages this is often difficult, but when the disease is well established the diagnosis is easy enough. It may be confounded with—

1. *Simple pharyngitis and stomatitis*, but the entire absence of any inflammatory appearances ought to make an error impossible.

2. *Simple paralysis of the palate and pharynx.*—Simple paralysis of the palate may cause food and liquid to return by the nose, but it never prevents the swallowing of the saliva. In simple paralysis of the palate contraction of the same by tickling cannot be obtained, but this possibility of reflex irritation is always present in glosso-labio-laryngeal paralysis.

Duchenne has never seen paralysis of the upper constrictor of the pharynx present in this disease. The manner in which food and drink are thrust forward in the mouth and nose at the moment of swallowing is a proof of the integrity of the function of this muscle. He does not deny the possibility of a paralysis of the pharynx, but as a matter of fact he has never seen it present in glosso-labio-laryngeal paralysis.

3. *Paralysis of the seventh pair* could only be confounded with glosso-labio-laryngeal paralysis in those rare cases where the facial paralysis is *double*. Even here error may be avoided by noting that electric contractility is usually unchanged in labio-glosso-laryngeal paralysis, but diminished or lost in double facial paralysis.

4. *Atrophy of the tongue in progressive muscular atrophy.*—In progressive muscular atrophy it is infinitely rare to have atrophy of the tongue before atrophy of other muscles. Out of 200 cases observed by Duchenne this never occurred, but he mentions one case of a Neapolitan in whom at the outset of progressive muscular atrophy the tongue and interossei were affected simultaneously.

Great stress is laid upon the fact that *in progressive muscular atrophy the tongue wastes but is not paralysed*, movement being preserved as long as a muscular fibre remains, while in *glosso-labio-laryngeal paralysis the tongue is paralysed, but does not*

waste. It follows from this that the troubles of deglutition and articulation are not usually so strongly marked in progressive muscular atrophy. In this latter disease the wasting sometimes spreads to the pharynx and œsophagus, so that the food bolus can only be forced down, as it were, by drinking water, and liquid may be heard to fall into the stomach with a noise as if it were being poured into a "carafe." This never occurs in glosso-labio-laryngeal paralysis.

5. *Associated diseases.*—"Glosso-labio-laryngeal paralysis may exist together with another muscular affection. From this it results that the observer, if he has not had opportunities for studying each of the diseases separately, and especially if he has not sufficient knowledge of these two 'morbid species,' may only recognise in their accidental association one single disease. I myself was exposed to this error of diagnosis in a case of progressive muscular atrophy coinciding with a progressive paralysis of the tongue." Duchenne has seen only thirteen cases of involvement of the tongue out of 150 cases of progressive muscular atrophy, the tongue being affected only in a late stage of the disease, but he records a case in which, in the early stage of progressive muscular atrophy, the tongue was completely paralysed without being wasted, and this he regarded as a concurrence of two "morbid species."]

Ætiology and Pathological Anatomy.—I have searched carefully, but in vain, for the possible causes of this affection. Once it seemed to occur under the influence of a deep sorrow. In two other cases which I have not related, there was a history of tertiary syphilis in one, and in the other a rheumatic condition which had shown itself for many years by rheumatic pains. In no other case have I been able to discover the least appreciable cause. The disease has come as it were insidiously, and has taken by surprise those whose lives gave promise of a long and happy period of health. I could give many examples of this. For example, M. le Comte de X. arrived at fifty years of age without ever having been ill. Muscular and with a fine strong constitution, he had passed a happy existence till, without suffering and without known cause, he was smitten with this terrible malady. Or again, to give another example, M. P., having always enjoyed good health, and with a fine constitution, retired from business at the age of forty-five, hoping to enjoy the large fortune he had

amassed. Then he was smitten without cause with this disease, of which no one suspected the gravity, and which was rapidly approaching its fatal termination when I was called to see him. Of a convivial nature; loving life, wine, and good cheer, and accustomed to taste like an epicure every morsel that passed his lips, he wept because, although perpetually hungry and thirsty, he no longer had the power to swallow, and was maddened by his inability to speak; while he was conscious of his approaching end, and felt himself, as he said, dying of hunger.

Glosso-labio-laryngeal paralysis is a disease of adult life.

The disease is, I think, tolerably frequent, for I had seen fifteen cases of it in 1861, and seven in less than a year. Within two months of the publication of my memoir fourteen new cases were communicated to me by professional brethren. This made a total of twenty-nine cases in 1861. At the present time (1871) I have seen thirty-nine cases, without counting a good number that I saw in London.

Object of my Icono-Photographic Researches on the Intimate Structure of the Healthy Human Medulla.—I had no sooner published my clinical study of this disease (in 1860), which had not up to that time been described, than I was seized with a desire to investigate the physiological explanation of the series of symptoms which it presented. My clinical facts had established—1. That glosso-labio-laryngeal paralysis affects first the articulation of lingual consonants, and a little later that of the labials, the loss of power being progressive. 2. That at the same time swallowing is affected, and becomes more and more difficult; that the voice becomes nasal, and that the "diduction" movement of the lower jaw is paralysed by degrees. 3. At a more advanced stage the power of expiration and phonation is weakened. 4. In the last stage the action of the heart is sometimes deeply affected, so that the heart suddenly ceases to beat, and death is instantaneous.

Writing in 1860 I said, "Why does the anatomical lesion in this disease always affect the muscles presiding over the articulation of words and deglutition? Can there exist a central lesion, either anatomical or dynamic, which explains the peripheral distribution of the troubles? *It would be necessary, in this morbid species, that one single lesion should affect the origin, without*

spreading to the nerves, of—1, *the hypoglossal;* 2, *the motor nerve-fibres of the soft palate;* 3, *those of the lips;* 4, *the spinal accessory, and possibly the pneumogastric.*" These words prove that clinical observation had already made me foresee that the central organs (the groups of cells) presiding over the mechanism of speech must be sufficiently close to each other to be successively, or sometimes simultaneously, affected by the same lesion. I did not conceal from myself the difficulties of solving this anatomical question.

I then sought enlightenment from the beautiful works of Stilling, Schroeder Van der Kolk, and Luys, but I could not find any certain agreement between my photographs from sections of the medulla and the pictures published by these authors.

I was thus brought to a standstill in my investigations, when in 1867 Lockhart Clarke, the celebrated English microscopist, who had come to visit our Exhibition, was good enough to show me, from beautiful sections of the medulla which he had prepared himself, the structure of this region of the cerebro-spinal axis.

He especially pointed out the nuclei or masses of cells forming the intra-medullary origin of the nerves, and the multiple fibres which emanate from them to put them in communication with other nuclei, *e.g.*, the *fasciculus teres* (the nerve fibres surrounding and adjacent to the seventh pair, and which go thence in many directions). Thanks to these ideas I was able to continue my iconographic and photographic investigations on the intimate structure of the bulb, and by the aid of the solar microscope to photograph from nature figures of great importance to this branch of study. I enter upon these details because I wish it to be understood that the chief merit of my iconographic and photographic investigation of the bulb is to confirm and—if I dare say so—to complete the recent beautiful work of Lockhart Clarke on the intimate structure of the human medulla (*Philosophical Transactions, 2nd Series, Part I.*, 1868).

Certain important micrographic details which there was need of showing have been brought to light by my photographs. It will be seen, for example, that in the white substance of the medulla there are many very fine nerve tubules ($0{\cdot}0033^{mm}$),

mixed with big and medium-sized tubules (·01^{mm} to ·02^{mm} and ·03^{mm}).

[Here follows a chronological account of Duchenne's photographic labours, beginning in 1864 with his first communication on the subject to the Academy of Science, and ending in 1869. It contains nothing bearing on the minute anatomy of the bulb, or of glosso-labio-laryngeal paralysis.]

First Hypotheses on the Pathological Physiology of Glosso-Labio-Laryngeal Paralysis.—By the help of photographs of anatomical facts the explanation of the symptoms and course of glosso-labio-laryngeal paralysis becomes very easy. Thus it will be seen—1. That usually the lesion, by attacking first the nucleus of the hypo-glossal, occasions the difficulty of articulating the linguals. 2. That soon this lesion, by spreading to the cells which form the termination of the seventh pair, renders difficult and then impossible the articulation of the labials. 3. That in a more advanced stage the same lesion attacks the nucleus of the spinal accessory, situated more posteriorly, and then bronchial expiration and phonation get gradually weak by the want of innervation of the muscles of Reissessen and those of the larynx. 4. The nasal tone, the paralysis of the palate, and especially the paralysis of the pterygoids, are accounted for by the fact that the cells of origin of the motor branch of the fifth descend in the medulla as low as (outside and in front of) the grey tubercle of Rolando. 5. It is easy to understand that the lesion usually travelling from below upwards, attacks the nucleus of the vagus in the last stage, causing the irregular action of the heart, and finally its stoppage. 6. Lastly, having, by the order in which the symptoms appear, established the fact that the lesion which characterises this disease attacks progressively in its ascending course the nuclei of the nerves situated in the upper part of the medulla, and further that it cannot pass over the nucleus of the pneumogastric without causing death, it will be understood why the functions of nerves whose central nuclei are situated above the nucleus of the vagus (the superior cells of the facial, the motor branch of the fifth, the sensory portions of the fifth, the glosso-pharyngeal, the auditory and the motor nerves of the eye) are not affected in this disease.

Investigations on the central anatomical lesion of glosso-labio-laryngeal paralysis. — The post-mortem examinations which have been made since I wrote the above, have taught us that the damage is in the bulb. M. Luys and I thought we had proved, in many of those who had died of this disease in the wards of Professor Trousseau, that there existed a *sclerosis of the bulb*, because we found a considerable number of amyloid bodies. Now, being better acquainted with the anatomy of the bulb, and more familiar with the preparation of microscopic specimens, I should like to know whether or not there is any change in the cells.

My first hypothesis on this disease is now a reality. It is in fact perfectly established by many recent autopsies, in which many microscopic examinations of transverse sections of the bulb and pons (*protubérance*) were made, that the fundamental lesion in this disease is in the nuclei of the bulbar nerves, as I had foreseen in 1860, when resting my hypothesis entirely on the symptoms.

This discovery is due to Professor Charcot, who has further attempted to prove that the lesion is characterised by a primitive atrophy of the cells.

In what does this change consist? "The accumulation of yellow pigment seems," says M. Charcot, "to play an important part, to be, as it were, the beginning; the atrophy of the cellular prolongations, of the nucleus, and lastly of the nucleolus, are secondary facts. Have we to do with a slow irritative process, or, on the contrary, with a progressive atrophy? Nothing can be decided by the anatomical characters alone, but I may be permitted to state my belief that the disease process, whatever it be, attacks the cell in the first instance." (Charcot, *Arch. de Physiol.*, Mars et Avril, 1870, p. 247. See also Charcot and Joffroy, *Arch. de Physiol.*, vol. ii., 1869.)

Pathogeny.—In M. Charcot's cases the paralyses symptomatic of the lesions of the bulbar nuclei were successive and progressive, as in all my cases. (I have seen thirty-five cases, and in one only the lesion travelled from above down, attacking in the first instance the seventh and sixth pairs of nerves.) It is not to be doubted that the innervating cells of the paralysed muscles have been smitten successively and in proportion to the amount

of paralysis. But at what height or in what point of the nuclei were situated the cells first attacked? Were they affected from below upwards, or, so to say, step by step, as appeared to me to be the case in 1868, from the morbid process, the topography and the connections of the bulbar nuclei? The value of this hypothesis cannot be definitely settled until after a great number of microscopic examinations made at different stages of the disease. Photography will assist in making this question clear. The points which are visible in many of the plates, from an album which I am now making designed to show the lesions of the bulb, and especially its anatomical elements, enable me to count upon such a result.

Further, photographs of sections made at different levels in the bulb of patients who had suffered from glosso-labio-laryngeal paralysis show—1. That changes in the cells already exist to a slight extent at the upper part of the nucleus of the hypoglossal and a little below the pons varolii. 2. That the lesions have a maximum intensity at the level of the middle part of the olivary bodies, and especially at the level of the tip of the calamus scriptorius. 3. That they get less marked in the sections made near the lower extremity of the hypoglossal nucleus. 4. That the nucleus of the facial shows changes still more general, and at the same level as those of the hypoglossal. 5. That in the same plane of the bulb the nuclei of the spinal accessory and vagus have an abnormal pigmentation, which is well marked, but that the cells are much less atrophied than those of the hypoglossal and facial.

The pathogeny of this disease is still unknown. Why does the anatomical lesion of this *morbid entity* localise itself in the cells of the bulb? Why does it—at least if we are to judge by the symptoms—begin below and spread upwards? Why, in this disease, does not the atrophy of the bulbar cells produce granular and fatty degeneration of the paralysed muscles, as in similar atrophies of those cells of the cord which are called motor, and which are trophic, as has been shown by clinical observation and pathological anatomy? Why is the paralysis of the muscles the only symptom of this morbid species? These questions as to pathogeny and disease process have still to be solved.

Note by the Editor.

The four preceding chapters, from the *Electrisation Localisée*, have been grouped together because they contain an account of the clinical phenomena due to changes limited (as the majority of pathologists are agreed) mainly to one tract of the cerebro-spinal axis, viz., the motor cells situated in the front horns of the spinal cord and their prolongation upwards in the medulla oblongata.

The extinction of any of the motor cells of this system causes a palsy of the muscular area supplied by those nerve fibres which issue from the extinct cells; and the muscular area paralysed, which is proportionate to the number of motor cells extinguished, may vary in extent from a fractional part of one small muscle to the muscles of all four limbs and the trunk.

The mode of extinction of the motor cells doubtless differs in different cases. Inflammation, congestion, hæmorrhage, embolism, thrombosis, sclerosis, or other degenerative change, pressure, new growths, &c., are all theoretically competent to destroy the cells.

In cases of infantile paralysis and acute spinal paralysis, the *polio-myelitis anterior acuta* of recent writers, the pathological change is assumed to be inflammatory, and with regard to progressive muscular atrophy, the *protopathic spinal progressive amyotrophy* of Charcot, this writer says (New Syd. Soc., vol. xc., p. 173), "We find here in chronic form alterations which we have described in connection with infantile paralysis; and there is reason to suppose that, as in the latter case, the limiting of the alterations to the anterior cornua of the grey substance is accounted for by the circumstance that the ganglionic elements are the primary seat of the affection."

M. Charcot suggests the name of *chronic parenchymatous tephro-myelitis* as calculated to impress the nature of the pathological change upon the mind. Beyond the fact, however, of the ultimate extinction of the motor cells of the front horns, which undoubtedly occurs in all the diseases grouped in the

preceding chapters, we know little; their mode of extinction is still a matter more of probabilities than certainties, and I would humbly suggest that M. Charcot's barbarous phraseology is not calculated to assist either the pathologist or the student.

Acute spinal paralysis is rarely fatal, so rarely that post-mortem examinations of recent cases are exceedingly uncommon. In the thirtieth volume of the Pathological Society's Transactions (1879) will be found a most interesting description of the spinal cord "from a case of acute anterior polio-myelitis in a child, fatal within six weeks from the onset." The case is recorded by Dr. Charlewood Turner, the autopsy having been made by Mr. R. W. Parker. In this case the anterior grey columns were the seat of well-marked inflammation, recognisable by the naked eye, and confirmed by the discovery of inflammatory products with the microscope. In the left front horn in the lumbar region there was a well-marked hæmorrhagic extravasation. The posterior cornua were affected, but to a less extent, and the lumbar section (at the level of greatest pathological change) showed that "the whole grey substance was abundantly infiltrated with leucocytes disseminated through it singly and in clusters."

The history of the case was to the effect that the child (æt. 2½ years) slipped down-stairs on November 1st, 1877, and fell on her back. Ten days later it seemed unwell; on November 14th it complained of being cold, and lost power in the legs; and on the 16th it began to lose power in the arms. She was admitted to the London Hospital, under Mr. Hutchinson, on November 29th. At this time the legs were quite powerless, and the arms almost quite. Bending the neck forward caused pain. Sensation was *completely lost in the legs*, and there was loss of power over the sphincter of the bowel. The temperature was slightly raised. On December 7th the temperature was normal, movement and sensation returning in the legs, and in the arms movement was quite free. The evacuations were still involuntary. On December 17th measles set in, and she died on the 25th, with dyspnœa and suppression of urine. The loss of sensation in the legs and the want of control over the bowels seem to me to make it doubtful whether this case should be considered as one of ordinary infantile paralysis, a doubt which is strengthened by the post-mortem. The able reporters of the case recognise this

doubt and they say, "The anterior grey columns being primarily the seat of such acute inflammatory lesions, the extent and degree to which adjacent parts of the grey centre are affected in any case must vary with the severity of the primary lesion." (This is very true: inflammation, as we understand the process, is rarely limited to one or other of the physiological "columns," and it is precisely because the lesion so rarely spreads beyond the limits of the front horns in spinal paralysis that we doubt whether the lesion in ordinary cases is really inflammatory.) "In the present case the changes in the posterior grey columns appear to have been of a secondary character, and although such as one might expect to be followed by some permanent impairment of the sensibility of the lower limbs, do not seem to afford any ground for removing the case from the category of polio-myelitis anterior acuta, or infantile paralysis."

"With the exception of the persistence of the usually more transient disturbance of the action of the sphincters, the clinical course of the case followed that so characteristic of infantile paralysis, in the rapid recovery of sensation (probably partially only in the lower limbs) and in great part of mobility in the upper limbs, and in some degree of the lower limbs also."

The above mentioned exceptions to the usual course of the disease seem to me so important as to warrant one in considering that the case was not one of infantile paralysis of the ordinary type.

In the same volume are two other cases recorded by Dr. F. Taylor and Dr. Henry Humphreys respectively. Dr. Taylor's case was one of paralysis of the left leg of nearly two years standing, in a child aged 3, which died of broncho-pneumonia in Guy's Hospital.

Post-mortem, the left side of the lumbar portion of the spinal cord was found to be smaller than the right, the left front horn especially so. Microscopically there was an almost complete absence of ganglion cells and axis cylinders in the left front horn, together with a felt-like appearance (composed of closely matted very fine fibres) of the tissue forming the basis of the grey matter, and scattered throughout were connective tissue nuclei "of the same size as, and no more numerous

than, in the normal grey cornu structure." The antero-lateral columns and the anterior nerve roots showed a similar sort of change, with diminution (in size and quantity) of nerve fibres and increase of fibrous tissue. The left posterior half of the cord was small, but otherwise healthy. The right side of the cord showed a change similar in character to, but infinitely less in degree, than that on the left side, notwithstanding that the movements of the right leg were said to be good.

The history of Dr. Humphreys' case was very like that of Dr. Taylor's, the child (æt. $3\frac{1}{2}$) having died in the Pendlebury Hospital of scarlet fever about two years after the paralytic attack which affected the left leg. Here too in the lumbar region the ganglion cells were found very deficient in number in the left front horn. The microscopic examination clearly established this fact, but, in the words of Dr. Humphreys, threw "no light upon the cause of the disappearance of the cells." In the same way it would be impossible to say that in Dr. Taylor's case the cause of the trouble had been inflammation.

It must not be assumed that, because the early stage of infantile paralysis is accompanied by elevation of temperature, the lesion is necessarily inflammatory. In very young children the temperature is most unstable, and those who have been connected with a children's hospital will know that a rise of temperature lasting a few days is often accompanied by no other symptoms, is followed by no appreciable effects, and is not unfrequently due to an error in diet or some such trifling cause. Any trouble tending to derange the circulation in the spinal cord (especially its upper part) would be especially likely to influence the temperature, and it is by no means necessary that such trouble should be inflammatory.

The fact that the paralysis is at first very wide-spread, and then tends to clear up more or less quickly and completely, points, I think, to the probability that congestion is an important factor in the early stage of the disease.

In the seventh volume of the Transactions of the Clinical Society, the late Dr. Anstie has recorded the following case. A young gentleman, aged 13, consulted Dr. Anstie for some swollen cervical glands on September 23rd, 1873. He went

back to school, sailed on the river, played football, &c. On September 29th there was stiffness of the legs, on October 4th he could not stand; the paralysis of the legs got worse, then the arms were affected, then the muscles of the neck and the respiratory muscles suffered, and he died suffocated on October 7th. Sensation in this case was scarcely affected, the pulse was quick (100—120), temperature normal, intellect clear, no affection of the sphincters.

Post-mortem, intense congestion of the spinal meninges was the only morbid change discovered. The cord was not examined microscopically.

The motor tracts were mainly affected in this case. Dr. Anstie was careful to exclude diphtheria as a cause, and indeed the symptoms are not altogether those of diphtherial paralysis. It has always seemed to me that this was a case of spinal paralysis fatal in its earliest stage from the implication of the respiratory muscles.

The doctrine of identity of the pathological changes in acute spinal paralysis and progressive muscular atrophy, the latter being merely a repetition in chronic form of the pathological changes seen in the former, places us under the necessity of believing that an identical lesion (destruction of the motor cells of the front horn) is capable of giving rise to two sets of symptoms which have hardly any points in common. Usually there is a strong family likeness between the acute and chronic form of a disease, but the symptoms of acute destruction of the motor cells of the front horn (spinal paralysis) are so very different from those said to be caused by their chronic destruction (progressive muscular atrophy) that one is driven to the conclusion that the microscope has not yet revealed the whole truth concerning these changes. The differences between the symptoms of these two forms of paralysis I have arranged in tabular form.

PROGRESSIVE MUSCULAR ATROPHY, OR CREEPING PALSY—("chronic anterior poliomyelitis") (?)	INFANTILE OR SPINAL PARALYSIS. (Acute anterior poliomyelitis.)
Chronic, and spreads gradually.	Acute and REGRESSES.
Muscles attacked individually.	Large groups of muscles usually attacked at first.
Attacks the upper limbs by preference.	Tends to settle in the lower limbs.
Often quasi-symmetrical in its distribution.	Very rarely symmetrical.
Weakness of muscles is proportioned to their wasting, and paralysis is not absolute till the wasting is complete.	Paralysis absolute from the first, and wasting follows gradually.
Fibrillary quivering of muscles is common.	No fibrillary quivering.
Muscles give normal reactions to electricity.	Muscles give "degenerative reactions" to electricity.

Thus we find, according to the above table, that in the acute form the disease is regressive, tends to settle in the legs, and is rarely symmetrical; many muscles are paretic and the palsy of a few is absolute from the first, they give degenerative reactions, are not the seat of fibrillary tremors, and waste rapidly.

In the chronic form the disease is progressive, tends to settle in the upper limbs, is usually quasi-symmetrical; individual muscles are picked out in which weakness and wasting march *pari passu*, they respond normally to electricity, and are the seat of fibrillary tremors. These two diseases never complicate each other, and the one never gives place to the other, and yet we are asked to believe that pathologically they are (except in respect of the time element) identical.

I do not mean to deny the accuracy of the statements made by pathologists as to the seat and nature of the lesions found in these two diseases, but I feel sure that the pathological fact which causes the difference between the two has yet to be discovered.

Some pathologists have held that the change in progressive

muscular atrophy is primarily in the muscles. Of this there is no sufficient evidence at present.

There are two features of progressive muscular atrophy which call for some remark. The fibrillary tremor of the muscles certainly occurs independently of spinal change. It is very common in the orbicularis palpebrarum, where it occurs as a phenomenon attendant on gouty dyspepsia. Quivering of the muscles in other parts of the body is not uncommon in gouty subjects. It is very common also as a result of bruising of the nerves. It is certain that the fibrillary tremor in progressive muscular atrophy must not be taken as an evidence of primary spinal change.

Neither I think must the quasi-symmetry so often seen in this disease be taken as an evidence of spinal origin.

Chronic pathological changes in the spinal cord tend to run vertically rather than transversely, and it is, possibly, scarcely more easy for a pathological change to travel from one front horn of the spinal cord to the other than from one half of the brain to the other. Symmetry of peripheral lesions is so common (*e.g.*, skin eruptions) in cases where there are no sufficient grounds for assuming that the spinal cord is implicated in the morbid process, that the quasi-symmetry of progressive muscular atrophy might almost be used as an argument in favour of the disease being primarily peripheral rather than central.

The fact also that muscles are picked out for wasting in such a remarkable way, that a wasted muscle is often surrounded by others which are apparently in perfect health, might also I think be used as an argument against the destruction of the motor cells being the primary change. The function of the plexuses seems to be, according to Professors Ferrier and Yeo, and according to the reasoning which I put forward in the Bradshaw Lecture of 1881 (*Lancet*, vol. ii., 1881, p. 405), to scatter, as it were, the effect of the motor cells. A study of the brachial plexus seems to show that most of the muscles supplied by it are innervated by a considerable length of the cervical enlargement, and that while limited lesions of the spinal cord would cause a very limited paralysis, the area of paresis would be larger. The phenomenon so often seen in spinal paralysis of a few paralysed muscles surrounded by many others which are paretic seems to find its explanation in the fact that, owing to the plexus, a very small

lesion in the cord makes itself widely felt, and if this explanation be accepted it is difficult to explain on the spinal origin theory the fact that, in progressive muscular atrophy, paresis in muscles, other than those which are actually wasted, is not seen.

The origin and order of development of the pathological changes which constitute progressive muscular atrophy must still be looked upon as questions which are *sub judice*.

Mention has just been made of the fact that in spinal paralysis the muscles when tested with electricity give the "degenerative reactions." This term does not occur in the writings of Duchenne, so that it may be as well to state briefly what is meant by it.

Muscles in a state of health contract when they are stimulated by electric currents. The induced or faradic current (which was used almost exclusively by Duchenne) causes an apparently continuous contraction of a muscle for as long as it is applied to it. The galvanic current, on the other hand, only causes contraction of the muscle at the moment when the current is made or broken. The readiness with which a muscle responds to the galvanic current depends upon the pole (positive or negative, *anode* or *cathode*) which is applied to it, and the mode in which the current is made to vary, whether by closing or opening the circuit. Contraction is most easily obtained when the cathode (negative pole) is applied to the muscle, and the circuit is suddenly closed. This is called the C. C. C. or cathodal closure contraction. With slightly stronger currents we may get, in addition, muscular contractions when the anode (positive pole) is applied to the muscle, and the circuit is either closed or opened. These are called the A. C. C. and the A. O. C. (anodal closure and anodal opening contraction), and sometimes the one and sometimes the other appears most easily. Lastly, with very strong currents C. O. C., or cathodal opening contraction, occurs. The order of occurrence of the muscular contraction (the strength of the current being gradually increased) is, therefore—

1. C. C. C.
2. { A. C. C.
   { A. O. C.
3. C. O. C.

To obtain these reactions one pole of the battery should be applied to a distant and slightly sensitive part of the body, such

as the back, while the other is accurately applied over the muscle or the motor nerve supplying it.

The alteration in pole and the sudden opening and closing of the circuit should all be managed by means of a commutator. These matters will be found in greater detail in the editor's *Text Book of Electricity in Medicine and Surgery* (Smith & Elder).

The term "degenerative reaction" means that the muscle ceases to respond to the faradic current, no matter how strong, but continues to respond to the galvanic current. The response to the galvanic current is more easily produced than in health, and it sometimes happens that A. C. C. is produced more easily than C. C. C.

The degenerative reactions are supposed to be due to the degeneration of the motor nerves supplying the muscle. The electric contraction of health is brought about by stimulating the motor twigs ramifying in the muscle. When these nerve-endings degenerate they are no longer excitable, and the contraction produced is due to the direct effect of the galvanism on the muscle tissue without the intervention of nerves. When the muscle is wasted and lost, of course no contraction occurs with any form of stimulation.

Degenerative reactions occur—

1. Whenever a muscle is cut off from all nervous communication with the spinal cord.

2. When the motor cells of the spinal cord from which the nerve-twigs supplying the muscle emanate are destroyed.

Degenerative reactions do not occur in brain paralysis, nor in paraplegia, provided the muscles which are paralysed are connected to a healthy bit of the spinal cord by means of healthy nerves. Duchenne was well aware of the dying out of faradic muscular irritability, and of the circumstances which produced it, but he apparently had no knowledge of the peculiar reaction to galvanism which such muscles afford.

A few words may be said as to the treatment of *glosso-labio-laryngeal* paralysis.

I believe that the sufferings and annoyances of these patients may be very greatly lessened by a judicious use of electricity. One of the great troubles of these patients is the constant dribbling of saliva. The orbicularis oris loses tone and wastes, and as a consequence of this the lower lip droops, and over the drooping

and inert lip the saliva (which the patient cannot swallow, owing to the paresis of the tongue and pharynx) falls in an almost continuous stream. Now it is a well-known fact that the tone of muscles which are utterly and irretrievably paralysed to the will may be greatly improved by the application of any form of electricity to which they will respond. In cases of paraplegia the tone of the sphincter ani may not unfrequently be sufficiently restored to lessen considerably the annoyance of incontinence of fœces, and in cases of incurable facial palsy sufficient tone may be given to the orbicularis palpebrarum to prevent the drooping of the lower eyelid, the exposure of the red conjunctiva, and that state of conjunctival catarrh which causes an incessant flow of tears over the cheek. So in cases of glosso-labio-laryngeal paralysis a judicious use of electricity will give tone to the lip, tongue, and pharynx, and by pursing up the mouth, will prevent the constant dribbling which is so annoying to the patient and his friends.

That form of current (galvanic or faradic) must be used to which the muscles most readily respond, and I am accustomed to employ a silver tea-spoon for one of the rheophores. Various rheophores for use in the mouth have been made by instrument makers, but none of them are so convenient as a tea-spoon, an article which we have all been accustomed to put into our mouths, and which is at once convenient, cleanly, and unobjectionable. If the conductors of the battery be made (as they always should be) of simple telegraph wire (copper, covered with gutta-percha), all that is necessary is to lay bare a couple of inches of the bright copper, and to twist it round the spoon just above the bowl. If the spoon be held by the handle in the dry hand of the operator, and the bowl be introduced into the mouth of the patient, the current will take the course of least resistance, *i.e.*, through the bowl to the moist mucous membrane.

If it is wished to act upon the orbicularis oris, the spoon should be attached to the positive pole (when galvanism is used), and being held in the right hand of the operator, should be introduced with its convexity towards the cheek, so as to cause a slight bulging of the angle of the mouth, cheek, and upper and lower lips. The skin of the cheek and lips should then be lightly stroked with the negative rheophore, for which

the most convenient form is the sponge-holder, invented by Kidder, of New York. This will be found to produce an effective contraction of the orbicularis oris, buccinator, and adjoining muscles, with the result, after a few applications, of increasing their tone, and thereby adding considerably to the comfort of the patient.

For acting upon the tongue and pharynx, it will be found convenient to reverse the position of the spoon, holding it by the bowl (which must be wiped dry), and introducing the handle into the mouth. Then, if the negative sponge-holder be held beneath the lower jaw, over the region of the hyoid bone, and the tongue and pharynx be stroked with the handle of the spoon, most efficient contractions of the muscles of the tongue and of many other muscles concerned in deglutition will be produced. I have had one case under my care in which a very remarkable amount of improvement in the patient's condition was produced by constant electrifying with the aid of a spoon-rheophore.

CHAPTER VI.

PSEUDO-HYPERTROPHIC PARALYSIS, OR MYO-SCLEROTIC PARALYSIS.*

[This disease was described by Duchenne in the *Archives Générales de Médecine* in January and February, 1868.]

PSEUDO-HYPERTROPHIC PARALYSIS is a disease of infancy or youth, which I believe to be not very uncommon, but which nevertheless has not been yet described. Its progress is in general towards a fatal termination, and for this reason it must be classified with diseases having a progressive tendency. This affection, serious from its very commencement, deceives the friends for a long time, and deludes them by giving the limbs an appearance of great muscularity.

This disease is mainly characterised—1. By feebleness of movement, usually situated at first in the muscles of the lower extremities and of the lumbar spine, ultimately spreading progressively to the upper limbs, and increasing in intensity till all movement is lost.

2. By the gradual increase in size of most of the paretic muscles.

3. By the increase of the interstitial connective tissue of the paretic muscles, and in the more advanced stages by an abundant production either of fibrous tissue or of fat globules.

The name of *pseudo-hypertrophic paralysis*, which I have given to this disease, seems to me a good one, because it has reference to the symptoms, and appeals, so to say, to the eye. It may also be called *myo-sclerotic paralysis*, a name which is more scientific and justified by pathological anatomy.

[It was in 1858 that Duchenne first suspected the existence of this disease, and his attention being once drawn to the concurrence of the characteristic symptoms he had little difficulty in collecting instances of it, and in the second edition of *Electrisation Localisée* (1861) he published his first description of the

* From the last edition of the *Electrisation Localisée*, pp. 595—616.

new malady, which he proposed to call "*hypertrophic paraplegia of infancy.*"]

The observations recorded by me, and which form the basis of my work, are thirteen in number.*

Pathological Anatomy.—1. *Nerve centres.*

The condition of the nerve centres in pseudo-hypertrophic paralysis has been examined in one interesting case, the clinical history and post-mortem examination of which were published by Eulenberg and Cohnheim in 1863, and I reproduced an abstract of them in my memoir in the *Archives Gén. de Med.* in 1868. As I considered that it was not wise to draw any hasty conclusion from a negative fact, I wrote as follows:—"Notwithstanding the great authority of this able micrographer, one of the most distinguished of the former assistants of Professor Virchow, and now well known as a master, this negative result, of great value in itself, must be confirmed by fresh autopsies before opinion can be definitely fixed with regard to the pathological anatomy of the nerve centres in pseudo-hypertrophic paralysis."

2. *Muscles.*

I have been much blamed abroad for having, in the different pathological investigations which I have published, neglected and despised pathological anatomy, that branch of science inseparable from all good clinical work. I recognised the reproach, and wished to expose myself to it no longer. I waited in vain for an autopsy to furnish me with an opportunity of examining the condition of the muscles whose size had increased so excessively, and seeking for an explanation of the two contradictory facts co-existing in my little patients, viz., paresis or paralysis and muscular hypertrophy.

The appeal which I made to observers in 1861 when describing this new disease was listened to in Germany, where my researches in muscular pathology and physiology, as I am bound to acknowledge, have always met with a most cordial reception, and have become popular sooner than in France. From 1861 to 1866 fifteen cases of pseudo-hypertrophic paralysis have been published there. Moreover, a great

* I could now bring together some forty cases of pseudo-hypertrophic paralysis, without counting those which I saw in 1871 in the London hospitals, where this disease of infancy appears to be tolerably common.

advance has been made in the pathological anatomy of this disease by the impulse given to it by Griesinger, who, with the assistance of Billroth, ventured to excise a portion of the deltoid muscle of a little boy in order to make an histological examination of it. . . . [To avoid a like serious proceeding I have devised a little instrument, which I have named the "tissue punch" (*emporte pièce histologique*), by means of which, with very little pain, I have removed minute portions from the deep parts of muscles. It has been of great use to me for many years, and has never caused any serious trouble. Thanks to this instrument I have been able to complete my clinical observations by researches into the pathological condition of the muscles of living patients.

The punch must be introduced at right angles to the direction of the muscle, for if the hook do not take the fibres transversely no tissue may be brought back with it.

In order to diminish the pain I keep the skin tightly stretched, and insert and withdraw the instrument very rapidly. The patient then feels nothing but a slight shock, and children will hardly cry if one is careful to conceal the instrument.

This little operation has never been followed by any accident. It is true that I have never neglected to displace the skin laterally while stretching it so that the cutaneous and muscular wounds should not coincide.

The instrument must be carefully cleaned after use. The dangers of punctured wounds are known too well to make it necessary for me to insist on this point; and further, if the instrument be not clean the microscopic preparation will be spoiled by foreign matter. To clean the instrument it must be taken to pieces, and the portions which have penetrated the skin wiped after steeping them in alcohol, which, unlike water, does not cause rust.]

In 1865 I first examined muscular fibres removed during life from different parts of one of my little patients, and from that moment I possessed all the necessary elements for a complete description of pseudo-hypertrophic paralysis. But since the results of this examination differed somewhat from those obtained in Germany I was anxious to wait for an opportunity of submitting them to the judgment of other micrographers. This

mode of investigation had been refused to me in many cases, when in 1867 M. Bergeron informed me that there was under his care at the Sainte Eugénie Hospital a little boy suffering from the kind of muscular hypertrophy of which I had shown a case in 1861. My learned colleague, who has made an interesting communication on the subject to the *Société Médicale des hôpitaux*, most obligingly aided my researches by allowing me to remove from this little patient, repeatedly and at different stages of the disease, portions of his hypertrophied muscles, some of which were examined by our most expert micrographers. The result of this examination having fully confirmed my previous observations, I decided to publish it. The following is an abstract of my conclusions.

1. Hyperplasia of the interstitial connective tissue, with production of more or less fibrous tissue, is the fundamental anatomical lesion of the muscles in pseudo-hypertrophic paralysis.

2. This hyperplasia is present in all the paralysed muscles when they begin to increase in size. This fact justifies the name *myo-sclerotic paralysis*, based upon the anatomical condition, a name which I proposed for it in opposition to, or complementary to (*en regard de*) the name *pseudo-hypertrophic paralysis* based upon its symptoms.

3. The increase in size of the muscles is in direct proportion to the hyperplasia of the interstitial fibroid and connective tissue.

4. This increase of fibroid and connective tissue is usually associated with a small number of fat vesicles, or, as has been observed in Germany, it may be replaced by a large number of fat vesicles. This latter condition appears to me to constitute the most advanced stage in the change of the interstitial connective tissue.

5. According to my own observations the transverse striation of the muscular fibres is preserved, in most of them, in all their length; but it becomes very faint and scarcely apparent. At the points where the transverse striation has disappeared are seen longitudinal markings, and sometimes, when these become effaced, the sheaths of sarcolemma appear to contain fat cells, which, however, are in reality derived from the surrounding connective tissue, and which otherwise differ from the fatty

granules which are characteristic of fatty degeneration of the muscle.

6. The hyperplasia of the interstitial connective tissue appears generally only in the second stage of the disease, and it appears to me to be preceded by a congested condition (*état fluxionnaire*) of the muscles, which occasions also a slight increase in their volume. At this period (first stage of the disease) the transverse striation is often exceedingly faint.

Contrary to the opinion of M. Heller and of MM. Eulenberg and Cohnheim, the interstitial fibroid and connective tissue is not composed of empty sheaths of sarcolemma; in other words, it is not produced by a fibroid degeneration. This fact has been perfectly established by the late M. Ordoñez, who wished to call this kind of alteration in the muscular fibres "fibroid substitution." The small number of primitive fasciculi found in the centre of the muscles in pseudo-hypertrophic paralysis might lead one to suppose that many of them have disappeared, and that empty sheaths are found in greater or less number amongst the connective tissue, or, indeed, that this tissue is produced by a fibroid degeneration of the muscular tissue. The small number of the primitive fasciculi has called forth the remark from M. A. Heller "that some of the muscular fibres disappear, for if the newly formed fatty masses were merely added to the normal volume of the muscles, the total volume ought to be more considerable than it is."

Symptoms and Course of the Disease.—Pseudo-hypertrophic paralysis may be divided into three distinct periods. 1. A period of feeble movements. 2. A period of apparent muscular hypertrophy. 3. A period of extension and aggravation of the paralysis. This, at least, is the result of my clinical experience.

First Period.—This is characterised by, (*a*) weakness, usually limited to the muscles of the lower limbs; (*b*) certain troubles in standing and walking, *i.e.*, separation of the legs, lumbo-sacral curvature, amounting very often to the saddle-back condition, and a waddling of the trunk during walking.

This first period is of short duration compared with the others (a few months or a year). In most of my cases my knowledge of this period has been derived from the statements of friends; but these statements have been so accurate, and have

shown so much agreement, that I cannot entertain the least doubt as to the reality of the paralytic condition at the outset. I have elsewhere had occasion to demonstrate this condition in two cases recorded in my original memoir.

The functional troubles observed in this first period continue to increase in the second, which is characterised by an excessive increase in volume of a certain number of muscles.

Second Period.—The apparent hypertrophy which is characteristic of this period shows itself first of all in the gastrocnemii, where it attracts the attention of those who have the care of the little patients. I have thus been able to learn that this hypertrophy does not commence till some months or a year after the outset of the paralysis. It spreads progressively from the gastrocnemii to other muscles, being either limited to some few of the weakened muscles, or invading nearly all of them. In one of my cases the deltoids began to increase in volume some months after the gastrocnemii.

The two first stages of pseudo-hypertrophic paralysis may be intermingled. In one case, at least, the weakness and enlargement seem to have started together, or to have dated from birth; but still the information which I have been able to get on the morbid phenomena observed in early infancy has been uncertain or incomplete.

Whatever may be the time of appearance and the mode of extension of the muscular hypertrophy, the increase in size takes place progressively, and requires a considerable time (a year or eighteen months) to attain its maximum. Then the disease remains stationary for several years (two or three, or sometimes more). This condition might be considered as a separate stage of the disease (*on pourrait en faire une période d'état*).

Third Period.—The third period is marked by an increase and extension of the paresis to the upper limbs, if the lower limbs only were affected in the first instance.

Then, function having hitherto remained intact, the elevation of the arm becomes difficult, and ultimately impossible, and the other movements of the limbs grow weak and are ultimately lost.

Nevertheless the muscles do not increase in size in this third period as in the second; on the contrary, they remain thin in comparison with some of the muscles of the lower limbs.

The enlarged muscles gradually lose what mobility they had, till the patients, arrived at adolescence, are usually obliged to remain always lying or sitting.

Since the disease is unaccompanied by fever, and the digestive, respiratory, and circulatory functions remain normal, the little patients may still live a long time. At length in the last stage they become greatly exhausted, and are carried off rapidly by some intercurrent disease.

Diagnosis.—The elements for a diagnosis are derived from the symptoms, and from a recognition of the anatomical change displayed by fragments of muscle removed by the tissue punch, and examined with the microscope.

Pseudo-hypertrophic paralysis might be confounded with—

1. Progressive muscular atrophy of children.

2. Atrophic (spinal) paralysis of children.

3. The halting gait, caused in children by an arrested development of the co-ordinating faculty which controls equilibrium and the instinctive movements of walking, or by certain cerebral lesions.

4. Certain other pathological and abnormal conditions.

The differential diagnosis from these diseases will be evident by a comparison of the diagnostic elements arranged in parallel columns :—

DIAGNOSIS OF

Progressive muscular atrophy of children } from { *Pseudo-hypertrophic paralysis.*

1. Commences usually between 5th and 7th year, in the face, where certain muscles, such as the orbicularis oris and the zygomatici, are the first to atrophy. After remaining stationary for some years (two or three) it invades the limbs and trunk, where it progresses as in the adult, *i.e.*, by a "descending progress," attacking first the muscles of the upper limbs and trunk, and only reaching	1. Begins by weakness in the lower limbs, and makes an "ascending progress," and only invades the upper limbs and certain muscles of the face (principally the temporals and masseters) in the most advanced stage.

the lower limbs in the most advanced stage.

2. The muscles waste partially and irregularly one after another, and feebleness only affects those movements which involve the wasted muscles, and in a degree directly proportioned to their atrophy. From this there results a partial abolition of many movements, and various deformed attitudes of the limbs during repose.

3. The muscular fibres undergo a fatty and granular degeneration, with a fatty substitution in the interstitial tissue, as in the progressive muscular atrophy of the adult.

4. Movement is not abolished until after the change in the muscle.

2. The feebleness invades, simultaneously at the outset, all the muscles which move many articulations; and at a later period some, or more rarely all, of the affected muscles increase in size.

3. The interstitial connective tissue increases with an abundant production of fibrous tissue, and ultimately of fat vesicles. The primitive fasciculi usually preserve their striation (which becomes very faint), but decrease in size.

4. Movement is weakened, or abolished, notwithstanding that the fasciculi retain their faint striation.

DIAGNOSIS OF

Atrophic spinal paralysis of children } from { *Pseudo-hypertrophic paralysis.*

1. In most cases there is pyrexia at the outset.

2. At the outset the paralysis may be *general*, or *paraplegic*, or *hemiplegic*, or *crossed*, or limited to *one limb* or to *part of one limb*, and the muscles are paralysed completely from the first. Those whose innervation is the least damaged soon re-

1. No pyrexia at any period.

2. At the outset the muscles of the lower limbs and the extensors of the back are affected, but only with feebleness. In the last stage only is movement completely lost.

cover their power of movement, but the others waste considerably, or undergo different degrees of textural change.

3. In the early stage the electric irritability of the muscles is weakened, or lost, although they may be texturally unchanged.

4. In an advanced stage the loss of movement is soon followed by wasting of the muscles, more or less rapid, and marked with tissue change in proportion to the nervous lesion.

5. The loss of power of voluntary contraction and electric irritability indicates the textural change of the muscles, in the advanced period (the fibres having undergone a fatty-granular degeneration, and the interstitial tissue a "fatty substitution" change).

6. Reason, analogy, comparative pathology, and some anatomical facts, demonstrate that atrophic paralysis of infancy is caused by a primary lesion of the anterior cells of the spinal cord.

3. In the early stage the electric irritability of the muscles is normal.

4. Feebleness of movement is followed before long by an increase in size of a greater or less number of the muscles, without destruction of their fibres.

5. When in the last stage the big muscles have, so to say, melted away, and the limbs appear wasted, the microscope shows that the primitive fibres retain their striation, and that the connective tissue is mixed with fibroid tissue and more or less fatty vesicles. These histologic characters serve to distinguish pseudo-hypertrophic paralysis from the fatty atrophic paralysis of childhood.

6. There are no facts in the pathogeny of pseudo-hypertrophic paralysis which point towards a primary spinal lesion. A new clinical fact, observed by MM. Bergeron, Charcot, and myself, has shown that the cerebro-spinal centre is without organic change.

DIAGNOSIS OF

The halting gait caused in children by arrest of development of the co-ordinating faculty which controls equilibrium and the instinctive movements of walking — from — *The halting gait of pseudo-hypertrophic paralysis.*

1. When the child begins to walk there is no separation of the legs, no saddle-back condition, nor waddling.

2. The striation of the muscular fibres is normal.

1. There is separation of the legs, saddle-back condition, and waddling.

2. The striation of the muscular fibres is faint.

DIAGNOSIS OF

The halting gait due to cerebral lesion — from — *The halting gait of pseudo-hypertrophic paralysis.*

1. Intelligence more or less obtuse, speech slow and difficult.

There is generally dribbling of saliva.

2. When the lesion is grave, voluntary movements of the lower limbs provoke reflex contractions.

1. Intelligence often obtuse, speech slow, and articulation a little defective.

No dribbling of saliva.

2. Voluntary movements cause no reflex contractions.

It is only necessary to mention certain other conditions which might be mistaken for pseudo-hypertrophic paralysis to put the reader on his guard against them. For example—

1. In some children with muscular lower limbs contrasting with their upper limbs, one might be disposed to attribute a weakness due to disease of the lumbar vertebræ or other troubles to the onset of pseudo-hypertrophic paralysis.

2. Polysarkia cannot be confounded with pseudo-hypertrophic paralysis, if it be remembered that in the latter disease the sub-cutaneous tissue contains very little fat, and is so thin

that the hypertrophied muscles look like herniæ beneath the skin.

Ætiology and Pathogeny.—Pseudo-hypertrophic paralysis is a disease of infancy. Up to the present time it appears to be more prevalent in boys than girls.

It has been observed in several children of the same family. This is the only kind of heredity which has yet been established with regard to it.

The pathogeny is obscure, and since pathological anatomy has not at present shown any appreciable change in the nerve centres, how is the progressive feebleness of movement to be explained? Neither the compression nor the separation of the muscular fibres by the hypertrophied connective tissue will serve to explain this derangement of voluntary contraction, because the latter precedes the former and is not in direct ratio to the overgrowth of the connective tissue.

The growth stimulus (*l'irritation formatrice*) which causes the abundant proliferation, and the other changes of the interstitial connective tissue of the muscles, appears to me to be in this case the probable cause of their weakness.

But how shall we explain the alteration itself of the interstitial connective tissue?

That is a problem which has yet to be solved.

Prognosis and Treatment.—The prognosis of pseudo-hypertrophic paralysis is grave. In fact, whenever I have met with this disease when it has reached the stage of proliferation of the interstitial connective tissue, I have always seen it progress steadily towards generalisation, and terminate by complete loss of movement and death during adolescence.

But it may be cured in the first stage, before the onset of the connective tissue overgrowth. For this reason I have not called it a *progressive* paralysis, *i.e.*, according to Requin (the inventor of this fatal name), a disease which, once established, always progresses towards a fatal termination.

I haved cured pseudo-hypertrophic paralysis in its early stage by muscular faradisation, assisted by hydropathy and shampooing. When this treatment has been used after the beginning of the second stage, it has only produced a temporary alleviation, and has not prevented the progress and fatal termination of the disease. Other and very varied therapeutic agents (strychnine,

ergot, iodide of potassium, &c.) have proved equally unavailing in the second stage. At the present time I am trying the effect of continuous currents.

Case No. 28.—Pseudo-hypertrophic paralysis. The patient is a boy aged 10. The father is strong and robust. The mother also is healthy, and having been abandoned by the father of the patient, married again, and has two children, both of whom have an exaggerated development of the lower limbs, and a gradually increasing arching of the spine like that of their elder brother. Their mother considers them to be suffering from the same disease. (This is another example of heredity in addition to those already mentioned.) She nursed the eldest till it was seven months old. It was then a fine child, and remained so, in spite of its premature weaning and coarse feeding. At two years of age the child was noted for its size, but still could not walk. At this time it was sent to the mother's family in the country, and the mother, who only saw it at long intervals, can only give very imperfect details of the progress of the disease. She says it ultimately walked like other children, but always in an awkward manner.

A lady with whom the mother was in service, and whose own daughter was being nursed in the same family as the patient, was able to observe it for many years whenever she went to see her own child. She has made the following communication to me:—At two years of age the limbs and trunk, which had been rather big since birth, did not exceed normal proportions. It was such a fine child that she wished her own daughter were similarly developed. It was only towards the age of four that the calves commenced to be appreciably enlarged. A little later she remarked that this excessive development showed itself in all parts of the body, and that parts of the surface of the skin stood out in relief. Meanwhile its walking, though always peculiar, did not become more difficult. In fact, whether standing or walking, he was obliged to separate his legs and bend his body backwards in order to balance himself. He waddled in walking, could not run like other children, could not go up-stairs, and often fell down. If he bent forward he could only recover his position by catching hold of the furniture, or by supporting his hands on his thighs. Up to the age of eight his power of movement, such as it was, remained sufficient for him to go

to school daily, a distance of one kilometre. Walking then became gradually more difficult as the limbs, which had always been very large, took on an unusual development. For about the last year movement has become almost impossible, so that when the child falls it cannot get up again. He can now only manage to traverse a few yards on a level floor, and that with great difficulty. It is a notable peculiarity that he cannot walk when held by the arm or hand, but manages better by himself to perform those voluntary movements which are necessary to maintain his equilibrium. These movements consist in an alternate lateral inclination of the trunk towards the limb which is resting on the ground. The word "*waddling*" perfectly expresses his mode of walking. I will only add that while standing or walking the boy can only maintain his balance by bending the back strongly forward, although when seated this curvature completely disappears.

All the muscles, with the exception of the pectorales, present for the age of the child a development so truly monstrous as to remind one exactly of the Farnese Hercules or the muscular studies of Michael Angelo. The facial muscles seem to have undergone the same change, and to exercise their functions as badly as those of the limbs, and one must attribute the lack of expression in the face, in part at least, to this fact.

M. Bergeron (who made a communication on this patient to the Société Médical des Hôpitaux, on May 24th, 1867) was struck not only by the extraordinary size, but also by the hardness of the muscles, even during repose. On pressing them the sensation was similar to that produced by palpating the skin of a child or adult who is the subject of sclerema, and this, combined with the powerlessness of muscles so strong in appearance, induced him to think that it was not the muscle itself which was hypertrophied, but that probably the connective tissue of the muscle was the seat of an hypertrophy and induration more or less analogous to sclerema; in short, that the paralysis was due to a separation of the muscular fibres by an abnormally developed cellulo-fatty tissue, and the microscopic examination appeared to him to justify this view. It must be added that the attachments of the muscles are not thickened, and that the tendons are clearly defined; that the joints are freely movable, and that the skeleton is in harmony with the age of the patient; that the skin is

normally thin, supple, and free from fatty pads, and that the muscles alone were affected by hypertrophy.

Electric contractility was weakened, but nevertheless persisted. There was no other functional trouble; the general health was excellent, and the intelligence ill-developed, though the patient was not an idiot. We will add one more fact, that he weighed about 75 lbs.

The patient died on February 13th, 1871, aged 14 years. Since the publication of his case in my memoir of 1868 he had gradually become weaker, and in about two years had become entirely paralysed, notwithstanding an increase in the muscular hypertrophy. On February 8th he was seized with moderately severe bronchitis, to which he succumbed in three days, although it was not accompanied by pneumonia or any other organic lesion, as was proved by the post-mortem examination. [The details of the bronchitis present no points of interest.]

This shows how, in the last stage of pseudo-hypertrophic paralysis, the patients are unable to resist even the most trifling intercurrent diseases, to which they generally succumb. It is in this way that I have always seen the disease terminate.

M. Bergeron placed at my disposal the spinal cord and a great many of the muscles of this patient, whose post-mortem examination was made thirty-six hours after death by M. Henszel.

The brain and cerebro-spinal meninges, examined when fresh, were perfectly healthy.

The cord and muscles were hardened in a solution of chromic acid. Being desirous of giving additional value to their histological examination, I requested my friend M. Charcot to be good enough to assist me in it.

A number of sections of the cord taken from the cervical and dorsal regions, and all prepared with great skill by one of his pupils, M. Pierret, were found to be typically healthy.

The hypertrophied muscles, when fresh, looked like fatty tissue, and had the same appearance as the little fragments removed by my tissue punch at various stages of the disease, more especially a few months before death. These muscles were preserved in a weak solution of chromic acid. I distributed some pieces of them to MM. Charcot, Vulpian, L. Clarke, and other pathologists accustomed to make histological preparations, and made transverse sections and teasings of them myself.

The microscopic examination of these specimens showed the muscular change of pseudo-hypertrophic paralysis in an extreme degree, *i.e.*, the muscular fibres smaller than usual, but still preserving as a rule a faint striation, and separated from each other by a large quantity of fatty tissue, mixed with fibrous tissue and wavy fibres in small quantity.

These recent facts go to confirm the ideas which I put forward in 1868 with regard to the distinctive anatomical changes and pathogeny of this disease, which show it to be a morbid entity clearly distinguished from other diseases which are characterised by lesions affecting muscular nutrition.

The following conclusions seem to me to result from the last case, corroborated as it is by similar facts previously observed by MM. Cohnheim and Eulenberg.

1. The irritation [*travail irritatif* (*irritation formatrice*)] which causes the multiplication of the interstitial connective tissue and its secondary fatty change is not symptomatic of any appreciable lesion in the nerve centres.

2. At the outset of this disease the muscular fibres grow pale and their striation becomes faint (their force diminishing at the same time); a little later the interstitial tissue increases; and in a more advanced stage fat cells, more or less big and plentiful, mingle with the interstitial fibroid tissue. It will be remembered that by the aid of the tissue punch I had shown by pathological research on the living subject, what had already been demonstrated on the living by Billroth and Griesinger, viz., that in the final stage the increased connective tissue is changed into fat.

The histological examination of this patient's muscles confirm, therefore, what Cohnheim had seen in the dead subject and Billroth in the living. But it will be generally admitted that the photographs which I have made from the beautiful carmine-stained preparations of M. Pierret, show better than has previously been done the transition of the fibroid interstitial growth into fat. M. Charcot has drawn the same conclusion from the microscopical examination of this case, in a communication made to the Société de Biologie in October, 1871. My photographs show also in the perimysium of the muscles many enlarged capillary vessels which prove the existence of irritation (*travail irritatif*) in this peripheral disease.

3. Numerous post-mortem examinations, strengthened by

microscopical investigation, have established beyond doubt that the change of muscular nutrition seen in a certain number of paralytic and other affections (acute and subacute spinal paralyses, progressive muscular atrophy) always corresponds with an atrophy of the anterior cells of the spinal cord. The anatomical and pathological facts which have just been given show that pseudo-hypertrophic paralysis cannot be ranged among the same morbid species.

Note by the Editor.

In his account of the typical case of this interesting disease Duchenne says, "All the muscles, with the exception of the pectorals, present, for the age of the child, a development so truly monstrous as to remind one exactly of the Farnese Hercules or the muscular studies of Michael Angelo."

There can be no doubt that this "Infant Hercules" was, as a type of the disease, almost unique. The features were so striking that they could not fail to excite the attention of Duchenne, and this exceptional case has been the pioneer of a clinical advance. This excessive pseudo-hypertrophy is very rare, and is now seldom seen in cases which we recognise as "Duchenne's paralysis."

In Gowers' able monograph on *Pseudo-hypertrophic Muscular Paralysis* (Churchill, 1879) notes of many cases are given, and one cannot fail to be struck with the exceedingly small share which the large muscles have in the general features of the disease.

The muscles forming the calf of the leg are the ones most usually enlarged, and these muscles, be it observed, are usually contracted and the tendo-achillis "drawn up." In twenty-six of the cases recorded and collected by Gowers in which details of the muscular condition are given, the calves are mentioned as being big in twenty-one of them, but in no single instance do the calves appear to have had "Herculean proportions," and it is a question whether in many cases they were not merely big by contrast with the general muscular wasting, for it must not be forgotten that in the cases observed in this country

wasting of muscle has been a far more important feature than seeming overgrowth.

Among Gowers' patients the biggest calves were found in a man æt. 33, and measured 14½ in. in circumference. He was a very fat man. This most certainly is not a big measurement.

Again, in a very stout boy, æt. 15, the calves were 13½ in. in circumference, a most moderate size; and in another case (a boy aged 15) the reporter (Mr. W. Adams) says: "Calves large and prominent, each 9½ in. in circumference!" Judged by inches the legs of this latter must have been the reverse of "fine."

In the five cases of this disease (four in one family) which I have seen there has been no undue enlargement of any of the muscles, although the calves have felt, perhaps, unduly solid, and I am strongly of opinion that the prominence which has been given to the "pseudo-hypertrophy" has led to the disease receiving a name which is, to say the least, misleading.

Among other muscles which have been found "firm" if not big are, the muscles of front of leg (out of thirty-one cases, *i.e.*, twenty-six collected by Gowers and five of my own) twice, vasti externi twice, front of thigh twice, masseters once, infra-spinati once, triceps once, deltoids twice.

On the other hand the wasting of muscles, or perhaps one might say the non-development of muscles or the shrinking of muscles, has been very evident in almost every case, and has attacked both upper and lower limbs and the trunk. Gowers says, "The infra-spinate and deltoid muscles are often increased in size, the former with especial frequency. The latissimus dorsi is commonly much wasted, and so also is the lower (sterno-costal) portion of the pectoralis major, the clavicular part being much less commonly affected." He further points out that there is a physiological association of these two muscles, which are depressors of the raised arm.

The irritability of the weak muscles to both forms of electrical current is lessened but not lost.

The weakness of the affected muscles is very evident, and there is a difficulty in performing any muscular act which involves their use.

Thus the straddling attitude and waddling gait are due to the want of power in muscles which keep the body balanced when standing or walking.

The lordosis is due, according to Duchenne, to the weakness of the spinal extensors; but Gowers is of opinion that it is to compensate the abnormal tilting forward of the pelvis on the thigh bones, which occurs owing to the weakness of the extensors of the hip.

Gowers has pointed out how frequently patients suffering from this disease will (on rising from a sitting posture) place the hands on the knees in order to assist the extension of the knee joints, and then climb up their thighs, as it were, in order to assist the extension of the hip joint. Such a trick as this he considers to be pathognomonic of the disease.

It is often, however, not observed, and even when present can hardly be regarded as pathognomonic, as it is often seen in at least one other condition, viz., caries of the spine.

The contraction of certain muscles leads to permanent distortions, the most common of which is talipes equinus, the result of contraction of the calf and shortening of the tendo-achillis. Permanent contractions and distortions of joints other than the ankle may occur, such as the knee or elbow, and there may be a permanent lateral curvature, with rotation, of the spinal column.

In a considerable number of cases there has been mental dulness, but in the majority the mind has been normal.

In twenty-four out of thirty cases collected by Gowers, death occurred between 10 and 20. The ages of the remaining six cases at death were, 6, 6, 21, 30, 40, 40.

As might be expected, the muscular condition produces death through the failure of some of the respiratory muscles to perform their duties. A very slight bronchitis or other lung trouble is often fatal to these patients.

The muscular substance of the heart also apparently suffers. In a recent case reported by Dr. Milner Moore, of Coventry, in which the microscopic examination was made by Dr. Byrom Bramwell, the latter says (*Diseases of the Spinal Cord*, p. 203), "In the wall of the left ventricle numerous degenerated patches could be seen with the naked eye; and on microscopical examination they were found to consist of wavy bundles of connective tissue."

As to the nature of the condition observed in the muscles in these cases there is agreement among histologists; but with regard to the condition of the cord the case is very different.

On one important point, however, there is unanimity, viz., that the motor cells in the front horn are not affected in pseudo-hypertrophic paralysis. Lesions, degenerative and inflammatory, have been found in the cord, but they have not been definite, nor the same in different cases, and in view of the fact that the cords of patients who have died of this disease are generally the cords of helpless and deformed invalids with distorted spinal columns, observers hesitate to link together the spinal and muscular lesions as cause and effect.

Dr. Byrom Bramwell, in the case before alluded to, found a peculiar change in shape of the grey matter, and an outgrowth from the lateral column in the cervical region. These, he says, "were probably, I think, congenital malformations, and not the essential lesion in the case. It is, however, very important to remember that a very similar outgrowth from the lateral column was present in Dr. Drummond's case; it is therefore probable that in pseudo-hypertrophic paralysis there is a strong tendency to congenital malformations in the arrangement of the grey matter, and in the shape of the cord."

There is, I think, much force in this suggestion. When we have regard to the fact that the disease in question is distinctly hereditary, and almost entirely limited to the developmental period of life, it seems impossible to escape from the conclusion that the essential lesion, whatever it may be, is congenital.

It may reasonably be expected that embyologists well accustomed to watch the evolution of the spinal cord will some day solve for us the mysteries of pseudo-hypertrophic paralysis.

CHAPTER VII.

PARALYSES FOLLOWING INJURIES OF MIXED NERVES.*

. . . . I shall divide the paralyses of which I have to speak into *recent* and *old*. Among the former I shall include those which have lasted from one to six weeks, and among the latter I shall include such as have lasted from six months to four years.

Case No. 29.—*Paralysis of the upper limb following a dislocation of the humerus, with wasting of the arm, forearm, and hand. Diagnosis of the degree of damage to each of the nerves of the arm established by localised faradisation. Prognosis established by means of the electric contractility of each of the muscles. Localised faradisation applied a month after the accident; rapid cure of those muscles whose electric contractility was retained; slow cure of those in which it was lost.*

[Vaubelle, a tailor, æt. 25, was admitted to the Hôtel Dieu on February 3rd, 1850, with a forward dislocation of the humerus. Movement of the dislocated limb caused him great pain, and provoked spasmodic contraction of the muscles. The reduction was postponed for twenty-four hours, and the next day the head of the humerus had shifted its position to below the glenoid cavity. The pain and spasm had disappeared, and the limb was almost completely paralysed. The sensibility of the skin remained intact. A month after the accident it was found that all the muscles of the arm, forearm, and hand had lost their faradic contractility, even to the strongest currents. The deltoid contracted feebly. Electro-muscular and cutaneous sensibility were notably diminished, as well as that of the nerve trunks. The limb was wasted, and its temperature was depressed.

A month later, in spite of active treatment with faradism, the wasting of the arm and forearm had reached a maximum, and the hand looked "as if dissected." In a little time the deltoid, however, recovered its voluntary power, and the sensibility of

* From *L'Electrisation Localisée*, pp. 311—381.

the nerve trunks of the arm notably increased. Towards the sixth month after the accident the sensibility of the muscles of the arm returned somewhat, and the patient complained of pain in the limbs. Next the power of flexing and extending the elbow began to return, and the muscular prominences began to reappear. Then there was a return of muscular sensibility, with attacks of pain in the forearm, which was followed by some power of movement in the wrist and phalanges, though many months elapsed before this power returned. The muscular prominences gradually reappeared and their normal power returned, *but nevertheless they did not contract under the influence of the most intense currents*. The recovery of the hand was most obstinate. In May (1851) there was no sign of the muscles of the thenar eminence, and it was only in February, 1852, that they began to recover. The treatment had also an obvious effect in increasing the temperature of the limb and restoring its circulation.

The arm of this patient was at first in the "cadaveric position," but after the recovery of the muscles of the forearm, it was "clawed," the first phalanges being extended and the two last flexed owing to the force of the extensors and flexors of the forearm not being antagonised by the action of the interossei and lumbricales which flex the first and extend the distal phalanges. Owing to the wasting of these latter muscles the cords of the flexor tendons were plainly visible in the palm. As the interossei recovered the clawing of the hand disappeared, and it resumed its normal position.]

. . . . .

Is this paralysis due to concussion of the brachial plexus, or to contusion or rending of its nerves? I shall not enter at length into this question, which has been treated by Malgaigne and Empis. I will merely remark that Malgaigne has not found post-mortem in patients who during life have had a dislocation of the humerus either rending or bruising of the brachial plexus, and he has never been able to produce a rending of the nerves on the dead body, no matter to what amount of traction he subjected the upper limbs. From these facts he has concluded that the paralysis following dislocation of the humerus is due to concussion of the nerves. I believe with Empis that concussion of the nerves was the cause of the paralysis in the

case just mentioned, and I can support Malgaigne's opinion with another case which seems to establish the fact that concussion of certain of the nerves of the brachial plexus sometimes causes wasting paralysis of the muscles supplied by these nerves. . . .

One may conclude from Vaubelle's case that the loss of electric contractility in the muscles supplied by a damaged nerve is a much more serious matter than the mere weakening of their contractility.

The absence of electric contractility in certain muscles might lead one to fear that it was lost for ever, but since the patient had some electric sensibility remaining—which indicated that the nervous connection was not completely broken—I was able to tell Professor Roux that sooner or later these muscles would recover under the influence of electric excitation. (I have noticed that contusion or concussion of nerves usually causes more trouble in contractility than in sensibility.) Before recovering their nutrition and motility Vaubelle's muscles experienced, as we have seen, some sensory excitement under the influence of treatment. *Muscular hyperæsthesia developed under the influence of faradisation in a limb paralysed by nerve injury is then a favourable sign.*

The order of succession of phenomena occurring during Vaubelle's treatment is all the more interesting because I have noticed the same order in similar cases. This order was, (1) rapid return of voluntary power in muscles which had not lost their electric contractility; (2) increased sensibility of those muscles whose electric contractility was seriously impaired, but whose sensibility was merely diminished; (3) return of nutrition and motility in the arm, forearm, and finally in the hand.

The curative action of localised faradisation on those muscles whose electric contractility was much impaired was very slow, for evidently localised faradisation could not give life to muscles deprived of nervous influence. In Vaubelle the faradism did not really begin to act on the muscles till the nerves by their regeneration allowed the nervous influx to reach them freely. The cure was so slow that one might be inclined to deny the utility of the faradism, and to attribute it to time, and with all the more reason that paralyses following dislocations of the humerus get well spontaneously. In such cases, however, the

muscles doubtless lose only a part of their electric contractility, *i.e*, the nerves are only slightly damaged. But in Vaubelle's case I believe I may affirm that temporising would have proved fatal to him. The proof of this will be found in the relation of other similar cases. . . .

I will finally note another peculiarity, important alike from a therapeutic and scientific point of view, viz., that in spite of the absence of electro-muscular contractions the curative effect of faradism was none the less evident in Vaubelle's case. The voluntary power and nutrition of the muscles recovered sooner than their electro-muscular contractility. This fact, *which I have noticed in all paralyses from nerve injury*, is opposed to the usually accepted notions, for no one would expect that a living muscle would not contract, or would contract only feebly, to faradism, while it is known that a muscle will contract for some hours even after death.

[Several other cases are given by Duchenne of paralysis from nerve injury, but since no points are elucidated in addition to those illustrated by the case of Vaubelle they need only to be alluded to.

Case No. 30.—*Wasting paralysis of the upper limb from injury to the nerves of the cervical and brachial plexuses. Differential diagnosis and prognosis of the condition of each muscle derived from the electro-muscular contractility. Localised faradisation employed many months after the onset. Slow recovery of those muscles whose electric contractility was seriously impaired.* (For details see Case VIII. in first and second editions of *Elec. Local.*)

Case No. 31.—*Wasting paralysis of upper limb from a dislocation forward and downwards of the humerus caused by a fall. Differential diagnosis and prognosis derived from electro-muscular exploration. Faradisation employed a few days after onset. Recovery rapid in those muscles which retained their electric contractility, and slow in those which had lost it.* (For details see *Elect. Loc.*, second edition, Case IX.)

Case No. 32.—*Wasting paralysis of the hand following an injury of the ulnar nerve.* (For details see *Elect. Loc.*, second edition, Case X.)

Case No. 33.—*Paralysis of deltoid and supinator longus, caused by a woman jumping from the second floor on to the*

patient's shoulder. (For details see *Elect. Loc.*, second edition, Case XI.)

Case No. 34.—*Wasting paralysis of the upper limbs coming on during sleep, attributed to a syphilitic tumour compressing the cervical and brachial plexuses. Diagnosis from the electro-muscular condition. Failure of faradisation.* (For details see *Elect. Loc.*, second edition, Case XII.)

With regard to this last case Duchenne was able to state that it was not of cerebral origin: 1. Because the muscles of the limb failed to respond to faradism, which is never the case in paralyses of cerebral origin; and 2. Because the limb wasted rapidly, "while muscular atrophy is not seen in cerebral paralysis, which merely causes a slight thinness of the limb from want of exercise."

"I localised the chief lesion in certain nerves of the cervical and brachial plexus. On closer examination a tender point was found at the level of the cervical plexus on the right side. Pressure here caused pain in the upper limb, and the patient complained of lancinating pains occurring spontaneously at this spot. It was found that certain muscles and portions of muscles supplied by the cervical plexus had preserved their electric contractility, which was evidence of the soundness of the nerves supplying them. Thus the patient, who could not raise his right shoulder by direct spontaneous effort by reason of the paralysis of the middle third of the trapezius which executes this movement, could raise the shoulder instinctively during deep inspiration, thanks to the soundness of the clavicular part of the trapezius and the levator anguli scapulæ (the sole agents of this instinctive respiratory movement). Voluntary outward rotation of the humerus by the infra-spinatus was still possible. This patient recovered under an anti-syphilitic treatment prescribed by Nelaton."]

Old paralyses following nerve injuries (treatment commencing from six months to four years from the date of injury).

Case No. 35.—Albert Musset, æt. 19, printer, lost the use of his right hand after a wound of the forearm on November 13th, 1846, having had his hand caught in a machine. A cutting instrument with blunt edges entered the skin of the front of the forearm 4 or 5 centimetres above the wrist, cut the front of the ulna, and came out inside the tendon of the *flexor carpi radialis* dividing and tearing everything before it. In

consequence the *flexor carpi ulnaris* muscle, the internal fasciculi of the superficial and deep flexors, the *palmaris longus*, the ulnar nerve and artery, and perhaps the end of the median nerve, must have been divided. His wound was healed three months after his admission to the Hôtel Dieu. His hand was then wasted. The two terminal phalanges were constantly flexed without power of extension. The fourth and fifth fingers could not be extended, being apparently fixed by the scar to which the tendons adhered. We succeeded by gradual and continued traction in breaking the restraining bands, and stretching them like the other fingers, but all voluntary power seemed to have been irreparably lost. On October 16th, 1850, he was admitted to the Charité, and his hand was then in a pitiable condition. The extensor and flexor tendons stood out in strong relief under the skin. The hand was "clawed," owing to the failure of the wasted interossei to oppose the tonic force of the flexors and extensors of the fingers. The heads of the metacarpal bones were hypertrophied, and the first phalanges were dislocated backwards, their flexion being limited by the heads of the metacarpal bones.

Treatment was commenced on December 22nd, 1850. During five or six sittings on alternate days, faradism was used solely to the muscles of the forearm without any benefit. It was then applied to the interosseal spaces, and to the thenar and hypothenar eminences, a strong and painful current being used. After the tenth application Musset experienced a burning sensation in the hand. The fingers, however, were still shiny, swollen, and painful. The hand looked less like a skeleton, and the interosseal spaces began to fill out. The first phalanges were less extended, and the second began to stretch out. The treatment was now suspended by reason of a febrile attack, at the end of which it was found that the hand had not lost ground, but that the fingers were warmer, and the pains which he had hitherto felt had disappeared. Faradisation of the skin with metallic threads was now added to faradisation of the muscles, and soon the sensibility of the skin of the hand was markedly increased, the veins began to re-appear, and the normal colour of the hand to return. The little muscles of the hand gradually regained their power, and the fingers resumed by degrees something of their normal attitude. The tonic power of the interossei showed itself long before the return of voluntary power, and when Musset left

the Charité (March 15th, 1851), there was scarcely any voluntary power over the phalanges; when he flexed the two end phalanges he could not straighten them, but when, having shut his hand, he ceased to contract his flexors, the two end phalanges extended of themselves, owing to the tonic power of the interossei, which I have shown flex the first phalanges and extend the two end ones. Musset suspended his treatment for two and a-half months, at the end of which time the condition of the hand remained unaltered. Treatment was resumed in June, 1851 (Duchenne says 1850), and faradism was applied twice or thrice weekly. The position and power of the fingers gradually improved, the improvement occurring earliest in the index and middle fingers. In August he was able to write and draw, and was soon able to take a situation as a railway clerk. The thenar and hypothenar eminences filled up, and the flexor and extensor tendons disappeared. This recovery was maintained when Duchenne saw him in 1869, when he was seeking employment as a short-hand writer.

The position of the wound, the deformity of the hand, and the loss of electric contractility in the muscles supplied by the ulnar nerve, all pointed to that nerve as the seat of injury. The return of tonic power in the muscles before the return of voluntary power is a phenomenon which I have seen in other similar cases.

The curative action of localised faradisation on the nutrition and movements of the muscles of the fingers and thumb was very rapid in Musset's case as compared with Vaubelle's, in which the nerves were certainly not divided. I have already given the explanation of this. I have said that a cure cannot take place till after the recovery of the nerve lesion, and the possibility of free nervous influx to the paralysed muscles. But such a recovery of nerves takes more or less time. Therefore the recovery of Vaubelle was slower than that of Musset, whose injury was of four years duration, and in whom the mental stimulus had already forced a passage through the scar and the extremities of the regenerated nerve. There was little doubt therefore that before faradisation was used the nerve force had arrived at the paralysed and wasted muscles, which doubtless were in need of the electric stimulus to enable them to react to the cerebro-spinal stimulus.

Case No. 36.—*Wasting paralysis from compression of the musculo-spiral (radial) nerve following necrosis of the humerus situated in the course of the nerve. Diagnosis by means of localised faradisation. Cure most rapid in those muscles whose contractility was least affected.* (For details see *Elect. Loc.*, second edition, Case XIII.)

Case No. 37.—*Paralysis and wasting of the interossei and lumbricales from a bruise or concussion of the ulnar nerve from the discharge of a gun, in the palm of the hand. Rapid cure by faradisation used ten years after the accident.* (For details see *Elect. Loc.*, second edition, Case XV.)

In this case the charge entered in front of the wrist, and lodged under the skin on the back of the forearm. The median nerve having escaped, the attitude of the first two fingers was less clawed than that of the last two, because of the integrity of their lumbricales muscles.

The ulnar nerve not having been destroyed, the patient might fairly expect that his paralysis would ultimately recover. He was deceived, for four years after the accident the paralysis was *in statu quo*, and the vitality of the hand was depressed. The temperature was diminished, and the circulation so feeble that the dorsal veins were no longer visible, and the usual perspiration of the hand was suppressed. . . . Recovery did not take place until after the application of localised faradisation. . . .

Case No. 38.—*Paralysis of the interossei following a bruise of the right ulnar nerve. Loss of sensation. Cured by faradisation in five sittings, after having persisted for five years.* (For details see *Elect. Loc.*, second edition, Case XVI.)

. . . "This case proves that paralyses from nerve injury, even when they are slight, are not always cured by time alone."

Case No. 39.—*Wasting paralysis of the muscles supplied by the external popliteal nerve from compression of the nerve. A club-foot in consequence of the faulty antagonism of the damaged muscles. Rapid cure by faradisation six months after the injury.* (For details see *Elect Loc.*, second edition, Case XVI.)

[In this case the damage to the nerve was not severe, although the paralysis of the muscles supplied by it was complete. The recovery after faradisation was very rapid. "This is another case in favour of the proposition I have already stated, viz.,

that in old paralyses from nerve injury, cure by muscular faradisation (provided the muscles be not completely destroyed) is more rapid than in recent paralyses of the same kind, because, the nerve lesion having recovered, the nerve-force has free access to the paralysed muscles."]

Review of the Symptoms of Paralysis from Nerve Injury.—Bruising, squeezing, stretching, concussion, in fact every kind of injury to a mixed nerve, causes more or less motor or sensory trouble, and change of nutrition and temperature in the limb supplied by that nerve. But there are points in these paralyses which are important as aids to diagnosis, and the knowledge of which is due to localised faradisation. I mean the loss of contractility and sensibility to faradism, which is always seen in the paralysed muscles. The following paragraphs are especially devoted to a study of these phenomena.

Impairment of electro-muscular contractility.—When the damage to a mixed nerve is so severe that all its fibres are affected, all the muscles supplied by it lose their electric contractility in equal degrees.

It sometimes happens that muscles which are equally paralysed are very unequally affected as regards electric irritability. Sometimes this inequality in electrical condition is seen in different fasciculi of the same muscle. . . . At other times muscles which seem to retain their voluntary power have lost a part of their electric contractility.

Finally, it is not uncommon to see in this kind of paralysis muscles retaining their normal electric contractility although they are more or less completely paralysed. I cannot think that in these cases the nerves have been really damaged, and further, the muscles speedily recover their power. But how does it happen that nerves which are not involved in the injury are nevertheless paralysed? Does not this pathological phenomenon compel us to admit the existence of a kind of *common responsibility* among the different nerves of a limb? Wherefore if one of them is seriously damaged, is it to be wondered at that the motor trouble is not always limited to the muscles supplied by that nerve, but spreads to other muscles whose nerves have not been injured?

Impairment of sensibility.—[In cases of nerve injury muscular

sensibility is usually much less affected than the electric contractility. Cutaneous sensibility is still less affected.] These facts show that in injuries of mixed nerves sensibility is much less affected than voluntary or electric contractility. But there are cases in which the sensibility of the skin and muscles is quite lost. These are cases where communication between the spinal cord and the limb is completely interrupted.

Differential diagnosis of paralysis from nerve injury derived from the electric contractility and sensibility of the paralysed muscles.

[There are cases in which the damage to the nerve may escape observation, and then the fact that the electro-muscular contractility is weakened or lost will prevent the case being confounded with one of brain palsy or rheumatic or hysterical palsy, in which the electro-muscular contractility is always normal. The diagnosis of these cases from cases of lead palsy will be dealt with elsewhere.]

In cases of paralysis from compression of the limbs electro-muscular contractility remains intact. The diagnosis of such cases cannot be doubtful, for the paralysis immediately follows the cause. I will give briefly two examples.

Case No. 40.—*Paralysis of the extensors of the wrist and fingers and supinators following compression of the forearm during a prolonged and strained attitude. Electro-muscular contractility normal. Sensibility a little weakened on the back of the forearm. Cure in sixteen sittings by localised faradisation applied four months after the onset.* (For details see *Elect. Loc.*, second edition, Case XVIII.)

Case No. 41.—*Paralysis of upper limb after severe compression by a land-slip. Preservation of electric contractility in the damaged muscles. Rapid cure by localised faradisation applied a month after the accident.* (For details see *Elect. Loc.*, second edition, Case XIX.)

Although in the paralyses from compression of a limb the electro-muscular contractility has been found normal, the cause of this can be recognised. May it not be attributed to an injury, probably limited, to the nerve tufts which spread in the compressed muscular tissue, and which is too slight to cause any great disturbance of the electro-muscular contractility?

1. PROGNOSIS OF PARALYSIS FROM INJURY OF MIXED NERVES DERIVED FROM THE STATE OF ELECTRO-MUSCULAR CONTRACTILITY AND SENSIBILITY. . . When we know that the muscles which, in these cases, have lost their electric contractility, remain for a long time paralysed, waste, and are in risk of textural damage; while those which preserve their electric contractility will rapidly regain their motor power, we understand the importance of electro-muscular exploration in arriving at a prognosis in cases of paralysis from nerve injury.

2. *It results from the preceding considerations that the prognosis of paralyses from nerve injury cannot be exactly established without electro-muscular exploration.* . . .

I have had to form a prognosis in the two following cases of paralysis of the same muscles from similar causes.

Case No. 42.—*Fall on the pavement on right shoulder. Immediate paralysis of shoulder-muscles and flexors of elbow. No pain, merely a little numbness of the hand. Two months after the accident, paralysis of the deltoid, trapezius, supra-spinatus, infra-spinatus, serratus magnus, flexors of elbow, and supinator longus, with abolition of electric contractility. Loss of electric sensibility. Long duration of the paralysis. Complete and rapid wasting of the muscles deprived of electric contractility in spite of firing in spots along the course of the brachial plexus and localised faradisation applied many times a week.*

Case No. 43.—*Fall on the right shoulder from the top of a waggon. Severe pain and swelling of the shoulder. Paralysis of deltoid, external rotators of humerus and flexors of elbow six weeks after the accident. Electric contractility of the paralysed muscles normal. Rapid cure by localised faradisation.* (For details see *Elect. Loc.*, second edition, Cases XX. and XXI.)

. . . . . .

[In the first of these cases my prognosis was to the effect that the muscles would waste, and that recovery could not be hoped for till the nerve injury was repaired (in eight or ten months), while in the second cases the muscles would probably not waste, and recovery would be rapid. The prognosis was justified in both cases.]

3. *The gravity of paralysis from nerve-injury is in direct proportion to the impairment of electric contractility and sensibility in the muscles supplied by the nerve.*

This proposition, formulated by me in 1852, is illustrated by he preceding facts, and receives a striking confirmation from many fresh cases, of which the following is an example.

Case No. 44.—L. Bellet, æt. 53, Hospital St. Louis, under M. Malgaigne. Paralysis of right upper limb from a dislocation of the humerus. Two months after the accident slight decrease of electric contractility of the deltoid, triceps, supinator longus, and the muscles on the back of the forearm, and complete absence of electric contractility in the muscles supplied by the median and ulnar nerves. Complete loss of electric sensibility in these last muscles. Cutaneous sensibility lost on the back of the forearm and in the regions supplied by the median and ulnar nerves. Recovery of those muscles which retained their electric contractility in fifteen or sixteen sittings. Return of movement in the muscles, which had lost their electric contractility nine months only after the accident, preceded by stabbing pains which were sharper after each sitting. Progressive return also of the warmth and nutrition of the limb although faradisation had been used seldom and irregularly (once a week). (For details see *Elect. Loc.*, second edition, Case XXII.)

[Duchenne says of this case that recovery would have been quicker if faradisation had been practised three or four times a week.]

4. *The prognosis of these cases is usually much less grave when with complete extinction of electro-muscular contractility the sensibility of the muscles is preserved or merely weakened.*

The complete demonstratlon of this proposition is shown in the following case.

Case No. 45.—*Wasting paralysis of the left upper limb following a dislocation of the humerus. Inequality of the nerve lesion recognised by electro-muscular exploration. The muscles whose sensibility was but little affected did not waste in spite of the loss of electric contractility, and recovered their motility towards the sixth month, while those which had lost both sensibility and contractility remained paralysed and much wasted.* (For details see *Elect. Loc.*, third edition, Case XXXIV.) . .

5. *The integrity of electric contractility in muscles paralysed by nerve injury is a favourable sign.*

The paralysis spreads sometimes by a sort of nervous sympathy to muscles whose nerves have not been injured and whose

electric contractility is normal. One then sees that these muscles have not suffered in their nutrition, and that they quickly regain their motility.

6. *The question of the regeneration of nerves.*

[This question, which has been under discussion for a century, may now be considered as settled. Among the early workers at this subject (from 1776 to 1838) are Cruikshank, the fellow-labourer of Hunter, Fontana (1781), Lorenzo Nanoni (1782), and Haighton (1795). In opposition to the doctrine of the regeneration of nerves many observers have held that the tissue which joins their cut ends is merely ordinary scar tissue. Among these were Monro, Richeraud, Boyer, Breschet, Magendie, and Jobert de Lamballe (1864). The recent history of this question begins in 1838 with a paper by Steinrueck, who repeated all the experiments of his predecessors, and includes the remarkable work of Waller (1850), who not merely showed that the union is nervous, but that every nerve section is necessarily accompanied by a degenerative process which destroys the nerve tubes, and by a process of repair which restores them, and that this occurs only in the far end of the nerve which is no longer in connection with a "trophic centre."

According to this author it is indispensable that the two ends of the nerve be joined by nerve material. Without this regeneration will not be definite. MM. Vulpian and Philippeaux assign to nerves a property which they call "neurility," independent of the nerve centres, and by virtue of which they can be regenerated by an intrinsic power, though connection with a nerve centre be not renewed.

I have seen many wasting paralyses the result of nerve injuries ultimately get well. In all these cases have the ends of the nerve, torn or carried away by a bullet for example, been united by a nervous regeneration? Has the recovery of the far end taken place, as Waller asserts, or has the nerve recovered in virtue of its "neurility" independently of the nerve centres, as Vulpian and Philippeaux assert?

. . . . . .

It is important to confirm experimental investigation by clinical observation. A case which I saw in 1866 in Professor Nelaton's wards, and in which microscopy showed the restoration of 5 centimetres of the ulnar nerve removed four years before by

M. Huguier, confirms the doctrine of the nervous regeneration of the far end.

Case No. 46.—*Section of the ulnar nerve for a length of* 4 *centimetres in the palm of the hand. Amputation of the forearm five years later; dissection of the amputated hand; demonstration by microscopy of the regeneration of the resected portion of the nerve and of its termination. Integrity of the interossei, which had been paralyzed since the resection of the nerve, and which had lost their electric contractility.*

Résumé.—A woman, æt. 35. In 1857, resection at the Beaujon Hospital, by M. Huguier, of 4½ centimetres of the right ulnar nerve in the palm of the hand for obstinate and frightful neuralgia of more than a year's duration. Paralysis and atrophy of all muscles supplied by the nerve below the section, and gradual development of the claw-like deformity. Loss of sensibility in the inner half of the hand, and disappearance of the pain. Six months after the resection return of sensibility, and a little later return of the pains as troublesome as before. At the same time there appeared a neuroma at the most painful spot, which M. Huguier removed. In 1866 she was suffering the same torture as before, and taking into consideration the presence of numerous neuromata in the hand, and yielding to the strong desire of the patient, M. Nelaton amputated the hand.

In this hand I established — 1. The clawed condition characteristic of a lesion of the ulnar nerve, but which had much diminished, according to the patient, for the last five or six months. 2. Absence of electric contractility in the muscles supplied by the ulnar nerve. 3. Loss of abduction and adduction of the little and ring fingers, and of the power of extending the middle and far phalanges.

After amputation the interossei were found wasted and rather pale, but otherwise healthy. The two last lumbricales were fatty. M. Beng Anger established by microscopy the fact of the regeneration of the resected portion of the nerve and of its far end.*

* I have given all the facts of the case as stated by Duchenne; but it will be observed that the facts are stated three times (in the introductory sentence, in the abstract (in italics), and in the *résumé*), and that these three accounts differ somewhat from each other; thus the length of nerve removed is stated to be 5 centimetres, 4 centimetres, and 4½ centimetres, and the time intervening between resection and amputation is stated to be four years in the introductory sentence, and five years in the abstract, in italics, whereas an appeal to dates (1857—1866) shows that it must in reality have been nine years.—EDITOR.

This case, in short, confirms Waller's doctrine of regeneration.

What was the condition of the interossei muscles?

This question was put to me before the operation by M. Nelaton. The persistence of their paralysis since the section of the ulnar nerve and the absence of electro-muscular contractility had at first led me to believe that they were in a state of fatty degeneration; but having learnt that for several months the "clawed" state of the hand had much lessened, which could only occur through the return of the tonicity of the interossei (as I had observed in analogous cases) before the return of their voluntary power, I expressed the opinion "that the texture of these muscles ought to be normal," an opinion confirmed after the amputation.

On the other hand, this return of tonic power had caused me to predict the regeneration of the far end of the ulnar nerve.

I conclude from the preceding facts that the prognosis in cases of paralysis following destruction of a nerve ought not to be too grave even when the muscles manifest great change in nutrition and electric contractility. It was in order to establish this proposition that I have made this digression on the regeneration of nerves.

7. *In cases of suture of a mixed nerve, electro-muscular exploration affords an excellent means of prognosis as to the paralysis and nutrition of the muscles supplied by the nerve below its division.*

The question of the immediate re-union of divided nerves was first sustained by the celebrated English surgeon Paget, who believes that he has obtained it twice (*Surgical Pathology*, 1853). It was revived in 1864 by Nelaton (who first applied the suture to nerves with the object of ensuring the union of the ends), by Langier in 1866, and by Richet in 1867. The question is far from being settled. In fact MM. Vulpian, Schiff, Eulenburg, Landois, Magnien, &c., have never got immediate union after section of nerves. The shortest space of time necessary for the return of function in their experiments was seven days in the youngest subjects. M. Vulpian has even declared that he has always found in the scar nerve-tubules in course of regeneration. On the other hand there is a want of agreement. In Nelaton's case, according to Houel, and in Langier's case

loss of sensibility immediately followed the division of the nerve. In Nelaton's case it re-appeared four days after the junction of the cut ends, and in Langier's on the evening of the same day. In Richet's case, on the other hand, sensibility was unaffected after the section of the median, even before the cut ends were sutured. After the suturing the muscles supplied by the median were paralysed and wasted.

M. Richet, relying on a fact discovered by Robin, offers the following explanation of the preservation of the sense of touch in the fingers. "M. Robin," he says, "shows by a figure that two nerve threads unite by their ends to form complete loops. From these loops other finer threads issue to terminate in the tactile corpuscles. Each of these corpuscles receive filaments from the loops formed by the anastomosis of the ulnar or radial with the median. The matter is explained, therefore, without difficulty. When one or other of these nerves is cut, sensory impressions travel by that which remain, and the tactile corpuscles, still sufficiently supplied with nerves, respond to external stimulation."

The paralysis of the muscles of the thenar eminence immediately following the division of the median, and its persistence with atrophy after the suture of the nerve, give additional weight to Richet's explanation. But since the facts recorded by observers of such repute as Nelaton and Langier cannot be rejected, one must conclude that the anatomical fact recorded by Robin (the anastomosis of nerve loops) is not the rule. The point has yet to be settled.

It further follows from the facts related by Nelaton and Langier that the almost immediate re-establishment of function which follows the junction of the cut ends of a nerve is an undoubted clinical fact, notwithstanding that it is not yet explained by experiment, that the mechanism of the mode of union has still to be discovered, and that it may not be possible to conclude that in these cases there is immediate reunion of nerves.

It must also be concluded from the persistence of the paralysis and wasting of the thenar eminence, which in Richet's case resulted in spite of the suture of the nerve, that nerve-suture is not always followed by an immediate return of muscular function, but that in such cases the recovery depends on the regeneration of the far end of the cut nerve. . . .

Case No. 47.—A woman aged 20, in whom M. Richet in the presence of several witnesses showed the preservation of sensibility in the far end of the median nerve, which had been completely divided by a wound of the forearm, situated about 3 centimetres above the bend of the wrist. Suture of the median having been effected about thirty-four hours after the accident it was important to know if the voluntary movement of the muscles supplied by the median below the wound would return as quickly as in the case of Nelaton and Langier. On the following day, *i.e.*, forty-eight hours after the injury, I found, on being invited to do so by M. Richet, that none of the muscles contracted voluntarily, and that their electric contractility was diminished. This was already to me an unfavourable sign, and when, on the third day, I found the electric contractility much diminished, I predicted that the paralysis would last till the far end of the nerve was regenerated, that the muscles of the thenar eminence would waste, and that the first metacarpal bone would take an unnatural position. This prognosis was fulfilled. On the fourth day the electro-muscular contractility was almost extinguished, the paralysis persisted, the thenar eminence got flatter by degrees, and the first metacarpal bone was drawn back by its extensor and placed on the plane of the second metacarpal bone.

In contrast to the persistence of sensibility and touch, in spite of the section of the median, was the loss of electro-muscular sensibility in the thenar eminence, which I discovered on the day following the suture. I do not doubt that this was due to the division of the median, and I could not understand how it could be otherwise. It is hardly needful to state that the preservation of sensibility in the muscles of the thenar eminence could not be explained, as in the case of the fingers and skin, by anastomoses of terminal loops of the median and radial, furnishing nerve filaments to the tactile corpuscles.

This loss of sensibility in the paralysed muscles lasted but a short time, for the patient soon felt pains in the scar radiating in the thenar eminence. At the same time the skin of this region became warm and red. This irritation lasted several weeks, and then treatment by faradisation with slow intermissions was commenced. In about two months electro-muscular sensibility began to return, and "after some months" the thenar eminence

began to fill out and the muscles to contract voluntarily. The patient left Paris before she was completely cured.

It is to be remarked that the third day after section of the median nerve the electric contractility of the muscles of the thenar eminence was already lessened. This fact stands in contradiction to the experiments of physiologists who have found in their vivisections that muscles retain their electric contractility for some time after the section of a nerve. . . .

How are these facts to be explained? J. Reid has stated that the atrophy and consequent loss of electric irritability after section of a nerve were due to the state of inaction of the muscles caused by the paralysis. Many other hypotheses have been proposed by physiologists to explain the same morbid phenomena. The most recent opinion formulated by Brown-Séquard in 1859 seems to me the best. "I have seen," says he, "at least some hundreds of animals live for months after section of the spinal cord, showing no other nutritive trouble in the paralysed parts than a wasting, which is usually slow to appear. In two cases, on the other hand, in which exostoses formed in the neighbourhood of the section and compressed the lower end of the cord there was considerable wasting in five or six days, and gangrenous ulceration of the sacrum and some points of the thigh. We must, therefore, distinguish the effects of excitation of the spinal cord and nerves from those of paralysis or simple cessation of action of the parts. In other words, we must distinguish between the effects of diseased action and want of action."

The preceding case seems to me to support Brown-Séquard's hypothesis. It will be remembered that at the moment of establishing the loss of irritability in the muscles of the thenar eminence there were symptoms which might be attributed to irritation of the divided nerve. Similar phenomena occurred in Case 48, where the median nerve, which was completely divided at the bend of the elbow, became inflamed in consequence of a bad first dressing, and there resulted a wasting paralysis with loss of electric contractility, which was not cured three years later.

Electro-muscular exploration made shortly after the re-union of the ends of a divided nerve enables a prognosis to be formed with regard to the condition of muscles situated below the wound.

It follows from the preceding case—1. That loss of electric contractility shows that the muscles will remain long paralysed and will waste. 2. That loss of electric sensibility of the muscles is the inevitable consequence of the division of the nerve, although in the hand the fingers may preserve their sensibility in virtue of the anastomoses of different nerves. 3. That the return of electric sensibility in the muscles is a favourable sign.

This last proposition is strengthened by the following case, which resembles the preceding, and was treated at the same time :—

Case No. 48.—A woman, æt. 23, divided the median at the bend of the elbow in 1863 ; immediate paralysis of the muscles supplied by the cut nerve ; inflammation of the wound in consequence of a bad dressing applied by a druggist. Three months later I found paralysis of the muscles, wasting and absence of electric contractility and sensibility. Inutility of two months treatment by faradisation. Fresh attempt at treatment three years later, return of some sensibility in a few sittings, and gradual slow recovery of motor power and nutrition.

Taken in connection with the preceding, this case is of great interest with regard to the difference of prognosis according to the absence or presence, even in a slight degree, of electric sensibility of the muscles. In Case 47, when the electric sensibility returned, I predicted a speedy recovery, and in fact from that time, both during and after faradisation, all the signs of returning nutrition (warmth, redness, hyperæsthesia) were manifest, the muscles soon increased in size, and recovery of tone was followed by return of voluntary power, while in Case 48 these signs appeared only after three years.

OBSTETRIC OR BIRTH PARALYSES.

1. To Smellie has been attributed the first observation of a case of bilateral paralysis of the upper limbs caused by an application of the forceps, but the relation is so incomplete that it is impossible to say whether or no this paralysis was traumatic, *i.e.*, caused by a compression of the brachial plexuses by the forceps.

The first undoubted case of this form of paralysis was communicated to the Société de Chirurgie by M. Danyau in 1851. Here are the details.

Case No. 49.—[The subject was a primipara suffering from albuminuria and puerperal convulsions. The child, delivered with forceps, was born apparently dead, and was resuscitated by artificial respiration. Thirty-six hours after birth it was noticed that it had facial palsy and paralysis of the left arm. Movement of the left upper limb was absolutely abolished, but there was apparently some slight sensibility.

On examining the left side of the neck there was a linear slough one centimetre in length, which separated and left a wound in the cellular tissue penetrating along the edge of the trapezius. This had been caused by the right blade of the forceps, which had penetrated the neck and squeezed the brachial plexus, as it had also squeezed (by the deflection and inclination of the head) the facial nerve as it issues from the stylomastoid foramen.

The child died in eight days, and post-mortem there was found bruising and effusion of blood round the brachial plexus and facial nerve.

In 1867 M. Guéniot published a case similar to the above, but without facial palsy.

These are the only two cases of paralysis of the upper limbs from application of the forceps which have been published in France. I do not know whether foreign literature furnishes other examples.

In cases of paralysis of the upper limbs or of the facial nerve from application of the forceps, the prognosis should be most guarded. It is evident that electro-muscular exploration would be of service in forming a prognosis in these cases. MM. Danyau and Guéniot regard these paralyses as of trifling importance. Duchenne, on the other hand, has seen not a few cases which have been permanent.]

In obstetric operations the disengagement of the arms often offers great difficulties, especially in foot or breach presentations, or after turning, or if the midwife is obliged, in order to extract the body after the birth of the head, to pull with one of his fingers hooked round the arm-pit. It may then happen even to the cleverest that one or more of the muscles become paralysed and wasted by the stretching or squeezing of their nerves or of the brachial plexus.

In support of this statement I will briefly allude to three

identical cases of wasting and paralysis of the upper limb, which I have under treatment by localised faradisation, and which were sent to me by Professor Depaul, M. Guibout, and M. Tarnier, and a fourth by M. Danyau.

[The children were all girls. In one case, a prolonged labour in a primipara, the body was extracted by hooking the finger round the arm-pit. Two other cases were breach presentations, and after the birth of the body there was difficulty in bringing down the arms, which are always raised in such cases.

In all these children one arm was completely paralysed, and hung with the hand prone from the internal rotation of the humerus. In all but one electric contractility was abolished, and the limbs were wasted.

The fourth case was exactly similar to the others.]

The preceding facts show that in this kind of paralysis of the upper limb from obstetric manipulations the arm falls motionless alongside the body, and is rotated inwards; the forearm remains extended, *but the movements of the hand are preserved.*

I leave to others the study of the anatomical cause, and to say why in these cases the same muscles (deltoid, infra-spinatus, biceps, and brachialis anticus) are always paralysed.

[Duchenne goes on to show that if these cases are recognised early and taken in time they yield to treatment by localised faradisation, and that prognosis is determined in exactly the same way as in ordinary cases of paralysis from nerve injury. He concludes thus: "In short, certain violent manipulations of the midwife, necessitated by the difficulty of bringing down the arms after the birth of the trunk, or the strong pulling on the shoulder by a finger hooked round the arm-pit after the birth of the head, sometimes cause paralysis of the upper limb, seated in the deltoid, infra-spinatus, and flexors of the elbow, characterised by falling of the limb alongside the trunk, inward rotation of the arm and extension of the elbow. The prognosis is usually grave. The paralysis may be cured by localised faradisation, but if left to itself it becomes incurable and causes wasting of the limb."

2. Birth Paralyses complicated by Fractures.—These paralyses may be complicated by a fracture caused by the manipulations of the midwife. On the other hand they may be masked by another kind of paralysis, as happened in the following case, in which a paralysis of the ulnar nerve (radial ? in text), which

had given a clawed form to the hand, was cured by localised faradisation after six years.

Case No. 50.—In 1855 I was called in consultation by M. Hérard to see a boy aged 10, whose right hand was deformed. The hand was smaller than the other and "clawed," especially in the third and fourth fingers. The interossei were wasted. The wrist was constantly flexed, and the hand abducted. On trying to straighten the hand and turn it outwards, a mechanical obstacle was found to exist in the contraction of the flexor carpi ulnaris (*muscle cubital antérieur*) and a deformity of the radio-carpal joint. The movements of the upper limb were feeble, difficult, and incomplete. Nevertheless the muscles of the arm and shoulder were not wasted. All voluntary movements of the right upper limb caused reflex spasms in the muscles, as is seen in cerebral paralysis. On electro-muscular exploration I found the contractility very weak in the muscles supplied by the ulnar nerve, in the muscles of the eminences, and in the extensores carpi radiales (*muscles radiaux*), although it was normal in the other muscles. In this case there was a brain paralysis co-existing with a paralysis from nerve-injury. This child was born under adverse circumstances. A shoulder presentation with the umbilical cord twisted round the neck. The labour was long. He was born asphyxiated, and resuscitated with difficulty. Then he had his ulna fractured close to the elbow. At birth he could not move his right upper limb, and in a few months the right limb was smaller than the left, and the deformity of the hand was complete. After some time of varied treatment the right arm recovered some movement, complicated, as I have said, by reflex spasm.

In this case we have a hemiplegia of the right arm from a brain lesion, caused perhaps by the compression of the neck by the umbilical cord, and a wasting paralysis, with deformity of the hand, the result of damage to certain nerves involved in the fracture of the forearm during the manipulations of the midwife. Such was my diagnosis, and the results obtained by faradisation tended to confirm it.

Under the influence of local faradisation the wasted and paralysed muscles gradually developed, and the hand regained its normal shape. This was due to the recovery of the interossei. The eminences regained their form as did also the

wasted muscles of the forearm. The functional troubles caused by reflex spasms during voluntary movements remained almost as they were before treatment. It is true that the movements of the upper limb had gained in power and extent, but they had not regained their independence. The movements were irregular, and were always hindered by the brain lesion. I nevertheless hoped that, by the aid of certain gymnastic exercises which I prescribed, these reflex spasms would lessen or disappear in time. Two years later the little boy came to show me that my hope was realised, this hand being nearly as useful to him as the other. This case, interesting from more than one point of view, shows that, when we have two paralyses in the same individual, one from brain lesion and another from nerve lesion, the differential diagnosis may still be made by the help of electro-muscular exploration. It is no less interesting as showing the cure by local faradisation in a child 7 years old of a wasting paralysis from nerve-injury occurring at birth.

3. Sub-spinous Obstetrical Dislocation (Sub-acromial of Malgaigne) Complicating or Causing a Wasting Paralysis of the Upper Limb.—The sub-spinous dislocation, hitherto very rare, has been met with more frequently of late, doubtless because its diagnosis is better understood. Malgaigne says he has collected thirty-four cases. But none of these were caused by midwives' manipulations. This cause, however, is not very uncommon, if I may judge by my own personal experience. In less than a month I have seen four cases, all of them sent to me as cases of congenital paralyses of the upper limb. The following is a case in point:—

Case No. 51.—In March, 1857, a little boy, æt. 6, named Théophile Bigot, was brought to me for a congenital paralysis of the right upper limb. Its movements were so hindered that the limb was of no use to him. All the muscles responded naturally to electricity. His right shoulder was deformed, and the position of the right arm was unnatural. At the back of the shoulder, a little below the posterior angle of the acromion, a projection caused by the head of the humerus was seen, while in front there was a slight hollow beneath the acromion, and the head of the humerus was absent from its proper place. The head of the bone seemed to be resting on the posterior edge of the glenoid cavity. It was certainly not in the infra-spinous fossa.

It was difficult of explanation, but it was incontestable that this incomplete dislocation had lasted six years. The elbow, carried a little forward and away from the side, could not be adducted, and the attempt caused pain. The humerus was rotated inwards, and the least attempt to rotate it outwards made the child cry. The elbow was slightly flexed. The voluntary power of the arm and forearm was limited and feeble. . . . In this and in the three other cases, which were quite like it, the cause of the dislocation seemed to be the same. The labours were tedious, and the child had in each case been extracted by long pulls with the finger, or crotchet, hooked round the arm-pit, and in each case the limb thus treated had been completely paralysed from birth. . . . I sent all these cases to M. Chassaignac, who easily reduced the dislocation. The head of the bone was held in its place for five or six weeks by means of a bandage, and the reduction afterwards proved permanent, and the head of the bone was then found to have its proper relations.

This dislocation was so obscure that it at first escaped my attention, which was mainly devoted to the troubles of movement. I nevertheless could not satisfactorily explain the functional troubles, and it was only after seeing three similar cases that I recognised by careful examination that the head of the humerus had not its normal relations.

The troubles caused in children by sub-acromial dislocations may easily be mistaken for a congenital brain paralysis. In both cases it is difficult to raise the arm, the hand is pronated and the arm rotated inwards. Finally, the contrary movements are difficult or impossible.

If there had been merely the troubles of movement proper to a sub-acromial dislocation a mistake in diagnosis would have been difficult, but in all these children there was also a true paralysis caused by injury to the brachial plexus, which was more or less complete and serious. Thus in one of them (J. T., æt. 8 months) some muscles were wasted and responded feebly to faradism (biceps and brachialis anticus), or had lost their contractility (flexors of fingers and interossei).

In another child (E. P , aged 15 months) all movement was lost, but electric contractility was normal, except in the extensor indicis.

In another the interossei and extensors of the wrist and

thumb were wasted and did not respond to electricity. The consequent disturbance of tonic equilibrium caused ultimately a deformity of the hand.

Since 1861 (when the foregoing cases were published) I have seen four fresh cases of sub-acromial obstetrical dislocation of the humerus, followed by paralysis limited to certain muscles of the corresponding limb.

Curative Action of Local Faradisation and Continuous Currents on Paralyses from Nerve-injury.—[The first effect of faradisation is on the sensibility, and paralysed limbs become (as we have seen) very sensitive after a few sittings. An over-sensitive condition of the muscles is always the herald of the return of movement and nutrition, and is always a good sign. This over-sensitiveness does not last long. It soon disappears, and the vitality of the muscles returns at the same time. It is not constant in old paralyses.

In these paralyses there is always a fall of temperature (5° or 6° C. as compared with the sound limb). The circulation is sluggish and the limbs blue. Faradisation always quickens the circulation and restores the temperature of the limb in a few sittings. Nutrition likewise improves, and in situations where the veins are plainly visible (as on the back of the hand) one may see them increase in size as the treatment progresses.

With regard to the return of muscular contractility, which follows close upon the return of sensation, warmth, and nutrition, it may be observed—

1. *That voluntary power is always preceded by tonic power.* Of this examples have been given.

2. *The curative action of local faradisation is quickly apparent in a muscle in proportion to its nearness to the nerve centres.* Thus in cases of injury of the brachial plexus the arm will recover first, then the forearm, and lastly the hand, and in each of these regions the same series of phenomena occurs, and in the same order, viz. :—

(*a*) Muscular hyperæsthesia.
(*b*) Quickened circulation.
(*c*) Increase of warmth.
(*d*) Improvement of nutrition.
(*e*) Tonic muscular power.
(*f*) Voluntary muscular power.

3. *The impairment of electro-muscular contractility persists after the cure and in spite of the return of voluntary power.*

This is a remarkable fact, but not the less incontestable. Electro-muscular contractility returns, however, after a time. Local faradisation cures the majority of cases of paralysis from nerve injury. This form of paralysis is assuredly that upon which local faradisation has the most happy influence.

I feel that the large proportion of cures which I can show from collected cases will seem to many practitioners an exaggeration; and I even fear that they may not obtain the like results. Local faradisation must not be blamed for this, but rather the want of attention to the rules which I have laid down and the numberless difficulties attending upon a treatment which needs both time and patience. It is a mistake, as has long been thought, and as some still believe, to think that the curative action of electricity must be rapid.

The mean duration of the treatment is from two to three months, but there are cases which require several years. I will give as an example the case of a workman in whom a dislocation of the humerus caused wasting and paralysis of the upper limb, and who was not quite cured at the end of two years of faradisation. In 1851, two years after the commencement of the treatment, I wrote, "His arm and forearm are well; in the hand the muscles of the thenar eminence have not yet recovered. How much time will be necessary for the recovery of these last muscles? I have no doubt that the muscles of the thenar eminence *will* recover, just as those of the arm and forearm have done, because the same signs of recovery are seen here as were seen in the other regions."

It has been urged that these cases would recover spontaneously, and Duchenne admits that some of them do, especially those in which the muscles retain their electric contractility. To place too firm a reliance upon the curative effect of time is, he urges, most dangerous for the well-being of the patient, and he insists that many hopelessly-crippled cases would have been in a much better condition if local faradisation had been used from the first regularly and systematically.

At what period should electricity be employed in cases of paralysis from nerve injury? It is evident from the preceding facts that we ought to act differently according as electric

contractility is retained or abolished. In the first class of cases it cannot be used too soon, and although such patients get well of themselves, still faradisation shortens the course of their trouble. It is certainly irrational to apply electricity for the purpose of recalling voluntary power, when the recently damaged nerves cannot give passage to the central nervous influx. But is it the same with muscular nutrition? Cannot the artificial stimulus replace the nervous stimulus to a certain extent? I used to think that local faradisation could not but produce good results, even when applied as early as possible. "But this opinion, expressed in 1852, which rested more on theoretical considerations than on exact experience, is unhappily not so well founded as I could wish, as many experiments made since then have shown me. No, the electric agent, however applied, cannot replace the spinal influence for the maintenance of muscular nutrition. It will be sufficient to recall cases given in this chapter in which we have seen muscles, whose innervation was seriously damaged, waste rapidly and almost quite vanish in spite of faradisation energetically used from the very onset of the trouble. It is only at a more distant time (after the regeneration of the far end of the nerve) that nutrition begins to recover in the muscles whose innervation has been damaged. Therefore I say *that the curative action of local faradisation is more rapid in old paralyses from nerve injury than in those which are recent.*

Seeing that in these cases the paralysis must last a certain time, and that recovery of the muscle is not possible before the recovery of the nerve, it is rather mischievous than otherwise to use faradisation too early, because the patient is fatigued thereby, and is unwilling to submit a second time to the treatment when it would be really useful to him.

Duchenne thus sums up his experience on this point.

1. Every paralysis from nerve injury, in which the electro-muscular contractility is not abolished, may be treated by local faradisation as soon as possible.

2. When electro-muscular contractility is lost, or at least is no longer appreciable to our means of investigation—a fact which shows that nerve-force no longer reaches the muscles—we must wait till the nerve regeneration has had time enough to be effected, and this is late in proportion to the lessening of muscular sensi-

bility. Then only has faradisation a chance of success; its application is even necessary.

Method of local faradisation.—Each muscle must be faradised in a special way according as it has suffered more or less in its contractility and nutrition. The more these faculties are in abeyance, the longer must be the application and the stronger the current. But when sensation begins to increase the treatment must be moderated, and the intermissions of the current be lengthened in order to repress neuralgic and even inflammatory symptoms. In all my cases I have excited the muscular sensibility as much as possible by means of intermissions more or less rapid, because I have found this the best means of acting on the nutrition of wasted muscles.

Nevertheless, too lengthy sittings and too rapid intermissions may fatigue and exhaust the muscle, just as forced exercise causes wasting, instead of favouring nutrition, like moderate exercise. I think that ten or fifteen minutes is enough for each sitting, and I rarely give more than a minute to each muscle. In order to avoid fatigue and cramp I pass rapidly from muscle to muscle, taking care to revisit each muscle many times during the sitting, and to leave time for repose between each stimulation. At the same time I lengthen the intermissions as much as possible, so as to render the application as little painful as possible, and not to run the risk by over stimulation of paralysing the nerves which control the local circulation and the nutrition of the muscles.]

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CHAPTER VIII.

ELECTRO-PATHOLOGY OF ACUTE SPINAL PARAPLEGIA.*

1. *Acute paraplegia of traumatic origin.*—[These cases are analogous to cases of paralysis from nerve injury, and the diagnosis and prognosis derived from the electrical exploration of irritability and sensibility are the same in both instances. In proportion to the loss of irritability and sensibility and the degree of wasting of the muscles is the difficulty of effecting relief or cure.]

Complete absence of muscular irritability and sensibility, together with anæsthesia of the skin, is a symptom far more serious in the case of traumatic injury to the cord than in similar injuries to mixed nerves. . . .

Case No. 52. *Summary.*—Fracture of spinal column in upper dorsal region caused by a fall of 50 ft. in a subject aged 19. Complete paralysis, loss of sensibility, loss of reflex contraction in the paralysed muscles. Loss of electric irritability and sensibility the sixth day after the accident, in the muscles of the anterior and outer part of the leg, in all the muscles of the thigh on the tenth day, and a few days later in the gastrocnemii, death on the thirty-second day.

The signs furnished in this patient by electro-muscular exploration showed a severe lesion of the whole of the lower part of the spinal cord. *It will be noted that from the sixth day a certain number of the muscles had completely lost their irritability and contractility, and that a few days later only a few muscles retained these functions, and that the sensibility of the skin was equally extinguished.* My prognosis was consequently very grave. The muscles, in fact, rapidly wasted. Sloughs were not slow to appear on the sacrum and heels, and the patient died a few weeks after my examination. I was unfortunately not

* From *L'Electrisation Localisée*, 3rd edition, pp. 447—458.

present at the autopsy, but I learned that the cord was completely softened for several inches in the dorso-lumbar region. The specimen was shown in M. Velpeau's Clinic. . . .

2. *Acute paraplegia of spontaneous origin.*—Acute paraplegia caused by a spontaneous and inflammatory lesion of all the constituent parts of the cord, no matter from what cause (diffuse myelitis, hæmorrhage, &c.), are characterised by the same troubles of sensibility and electro-muscular irritability as are the paraplegias of traumatic origin. . . .

Case No. 53. *Summary* —Sudden paraplegia in a young man, aged 26, without appreciable cause. Lumbar pain, formication in the lower limbs, anæsthesia of the legs, paralysis of bladder and rectum. Prognosis grave in consequence of the loss of electro-muscular contractility and sensibility. Sacral slough. Death on eighteenth day. Post-mortem; hæmorrhage and *red softening* of the cord from the level of the ninth dorsal vertebra, to its termination. (For details see *Union Médicale*, 17th February, 1859, p. 308. *Electris. Loc.*, second edition, obs. xxxi.)

After the fourth day I examined nearly every day the state of the electro-muscular contractility and sensibility in this patient till its abolition on the ninth day. I did not content myself by using moist rheophores applied to the skin, but I often drove needles (connected with my induction apparatus) into the gastrocnemii, and even thus failed to get any contraction. This rapid diminution of electric irritability in the muscles innervated by that part of the cord which was the seat of hæmorrhage is explained by the consequent inflammation. It will be remembered that microscopic examination demonstrated the existence of red softening of the cord from the level of the twelfth dorsal vertebra to the end of the cauda equina. This fact confirms the opinion of Brown-Séquard on the cause of the rapid diminution of electric contractility in lesions of the spinal cord in man. It will further be remarked that it is in perfect accord with the electric signs observed after lesions of mixed or motor nerves, and that the cause of the rapid diminution of electric contractility in these different cases is the same, viz., *irritation or inflammation.* Here then clinical observation appears to be at variance with experimental physiology, which after section of a motor nerve shows a persistence, more or less prolonged, of the electric properties of the muscles.

M. Charcot, at the Salpêtrière, endeavours to show that this want of accord is only apparent. He says, among other remarks, that in recent experiments made in Germany, Erb and Ziemssen, by bruising nerves, caused a neuritis more or less intense, rapidly followed by lessening and loss of faradic irritability in the muscles supplied by the damaged nerves. If in these experiments complete section of the nerves had been practised instead bruising them, the results obtained would have been very different. It is necessary, in fact, according to Charcot, in all that concerns the effects caused by nerve lesions on muscles, to take into account the fundamental and absolute difference between the effects of *want of action* and those of *morbid action.*

. . . . . .

Paraplegia from compression of the cord.

Compression of a mixed nerve always causes, besides paralysis, diminution or rapid abolition of electro-muscular contractility in consequence of the inflammation of the nerve, together with loss of sensibility and impaired nutrition of the muscles placed under the dependence of the nerve. I know no exception to this rule, which may be regarded as a law. It is not so with compression of the cord, in which the symptoms differ from those of compression of a nerve. Let us suppose, for example, that the cord is moderately compressed by an osseous tumour, by a neuroma, by acute incurvation of the spinal column, as in Pott's disease, or by some lesion of the membranes, the result of rheumatism or meningitis. The voluntary movements are, under such circumstances, weakened or abolished. Contractions, symptomatic of a sclerosis of the antero-lateral columns, might set in. Sensibility will be more or less interfered with, sloughs may occur on the sacrum, but electro-muscular contractility will remain normal, and the muscles will not waste at all, or only a very little, provided the grey matter of the front horns be not damaged. This kind of spinal paraplegia seems to me to be the most common of all.

. . . . . .

All these forms of paraplegia (from compression) are distinguished by a common sign of great value, viz., the reflex contraction of the muscles receiving their innervation from the parts of the cord below the point compressed, " and the integrity of the

sensibility" (*sic*). Thus in patients whose lower limbs are completely deprived of voluntary movement, simply by the compression of the lower part of the cord one may see, from a tickling or pinching of the skin, or from the impression of cold, the different sections of the limbs move suddenly and with more or less force, and quite unchecked by the will. The point where tickling of the skin ceases to produce reflex contractions shows the level of the compression of the cord. On the other hand, I have never been able to produce these reflex movements in limbs the muscles of which showed a weakened electro-contractility. The value of electro-muscular contractility as a sign will now be apparent, for its integrity in paraplegia shows that the anterior part of the cord is not materially damaged, and that therefore the nutrition of the muscles is not menaced. I have, often enough, made a happy application of this knowledge in the diagnosis of spinal paraplegias.

In support of this I quoted in the last edition a case of acute paraplegia, in which M. Oulmont, Physician to the Lariboisière Hospital, asked me to diagnose, by the aid of electricity, the anatomical state of the cord in a patient under his care already affected with sacral slough. The electro-muscular contractility being perfect, and reflex movements being present, I declared that the part of the cord which innervated the paralysed muscles was intact. The exactness of this diagnosis was verified by post-mortem examination.

Case No. 54.—In 1859 M Trousseau asked me to investigate, in a case of paraplegia under his care, the state of electro-muscular contractility, and to form a diagnosis therefrom. In this patient voluntary movement and sensibility were completely abolished in the lower limbs, and there was a large slough on the sacrum. From the integrity of electro-muscular contractility and reflex action I concluded that the part of the cord animating the muscles (the cells of the front horns) was intact. The truth of this was proved post-mortem. This patient, like the preceding, succumbed to the sacral slough ; the cord was perfectly sound. (For details see a paper by M. Moynier in *L'Union Médicale*, 1859.)

In short, electric exploration allows us to distinguish paraplegias caused by certain anatomical changes of the medullary substance of the cord from those which are symptomatic of a simple compression of the cord. In the first electro-muscular

contractility is weakened or extinguished; in the second it is intact.

Therapeutic Action of Localised Faradisation.—Acute spinal paralysis of the adult from wasting of the cells of the front horns, paraplegias from traumatic lesions of the cord (diffuse myelitis, hæmorrhage, &c.), can, like the traumatic lesions of nerves, be cured or improved by localised faradisation. No one will think of vaunting localised faradisation as a curative agent. In those cases which are caused entirely by inflammation of a part of the cord, electricity is merely an aid to the nutrition of the periphery cut off from central innervation. But it may happen that the inflammation, or the cause of the compression, may improve or get well, and yet the paralysis remain more or less complete. The paralysis is then peripheral, the muscle having merely lost, wholly or in part, its power of responding to central innervation (its nervous excitability). Now is the time when localised faradisation may re-establish movement. Cases of this kind are not rare in electro-therapeutics.

It is difficult, no doubt, to form a judgment of the opportune moment for interfering with localised faradisation in this kind of paraplegia. How are we to judge if the compression has diminished or vanished, and whether the affection causing the compression has been modified by suitable internal treatment? Faradisation can then only be used tentatively at first. I always interfere tentatively in this kind of paraplegia. When I do not get a complete result with faradisation, and even when I fail at first, I advise a trial to be made again later, after a continuance of suitable internal treatment. I have sometimes happened, by proceeding in this way, to hit on the right moment to cure or improve cases of paraplegia, which by their long duration drive patients to despair.

We may reasonably suppose that the cord, whose motor or trophic elements (the anterior cells) have been changed by inflammation or by too long compression, has lost its excitability to a greater or less degree. It is in these cases that strychnine, phosphorus, &c., have often re-established movement; and might we not with equal reason use electricity (faradisation or galvanisation) for acting on the spinal nerve centre, applying it in the continuity of the cord or nerves? Although I have tried these two kinds of electrisation, without success up to the

present time, in paraplegia resulting from acute myelitis or compression, I recommend them, and continue to use them after having failed with localised faradisation of the paralysed muscles.

Note by the Editor.

In his admirable account of paralyses from nerve injuries and paraplegia, it will be observed that Duchenne, although fully alive to the importance, from a diagnostic point of view, of a failure of muscles to respond to the faradic current, makes no mention of the *degenerative reactions* which muscles give under certain conditions, and for an account of which the reader is referred to an editorial note on page 169.

It need hardly be said that, since the object of electrical treatment in these cases is to maintain the nutrition of the muscles until the repair of the nerve lesion allows the voluntary stimulus again to reach them, the best form of current is always that to which the muscles most readily respond. This in almost all cases of nerve injury is the galvanic current. And further, since the galvanic current stimulates the cutaneous nerves quite as powerfully as the faradic, it is from the galvanic current that the most satisfactory results are likely to be obtained in the treatment of paralyses from nerve injury.

It will be observed that Duchenne gives some degree of adhesion to the theory that muscular wasting as well as extensive "tropho-neuroses," such as sacral slough, are dependent upon nerve *irritation*, a theory held by Charcot, Brown-Séquard, and other eminent authorities. Charcot even asserts that muscular atrophy (among other trophic changes) is more likely to occur "after *contusions, punctures, and incomplete sections of the nerves, i.e.*, after traumatic causes which are most competent to produce neuritis, or at least the neuralgic condition, than after complete sections of nerves."

It seems to me that this theory of the effect of "irritation" is very questionable. It certainly has never fallen to my lot to witness any case of nerve injury in which motor conduction in the nerve has been arrested for any considerable time without wasting of the muscles supplied by the injured or divided nerve.

It is possible to conceive the two ends of a divided nerve uniting so quickly that conduction is scarcely interrupted, or is interrupted for a time too short to allow of appreciable muscle wasting. Such cases, however, are rare, and it is now acknowledged that muscles certainly waste if the conductivity of their motor nerves be interrupted for a sufficient time. The so-called *irritative* lesions are for the most part inflammatory and destructive, and they tend to absolutely destroy the conducting power of the nervous tissues in which they are seated, and to replace a highly organised and irritable tissue by a hard, impenetrable scar-like gristle in which nervous repair is next to impossible.

It is a well-established fact that in ordinary cases of hemiplegia the paralysed muscles do not waste, and that in paraplegia from a limited injury to the cord the muscles supplied by the nerves emanating from the healthy cord below the lesion neither waste nor degenerate, although they are absolutely paralysed to the will. The reason for this I believe to be that the motor nerves and muscles of the paralysed area, although cut off from mental stimuli, are still accessible to stimuli reflected to them by the sensory nerves of the whole body, and this last form of stimulation, from which there is no possibility of escape, is sufficient to keep up the nutrition of the paralysed parts. Thus we find that in certain cases of paralysis there is neither wasting nor degeneration, while in certain other cases wasting and degeneration are a marked feature, and the following rule is one which I believe is capable of all but universal application.

As long as a paralysed muscle is in communication by means of a healthy nerve with a healthy portion of a nerve centre, it will neither waste nor degenerate; but if the nerve be divided or have its conductivity for motor impulses destroyed by disease, or if the portion of the centre from which the nerve originates has undergone destructive change, then wasting and degeneration of the muscle are sure to occur.

Duchenne's advice to us to persevere in the treatment of nerve injuries is, I feel sure, sound, and his statement that the chance of recovery in some cases increases, within certain limits, with the lapse of time is, I believe, true and in accordance with the teaching of pathology.

The following case and the reasoning upon it are taken from the Bradshaw Lecture which I delivered in 1881, and since it seems to me to bear directly upon the subjects of the previous chapters, I make no excuse for inserting it here.

In March, 1877, Miss F. W., aged 50, fell down a flight of steps, alighting on her right shoulder. Shortly after the accident the arm was devoid of motion and sensation from the shoulder downwards. When I saw her, three months later, the arm was much wasted; the deltoid, triceps, biceps, and all the other muscles of the limb gave degenerative reactions. The limb was absolutely useless, and sensation was entirely wanting below the middle of the forearm, and impaired elsewhere. On scratching the forearm, it was noticed that vascular reaction occurred far more readily on the sensitive than on the insensitive part. The galvanic current, applied to the sensitive parts, caused an immediate uniform redness, but on the insensitive parts it slowly produced an irregular patchy redness like a lichenous rash. The fingers were scurfy and bulbous at the tips, and the patient complained that they got hot at night. Galvanisation was efficiently and methodically applied to the arm by the patient's sister. By November, sensation had returned down to the finger-tips (imperfectly). The wrist was movable; the hand clawed and paralysed; the nails furrowed and white. During the winter the hand was covered with chilblains. In April, 1878, the arm was well, but the intrinsic muscles of the hand remained paralysed; the nails were a dead white. In July, 1878, the motion and sensation of the hand were still impaired, and the muscles of the hand gave degenerative reactions. In June, 1879, the intrinsic muscles of the hand responded to faradism. There were glazy spots at the roots of the finger-nails; nails harsh, hard, and grooved. In June, 1880, the hand was fairly useful, though still liable to chilblains, and the sensation still imperfect. In a letter dated August, 1881, this patient writes: "It is really quite a natural colour. Nails of thumb, first and third fingers well; and the others much better. Performance on the piano quite grand; I can reach an octave, but cannot strike it without holding the thumb. The thumb and first finger are the worst parts, as the thumb will not go out quite far enough, and the first finger is inclined to bend back. The chilblains last winter were nothing to what they were before."

This case is one of surpassing interest, and is most instructive from the point of view of nerve pathology. First, as to the time of duration of the symptoms. The accident happened in March, 1877, and in August, 1881, her recovery, though still progressing, is yet not complete, so that it has already been protracted over a period of nearly *four years and a half*. During the whole of this time there has been a steady, slow, evenly progressing recovery, so that her improvement was recognisable from month to month. Recovery has taken place in a regular order: sensation returned first, and always preceded the return of motion; and muscles situated nearest to the trunk recovered before those which were more distant from it. At present two muscles of the hand, the abductor of the thumb, and the first dorsal interosseous, alone remain paralysed. From what is known of nervous degeneration and regeneration one could hardly have expected the course of events to have been otherwise. The injury in this case was severe. The nerve-trunks were probably bruised and torn by the head of the humerus, so that immediately after the accident they were completely "blocked" to upward sensory impressions or downward motor stimuli. A disused nerve degenerates along the line of function, and the motor nerves in this case rapidly degenerated from the point of blocking down to their terminations, for no stimulation could possibly reach them either directly from the brain or be reflected to them through the spinal cord. Accordingly, within a month or so after her accident, it is probable that each motor nerve had been converted into a degenerated cord some three-quarters of a yard long, as incapable of conducting stimuli as the skin, fat, or connective tissue. Now, as the degeneration of a nerve takes place from trunk to end, so its regeneration takes place in the same direction, for there can be no regeneration unless physiological or artificial stimuli reach the point to be regenerated. Motor stimuli, both direct and reflex, impinging against the block, gradually induce molecular regeneration and the power of conducting stimuli; and thus little by little, as the stimuli are able to travel further, we get a complete regeneration of the nerves (a process which in this patient has already taken four and a-half years). It is quite inconceivable that the stimulus which seems necessary for the regeneration of a nerve should be able to pass by any unregenerated portion. One might as well expect the makers of a well to begin

their work in the middle. Just as a well has to be tediously bored, so is a degenerated motor nerve slowly opened for the traffic of stimuli from above. Provided stimuli can reach the nerve the power of regeneration seems almost unlimited, and, indeed, when pieces are cut out of a nerve-trunk the severed ends seem capable of worming their way, as it were, towards each other, and eventually effecting a junction in spite of every adverse circumstance. It seems probable that the bulbous ends in amputation stumps must be looked upon as an overgrowth due to the arrival of stimuli in the stump of the nerve, which are unable to produce their proper physiological effect. Bulbous nerve-ends, according to Weir Mitchell, are physiological rather than pathological, and are almost invariably present in stumps.

The earlier return of sensation in all cases of nerve-injury is due to the following facts.—1. That the sensory branches from the periphery to the obstruction are not deprived of their natural stimuli, and presumably do not entirely degenerate, and are consequently ready to resume their full functions directly the obstruction on the up-path is removed. 2. That in the parts above the block the incidence of impressions of various kinds probably serves to keep up in some degree the healthy condition of the nerve; it is, in short, next to impossible to deprive a sensory nerve of its natural stimuli. 3. That, owing to the anastomoses which exist between sensory nerve branches, impressions have the power, to some extent, of choosing the path of least resistance. Hence, happily, it results that sensory impressions, so important for the nutrition of the limb, are able to produce their physiological effects in spite of very serious injuries.

A few words as to the trophic troubles observed in this patient. These were of three kinds:—1. Muscle-wasting. 2. A sluggishness or absence of vascular reaction when the skin was stimulated. 3. Scurfiness of the skin, loss of nails, and a tendency to chilblains.

Why does it happen that muscles paralysed from a brain-lesion scarcely waste at all, while those paralysed from destruction of the motor cells of the cord, or from injury to a nerve-trunk, rapidly waste and degenerate? Because, in the first case, physiological stimuli still reach it through the cord, while in the second case it is completely cut off from every source of

physiological stimulation. So long as a muscle be stimulated it will maintain its size, no matter whether the stimulus come to it from the brain along the will-path, or from the surface or deep parts along the path of reflected impressions, or be artificially applied to it by means of a galvanic battery. If, on the other hand, a muscle be cut off from all sources of stimulation, it will waste. I do not, of course, mean to say that there may not be other causes for muscle-wasting, but I have never encountered an exception to the rule I have enunciated.

With regard to the trophic changes, I speak with less confidence, but I must express my absolute disbelief in the existence of special trophic nerves; and we clearly ought to exhaust every possible explanation before we proceed to do what is far too common in nerve pathology, viz., to give "to airy nothings a local habitation and a name." I believe that tissue-changes other than muscle-wasting are often due to the cutting off from the vessels which supply those tissues those physiological stimuli which produce the contraction and dilatation of the vessels, and thus exercise a local control over them. Every paralysed limb is deprived of one important aid to circulation, viz., the muscular contraction, which is a material aid to the circulation, especially in driving the blood towards the heart. Every paralysed limb must therefore be at a nutritive disadvantage, but yet in hemiplegic limbs whose connections with the spinal cord are normal, no trophic changes usually occur, except a little congestion. When the motor cells of the cord are alone destroyed, no trophic changes occur. When a mixed nerve is destroyed, trophic changes are very liable to occur to some extent. When the posterior roots and sensory paths of the cord are damaged, trophic changes often occur, as witness the joint affections and occasional zona of locomotor ataxy. When a purely sensory nerve, such as the fifth, is damaged, trophic changes are common, but not invariable; their occurrence, according to some, depending upon the implication or otherwise of the Gasserian ganglion.

Now, in the case we have been considering there were trophic changes. There was impaired vascular reaction on stimulating the insensitive parts of the limb, and it is tolerably certain that cutaneous impressions were not reflected to the vaso-motor nerves of the limb. The local circulation was therefore cut off from

the stimulus of muscular motion and from the stimulus of cutaneous impressions. It must not be forgotten, however, that impressions made upon one limb seem capable of being reflected to the vessels of the other, so that impressions made upon the left arm and other parts of the body were probably able to reach the paralysed limb. This fact is very suggestive of the importance which it is in the animal economy that cutaneous impressions should reach the vaso-motor nerves. A sensory impression is felt, if I may be allowed the expression—(1) by the brain; (2) by the muscles, being reflected to them by the spinal cord; (3) by the vessels, being reflected to them by the cord or the ganglia on the posterior nerve-roots. It must be remembered that conceivably any one of these paths for sensory impressions may be blocked, while the other two may remain open; that any two may conceivably be blocked; or that all three may be blocked. The question is very complicated and very difficult to study. The dependence of nutrition upon a due connection between the sensory and vaso-motor nerves combined with muscular motion seems to me extremely likely. Its absolute proof or disproof seems almost impossible.

CHAPTER IX.

LEAD PALSY AND "VEGETABLE PALSY."*

I devote this chapter to the combined study of lead palsy and the paralyses following the so-called "vegetable colic" of Madrid, because, in character, course, and treatment they are as much alike as if the causes were the same.

LEAD PALSY.—I have studied the muscular state in lead palsy with the help of local faradisation in more than 150 cases. . . . Lead palsy is usually limited to the upper limbs; sometimes, however, it is general from the first.

Case No. 55.—Lead palsy of the two forearms (under M. Andral at the Charité).—Gervais, a typefounder since 1835, had for the first time in November 1845, an obstinate constipation with fever and vomiting. The constipation yielded in seventeen days, after leeches to the abdomen and purgative enemata. A month later he was unable to extend the right index and thumb, and this disappeared in three months after a course of sulphur baths. In November 1846, he had a second similar attack. In 1847 he had violent pain along the back of the forearm increased by pressure especially at the lower fourth of the radius. He came into the Charité and left cured at the end of a fortnight. He soon noticed that he could not extend the middle and ring fingers of the right hand. He continued at his work, though the paralysis increased, spreading to the other fingers and to the muscles of the front of the forearm. In January 1849, the paralysis attacked the middle and ring fingers of the left hand, and in April there were pains in both arms followed by paralysis of the other muscles on the back of the left forearm, except the extensor secundi internodii, extensor ossis metacarpi pollicis, extensor of the little finger, and supinators. He was then admitted to the Charité, and for the first three weeks was treated with blisters, sulphur baths, and strychnine (both by the mouth and hypodermically) without any appreciable

* From *L'Electrisation Localisée*, 3rd ed., pp. 671—691.

result. As the strychnine caused trouble, local faradisation was had recourse to.

On his admission I established the following facts. On the *right side* there was forcible flexion of the wrist and fingers without any power of voluntary extension, but if the near phalanges were straightened for him he could extend the two end ones. Adduction of the wrist and supination were preserved. There was considerable wasting of the muscles of the back of the forearm, so that the outline of the muscles had disappeared, leaving a depression between the radius and ulna. On the *left side* there was wrist-drop, with some slight power of extension by dint of effort, and loss of extension of the first three fingers; movements of thumb and little finger, and adduction of wrist preserved. The other muscles were less paralysed than on the right side.

Under the influence of local faradisation, I noted *on the right side* that the extensor communis digitorum, extensor minimi digiti, extensor secundii internodi, and the extensores carpi radiales (*les radiaux*), did not contract to a maximum current with moist rheophores, and even electro-puncture (a needle being plunged into the muscles) only caused a few fibrillary contractions with the most intense current. The extensor ossis metacarpi still contracted, having lost only about a third of its electro-muscular contractility. The other muscles of the forearm were (electrically) normal. On the *left side* the extensor communis digitorum and the extensor secundi internodii contracted as feebly as on the right. The extensores carpi radiales had lost about half of their contractility, but the other muscles of the forearm and hand were normal. In the muscles which failed to contract to faradisation the patient experienced a sensation like that which accompanies the contraction of healthy muscles, but this sensation was very feeble.

I have given this case in detail because it is a true picture of one of the most frequent forms of lead palsy.

[In a similar case of lead palsy affecting the left arm, it was found that—1. The extensor communis digitorum and the extensores carpi radiales did not contract to intense currents. 2. That the other muscles of the arm and forearm retained their contractility, except the middle part of the deltoid, which did not contract to the strongest current,

although the corresponding muscle contracted perfectly to a feeble current. 3. That electro-muscular sensibility was only feebly diminished even in those muscles which had lost their contractility.]

. . . . . .

In these cases there is one striking fact, viz., that the loss of electro-muscular contractility always seems to affect certain muscles by preference, even when the whole limb is paralysed.

If close attention be given to the course of this disease, and the order in which the muscles are affected, it will be found that the *extensor communis digitorum*, *extensor indicis*, *extensor minimi digiti*, and the *extensor secundi internodii*, are the first to suffer in their electro-contractility.

When the common extensor alone is paralysed, the patient loses the power of extending the near phalanx of the middle and ring fingers, the index and little fingers retaining some power, thanks to their proper extensors. The trouble may affect singly and unequally not only each of these extensor muscles, but even the separate bundles of the extensor communis. Thus in a patient with partial paralysis of the hand I noted a considerable loss of electro-contractility merely in the extensor indicis and the extensor secundi internodii, muscular power being generally weak in the forearm. In other cases the extensor minimi digiti retained its contractility even when the other extensors had lost it in whole or in part.

From the extensors of the fingers the loss of electro-muscular contractility spreads to the extensores carpi radiales. They may be affected singly or together. In the first case it is always the *extensor carpi radialis brevior* which is first attacked, and when they are affected together they are unequally affected, and the "*brevior*" is always the worst.

Finally, the extensor carpi ulnaris (*cubital posterieur*), the extensor ossis metacarpi pollicis (*long abductor*), and the *extensor primi internodii*, are almost always the last muscles of the back of the forearm which lose their faradic contractility. In all my patients the supinators and the anconeus have preserved their contractility intact. I will not try to explain the immunity of these muscles, which, equally with others, are supplied by the musculo-spiral nerve.

In lead palsy the muscles of the front of the forearm and palm

retain their normal contractility, although their voluntary power suffers slightly.

Although I have just said that the muscles of the palm are not ordinarily attacked, I have nevertheless six times seen the muscles of the right thenar eminence deprived of electro-contractility, and much wasted in painters suffering from lead palsy. I formerly attributed this wasting to the pressure of the handle of the brush and not to the lead, but I now believe that the lead is the real cause of it. I have noticed that this form is often seen in slow cases. I have seen two examples, among others: one in a designer who was poisoned by holding his brushes in his mouth, and who had double wrist-drop and an incomplete wasting of the thenar eminence on the right side; the other in a colorist in whom the disease was limited to the right side. In the last case the paralysis of the extensors was soon cured by local faradisation, but the wasting of the thenar eminence persisted. In both these patients, as well as in those in whom I have noticed a similar wasting, the lead poisoning was produced slowly by small doses.

The altered electric contractility is not always limited to certain muscles on the back of the forearm. It often affects the deltoid without affecting the arm. In the arm the triceps is more often affected than the biceps. Finally, when lead palsy affects all the muscles of the upper limb, in addition to the supinator and the muscles on the front of the forearm, I have always noticed that the pectoralis major, the trapezius, and the muscles attached to the infra-spinous fossa (*les muscles qui s'insèrent dans la fosse sous épineuse*) contract naturally to electricity . . .

The deltoid is sometimes the first muscle to be attacked. . . . Does the loss of electric contractility precede or follow the loss of voluntary power? I have not yet the material for answering this question; but I have, nevertheless, noticed even in the most recent cases that the loss of electric contractility had reached its maximum, and I have never known it to increase progressively, as in other cases of paralysis.

Although the muscles lose their contractility they retain a great part of their sensibility.

A limb the seat of lead palsy is usually thin, but it is noteworthy that wasting attacks mainly those muscles which have

suffered in their contractility, and I believe it is these last which have undergone the action of lead poisoning. The other muscles are less damaged, and quickly recover their power under treatment.

Pathological anatomy confirms this view, as the following case shows:—

Case No. 56.—In 1851 a man died in the St. Louis Hospital, under M. Briquet, who had had a bilateral lead palsy of some muscles on the back of the forearm for more than twenty years. I had proved during life that these muscles had lost their contractility, while those on the front of the forearm retained it. These latter were weak and wasted, it is true, but to a much less extent than the paralysed muscles. Post-mortem, those muscles which had lost their electric contractility were found to be of a pale yellow colour, and, with the microscope, were shown to be partly in a state of fatty degeneration. The muscles on the front of the forearm, on the contrary, were in a normal condition.

It is only after many years that the change in the texture of the muscles apparently begins, for I have seen, in the wards of my late friend M. Aran, the muscles of a patient who died of fever, but who had had lead palsy limited to the forearm for six or seven months. The muscles, in which I had established a considerable diminution of electric contractility, and which were much wasted, were shown, by the aid of the microscope, to have perfectly normal fibres.

Lead palsy sometimes attacks the whole system at once; and I have seen in such cases the diaphragm suffer with the rest of the body. . . . Even when the paralysis extends to all the muscular system I have noted that the loss of electric contractility has its seat of election in certain muscles.

[*Case No.* 57 is that of a worker in a white lead factory, who was entirely paralysed, except in the face, and whose muscular system had rapidly wasted. Duchenne proved that electro-muscular contractility was

LOST IN	RETAINED IN
Extensores communes digitorum.	Supinators.
	Muscles of front of forearm.
Extensores carpi radiales.	
,, pollicis.	
,, carpi ulnares.	

DIMINISHED IN

Extensores secundi internodii.
Deltoids.
Muscles of arm.
Extensors of knee.
Extensors of toes.
Pectorales majores.
Recti abdominis.

This patient recovered rapidly under treatment, excepting in those muscles which had lost their electric contractility, and even when these last recovered their power of voluntary contraction it was found that they still failed to respond to the strongest electric currents. This singular condition is referred to elsewhere.] . . .

PARALYSIS FOLLOWING THE SO-CALLED VEGETABLE COLIC OF MADRID.

This disease in all particulars closely resembles lead palsy.

. . . . . .

[Duchenne quotes several cases which are nothing more nor less than cases of lead palsy, presenting all the symptoms, inclusive of the grey line on the gums. Many of the cases occurred in persons employed on board ship, and Duchenne is inclined to think that the distilling apparatus (the worm of which is made of lead) may have been the source of the lead poisoning.]

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TREATMENT.—[Duchenne insists on the importance of employing local faradisation, which he has found of great service when all other means of treatment have failed.]

Lead palsy interferes with the use of the hand in proportion to the damage to the extensors. For their individual treatment it is important to diagnose exactly the condition of each of these extensors, which, without counting the extensors of the fingers, are three in number; the extensor carpi radialis longior (*premier radial*), the extensor carpi radialis brevior (*second radial*), and the extensor carpi ulnaris (*cubital postérieur*). Electric exploration is the best method of determining their

condition, but there are other ways which I will indicate. . . . If (in a healthy individual) one causes the alternate contraction of these three muscles, it will be observed—1. That the extensor carpi radialis longior extends and abducts the wrist. 2. That the extensor carpi radialis brevior extends the wrist without causing any lateral movement. 3. That the extensor carpi ulnaris causes a feeble extension of the hand with adduction. If, with the hand adducted, the extensor carpi radialis longior is made to contract while the extension of the hand is opposed one sees it produce a great lateral movement until the maximum of abduction is reached. If the extensor carpi ulnaris is then made to contract, the extension of the wrist being prevented, a great movement is produced in the opposite direction. The extensor carpi radialis longior is the *extensor abductor*, the extensor carpi ulnaris is the *extensor adductor*, and the extensor carpi radialis brevior is the *direct extensor*. When these three muscles are paralysed together the wrist drops and the patient is quite unable to extend it, no matter what efforts are made. If the extensor carpi ulnaris remains sound, while the other two are paralysed, the wrist drops and is dragged in the direction of adduction, so that the fifth metacarpal bone makes with the ulna an angle, more marked than in health, because the adducting power of the extensor carpi ulnaris is not antagonised by the extensor carpi radialis longior.

The paralysis of the extensor carpi radialis brevior causes the wrist to drop, but the patient by shutting the hand (*i.e.*, by shortening the flexors of the fingers and neutralising their antagonism) can raise the wrist, which falls again directly there is the slightest extension of the fingers.

If the paralysis of the extensor carpi radialis brevior is complicated with paralysis of the extensor carpi ulnaris; the extensor carpi radialis longior, which is no longer checked by its direct lateral antagonist, keeps the wrist forcibly abducted (*adduction* ? in text).

There is no need of insisting on the importance, in diagnosis and prognosis, of a knowledge of these physiological and pathological facts revealed by electricity. On first examining a patient with lead palsy of the arms I always make him hold his arms in front of him, and by the attitude of his wrists alone I can tell if his extensors are wholly or partially paralysed.

1. If the wrist drops and is not abnormally abducted I am sure that all three muscles are paralysed.

2. If the wrist drops and is adducted I conclude that the extensor carpi ulnaris is sound, while the extensores carpi radiales are paralysed.

3. If the patient can raise his wrist only after shutting the hand I know that the paralysis is limited to the extensor carpi radialis brevior, if the wrist is extended straight on the forearm.

4. Finally, if when the wrist is raised in this way it is abducted I know the extensor carpi ulnaris is paralysed as well as the extensor carpi radialis brevior.

These signs are invariable, and have never deceived me.

An exact knowledge of the amount of damage to the muscles in lead palsy is important, because the electric stimulation of each of them ought, both as regards intensity and duration, to be proportioned to the intensity of the paralysis.

The radials are the first to recover under the influence of faradism. This is fortunate, for without them the hand is almost useless. And further, the flexors of the fingers can only act provided the flexors and extensors of the wrist maintain it as steady as if it were part of the forearm.

The extensor carpi radialis longior is the first to recover, and this almost always long before the extensor carpi radialis brevior. I have seen cases in which the latter has remained persistently very weak.

It would have been unfortunate if the extensor carpi radialis longior had shown itself the most sensitive to the poison of lead and the most stubborn under treatment, for this muscle is more indispensable than the extensor carpi radialis brevior for the utility of the hand. I may quote the case of young M. X., who had been long deprived of his extensor carpi radialis longior, but managed nevertheless to extend his wrist with his extensor carpi radialis brevior and extensor carpi ulnaris. But his wrist was so much adducted that certain uses of the hand were much hindered thereby. Thus, if he wanted to touch his mouth or the opposite ear with his fingers, the attitude of his hand thwarted the movement by its inclination towards the ulnar side.

Almost as soon as patients regain the power of extending the wrist they resume their usual occupations in spite of the paralysis of the extensors of the near phalanges (loosely styled

the extensors of the fingers) and the extensors of the thumb. Thus they write easily enough provided there be no paralysis of the *extensor primi internodii*, and painters hold their brushes firmly. [These patients are often sent back to their work before their recovery is complete, and then they speedily relapse. If, however, they can be persuaded to give up their dangerous occupation their cure is lasting.]

I have always noticed that the deltoid soon recovers under local faradisation. . . . [When the paralysis is general those muscles recover easily enough which are not the ones usually affected in lead palsy.]

The method of faradising which I have found most useful in the treatment of lead palsy is almost precisely that which I use for paralysis from nerve injury. Thus—

1. The current of the primary coil, as strong as possible and with rapid intermissions, must be used, and mainly to those muscles whose contractility and sensibility are the weakest. The current of the primary coil with rapid intermissions is, as one knows, that which acts most powerfully on muscular nutrition and sensibility at the same time that it recalls voluntary power.

2. The sittings must not last more than ten minutes. Longer sittings may cause cramp, and be followed by pain, and produce an opposite effect to that desired.

3. Sittings renewed every other day have been found to answer well with my patients.

4. They were treated solely by local faradisation. Nevertheless the association of sulphur baths, strychnine internally, gymnastic exercises (of the kind which I have called *nervous*), and different forms of shampooing, cannot fail to hasten the cure.

[*Continuous currents*, which were found most useful by Remak in the treatment of lead palsy, proved far less useful than faradisation in the hands of Duchenne.]

CHAPTER X.

PARALYSES FROM COLD.*

I HAVE seen the continued impression of cold cause an acute spinal paralysis, or a paralysis following a neuritis or primary nerve paralysis (usually of the musculo-spiral), or a paralysis following muscular rheumatism, especially of the deltoid. Having elsewhere described acute spinal paralysis, I need only discuss the other forms in this chapter.

Paralysis following Neuritis the result of Cold.

After a neuritis caused by cold the pains sometimes disappear almost suddenly, and the regions supplied by the inflamed nerve are stricken with paralysis of sensation and motion. A few days later the muscles lose their electric contractility and waste.

SYMPTOMS AND COURSE.—At the commencement there is pain along the course of a nerve, feeble at first, and then increasing rapidly. I have often known the pains violent from the first. They are usually continuous, but with paroxysms of severe pulsating stabbing pain radiating from the damaged nerve. They are accompanied by over-sensitiveness, creepings and prickings in the end of the limb supplied by the nerve. They are increased by movement or by pressure of the inflamed nerve, the size of which is increased. Some patients have complained of a little fever at the outset, and almost all have said that the affected limbs were too hot.

These pains, difficult to diagnose, have been attributed to neuralgia. The only sign which could lead one to suspect or recognise the existence of neuritis is the increase of temperature in the region where the pains are situated.

But there soon appear unmistakeable signs of the organic mischief in the painful nerve. In fact after lasting some time (it may be months) the pains cease, usually suddenly, and give place

* From *L'Electrisation Localisée*, 3rd ed., pp. 692—710.

to paralysis of the muscles supplied by the affected nerve. At the same time there is a lessening or loss of the sensibility of the skin and muscles of the paralysed area, and the electric contractility of the muscles is also lessened or lost. Lastly, the muscles waste by degrees, and there is a fall of temperature in the disturbed region, with pallor of the skin, and a lessening in the size of the veins.

Cause, &c.—The name shows that the impression of cold and prolonged chill during sweating are the usual causes of this kind of paralysis. . . . I have met with paralytic neuritis in most of the nerves of the limbs. I will give only one example.

Case No. 58.—My worthy *confrère* and friend M. Campbell, sent me in 1870 a workman from the Mint, who had a wasting paralysis, with loss of electric contractility and sensibility in all the muscles supplied by the musculo-spiral (radial) nerve. It had been preceded by frightful pain in the region of this nerve and its branches. At the outset there had been one or two days of fever with burning pain in the affected limb.

After three months the pains disappeared suddenly, and the muscles supplied by the nerve were paralysed and wasted rapidly. From this time the temperature of the limb fell. Blisters having been used to the back of the forearm without effect the patient was sent to me to be treated for his paralysis.

By reason of the long duration of the neuritis, the rapid wasting of the muscles, and the loss of electric contractility the prognosis seemed to me to be grave, but experience had already taught me that this kind of paralysis could be cured by local faradisation. This, in fact, happened in this case: after fifteen sittings I saw movement and nutrition begin to return.

This case was seen by some who attend my clinic (among others by M. Charcot, who was present on one occasion). It may serve for instruction as to the prognosis of paralytic neuritis, and the best way of treating it electrically.

Rheumatism of the Deltoid.

[The chilling of the shoulder when the skin is sweating often causes a trouble called rheumatism of the deltoid, and of this I have noticed three forms, viz. :—

1. *Simple rheumatism of the deltoid.* This is characterised by pain only, often deep, tearing and stabbing, and increased by movement. The whole or part only of the deltoid may be

affected. The pain is so great that the patient dares not move his arm, and believes himself paralysed. The trouble may last days, weeks, or months.

2. *Wasting rheumatism of the deltoid*, in which the pains are succeeded by wasting of the whole muscle, or of that part of it which is affected, although voluntary and electric contractility are preserved. After the pains have disappeared there is weakness of the muscle proportioned to the wasting.

3. *Paralytic rheumatism of the deltoid*, in which the pains are followed by paralysis. In this form there is loss or lessening of electro-muscular contractility and sensibility.

The last form is undoubtedly due to a true neuritis, for not only are all the symptoms present, but this opinion has many times received support from the fact that undoubted neuritis of other cords or filaments of the brachial plexus has been present at the same time.

The first two forms are not caused, Duchenne thinks, by an organic change in the circumflex nerve, but rather by neuralgia, irritation, or inflammation of its ultimate twigs (*travail irritatif ou inflammatoire*).

For the *treatment* of the first two forms Duchenne strongly recommends faradisation *locally applied to the skin over the deltoid*, care being taken not to allow the muscle to contract. The skin should be powdered with starch, and the rheophores should be dry, or the hand of the operator previously powdered may be used as a rheophore.

He has found faradisation far more useful than continuous currents, and although he has often known the former succeed where the latter failed, he has never noticed the reverse effect.

In the wasting form Duchenne recommends the alternate use of faradism to the skin, and the continuous current, "the first in the hope of curing the rheumatic pains, and the second to quicken the nutrition of the wasted deltoid. This method of mixed and rational electrisation has appeared to me the best. By faradisation of the skin I have conquered the pains which did not yield to the continuous current, and by applying the latter so as not to cause muscular contractions in the deltoid I have aided the return of nutrition without running the risk of bringing back the pains, which might have been done by muscular faradisation."]

Paralysis of the Musculo-spiral (radial) Nerve from Cold.

Symptoms.—There are no premonitory symptoms. It is usually after sleeping with the arm exposed to a cold draught, or resting on the damp ground, that the patients feel on waking a numbness and creeping to the ends of the fingers. They can neither raise the wrist nor extend the fingers. There is complete paralysis.

A. The physiological investigation of the voluntary movements shows that the paralysis affects all the muscles supplied by the musculo-spiral, and is strictly limited to these. This is shown by the following facts:—

1. With the elbow half flexed and the forearm half prone, if the patient be asked to bend the elbow more while the movement is gently opposed, there is not felt any contraction or hardening of the supinator longus. This shows a paralysis of the muscle which I have experimentally demonstrated to be a flexor of the half-pronated forearm (*fléchisseur semi-pronateur de l'avant bras*).

2. When the arm is extended and pronated, supination cannot occur, unless the *biceps* energetically contract and half flex the elbow. This would not occur if the *supinator brevis* were able to act, for it is the only independent supinator muscle, while the *biceps* causes flexion and semi-supination concurrently. I have never noticed this evidence of paralysis of the *supinator brevis* in lead palsy.

3. The flexed wrist can neither be raised nor moved sideways by the patient when it rests on a flat surface. This shows paralysis of the extensores carpi radiales and extensor carpi ulnaris.

4. The patient cannot extend the near phalanges (which are flexed on the metacarpals), owing to the paralysis of the extensor communis digitorum.

5. While the fingers are thus flexed in the palm he cannot move them sideways. From this one might think that the interossei were weak or paralysed, but if the hand be placed on a flat surface it will be seen that the fingers can be made to move to and fro (sideways). It is easy in another way to show that the interossei are sound, for when the near phalanges are straightened (passively) it is seen that the patient can extend the two last phalanges on the first. To understand why he can do

this when the extensor communis is paralysed, it must be remembered that I have shown the interossei to be the only extensors of the two end phalanges.

6. When the patient closes his hand it can be felt that the flexors of the fingers have very little power. Nevertheless these muscles are not paralysed. To prove this it is sufficient to hold the wrist straight firmly, when the flexors of the fingers will be found to contract forcibly. The weakness of these muscles was merely apparent, and was due to the slackness of the tendons produced by the flexion of the wrist from the paralysis of its extensors.

The paralysed muscles retain their normal electric irritability, and usually also their sensibility. Most of the uses of the hand are brought to naught by palsy of the musculo-spiral. I have only once noticed that the sensibility of the skin of the forearm and hand was weakened, although the muscular sensibility was maintained.

Such are the symptoms at the outset of this form of paralysis.

B. In a more advanced stage the muscles gradually waste. This wasting may in the end be considerable, but I have never seen the muscles changed in texture.

In the end also other muscles of the forearm and hand, especially the flexors of the fingers, fall into a state of semi-paralysis. Thus the patient closes his hand very feebly, even when the wrist is straightened for him, and can hardly extend the two last phalanges when the first are passively extended. This feebleness is caused by the inaction and continued shortening of these muscles, for if their exercise be encouraged by an apparatus for keeping the wrist and first phalanges extended we soon see their power return.

C. Paralysis of the musculo-spiral from cold is in itself painless (*indolente*). Nevertheless it often happens that the continued flexion of the wrist ends by causing great fatigue of the carpal joint and a painful swelling on the back of it, which seems to me to be produced by a distension of the extensor tendons.

Cause.—The form of paralysis which I am describing is caused, as I have already said, by exposure to moist cold, or to a draught, &c., acting specially on the forearm. I have collected more than 100 cases of it. In almost all a continuous slight chill of the forearm caused the immediate and complete paralysis of the

musculo-spiral nerve, and strangely enough it almost always occurred during sleep.

It is important, therefore, to know how this palsy may be produced.

Workmen, for example, when taking their accustomed after-dinner nap go to sleep under a shed with one of the arms, generally naked, exposed to a draught. They may sit with their arms crossed, or lie on the cold or moist ground with one of the arms under the head. They remain thus for an hour or two, and when they wake the arm is paralysed.

I have seen several persons who, having in hot weather fallen asleep on the shaded turf, have suffered in a like way.

It is not less dangerous to fall asleep with the naked arms outside the bed, placed beneath the head, for if a draught strike upon one of the sweating arms this same paralysis may be produced. Of this I have many examples.

Finally, I have known this palsy produced in the following way. Two people fall asleep with their naked arms crossed on the chest near a stove or in a hot room while the window or door is open. In one case it was a portress who fell asleep in her chair after doing a little washing, with her wet arms crossed on her chest. In another case it was a barber of Villette who, in his shirt sleeves, was taking a nap in his chair. On awaking both these were paralysed like the others, and that arm was paralysed which had been exposed to the draught, without having been in the least degree pressed upon. These examples suffice to show that exposure to cold or draught is a cause of this form of paralysis. I agree that in some cases the compression of the back of the forearm or of the musculo-spiral nerve has had some influence, but it seems to me certain that in the large number of cases I have seen in the last twenty years (about 100) exposure to cold has been the chief factor in the causation.

Does the damage which causes this paralysis lie in the nerve or is it peripheral? If the cause (draught and contact with the cold moist earth) really acted on the periphery, *i.e.*, directly on the muscles, the superficial muscles of the back of the forearm would suffer more than the deep muscles. But all the muscles supplied by the musculo-spiral nerve are always equally affected. And again, I do not see why muscles adjoining those which are

supplied by the musculo-spiral, and which have been equally exposed to cold, &c., should not be equally paralysed. The sudden paralysis of the musculo-spiral from cold seems to me to be caused by a congestive hyperæmia of an irritative nature (*par une hyperhémie congestive de nature irritative*). But if the damage really exists in the nerve, why do not the muscles lose their irritability, as happens in rheumatic paralysis of the seventh, after exposure to cold? It is possible, I think, to explain the difference existing between these two forms of paralysis, caused in the same way, by anatomical considerations. In fact, if it be true that rheumatic nerves grow big, the bony canal (Fallopian aqueduct) traversed by the seventh must oppose this increase in size, and consequently squeeze the nerve so as to diminish the irritability of the muscles supplied by it. As this cause for compression does not exist in the musculo-spiral, it is intelligible how there may be a paralysis of the nerve without any weakening of electro-muscular irritability.

Diagnosis from Lead Palsy.—[This is important, and sometimes difficult. Duchenne asserts that in lead palsy electric irritability is lessened or lost, whereas in paralysis of the musculo-spiral from cold it remains normal. "I declare," he says, "that up to the present time I have not met with a single exception to the law which I have formulated, viz., that *in lead palsy electric irritability is lessened or lost in certain muscles*." He warns his readers that the establishment of slight degrees of lessened irritability requires very great care and good instruments capable of fine graduation.

Another important aid to diagnosis is the fact that in lead palsy the supinator longus invariably escapes, whereas in musculo-spiral paralysis from cold it is paralysed equally with the other muscles. The following case illustrates this point:—]

Case No. 59.—A man with paralysis of one forearm came to a consultation at the Hôtel Dieu, at which I was present. The patient suffered from constipation and colic, had wrist-drop, and could not open his fingers. A diagnosis of lead palsy was made. The occupation of the patient did not favour this diagnosis, but it was supposed that he had been poisoned by drinking fluids. My opinion was asked. Having flexed the elbow, and placed the forearm semiprone, I asked him to maintain this position while I tried to straighten the arm. I found that the supinator longus

(which I have shown to be a flexor-pronator) remained flaccid and inert, although on the opposite side, under similar conditions, it contracted strongly. I said that this was no case of lead palsy, because of the impotence of the supinator longus, which I had never observed in that form of paralysis. Since his paralysis had only lasted a few days, and had not been preceded or accompanied by pain, I attributed it to a sudden paralysis of the musculo-spiral from cold, and expressed an opinion that his arm had been exposed to cold during sleep. I questioned him on this point, and his answers confirmed my diagnosis. In fact he was a dock labourer, and had taken a nap on a barge after his *déjeuner* with his two arms naked. When he awoke his right arm was cold and numb, and he had not since been able to move his wrist or fingers. In support of my diagnosis, I stated that the paralysed muscles should have retained their electric irritability, and this point was also immediately confirmed.

. . . . . .

Prognosis and Treatment.—[Paralysis of the musculo-spiral from cold always gets well, says Duchenne, when treated by local faradisation, although it resists ordinary treatment, such as blisters, strychnine, &c.

Cases are given of this paralysis in which Duchenne found the electric irritability of the muscles normal, although the paralysis was complete, and had lasted for two or three months. A rapid recovery followed local faradisation in each case.

In spite of the number of cases which have yielded to local faradisation in Duchenne's hands, we are not to conclude that they are only to be cured by this means, for he says, "I have, indeed, known many cases which have yielded to the application of a few blisters."]

CHAPTER XI.

PARALYSIS OF THE SEVENTH NERVE.*

Before C. Bell discovered the motor properties of the seventh nerve, facial palsy was regarded as due to a lesion of the opposite side of the brain. Ph. Bérard, a professor of physiology, was the first to give a good description of the paralysis of this nerve at its point of emergence or in its course.

The ordinary course and symptoms of this paralysis are too well known to need description. I shall merely give some of my electro-pathological and therapeutic researches on lesions of this nerve and on rheumatic paralysis from cold, which I have seen and treated more than 200 times.

1. *Paralysis of the Tensor Tarsi* (*muscle of Horner*) *in paralysis of the seventh nerve.* . . . .

There is a little muscle of the face whose function (hitherto, as it seems to me, completely misunderstood) needs to be accurately determined; for its paralysis causes much trouble in the absorption of the tears. I mean the tensor tarsi, or muscle of Horner. The following are the circumstances in which I have been able to study the action and use of this muscle.

I have long remarked that among patients suffering from facial palsy, and whose eyelids are motionless, in some the tears are not absorbed but run down the face, while in others this overflow is scarcely noticeable.

In the latter the internal canthus (*le grand angle*) is rounded and scarcely deformed, while in the former the angle is acute and the puncta are drawn outwards to the extent of 1 or $2^{mm.}$

In these cases the palpebral muscles have lost their electric contractility; but if a small rheophore be placed on the inside of the eyelids internal to the puncta it will be seen, in those cases in which the internal canthus has retained its normal rounded form, that the lachrymal points move inwards to the extent of $1^{mm.}$ or $1{\cdot}5^{mm.}$, and dip into the lachrymal sac; while in those

* From *L'Electrisation Localisée*, 3rd ed., pp. 853—884.

cases in which the internal canthus is deformed no movement of the puncta takes place.

In those cases, then, in which the tears run down the cheek the internal canthus is deformed, and electric excitation causes no internal movement of the puncta, (which are pulled slightly outwards,) the tensor tarsi as well as the rest of the orbicularis is paralysed; while in those cases where the angle of the internal canthus is normal, and the puncta retain their proper motion and position, the tensor tarsi has escaped the paralysis which affects the rest of the orbicularis.

It is shown by the pathological facts and by the electric experiment—1. That the muscle of Horner causes the lachrymal puncta to project and move inwards and dip into the lachrymal sac, whither they conduct the tears (a function which takes place during the winking of the eyes). 2. That the tonic force of this muscle gives to the inner angle of the eye its rounded form. 3. That by its paralysis the inner angle of the eye becomes acute, and the lachrymal puncta being drawn outwards and no longer able to dip into the lachrymal sac, the tears flow over.

As to the supposed squeezing of the lachrymal sac by the tensor tarsi (the only use hitherto assigned to it), I do not believe it to be possible under normal conditions. For this it would be necessary, that the outer attachment of the muscle should be fixed like its inner attachment. Then one could understand how its tension would squeeze the sac. But this is not what is seen when the muscle of Horner is made to contract alone under the circumstances just detailed. It is then observed, on the contrary, that the external attachment of this muscle is very movable, and that all its power is used to move the lachrymal puncta. Indeed, it is hard to see how it can compress the lachrymal sac.

The importance of these physiological and pathological facts from the point of view of diagnosis and treatment will be shown further on.

2. *On the electric irritability of the muscles after lesions of the seventh, at its real and apparent origins.*

I have long established by clinical facts and by localised faradisation that every lesion of the seventh nerve beyond its emergence from the pons destroys or lessens the irritability of the muscles supplied by it; that this change in irritability may show

itself within a week, and often lasts after voluntary power has returned. But these researches had reference merely to lesions of the facial nerve *at its apparent origin* (its emergence from the *fossette sus olivaire*) and in its course. When the first edition of this book appeared in 1855, I was not sure as to the state of irritability in facial palsy following lesions of its deep origin.

A knowledge of the facts brought to light by Stilling, Clarke, Van der Kolk, Vulpian, and Philippeaux, on the minute structure of the medulla has since assisted me to understand the symptoms of that form of paralysis in which the muscles of one side of the face and of the opposite side of the body are affected, and often at the same time one or more of the cranial nerves, such as the third, fifth, or auditory. It is of these paralyses, seemingly symptomatic of multiple lesions, but in reality due to a single lesion of the pons, that I wish to speak.

Before I had become acquainted with the last anatomical and physiological researches on the real origin of the seventh pair, even before the concurrence of palsy of one side of the face and the opposite side of the body had received the name of crossed paralysis (*paralysie alterne*), this form of palsy had come under my notice. In all these cases the irritability of the muscles of the face was lost or lessened, but normal in the muscles of the trunk. I admit that I then thought the co-existence of these two palsies to be a mere coincidence. But since anatomy has clearly established the course of the real root of the seventh across the pons, and its intra-spinal relations to other cranial nerves, and since experimental physiology has shown that certain lesions of one half of the pons cause a facial palsy of the same side, I have been able to define accurately the exact seat of the lesion in these cases of crossed paralysis.

Although among my private patients it has been impossible to obtain post-mortem proof, my cases are none the less interesting from a clinical point of view. *In these latter as well as in the former cases I have always established the fact that there is loss or lessening of the irritability of the facial muscles, although the paralysed muscles of the opposite side of the body enjoy their normal irritability.* . . .

Case No. 60.—In March, 1858, Count P. was sent to me by Professor Bouillaud to be treated for a right facial palsy of some weeks duration, accompanied by neuralgic pains on the same side

dating from the previous December. The pains were increased by pressure on the infra-orbital and mental branches of the fifth. The facial palsy was complete, and faradic *irritability was almost lost in all the facial muscles*, sure sign to me of a lesion at the origin or in the course of the facial nerve. As there was a deviation of the uvula it was evident that the lesion was above the stylomastoid foramen. Knowing that the big root of the fifth traverses the pons (*protubérance*) perpendicularly, and close to the root of the seventh, I thought the pains were caused by a single lesion in the front wall of the fourth ventricle above the crossing of the fibres of the seventh. Occasional attacks of dizziness and deafness and certain troubles in speaking and swallowing, signs of a diseased state of the medulla (*bulbe*) confirmed me in my diagnosis. I told my fears to M. Bouillaud and to the family. These fears were soon realised. In April, 1859, there occurred a paralysis of the right sixth nerve, and the right side of the face completely lost its sensibility, although the neuralgia was more persistent than ever. These signs showed an extension of the lesion. On March 10th, 1860, M. de P. was suddenly seized with paralysis of the left side of the body (the side *opposite* to the other paralyses), which was probably due to an extension of the lesion of the fourth ventricle (softening, hæmorrhage, cyst, tumour), to the pyramidal fibres of the "pons." . . . .

The considerable number of cases of crossed paralysis which Gubler has given in his interesting work are more complete than mine, because they have mostly been proved post-mortem to have been due to lesions of one half of the pons below the level of the crossing of the fibres of the seventh. As in my cases the symptoms were the same as in those of M. Gubler, we may suppose that in the cases of which I have studied the clinical aspect there was an anatomical lesion at the same point of the pons, *i.e.*, below the crossing of the seventh pair.

Finally, I believe I have shown that after a lesion of the fibres of origin of the seventh nerve there is loss or lessening of the irritability of the muscles supplied by that nerve.

Anatomy is unable to follow the seventh pair beyond the crossing of the fibres of origin; but pathology reveals its passage in the part of the pons above the point of crossing, which, according to Vulpian, is $16^{mm.}$ below the upper border. At this

spot the seventh can no longer be considered as a nerve, not only because its material presence escapes all observation, but because it has lost part of its properties. To maintain the contrary would be as much as to say that the spinal nerves still exist as such in the brain because a lesion of one hemisphere destroys the motion of the limbs supplied by those nerves.

In the same way I have shown that a lesion of the spinal motor roots weakens or abolishes the irritability of the muscles which they supply, while a lesion of the pyramidal fibres of the pons (which are the continuation of the spinal motor fibres to the peduncles and hemispheres, but above the crossing in the medulla) leaves the electric irritability unchanged. It is in the same way that a lesion of the pons (*protubérance*) (above the crossing of the fibres of the seventh) causes no alteration in the electric irritability.

Diagnosis assisted by faradisation.—A facial palsy may be evidence of a lesion of the brain, or upper part of the pons (above the crossing of the fibres of the seventh), or of the part of the pons (below the crossing of the fibres), or of the facial nerve at its origin or in its course.

1. If the muscles retain their normal irritability there is a lesion of the hemisphere or upper part of the pons on the opposite side. The palsy in such a case is likely to affect the body on the side of the facial palsy.

Facial palsy of cerebral origin, without being frequent, is not very rare. I gave two cases of it in a former edition, in which a patch of softening was found on the floor of the lateral ventricle of the opposite side.

. . . . . .

How are we to distinguish facial palsy following a lesion of one cerebral hemisphere from that caused by damage to the upper part of the pons, which leaves the electric irritability intact, and affects the face and limbs on the side opposite the lesion?

If it is borne in mind that Vulpian has followed the radicles of the third pair in the upper part of the pons, and that these nerves have been seen *to cross in the middle line below the membrane which forms the floor of the sylvian aqueduct, i.e., above the upper border of the pons,* it will be conceivable that a damage to the upper part of the pons (as well as a lesion of the

cerebral peduncle) can bring together a palsy of the *motor oculi* on the same side as the damage, and a palsy of the face and limbs on the opposite side. [Allusion is made to two cases in which this coincidence was observed.]

2. Faradisation, which enables us to distinguish facial palsies due to a lesion of the nerve from those due to a lesion of the brain, peduncle, or upper part of the pons, does not enable us to say whether (when the palsy is limited to the face) the nerve is damaged at its deep or superficial origin, or in its course within the brain. Nevertheless, if this palsy be complicated by pains in the course of the fifth, one may suspect that the origin of the fifth nerve as well as of the seventh is affected, and if the face lose its sensibility this suspicion is strengthened. (See Case 60.) . . . If there be paralysis of the *sixth* the suspicion becomes stronger still, and there seems to me no longer room for doubt when a palsy of the opposite side of the body is added to the other symptoms. We may then reasonably suppose that a lesion beginning in the fourth ventricle at the level of the origin of the seventh has gradually spread upwards to the pyramidal fibres of the pons. This happened in the case reported.

When the primary lesion affects the front of the upper part of the pons, the crossed palsy ought to declare itself at once; for the damage cannot reach the origin of the seventh without affecting the pyramidal fibres of the pons at the same time. The diagnosis does not then remain doubtful, but caution is necessary, because a crossed palsy may be due to the simple coincidence of a cerebral hæmorrhage and a local damage to the facial nerve. The following case is an example of this:—

Case No. 61.—In 1859 M. M. was struck upon the head and shoulder by the débris of a falling house. A sharp bit of wood had pierced the skin below the right ramus of the lower jaw and had penetrated backwards as far as its angle. In falling he had also put his left shoulder out. Notwithstanding a considerable loss of blood, he managed to get home, and although stupefied by the blow, did not lose consciousness. When the wound was dressed a *right* facial palsy was noticed, and the next day a palsy of the *left* arm and leg was observed. In 1860 M. M. came to Paris, and I saw him in consultation with Dr. Leperre. There was complete right facial palsy, causing distortion of the features and preventing the closing of the right eye, but the azygos

uvulæ (*palato staphylin*) and the other palatal muscles acted perfectly. The facial muscles had lost their faradic irritability, but this was retained in the paralysed muscles of the limbs.

In this case the electric examination showed that the palsy of the facial muscles was due to an injury of their nerve, while the palsy of the limbs was due to damage of the opposite half of the brain.

Cause and Nature.—It is quite as important to know the cause and nature of the lesion as its exact anatomical seat. Thus, is a facial palsy due to organic damage or to cold?

. . . . . .

I shall merely touch upon the diagnosis of the so-called rheumatic paralysis, especially that caused by a draught, because it is very frequent.

I have not seen a single case of facial palsy from this cause in which the faradic irritability was not lost or lessened. Electricity, then, does not assist the diagnosis of rheumatic palsies from those which are due to a damage to the nerve trunk. The history of the case must decide the diagnosis.

. . . . . .

Case No. 62.—Some years ago I was called in consultation by my friend and master, M. Louis, to a family in which two sisters had been seized with facial palsy during sleep, the palsy affecting the same side in each, a year having intervened between the cases. In both cases the electric irritability was lessened. The palsy was due therefore to a lesion of the seventh nerve, but what was the cause of it? The young ladies stated that they had not been exposed to chill or draught. I was beginning to fear that the trouble was due to damage to the source or course of the seventh nerve, when, on visiting their bedroom I discovered the cause. A draught from a door which was always open, in passing between the wall and the bed-curtains, exactly struck one side of the face during sleep, and it was this side which was affected in each case. This room was first used by the elder, who was the first to suffer, but after her marriage the younger sister occupied the room, and suffered in the same way.

Prognosis.—[It is needless to say that the general prognosis is necessarily grave if the lesion be central, but not necessarily so if it be peripheral. The local prognosis depends on the amount and nature of the damage to the nerve, which must

be judged of by the degree to which the electric irritability is altered.]

. . . . . .

. . . . . .

As regards rheumatic paralysis of the seventh, I recognise two degrees. In the first, there is a slight lessening of the faradic irritability; in the second, the irritability is more gravely affected or lost. I long ago showed that the prognosis depends upon the degree to which the electric irritability is lost, and this sign has never yet deceived me.

My cases show—(1.) That some palsies, apparently severe, get rapidly well, irrespective of treatment, whilst others, just like them, resist all treatment, or remain incurable. (2.) That the electric test enables one to distinguish between these different degrees of facial palsy.

Contracture of the Muscles in Facial Palsy.

. . . . . .

I had hardly begun my electro-pathological studies when I noticed that certain patients suffering from facial palsy due to nerve-lesion were affected during or after their treatment with various deformities of the features, which evidently depended on contracture of a variable number of muscles. I noticed muscles which, after recovering their tone, and that often quickly, at first re-established the regularity of the features and then gradually accentuated them unnaturally. Thus the zygomaticus minor defined and hollowed the naso-labial line, giving a chagrined look; or the zygomaticus major raised the angle of the mouth and gave a look of gaiety, or the depressor labii inferioris (*carré des lèvres*) pulled down and everted the lower lip, or the orbicularis palpebrarum lessened the opening of the eyes, or the face looked as if shrivelled with cold owing to the contraction of all its muscles. This occurred in patients whose features had been effaced for one or more months from the absence of tone in the paralysed muscles; but in all these cases the deformity and contracture of the face had been so slight that it might have escaped notice, or have been attributed to that slight want of symmetry often observable in the features. I have known some observers who doubted that these appearances were really due to muscular contraction.

Examples given in former editions enable me to conclude that

partial or general contraction of the muscles is a tolerably frequent ending of facial palsy *from cold* (*a frigore*).

The Signs which give warning of Contraction.

Principiis obsta, resist the beginnings, is certainly a maxim applicable to facial contracture; for, to a certain degree, it is incurable, and may be made worse by treatment. To prevent it, it must be discovered early, which is not always easy. One must search for the signs of oncoming contracture among some of the following symptoms.

A. *Spasm occurring in a muscle under the influence of artificial stimulation, such as faradisation with rapid intermissions, or which is provoked by friction of the mucous membrane of the mouth, is a warning sign of contracture.*

. . . . . .

B. *The quick return of tone in a muscle which is palsied and without electric irritability is usually a warning of its approaching contracture.*

. . . . . .

I have elsewhere said that muscles whose electric contractility was abolished recovered their tone before their voluntary power. This first stage of return of tone usually takes place in a certain order. Two or three weeks is usually necessary before the first sign of tonic force appears. The buccinator usually is the first to show a tendency towards the recovery of its tone, and after it come in the following order:—

Zygomaticus major.
Zygomaticus minor.
Levator labii superioris alæque nasi.
Attollens auriculam? (*le pinnal radié*).
Depressor labii inferioris (le carré, quadratus menti).
Depressor anguli oris (triangulaire des lèvres, triangularis menti).
Levator labii inferioris (le releveur de la houppe du menton, levator menti).
Orbicularis oris (orbiculaire des lèvres).
Orbicularis palpebrarum (orbiculaire des paupières).
Frontalis (anterior belly of occipito-frontalis, le frontal).
Corrugator supercilii (le sourcilier).
Compressor naris (triangulaire du nez).
Dilatator naris (dilatateur de l'aile du nez).

These details have a real importance, for, as I have said, if one of the muscles recovers its tone too rapidly (within a week), and, as it were, out of its turn, this, so far from being a good sign, is in reality a warning of approaching contracture, which will not be slow in becoming marked, and in steadily getting worse.

This unusually rapid return of tone in a muscle paralysed to the second degree is a probable sign of approaching contracture, and a valuable indication of it in the absence of spasm.

Some patients have suffered from neuralgia of the fifth, and others from tenderness of the muscles, but these two symptoms have no diagnostic value.

[The ease with which a diagnosis of contracture can be made depends upon the muscle first attacked. In muscles much used for expression it is easily seen, but if such muscles as the levator menti be attacked first its detection is difficult.]

Together with or soon after the occurrence of contracture voluntary power returns.

I have only seen one case of "rheumatic" facial palsy in which there was contracture, notwithstanding that the paralysis was still absolute after a lapse of four years.

Shortening of the contracted muscles will sometimes take place, and then to the deformity and distortion is added a considerable difficulty of movement.

Case No. 63.—In 1851 a colleague showed me a patient who had suffered from left facial palsy (from cold) two years previously. Movement had completely returned. His cheek, at first slack and puffy, had become stiff and had steadily flattened. This stiffness caused a constant difficulty in all facial movements, and the separation of the jaws was limited by it. On putting the finger into the mouth I felt a sort of hard tight cord evidently formed by the contracted buccinator, and projecting inward to such an extent that the patient often bit it, to his great annoyance, during mastication. I have reported a similar case of contraction of the buccinator in which there was also so great a contraction of the orbicularis palpebrarum that the cornea was almost invisible.

I need not describe the signs of shortening of each of the muscles of the face, because they are merely those of contracture

in an extreme degree. They differ only in this, that they do not yield to attempts to stretch them.

. . . . . .

Treatment by Localised Faradisation.—[There are many slight cases which get rapidly well spontaneously. In those cases even in which faradic irritability is abolished Duchenne recommends the application of the faradic current. Tonic power returns before, and generally heralds the return of voluntary power.

When the faradic irritability of the muscles is completely lost it is not generally advisable to begin the electric treatment too early. The inflammation of the sheath of the nerve should first be combated by leeches or blisters behind the ears, and it is only after the inflammation has ceased, "when the central nerve force can reach the muscles," that electric treatment is indicated. It is recommended that the current be applied directly to the muscles and not to the nerve trunk, and for this reason, that "sometimes the muscles are partially and irregularly paralysed, or sometimes a palsy which was uniform at first becomes modified in the course of treatment," and then it is necessary to direct one's attention especially to those muscles which seem most gravely affected and are slowest to recover. If this be not done deformity of the features is very likely to occur.

Among the muscles which resist treatment and which remain longest paralysed are the anterior belly of the occipito-frontalis, the depressor labii inferioris, the platysma (*le peaucier*), the orbicularis palpebrarum, and the tensor tarsi (*muscle de Horner*). It is necessary to localise the current in those muscles which most resist treatment.

When signs of impending contracture occur the intervals between the intermissions of the faradic current should be lengthened as much as possible.

The question whether, when contracture has occurred, the treatment should cease, is answered in the negative, but the advice is given to treat contracted muscles with a current of very slow intermissions, and to take care that it be not applied too often or for too long at a time.]

When in the course of the disease contracture occurs in a paralysed muscle, I never neglect trying to overcome it by

stretching, while at the same time it is being faradised at long intervals with a current of slow intermission. To effect the stretching the patients sometimes pull upon their lips, cheeks, or eyelids in the direction of the muscular fibres, at other times they have placed a big ball in the mouth in order to distend the cheek as much as possible, and thus put the contracted muscles on the stretch. This must be practised continuously and for many hours each day.

Sometimes the chief annoyance of contracture is in the striking want of symmetry between the two sides of the face. In a case where I had failed to prevent the contracture of the zygomaticus minor, which gave the patient a chagrined look, I successfully restored the symmetry of the features by bringing about (by means of rapidly intermittent faradism) a similar contracture of the corresponding muscle on the healthy side.

Treatment by the Continuous Current.*—There is no paralytic trouble which is more easily cured by localised faradisation than facial palsy *a frigore*. It is a therapeutic fact established by numerous investigations made by me during the past twenty years. I admit that it is not without surprise that I see pathologists and electropathists, whose experience is limited or who have never tested the therapeutic value of localised faradisation, dispute the accuracy of my assertion, and assert that rheumatic facial palsy is cured more easily by continuous currents than by faradisation.

If faradisation has failed in their hands it is because they have neglected the rules I have laid down. Maybe they have made the facial distortion worse, for, I must repeat, faradisation ill applied may cause contracture of the muscles.

I have no wish to deny that galvanism may cure a rheumatic facial palsy. I believe the results which "they" have got by this method. But what is the real value of their cures? Everybody knows that a good number of these palsies get well with ordinary treatment (blisters, &c.), or even of themselves. In judging of the value of faradisation, I have taken the trouble to tabulate the cases according to the gravity of the prognosis as

* [The following passages are of interest as showing how completely Duchenne was wedded to one method of electrical treatment. It is not necessary to say that many of the opinions expressed are in direct opposition to those which are now generally held.—Ed.]

established by the state of the electric irritability of the muscles. Now this was not done by the observers I am criticising, for probably they did not know how to do it. However, I have been at this trouble in my investigations on the comparative value of faradisation and continuous currents, and I affirm that the continuous currents were so useless in the treatment of this affection, that I had to fall back upon localised faradisation.

It is well known now-a-days that the continuous current is able, in some degree, to awaken the irritability of paralysed muscles which do not respond to faradism. This is a fact which I have seen many times and established long ago. (It is due no doubt to the action of the continuous current on the local circulation; but this is not the place to discuss the point.)

This power of continuous currents to increase the electric irritability of paralysed muscles—a power which they only exercise for a time, and by no means invariably—does not one whit increase their power of curing a palsy. This at least follows from my already numerous experiments, not only in rheumatic facial palsy, but also in other kinds of palsy in which the electric irritability is, in like manner, changed.

Even if we admit that continuous currents are as useful in treatment as localised faradisation, the inconvenience and danger of applying them to the face should cause them to be rejected for the treatment of facial palsy. If it is borne in mind that, in a case of facial palsy in which I tried the effect of a galvanic current, I caused a blindness instantly, the danger of the powerful action of the current on the retina will be understood.*

An accident similar to this happened to Magendie himself.

Case No. 64.—One of Magendie's near relatives suffering from facial palsy was treated by him with galvano-puncture (Cruikshank's battery was then unknown). A few years later this patient was sent to me by Professor Ph. Bérard, when I found the sight much weakened on the paralysed side, and I learnt that this mishap occurred when the galvano-puncture was being used, and that the flashes of light caused by it were so strong as to tire the eyes and compel the treatment to be suspended. The palsy in this case remained incurable.

Most of the patients in whom I have applied galvanic currents

* Duchenne does not state the strength of current used upon this unfortunate occasion.

to the face have complained of dizziness, and once the patient fell down in a faint.

A final objection to continuous currents is that they cause a general excitement, and seem to me to provoke those contractures which are the usual ending of facial palsy. . . .

PARALYSIS OF THE OCULAR MUSCLES.

[In this chapter Duchenne reminds the reader that these paralyses are not unfrequently among the early symptoms of central troubles, such as locomotor ataxy, and that many of the reported cures of strabismus have been due more to the natural course of events than to the efforts of the oculist. He claims, however, to have cured some cases of strabismus by localised faradisation, which he applied in the following manner :—

A slender metallic stem (5 or 6 centimetres long and half a millimetre in diameter), insulated within a centimetre of its ends (of which one is olive-shaped for introducing beneath the eyelids, while the other has a screw terminal for making connection), is used for the purpose. This rheophore is placed on the conjunctiva over the muscles to be stimulated, care being taken that it does not touch the free edge of either eyelid, which if necessary must be held off by pinching up the skin. The commissures of the eyelids are very sensitive, and must also be avoided. The current used must be feeble, and it is recommended that the current of the primary coil be employed, and that the intermissions be half a second apart.]

In cases of dilatation of the pupil, its contraction may be brought about by faradisation with two olive-headed rheophores placed just outside the free edge of the cornea, and on a level with the ends of the imaginary equator of the pupil. It is a painful proceeding. This manœuvre will cause the contraction of the pupil in persons just dead.

CHAPTER XII.

PALSIES OF THE HAND.*

1. *Palsy of the extensors of the fingers* is seen after injuries to the musculo-spiral (radial) nerve, or one of its branches, or in lead palsy, or as a result of rheumatism of the musculo-spiral nerve. All or part only of the muscles supplied by the musculo-spiral may be affected. In some rare cases progressive muscular atrophy may begin in the extensors of the fingers. From whatever cause arising the functional troubles are the same.

The extensors of the fingers do not, as has been taught, straighten all three phalanges. They act only on the first (near) phalanges, and might well be called the *extensors of the near phalanges*. Although the tendons end on the backs of the near ends of the middle phalanges, electro-muscular experiment proves that they act only feebly on the middle phalanges, while they extend the near ones powerfully. This action of the extensors of the fingers is limited to the near phalanges by reason of the little aponeurotic fibres which leave the front of the tendon, and are attached to the metacarpo-phalangeal joint and the near end of the back of the first phalanx.

This limitation of the action of the extensors is necessary, because in using the hand there is constant need of extending the near and flexing the middle and far phalanges at the same time, as in making down strokes in writing. These movements are caused by the simultaneous contraction of the extensors and the superficial and deep flexors of the fingers, and would have been impossible had the extensors acted upon all three phalanges. The extensors of the fingers cannot act on the phalanges without at the same time extending the wrist. To avoid this inconvenience the flexors of the hand (*palmaires*) contract at the same time and in proportion to the power exerted by the extensors of the fingers. Hence it follows that the hand

* From *L'Electrisation Localisée*, 3rd ed., pp. 964—982.

is held steady between these antagonising forces. This contraction of the flexors of the hand is instinctive, so that we are powerless to prevent it whenever we wish to straighten the fingers.

Palsy of the extensor communis digitorum merely abolishes the extension of the near phalanges. When in such a case the wrist is kept raised (*relevé*), and the patient is asked to open the hand, one sees that he cannot raise the near phalanges on the metacarpal bones. But if the near phalanges be kept extended, the patient easily extends the two last phalanges. This is shown by electro-muscular exploration to be brought about by the action of the interossei and lumbricales. I shall return to this subject. Every attempt to straighten the fingers, as in opening the hand, causes in these patients a flexion of the wrist proportioned to the force of the effort. The reason of this is found in the mechanism of the function which I have just explained. The instinctive brain stimulus by which it is accomplished arrives at once at the extensors and flexors of the hand. And as the former are palsied the latter can alone respond, and the hand is flexed without the patient having power to stop it, notwithstanding that the extensors of the wrist are sound.

When the extensor communis digitorum is alone paralysed the index and little fingers can still be extended by their proper extensors, which extend their near phalanges and direct them towards the ulnar side of the hand.

Palsy of the interossei.—I have only noticed this as a result of injury to the ulnar nerve or one of its branches, and in progressive muscular atrophy. . . . The interossei perform three movements at once, viz., 1. Extension of the middle and far phalanges; 2. Flexion of the near phalanges; and 3. Abduction and adduction of the fingers. Modern writers since Bichat only assign to the interossei the movements of abduction and adduction. The ancients, however, recognised the power of the interossei to flex the near and extend the middle and far phalanges; but this action, they considered, was of secondary importance, and was mainly performed by the extensors and flexors situated in the forearm. To them also, as to the moderns, the utility of these movements, in opposite directions and peculiar to the interossei, and the troubles and deformities consequent on lesions of these muscles, was completely unknown.

Electro-muscular exploration, controlled by pathological observation, has shown me that the interossei are, physiologically, the sole extensors of the middle and far phalanges, and the sole flexors of the near phalanges. The lumbricales help in these movements, but they have no action on the side movements of the fingers.

The direction of the tendons of the interossei accounts for their acting upon the phalanges in opposite directions. It follows from recent anatomical investigations;

1. That the tendon in the first part of its course (from the metacarpo-phalangeal joint to the near end (*partie supérieure*) of the near phalanx) is directed obliquely from above down and from before back, whence there results a flexion of this phalanx on its metacarpal bone during the contraction of the muscle.

2. That the second part of the tendon joined to the extensor tendon by an aponeurotic expansion, as well as to the lateral bands by which it is continued to its ending on the far phalanx, is placed behind the phalangeal joints, and consequently extends them. The side movements of the fingers are mainly due to the attachment of a phalangeal fasciculus of these muscles to the side of the near end of the near phalanges.

In the hand with interosseal palsy abduction and adduction of the fingers is no longer possible, and the power of bringing the phalanges into the plane of the metacarpal bones is also lost. When a patient affected with this palsy tries to straighten the fingers the near phalanges bend back on the metacarpal bones, and the middle and far phalanges are flexed, so that the hand takes the clawed form. This deformity, which is evident with the hand at rest, ultimately increases. The near phalanges get dislocated backwards on the metacarpal bones and the middle and far phalanges forwards. The mechanism of this clawed hand is as follows. When the hand is opened and the fingers straightened, the so-called extensors of the fingers (*i.e.*, of the near phalanges), and the interossei (extensors of the middle and far phalanges) act in consort to cause this extension. But these muscles are antagonists as far as their action on the near phalanges is concerned, and each moderates the action of the other to give to the near phalanx a direction parallel with the metacarpal bone. When the interossei fail the unopposed extensor causes an exaggerated extension of the near phalanges,

and the extension of the middle and far phalanges does not only not occur, but they are on the contrary flexed in direct proportion to the degree of extension of the near phalanges. The clawing of the hand when at rest is explained by the upsetting of the balance of power by which the normal position of the phalanges is maintained. Since the interossei (flexors of the near and extensors of the middle and far phalanges) no longer moderate the tonic force of their antagonists, these latter (extensors of the near and flexors of the middle and far phalanges) gradually shorten and bend the near phalanges back upon the metacarpal bones and keep the middle and far phalanges continually flexed, so that at last they are dislocated, and their ligaments are shortened in the direction of their pathological position.

I have seen another case of clawed hand following interosseal palsy, in which the subluxation of the phalanges was still more remarkable.

Case No. 65.—This was the case of a man in whom the interossei, lumbricales and muscles of the two eminences had been paralysed for a year, as the result of a wound on the lower and internal part of the forearm, and gave no signs of life. The middle and far phalanges of all his fingers were constantly flexed at a right angle, and the near ends of the middle phalanges had slipped forwards over the heads of the near phalanges so as to form an irreducible dislocation. When he tried to straighten his fingers the near phalanges, which were always extended, bent back still more over the metacarpal bones. These serious disorders had developed in this patient in spite of the integrity of the extensors and flexors of the fingers.

I have recorded other examples of this palsy in this book, and it is easy to understand how the clawing of the hand is brought about, and how serious are the hindrances to using the hand which it causes. I have said, and I repeat, that when this palsy reaches a maximum, the hand is a claw which is more inconvenient than useful.

Palsy of the superficial and deep flexors.—I have only noticed this as a result of injury to the median or one of its branches, and in progressive muscular atrophy. These muscles are not intended, as has always been taught, to flex all three phalanges. Their real action is merely upon the middle and far phalanges, as is proved by experiment and pathological observation, and

when these muscles are paralysed the near phalanges can nevertheless be strongly flexed. (M. Chassaignac has kindly shown me a patient in whom the tendons of the superficial and deep flexors had been divided at the wrist and had not united. Although he had as a consequence of the injury lost the power of flexing his middle and far phalanges, I found that the patient could strongly flex his near phalanges by means of the interossei, this power of flexion in the index amounting to 15 kilos.) If the interossei, the true flexors of the near phalanges, are wanting, the middle and far phalanges can alone be forcibly flexed. The action of the superficial and deep flexors on the near phalanges is so weak, that without the help of the interossei they cannot counteract the tonic force of the extensors, which ultimately cause an exaggerated extension of these phalanges, and cause them to be half dislocated backwards.

It is easy to see the end which nature has in view in this arrangement. As in the use of the hand there is frequent need of flexion of the middle and far with extension, at the same time, of the near phalanges, it was necessary to limit the action of the superficial and deep flexors to the middle and far phalanges, just as the action of the extensor communis has been limited to the near phalanges, for these opposite movements of the phalanges can only be obtained by the simultaneous contraction of these muscles.

Patients with palsy of the superficial and deep flexors can no longer flex the middle and far on the near phalanges. They nevertheless preserve the power of flexing the near phalanges, but this power of flexion is inseparable from extension of the middle and far phalanges.

If the two end phalanges of a patient whose superficial and deep flexors no longer act be passively flexed, while the near phalanges are kept raised, it will be found, on asking him to shut the hand, that the near phalanges are flexed, while the middle and far are straightened with a force in proportion to his effort to flex the three phalanges. Need it be said that this movement is due to the action of the interossei and lumbricales ?

As a result of a palsy of the superficial and deep flexors extension of the middle and far phalanges becomes exaggerated, so that at length they bend backwards.

I have once seen an atrophy of those bundles of the flexor sub-

limis which supply the middle and ring fingers. The patient could only move the metacarpo-phalangeal joint and the "*phalango-phalangienne*" joint.

Palsy of the flexor profundus is recognised by the inability to flex the far phalanx. The functional troubles thus caused are less grave than those due to palsy of the flexor sublimis, although they cause serious bother in some professions. Thus I have seen wasting of the flexor profundus in a pianist of talent, who consequently could not extract the same amount of sound from his instrument as formerly, notwithstanding that he retained all the pristine nimbleness of his hand and fingers. This was due to the fact that his far phalanges bent back on the middle ones when he went to place them on the keys, by reason of the weakness or inertion of his flexor profundus.

Palsy of the muscles which cause opposition of the thumb.—I have often seen a partial palsy of the muscles of the ball of the thumb following injury of the end branch of the median nerve distributed to these muscles. The palsy is then limited to the short abductor, opponens and outer head of the flexor brevis pollicis, but since all the fibres of the nerve may not be injured, the palsy may be limited to one or other of the muscles.

(I have long noted by means of electrical testing that in injuries of the median all the muscles of the ball of the thumb are palsied or wasted, except the inner head of the short flexor which goes to the inner sesamoid bone. On the other hand, I have noticed that a lesion of the ulnar nerve affects at the same time all the other muscles supplied by it. I have concluded from this that the inner head of the short flexor is supplied by both the ulnar and median. Anatomists, M. Cruveilhier among others, to whom I have communicated this hypothesis, founded on clinical observation, say that they have never been able to find a filament of the ulnar nerve going to this muscular bundle. Pathological anatomy now explains my hypothesis. A pupil of M. Charcot has established the fact by microscopic examination that a fatty degeneration of the inner head of the short flexor followed an injury of the ulnar nerve in a woman who died in M. Charcot's wards. This pathological fact was communicated to the Société de Biologie on the 17th of April, 1872. See also the *Revue Photographique des Hôpitaux* for March and April, 1872.)

Lead palsy sometimes attacks the muscles of the ball of the thumb, which is also the ordinary starting point for progressive muscular atrophy. The last-mentioned disease has given me frequent opportunities of studying the physiological facts which I am about to discuss.

Three muscles help in opposing the thumb, viz., the opponens, outer head of the short flexor, and the abductor (*court abducteur*). The opponens merely acts on the first metacarpal bone. It has the least opposing power of the three even when it places the first metacarpal bone in the extreme of opposition. The end of the thumb, when not acted on by any other muscle, remains outside the index with its palmar surface looking inwards. I call this muscle, which merely assists in the opposition of the thumb, the opposer of the first metacarpal bone.

The outer head of the short flexor acts on the first metacarpal bone like the preceding ; and it further gives a side movement and a rotation on its long axis to the near phalanx, so as to oppose the pulp of the thumb to each of the fingers ; finally, it extends the far on the near phalanx.

From these three simultaneous movements results the real opposition of the thumb, but the muscle producing them has not the power to put the pulp of the thumb against the pulps of the fingers when these are inclined towards it. The pulp of the thumb under these circumstances scarcely touches the near end of the middle phalanx. The name of this muscle is not justified by its action. It should be called the *phalangeal opposer* (opposant phalanginien) of the thumb.

The abductor (*court abducteur*) also causes opposition of the first metacarpal bone and extension of the far phalanx. Its action differs from that of the short flexor by bending the near phalanx more forward so that the thumb can only be opposed to the two first fingers.

But there is one advantage which it alone possesses, viz., that the end of the thumb can reach the ends of those fingers whose extended phalanges are inclined towards it. It is hence the most important of the muscles of the ball of the thumb. The muscle is no abductor, as its name implies ; it should be called the opposer of the far phalanx (*opposant phalangettien*).

In the use of the hand the far phalanx of the thumb needs to be frequently straightened during opposition, so as to oppose its

pulp to that of the two first fingers. This extension cannot be done by the long extensor (extensor secundi internodii) because, as we shall soon see, it antagonises opposition. The following is the arrangement which allows of opposition and extension being effected by the same force. The muscular bundles going to the outer sesamoid bone, and which are fixed to the near end and side of the near phalanx, send an aponeurotic expansion to join the tendon of the extensor secundi internodii, on the near phalanx. It is easy to see that these bundles in contracting extend the far phalanx, while they move the near phalanx sideways and oppose the first metacarpal bone. The different direction of the two bundles running to the external sesamoid bone perfectly explains why one (the abductor) acting from behind forward can oppose the pulp of the thumb to that of the fingers, while by the other acting more from without in, the thumb is successively opposed to the four fingers, but cannot by it be made to reach the far phalanges.

By the power of the abductor the pulp of the thumb is brought in contact with the far phalanges of the flexed fingers; and hence palsy of this muscle causes great trouble in using the hand, (notwithstanding that the thumb can still be opposed to the digits by the short flexor,) because its pulp cannot reach the far phalanx. If the patient wishes to hold anything between the thumb and forefinger he is obliged to keep the near phalanx of the latter extended while he flexes the middle and far phalanges. This position is very tiring, causes clumsiness, and makes many functions of the hand either difficult or impossible.

Although the loss of the outer head of the short flexor prevents the opposition of the thumb to the ring and little fingers, the trouble is not great, because the abductor allows of opposition to the index and middle fingers, which are of the most importance in the use of the hand.

The position of the thumb at rest becomes changed by wasting of the muscles of the ball of the thumb. The thumb obeying the tonic action of the extensor secundi internodii (*long extenseur*), its metacarpal bone is placed in the same plane as the second metacarpal bone, and its pulp, like that of the fingers, looks directly forwards.

The hand of man then loses its characteristic, *i.e.*, the position of the thumb destined to subserve the intelligence, a position

of half opposition during rest, so that by bringing the thumb into opposition with the index and middle fingers, he is always ready to hold a pen, the servant of his thoughts, or any other instrument for displaying his manual dexterity.

This deformity of the human hand recalls what in the ape is the sign of the beast, showing that he is meant to go on all fours when not climbing. In short, the position of the thumb due to the predominance of the tonic power of the extensor ossis metacarpi allows the ape to put his hand flat on the ground without effort or fatigue.

Palsy of the adductors of the thumb occurs after injuries to the ulnar nerve, and in progressive muscular atrophy.

Adduction of the thumb is produced by the muscles inserted into the inner sesamoid bone, viz., the adductor pollicis, and the inner head of the flexor brevis pollicis. At the same time these muscles cause extension of the far phalanx by means of an aponeurotic expansion going from the inner sesamoid bone to the tendon of the extensor secundi internodii.

Finally, they make the first metacarpal bone turn from within out on its long axis. These muscular bundles, which form what should be called the adductor, are antagonists of the muscles attached to the outer sesamoid bone, to the extensor ossis metacarpi, and to the extensor primi internodii.*

I have only once seen a palsy limited to the adductors of the thumb, and the following symptoms were then noted. The metacarpal bone of the thumb could not be made to approach the metacarpal bone of the index, but was constantly separated from it by the tonic action of the antagonists of the paralysed muscles. The main functions of the hand were preserved because the adductors are not very important. The patients could write, for the muscles concerned in this function were healthy, but they experienced fatigue in holding the pen. The power of squeezing objects placed in the hand was feeble.

Palsy of the extensors and long flexor of the thumb may occur as the result of injury, lead palsy, or progressive muscular

* The names of some of the thumb muscles are not identical in French and English. Thus—
Long abducteur du pouce (Sappey)=Extensor ossis metacarpi pollicis (Ellis).
Court extenseur du pouce=Extensor primi internodii pollicis.
Long extenseur du pouce=Extensor secundi internodii pollicis.
Court abducteur du pouce=Abductor pollicis.—(Ed.)

atrophy. I have only once seen an isolated palsy of the long flexor.

The extensor secundi internodii (*long extenseur*) is the only muscle of the thumb which is properly named, for it extends both phalanges and the metacarpal bone, which it causes to approach the second metacarpal bone. It is the only muscle which places the first metacarpal bone on the same plane as the second.

The extensor primi internodii is the only true abductor of the first metacarpal bone ; its extensor action on the first phalanx is weak.

The flexor longus pollicis flexes the far and near phalanges, but especially the former. It has no action on the first metacarpal bone.

The extensor ossis metacarpi (*long abducteur*) bends the first metacarpal bone forward, (?) but it does not cause abduction. (Quant au long abducteur du pouce, il incline le premier métacarpien en avant mais il ne produit pas l'abduction.) . . .

Palsy of the extensor secundi internodii (which I once saw as the result of a bruise of the lower and hinder part of the forearm) causes the thumb to be always "opposed." The impossibility of raising it caused clumsiness and bother in many manipulations, but nevertheless most of the functions of the hand were preserved, for extension of the far phalanx could be brought about during opposition of the thumb by the muscles which produce the latter movement, as I have demonstrated above.

Palsy of the extensor ossis metacarpi causes no great trouble, although the first metacarpal bone is then dragged on by all the muscles which cause opposition and abduction. But if a palsy of the extensor secundi internodii be superadded (as is often the case in lead palsy) the first metacarpal bone is continuously in extreme opposition and adduction, while the two phalanges are flexed on the palm. Hence it happens that the thumb is always thus shut on the palm either when the hand is closed or when the patient wishes to seize an object.

The absence of the *extensor primi internodii* (the only real abductor of the first metacarpal bone) causes difficulty in certain acts, such as making down-strokes in writing or drawing. Clearly, in this movement the first metacarpal bone is abducted while the two phalanges are flexed, or in other words, this, the opposite

of the up-stroke movement, is caused by the *extensor primi internodii* and *flexor longus pollicis*.

When the flexor longus pollicis is palsied, the movements of the far phalanx of the thumb are completely abolished, and the phalangeal joint (*l'articulation phalango-phalangienne*) soon becomes fixed. The use of the hand in such cases is restricted and hindered.

*Palsy of the extensor carpi radialis longior (premier radial externe, extenseur abducteur) extensor carpi ulnaris (cubital postérieur, extenseur adducteur), and extensor carpi radialis brevior (second radial externe, extenseur direct).**

Palsy of these muscles may result from injury to the musculo-spiral (radial) nerve or its branches, from lead palsy, or from rheumatism of the musculo-spiral. The extensor carpi radialis longior causes extension and abduction of the wrist, the extensor carpi ulnaris causes extension and adduction, while the extensor carpi radialis brevior causes direct extension.

The extensor carpi radialis longior is the most important of these extensors of the wrist, because it alone produces that abduction so needful in most uses of the hand, and since it moderates the adduction of the extensor carpi ulnaris, it produces when paralysed a deformity of the wrist, which keeps the hand in a state of exaggerated adduction, and thus hinders or annuls certain of its functions.

When all three extensors of the wrist are palsied the diagnosis is easy, because there is always wrist-drop, even when the extensors of the fingers are sound, for these latter are unable of themselves to produce extension of the wrist with any force. But a partial palsy (such as I have often seen in lead poisoning) might well be misunderstood, because of the power of extending the wrist.

If the extensor carpi radialis longior be alone affected, extension of the wrist cannot be made without adduction, while, if the extensor carpi ulnaris be affected, abduction always goes with extension of the wrist.

Palsy of the extensor carpi radialis brevior does not seem to

* The extensors of the wrist are thus named by Sappey and Ellis:—
Premier radial externe (Sappey)=Extensor carpi radialis longior (Ellis).
Second radial externe=Extensor carpi radialis brevior.
Cubital postérieur=Extensor carpi ulnaris.

interfere with wrist extension, because direct extension is then produced by the acting together of the extensor carpi radialis longior and the extensor carpi ulnaris, and abduction or adduction take place according as one or the other predominates. The palsy is recognised by the fact that the tendon of the extensor carpi radialis brevior does not tighten during extension of the wrist, which movement also lacks a certain degree of force.

The troubles caused by inability to raise the wrist are too well known to need mention; but the troubles caused by partial palsy of the wrist extensors are not so well known.

Palsy of the extensor carpi radialis longior, owing to the hand being in continued adduction, renders it difficult for the hand to minister (*servir*) to the front of the body. It is only needful to try on oneself, by attempting with the wrist adducted, to carry the fingers to the mouth or face, to put on a hat or tie a cravat, and the difficulties of these functions will be evident. . . . I must add that ultimately the adductor muscles get contracted, and the wrist joint becomes deformed.

Palsy of the extensor carpi ulnaris, in which the hand is abducted, causes much less trouble in using the hand.

As for palsy of the extensor carpi radialis brevior, the patients scarcely notice it, although extension of the wrist is weakened. It is only the careful examination of the physician which leads to its discovery.

I have occasionally seen the flexor carpi radialis and the palmaris longus * (*les palmaires*) partially destroyed by progressive muscular atrophy, and the only functional trouble of interest which I noted was the following: when the patients straightened the fingers with the hand in pronation, the wrist was bent backwards in spite of their efforts to prevent it. This was caused by the extensors of the fingers (which are also extensors of the wrist) whose action was not antagonised by the flexor carpi radialis and the palmaris longus.

Palsy of the pronators and supinators may follow injuries, or occur in progressive muscular atrophy.

There are three chief pronators. Two of these, the pronator radii teres and the pronator quadratus, *are independent;* the

* Grand palmaire (Sappey)=Flexor carpi radialis (Ellis).
Petit palmaire ou palmaire grêle=Palmaris longus.
Palmaire cutané=Palmaris brevis.—(Ed.)

third, which causes semi-pronation along with flexion of the elbow, is known as the *supinator longus* (a name which is inaccurate). I may add that all the muscles attached to the *epitrochlea* help in pronation.

Two muscles effect supination: (1) The *supinator brevis*, which is a pure supinator; (2) The biceps, which is at once a semi-supinator and a flexor of the elbow. Pronation and supination can therefore be performed independently of flexion or extension of the elbow, although a single muscle may perform at once either pronation and flexion (supinator longus), or supination and flexion (biceps). I should add that in order to give an independent flexion of the elbow, nature has provided the brachialis anticus.

The muscles concerned in pronation and supination are so numerous that when one is palsied another acts for it. Their partial palsies are not easy to recognise, and sometimes a diagnosis is impossible.

No one could say that the pronator quadratus is palsied as long as the pronator radii teres is sound, and *vice versâ*, because their actions are identical. Possibly one might say that the pronator radii teres was acting because of its enlargement during contraction, but this sign is of doubtful utility.

When both these muscles are palsied pronation can still be performed by the supinator longus when the forearm is flexed at the same time; but as this muscle only half supinates (pronates) the patient is obliged to help the action by turning the humerus with his subscapularis, the elbow being separated from the trunk. This pronation is but feeble.

When the supinator brevis is palsied, supination can still be brought about by the biceps, which flexes the elbow at the same time. The patient tries to supplement this supination by rotating the humerus outwards, by the infra-spinatus, and bringing the elbow close to the trunk.

To be sure that supination is due solely to the supinator brevis, the elbow must be extended and firmly fixed. The same plan must be adopted when testing pronation. If pronation and supination can be done under these conditions, one may be sure that these movements are performed by the muscles specially charged with this function (the pronators and the supinator brevis).

CHAPTER XIII.

PARTIAL PALSIES OF THE FOOT.*

THESE palsies may affect the muscles of the ankle or the toes. It is necessary to remind the reader of certain general principles of the muscular physiology of this region, principles which in great part are due to my clinical and electro-physiological researches, and without which no one can make any advance in the muscular pathology of the foot.

There are six muscles which are specially destined to move the ankle. These are: 1, gastrocnemius and soleus (triceps sural); 2, peroneus longus; 3, tibialis anticus; 4, extensor longus digitorum; 5, tibialis posticus; 6, peroneus brevis.† 1 and 2 extend, 3 and 4 flex, 5 and 6 give lateral movements (independent of flexion and extension) to the ankle.

No muscle causes simple flexion and extension of the foot without at the same time producing abduction, adduction, or movements in or out. Direct extension and flexion can only be caused by combinations of muscles. Thus the gastrocnemius and soleus is the extensor-adductor, and the peroneus longus the extensor-abductor, but combined they cause direct extension.

The tibialis anticus is the flexor-adductor, and the extensor longus digitorum is the flexor-abductor, but combined they cause direct flexion of the foot.

* From *L'Electrisation Localisée*, 3rd ed., pp. 983—1016.

† Triceps de la jambe, or triceps sural (Sappey)=Gastrocnemius and soleus (Ellis). The gastrocnemius is also called *les jumeaux*, the two "heads" being known as *le jumeau interne* and *le jumeau externe*.

Long péronier latéral=Peroneus longus.
Court péronier latéral=Peroneus brevis.
Péronier antérieur=Peroneus tertius.
Jambier antérieur=Tibialis anticus.
Long extenseur commun des orteils=Extensor longus digitorum.
Extenseur propre du gros orteil=Extensor proprius pollicis.
Jambier postérieur=Tibialis posticus.
Le pédieux=Extensor brevis digitorum.—(ED.)

It would be rational to name the muscles according to their use, and this I have elsewhere done, but I now see that by so doing confusion is caused in medical language, and that by changing old terms I am liable to be misunderstood. . . .

Besides movements of the foot on the leg these muscles cause many partial movements. The study of these, though of great practical interest, has been too much neglected. Without a knowledge of these partial movements it is hard or impossible to understand not only the ordinary mechanism of the physiological movements, and the normal position of the foot, but also the mechanism of pathological movements and deformities which follow certain injuries to muscles.

Palsy and Wasting of the Gastrocnemius and Soleus.

The physiological study of these muscles is inseparable from that of the peroneus longus, and I shall therefore deal with them collectively.

The gastrocnemius and soleus forcibly stretch the back part of the foot (*l'arrière-pied*) and the outer half of the fore part (*l'avant-pied*); they have no action on the inner half of the latter. Having caused the maximum extension of the foot at the ankle-joint they rotate it on the leg as a pivot, so that the toe is carried *in* and the heel *out*. At the same time it (the foot) turns on its long axis so that the outer edge is lowered and the inner raised. The sole of the foot, therefore, looks inwards.

The tarsal movements resulting from the action of the gastrocnemius and soleus may be divided as to time—viz., 1, the movement at the tibio-tarsal joint, and 2, the movement at the calcaneo-astragaloid joint. In the first period the calcaneum in stretching makes the astragalus move in its socket, and drags powerfully, in the extension movement which results, on the cuboid and two last metatarsal bones as if they formed a single bone with it (being united to it by the lower calcaneo-cuboid ligament very firmly, so that these bones have only a very limited movement from below up). But as there is no ligament on the sole to prevent, during extension of the heel, the inner part of the foot from rising (*remontés*) if a force, opposed to extension, act upon it, the first metatarsal bone, the internal cuneiform and the scaphoid yield to the least resistance offered

by the ground, in spite of the powerful extension exercised by the gastrocnemius and soleus on the other parts of the foot.

The second period begins when the astragalus has reached its maximum of extension. At this time, the fore-back diameter of the articular surfaces of the calcaneum being oblique from below up and from before back, the least dragging on the tendo achillis makes the calcaneum glide on the astragalus. As this gliding cannot take place from before back under the influence of the gastrocnemius and soleus, because it is opposed by the ligaments joining the calcaneum to the astragalus and scaphoid, the calcaneum moves obliquely on the astragalus, following the oblique direction from behind forwards and from within out of the sub-astragaloid articular facettes. This gliding of the calcaneum on the astragalus causes a double rotatory movement of the calcaneum, on its long axis and on the axis of the leg. It is from this double movement of the calcaneum that results the adduction of the foot and the turning of its dorsum outwards.

The *peroneus longus* firstly depresses the inner edge of the fore-foot and hollows the sole. It holds the first metatarsal bone in its depressed position like a ligament, while the gastrocnemius and soleus forcibly extend the heel and the outer part of the fore-foot. Secondly, it gives to the foot a double rotation, so that it is abducted while the outer edge is raised; and thirdly, the muscle acts feebly as an extensor of the ankle.

The depressing of the inner edge of the foot by the peroneus longus is the result of a succession of little movements of the joints. Thus the first metatarsal bone is depressed on the internal cuneiform, the cuneiform on the scaphoid, and the scaphoid on the astragalus. The head of the first metatarsal bone (the sub-metatarsal prominence or front heel) is thus lowered in the adult, whose sole is normal, about $1\frac{1}{2}$ cm. by the first movement, and 1 cm. by the second. The last movement is less extended.

When the action of the peroneus longus is at a maximum, the head of the first metatarsal bone is on a lower plane than the head of the second. The movement of the inner edge of the fore-foot then taking place obliquely downwards and outwards, the head of the first metatarsal bone executes a sort of opposition movement so as to slightly cover the head of the second.

When the most extreme contraction of the peroneus longus

takes place the three cuneiforms are crowded together at their lower aspect, giving a twist to the fore-foot, which extends to all the metatarsal bones and lessens its width.

The abduction and raising of the outer edge is due to the sliding of the calcaneum on the astragalus in an opposite direction to that which takes place through the action of the gastrocnemius and soleus.

This sliding is favoured by the surface of the calcaneo-astragaloid joint, but it could not occur without the deep triangular fossa which forms the outer end of the interosseous groove on the upper side of the calcaneum. In fact while the astragalus moves on the calcaneum by the action of the peroneus longus the front edge of the hinder articular facet of the astragalus sinks into this triangular fossa, pushing before it the corresponding part of the interosseous ligament.

When the gastrocnemius and soleus cease to act the extension of the foot at the ankle becomes very feeble, and scarcely goes beyond a right angle in spite of the energetic contraction of the *peroneus longus* and *flexor longus digitorum*, which, as I shall show, have a slight action on the tibio-tarsal joint. While the patient is thus trying to stretch his foot no tension can be felt in the tendo achillis.

By reason of palsy and atrophy of the gastrocnemius and soleus the foot undergoes the following deformity. The heel is depressed, while the fore-foot flexes on the hinder part, and thus there results a hollow club-foot (*talus pied creux*). The genesis of this form of club-foot is explained in the chapter on the wasting palsy of childhood (p. 108), to which I must refer the reader, as well as to figs. 19 and 20. I will merely recall the fact that the form varies somewhat according as the muscles acting on the fore-foot are more or less damaged. If they be all sound the front of the foot is flexed *en masse* on the back part, and we get a direct club-foot (*talus pied creux direct*, fig. 19). If the peroneus longus is affected the inner edge of the foot is kept raised by the tibialis anticus, and the long flexor of the toes alone acting, the sole looks inwards and we have a hollow club-foot with inward twist (*talus pied creux tordu en dedans—varus de l'avant-pied*), see fig. 20. Lastly, when, in this same case, the peroneus longus acts alone on the fore-foot the sub-metatarsal prominence is lower than the head of the last

metatarsal bone and the fore-foot looks outwards; this is a hollow club-foot with outward twist. All these club-feet are "valgus" (*tout talus est valgus*). The reason of this is plain, because the gastrocnemius and soleus being an adductor, as I have shown by experiment, and as Delpech had long ago demonstrated clinically, palsy of this muscle necessarily allows the abductors of the hinder foot to predominate.

The movement causing this deformity takes place, as I have shown, at the tibio-tarsal joint, so that when the deformity is at its maximum the astragalus has reached its extreme of flexion. At this time the tibio-tarsal joint no longer acts, but the patient instinctively substitutes for it a calcaneo-astragaloid movement by means of the extensor longus digitorum, which causes flexion and abduction. (I shall show further on that the abduction movement of the calcaneo-astragaloid joint also raises the point of the foot. This abduction flexion is more marked as the deformity is more advanced.)

The deformity of the joints is in proportion to the degree of hollow club-foot (*talus pied creux*). The supplementary flexion of the extensor longus digitorum, repeated incessantly, wears the joints which rub together, *i.e.*, the front edge of the hinder articular facet of the astragalus hollows by friction the three-cornered fossa which forms the outer end of the interosseal groove on the upper side of the calcaneum.

The joint deformities caused by the flexion of the front on the back of the foot are not less important. They chiefly affect the mid-tarsal joint (*médio-tarsienne*). The joints which half slip out, so to say, are worn by the flexion, and the ligaments shorten on the plantar surface, while they are stretched on the back of the foot. These deformities, seen in all forms of "hollow feet," have been perfectly described by M. Bouvier in the feet found in the second and third degrees of equino-varus. To his work (Bouvier, *Leçons cliniques sur les maladies chroniques de l'appareil locomoteur*, 1858) I refer the reader.

Having read the description of the troubles caused by palsy or wasting of the gastrocnemius and soleus in the motion and shape of the foot, one would suppose that mistakes were impossible. Unfortunately this is not the case, for I have seen grave mistakes made at the onset, or in the early days of this paralysis, when the changes in shape and the trouble in walking

are slight. There is merely a slight limp, and if care be not taken to examine the partial movements of the foot on the leg the palsy escapes observation. I have seen the limp caused by this palsy attributed to hip disease! It is only ultimately, and when it is too late, that the local palsy is recognised as the cause of the deformity and trouble of the foot.

Palsy of the peroneus longus has been misunderstood till now, because in order to diagnose and describe it an exact knowledge was necessary of the action of the muscle and the part played by it in standing and walking, and in the shape of the foot. I may say, without exaggeration, that the ignorance on this point was absolute before my electro-physiological researches.

I have seen this palsy in patients whose feet had been too long in very cold water. As the electric irritability was normal in these cases, one might regard them as "rheumatic." Often it has followed a blow on the outside of the leg. Usually, however, it has come on without any cause, other than great fatigue after a long walk, or standing too long, and in patients whose plantar arch was poorly developed. . . .

I have already given the action of this muscle, and need now merely add the following *résumé*. . . . The plantar arch is the support of the lower limb. Its back pillar is formed by the tuberosity of the calcaneum (*talon postérieur*), and the front pillar mainly by the head of the first metatarsal bone (*talon antérieur*). The peroneus longus is the only muscle which keeps the head of the first metatarsal bone and the other bones (internal cuneiform and scaphoid) which help to form the front half of the plantar arch (of which it is the active ligament) firmly depressed.

When the foot is flat on the ground the body rests on the two pillars of the plantar arch, but in standing on the front of the foot, and in leaping the front pillar supports the weight of the body by itself.

The action and functions of the peroneus longus being known, the functional troubles caused by its palsy are easily foreseen. I will describe them.

The first metatarsal bone gradually rises, dragging the internal cuneiform and scaphoid with it, so that the plantar arch lessens by degrees, and gradually disappears, and we get a flat

foot. On looking at the sole it is seen that the front half looks inwards, *i.e.*, the foot is "varus" in front. From this it results that the foot cannot rest flat on a level surface, and that when standing erect the outer edge of the foot only touches the ground. The patient tries in vain to depress the head of his first metatarsal bone, and one sees him instinctively flex his big toe by the energetic contraction of the adductor and short flexor, as he vainly tries to find a point of support for the inner edge of his fore-foot. The sub-metatarsal prominence remains raised, and cannot touch the ground.

After a long walk the patient suffers as follows: first he feels numbness and creepings, then pricking and fatigue in the sole. Then he complains of pain in front of and below the external malleolus, which he usually attributes to a strain (the foot, as he says, having twisted). From this time he finds his foot turn out more and more, and remain so. Then, on examining the patient, one finds a "painful flat valgus foot" (*pied plat valgus douloureux*), and that the peroneus brevis, or extensor longus digitorum, or both, are contracted.

Thus the "*painful flat valgus*" is developed after palsy of the peroneus longus.

The signs which mark the different periods are in perfect accord with electro-physiological facts. The ones, mutually confirm the others.

Thus in the first stage of palsy of the peroneus longus the tonic force of its antagonist, the tibialis anticus, gradually raises the head of the first metatarsal bone, and destroys the plantar arch. The foot then has the form of a "varus" flat-foot in its front half, the outer edge of which can alone be placed on the ground in walking and standing. Hence the numbness, creepings, and fatigue caused by the compression of the outer edge of the sole during long walking or standing.

This faulty point of support is the cause of the accidents which bring about the second stage of valgus flat-foot. The body cannot rest for long on the outer edge of the sole with impunity, and for the following reason. The force which acts from below upwards on the sub-metatarsal prominence . . . . acts on the tibio-tarsal articulation in the direction of flexion; while the same force equally acting from below upon the last metatarsal bone tends mainly to move the calcaneo-astragaloid joint in the

direction of abduction. But do we not know that the large gastrocnemius and soleus muscle, whose attachment to the calcaneum is so advantageous, strongly opposes the flexion of the tibio-tarsal joint, while the tibialis posticus by itself opposes the abduction of the calcaneo-astragaloid joint? It will be seen, therefore, that if the fore-foot be only supported on its outer edge, the weight of the body causes the calcaneo-astragaloid joint to twist in the direction of abduction. It is also obvious that the tibialis posticus has not the power to constantly oppose this movement, as the strong gastrocnemius and soleus constantly opposes the flexion of the tibio-tarsal joint caused by the weight of the body. This is the cause of the pain which, in palsy of the peroneus longus, is situated in front and below the outer malleolus. It is due to the strong and repeated pressure of the articular surfaces of the calcaneum and astragalus, and of the nervous and vascular tissues placed between them, a pressure which is inevitable in the exaggerated abduction of the joint. The irritation caused by these joint strains causes, by reflex action, the contracture of the muscles nearest to the painful spot (the extensor longus digitorum and the peroneus brevis), which then maintain continuously and excessively the valgus flat-foot. Need one add that in time the ligaments fall in with the deformity of the foot, the one set lengthening, while the set opposed to them shorten? Finally, the surfaces of the joint are changed.

The proof of my assertions will be much more complete if I can show by cases that the functional troubles (the pains during standing and walking) and the deformity of the foot (the flat-foot) disappear when the palsy or weakness of the peroneus longus is cured. I have, indeed, obtained these results by local faradisation.

I have followed the various phases of valgus flat-foot often enough to believe that the mode of production just given is as a rule applicable to the accidental and even to the congenital valgus.

This genesis of the valgus flat-foot is no mere theory, but is supported by electro-physiological proofs given in my first memoir in the *Archives Générales de Médecine*, 1856. . . .

The following is a summary of one of the cases of which the details were given in the last edition (Case cxcvii., 1861):—

Case No. 66.—Accidental painful valgus flat-foot. Palsy of the peroneus longus. Contracture of the extensor communis digitorum, and peroneus brevis. Cure of the pains and recovery of the plantar arch by faradisation in spite of the persistence of the deformity, which was only cured later by forcible movements in a direction opposed to the valgus. Disappearance of the abnormal corns.

This case, which is most creditable to treatment by faradisation, shows the truth of the theory which I have just given of the mechanism of flat-foot, and the functional trouble which it causes in standing and walking.

I attributed, in this case, the flat-foot and the difficulty or impossibility of bringing the sub-metatarsal prominence down upon the ground to the palsy or weakness of the peroneus longus, so that the outer edge of the foot was forced upon the ground during walking and standing. I said that in consequence of the body having this wrong point of support the sub-astragaloid joint had been forced into a vicious attitude of abduction between the weight of the body above and the resistance of the ground below, and thus secondarily a valgus had been produced. I held that hence had resulted a painful pressure on certain points of the joints of the calcaneum and astragalus, and that these joint pains had provoked reflex muscular contractures, which had made the valgus worse. Lastly, the corns seemed to me to be caused by the wrong points of support of the foot on the ground. Well, if all these troubles were really caused by palsy of the peroneus longus, the cure of the muscle ought to cure the troubles. This in fact took place.

It was remarked that the happy effect of the gradual development of the force of the peroneus longus was soon seen by the ending of pain, and by the return of the plantar arch, although the valgus was still maintained by old adhesions. The foot being able, thanks to the recovery of the muscle, to rest on the sub-metatarsal prominence during walking and standing, the weight of the body was chiefly borne by the tibio-tarsal joint, and thus the crushing of the tissues between the calcaneo-astragaloid joint no longer took place. A few forcible movements were then alone necessary to break down the contracted bands which maintained the valgus. The valgus never returned, although the patient no longer used any apparatus

and continued to walk. The chief cause of the valgus, the resting on the outer edge of the foot, no longer existed.

And yet the cure of valgus by tenotomy of the retracted muscles and certain manipulations is almost the only mode of treatment which has hitherto occupied the mind of the surgeon. While I acknowledge the usefulness if not the necessity of having recourse to surgical treatment sooner or later, the above case shows that the deformity of the foot is not the chief thing which demands attention. I may add that even when this secondary lesion has been overcome little has in reality been done.

The following case shows the truth of this assertion even better than the last:—

Case No. 67.—*Accidental, painful valgus flat-foot of two years standing. Contraction of the peroneus brevis and extensor longus digitorum. Palsy of the peroneus longus. Cure of the palsy by faradisation, and as a consequence disappearance of the pains and of the flat-foot, which had persisted after tenotomy in spite of the improvement of the valgus.*

Experiment goes to confirm clinical experience as to the genesis of the secondary valgus with which I have been dealing. Neither have I hitherto seen a congenital painful valgus in which the pain caused by walking or standing did not disappear quickly on faradising the peroneus longus. The patients have noted that they could rest better on the sub-metatarsal prominence of the big toe, on which spot moreover the cuticle began to grow thick.

I have no wish to enter hurriedly into the question of treatment, of which the importance as well as the novelty is plain enough. I have merely dealt in this place with the pathological problem which I had to answer, viz., 1. That the peroneus longus is the only muscle which keeps the sub-metatarsal prominence (which I call the front pillar of the plantar arch) firmly depressed. 2. That the failure of this muscle causes flat-foot (whether accidental or congenital) and secondarily valgus. 3. That the flat-foot causes special troubles during walking and standing. 4. That it causes deformities and pains attributable to the wrong point of support on the outside of the foot.

. . . . . .

Palsy of the tibialis anticus and extensor longus digitorum (flexors of the ankle).

The tibialis anticus causes at once the three following movements: 1. Flexion of the ankle; 2. Raising of the inner edge of the foot; and 3. Adduction of the foot.

The flexion is forcible, and takes place at the tibio-tarsal joint.

The raising of the inner edge of the foot is due—1. To a succession of small movements (of the first metatarsal on the internal cuneiform, of the latter on the scaphoid, and of the scaphoid on the astragalus) in a direction opposed to those which are caused by the proper action of the peroneus longus. 2. To the rotation from within out and from below up of the calcaneum on the astragalus. The adduction is due to the gliding of the articular facets of the calcaneum on those of the astragalus, of which the mechanism is the same as that of the movement produced by the gastrocnemius and soleus. But we know that this movement of the calcaneo-astragaloid joint cannot cause the calcaneum to turn on its long axis, without making it at the same time turn on the long axis of the limb, and it is from this double movement that results the adduction of the foot by the action of the tibialis anticus. But the adduction caused by the tibialis anticus is feeble when compared with the force of the flexion at the tibio-tarsal joint which it causes.

The general shape of the foot is changed by the action of the tibialis anticus. The sole looks in and the phalanges, especially that of the big toe, bend (*s'inclinent*) on the metatarsal bones.

The extensor longus digitorum bends the foot on the leg and abducts it. It also extends the toes, but this latter action is far less pronounced than the former.

The flexion of the foot is the product of two articular movements, tibio-astragaloid and calcaneo-astragaloid, which take place together. In the adult, the extent to which the end of the foot is raised by the calcaneo-astragaloid movement averages $4\frac{1}{2}$ centimetres at its outer edge and $1\frac{1}{2}$ centimetres at its inner edge, above the horizontal; the calcaneum is then depressed to the extent of half a centimetre.

The mechanism of the calcaneo-astragaloid movement caused by the action of the extensor longus digitorum is the same as that described *a propos* of the peroneus longus; the joint moves, however, more in the first than in the second case, so that the

trochlear movement (*mouvement de trochlée*), oblique from within out, and from back to front, (which is peculiar to this joint and causes a slight raising of the foot, at the same time that it is abducted,) is much more pronounced.

The extensor longus digitorum acts more powerfully on the calcaneo-astragaloid than on the tibio-tarsal joint.

From the co-operation of the tibialis anticus and extensor longus digitorum results a direct flexion of the foot on the leg, or a flexion with abduction or adduction, or movements of circumduction. But during the instinctive flexion of the foot on the leg which occurs in the second period of walking, the combined muscles act so as to cause slight abduction. The use of this is evident, for the flexion with adduction which results from the moving at once of the tibio-tarsal and calcaneo-astragaloid joint is greater than the direct flexion which is caused solely by the action of the tibio-tarsal joint.

The peroneus tertius is merely the assistant of the extensor longus digitorum. It is often absent, but notwithstanding this, the extensor longus digitorum possesses its normal amount of power.

The extensor longus digitorum also extends the toes. This action is much less pronounced than that which it exerts over the calcaneo-astragaloid and tibio-tarsal joints. On the other hand its usefulness as a flexor-abductor is much greater, since it is the only muscle which can neutralise the flexor adductor (*tibialis anticus*), to cause either direct flexion or flexion with abduction, while the extensor brevis digitorum without its help can extend the toes with force.

In palsy of the tibialis anticus flexion of the foot on the leg can still be brought about by the extensor longus digitorum, and the foot during flexion is abducted and turned on its long axis, so that the outer edge is higher than the inner. Flexion with adduction and direct flexion are no longer possible. It is true that the *extensor proprius pollicis*, which helps the tibialis anticus to adduct the foot during flexion, at first neutralises the abduction of the extensor longus digitorum, but it is unable by itself to antagonise the latter muscle and abduction of the foot is not slow to appear. Normally the extensor proprius pollicis is but a feeble flexor of the foot, but in the absence of the tibialis anticus it acts with much force. We see also in such

cases the near phalanx of the big toe bend back on its metatarsal bone almost to a right angle, and at last this muscle overgrows, and when it contracts its thick tendon forms a prominence at the inner side of the foot, nearly as marked as that of the healthy tibialis anticus.

In spite of the loss of the tibialis anticus patients can easily give to the foot movements of abduction and adduction, or rock it in or out on its long axis, provided they be not called upon to flex it beyond a right angle. They can also hold the foot firmly so as to prevent it twisting in or out. It could not be otherwise, for they still have the peroneus brevis and tibialis posticus, which have the power of producing these movements. How do we explain, then, that when the tibialis anticus is lost the point of the foot is turned more outwards in walking and standing? The following is the explanation. When, in walking, the rear leg quits the ground to swing forward the foot flexes on the leg. But since this flexion, in cases of loss of the tibialis anticus, cannot be effected without abducting the foot, the outer edge being raised more than the inner, it happens that the foot is still in the same position when it is placed on the ground and remains so till it quits it again. This faulty position when the weight of the body rests on the foot causes a certain weakness in walking and a little limping; it is conceivable also that the faulty position tends to increase, and that certain joints may thereby be progressively depressed. The patient with loss of the tibialis anticus can still, by giving attention, flex the foot forcibly if the extensor longus digitorum still act powerfully, and if the extensors of the foot be not contracted. One can feel in such a case, by opposing the flexion of the foot, how powerfully it is made. Why then does the toe knock against the inequalities of the ground in walking? It is because the instinctive nervous stimulation which produces the automatic muscular contractions of walking is normally unequal to producing sufficient flexion of the foot by means of the extensor longus digitorum, *i.e.*, a degree of flexion which the patient can only bring about by a slight effort of the will.

The tibialis anticus is big, and constitutes nearly half the mass of muscle which helps to flex the foot. The tonic force which tends to keep the foot in its mid position and antagonises the extensors is lost when the tibialis anticus is palsied, and a talipes

equinus is the result. (It is known that the continued shortening of the extensors, which takes place in these cases during rest, causes their permanent retraction in the long run.)

Palsy of the extensor longus digitorum causes, during the exercise of function or at rest, the same troubles as palsy of the tibialis anticus, with this difference, that during flexion the lateral and rotatory movements of the foot on its long axis are in an opposite direction. Thus a patient with palsy of the extensor longus digitorum cannot flex the foot without adducting it and turning the sole in. During walking and standing the foot turns so that the outer edge comes to the ground. Voluntary flexion of the foot is forcible in spite of the loss of the extensor longus digitorum, but the instinctive flexion during walking is so feeble that the point of the foot almost always trails on the ground. Finally, the tonic force of the extensors of the foot predominates during rest, so that the foot which in walking and standing looked like a *varus*, ultimately takes the position of a direct *equinus*.

When the extensor longus digitorum is palsied the foot bends more or less at the "medio-tarsal" joint; the fore-foot curves down and in, and one sees on the instep a lump, more or less marked, formed by the heads of the astragalus and calcaneum.

Palsy of the tibialis posticus and peroneus brevis.—The tibialis posticus adducts the foot by acting at once on the calcaneo-astragaloid and "medio-tarsal" joints. The calcaneo-astragaloid movement causes rotation from below up, and from within out of the calcaneum on its long axis, and makes this axis turn on that of the leg.

The adduction caused by this double movement cannot consequently take place without the inner edge of the foot rising higher than the outer edge, which gives a position of varus to the foot. The mechanism of this movement is the same as that due to contraction of the tibialis anticus and gastrocnemius and soleus.

During the transverse movement given by the tibialis posticus to the fore-foot on the after-foot, the scaphoid may be carried inwards till the inner edge of the hinder articular facet is in contact with the inner edge of the corresponding articular facet of the astragalus.

When the foot is in the extreme of flexion or extension the

tibialis posticus pulls it into a position nearly midway between flexion and extension. But this muscle has a very weak action on the tibio-tarsal joint, although it has a more powerful and more extended adduction action than the tibialis anticus and gastrocnemius and soleus.

The muscle may therefore be looked upon as a true adductor of the foot independently of flexion or extension. The movements given to the calcaneo-astragaloid and medio-tarsal joints by the peroneus brevis are in a direction directly opposite.

In short, the tibialis posticus and peroneus brevis being able to act independently of extension or flexion are meant to hold the foot firm while standing, and to prevent it by their joint action from turning in or out. It is only during great efforts that the other muscles, which produce the same lateral movements combined with flexion and extension, are called into action.

I have only twice seen wasting of the peroneus brevis alone. The patients could not abduct the foot without flexing or extending it by contracting the extensor longus digitorum or the peroneus longus. Sometimes they managed by great efforts and by the joint action of these two muscles to turn the foot a little out by keeping it midway between flexion and extension. When standing the foot often turned on its outer edge.

I have never yet seen palsy of the tibialis posticus alone, but arguing from experiment, one may suppose that it would cause functional troubles in a direction "inverse" to those caused by wasting of the peroneus brevis. When these two muscles are palsied together the foot is far less firm when standing, *i.e.*, it turns easily in or out. The functional troubles are much more considerable when *one* than when *both* these muscles are damaged together.

When the peroneus brevis is palsied the foot tends towards varus when at rest, by reason of the predominance of the tibialis posticus. This movement occurs at the calcaneo-astragaloid joint. Ultimately deformity of the medio-tarsal joint is produced, the fore-foot yielding to the action of the tibialis posticus, and the tibialis anticus moves on the after-foot, which is then carried inwards so that the outer edge forms a curve with the convexity outwards.

Palsy of the tibialis posticus is followed by deformity of the

calcaneo-astragaloid joint, the foot taking the position of valgus. The medio-tarsal joint has not appeared to me to be altered.

Palsy of the Muscles of the Toes.—Experiment shows that these muscles act on the phalanges like their fellows in the hand. Thus, as in the hand, the interossei not only move the toes sideways, but they strongly flex the near phalanges while they extend the mid and far phalanges. The adductor and short flexor of the big toe are also flexors of the near and extensors of the far phalanx, but they have no action on the first metatarsal bone, which is depressed, as I have shown, solely by the peroneus longus. The extensors of the toes (extensor longus and extensor brevis digitorum and the extensor proprius pollicis) only act physiologically on the near phalanges, and the flexors of the toes have a true action only on the far phalanges. All these facts are confirmed by clinical observation. The uses of the foot and hand being essentially different, I cannot say that the functional troubles caused by local palsies of the two members are similar; nevertheless the changes in movement and position of the toes are exactly the same. As in the hand, so in the foot we see—1. *After palsy of the extensors of the toes*, continued flexion of the near phalanges without the power of straightening them. 2. *After palsy of the flexors*, a slight bending backwards of the far phalanges without the power of flexing them, every effort at flexing merely causing (by the action of the interossei) the depression of the near phalanges and the extension on them of the far phalanges. 3. *After palsy of the interossei* (and adductor and short flexor of the big toe), the toes take a clawed position, *i.e.*, the near phalanges are extended on the metatarsal bones, while the mid and far are flexed on the near phalanges by the action of their antagonists.

Palsy or wasting of the muscles of the toes usually complicates similar lesions of the muscles of the ankle. I have seen many such cases in infantile paralysis. Thus in one case there was a direct hollow club-foot from wasting of the gastrocnemius and soleus. The interossei were also wasted, and there resulted a claw which increased the hollow of the sole.

As to deformities limited to the big toe, or to one of the other toes (turning in or out, a crossing, a continued flexion or extension), they are, I say, nearly always caused by wasting or palsy of a part or whole of one of the muscles which act upon

them. At least this has always been my experience in those vicious formations and positions of the toes which so hinder walking that the "*conseils de révision*" have made them a cause of exemption from military service.

There is a faulty form of the foot, usually congenital, which is common enough, and of which the genesis has remained unknown till now.

It is a kind of club-foot, which I shall call the hollow clawed foot (*griffe pied creux*), from palsy or wasting of the interossei and adductor and short flexor of the great toe. This kind of foot occurred in a patient of M. Nelaton's admitted to the Clinical Hospital to be treated for pains in the sole. I proved by electrical observation in this case the absence of the interossei, adductor and short flexor of the big toe. It was evident—1. That the near phalanges were extended to the point of dislocation on the metatarsal bones, while the far phalanges were flexed, and thus the "claw" was formed. 2. The plantar arch was much increased.

When the interossei are palsied or wasted, the tonic force of the muscles which straighten the near phalanges and flex the middle and far phalanges being no longer restrained, the claw of the foot just described gradually increases. The near ends of the near phalanges push down the heads of the metatarsal bones with a force proportioned to the extent to which they are dislocated on to the heads of these bones; then the plantar arch greatly increases, and at length the plantar fascia shortens; then certain joints and their ligaments become deformed, as in all hollow feet. The mechanism of this claw is thus precisely the same as in the hand when the heads of the metacarpal bones are driven back by the near phalanges, causing a sort of hollow in the palm.

Clawing of the hand destroys its use. In the foot it is less serious, as it merely makes prolonged walking and standing painful, and for the following reason. Normally, when standing, the support of the fore-foot is shared by the heads of the metatarsal bones and toes, and when in the first period of walking the foot rocks from heel to toe, the toes, and mainly the near phalanges of the first two toes, strongly pulled downwards by their flexors (interossei and short flexor and adductor of big toe), are the last to give the forward push to the trunk. Palsy of the inter-

ossei makes this function impossible; for the foot, in fact, can only rock from the heel to the heads of the metatarsal bones, or rather to the sub-metatarsal knob of the big toe, and the phalanges remain raised on the metatarsal bones, and clawed. It will be readily understood how the parts in the sole corresponding to the heads of the metatarsal bones become painful to pressure, especially after prolonged walking or standing. This was exactly the case with M. Nelaton's patient. This unfortunate man, in whom this deformity was congenital, began to have pain in the sole over the heads of the metatarsal bones, and mainly over the sub-metatarsal knob of the big toe, when he was first apprenticed to a mason, at the age of ten. His pains always began after a long walk, or a hard bit of work which obliged him to stand.

Formerly the pains did not oblige him to stop his work, a little rest making them disappear; but lately they had prevented him from following his occupation for more than a week or a fortnight at a time without having to seek shelter in a hospital for a month or so to be cured of his pains by rest and other means.

The hollow clawed foot was double in this individual, but less marked on the right side. The interossei were merely weakened on this side, for they responded a little to electric excitation, and under the influence of a strong current they were able to straighten the far phalanges and bend the near ones. On the right side also the sole was much less painful than on the left.

I have always noted that the hollow club-foot from interosseal palsy has a tendency towards equino-varus, *i.e.*, that flexion of the ankle is incomplete during walking, and that the tibialis anticus (flexor adductor) predominates over the extensor longus digitorum (flexor abductor), or, in other words, that during flexion of the foot the sole is carried in, while the dorsum looks a little outwards. Nevertheless a careful examination shows that there is really neither equinism nor weakening of the muscle which causes flexion and abduction. This abnormality is readily accounted for if one bears in mind the unfavourable conditions under which, after palsy of the interossei, the extensor longus digitorum (the flexor abductor) is obliged to work. The lower attachment of the muscle is made to a movable point, the near upper side of the far phalanx. The object of this was explained in my memoir on the foot (*Physiologie des*

Mouvements, p. 476), where I showed the utility of a muscle destined to raise the foot being able at the same time to extend the toes (an extension which was moderated by the interossei so as to afford a fixed point for the flexion of the foot by this same muscle).

But when the near phalanges are dislocated on the metatarsal bones, as the result of palsy of the interossei and of the adductor and short flexor of the big toe, the lower attachments of the extensor longus digitorum and of the extensor proprius pollicis become very movable, and when the foot is flexed the near phalanges are bent still more backwards on the metatarsal bones, the heads of which are more depressed, and thus the hollow of the foot is intensified. The action of the extensor longus digitorum as a flexor abductor of the foot is thereby weakened, and hence the predominance of the tibialis anticus and the flexion with varus when the foot is instinctively flexed during the second stage of walking.

In short, the kind of hollow foot just described is caused by excessive and prolonged action of the extensor longus digitorum and extensor proprius pollicis (extensors of the near phalanges) as a result of weakness or palsy of their antagonists, the interrossei, adductor and short flexor of the big toe. It follows that any excessive action of these extensors of the near phalanges from no matter what cause would produce results absolutely identical.

A knowledge of this fact throws light on the genesis of hollow foot which occurs in cases of equinus, when the extensor longus digitorum preserves its voluntary contractility.

I may perhaps be allowed to show the mechanism of the hollow clawed foot in equinus. I have not wished to separate the study of it from that of the hollow clawed foot from palsy of the interossei, &c., because in both cases the mechanism, as I shall show, is the same.

As soon as the equinus begins to oppose the flexion of the foot, the muscles producing that movement act with exaggerated force, proportioned to the degree of equinus. This increased effort is shown by an extreme extension of the near phalanges, to which many of these flexors of the foot (extensor longus digitorum and extensor proprius pollicis) are attached. Thus the near phalanges are bent back, and at length dislocated on the heads of the metatarsal bones, and hence arises a

progressive increase of the plantar arch (proportioned to the degree of equinus) owing to the depression of the heads of the metatarsal bones, especially the first, by the near phalanges of the toes. The attitude of continued extension during rest, which the foot maintains in equinus, helps more strongly still to form this hollow foot by stretching the extensors of the toes, which then drag the near phalanges into a state of continued extension, since the tonic force of their antagonists (the interossei and the adductor and short flexor of the big toe) are no longer sufficient to keep the near phalanges depressed.

The mechanism which I have just detailed is quite correct. It may be proved thus. Ask a patient affected with the highest degree of hollow clawed equinus to flex the foot on the leg. It will be seen, when he makes the necessary effort, that the near phalanges bend still further backwards on the metatarsal bones, and that, the heads of these latter being depressed to the extent of one or two centimetres, the hollow of the plantar arch is thereby increased, so that the tip of the foot is depressed instead of being raised.

CONTRACTURES OF THE FOOT.

Contracture of the peroneus longus, causing a hollow valgus foot (a club-foot not hitherto described).

When first I made the peroneus longus contract by the help of faradism, I saw that there might exist a kind of hollow valgus from pathological contracture of this muscle. . .

Case No. 68.—*Hollow valgus from contracture of the peroneus longus.*—In May, 1857, Eugenie Diekman, æt. 9, suffered from chorea of the left side, of which she was cured by gymnastic exercises in twenty days. Her parents then noticed that her foot turned a little out, but as she suffered no inconvenience they did not attach much importance to it. As, however, the deformity got worse, they consulted M. Bouvier at the Hospital for Sick Children in 1858, who sent her to me, and I then discovered the existence of a hollow valgus from contracture of the peroneus longus.

The following were the main characters :—

1. The plantar arch was much more marked than on the

healthy side, from the depression of the different bones which form the inner edge of the fore-foot. The radius of the curve (*la flèche de sa courbe*) measured 3·1 or 3·2 centimetres. The transverse diameter of the fore-foot had diminished about half a centimetre at the line of the heads of the metatarsal bones, and the fore-foot had become twisted on the hinder foot, so that the folds of skin on the sole were oblique from within out, and from behind forward. The foot was turned over on its inner edge, and the tip turned out. This abduction movement, which took place at the calcaneo-astragaloid joint, had caused the inner malleolus to be prominent. On examining the foot when hanging at rest, the front end of the outer edge was from 3½ to 5 centimetres higher than the sub-metatarsal prominence. This raising was due to the lowering of the head of the first metatarsal bone below the others, and to the rotation of the foot on its inner edge at the sub-astragaloid joint (*sous-astragalienne*). The heel was no longer in the axis of the leg, but had an oblique direction from above down, and from within out. The contracted tendon of the peroneus longus stood out above the outer malleolus, and the want of tension in the peroneus brevis at its attachment to the fifth metatarsal bone enabled one to say that the peroneus longus was alone contracted. This was confirmed by feeling the tendon of the peroneus brevis grow tight on faradising the muscle. Walking, continued standing, and even jumping, caused no pain, which was only produced by trying to bring the foot to its natural position, which movement was resisted by the peroneus longus.

I thought of trying in this case a method of electric treatment which I put in practice many years ago, and which is now common, viz., the faradisation of the muscles antagonistic to the contracted muscle. I was encouraged by the knowledge that M. Debout had cured a similar case by faradising the tibialis anticus. In a few sittings the contracture of the peroneus longus had disappeared, the curve of the plantar arch had lessened, but nevertheless the valgus remained, although it could be felt that the peroneus longus no longer opposed its reduction. Being sure then that the opposition to the reduction was due entirely to old bands, which had doubtless formed during the two years that the foot had been in a wrong position, I broke these down by forcible manipulation, and the cure of the valgus was

quickly completed. The foot was kept in its right position by a simple bandage, and after eight or ten days' rest Eugenie Diekman was quite well.

It is clear from the foregoing case that contracture of the peroneus longus must cause increase of the plantar arch as well as valgus, which, for the matter of that, had already been proved by electro-physiological experiment.

It is remarkable that this valgus, though very marked and of two years duration, had never caused any pain, nor even the slightest trouble in walking. This seems to me to prove that the pains at the front outer part of the sub-astragaloid joint are not caused by the valgus, but rather by the crushing of certain points in the joint, upon which all the weight of the body bears when standing. But this crushing cannot occur in the hollow valgus from contracture of the peroneus longus, for then, the foot resting on its normal points (the heel and the sub-metatarsal prominence), the weight of the body and the resistance of the ground act upon the tibio-tarsal joint.

It must not be thought, however, that this form of club-foot is always painless. M. Bouvier has shown me a case in which the abduction was so great, that the foot, resting chiefly on the inner edge of the scaphoid, had caused the skin to become painful to pressure over that spot, so that walking and standing were prevented. In two other cases I noted rheumatic pains, which seemed to cause the contracture, and which got worse on walking. Certain forms of tarsal arthritis may be linked with this kind of contracture.

For the sake of brevity I shall not give another case of this form of hollow valgus from contracture of the peroneus longus, but the following is a summary of the chief symptoms noticed in all the cases :—(1) Lowering of the sub-metatarsal prominence, and increase of the plantar arch; (2) lessening of the width of the foot across the heads of the metatarsal bones, and a twisting of the fore-foot on the after-foot, causing oblique folds on the sole; (3) valgus movement in the calcaneo-astragaloid joint; (4) the tendon of the peroneus longus standing in relief above the outer malleolus; and (5) contracture of the peroneus brevis and extensor longus digitorum, complicating the contracture of the peroneus longus.

From the facts given in connection with palsies and contractures of the foot, it follows that there are three kinds of hollow foot, viz., 1. The hollow foot from contracture of the peroneus longus. 2. The clawed hollow foot from excessive action of the extensor of the near phalanges, following either palsy of the interossei and the short flexor and abductor of the big toe, or as a result of equinus (contracture of the gastrocnemius and soleus). 3. The sloping hollow foot (*pied creux talus*) from bending of the fore-foot on the after-foot, as a result of palsy or wasting of the gastrocnemius and soleus. All these hollow feet have a certain resemblance, and although their genesis is essentially different, one might at first sight attribute the hollowness of the sole to excessive action of the peroneus longus. Hence the necessity for enumerating the signs by which the hollow foot due to contracture of the peroneus longus is distinguished from the other kinds.

(*a*) In the clawed hollow foot from palsy of the interossei, &c., the mechanism of which has been explained elsewhere, the curve of the plantar arch resembles that caused by contracture of the peroneus longus. It must be so, for the increase of the arch is caused by the first metatarsal bone in both instances. In the clawed foot the head of the first metatarsal bone is pushed down by the near phalanx of the big toe, which is half dislocated upon it, while in the other case the first metatarsal bone is depressed at its joint with the internal cuneiform. The direction of the force which acts on the first metatarsal bone is different in the two cases and enables a diagnosis to be made. Thus in one case it acts directly downwards, and only acts on the first metatarsal bone, the head of which it depresses; while in the other it acts obliquely from within out on this bone, so that by depressing it together with the other bones of the inner edge, it lessens the width of the fore-foot and abducts the foot. These oblique outward displacements distinguish the hollow foot due to contracture of the peroneus longus from the clawed foot resulting from palsy of the interossei, &c. Finally, in the first form clawing of the toes is not seen, and in the second there is no prominence of the tendon of the peroneus longus.

(*b*) No one can confound contracture of the peroneus longus with contracture of the gastrocnemius and soleus. The distinguishing mark of the latter is equinism from resistance of the

tendo achillis to the flexion of the ankle. The signs of peroneal contracture are absent. . . .

(*c*) When an equinus has been cured by tenotomy or mechanical means, the clawed hollow foot still persists, because the tissues surrounding the deformed joint have gradually come to accommodate themselves to the faulty position. This clawed hollow foot is distinguished from the hollow foot of peroneal contracture by the absence of abduction, twisting of the foot, and prominence of the peroneal tendon.

(*d*) When the gastrocnemius and soleus is wasted or palsied the heel gradually falls, and the fore-foot is flexed on the after-foot at the medio-tarsal joint. This foot (*pied creux talus*) is known by the fall of the heel and the difficulty of straightening the foot at the tibio-tarsal joint, or of making the tendo achillis stand out when the foot is forcibly extended.

(*e*) When contracture of the peroneus longus occurs in a patient with congenital flat-foot, the diagnostic signs are a little different and much less evident, nevertheless they will not escape an attentive observer. . . .

[From a case of this kind seen by me in M. Nelaton's wards it follows that contracture of the peroneus longus in an adult affected with congenital flat-foot gradually depresses the first metatarsal bone, which is subluxated at the internal cuneiform; but as the dorsal ligaments of the other joints of the inner edge are shortened they resist the action of the peroneus longus, and the metatarso-cuneiform is the only joint which yields to the contracture of the muscle. This joint becomes painful and inflamed, as well as the skin over it, and the patient very soon is not able to wear a shoe or to rest on the foot. The foot is not so flat as usual, because the sub-metatarsal prominence is a little depressed by the contracture of the peroneus longus, but this is not enough to produce a hollow sole; the first metatarsal bone forms an acute angle with the internal cuneiform. . . .]

(*f*) The diagnosis of contracture of the peroneus longus is easy enough, but nevertheless one might, unless careful, confound it with valgus flat-foot. Patients with this kind of valgus are usually examined either standing or walking. Since in contracture of the peroneus longus the inner edge of the foot seems to touch the ground in all its length (the weight of the body and the resistance of the ground diminishing the arch of the foot),

one might diagnose a valgus flat-foot. If, however, the foot be examined, suspended and at rest, the signs above described are soon recognised, and the flatness of the foot is seen to be only apparent. I have known this mistake made by men of great knowledge, a mistake which would have been avoided had they known the signs of hollow valgus from contracture of the peroneus longus, and had they been upon their guard.

CHAPTER XIV.

LOCAL PALSIES AND SPASMS.*

I PROPOSE in this chapter to study partial palsies, arising from no matter what cause.

The exact knowledge of the individual action and functions of the muscles of the limbs, a knowledge which results from my electro-physiological researches, throws great light on the pathological movements and the numerous and diverse deformities which are seen to follow palsy or wasting of muscles.

I shall show the value of these researches by applying them to the study of the diagnosis of these pathological movements and deformities.

Although the cases of partial paralysis reported by authors have an undoubted value, and have shed some light upon the subject, it cannot be denied that there is still great obscurity as to the differential diagnosis of these affections. Some of them are yet unknown or ill-defined.

Far be it from me to accuse my learned *confrères* of want of ability, for most of them are justly celebrated in the domain of muscular pathology. It is merely that they were in need of that which has enabled me to come to an exact knowledge of these troubles. I mean *electro-muscular exploration*, by means of which a knowledge of muscles can be as surely obtained upon the living as with a scalpel on the dead.

I do not propose to give a complete description of the muscular affections of the limbs, which would take me far beyond just limits. I intend merely to give rapidly the chief diagnostic signs which serve to establish and distinguish them. I shall review most of the palsies and wastings of muscles, and parts of muscles, when these have special functions, which should almost give them the right to be considered as separate

* From *L'Electrisation Localisée*, 3rd ed., pp. 932—949.

muscles. My investigations show that one must not infer the true function of a muscle from the study of its isolated action, since muscular function is always the resultant of the action of more than one muscle; and in the same way the knowledge of the isolated action of a muscle is not sufficient to establish the diagnosis of its palsy or wasting. I need merely give one example of this. The serratus magnus acting by itself raises, as we know, the point of the shoulder by causing the scapula to rotate on its internal angle, and it also carries the scapula bodily forwards. Certainly the palsy or wasting of the muscle could not be described from a knowledge of these facts. It will be seen anon that we must put the deltoid into action by raising the arm in order that the failure of the serratus magnus may become evident. I might take other examples from the muscles of the hand or foot. . . .

Whether the failure of movement be due to the destruction or palsy of a muscle the result is the same. The motor troubles, therefore, serve equally for the diagnoses of wastings or palsies. But the same does not hold good in regard of the deformities caused by changes in contractility. It has been well established that in paralysis without nutrition-change the tonic force of the palsied muscles, although diminished, is still sufficient to keep the limb in its normal position. Here is the proof. A patient had lost movement in all the muscles which move the shoulder on the trunk and the arm on the shoulder, but after six months these muscles were almost as big as their corresponding fellows. And further, these same muscles had not lost their tonic force so that the shoulder was almost in its normal position, the diseased side being merely a little depressed by the weight of the arm. Compare this with cases of wasting of the shoulder muscles, and it will be seen that in these a wasting of the trapezius causes, besides a drooping of the point of the shoulder, a separation of the spinal border of the scapula to the extent of some 10 centimetres, and a tilting (*mouvement de bascule*) of the bone which causes the lower angle to move *up* and *in*, and to project beneath the skin; and that in wasting of the rhomboids the spinal border of the scapula is detached from the thorax. May we not conclude from these considerations, that although it is true that palsy or wasting produce indifferently the same functional troubles during voluntary movements, wasting or the

loss of tonic force causes in addition faulty attitudes of the scapula during muscular repose? . . . .

In the course of my exposition I shall recall those fundamental physiological facts brought to light by my electro-physiological experiments, without which it would not be possible to understand the functional troubles caused by partial palsies of the limbs and the deformities resulting from them.

Atrophy and paralysis of the clavicular part of the trapezius are easily diagnosed. Its contraction is seen during strong breathing efforts and in raising the shoulders.

The clavicular (respiratory) part of the trapezius may lose its power of voluntary movement, while it still contracts during great instinctive breathing efforts.

This proposition is supported by cases of damage to the cervical and brachial plexuses, in which I have seen inability to raise the shoulders voluntarily, although the clavicular part of the trapezius could be seen to act strongly during forced breathing.

On the other hand, I have seen cases in which instinctive (respiratory) action was abolished, although voluntary power remained.

Lastly, after wounds of the neck which have damaged certain nerve fibres, I have seen the clavicular portion of the trapezius fail to act to either voluntary or instinctive (respiratory) efforts.

From this I conclude that, for the complete paralysis of the clavicular part of the trapezius, its own proper nerve, as well as the outer branch of the spinal-accessory, need to be paralysed.

The middle (raising) third of the trapezius (portion élévatrice) is known to be palsied or wasted by the falling of the point of the shoulder during rest.

In order that the parallelism of the spinal border of the scapula shall be abolished, the middle part of the trapezius, and especially the acromial fibres, must be completely destroyed. This tilting is more marked when the lower part of the muscle, which keeps the inner angle of the scapula depressed, is also atrophied. During rest, and with the arms by the side, the shoulder tip is lower on the affected side. The lower angle of the scapula is more *up* and *in* towards the middle line, and its

spinal border is oblique from *within out*, and from *below up*. In this case the scapula is suspended, so to say, by its inner angle (to which the levator anguli is fixed) like a triangle by its apex. Voluntary raising of the shoulder is still possible, but it is weak and limited in extent. The inspiratory part of the muscle contracts with more force, as if to make up for the loss of the middle third, which is wasted, and in this it is helped by the levator anguli scapulæ (*triangulaire de l'omoplate*). When voluntary raising of the shoulder is resisted it is still effected with sufficient force because of the powerful help of the upper third of the pectoralis major.

The position of the scapula in complete wasting of the raising part of the trapezius is something like that seen in contracture of the rhomboid. I shall give the differential diagnosis of these two conditions later on.

The adductor (lower) third of the trapezius has been the first to suffer in almost every case of atrophy of this muscle which I have seen. . . . As a result of this atrophy the scapula is dragged outwards and forwards. To understand this it must be remembered that the function of the wasted fibres is to hold the scapula at its proper distance from the middle line. As a result of this morbid position of the scapula, the back gets rounded transversely, the clavicle and the shoulder tip are more prominent in front, and the front of the chest is hollowed transversely. These irregularities are most easily seen when the wasting exists only on one side. Thus, in a patient suffering from loss of the adductor portion of the right trapezius, the spine (spinal border ?) of the right scapula is 10 centimetres from the middle line, while that of the left scapula is only 6. If an attempt is made to make the scapulæ approach each other the right scapula rises and turns on its external angle, while the left scapula moves naturally.

In a patient who had lost the adductor part of both trapezii, the spinal borders were each 10 centimetres from the middle line. The back was rounded, and the front of the chest seemed hollowed out by reason of the prominence of the clavicles and shoulder-tips. When he tried to make his scapulæ meet behind they were seen to rise and turn by the action of the rhomboids, but nevertheless the scapulæ approached each other owing to the action of an over-developed latissimus dorsi, which has the

power of making the scapula approach the middle line, as I shall show further on.

When muscular wasting attacks both the elevating and adducting parts of the trapezius, the signs of paralysis of these two parts are conjoined. The shoulder seems ready to fall from the trunk, and the weight of the upper limb often causes painful twitchings at the points corresponding to the attachments of the trapezius, so that the patient is obliged to lie down in order to relieve himself from the weight of his arm.

Finally, I ought to say that wasting of the trapezius causes no trouble in any arm-movements, except vertical elevation, which is weakened by the loss of the middle third.

Wasting and palsy of the serratus magnus. — A complex affection, in which many muscles are implicated, has been comprised under or confounded with paralysis of the *serratus magnus*. . . .

In order to clear up the differential diagnosis I shall first give the signs peculiar to paralysis of the serratus magnus, and shall then group round them the symptoms which have been mistaken for it, and those which often complicate it.

The true signs of paralysis of the serratus magnus are brought into relief by giving certain movements to the limb which is affected.

Directly the patient separates the arm from the trunk, and especially when he carries it forward, the scapula executes two chief movements: 1. A rotation on its vertical axis, so that the spinal border separates from the thoracic wall; 2. A tilting movement, so that the lower angle rises and nears the middle line, while the outer angle is depressed.

These wrong movements are directly proportionate to the degree of wasting or palsy. In its most extreme form the scapula projects like a wing from the thorax, and the skin of the back is tucked round the spinal border so as to form a gutter some 4 or 5 centimetres deep, which, when the trapezius and rhomboids are also wasted, may extend between the costal surface of the scapula and the ribs, presenting the appearance of a deep hollow, reaching forward to the arm-pit, and big enough to hold the entire hand.

The depression of the external angle of the scapula is also so great that the arm can scarcely reach the horizontal position, and

in order to raise it higher, the patient instinctively inclines his trunk to the opposite side.

This limitation in the raising of the arm might incline one to think that the deltoid was implicated, were it not for the fact that when the scapula is supported by the hand and its lower angle pushed forward and held against the chest, the patient can raise his arm vertically without difficulty.

These signs are far less evident when the wasting of the serratus magnus is less advanced. The early symptoms are therefore important. If when the arms are held out one scapula is seen to project 2 or 3 centimetres, while its lower angle is less advanced than that of its fellow, we may feel sure that the serratus magnus is beginning to be paralysed, notwithstanding that the arm can be raised vertically.

I have seen this early stage among other cases, in a girl of 14. As soon as she separated the arm from the trunk, the spinal border of one scapula projected from the chest wall; but beyond a certain degree of separation of the arms from the trunk the scapula became applied to the thoracic wall, and during the vertical raising of the arm this bone performed its natural rotation movement. I have noticed also in two other girls, aged 13 and 9, during a slight separation of the arm this same behaviour of the scapula, which tells of commencing wasting or palsy of the serratus magnus.

In all the cases I noticed that the muscle contracted far less forcibly to electricity on the diseased side. The muscular lesion in this stage causes neither suffering nor trouble in moving the upper limb, but merely a deformity which occurs in certain attitudes and in certain movements.

During rest, with the arms by the side, palsy of the serratus causes no deformity, provided the lower two-thirds of the trapezius be not at the same time wasted. We shall see later what is the attitude of the scapula under these circumstances.

In an extreme case of atrophy of the serratus magnus, when the arms were extended, the scapulæ projected like wings from the thoracic wall. The difficulty in raising the arm was well shown on the right side. . . . .

Wasting and palsy of the deltoid and its complications.—The symptoms of wasting and palsy of the deltoid are so sure that I should not trouble to consider them were they not often

masked by complications which render the diagnosis difficult, and which appear to me to have been hitherto neglected or misunderstood.

Wasting or palsy of both the deltoid and serratus magnus is a complication which I have many times seen, but which I should not have recognised without electro-muscular exploration. When the arm cannot be raised (by the deltoid) it is impossible to develop those movements which alone serve to show the lesion of the serratus magnus. Passive movements of the arm are of no use for this purpose, for it is the unopposed action of the deltoid which gives the scapula the attitude which reveals a palsy of the serratus.

Although simultaneous palsy of the deltoid and serratus is not very rare, it is no wonder that it has escaped observation. Electrical exploration clears up the diagnosis. If the serratus be wasted, the placing of the rheophores over the digitations of this muscle fails to produce that forward movement of the scapula which is readily produced on the sound side by similar stimulation.

It must be borne in mind, however, that the serratus may still contract to electricity, but nevertheless be paralysed to the will.

I have therefore sought some other voluntary movement which might show that the serratus is paralysed, and I have noticed that during the forward movement of the shoulder the serratus drags the lower angle of the scapula forwards and outwards, while the pectoralis major drags indirectly upon its outer angle by its action on the humerus. It is evident that if the serratus be paralysed, this voluntary movement will cause a forward movement of the outer angle of the scapula, while its spinal border remains stationary.

In the first patient upon whom I established these facts, the right serratus magnus was paralysed, while the right pectoralis major was sound. When he advanced his shoulder I noticed —1. That the tip of the right shoulder was dragged forward; 2. That the spinal border of the right scapula remained in place, but was raised a little from the chest wall in consequence of the rotation of the bone on its long axis. Its position was in striking contrast to that of the spinal border of the left scapula, which executed its proper movement outwards and forwards and in close contact with the chest wall.

Every one will recognise the importance of this sign of impotence of the serratus, seeing that no raising of the arm is required, and it is quite as certain as the evidence got by raising the arms.

The retraction of the adductor portion of the trapezius might, it is true, hold the spinal border of the scapula towards the middle line in spite of a healthy serratus, but this should cause no confusion, because in such a case the spinal border would be in close contact with the chest wall, and would take a direction opposite to that seen in palsy of the serratus. So at least I judge from causing artificial contraction of this part of the trapezius by means of faradism. . . . .

The following signs may further aid diagnosis. Place a hand on either shoulder and push them in opposite directions, and it will be observed that the scapula on the damaged side offers far less resistance than the other if the serratus be palsied, and the scapula may be felt under these circumstances to kick up and cause its spinal border to project beneath the skin.

Finally, if the deltoid, pectoralis major, and serratus magnus be all three paralysed on the same side, the shoulder remains motionless when the patient tries to push it forward.

Simultaneous palsy of the deltoid and rotators of the humerus.—The external rotators of the humerus are the *infra-spinatus* and the *teres minor*. From a physiological point of view these two muscles are one, and I shall call it the *infra-spinatus*. *Wasting and palsy of the infra-spinatus* is a frequent complication of palsy of the deltoid. The diagnosis is easy, and the troubles which it causes are great, although they have not yet been described. The following is the first case I saw of this combined palsy:—

Case No. 69.—A patient with atrophy of many of the shoulder muscles was sent to me in 1850 by M. Bouvier. At first sight I thought that a lesion of the deltoid was the main cause of his trouble. The deltoid was partially wasted, the middle part being still big enough to allow him to separate the arm from the side to a slight extent. What bothered him most was a difficulty in writing, for he was a schoolmaster. The hand could hold the pen and form the letters, but having written one or two words he was unable to follow the line, and was obliged to stop and drag the paper to the left with his left hand, and

this had to be repeated every one or two words. I at first thought that this trouble was due to the lesion of the deltoid, but having seen a few days later a patient whose deltoid was palsied, and who suffered no difficulty in following the lines of his writing, I recognised that the functional difficulty in my first patient was due to something more than the lesion of the deltoid.

What muscle was it, then, which proved so essential for writing, and the paralysis of which caused the above trouble ?

I have noted that patients with palsy of the deltoid and infra-spinatus suffered in the same way, and that they could not give an outward movement to the forearm when flexed on a table in the writing position. They could scarcely draw a line from 3 to 4 centimetres long. If by means of an induced current I caused the infra-spinatus to contract, the forearm and hand then continued their outward movement. In this way, and by placing a pencil in their hands, I made them trace lines of 28 or 29 centimetres. This experiment shows, therefore, clearly that in the above case it was the infra-spinatus which was palsied with the deltoid.

When making these researches I noted that the privation of the help of the infra-spinatus caused serious trouble in other professional exercises. I will only allude to the use of the needle. When in sewing or embroidering the needle is drawn from within outwards, the deltoid and infra-spinatus contract together, the first for separating the arm from the trunk, the second for rotating outwards the forearm and hand, the forearm radiating on the humerus as an axis. This last movement is the most important, for patients who have merely palsy of the deltoid can sew almost perfectly, while it is far otherwise if the infra-spinatus be alone affected. Then abduction is the only movement of the arm, and in order to compensate for the loss of the infra-spinatus, the abduction is more extended, and there results a fatigue which prevents the use of the needle for long together. I could quote other instances, but those I have given show sufficiently the great difference as regards functional trouble which exists between a simple palsy of the deltoid and one complicated by palsy of the infra-spinatus.

Till now the *sub-scapularis* and *teres major* have alone been considered as internal rotators of the humerus. My researches show that the *supra-spinatus* has also this power to a less extent,

but only when the humerus falls by the side and parallel to the direction of the trunk.

Normally, the humerus, equally acted upon by the tonic power of its rotators, is, during rest, midway between external and internal rotation, so that when one goes to flex the forearm on the arm either the internal or external rotation may be completed. But if the internal rotators be palsied, this balance of power is upset and the humerus is turned outwards to the utmost degree, so that the external rotators can no longer act. This shows how serious is the loss of the internal rotators, for it is really equivalent to a loss of all the rotators.

From what precedes we see that the diagnostic signs of palsy of the internal rotators belong also to palsy of the external rotators. What I have already said of these signs makes it unnecessary to recur to them.

When the external rotators are alone palsied, the hand of the affected side is still of use to the opposite side of the trunk and head. But when the internal rotators are palsied it is otherwise, for neither the upper part of the pectoralis major nor the front part of the deltoid can supply their place, and the uses of the upper limb are much more restricted.

It is no exaggeration, therefore, to say that palsy of the internal rotators is far more serious than palsy of the external. The former is far more rare than the latter.

I have often had occasion to notice atrophy of the external rotators, and this seems to show that progressive muscular atrophy usually attacks the internal rotators after the external, and that in palsies from injury a far deeper and more serious lesion would be required to affect the internal than the external rotators. This is perhaps the explanation. The deltoid can hardly be injured without the teres minor suffering to some extent, since the same nerve (circumflex) supplies the two muscles. Now every one knows how easily and often this nerve is damaged because of its superficial position, and the constant risks of injury which it runs. (This also explains the integrity of the sub-scapularis in infantile obstetrical palsy of the deltoid and infra-spinatus, described p. 211.) The anatomical condition of the nerves supplying the sub-scapularis (the upper, middle, and lower sub-scapular nerves), which are deeply placed, protects this muscle from similar dangers.

It is easy to know whether the external rotators are alone palsied, or whether all the rotators are palsied, notwithstanding that in both cases the functional troubles are nearly the same. When, the forearm being half bent, the affected arm is placed in external rotation, the patient will be able to turn it in if the internal rotators be sound. By a similar experiment we can learn whether or no the external rotators are sound.

CHAPTER XV.

CONTRACTURES.*

Contracture of the rhomboids causes a deformity of the shoulder, which has been attributed by authors to another lesion, viz., palsy of the serratus magnus. . . .

The girl whose case I am about to give was shown to the Société de Médecine of Paris in 1852, before the cure of the deformity from which she suffered. I then established the diagnosis of this deformity by means of comparative electro-physiological experiments made upon this patient and another whose serratus magnus was wasted.

Case No. 70.—Aglaé Prude, of Bagnolet, æt. 13, of good constitution, felt in February, 1849, a pain in the middle of the right side of her neck. This pain was increased by pressure or by inclining the head to the opposite side. There was slight swelling, thought by the local doctor to be inflammatory. As the patient did not suffer it was hoped that the trouble would subside spontaneously. No notice was taken at this time of certain difficulties in the free movement of the head, and up to October, 1850, there was no abnormal attitude of the shoulder. It was not till April, 1852, that this was noticed, when I saw her in consultation with MM. Marjolin and Bouvier.

With the arms by the side, the lower angle of the right scapula was seen to be close to the middle line, nearly on a level with the outer angle, and to project beneath the skin. By using considerable force the lower angle could be pressed into its proper position, but directly the force was removed the scapula sprung back into its faulty attitude. During these movements a crepitation could be heard and felt, which seemed to have its origin between the scapula and thorax. Above the spinal border of the scapula, which was oblique from within out and from below up, a considerable tumour could be seen, caused by the

* From *L'Electrisation Localisée*, 3rd ed., pp. 950—963.

contracted rhomboid standing out in relief. This tumour was connected with another on the right shoulder due to the inner angle of the scapula, which could be plainly felt, and to the prominence of the levator anguli scapulæ, which was seen stretching to the top of the supra-clavicular triangle. Lastly, the head was slightly inclined to the right, and on turning it to the opposite side pain was produced, which Aglaé referred to the tumour in the supra-clavicular space.

It was evident that this pathological attitude was identical with that which I could produce at will by faradising the rhomboids and levator anguli scapulæ, either singly or together. Nothing, not even the muscular prominences, was wanting, and accordingly I did not hesitate to diagnose at first sight a contracture of the rhomboids and levator anguli scapulæ.

I could not agree with the majority of my *confrères* that the position of the scapula was due to palsy of the serratus magnus, and that the contraction of the muscles was due to want of antagonism.

Observation had taught me that the serratus may be completely wasted without any contracture of the rhomboids occurring as a consequence. The proof of this is that in cases of complete wasting of the serratus, the spinal border of the scapula maintains its normal direction during rest in spite of the soundness of the rhomboids. The scapula is merely a little lower and further from the middle line than usual, in consequence of the wasting of the trapezius.

Something more than an unopposed rhomboid is therefore necessary to drag the scapula into this position. A pathological contraction, a continued spasm, a veritable *contracture* is needed. This was the case with our little patient.

As this contracture might possibly be complicated by palsy of the serratus it was necessary to ascertain the state of this muscle. I therefore made her put her two arms in front of her, when the scapula, by the action of the serratus, took its normal position and moved like its fellow.

On comparing contracture of the rhomboid with palsy of the serratus, it will be found that in the first the deformity is seen only when the arm hangs by the side, and disappears when it is raised; while in the second the deformity appears when the arm is separated from the side, and is not seen during repose.

Contracture of the rhomboids combined with contracture of the levator anguli was my diagnosis in this case; and that the contracture was of rheumatic origin appeared to me to be established by the history. The serratus magnus may possibly have been weakened by being elongated through the faulty position of the scapula, but it was not paralysed.

Contracture of the rhomboids and levator anguli does not cause depression of the point of the shoulder.

It was formerly thought that these muscles caused the scapula to turn on an imaginary central axis, so that the outer angle sank while the two other angles rose. My electro-physiological experiments have shown, however, that these muscles cause the scapula to turn round its outer angle, which remains fixed, and that when they contract the bone rises *en masse*. These physiological facts are confirmed by pathological ones. In the case of little Aglaé the point of the shoulder was not depressed.

But why was there not a raising *en masse* of the whole bone in this case? Was it that the muscles had not reached their maximum of contracture, or was it that the serratus magnus, weakened by being continually kept upon the stretch, no longer offered sufficient tonic resistance to cause, by its combined action with the rhomboids and levator anguli scapulæ, the elevation *en masse* of which I have exposed the mechanism? This last explanation appears to me to be nearest the truth.

The following is a *résumé* of the signs of *rhomboidal contracture*. 1. More or less raising of the lower angle of the scapula, which approaches the middle line, while the point of the shoulder is not lowered. 2. The spinal border of the scapula is oblique from below up, and from within out. 3. A swelling internal to the spinal border of the scapula and prolonged into the supra-spinous fossa. 4. Occasional crepitation, which can be felt and heard in this swelling during movements of the scapula or arm. 5. Lastly, the disappearance of the deformity of the scapula and of the swelling when the patient voluntarily raises the arm on the affected side.

Contracture of the levator anguli scapulæ adds to the foregoing symptoms inclination of the head to one side. In many cases I have seen simultaneous contracture of the rhomboids and two upper parts of the trapezius. Then, in addition to

the other symptoms, we get a raising of the shoulder-tip and a turning of the head to the opposite side. . . .

(Immovable) retraction as well as (spasmodic) contracture of the rhomboid may occur, and I have proved that artificial contraction of the rhomboid, and consequently its retraction, does not allow the humerus to reach the horizontal position. In this case the *teres major* restrains the humerus, and the acromion prevents its elevation. If one were then to divide the teres major the humerus could certainly be raised more readily, but the head of the bone would rub against the acromion, and would be pushed by it out of the glenoid cavity.

Finally, electro-physiology shows very clearly that simultaneous contracture of the rhomboids and teres major fixes the arm against the trunk, and pulls it a little back.

Diagnosis.—There is a position of the scapula caused by palsy of the upper part (*portion élévatrice*) of the trapezius which somewhat resembles that caused by contracture of the rhomboids. This resemblance is still stronger if, at the same time, the serratus magnus has lost its tonic power, as may happen in cases of progressive muscular atrophy.

The following sign serves to distinguish two conditions apparently so similar. In palsy of the upper two-thirds of the trapezius, whether it be or be not complicated by palsy of the serratus, *the tip of the shoulder is always lowered, while in contracture of the rhomboids the shoulder-tip maintains its normal level or exceeds it.*

Further, the swelling corresponding to the contracted rhomboids is not present in cases of palsy of the upper part of the trapezius.

The scapula may take a position a little like the above in cases of contracture of the supra-spinatus, or in certain ankyloses of the shoulder joint. In these cases, however, the movements of the humerus are restricted or abolished, and hence confusion of these conditions with contracture of the rhomboids or palsy of the trapezius ought not to occur.

The faulty position of the scapula, seen in commencing scoliosis, may be mistaken for contracture of the rhomboids, especially because in this stage of spinal curvature the spinal processes retain their normal position. Pathological anatomy shows that the bodies of the vertebræ merely undergo a lateral inclination, and it is the rotation of the vertebral column on its

axis which causes a deformity of the thorax, so that on the side of the dorsal convexity it is hollowed behind and flattened in front, while the reverse condition is seen on the side of the dorsal concavity. It is the bulging of the thorax behind which causes the faulty position of the scapula. . . .

Contracture of the trapezius.—(*a*) Middle portion. My electrical experiments alone would give the signs of contracture of this part of the trapezius, even though no cases of it had occurred in medical practice. The following are the signs: raising of the shoulder-tip; spinal border of scapula too near to middle line, and slightly oblique from within out. and from above down; separation of the lower angle from the middle line; middle part of the trapezius felt like a resisting muscular cord; direction of the clavicle too oblique from within out, and from below up.

The unopposed tonic action of this part of the trapezius following wasting of the lower part of the latissimus dorsi gives to the scapula and clavicle the same position as was observed during my experiments.

This, however, is only the first degree of the trouble, for, when the middle part of the trapezius is more contracted, there is added to the other symptoms a raising *en masse* of the scapula.

This form of contracture is well known, and has been described under the inexact name of contracture of the upper part of the trapezius, and under this name contracture of the clavicular part has been included with contracture of the middle part, although in the reported examples none of the signs of the latter affection (signs established by electro-physiological experiment) are mentioned.

(*b*) The *clavicular part* of the trapezius, I have said, bends the head to the same side, and a little back, while the chin is turned slightly to the opposite side.

These signs were observed in a case reported by me in 1852, in which contracture of the clavicular part of the trapezius had been brought about by exposure to a cold draught. The faulty position of the head, which could not be turned from left to right, was evidently due to contracture of the clavicular part of the trapezius, which could be seen and felt as a resisting cord.

The prominence of the sterno-mastoid, so evident on the left side, could be produced on the right, where the muscle was habitually relaxed, by moving the head without rotating it, so

that its vertical axis was to the right of the vertical axis of the body. Then the prominence of the left sterno-mastoid disappeared. If the vertical axis of the head and trunk were then placed in the same plane the sterno-mastoids became equally prominent on the two sides. The prominence of the left sterno-mastoid was therefore evidently due to the displacement *en masse* of the head to the left, which displacement seemed intended to counteract the shifting of the centre of gravity of the head which was bent towards the right side. The signs which distinguish wry neck due to this cause from that due to the sterno-mastoid are as follows: in the trapezial form the head is bent back towards the contracted side; it is rotated to the opposite side; the sterno-mastoid is relaxed, and the clavicular part of the trapezius forms a cord under the skin. In the sterno-mastoid wry neck the head is also rotated to the opposite side, but it is bent laterally and forwards, and the sterno-mastoid stands in relief on the side to which the head inclines. . . .

Contracture of the splenius.—When one splenius is faradised at the point where it is subcutaneous, the head turns to that side and bends back, while the skin on that side forms folds towards the upper attachment of the muscle. If both splenii are faradised the head is bent directly backwards, and the skin is thrown into transverse folds below the nucha. Thanks to a knowledge of these electro-physiological phenomena I was able to diagnose the first case of contracted splenius which I saw, and to establish its diagnosis from other contractures of the same region.

Bending of the head backwards and to the contracted side, and swelling and hardening of the splenius, which can be felt at its upper third, are the signs which I have observed in many cases. It is distinguished from contracture of the clavicular part of the trapezius by the fact that the head turns to the affected side and not to the opposite side, and also by the difference in the muscular prominences. From contracture of the sterno-mastoid it is known by the fact that the head is bent back and not forward, and turned towards the affected side and not from it. The muscular prominences also differ.

Only once have I seen a contraction limited to the splenius, and even then I could not say whether the small deep-seated rotators were affected. Sometimes the levator anguli scapulæ, or

the upper part of the trapezius, or the deltoid, were affected at the same time, and then the signs of these conditions were superadded.

. . . . . .

Contracture of the deltoid.—The deltoid, it is well known, is the chief elevator of the arm. It separates the arm from the trunk either directly outwards by its middle third, or forwards and inwards by its front third, or backwards and inwards by its hinder third. It is not generally known that an isolated contraction of this muscle may cause a faulty position of the scapula.

While the upper limb is separated from the trunk by the contraction of the deltoid it weighs upon the outer angle of the scapula, producing in that bone, (*a*) a tilting movement whereby the acromion is depressed while the lower angle moves up and towards the middle line; and (*b*) a rotation of the bone on an imaginary axis passing through the outer angle, so that the spinal edge is separated from the chest-wall, and a gutter, of varying depth, is formed between the edge and the corresponding part of the thorax. . . . I have seen one case of contracture *en masse* of the deltoid in which the arm was always out from the trunk, and the scapula was in the faulty position just described.

The patient could make the humerus approach the body by means of the latissimus dorsi and pectoralis major, but he could not make it actually touch it, and at such times the tilting of the lower and outer angles of the bone was very marked. When the arm was held forward the scapula took its proper position, the spinal border being in contact with the thorax, and the outer angle was raised up owing to the action of the serratus magnus.

Without doubt contracture is very seldom limited to the deltoid.

Case No. 71.—I lately saw a case with M. Bouvier of combined contracture of the front third of the deltoid and of the sub-scapularis. The arm was carried forwards and outwards, and could not be made to approach the trunk. This was slight in degree, and the position of the scapula was not much affected by it. The spinal border, nevertheless, was separated from the chest wall to the extent of 2 centimetres, so that the skin was tucked under the edge to form a gutter, and the lower angle approached the middle line. Finally, the humerus was strongly

rotated inwards by the contraction of the sub-scapularis. The internal rotation of the humerus increased the abduction of the elbow.

In wasting of the serratus magnus, the scapula, when the arm is raised, takes a position, like that caused by contracture of the deltoid. It could not be otherwise, for in palsy of the serratus the deltoid contracts by itself (and not in concord with the serratus, as is the case normally). The two conditions cannot, however, be confounded, for in palsy of the serratus the deformity of the scapula occurs *only when the arm is raised*, while in contracture of the deltoid it is present with the arm at rest, and lessens or vanishes when it is raised. Further, the soundness of the serratus magnus might be demonstrated by electricity.

CHAPTER XVI.

PALSIES AND CONTRACTURES OF THE MUSCLES WHICH MOVE THE THIGH ON THE PELVIS, AND THE LEG ON THE THIGH.*

[The principal conclusions drawn from my experimental investigations are merely given, and I refer the reader for further information to the *Physiologie des Mouvements*, published in 1868.]

Extensors, abductors, and rotators of the hip (gluteus maximus, medius, and minimus).

A. In the erect position of the lower limb contraction of the gluteus maximus, no matter what part of it is excited, merely extends the hip without abducting it in the least.

B. In the sitting attitude, with the legs resting on the ground, stimulation of the gluteus maximus causes abduction of the hip. This muscle causes slight rotation outwards of the thigh, at the same time as extension, but feebly.

C. The gluteus maximus has nothing to do with defecation or labour.

D. The action of the lower part of the gluteus medius is in two periods: 1. Strong rotation in of the thigh; 2. An oblique movement forwards and outwards. The independence of the first movement is useful.

E. The different bundles of the gluteus medius abduct with greatest force as they approach the middle. They cause the thigh to perform movements of circumduction from before back, and from behind forwards.

Clinical observation shows us—

1. The degree of utility of the gluteus maximus.

2. The part it plays in standing and walking.

3. That it acts vigorously in great efforts, such as leaping, or in walking, or going up-stairs when a burden is carried on the shoulders.

4. That the gluteus medius and gluteus minimus are mainly for preventing the pelvis from leaning to the side opposite to the limb which supports the body in walking or standing.

From *L'Electrisation Localisée*, 3rd ed., pp. 1016—1021.

Flexors of the hip (psoas and iliacus and tensor vaginæ femoris).

A. Experiment confirms the opinion of modern authors as to the action of the psoas and iliacus.

B. It shows their rotatory action (feeble indeed), of which Winslow has expressed a doubt.

C. The tensor vaginæ femoris helps the psoas and iliacus to flex, but antagonises them in rotating the thigh; consequently when acting with those muscles it causes direct flexion of the thigh on the pelvis.

D. This muscle causes neither extension nor abduction of the leg. It is merely meant to help flex the thigh.

E. It is a very feeble abductor.

F. It flexes the thigh on the pelvis obliquely forwards, and a little outwards.

Clinical observation shows, (1) that the tensor vaginæ femoris is mainly intended to act with the psoas and iliacus in order to neutralise the external rotatory action of the latter, and to cause direct flexion of the hip during the second stage of walking; (2) that the tensor vaginæ femoris is a strong flexor of the hip; and (3), that it does not extend it; (4) that the flexors of the hip are absolutely necessary for progression, although a very feeble flexing power is enough to make the lower limb swing forwards in walking; (5) that in walking the lower limb does not merely swing like a pendulum, as the brothers Weber taught, but is brought forwards by muscular action.

Muscles which cause adduction of the thigh.

A. The pectineus flexes enough to place one thigh on the other.*

* Grand fessier (Sappey)=Gluteus maximus (Ellis).
Moyen fessier=Gluteus medius.
Petit fessier=Gluteus minimus.
Le pyramidal=Pyriformis.
Jumeau supérieur=Gemellus superior.
Jumeau inférieur=Gemellus inferior.
Carré crural=Quadratus femoris.
Tenseur du fascia lata=Tensor vaginæ femoris.
Couturier=Sartorius.
Triceps crural=Triceps extensor cruris.
Droit antérieur=Rectus femoris.
Droit interne=Gracilis.
Premier ou moyen adducteur=Adductor longus.
Second ou petit adducteur=Adductor brevis.
Troisième ou grand adducteur=Adductor magnus.—(Ed.)

B. The adductor longus adducts more than it flexes; the adductor brevis acts similarly, and the adductor magnus causes direct adduction.

C. All the adductors are outward rotators, but the lower part of the adductor magnus causes inward rotation, an action which is useful on horseback.

Clinical observation shows—

1. That the lower part of the adductor magnus checks the outward rotation caused by the other adductors during adduction of the thigh.

2. That the front part of the gluteus medius and minimus, which causes inward rotation, does not help to cause adduction.

3. That the tonic force of the lower part of the adductor magnus influences the normal position of the thigh between outward and inward rotation.

4. That the adductors are necessary for the direct forward flexion of the hip.

Extensors of the knee.

A. The exaggerated oblique action of the vastus externus exposes the patella to outward displacement.

B. The vastus externus and vastus internus must be looked upon as a single muscle.

C. The attachment of one of the extensors of the knee to the anterior superior iliac spine is of service.

D. Under certain conditions this pelvic attachment increases the extensor power of the rectus femoris, and makes a sort of active ligament of this muscle.

Clinical observation shows—

1. That the patella may be displaced outwards in walking or jumping when the vastus externus contracts by itself, and it is probable that recorded cases of outward displacement of the patella were caused by the predominating tonic action of this muscle.

2. That the rectus femoris has no part in the swinging forward of the leg in the second stage of walking.

3. That the extensors of the knee are inactive during standing.

4. That the intervention of the triceps is necessary during

extension, when the leg and thigh form a projecting angle at the knee.

5. That the gracilis (*droit interne*) and "external rectus" (*droit externe*) may cause extension of the knee without the help of the patella or its tendon.

6. That the utility of the small articular muscles of Winslow is not apparent.

Flexors of the knee (sartorius, gracilis, semi-tendinosus, biceps femoris, popliteus, semi-membranosus).

A. The sartorius acts upon the femoral fascia and the tissues of the thigh like the tensor vaginæ, but in an opposite direction.

B. This action takes place during the first stage of its contraction.

C. The sartorius does not cause all the movements which place the leg in the tailor's attitude.

D. The external rotation caused by the sartorius is more feeble than is generally supposed.

E. Its direction is not slanting enough to cause strong rotation outwards, an action, by the way, which would do more harm than good.

F. By reason of its reflection from within out the lower tendon of the sartorius rotates the leg inwards and also the thigh.

G. Under some conditions the sartorius acts as a flexor of the hip.

H. The gracilis is more powerful as an adductor of the thigh than as a flexor of the knee; contrary to the assertion of Winslow, it may begin to flex the knee even when the thigh is not rotated.

I. The gracilis rotates the thigh inwards.

J. The rotating action of the popliteus is stronger than its flexing action.

Clinical observation shows—

1. That the sartorius cannot swing the leg completely forward in walking.

2. That the semi-tendinosus, semi-membranosus, and biceps femoris are mainly meant to cause and maintain the extension of the body in walking and standing.

3. That the knee must be bent when the foot leaves the ground to swing forward, and that this is chiefly done by the semi-tendinosus and biceps.

4. That the muscular contraction necessary to bend the knee during the second stage of walking is brief.

What is the proper action of the biceps, and what are the troubles it causes when, as an outward rotator of the leg, it is not checked by antagonists?

5. As a result of continued and unchecked action of the biceps, the leg turns on the thigh during extension.

6. Firmness in standing and walking is impaired by lengthening of the lateral ligaments.

7. The anatomical arrangements of the crucial and lateral ligaments of the knee are useful.

8. A certain balance of power among the rotators of the leg is necessary for the natural form of the knee, and the crooked knee (*genou cagneux*) is mainly due to the predominance of the biceps, which rotates it outwards.

9. Continued exaggerated action of the inward rotators causes troubles like the above, but in an opposite direction.

10. The inward rotators of the leg are a sort of needful active ligaments for checking the outward rotation of the biceps.

11. The semi-tendinosus, semi-membranosus, and biceps femoris are necessary active ligaments for checking the extension of the knee. The happy applications in diagnosis which may be made of these physiological ideas, and the deductions which may be drawn from them in regard to the prognosis and treatment of the palsies of the muscles of the thigh and leg must be readily apparent.

CHAPTER XVII.

PARALYSIS AND CONTRACTURE OF THE DIAPHRAGM.*

BEFORE beginning the pathological study which forms the subject of this article, it seems advisable to recall, in the form of propositions, the fundamental physiological facts derived from my electro-physiological researches on the respiratory movements. These propositions were demonstrated by experiment on man and animals, and by numerous clinical facts. The reader will find these experiments fully given in my work on the Physiology of Movements. The knowledge of the physiological facts which resulted from them will help him to understand the mechanism of the functional troubles and deformities of the trunk seen in paralysis or wasting of the diaphragm. It will serve also to explain the troubles caused by palsy of other respiratory muscles, of which examples are given in this book, and which must be distinguished from those caused by palsy of the diaphragm, with which they may be confounded.

(A) *Diaphragm.* . . . —1. My experiments on men and animals show that the contraction of the diaphragm caused by electrifying the phrenic nerve, isolated or otherwise, from its nervous connections (*communiquant ou non avec l'arbre nerveux*) causes an elevation and outward movement of the diaphragmatic ribs as long as the abdominal walls are intact. The antero-posterior diameter of the base of the thorax is also slightly increased. In some cases the outward movement of the lower ribs is communicated to the upper.

This observation has been confirmed by clinical experience.

2. If the belly be opened and the bowels pulled downwards the contraction of the diaphragm by itself causes an *inward* movement of the diaphragmatic ribs.

3. Although the diaphragm, when the adjacent intestines are removed, becomes an expiratory muscle in respect of the move-

* From *L'Electrisation Localisée*, 3rd ed., pp. 901—921.

ment which it gives to the lower ribs, it does not the less by its descent increase the vertical diameter of the chest.

The lowering of the middle of the diaphragm is very limited, for at the most the diaphragm assumes the form of a truncated cone, and never, as Haller thought, becomes convex on its under side.

4. The concentric movement of the lower part of the chest seen during contraction of the diaphragm when the belly is opened and the bowels pulled down proves, contrary to the opinion of MM. Beau and Maissiat, that the pericardium does not afford a point of resistance which allows the diaphragm to cause an expansion of the ribs to which it is attached. The movements, when the diaphragm descends, of the base of the chest in opposite directions, according as the bowels are pulled down or not, shows, as Magendie thought, that the expansion of the lower ribs during the contraction of the diaphragm is due to the resistance afforded to the muscle by the bowels.

5. The resistance of the bowels would not help the expansion of the diaphragmatic ribs unless they offered a large and convex surface to the diaphragm. This assertion is based on an experiment in which no expansion of the ribs was seen when the descent of the diaphragm of a dead horse was opposed by applying the hand to the centre of the muscle (the contraction of the diaphragm being caused by faradising the phrenic nerves).

6. The knowledge of paralysis and contracture of the diaphragm is due to my electro-physiological experiments.

(B) *Intercostals.*—1. The electro physiological experiments which I have made on man, either by faradising the external intercostals along their entire surface, and the internal intercostals in the space between the cartilages, or by localising the stimulation in an intercostal nerve, show that all these muscles are *inspiratory*—(*a*) by pulling each rib towards the rib above it which remains fixed; (*b*) by giving an excentric movement to the lower of the two ribs by causing them to rotate on their extremities.

2. The inspiratory action of the intercostals shown in the living by means of electricity has been confirmed (*a*) by clinical cases in which the auxiliary muscles of inspiration being wasted as well as the diaphragm, costal respiration was nevertheless ample, and by putting the fingers in the intercostal

spaces the intercostal muscles could be felt to swell and harden during each inspiration; (*b*) by clinical cases in which, as a consequence of wasting of the intercostals, the upper costal (*costo-supérieure*) respiration is abolished, and the capacity of the chest lessened. This is the counter-proof of the preceding facts.

3. Experiment thus confirms the opinion of Winslow, Haller, and others that the intercostals raise the lower rib because the upper is fixed; it is proved by clinical observation that these muscles cannot pull down the upper rib during natural "costo-superior" breathing because these are strongly raised by the sterno-mastoids and scaleni.

4. The fact that the intercostals are lengthened during inspiration is no reason against their assisting in the inspiratory act, as Hamberger pretended; for I have shown that certain muscles when they are called upon for vigorous voluntary action are lengthened by the contraction of other muscles for the purpose of increasing their power.

This has been shown in the case of the flexors of the fingers, which lose their power when, owing to paralysis of the extensors (radial and ulnar) of the wrist, the hand can no longer be extended when the flexors contract. In the same way lengthening of the internal intercostals during inspiration can increase the power of these (inspiratory) muscles (*de même l'élongation des intercostaux internes, pendant l'inspiration, peut augmenter la puissance de ces muscles qui concourent à la produire*).

5. The oblique direction of the intercostals shows that they are meant for inspiration, for it is only during inspiration that they become perpendicular to the levers (ribs) which they move. If they contracted during expiration they would be still more oblique to these levers, which would be contrary to the general rule "that muscles in contracting tend to become perpendicular to the levers which they move, so that their power increases in proportion to the amount of their contraction."

6. The opposite direction of the slant of the inner and outer intercostals shows that they are meant to contract together, for if they acted alone, the first for expiration and the latter for inspiration, the ribs would glide obliquely over each other during each respiratory act.

The simultaneous contraction of all the muscles neutralises

this oblique action, which is converted by composition into a movement perpendicular to the ribs which have to be moved.

7. Contrary to the deductions made from vivisections by Galien, who rendered a pig voiceless by ligaturing all its intercostals, clinical observation shows that these muscles have no great influence on the voice.

8. The utility of the intercostals cannot be compared to that of the diaphragm, for their paralysis does not endanger life. Nevertheless when the diaphragm fails the intercostals are the only muscles which can supply its place, and the patient may continue to live, provided no bronchial or chest affection supervene.

On this account the intercostals ought to be considered as essential muscles of inspiration, but second in importance to the diaphragm.

(C) *Auxiliary Inspiratory Muscles.*—1. The scaleni, sterno-mastoids, clavicular parts of the trapezii, pectorales minores, and subclavians, all help in costo-superior respiration, as may be proved both by eye and touch.

2. These muscles are less important than the intercostals, to which they act merely as auxiliaries, for clinical observation shows that in spite of their absence costo-superior respiration can be effected without appreciable trouble.

3. Their power and use in rib-breathing is, nevertheless, not to be denied, especially when we see one of them (the sterno-mastoid) alone able to sustain sufficient costo-superior respiration to accomplish incomplete aeration of the blood.

These muscles are especially useful in fixing and raising the upper attachments of the intercostal muscles; but the sterno-mastoids are only used when the breathing need is great.

4. The sterno-mastoids can only act as helpers of inspiration when the head is firmly extended. The extensors of the head, therefore, and especially the *splenii*, ought to be ranked amongst the helpers of inspiration.

5. Other muscles help in a secondary way in upper rib-breathing during extraordinary efforts, such as the *serrati magni*, the *rhomboidei*, &c.

(D) *Expiratory Muscles.*—1. The expiratory muscles are extrinsic and intrinsic: the first move the diaphragm and the chest walls, such as the muscles of the belly, the *serratus posticus*

inferior, and the *triangularis sterni;* the second chiefly act on the bronchi, and are the bronchial muscles of Reisseissen.

2. The muscles of the wall of the belly by acting together depress it and tighten it, and thus push the bowels, and consequently the diaphragm, upwards.

3. The help of these muscles is not wanted in ordinary breathing, for the abdominal muscles, the most powerful of them, may be wasted without any appreciable expiratory difficulty resulting. They only contract during forced expiration, as in singing, crying, coughing, &c.

4. My clinical observations establish, on the other hand, that the bronchial muscles (expiratory, intrinsic) are the ordinary essential expiratory muscles; for they show that the column of air which arrives freely in the lungs during normal inspiration is expelled from them in small quantity, and with little force, in spite of the energetic contraction of the extrinsic expiratory muscles, whence arises a constant need of expiration caused by the too long sojourn in the little bronchi, of air which is no longer breathable, and this feebleness in breathing out makes coughing difficult or impossible, so that a simple bronchitis may endanger the patient's life.

5. Experimental physiology has perfectly established the fact that by an electric current the bronchial muscles could be made to squeeze up the membranous bronchi so as to entirely close them; but the trifling power which is assigned to these muscles (one-fifth of the elastic force of the lung) does not seem to me to be proportioned to the important part which they play in the expiratory act as clinically demonstrated.

Paralysis of the Diaphragm.

. . . . . .

There are many kinds of diaphragmatic palsy. I have collected many cases to prove this, and some of them have been given in previous editions.

These cases presented the following types:—

1. Progressive wasting.
2. Lead palsies.
3. Hysterical palsies.
4. Palsies due to the inflammation of neighbouring organs.

. . . . . .

Symptoms.—These are noticeable during respiration, and are as follows:—*At the moment of inspiration the epigastric and hypochondriac regions are depressed although the chest dilates; during expiration these movements are reversed.* When the diaphragm is merely weak these signs are seen only during forced respiration, but during tranquil breathing the bulging of the belly and the expansion of the chest, and *vice versâ*, occur together, as in health.

When the diaphragm is palsied on one side only, these signs are seen only on the palsied side. . . . Respiration is quicker than usual, but is without serious difficulty during repose. When the patient is asleep one would never suspect so grave a lesion, for the chest movements are performed without the help of the extraordinary muscles of respiration. They are evidently caused by the scaleni (which can be felt to contract under the finger) and the intercostals. But directly the patient makes the least effort to walk or speak, or suffers the least excitement, his breathing quickens at once, and the trapezii, sterno-mastoids, serrati, pectorales, and latissimi muscles begin to contract. The face reddens, the patient feels stifled; he is obliged, if he tries to walk, to sit down after a few steps, or, if he tries to talk, to pause for breath in order to finish what he has to say.

The patient whose diaphragm no longer contracts cannot take a long breath without feeling suffocated. If he sighs he feels, as he well expresses it, his bowels rise in his chest and smother him. Instead, therefore, of trying to breathe deeply, as is the case in other chest troubles when the need of air is felt, he instinctively tries to prevent too great expansion of his chest. The voice is not lost, but weak, and the least emission of sound puts him out of breath. In one case only have I seen complete aphonia, but I am not sure that in this case the laryngeal muscles were not paralysed as well.

I need not say that coughing and sneezing occasion great difficulty in breathing. Spitting and blowing the nose (*l'expuition*) are difficult or impossible; and, lastly, defecation requires great effort, and is accomplished with difficulty.

Such is the imperfect picture of the troubles caused by loss of power in the diaphragm. It will be understood that this loss of power may be present in any degree between complete

loss of function in the whole muscle and a trifling weakness of only a part of it.

[The diagnosis is usually not difficult. Confirmatory evidence might be got by faradising the phrenic nerve in the neck, which would cause a descent of the diaphragm, and thus produce those movements which are wanting.]

. . . . . .

Prognosis.—Respiration occurring only in the upper part of the chest would not be sufficient for the aeration of the blood, and asphyxia would inevitably result. . . . But other muscles (intercostals), happily, are able to produce expansion of the lower part of the thorax, even when the diaphragm remains inactive, so that in spite of the failure of vertical expansion, the expansion of the thoracic wall in all its extent is enough for breathing. The life of the patient is not, therefore, in immediate danger if no complications arise, but it is known that a simple bronchitis may cause death in these cases, by reason of the impossibility or difficulty of expectoration. Paralysis of the diaphragm is not mortal by itself, as has been written and taught hitherto; but is merely a cause of functional trouble (in talking, breathing, &c.), which embitters the life of the patient.

Treatment.—[When diaphragmatic palsy is due to progressive muscular atrophy, Duchenne strongly recommends faradisation of the phrenic nerves.]

. . . . . .

Artificial Respiration by Faradising the Phrenic Nerves.—. . . The phrenic nerve arising from the third, fourth, and fifth cervical descends in front of the anterior scalenus in a direction from without in, and sinks into the anterior mediastinum to reach the pillars of the diaphragm. The phrenic nerve must be reached and put in connection with the rheophores of an induction battery as it crosses the anterior scalenus. The induction battery must admit of fine graduation, and the rheophores must be conical, and covered with moist wash-leather. Local faradisation of the phrenic is not easy, because the scalenus is covered by the *sterno-mastoid* and *platysma* (*peaucier*), but it may be accomplished as follows: with two fingers placed on a level with the outer edge of the clavicular part of the sterno-mastoid press upon the skin from

without in, and when the anterior scalenus is felt separate the fingers while the pressure is maintained, and insert the rheophore between them.

Then, while an assistant keeps this rheophore in position, place the other on the corresponding point on the opposite side. This done, seize the two rheophores by their isolating handles, and keep them firmly fixed while the apparatus is put in action. Immediately the lower ribs separate, the belly rises, and air is heard to enter the lungs. After a few seconds the current is stopped, and immediately the chest and belly subside as in expiration. Expiration may be helped by pressure exercised upon the chest and abdomen by an assistant. After a few seconds' interval, the current is again allowed to pass, and the succession of inspirations and expirations should be made to imitate normal respiration.

The manœuvre just described does not always succeed at first, for if the platysma is very well developed it may contract and move the rheophores off the phrenic; the phrenic nerves again may be anomalous, and pass internal to the scaleni. These difficulties, however, are not insurmountable, for on shifting the position of the rheophores a little, we may at last succeed in hitting the phrenics. Excitation of the brachial plexus is as much as possible to be avoided.

I only have recourse to the method just described when I want to cause contraction of the diaphragm by itself, in order to show the action of this muscle on the diaphragmatic ribs.

The following is a simpler method of producing artificial respiration. Rheophores of large surface (cylindrical sponge-holders) are used, and are placed on the sides of the neck, at the points above indicated. In this way the passage of the current stimulates not only the phrenic, but the cervical and brachial plexuses, and the external branch of the spinal accessory, and hence there results more movement of the chest and raising of the shoulders, which cannot but help the respiratory movement which it is wished to provoke.

Artificial respiration thus induced causes a great mass of air to enter the lungs. Since electric excitation of the phrenics can make a corpse inspire, even noisily, for some time after death, what may not be expected of it in cases of asphyxia, no matter how desperate they may seem? To prolong life in

asphyxia is to save the patient, for without respiration how can any appropriate medication be applied ?

. . . . . .

Contracture of the Diaphragm.

Chronic spasm of the diaphragm causes hiccup, or a sort of barking, or simple shocks without noise. These symptoms have no gravity in themselves, and are merely evidence of other affections.

But if the spasm of the diaphragm become tonic, and continue for one or two minutes, accidents not hitherto described may supervene, which ought to be understood, because they may rapidly cause death. [In these cases, which seem to be exceedingly rare, the chest and belly are fixed in the inspiratory position, while the extraordinary muscles of respiration are brought into play, and the patient presents all the appearances of impending suffocation. In 1853 Duchenne had asked himself the question, if in tetanus death were not due to contraction of the diaphragm. This contracture may also cause the muscular trouble described by Dance under the name of *intermittent tetanus*, by Tessier and Hermel as *idiopathic contracture*, and by Corvisart as *tetany*. Duchenne describes the case of a girl of 18 who was suffering from tetany, and in whom contracture of the diaphragm supervened to an extent which endangered life. Relaxation of the contracted muscle was brought about by applying cloths wrung out of boiling water round the base of the thorax on a level with the insertion of the diaphragm.]

CHAPTER XVIII.

CLINICAL INVESTIGATION OF AFFECTIONS OF THE BRAIN AND CEREBELLUM.*

State of muscular contractility.—Marshall Hall has written that "in brain paralyses the irritability is increased." I have shown elsewhere that by the method pursued by this celebrated physiologist one could only learn the state of reflex excitability of the cord, and that for the exact determination of the state of electro-muscular contractility in paralysis it is absolutely necessary to localise the current in the paralysed muscles. In this way I have investigated whether or no it be true that irritability is increased in brain paralyses. In these investigations we must guard against several sources of error.

1. The contractions which often complicate brain palsies may mask the state of the electric irritability of the paralysed muscles, and cause us to think that it is even lessened.

If, for example, we examine the extensors of the hand in a case of hemiplegia where the flexors are contracted, the muscles will contract feebly to the current as compared with the corresponding muscles of the sound side. On the other hand the contracted muscles respond no better. We must not conclude that contractility is lessened, because, in the first case the extensors, kept on the stretch by the contracted flexors, cannot show their electric contractility, and on the other hand the already contracted flexors can contract no more. In this class of cases we must experiment on completely relaxed limbs without complicating contractions.

2. [Very slight differences of irritability between the two sides of the body are not of much importance in hemiplegia, because a slight difference may often be found between the two sides of the healthy body. This depends probably on slight differences of resistance offered by the skin, due to extra thickness, œdema, &c. . . .]

* From *L'Electrisation Localisée*, 3rd ed., pp. 729—759.

Avoiding all the causes of error which I have just mentioned I have been unable to establish in the many cases which I have submitted to electro-muscular exploration that the irritability is increased by brain paralysis, as stated by Marshall Hall. I have always found it normal. Finally, electro-muscular sensibility is usually intact in brain paralysis.

[With regard to *diagnosis* I have shown that "*in paralyses following lesions of the spinal cord electro-muscular contractility diminishes only when the motor cells are altered or wasted, as in the spinal paralyses of the child and adult from wasting of the anterior cells; electro-muscular contractility remains normal in brain paralyses.*" Brain paralyses, by the fact of the normal irritability of the muscles, are thus readily distinguished from *anterior spinal paralyses*, from *paralyses* from *nerve-injury*, and from *lead palsies*.]

Treatment by Local Faradisation and Continuous Currents.—[Duchenne has been occasionally surprised to find local faradisation of great use in brain paralyses. "It is not usually till after six months from the attack that I have seen faradisation of any service. I shall show anon that the too early application of it is not without danger to the patient."

After the absorption of the brain clot the mental stimulus can return, more or less, to the muscles, and the paralysis is then partly due to the muscles having lost their ability to respond thoroughly to the mental stimulus which has been so long in abeyance. Local faradisation seems to restore to them this lost faculty.

Duchenne faradised many patients in the Hôtel Dieu in the early stages of brain paralyses before the absorption of the clot, but never with any good result. "I have noticed, on the contrary, that its application too soon after the attack is not without danger. Certain accidents caused by premature applications have made me very cautious in the employment of this mode of treatment in the early stages of brain hæmorrhage."

We know that during the absorption of the effused blood, and the formation of the apoplectic scar, movement returns gradually in the palsied limbs, first in the leg and then more slowly in the arm, though in rare cases the arm recovers first. If faradisation were used at this period one might attribute to it an improvement which is the mere result of the natural course

of events. It is then wise (and honest) not to have recourse to local faradisation until the improvement has come to a standstill, and fair time has been allowed for the absorption of the clot. When faradisation has been used a long time after the accident Duchenne says, "I have seen the paralysis completely cured in a very small number of cases; often enough it has been improved; still more often I am forced to confess that I have noticed no kind of appreciable result."

The recovery in these cases depends necessarily upon the extent of brain damage, and the amount of healing or secondary damage which takes place. Duchenne's experience leads him to say:—

"The patients who, five, six, eight, or more months after the attack, are still more or less completely paralysed, but without any muscular contraction, have generally recovered rapidly under local faradisation. Those, on the other hand, who had stiff joints and contracted muscles did not derive any benefit from local faradisation. I need not give the too long list of failures scored to the account of faradisation when employed in such cases."

When the limbs are permanently contracted (a condition attributed to a fascicular induration descending from the scar into the cord) local faradisation is contra-indicated, and Duchenne states that accidents have occurred concerning which he is sure that they are to be attributed to the unwise use of faradisation in such circumstances.

Faradisation is often beneficial in curing the paralysis of the face and tongue in cases of hemiplegia, but its application is not without danger.

Case No. 72.—Under the care of M. Cruveilhier (Charité, 1848), right hemiplegia of two years standing, in a man aged 38, who had had many attacks. Before faradisation, the commissure of the lips was more depressed on the right side than on the left. The tongue deviated to the right. Active intermittent contraction of the pectoralis major and flexors of the upper limb, disappearing with the warmth of the bed and during the night, and increasing when the patient is touched or irritated (*impressionné*); similar contraction of the lower limb causing pain and turning the foot inwards. Complete absence of movement in the upper limb since the first attack; movement in the lower limb sufficient to allow him to walk with a stick, but with considerable mowing of the foot and much stiffness and jerking

owing to the contraction. The foot cannot be placed flat on the ground. Faradisation of the trapezius, deltoid, and other muscles for ten sittings, after which the patient could move his arm nearly to a right angle with the trunk, mowed less in walking, and placed his foot better on the ground, when the treatment had to be suspended owing to a serious and unforeseen accident. The faradisation of the face and tongue were being practised with some energy when the patient was almost immediately stricken with a fresh attack of apoplexy, which necessitated many blood-lettings.

Faradisation of the face seems to me to be the probable cause of the accident which endangered this patient's life. In ten cases of facial hemiplegia (from brain lesion) in which I applied faradisation in the years 1847—48, I have seen more or less serious brain trouble follow the application in three cases.

It becomes therefore very necessary to distinguish between facial palsy from brain damage and facial palsy from damage to the facial nerve. In the former the orbicularis palpebrarum almost always escapes and the electro-muscular contractility remains normal, while in the latter the orbicularis is affected and the contractility is lessened and ultimately vanishes.

How should Faradisation be practised in these cases?—We must always bear in mind that the patient who has had one attack of cerebral hæmorrhage is liable to another. Faradisation must therefore be strictly localised to the muscles, and gentle currents with long intermissions must be used. Do not let either rheophore approach the nerve centres, and always remember that the patient or his friends, reasoning by the *post hoc* fallacy, will always attribute any accident occurring during treatment to the treatment itself.

There is no use in continuing the treatment too long, and if movement has not returned after fifteen or twenty sittings it should be discontinued.]

I have too long considered it dangerous to use faradisation for the brain paralyses of infancy, fearing that it might provoke fresh accidents by reason of the great excitability of children. Consequently, although I often advised this treatment for adults I always forbad it for children. Such was my teaching in the first edition, founded on prejudice, and not then established by experiments which now are completed.

I have used it on a great many poor children sent to me from the hospitals, and I can declare that I have never seen a single mishap to the brain occur from the employment of muscular faradisation to children, no matter how young. But I must say at once that I have never departed from those principles of procedure which I have given above for the use of localised faradisation in the brain palsies of adults.

The results obtained in children were always encouraging, even in congenital cases. Thus facts have shown me that certain congenital brain palsies, which have remained a long while stationary, may be singularly improved by this method of electrisation. Need it be said that hemiplegias dating from intrauterine life or early infancy differ from those occurring in the adult in nature, course, and prognosis, and that they resemble them only in so far as they are symptomatic of secondary induration to a variable extent of certain bundles of the antero-lateral columns of the cord following various primary lesions of the brain?

It must be recognised that hemiplegias following severe congenital lesions of the brain are incurable; but it is also established by my clinical and empirical observations that many of them may get well, or improve. I have recently had a fresh proof of this, which I will give in abstract in spite of the almost too marvellous result with which, so to say, it is tainted (*entachée*).

Case No. 73.—Léocadie Desplanches, 4, Rue de Marignan, aged 12½, appeared to be in good health till the age of six months. At this time she seemed to have an intestinal trouble without fever or convulsion, and at the time her mother noticed that her left limbs were stiff and contracted in the direction of flexion, and painful when they were straightened. Standing and walking were consequently difficult. Towards the age of 5 the contraction diminished and the pain disappeared, and the improvement had been progressing until the time of her first seeing me (November 1st, 1871). At this time the left hand was constantly closed by contraction of the flexors of the fingers in spite of all efforts to open it. The flexors of the wrist and elbow and the adductors of the humerus were likewise contracted. These contractions increased during voluntary efforts, so that the limb was completely useless. Sensation was everywhere perfect.

In the leg many muscles were contracted to a certain degree, and voluntary efforts or emotion increased the contraction and produced reflex tremor. Walking was thereby hindered. Faradisation was begun on November 1st, and consisted in moderate stimulation with slow intermissions of the muscles antagonising those which were contracted. After the fourth sitting there was already a notable improvement. The contraction had diminished, the hand could be opened, movements of the arm could be made, and the hand could be placed behind the head. The strength had also increased. The patient seized the handle of a pail full of water, and easily carried it for a certain distance. This improvement went on increasing. After the ninth sitting the hand could be opened voluntarily. Using it became more easy, and for the first time since birth the little girl was of some use in the house. For example, she could wash the plates and dishes, clean the boots, and even managed to hold things firmly enough to learn to sew. When standing the contraction of the flexors of the hip caused her to bend forward. Now she can stand straight and walk without limping but with a little dragging of the leg.

. . . . . .

Therapeutic value of continuous currents in paralyses following a hæmorrhage in the brain.—[Duchenne believes that the early application of these currents is not without danger. He doubts Remak's assertions as to their value, and has no facts of his own to offer.]

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FUNCTIONAL TROUBLES THE RESULT OF CEREBELLAR DISEASE.

It follows from my clinical observations, which will be summarized in this article, that the chief functional troubles symptomatic of cerebellar disease are—

1. Staggering, without motor inco-ordination, while standing or walking.
2. Giddiness, increased or caused by the upright position, or by standing or walking.
3. Nausea or vomiting often accompanying the giddiness.
4. Double vision or squint (not paralytic) during a fixed gaze.
5. Integrity of electro-muscular contractility.

To begin with, I will quote two cases of cerebellar disease in which the diagnosis formed during life was proved after death. . . . .

Case No. 74.—C. Robinet, æt. 28, working watchmaker, was admitted to the Lariboisière on May 8th, 1863. The patient, who has apparently a sound constitution, says that between the ages of two and ten he was liable to epileptic fits, which grew more rare and less severe as he grew older. His health was good till the age of twenty-three. At this time, having been three years in America, he had a hard chancre, which disappeared under treatment after fifteen or twenty days. Since then he has had sore throat, soft tumours scattered over his body, and has been subjected to antisyphilitic treatment for two months.

Towards the end of 1862 he entered the Midi Hospital, under M. Cusco, for vomitings, which occurred many times daily, and usually after meals. There was then a slight squint, and he returned to the hospital again shortly afterwards for the same symptoms. The vomitings continued.

Present state.—There is a condition of mental torpor, and sometimes his answers have no relation to the questions put. Generally he understands and replies almost correctly with a lagging speech, but turns away with indifference directly one ceases to fix his attention. Sensibility all over the body is slightly blunted. The squeeze of the hand is feeble, and the left arm and hand are weaker than the right. When the arm is extended it trembles notably. The gait is that of a man half asleep, staggering and uncertain. This has no relation to the muscular force, which is good enough, as is shown by a dynamometrical record made by M. Duchenne.

The gait was not made worse by shutting the eyes. The patient had not vomited for many days, but said that he felt sick when he hung his head down. Digestion fairly good. Complete impotence since two months. Urine expelled with fair force, and retained only once in two days. It contains neither sugar nor albumen. Senses all rather blunted. Sight obscured by a fogginess of varying intensity. To read comfortably the patient is obliged to shut the right eye; with the two eyes the letters look bigger but less distinct. There is slight squint, which has lasted some time, but otherwise the eyeballs move

naturally in the orbits. The ophthalmoscope reveals slight sub-retinal œdema.

The diagnosis was a tumour (probably syphilitic) situated in the cerebellum (*cervelet*) and spreading towards the brain. The patient was treated with mercury and iodide of potassium. Things remained stationary for some days; then the patient was seized with acute delirium, and died at the end of two months without having recovered his mental power, and having shown intervals of alternating excitement and depression.

The post-mortem examination thirty hours after death showed no lesion of the cord nor of the cavities of the chest or belly. There was marked injection of the pia mater on the convex surface of the brain, and on trying to remove the membranes a portion of the substance of the right lobe of the cerebellum was found to be adherent over an area about two centimetres in circumference. There were no adhesions elsewhere. There was nothing else observed, unless it be some hyperæmia of the entire brain, most marked in the cerebellum.

With regard to the diagnosis of this case from locomotor ataxy Duchenne observes: "While the patient was standing the body oscillated in every direction. It was a sort of balancing of the body not interrupted by those sudden movements which are produced by the efforts to balance in locomotor ataxy. There were no sudden muscular contractions of the limbs or trunk, and the feet rested quietly on the ground. When walking his swaying increased in extent; he described arcs of circles or zigzags, and the limb which swung from behind forward did not advance too far (*n'allait pas au delà du pas*), and did not knock with the heel on reaching the ground. He walked, in short, as if he were drunk."

While standing he suffered from giddiness and nausea, and during walking the giddiness increased and the sight became confused. The signs which collectively so closely resembled drunkenness are such as I have seen in many cases of cerebellar disease, and should not be confounded with those of locomotor ataxy.

In 1853, M. Vigla, physician of the Hôtel Dieu, kindly directed my attention to a patient under his care, whose troubles might at first sight have been regarded as locomotor ataxy. But on careful analysis I found that they corresponded with those of

the preceding case. I attributed the giddy staggering of this patient to a lesion of the cerebellum. Further, as there was muscular weakness, more marked on one side, I recognised that there was possibly also a lesion of the brain. The diagnosis was confirmed, as will be seen, post-mortem.

Case No. 75.—(Hôtel Dieu, under the care of M. Vigla, 1863.) Bodin, æt. 28, a whitesmith, usually enjoyed good health, but was in the habit of masturbating. For four months there had been deafness of the left ear, which began with buzzings and tinklings, but without discharge, pain, sore throat, or nose catarrh. Two months previously, suddenly and without known cause, he had had a dazzling of the eyes which had obliged him to cease working for ten minutes. Soon this dazzling became constant, and at times he saw double, but only with the left eye. With the right eye sight was dim, and he could not recognise objects at a distance of 20 centimetres, although he could distinguish the light. When his deafness came on he suffered from vomiting for a fortnight, with pain in the forehead and eyes. Later he experienced a sense of emptiness when bending the head forwards or backwards, but no nausea. There was no loss of memory. The difficulty of walking had lasted two months. At first he could direct his steps with his eyes, but in a few moments sight became confused. The patient walks, as he says, like a drunkard; his head swims and his sight gets obscure; he separates the legs, holds on by near objects, and cannot keep to a line; he wants balance, but there is no knocking with the heel. While standing with his feet close together he sways more. After walking a few minutes he falls from failure of his sight. In the left leg there is notable weakness in the movement of the flexors of the ankle and hip, and there is some lessening of sensibility. There is no weakness of the arms nor of the right leg. He does not hear a watch placed on the left temple or behind the left ear. There is no difficulty in speaking. In a few weeks his sight was completely destroyed and his pupils were strongly dilated, although at times they contracted spasmodically. An ophthalmoscopic examination by M. Galezowski showed the optic papillæ projecting and swollen, their outlines irregular, and hidden by a whitish exudation. The vessels were enlarged and varicose, and at the edge of the papillæ were buried in the exudation, re-appearing on the retina at a distance of 3 or

4 millimetres beyond the edge of the papillæ. The capillary vessels of the papillæ were very big, so that in some spots they looked like effusions of blood.

In short, the two cases just given are very much alike as regards the symptoms of cerebellar disease. The slow course and syphilitic antecedents had in both cases caused a recognition of the possibility of tumours compressing both cerebrum and cerebellum. Iodide of potassium was given on this hypothesis, nevertheless post-mortem examination showed that tumours existed only in the second case, while the first showed only a chronic inflammation of the brain and cerebellum, most pronounced at those points where there were adhesions. None of the symptoms would have enabled us to foresee this difference in the nature of the two cases.

In the second case fibro-plastic tumours were found; one situated above the corpus callosum was a good size (half as big as a fist), and slightly compressed both hemispheres, but especially the right. Two other tumours (neuromata) as big as a walnut were each astride of the middle cerebellar peduncle. Finally, a multitude of other tumours of the same nature were situated along the course of the spinal cord, the roots of the nerves, and the nerves of the periphery. I will remark that these numerous neuromata, which in the nerves were situated between the neurilemma and the nerve tissue, did not cause any nervous trouble (loss of electro-muscular contractility, sensibility, or pain). Microscopic examination showed, in fact, that they had merely unplaited and separated without causing wasting of the neighbouring nerve-elements, which seems to me to show that they were slowly developed. The absence of those grave disorders usually symptomatic of injuries of nerves, and the peculiarities observed in locomotion, permitted the diagnosis of a lesion of the brain and cerebellum.

Just a few words may be added on the singular eye-phenomena observed with peculiar care in one of these cases. When I made him read alternately with each eye I noticed that his sight was very weak on the left side, and normal on the right. If he looked at an object with both eyes his sight became confused in a few seconds, and became double, and that in proportion to the fixity of his gaze. Thus I made him look at one of my fingers, and usually after eight or ten seconds he saw two

confusedly and gradually separating from each other to a distance of 20 or 30 centimetres. If this experiment were too prolonged it caused giddiness and nausea. Nevertheless none of the nerves of the eye were paralysed. *On the other hand this double vision was equally produced when he looked with the left eye only.* This phenomenon of double vision with one eye cannot be denied, for I have made the same experiment many times in the presence of witnesses.

I have asked M. Galezowski to confirm this curious fact, and he has authorised me to publish the following note, in which he gives the results of his ophthalmoscopic examination and their interpretation.

"On examining this patient, I found that there was no trace of paralysis of any of the muscles of the eyeball, but the pupil, nevertheless, was less dilated than in the sound eye. It dilated very incompletely even under the influence of atropine. The patient saw double with this eye, and the false image was fairly plain. Light was very confused at ten paces, although before he was ill the patient could see long distances.

"The papilla was infiltrated at its external part (the right side of the image), and there was a slight whitish streak stretching to the macula. This infiltration was very slight, but at the same time all the veins were sensibly swollen, and the capillary vessels increased in size.

"It was an *acute serous neuritis* such as we have already described under the name of œdema or infiltration of the optic nerve. But how are we to explain the infiltration of the optic nerve in a cerebellar affection, and the double vision with one eye? To answer the first question it is sufficient to examine the researches of Magendie, Flourens, Béclard, Longet, &c., and one will understand that, as the visual functions reside in the corpora quadrigemina, sight ought to be affected or lost when these are congested, inflamed, or compressed by mischief in a neighbouring organ. But all cerebellar mischief may easily communicate by means of the *processus cerebelli ad testes* with the corpora quadrigemina, and consequently produce dim sight or complete blindness, as has already been shown by the observations of Andral and others.

"The question of the double vision with one eye is more difficult to solve. M. Desmares père has said, 'Exceptionally it is only

the retina that collects the two images, but then the double vision is always with one eye only.'

"It is easy to understand double vision with one eye when there is displacement or opacity of the lens or aberration of sphericity following failure of refraction, &c.; in this case we must seek another explanation, and I believe it is to be found partly in the failure of accommodation and partly in the functional troubles of the retina.

"With regard to accommodation we find a failure to adapt the eye to long distances. This was due probably to a spasm of Brücke's muscle which makes the eye myopic. We know, on the other hand, the power of the lens to give double or multiple images as soon as the limits of accommodation are passed. 'Then by placing the candle 5 or 6 metres off,' says M. Giraud Teulon, 'the myopic eye will see the flame multiplied.'

"Another circumstance might contribute to the double vision, and this was the localised infiltration of the retina depending on the cerebellar trouble. We know in fact that the cerebellar trouble communicates itself to the posterior corpora quadrigemina, and thence by the external *corpora geniculata* to the external parts of the retina of the corresponding eye. An image projected on the infiltrated part of the retina will be feebly perceived, while the neighbouring fibres, and notably those which come from the corpora quadrigemina interna and the opposite hemisphere, will perceive the image clearly and distinctly. From the spasmodic contraction of the accommodating muscle the eye has become myopic, and disposed to double objects seen from afar, and the retinal trouble would render the double images still more complete."

I have noticed the same fact (double vision with one eye) in another patient. It would be interesting to investigate whether it is often present in cerebellar troubles, and whether it is a special symptom of them. I could mention other cases of cerebellar trouble . . . but they so closely resemble those just given that I should merely tire my readers by doing so.

It results, in short, from my cases that the locomotor troubles in cerebellar affections like those of alcoholic drunkenness are caused solely by giddiness, or in other words that they are characterised by giddy staggering.

Diagnosis of Cerebellar Trouble from Locomotor Ataxy. —In my early investigations I confounded, as I have admitted,

the symptoms of motor inco-ordination due to locomotor ataxy with the troubles of standing and walking caused by cerebellar mischief. I should certainly not have made such a mistake, if, instead of trusting to appearances, I had more closely analysed the functional disorders presented by these diseases. I am now able to describe exactly the locomotor troubles which characterise both of them.

A. Let us look at them first when standing. As soon as the *cerebellar* patient begins to stagger, his body, while standing, begins to reel in every direction, laterally, from before back, or *vice versâ*. The extent of the reeling is directly proportioned to the amount of damage. He seems to abandon himself gently and quietly to the balancing process.

In the *ataxic* patient the oscillations have a very different character. They are sudden, short, and more rapid, like those of a tight-rope dancer who tries to maintain his equilibrium without a balancing pole. Nothing can be apter than this comparison. The muscular contractions which in the upright position cause the sudden swayings of the trunk are like spasms. To establish this fact the lower limbs must be exposed; then it is plain that when the body is strongly jerked by these oscillations, certain muscles of the limbs contract strongly and suddenly for a moment. It is these contractions which cause the little sudden swaying movements of the body.

These contractions, however, are not mere spasms; they are also purposive with the object of maintaining the body in the line of gravity. I have in fact established that when the patient sways in one direction it is always those muscles which drag the body the opposite way which enter into contraction. These contractions are as much instinctive as voluntary. They sometimes occur at the very beginning of the muscular inco-ordination when the patient sways slightly without knowing it, and one can already see little short contractions of the muscles of the feet and toes.

I have lately heard a very sharp observation from the wife of a patient with locomotor ataxy, to whom I was called in consultation by my friend Dr. Contour. "For some months," she said, "when he tried to stand without his shoes she had remarked that his feet, instead of resting quietly flat on the carpet, were jerked by continuous little movements, and that his toes were incessantly raised and depressed." She had also noticed a slight

swaying of the body. But these instinctive contractions soon get mixed with voluntary contractions. As soon as the patient recognises his loss of balance, one sees that he is incessantly preoccupied with it, and makes efforts to preserve it, which soon exhaust him. Neither can he stand for long without suffering fatigue.

B. It is only when walking that we can distinguish the cerebellar patient from the ataxic patient arrived at the stage of muscular inco-ordination (*asynergie*). The first, as he walks, describes curves, alternately right or left, or zigzags, and cannot walk straight on. He leans slightly from the side towards which he is swaying, and then seems to be dragged laterally by the weight of his body. It sometimes happens that the inclination of the body is so great that he falls on that side. Although he reels in this way when walking, he keeps time naturally, and the limb swinging from behind forwards does not overstep the mark, nor does the heel come noisily to the ground. On the contrary, he often drags his legs when walking. . . . .

The ataxic patient, on the other hand, walks straight before him, wavering, but without describing curves and zigzags like a drunken man. His swayings have the same character when he is standing, but walking increases them. They are proportioned to the amount of inco-ordination. The patient is very uneasy when walking. He is so afraid of losing his balance that his eyes are fixed on his legs or the ground, and he dares not look away from them for fear of falling. (*I have never seen this in cerebellar staggering.*) Finally, there comes a time when the inco-ordination reaches such a pitch that he can no longer keep time when walking. In this way the foot, swinging from behind forwards, oversteps the mark and the heel comes noisily to the ground. Further, during this swinging of the lower limbs the flexion of the foot on the leg is exaggerated.

The walk of a man with cerebellar disease is not at all like this.

The diagnostic value of the Giddiness and Double Vision in Cerebellar Disease.—Giddiness is usually the first symptom of cerebellar disease. I have asked many of these patients if they felt giddy, and they have told me that their head seemed to turn, or that they saw everything turning round them, and this it was which prevented them from standing steady or

walking straight. Their staggering is not caused by want of muscular co-ordination, but by giddiness, and therefore I have called it *vertiginous staggering* (*titubation vertigineuse*). Locomotor ataxy, on the contrary, causes no giddiness. The want of balance and staggering present at a certain period of the disease cannot be attributed to giddiness. I have inquired if they were giddy while standing or walking; if their sight troubled them; if they experienced nausea; if, in a word, they felt their heads stupid and giddy, as is the case after too much wine. They say that their heads are perfectly clear, and *the want of balance is only in the legs*. I have seen some who had been drunkards, and these well understood the difference. They knew that after drink it was the turning of the head which prevented them from walking a line or standing steady, but that in the disease it was the fault of the legs only that prevented their steadiness. One of them said to me, "When I was drunk I could not balance with my head; but in the disease the head is all right; it is down below that I lose my balance."

It is well understood that in taking the state of vision into account, we must remember that double vision due to paralytic squint causes dazzling and sometimes giddiness during walking, and deviation from the straight line. But since all these troubles disappear when the squinting eye is shut, it is always easy to avoid this source of error in ataxies.

In short, it is manifest that the staggering in locomotor ataxy is not caused by giddiness as in cerebellar disease, but is entirely explained by the loss of the co-ordinative faculty. For this reason I have called it the *staggering of inco-ordination* (*titubation asynergique*), the signs of which are essentially different, whether in standing or walking, from the *staggering of giddiness* (*titubation vertigineuse*) caused by alcoholic drunkenness.

CHAPTER XIX.

HYSTERICAL PARALYSES.*

Local faradisation applied to the study of hysterical paralyses has shown me that there are certain changes in the properties of the muscles. These symptoms, which I am going to state briefly, may be of use in establishing the differential diagnosis of these paralyses.

Electric contractility in hysterical paralysis is normal, but electro-muscular sensibility is diminished or abolished.

. . . . . .

[This fact distinguishes hysterical paralyses from paralyses from nerve injury (in which irritability is lessened or lost), and from brain paralysis (in which irritability and sensibility are both usually normal).]

Treatment.—[Local faradisation is very potent in these cases, and usually, but not always, succeeds. Duchenne's experience has enabled him to draw up certain rules for the treatment of these cases.

1. *The electric stimulation should be localised in each of the affected organs, and the treatment should be continued for some time after the return of movement, in order, so to say, to establish the cure.*

Sometimes a single application is sufficient for the complete recovery of the patient.

Between 1848 and 1860 I saw at the Charité, under M. Briquet, a good number of hysterical patients whose left limbs (especially the arm) were more or less anæsthetic. This was usually accompanied by muscular weakness. It was almost always sufficient in these cases to recall the sensibility in order to re-establish the normal muscular power.

2. *It is doubtless by exciting the nerve-centres by a kind of reflex action that faradisation of the skin recalls movement in*

* From *L'Electrisation Localisée*, 3rd ed., pp. 711—728.

hysterical paralyses. There are even cases in which it seems to succeed better than muscular faradisation.

The success of faradisation is often brilliant, but sometimes in quite similar cases it fails altogether.

3. *The cases which I have collected have not enabled me to recognise the signs which tell whether or no faradisation will cure a case of hysterical paralysis.*

4. *The paraplegic form of hysterical paralysis seems to be that in which the chances of success by local faradisation are the least favourable.*

M. Briquet has shown that the various manifestations of hysteria, both motor and sensory, are most common on the left side, and Duchenne has observed the same fact, and even when the paralysis is limited to the legs it is the left leg which suffers most.]

HYSTERICAL CONTRACTIONS.

These usually follow some lively emotion, shock, or fright. There is at first an hysterical attack, followed by a local contraction in one or more limbs, which are at the same time affected with paralysis or paresis, combined with increase or loss of sensibility. Sometimes the paresis, accompanied or followed by contraction, is the first to appear, and the hysterical attacks only take place at a more advanced stage of the disease.

The contraction or stiffness is usually permanent, but may be temporary. In the latter case it is brought back by emotion or any excitement acting in a reflex manner, and lasts from a few minutes to some hours. . . . These two forms of contraction (the lasting and passing) are sometimes found in the same patient.

The contractions usually affect certain groups of muscles in the limbs or segments of limbs which are attacked. In the upper limb the muscles supplied by the median, in the leg those supplied by the sciatic, and in the thigh those supplied by the crural are attacked. I have almost always seen them in the gastrocnemius and soleus, causing equino-varus, and ultimately a true club-foot, with deformity of the joints. Often, when I have tried forcibly to bend the ankle, the contracted muscles have been agitated by convulsions and tremors, which extended to the whole limb if the experiment was continued. These signs so

closely resemble those which are symptomatic of lateral sclerosis of the cord that one is inclined to allow the existence of some hyperæmia of the lateral columns. . . .

Diagnosis.—Hysterical contractions for the most part occur suddenly after a lively emotion; in lateral sclerosis they occur slowly and progressively, having been long preceded by paresis. If these signs furnished by the course of the disease are not plain enough, the hysterical symptoms will help the diagnosis, or even settle the question.

Case No. 76.—A girl, aged 24, a baker's assistant, usually healthy, was in the habit of carrying bread daily to a customer. One day she found him dead in his bed, and the shock was so great as to cause an hysterical fit, lasting several hours. After this she remained deprived of movement, the lower limbs being tetanised, and presenting a well-marked equino-varus. Her menstruation was suppressed, and she became blind. Certain senses were strangely perverted. If she were pinched or spoken to on the right side, she felt and heard on the left.

The contractions of the legs lasted several years, long after the disappearance of the other troubles, and this persistence might have caused a fear that they were symptomatic of damage to the cord. Nevertheless the whole group of symptoms just given made me certain of its hysterical origin.

This diagnosis was completely justified by her spontaneous and sudden recovery, only some deformity of the joints, caused by the long-sustained faulty posture of the feet, remaining.

Case No. 77.—(For details see *Elect. Loc.*, 1855, Case iv.) A girl who had an hysterical attack in 1848 (from the shock of hearing the call to arms in the streets of Paris), followed by a contraction of the left elbow, wrist, and fingers, which lasted several years, in spite of a variety of treatment. The least application of faradisation was enough to bring on hysterical symptoms. Continuous currents were not used. After lasting two years, this contraction disappeared suddenly one day after a douche of cold water over the surface of the body.

In this case the hysterical nature was not to be doubted, but I have met with cases where the diagnosis was uncertain, from the insufficiency or slow appearance of the hysterical symptoms.

Treatment.—[In most of these cases faradisation is contra-indicated. The application of it often causes hysterical attacks,

and only once did Duchenne obtain a satisfactory result (by faradising the antagonists of the contracted muscles). In one case a good result followed a six-weeks treatment with the continuous current, a "*descending stabile*" current being passed along the nerves of the affected limbs; but Duchenne says, "I ought to add that in order to conquer the excitability of this young person I gave chloral internally during the last fortnight, which she has continued to take since her recovery."]

ASCENDING REFLEX CONTRACTIONS FROM INJURIES TO JOINTS.

[This form of contraction comes on after violence done to certain joints, especially to the wrist, as by falls upon the palm or back of the hand. The violence causes slight inflammation, or a painful condition of the joint. This form of contraction has, as far as I know, not been hitherto described. The contraction affects certain of the muscles acting on the joint, and comes on when the joint is no longer painful and seems to be quite well. The trouble then spreads to other joints of the injured limb. The pain, at first slight, is limited to the contracted muscles; it then spreads to other muscles, but is always worst in those which were first affected; it then attacks the trunks of the nerves, and at last the origin of the brachial plexus. Often, indeed, after the contraction has ceased there remains a painful condition (sometimes for years) near the origin of the nerves of the affected limb indicating apparently a diseased state of the spinal cord at this level. Finally, the power of the affected limb is usually lessened, and the sensibility diminished.

I have cured many cases of this reflex ascending contraction by faradising in a painful manner (with the "extra current" and rapid intermissions) the antagonists of the contracted muscles.

A typical case is given. Mdlle. X., æt. 16, fell upon the back of her right hand when 11 years old (in 1861). For two months the arm was carried in a sling. The wrist was painful at times, there was weakness of the arm, and "when she attempted to write the weakness quickly increased." Two years later, after dancing, the wrist became so painful that the hand was useless, and in spite of blisters, splints, &c., the condition got worse. In 1864 a contraction of the "radials" and pronators appeared, and continued in spite of blisters, cauterisation, sea-

baths, &c., &c. In 1866 she was sent by Nelaton to Duchenne. There was then permanent flexion of the wrist, with pronation of the hand, and inability to straighten the wrist or supinate the hand voluntarily. When the wrist was straightened by force, the patient cried with pain, and when left to itself the old position was at once resumed. The hand was useless; the shoulder and elbow moved naturally, and the size of the limb was maintained. Continuous currents were ordered, with iron internally, but when the patient returned home the condition became aggravated and more extended, and this in spite of cupping, &c., and when next seen the superficial and deep flexors of the fingers were found contracted, so that the nails were driven into the palm of the hand. There was pain in the dorsal spine sufficiently great to keep the patient awake. The limb remained of normal size. The continuous current applied to the contracted muscles produced a certain amount of good, but the cure was completed by the vigorous faradisation of the antagonists of the contracted muscles. The improvement was maintained from April to December, 1866, but her troubles returned with the cold weather, and the pain in the cervico-dorsal region of the spine was intense, and yielded neither to narcotics, blisters, nor cupping. This time it was the supinator brevis which was contracted, and the arm was in a state of forced supination. In September, 1867, a vigorous faradisation of the supinators again effected a cure, and the spinal pain was relieved by faradising the skin of the neck and back. "Since then the contractions have not reappeared, but in a few weeks the spinal pains came back, and were worse than ever, with increase at night. They still persisted in 1869, in spite of all kinds of treatment."]

CHAPTER XX.

DIPHTHERIAL PARALYSIS.*

I HAVE treated many cases of palsy of the palate with localised faradisation. The causes of this paralysis are many. Thus I have seen the azygos uvulæ paralysed in *rheumatic affections* of the seventh, which reached a certain height in the Fallopian canal or higher; I have seen it as a symptom of *glosso-labio-laryngeal* paralysis; I have collected instances of atrophy of the palate in the last stage of *progressive muscular atrophy*, and some few of palate-palsy pure and simple. I have seen it follow a simple pharyngitis, as Trousseau was the first to point out, and of which Maingault gave confirmatory evidence; and lastly, a considerable number of cases following diphtheria have been sent to me. I have, therefore, had sufficient experience to write an interesting treatise on palate palsies, but since these palsies have been perfectly studied by MM. Orillard, Bretonneau, Trousseau, Lasèque, Faure, Moynier, &c., and more especially by Maingault, what I should have to say on the subject would in the main be merely confirmatory of facts exposed by others. But as I wish to restrict myself to what is new or has been incompletely studied, I shall not speak of my own researches into the pathology of palate palsy, but shall merely take the opportunity of giving my views on the pathology of that form which is caused by diphtheria.

It can easily be shown that this form of palate palsy is distinct and special. It results from facts which have been collected and published that diphtheritic palsy is rarely limited to the palate, but spreads to the limbs, causing loss or lessening of sense and motion. Sometimes sensibility is alone affected, while motor power remains.

Case No. 78.—In 1859 I saw a case of loss of sensibility in the limbs following a diphtherial palate palsy which had been cured

* From *L'Electrisation Localisée*, 3rd ed., pp. 884—901.

by localised faradisation. Muscular power was normal, but the patient allowed objects to drop from his hand unless he looked at them, and he could not walk in the dark. He had lost touch and muscular sense in the hands, feet, and legs. Localised faradisation quickly cured the palate palsy, but the loss of sensibility in the limbs resisted treatment for many months, as has happened also in all the cases I have seen of loss of sensibility following diphtheria, whether combined or not, with loss of movement.

I do not think that the facts of the above case are exceptional. To establish them the force of partial movements must be carefully examined, when it will be seen that there is no motor palsy, but that the patients let objects drop from their hands and walk with difficulty only when they cannot see. This condition is not so rare as is usually thought, but nevertheless loss of sense and motion generally occur together. In most of the cases I have seen, loss of movement did not exist to the same degree as loss of sense, but motor power was merely lessened. This might lead one to think that it was not a true paralysis, but rather a general weakness. Such a view, however, is not tenable in view of the following facts:—1. I have noted cases in which one side was weaker than the other, which could only happen in a true paralysis. 2. I have communicated to M. Maingault a case of diphtherial palsy limited to one leg, of which the following is a summary:—

Case No. 79.—A little girl of 10, after severe diphtheria, had palate palsy, which was quickly cured by localised faradisation. Then she began to suffer from "creepings," limited to the right foot and leg, which were found to be almost senseless. This little patient had great difficulty in moving this leg. She could not stand upon it without its bending, and when she walked her foot trailed along the ground, because she could not bend the leg. Electric irritability normal; no wasting. She was cured in a few weeks by localised faradisation.

These facts show that diphtherial paresis is a true paralysis.

To cases given in previous editions I am now able to add others in which troubles of the heart and breathing occurred, which teach us the pathology of diphtherial paralysis.

Sensory troubles with palate palsy have, as a fact, been already pointed out as among the chief symptoms of this form of paralysis.

The latter might be looked upon as peripheral, and due to the inflammation of the region, but with it, or soon after, appear, but too often, other formidable symptoms which have not as yet received sufficient attention, and which, when carefully analysed, teach us, as I believe I have shown, that they are probably signs of disease of the medulla oblongata.

The following case is interesting :—

Case No. 80 (summary).*—Diphtheria of wide extent in a pregnant woman, æt. 21. Palsy of the pharynx and palate on the sixteenth day; on the twenty-seventh day the patient aborted, and this was at once followed by other paralyses symptomatic of disease of the medulla oblongata, *i.e.*, 1. Serious troubles of the heart's action caused by palsy of the vagus (pulse 136, small, irregular, intermittent—valvular sounds not recognisable), which were dissipated by slight faradisation (with the "electric hand") of the præcordial region. 2. Two days later double vision lasting an hour, followed by loss of sense and motion on the left side, which was cured in an hour by faradising the skin of the paralysed parts. 3. Two or three hours later there was a return of the heart trouble, relieved by faradising the skin of the præcordial region at frequent intervals night and day for forty-eight hours. 4. Grave respiratory troubles, indicative of a palsy of the intrinsic expiratory muscles (bronchial muscles of Reissessen), or in other words a palsy of the lung, which was many times relieved for half an hour or an hour at a time by faradising the skin of the *back* of the chest. 5. Finally, a choking of the lung with its secretions, owing to failure of expiratory power, and death on the thirty-eighth day at two A.M.

In the foregoing summary, it is observable that, when convalescence was apparently established, and the palate palsy was almost well, there occurred heart troubles suggestive of palsy of the pneumogastric. Faradising the skin of the præcordial region (reflex stimulation of the origin of the vagus) quickly triumphed over these attacks, whose frequent recurrence for several days endangered the patient's life. . . . . Unhappily a paralysis of the intrinsic expiratory muscles evidenced a lesion

* This case, taken from Part I. of *L'Electrisation Localisée*, where it is used to show the value of the reflex effects of localised electrisation, is interpolated here for the sake of convenience.

of another spot in the medulla (doubtless the nucleus of the spinal accessory) and produced asphyxia.

Such is a sketch of this form of diphtheritic palsy, which I propose to call *bulbar diphtheritic palsy.*

Its physiological analysis has taught me—

1. That it is symptomatic of disease of the medulla oblongata, principally the bulb.

2. That the symptoms vary according as the nuclei are attacked successively, or together, or partially.

3. That they are fugitive, but that many of the cases prove fatal.

I saw with MM. Millard and Aug. Ollivier another case of bulbar diphtheritic palsy, of which the following is a summary:—

Case No. 81.—A father who kissed his child dying of diphtheria suffered in his turn. Having passed through the stage of the formation of false membranes, which endangered his life by the obstruction of his bronchi, the patient was attacked towards the twenty-fifth day, after convalescence had set in, by certain palsies which showed the period of the onset of diphtheritic intoxication (palsy of palate and pharynx, double vision, palsy of the fifth and seventh nerves on one side, and of the ninth and inferior laryngeal). Suddenly, on the twenty-eighth day, the intrinsic expiratory muscles became paralysed. The bronchi became choked with mucus which could not be expelled, and he became threatened with asphyxia. This palsy unaccompanied by fever caused fears of his rapid death.

In this case again the electric excitation of the skin of the back of the chest quickly re-established expiratory power, and caused the immediate expulsion of the bronchial mucus. It had to be continued many days in order to completely conquer the palsy of the lung.

Case No. 82.—I have now under treatment by localised faradisation two patients suffering from diphtheritic palate palsy which resisted ordinary treatment. One was sent to me by M. Millard and the other by M. Contour. The palate palsy was cured in a few sittings, but there came on sensory troubles with creepings and numbness in the limbs, and a loss of power which became general and indicated a derangement of the anterior pyramids.

In M. Millard's case there was also an incomplete palsy of the left facial nerve, with a little anæsthesia of the same side,

showing that the trouble extended to the front part of the pons (*protubérance*), a dangerous situation because of its nearness to the nucleus of the vagus which innervates the heart. At present all is well, except the anæsthesia of the limbs, which is improving daily.

In M. Contour's patient the palate palsy was complicated by blindness of the left eye, respiratory troubles, and a slight tendency to faint. These troubles, though slight, alarmed me seriously, for they seemed to show that the diphtheritic poison was gaining on the centres which control respiration and circulation, and which if attacked more seriously would threaten life. Recovery was complete.

The concurrence of the foregoing symptoms seems to me to justify the name which I have given to this new form of diphtheritic palsy, which could not be confounded with any other form of paralysis.

THE ACTION OF THE PALATINE MUSCLES.

I propose to relate shortly some experiments which I have made on the individual action of many of the palate muscles, for the better comprehension of the functional troubles caused by their paralysis, and the distinctive signs of these palsies. I shall then give certain facts which show the value of treating these palsies by faradism.

(My experiments were made upon patients in whom the motion and sensibility of the palate were extinct, contact causing no reflex contraction.)

A. The *palato-pharyngeus* (pharyngo-staphylin) forms the posterior pillar of the fauces, and has a triple action; it approaches its fellow like a curtain, leaving a gap of about a centimetre between them; it pulls down the soft palate and at the same time pulls up the pharynx.

[These actions, which have been doubted by some, have been demonstrated by placing a rheophore on each of the muscles at the level of the isthmus of the fauces.] . . . .

B. The *azygos uvulæ* (palato-staphylin) does not merely shorten the uvula; it holds it up forcibly (*il la redresse avec force*), while it carries it horizontally from before backwards. This fact was very evident in a patient upon whom I experimented

in the presence of M. Martin-Magron (because the uvula was long and a palsy of sense and motion in the entire region enabled me to excite the muscles without producing reflex movements.) The uvula was seen to shorten, and then, in moving from before back, the tip was seen to touch the posterior pharyngeal wall. When I tried to pull it down with a hook I felt it resist strongly. The uvula in contracting under the influence of electricity involves the middle part of the soft palate.

C. The *levatores palati* (*péristaphylin interne*) raise the soft palate by acting chiefly on its middle part, so that the membrane forms a curve with its concavity downwards. When these muscles are contracted to their full extent the highest part of the vault formed by the soft palate is about a centimetre above the horizontal direction, so that it does not then close the hinder opening of the nasal fossa, although it lessens the size of it.

By the light of these facts let us now examine the mechanism of the second period of swallowing, *i.e.*, what takes place after the morsel passes the isthmus. The levatores raise the soft palate, giving it the form of a vault, with its summit about a centimetre above the edge of the hard palate. The size of the pharynx is thus increased for the reception of the morsel. . .

The superior constrictor of the pharynx ought to contract at the same moment as the levatores palati, otherwise the morsel would pass upwards to the nasal fossæ, for I have shown that during the contraction of the levatores *per se*, a tolerable space is left between the edge of the soft palate and the posterior pharyngeal wall, but when the superior constrictor and the levatores act together the posterior pharyngeal wall is brought in contact with the free edge of the soft palate.

For the proper understanding of this action of the superior constrictor it must be remembered that its front upper edge is attached to the lower part of the internal pterygoid plate. It is especially at this level, which is above the soft palate, that I have felt with my fingers the curved fibres of the superior constrictor become tightly stretched like a straight cord during the contraction of the muscle.

The pulling downward of the soft palate immediately succeeds the action I have just described. This movement is brought about by the tensor palati (peristaphylin externe) and then by the

palato-pharyngeus (pharyngo-staphylin). The tensor palati in stretching the palate transversely, and in pulling it down to the level of the hook of the internal pterygoid plate, gives it a horizontal direction; this lowering movement is continued or simultaneously assisted by the more powerful action of the palato-pharyngeus. I have shown above that the palato-pharyngeus in contracting strongly depresses the soft palate so as to give it an oblique direction from above down, and from before back. Thus the soft palate, which till now was thought to be passive during swallowing, is on the contrary active till the morsel is driven down towards the gullet.

But the morsel pressed upon in front by the base of the tongue, behind by the constrictor of the pharynx, and above by the depressed soft palate, might yet escape between the free edge of the palate and the back of the pharynx. This is happily prevented by the curtain-like movement of the posterior pillars of the fauces caused by the contraction of the palato-pharyngei so as to divide the pharynx into two parts—an upper, communicating with the nasal fossæ, and a lower, which holds the morsel. The pillars, however, do not completely meet, but leave a space of about a centimetre between them, occupied by the uvula, through which part of the morsel might yet pass. Having, in fact, caused the palato-pharyngei and superior constrictor to contract together, I could easily pass a caoutchouc sound, several millimetres thick, between the relaxed uvula and the back of the pharynx. But if I then caused a contraction of the azygos uvulæ, the uvula, in becoming shorter and forcibly stiffened, touched the back of the pharynx, which was itself put upon the stretch by the superior constrictor, and prevented the passage of the sound. These experiments seem to me to prove that the azygos uvulæ takes an active part in the second period of the act of swallowing by completely closing the free space which still exists between the posterior pillars of the fauces during the contraction of the palato-pharyngei.

Application of the above facts.—The ideas which I have just advanced make the study of partial palate palsies singularly easy, by letting us know exactly what should be the changes in form of this membrane and its pillars in action and at rest, and what are the functional troubles resulting from paralyses. I shall give a rapid survey of the palate palsies, and we shall see

as we progress whether all my ideas have been confirmed by pathology.

Partial palsy of the azygos uvulæ (palato-staphylin). — In one-sided palsy of the azygos uvulæ the uvula ought to bend towards the sound side. This is caused by the preponderance of the tonic force of the healthy muscle during rest, and it is necessarily increased when the sound muscle is made to contract. Pathological observation confirms these deductions from a knowledge of the healthy action of the muscle. It is not possible to mistake a one-sided palsy of this muscle as it occurs in certain palsies of the seventh, when the uvula always bends to one side when at rest. A palsy affecting the muscles on both sides is not so easy to detect, because in this condition the uvula is merely lengthened when at rest. It is only by tickling it, and noting its immobility, that we recognise that the muscles which pull it up are paralysed. One-sided palsy of the uvula is not followed by appreciable difficulty in swallowing or speaking, although a palsy of the azygos uvulæ on both sides almost always causes a lasting bother from the contact of the uvula with the base of the tongue, which causes incessant swallowings, as if there were a foreign body in the pharynx. On carefully questioning these patients we learn that occasionally fluids pass through the nose, and that they have a slight nasal twang. The patients complain most of the falling down of the uvula, and to relieve them of this trouble we go so far as to cut it off. In similar cases I have tried the effect of local faradisation, which has always cured the trouble by restoring the tonic force of the azygos uvulæ and giving it its normal contractile power.

Paralysis of the LEVATORES *and* TENSORES *palati*.—Palsy of the levator palati (*peristaphylin interne*), which is the most powerful raiser of the palate, ought to cause a depression of it during rest, but this depression is slight if the tensors are sound. If, in these cases, we excite the contraction of the muscles by tickling the uvula, the palate does not form a vault, with the concavity downwards, but is merely stretched transversely by the tensors, while its posterior edge is a little depressed by the palato-pharyngei.

Such ought to be the signs of a localised palsy of the tensors of the palate, but I cannot with certainty say that I have ever seen these muscles paralysed alone. I have merely presumptive

evidence of it. Thus—1. I have seen incomplete palsies of the palate in which, on tickling the uvula, the palato-pharyngei and the palato-glossi contracted together, while the palate remained completely depressed. 2. I have seen other cases in which a similar excitement of the uvula caused, besides a contraction of the palato-pharyngei, a little raising and horizontal stretching of the palate, doubtless caused by the tensors, but no raising in excess of this, and no vaulting with the concavity downwards. Was I deceived in thinking that in this case the levatores palati were alone paralysed? In this last case fluids returned through the nose and the voice was nasal; but these troubles were much more pronounced when the complete depression of the palate announced the paralysis of the *levatores* and *tensores palati* (*peristaphylins internes et externes*).

Paralysis of the palato-pharyngei (*pharyngo-staphylins*).—The lessening or loss of the tonic force of the palato-pharyngei of necessity enlarges the curve described by the posterior pillars of the fauces, and tends to diminish or abolish the prominence of these pillars. The mechanism of this change in the form of the faucial isthmus is explained by our knowledge of the action of these muscles.

By provoking a reflex contraction of the palato-pharyngei, the signs of this paralysis are made plain. For their curtain-like movement no longer takes place, or is incomplete or limited to one side.

Once only I believe I have seen a paralysis of the palato-pharyngei alone, in a case where the other muscles of the palate had recovered their movement by faradisation. The posterior pillars and their curves were scarcely to be seen; tickling the palate caused it to be stretched and raised, and the uvula to be shortened and moved back; but the posterior pillars remained widely separated from each other and failed to form by their above described curtain-like movement a partition between the mouth and nose. The voice was not nasal, but swallowing, especially of drinks, was awkward. Fluids returned through the nose, but to a much less extent than when all the palate muscles are palsied.

Therapeutic Action of Faradisation.—Localised faradisation usually cures, quickly enough, simple palate palsies which are not symptomatic of *glosso-labio-laryngeal paralysis*. I have

applied faradisation to many cases of diphtherial paralysis, and although they had resisted (one of them for eight months) all previous treatment, they were all cured in from fifteen to thirty sittings. . . . . I advise the use of the current of the primary coil with slow intermissions.

. . . . . .

Nervous Aphonia.

[Two cases of nervous aphonia are recorded in which, after the failure of various modes of treatment (leeches, blisters, purgatives, cauterisation, gargles, and Mont-Dore waters), a cure was affected by localised faradisation. The faradisation was applied by placing both the rheophores externally over the laryngeal region, or by placing one externally and introducing the other into the pharynx. "By directing the end of the rheophore over the sides of the inferior constrictor, it is possible to reach the *recurrent laryngeal* nerve which animates the whole of the intrinsic laryngeal muscles."]

CHAPTER XXI.

TROUBLES OF SENSIBILITY AND OF THE SENSES TREATED BY ELECTRISATION.*

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1. *Neuralgia. A severe and instantaneous pain artificially produced on some part of the skin may greatly modify, and even cure, neuralgia.*

[This proposition is considered to be proved by the success of many modes of treatment. Faradisation is a very effectual means of producing the necessary effect upon the skin.]

Sciatica is that form of neuralgia in which I have most often had the chance of trying the therapeutic use of faradisation of the skin. . . . Very few sciatic neuralgias do not at once yield to its influence, no matter on what point of the skin we act, but in order that the good influence may be felt the impression must be lively and sudden. It is not uncommon to meet with phlegmatic patients on whom the most intense currents only cause a slight sensation. In these, skin faradisation has no influence on sciatica, and it becomes necessary to act upon some very sensitive organ. Thus, having placed the rheophore on the root of the helix (*la racine de l'hélix*) in many patients without being able to produce a lively sensation, and consequently to modify the neuralgia, I have seen it immediately disappear by faradisation of the septum naris (*sous-cloison nasale*). (Nothing can compare with the pain caused by the stimulation of this region. We should, therefore, practise it with care, and only in extreme cases.)

This, of course, does not refer to sciaticas which have their source in a purely dynamic trouble (*dans un trouble purement dynamique*), nor to those sciatic pains which are due to inflammation or organic change in the nerve, such as cancer, or the compression of the nerve by a tumour. These latter troubles cannot be called neuralgias, and it would be absurd to expect to cure them by faradising the skin.

* From *L'Electrisation Localisée*, 3rd ed., pp. 798—826.

[Duchenne recommends that faradisation should be applied in sciatica, *loco dolenti*, and that the skin should be first powdered to prevent the penetration of the current. After the first application the pain is usually relieved, but one sitting is seldom sufficient for a cure. This treatment, which Duchenne has found so useful in sciatica, is, he says, equally applicable to all forms of neuralgia.]

. . . . . .

Case No. 83.—In 1860 M. X. was sent to me by Professor Nelaton. He had suffered from neuralgia of the sole of the left foot for two years, which he attributed to a tight boot. The "painful spot" (*point douloureux*) was over the sub-metatarsal prominence ("*saillie sous-metatarsienne*"), so that during standing and walking the pressure of the ground caused intolerable pain, which radiated to the sole of the foot. To avoid this pain he carried his foot in the attitude of "*varus*," and walked, with a limp, on the outer side of it. The neuralgia had resisted a variety of treatment. Velpeau had caused him to keep his foot horizontal for a whole year. M. Nelaton had ordered an apparatus to enable him to walk. Encouraged by success in other cases of neuralgia, I tried the effect of faradisation of the skin. After some minutes of very powerful faradisation the painful spot disappeared, and the patient was able to put his foot to the ground and to walk without the least pain, which he had not been able to do for a year. After walking for three hours the pain re-appeared, but less severely. A second application on the morrow caused the pain to disappear permanently. The contractions which had taken place in order to avoid placing the foot flat on the ground then disappeared, and the uses of his foot became normal.

. . . . . .

Nothing is more difficult to foretell than the effect of faradisation on neuralgias. Thus one case, apparently simple and recent, is not at all affected by it, while another, on the contrary, after resisting for years every method of treatment will yield as by enchantment to faradisation of the skin. I have, however, remarked that those cases upon which faradisation has produced an immediate effect have often been cured, whereas most of those which have not been influenced by the first application have not been appreciably modified, and have not yielded to this mode of treatment. I have found this a sign of some value in forming

a prognosis, and it has often put me in the way of finding an organic lesion or inflammation of the nerve which was the cause of the neuralgia.

Case No. 84.—My lamented master, Chomel, who had suffered from obstinate sciatica for two years, thought that faradisation might relieve him. When I was called to him I shared this hope, all the more so because at that time I had been curing several obstinate cases of neuralgia by faradisation of the skin. I was soon undeceived, for the treatment which I at once applied was followed by no relief. This was an ill omen, nevertheless I hoped that a second, third, or fourth application might be more useful, but I was deceived. This complete failure of faradisation, even to modify the pain, made me doubt the nature of the lesion of the sciatica, and these doubts I expressed to my learned *confrères* who were in attendance. A dozen applications having been made without any result, a rectal examination was made, and a malignant tumour was found in the pelvis on the side of the sciatica, which was the cause of the pains felt along the course of the sciatic nerve, and eventually proved fatal.

[*Tic douloureux.*—The so-called epileptiform neuralgia of the fifth yields to treatment by faradisation of the skin only with great difficulty and occasionally. Duchenne records two severe cases which were cured by this method of treatment, but adds: "I am obliged, alas! to counterbalance the marvellous cures just mentioned by saying that in my experience such cures are rare, and that there is no means of foreseeing the result of treatment."] . . .

Paralyses following neuralgia.—I may here state my opinion on the nature of the paralyses which we sometimes see after certain painful affections styled neuralgia. It is briefly this: "In all paralyses which I have observed following so-called neuralgic pains in mixed nerves, I have established the fact that electro-muscular contractility is lost or lessened."

This is one of the signs of an anatomical lesion of motor nerves, as is shown in the chapter devoted to paralyses of mixed nerves. Consequently it is not possible for me to admit that a paralysis can be produced by a simple neuralgia. I have long held this opinion, and it is expressed in my first edition of the *Electrisation Localisée.* I said, and I still believe, that these

would be neuralgias are due to some organic change in the nerves. . . .

[*Angina pectoris.*—Duchenne has found that faradisation of the skin of the nipples and front of the chest has had the effect of checking the attacks of angina in some few cases. His most remarkable case was the following, of which we give an abstract:—

Case No. 85.—Pérone, a currier, æt. 50, was seized with his first attack of angina on November 29th, 1852. On April 28th, 1853, he came to consult Duchenne. "To come from Belleville to my consulting-room Pérone was obliged to take a carriage; he could not come up the stairs to my room without stopping at each step, feeling a tightness of the chest, &c. After a quarter of an hour's rest he was perfectly calm. Auscultation and percussion revealed nothing abnormal in the lungs, heart, or vessels, and the pulse was natural. Pressure on the chest at various points caused no pain."

"I then asked him to provoke a fit of angina. It was only necessary to stoop down as if to seize something, when the following series of phenomena developed:—Acute deep burning pain, with a feeling of tightness at the level of the upper part of the sternum, radiating to the left upper limb, and following the back of the arm and outer part of the forearm to terminate in the index. There was numbness and creeping of the limb. The two hands were kept folded on the upper part of the chest, as if to soothe his pain. The head was bent forward, and the shoulders raised and pushed forwards by the contraction of the trapezii and pectorales. When he tried to straighten himself or depress his shoulders, the pain increased. I asked him to walk, but he could not move two steps without sitting down, on account of the increase of pain. Respirations short and hurried, heart's action violent, pulse frequent, face red and injected, eyes wide open, body covered with abundant sticky (*visqueuse*) sweat, expression of extreme anxiety. The sounds of the heart and lungs were, meanwhile, quite natural. The heart was of normal size, and percussion revealed nothing wrong in the chest. When the patient tried to talk his words were clipped, and phonation, which was difficult and feeble, increased the pain. Movement of thoracic walls normal. No pains round the base of the thorax. In eight or ten minutes the symptoms began to subside."

This patient was relieved instantaneously by faradisation of

the nipples and sternal region, and attacks provoked at will could be suppressed at will by faradism. A fortnight after seeing Duchenne for the first time, Pérone returned to his work, and had had no further attack for at least a year afterwards.

Caution must be exercised in treating cases of angina with electricity, because patients sometimes die in an attack, and death may in such case be attributed to the treatment. Duchenne mentions the case of a general who died as he was entering the consulting-room, and before any application of electricity had (luckily) been made.]

"Perhaps," I wrote in my first edition (1855), "it will be said that I have established the good influence of electrisation of the skin in angina only by two cases. Faithful to the principles which have always guided my investigations, I ought, no doubt, to have waited before publishing them, till time and fresh experiment had given them additional value, if angina had not been so rare a disease." . . . Since I wrote this I have obtained, in my civil practice, results similar to the preceding, and which confirm the value of faradisation of the skin in cases of angina. Dr. Boullet has applied it successfully in many cases of this kind of neurosis, which he has communicated to the Academy of Sciences, and M. Ed. Becquerel has also communicated to the Academy (February, 1869) many cases of cure of angina obtained by the same measures.

I have also made some researches on the influence of faradisation of the skin in nervous asthma; visceral pains; and in a case of stabbing pain in the skin, appearing now in one spot, now in another. I have noticed, in these cases, that faradisation, applied to the painful spot at the moment of the attack of pain, has succeeded after all other remedies had been exhausted. I do not give these cases, interesting though they be, because they are but isolated facts which will encourage me to repeat my experiments, but which are too few to allow of any conclusions being drawn from them.

[Duchenne has found local faradisation of use in the treatment of hysterical anæsthesia, hysterical hyperæsthesia, and hyperæsthesia of the muscles, as well as in certain affections of the special senses (*e.g.*, paralyses of taste, smell, and vision) of "hysterical" origin.]

CHAPTER XXII.

NERVOUS DEAFNESS.*

The title of this section obliges me to say before beginning the subject that I have no taste for purely therapeutic experiments. It was with regret that I saw myself engaged in the electro-therapeutic experiments which I am about to detail, and a word of explanation is necessary. One day I conceived the idea of investigating the functions of the chorda tympani, by stimulating it through the external auditory meatus. My own ears and those of my friends not being sufficient for my experiments, I borrowed the ears of deaf gratuitous patients, to whom I held out the inducement of possible improvement. By chance many of these patients were cured or bettered. This is how I was led to be a curer through my physiological curiosity. These cures were quickly known, and then deaf and deaf-mutes were sent to me in numbers, and whether I would or no, I was obliged to continue these empirical experiments. These results seemed to me sufficiently important to form the subject of a monograph which I published in 1851. The facts upon which this monograph was based are incontestable and carefully observed, and they have been confirmed by fresh observations performed in public. The protestations of certain specialists who still deny the power of electric stimulation of the organ of hearing in nervous deafness compel me to briefly recall these facts. . . . .

Mode of procedure.—The head is so placed that the external auditory meatus is perpendicular. The meatus is then half-filled with tepid water, and into this is plunged a metallic wire, taking care not to touch the walls of the meatus nor the membrana tympani. To avoid this contact, which may be accidentally produced by the slightest movement of the head, M. Charrière has made for me a special rheophore, in which the

* From *L'Electrisation Localisée*, pp. 826—852.

wire is isolated by an ivory covering, and is prevented from touching the membrana tympani. Having waited till the buzzing caused by the pressure of the water on the membrana tympani has disappeared, the ear-rheophore is connected with one of the conductors of an induction apparatus, and the circuit is closed by placing another moist rheophore on the mastoid process, and connecting with the second conductor.

[The induction apparatus used for this purpose must be capable of very delicate graduation.]

. . . . . .

Electro-physiological Phenomena.

1. The rheophore having been placed in my own external auditory meatus (previously half-filled with water), and the apparatus being at its minimum, I perceived, on the instant that the intermission of the current took place, a little *dry parchment-like sound*, a crackling which I referred to the bottom of the external auditory meatus. When the intermissions were very rapid the sound resembled a crepitation, or the noise produced by the wings of a fly flying between a window-pane and the blind. The intensity of these sounds increased with the force of the current.

2. To the auditory phenomena was added a sense of tickling in the bottom of the ear, proportional to the strength of the current, and absolutely limited to the point at which the sound seemed to originate.

3. After a certain time, and with a certain degree of tension of the current, I felt very plainly a *tickling in the right side of my tongue* at the junction of the middle and posterior thirds. As the strength of the current increased, the tickling reached the point of the tongue, where I then felt *a numbness and a disagreeable pricking* which was not actually painful. This experiment is often followed by a numbness, and sometimes by an over-sensitiveness of the two front thirds of the edge of the tongue, which persists a considerable time.

4. It seemed also as if my tongue were dry and rough (*râpeuse*) on the side operated upon.

Such were the phenomena which first attracted my attention, and which appeared almost in the order I have indicated in the patients who were submitted to this experiment.

5. I must mention a very important phenomenon which, often enough, appears when the stimulation is sufficiently energetic, viz., the production of a *peculiar taste*. It was the last phenomenon to attract my attention, because it is masked by the tickling and pricking which accompanies it. It would pass unobserved if attention were not directed to it. Although the *taste* is feeble, it can be recognised to be of a *metallic kind*.

6. Finally, some patients perceived with each intermission a luminous sensation (*le phosphène electrique*) on the side stimulated.

7. I reserve the examination of the effects of the stimulation of the chorda tympani on the secretion of the saliva in man.

[*Analysis of the above phenomena.*—The peculiar sound is caused by the shaking (*ébranlement*) of the deep parts of the ear (ossicula, membrana tympani, fenestra ovalis). It might be supposed that this sound is caused by the direct stimulation of the auditory nerve. But it is sufficient to refer to the anatomical conditions of the region to see that the nerve, enclosed in the petrous bone, is incapable of direct stimulation. In fact the membrana tympani is only joined to the internal wall of the tympanic cavity (*la caisse*), or the membrane of the fenestra ovalis, from which it is distant 4 or 5 millimetres, by the chain of ossicles, which are bad conductors of the electric current. A much more intense current than that employed would be necessary, in my opinion, to reach the internal ear, and even then it would have to be proved that such stimulation could produce a sound independently of all vibratory movement.

I have proved by experiment in animals that the vibration of the chain of bones is caused by the contraction of one or more of the muscles of the ossicles. It would be hard to believe that this vibration should not equally be produced by faradising the ear of man, for the external muscle of the malleus, which is situated in the upper part of the auditory canal, is in contact with the liquid into which the rheophore is plunged.

The existence of this muscle is not universally admitted, but is not the constant presence of the short apophysis of the malleus, which is evidently meant for the reception of the tendon of the external muscle, sufficient to show that the absence of this muscle is merely an anomaly? It is possible that the internal and anterior muscles of the malleus are also

stimulated by the current, but it is enough to show that the chain of bones and the membranes to which they are joined are set vibrating. The movement of the fenestra ovalis agitates the fluid of the labyrinth and, consequently, the acoustic nerve.

The common and gustatory sensations produced in the tongue are explained by the relations of the chorda tympani to the membrana tympani. The chorda tympani separates from the seventh nerve just before its exit from the stylo-mastoid foramen, and travels by a special canal towards the tympanic cavity, into which it penetrates by a hole on a level with the back end of the transverse diameter of the frame of the membrana tympani. It touches the membrane before passing the vertical branch of the *incus* and the handle of the *malleus*. Finally, after its exit from the cavity (*la caisse*) by the fissure of Glaser, it goes to join the lingual nerve with which it ramifies in the two front thirds of the tongue. It is therefore easy to see how, in our experiment, the chorda tympani is stimulated.

It is this stimulation of the chorda tympani which causes the numbness and pricking, &c., of the side of the tongue; for this sensation occurs exactly at the point where the nerve sinks into the tongue, after joining with the lingual.

As for the nerve of Jacobson, the branch of the glosso-pharyngeal which constitutes the tympanic plexus, and which creeps along the posterior wall of the tympanic cavity and the great and little petrosal nerves, it would probably require a very intense current to reach them, such as could not be applied without danger. And besides, if the current reached these nerves, the excitement would be evident elsewhere than in the tongue, and the velum palati would be seen to move, which is not the case in these experiments. . .

From various experiments and from pathological experiences Duchenne concludes thus: "*The chorda tympani presides over the general sensibility, and the gustatory sensibility of the front two-thirds of the tongue and the integrity of this nerve is necessary for the complete exercise of these functions.*"

. . . . . .

The influence of stimulation of the chorda tympani on the sub-maxillary gland.—M. Claude Bernard has shown that section of the chorda tympani dries up the secretion of this gland.

My experiments, although repeated a hundred times, had not made me even suspect the existence of this fact. But when I knew of Bernard's important discovery, I noticed that, by my process of faradising the chorda tympani in man, salivation is sometimes increased, but is always preceded by a feeling of dryness on the side of the tongue excited. (This I had already noticed in my memoir of 1851.) Yet the salivation—which rarely occurred—was never sufficient to attract the attention of the patient. This is due no doubt to the fact that the stimulation of the chorda tympani was too feeble in the experiments I made on man. . . .

"The salivary secretion," says Bernard, "is explained by a reflex act in which, as in all similar cases, a sensory and a motor nerve take part." The sensory nerve which causes the motor reaction on the sub-maxillary gland is, according to this learned physiologist, the lingual, while the chorda tympani is purely motor, a power which it derives from the nerve of Wrisberg, which constitutes a sympathetic root of origin. . . .

Thus we can no longer say with the late Professor Bérard that "the chorda tympani is an enigma to be solved by the sagacity of physiologists," for, in fact, electro-physiological experiments in man and animals, mutually complementary, show—

1. That the chorda tympani presides over common sensibility and taste in the front two-thirds of the tongue.

2. That it has, probably by reflex influence, an effect, probably motor, on the sub-maxillary gland, without which it cannot secrete.

Therapeutic deductions. . . I knew that by electrifying the chorda tympani effects were produced on the general sensibility and taste in the tongue, and that the secretion of the sub-maxillary gland was stimulated. But what influence could such a stimulation produce on the hearing?

The close connection between the chorda tympani and the acoustic nerve has been shown by microscopic research. Transverse sections of the pons made at the level of the true root of the seventh pair show that there is a branch which detaches itself from the intra-bulbar root of this nerve, and courses from within out to join the internal border of the true root of the eighth pair, and thence passes from behind forwards in

the internal nucleus of this nerve, where it ramifies. This anatomical disposition is clearly shown in Lockhart Clarke's Atlas, and in fig. 4, pl. 9, of Roudanowsky's *Etudes photographiques sur le système nerveux de l'homme* (Paris, 1870). This plate is from a transverse section of the bulb of a cat, and Roudanowsky proposes to call this connecting branch between the seventh and eighth pair the chorda tympani.

The vibration of the membrane of the fenestra ovalis is mainly due to the motion of the chain of bones caused by the alternate sudden tightening and loosening of the muscles, and the consequent agitation of the fluid of the labyrinth. To these movements I chiefly attribute the powerful therapeutic action on nervous deafness of this mode of electrisation.

Look at the care with which nature has surrounded the acoustic nerve in order to keep it from external harm! Not only is it shut in the petrous bone, away from all outside communication, but it is also immersed in a fluid to check movement. From this we can understand how this nerve, soft and pulpy, should be sensible to the lively agitation of the labyrinthine fluid which usually moves but feebly under the influence of the sound waves which knock against the membrana tympani. I cannot explain in any other way the curative action exercised on the organ of hearing by an electrical excitation apparently so feeble.

Cases showing the curative value of Faradisation of the Muscles of the Ossicles and of the Chorda Tympani.

The cases in which I have successfully tried this mode of electrisation may be classed among the *nervous deafnesses*, *i.e.*, cases of deafness in which during life no organic lesion is found. These cases are numerous enough, and have been carefully collected. They belong to various classes of nervous deafness which I shall rapidly review.

1. *Hysterical deafness.* In the first rank of nervous deafness, and most certainly curable, I shall place deafness belonging to the hysterical state. In a former edition I gave a single case as an example, of which the following is a summary:—

Case No. 86.—Hysterical deafness of many months standing rapidly cured by faradisation of the ossicles and chorda tympani. . . . . This form of deafness is often complicated by noises

in the ear, and it is not always complete. Usually the trouble exists only on the left side like the other sensory and motor troubles. Sometimes, however, it is double, but worse on the left side. . . . About eight cases out of ten are cured by this treatment.

[Faradisation of the external ear, or even of distant parts of the body, is sometimes effectual in these cases, and sometimes any sudden stimulus, such as the forcible injection of water into the ear, will cure the case, but faradisation of the ossicles and chorda tympani in the manner indicated is on the whole the best way of treating them.]

2. *Quinine deafness.* — [Duchenne has cured one case of deafness from quinine by faradisation.]

3. *Deafness following continued fevers, or of unknown cause, dating from ten to twenty years, and resisting all previous treatment.*

[In the previous edition Duchenne has given the history of a few cases in which the recovery by faradisation was remarkably rapid, even when all other methods of treatment had failed.] . . .

Prognostic signs obtained by faradising the ossicles and the chorda tympani. . . .

The diagnosis of nervous deafness is not so simple a matter. We can no longer say with Kramer, "When the absence of all visible change in the external meatus or the middle ear has been established, one may, without fear of deception, diagnose a nervous deafness." There are undoubtedly changes of the internal and middle ear, the diagnosis of which is yet unknown, and which till now have been inevitably confounded with nervous deafness. It is obvious how important it would be in practice to distinguish these cases from each other. This has been felt by M. Philippeaux (de Lyon) (*Bulletin de thérapeutique*, t. liii. p. 456), who first recognised "that there were a certain number of deaf persons, who in spite of their trouble perceived very distinctly, under the influence of electricity, the characteristic pain at the point of the tongue, while in others this sign was completely absent." Having then remarked that he could not obtain either cure or improvement in the latter cases, he concluded therefrom (as was very rational) that in these cases deafness is incurable. "I am," says he, "so little encouraged by the success obtained in those deaf patients who have not, under electric exploration,

given evidence of the characteristic sign which I am trying to bring to light in this memoir, that now I absolutely refuse to treat those cases of old deafness in which the patients do not feel, under electric stimulation, the peculiar sensation which in health is felt at the point of the tongue."

[Duchenne says that the conclusions drawn by Philippeaux from the absence of the lingual sensation go beyond what is logically permissible. He urges that the chorda tympani may be damaged while the acoustic nerve remains sound, and he has known the tongue sensation absent in persons who heard perfectly well, and he records an instance of its absence in a case of facial palsy of "rheumatic origin," although the hearing was perfect. He also alludes to cases of deafness in which the tongue sensation was absent, which were completely cured by faradisation.

This new prognostic sign would have a much greater value if thereto were added an absence of the special noise which is heard at the bottom of the ear, because this noise is caused by the vibration and crackling of the membrana tympani and fenestra ovalis. If this noise is not noticed during the operation, the anatomical or dynamic lesion of the acoustic nerve must be of a serious nature.]

To conclude; from the previous facts and considerations it follows—

1. That hysterical nervous deafness is usually cured by this method of faradisation.

2. That some forms of deafness, following eruptive and continued fevers, may be cured by faradisation, although the cases be old and their resistance to previous modes of treatment may have led them to be regarded as incurable.

3. That the effect of this mode of treatment is probably due to the waves caused in the fluid of the labyrinth by the vibration of the chain of bones and the fenestra ovalis.

4. That electric exploration of the ear furnishes no pathognomonic sign of the incurability of deafness.

Deaf-mutism.—After the facts reproduced above, I related a case of half-cure of a congenital deaf-mute by the same process of faradisation. "This case," I then wrote, "has been rigorously and scientifically observed, and affords every guarantee of honesty and authenticity. It has now been tested by time. The child

became half-deaf, developed speech, and was educated with the help of its hearing. It becomes, then, a duty to publish it, although I shall bring many jeers upon myself by doing so."

[It is admitted that in some cases of congenital deaf-mutism no organic deformity or lesion is discoverable in the ear or brain, and Duchenne protests against the hasty assertion of Ménière (*Gazette Médicale de Paris*, 1848) that such cases depend on a change in the nervous system, and are necessarily incurable. Duchenne, in 1861, stated that out of seven deaf-mutes upon whom he had practised faradisation of the chorda tympani and ossicles, one became half deaf (alluded to above), two improved remarkably as regards their hearing, and four remained unchanged. It would not be rational to treat a congenital deaf-mute without first diagnosing the cause as far as possible. We should, therefore, ascertain—(*a*) If the external meatus and the eustachian tube are in a normal state; and (*b*) if faradisation produces the lingual sensation and the buzzing noise. If the latter phenomena are present we may assume the integrity of the chorda tympani and acoustic nerve.]

Duchenne draws the following conclusions:—

1. There is a kind of deaf-mutism, congenital or otherwise, which is entirely independent of all appreciable anatomical change. No one, therefore, is justified in saying that this kind of deaf-mutism is incurable.

2. Electrical exploration enables us to decide almost with certainty that the deaf-mutism is not caused by organic change.

3. Facts show that nervous deaf-mutism can be improved by faradisation of the chorda tympani and the muscles of the ossicles; but till now this mode of treatment has never produced more than a half-cure.

4. This state of half-deafness obtained in the congenital deaf-mute has a rapid and happy influence on the child's morals and emotions (*sentiments affectifs*), and on its capacity for education. It allows speech to be taught by the aid of hearing.

CHAPTER XXIII.

PARALYSIS OF THE MUSCULAR AND ARTICULAR SENSIBILITY.*

Proofs of the existence of an electro-muscular sensibility.—Mention is often made of "electro-muscular sensibility" in this book. I first tried to show experimentally the existence of this muscular faculty. I next showed its importance in the diagnosis and prognosis of many forms of paralysis whose electro-pathology I have studied. To deny in the present day that muscles are endowed with this sensibility is to doubt a fact firmly based on experiment.

Nevertheless, a German pathologist, Remak, whose heresy concerning Hallerian irritability I have already had to contest, has also denied the existence of electro-muscular sensibility. I feel bound to defend this doctrine.

A single experiment is enough to show that muscle is sensible, viz., that which I made at the Hôtel Dieu by faradising the bare muscle of a wounded man. I said that this patient felt a dull (*sourde*) sensation during the faradisation of his muscular fibres. Remak has concluded from it that muscle is not sensible, as if a "dull sensation" meant "want of sensation."

In truth this way of reading and interpreting an author is enough to drive one to despair! In point of fact I have stated (and proved by facts) that muscular sensation is dull when the intermissions of the current are long, but that the sensation increases as the intermissions become so rapid as to produce tetanus. And Remak has not understood a word of it, and has made me say that the direct excitation of the muscle is always dull and painless (*indolente*), whence he concludes that muscle is not sensible!

In order that no one shall be able in future to interpret in this strange way the experiment which I made upon the naked

* From *L'Electrisation Localisée*, 3rd ed., pp. 760—797.

muscle, I have repeated it publicly at the Hôtel Dieu, under the eyes of M. A. Richard (provisionally in charge of M. Langier's surgical wards), by acting on a portion of the bare pectoralis major in a patient whose skin and subcutaneous nerves had been removed during a surgical operation.

In this case the rheophore was applied to the muscular tissue at a distance from the points of entrance and exit of the nerve. The sensation caused by the contraction was dull when the current was moderate and the intermissions equal to a second; it increased in severity in proportion to the shortening of the intermissions, so that the pain became intense when the intermissions were sufficiently rapid to cause tetanus of the muscle.

Passing to pathological observations we must bear in mind cases of injury to the musculo-spiral nerve. If the wet rheophores are placed on parts of the skin which have preserved their sensibility and over muscles supplied by the damaged nerve, the patient feels nothing; but if, on the other hand, the nerves of the skin are damaged and the nerves of the muscles are sound, sensations are caused by the moist rheophores which can only be attributed to the sensibility of the muscles. These are facts which speak for themselves. . . . .

Nothing is easier to observe, with the help of electro-muscular exploration, than the phenomena which prove the existence of muscular sensibility. My long researches on these facts are well known, and the deductions which I have drawn from them for aid in the diagnosis and prognosis of muscular troubles have been thoroughly appreciated. The accuracy of many of these observations has been established by other observers.

Well, Remak suppresses all my work with a stroke of his pen and says that muscles have no sensibility! And upon what does such an assertion rest? Not upon numerous pathological researches, for he has not made one, nor upon long meditations, but upon a few experiments (of which I have given as an example in the first part of this book that which he made upon the trapezius), experiments upon which no serious discussion is possible.

I cannot help doing prompt justice to many other inaccurate assertions deduced by Remak from his experiments. According to this observer, the contraction of a muscle caused by exciting its nerve is not accompanied by sensation (from this fact he has

concluded that muscles are not sensible!) In this Remak falls into a material error, as can be proved by faradising the nerve-twig of a muscle with a current of moderate intensity, when a fairly strong contraction is produced with a dull painless sensation. If the current is increased and the intermissions are quickened the sensations increase in proportion until they become painful. These sensations may last after the operation in the form of neuralgic pains.

It follows from these observations, taken together, that the muscular nerves, and consequently the muscles, are endowed with sensibility. This is in accordance with anatomy, for muscular nerves are mixed nerves. But this muscular sensibility, whether painful or not, is of a special kind, and difficult to excite except by electric currents. Having denied the existence of muscular sensibility Remak attributes the sensation caused by the direct faradisation of a muscle (by moist rheophores applied to the skin over the muscle) solely to the stimulation of the nerves of the skin.

[Duchenne asserts that the so-called "muscular sense," the "sense of muscular activity," the "sixth sense," described by Charles Bell, is identical with the common muscular sensibility described above. He asks why it is that Bell's work is so little known to French physiologists, and says, "It is because the admirable chapter which treats of the 'muscular sense' is lost in a book which is little known, and in which one would not expect to find it. A lord, who had lost the use of his hand, promised a prize of 30,000 francs to whoever should write the best treatise on the hand. Charles Bell, whose name was already well known in science, sent to this competition a treatise on the mechanism of the hand.* This book, in which there is much comparative anatomy and physiology, adds really nothing to our knowledge of the use of the hand, but there is in it a little chapter (the pearl of the book) which treats of the '*muscular sense*.' This book, illustrated by very many plates

* It is unnecessary to inform the English reader that Sir Charles Bell's monograph entitled "*The Hand: its Mechanism and Vital Endowments as evincing Design*," was one of the Bridgewater Treatises on the Power, Wisdom, and Goodness of God. It was written in 1832. The theory of a muscular sense is fully enunciated in the 9th chapter of this book, but had long been taught previously by Bell. It is difficult to say where Duchenne could have heard the tale of "The lord who had lost his hand," &c.—(Ed.)

designed by the author (who had also a great artistic talent), has not been translated into French, which accounts for its being so little known among us. If I had not consulted it before publishing my researches on the hand I should still have been ignorant of the curious chapter to which I have alluded."]

. . . . . .

The functional troubles attributed by Charles Bell to paralysis of the "muscular sense" are chiefly caused by paralysis of articular sensibility.

I have long blindly believed in the physiological importance in locomotion which Bell ascribed to the muscular sense. The following case first showed me that its importance had been greatly exaggerated :—

Case No. 87.—A young lady, the daughter of a celebrated physician, had been twice stricken within two years with complete hysterical hemiplegia. Movement, and all forms of sensibility (touch, pain, the sensibility of bones and joints) were completely abolished. After resisting all forms of treatment for six months, the first attack yielded to faradisation, and the second attack yielded to the same treatment in a few sittings. Four years later the upper limb and trunk on the left side were smitten with insensibility, while motor power was preserved. I proved the absence of the sensibility of the skin to touch and pain, and of the muscles to electricity and pressure. Yet the patient, even with the eyes shut, had a knowledge of the movements (whether passive or active) of each part of the anæsthetic limb. She appreciated the weight of objects placed in her hand and did not let them drop.

. . . . . .

A careful consideration of this case led me to believe that the knowledge of the attitude and movement of the limbs was due to the preservation of articular sensibility. In fact, each of the movements impressed by turns upon the joints of the anæsthetic limb while the patient's eyes were closed "were perceived at the level of the joints put in movement" (*furent perçus au niveau des articulations mises en mouvement*). The patient was conscious of all the movements which the limb assumed in bed at night, and she referred the sensation of them to the joints. The knowledge of the attitude of this limb was, however, less

clear than on the sound side. The only functional trouble caused by the insensibility of the limb was a great clumsiness, and a sensation of extreme heaviness and fatigue which was constant.

Once upon the scent for functional troubles caused by the loss of articular sensibility, I was not long in finding other cases. I have often demonstrated these phenomena in different hospitals. I have shown that articular insensibility is sometimes partial, and may be limited to one or more of the little joints of the fingers whose movements are no longer perceived. I have on the other hand discovered that, in certain cases of locomotor ataxy, patients who have preserved to a fair extent the sensibility of the skin and muscles of a limb could no longer perceive in the dark the movements given to the joints of the limb, nor the various attitudes given to it.

Since the case mentioned above, I have collected many like it which afford fresh proof of the importance in locomotion of articular sensibility. I have quite recently (while correcting the proofs of these pages) seen a fresh case at the Lourcine Hospital under M. Fournier. This pathologist, who has made interesting investigations on the sensory troubles of secondary syphilis (Alfred Fournier, *de l'Analgésie syphilitique secondaire*, 1869), having invited me to analyse these disorders, I noticed in many of his patients the different forms of sensory paralysis which form the subject of this chapter. The following is a summary of a case showing in the same subject the relative importance of articular and muscular sensibility in locomotion :—

Case No. 88.—L. N., a woman aged 22, not hysterical, admitted August 3rd, 1871, for a chancre on the vulva. About three weeks after infection there appeared with the secondary phenomena sensory troubles, which became general instead of being localised, as usually happens in the seats of election pointed out by Fournier. Thus the limbs had lost the sense of touch and pain, but preserved the sense of temperature; pressure on the muscular masses was not felt, neither was electro-muscular stimulation, yet as long as she could see, locomotion was normal; but as soon as she was deprived of sight she manifested the troubles which have just been described, and which have no relation to those seen in the inco-ordination of locomotor ataxy. If, when she was lying down with her eyes

closed, movements were impressed upon the joints of her legs, she did not feel them, and had no ken of the changes in position. The same experiments repeated on the arms gave different results, and although the sensibility of the muscles and skin was profoundly affected, she perceived the slightest movement communicated to the joints.

In short, the experiments made upon this patient left no doubt in the minds of those present—1. That, in spite of the absence of muscular and cutaneous sensibility in the upper limbs, she perceived the movements impressed upon the joints, and was aware of the attitude of her limbs. Consequently the faculty known as "*muscular sense*" and the "*sense of muscular activity*" has no significance, and is only a myth imagined by Charles Bell. 2. That in the same patient (the muscular and cutaneous sensibility of the legs being in the same condition as that of the arms) the sense of movement of the joints of the legs and of the attitudes impressed upon them was entirely abolished. Consequently the faculty called *muscular sense* or *the sense of muscular activity* belongs in reality to the surfaces of the joints.

The following case shows the absolute independence of the sense of touch and the sense of pain, first discovered by Bean :—

Case No. 89.—I saw with M. Gèry, senior, a female colorist, whose hands were completely deprived of sensibility, but in a different manner on the two sides. The right hand had lost *touch* and preserved *pain*, while on the left side the reverse was the case. On the left side the strongest electrical currents and actual cautery produced not the slightest sensation, although she perceived the slightest rubbing of the skin, and recognised objects given to her without looking at them. On the right side she was sensible to painful stimulation, but had lost muscular sensibility; she did not recognise objects placed in the hand, and if deprived of sight she let them drop. These phenomena of touch and pain quite isolated in each hand and in the same person are very remarkable. The nature of this affection is not known to us. This woman was not hysterical. She had, from some cause unknown, suffered for some years from pain in the forearm. In spite, however, of the loss of muscular sensibility all the movements of the hand and fingers were perfectly normal, and were performed wisely and neatly when she was allowed to look.

General conclusions.

1. The existence of muscular sensibility cannot be doubted. Theoretically admitted by Charles Bell, who proposed to call it "*muscular sense*," it is demonstrated both by electro-muscular exploration and by pathological observation.

2. Muscular sensibility and the muscular sense of Bell (called the "feeling of muscular activity" by Gerdy) are one and the same thing, which is only a form of common sensibility, and has no right to take rank with the special senses.

3. Strict observation of cases collected by myself proves that muscular sensibility has not the importance assigned to it by Ch. Bell; and that troubles in locomotion attributed by him solely to a paralysis of this faculty, were, in reality, mainly caused by a concomitant paralysis of the articular sense.

. . . . . .

Symptoms.—Paralysis of muscular sensibility is usually accompanied by paralysis of voluntary contractility. It is met with, however, among patients who have motor power, sufficiently often to observe the functional troubles which it causes in their movements.

When paralysis of muscular sensibility exists alone, we see that the patient does not perceive the contractions caused by the most intense and most rapid induction currents localised in the muscles, and that he does not, even in the dark, suffer from any apparent trouble when using the limbs, which are the seat of this paralysis. He merely feels a kind of heaviness and great fatigue. With his eyes shut he has knowledge, as usual, of the movements impressed upon his limbs and their attitudes. He appreciates the weight of objects, but with less accuracy than on the sound side.

If, as is usual, the paralysis of muscular sensibility is complicated by loss of touch and pain in the skin, there is added to the above signs a difficulty and clumsiness in muscular actions, but then the movements and different attitudes of the limbs are still perceived. It is not the same, however, when paralysis of joint-sensibility is added to paralysis of muscle-sensibility. Then in the dark, or with closed eyes, there appear great troubles in locomotion, which have been erroneously attributed, and for a long time by myself also, solely to the loss of muscular sensibility. In this condition the patient can move his limbs naturally,

provided he can see them, and he can walk with precision and in time, and can use his upper limbs adroitly for delicate manual work, although the want of touch bothers him, and he cannot appreciate the weight of objects which he holds.

But directly he is deprived of sight he can no longer regulate his steps when walking, and this, as well as the maintenance of his balance when standing, becomes difficult, if not impossible. He no longer has knowledge of the movements of his limbs, even when he moves them himself, and if he keeps them forcibly flexed or extended he does not feel any force with which one may oppose their position, and finally he has no longer any knowledge of the changes in position which one may give them.

These functional troubles are chiefly caused by loss of articular sensibility, for they are seen in cases where this is at its maximum, and the loss of muscle and skin sensibility is incomplete, while, as we have seen, the loss of muscle and skin sensibility does not cause the same troubles.

Diagnosis. . . . This condition is most likely to be confounded with locomotor ataxy.

Before I had studied locomotor ataxy sufficiently deeply I attributed the loss of co-ordination to loss of muscle and joint sensibility, but a comparison of the movements in the two conditions has enabled me to show the inexactness of this explanation. I shall show that such a confusion is not possible when the phenomena are carefully studied. . . .

Co-ordination of movement is composed of two kinds of muscular functions, either of which may be damaged, and thus cause two degrees of co-ordinative trouble.

To the first I give the name of "*harmony of the antagonists*," while the second is constituted by the *instinctive or voluntary muscular associations* which govern all physiological movement. . . .

Harmony and discord of the antagonising muscles.—Contrary to the doctrine of Galen on the inaction of the antagonising muscles during voluntary movements, a doctrine which has spread from age to age to the present time, I have shown experimentally that every voluntary movement is the result of a double nervous excitation, in virtue of which two kinds of muscles having a contrary action (*e.g.*, flexor and extensor) are

put in action at the same time, the one set to cause the movement and the other to check it. Without this kind of co-operation (*solidarité*) of the antagonists the movement inevitably loses its precision and sureness. This must be an elementary principle in mechanics, and it is remarkable that it should have escaped the deeply-thinking author of *De usu Partium*. If, in fact, a little force be applied to a lever, you will see that it is impossible to stop exactly at a given point unless it be checked by a moderate opposing force.

This harmony of antagonising muscles, on which depends the precision and certainty of movements, soon suffers in locomotor ataxy. As soon as it is lessened or lost, the patient loses the instinctive power of regulating the range and extent of his movements, although the association of muscular contractions necessary for physiological movements is still naturally made. Hence, for example, we see, in this discord of antagonising muscles, the patients exaggerate most of the movements of walking, while they are still able to perform all its stages. It is especially in the second stage of walking that they throw the leg abruptly forward without power of holding it in, and thus they exceed the normal length of the step. This power of projection, which they can neither control nor moderate, gives them such an impulse that they dare not walk quick or run, from fear of losing their balance, and they feel, as they say, "pushed onwards by an unseen power." Similar troubles occur in the upper limbs, and we have recorded a case in which the patient, while carrying a glass to his lips, was obliged to watch it carefully in order not to break it against his teeth, because his movements went beyond his will.

I may be allowed, in passing, to draw from what goes before the following physiological deduction:—"If the harmony of antagonists had not been shown by my previous experiences, the pathological facts just analysed would suffice to throw light upon this muscular faculty, which is, so to say, hidden in the natural state."

The union and disunion of muscular acts.—Isolated muscular contractions do not exist in nature; they are only obtained by artificial means, such as local faradisation, for example. If we could produce them at will, one would see that they would often produce deformities and cause accidents.

On the other hand, muscular functions need a greater or less number of simultaneous movements, each of which is the resultant of component forces. These complex muscular unions are, nevertheless, made instinctively and without effort; but if locomotor ataxy, after destroying the harmony of the antagonists, gets worse, the muscular contractions are disassociated, and then the patient soon loses the power of instinctively performing the clever muscular combinations on which depend his power of balance, power of walking, use of the hand, &c.

The symptoms which mark this last co-ordinative trouble (the dissociation of muscular contractions) must be carefully studied afresh, because they have a certain likeness to other pathological movements. But as I cannot clearly analyse these phenomena without comparing them to normal muscular functions I must refer briefly to the mechanism of the latter. And besides, these ideas ought always to be present in the mind of an observer who wishes to study locomotor ataxy. In walking naturally the hind leg executes its half swing from behind forwards round the hip-socket as a centre, not merely like a pendulum in obedience to gravity, but mainly by the contraction of the flexors of the hip. Since the limb in swinging would not clear the ground if it were extended, its three segments are flexed the one on the other by muscular contraction, and not by the mere action of the swinging limb, which the brothers Weber regard as a pendulum composed of three segments of different lengths.

I have already contested the theory of these learned physiologists, who would, in this act, replace vital force by physical force, and I believe I have proved that this theory is in evident contradiction with pathological observation. If it were true, we should not see patients with locomotor ataxy executing disordered movements in the second stage of walking from the fact of the dissociation of their muscular contractions, because according to them the muscles in this second stage fall into disuse. After the second stage of walking, when the lower limb completes its half swing, the foot places itself on the ground, proceeding from the heel to the toes; then the phenomena which constitute the first stage of walking are produced, during which the pelvis, and consequently the whole lower limb of the opposite side, already raised from the ground, is pushed forward. I

need not here describe phenomena concerning which there is a general agreement.

One knows that these complex movements of walking are made instinctively and with admirable precision; but as soon as locomotor ataxy dissociates the muscles which usually act together for the performance of movements, instinct no longer suffices for walking, and the attention must be constantly kept awake. The patient is then no longer able, even by the help of his eyes, to execute with regularity the different stages of walking. Sometimes, in fact, instead of placing his foot quietly and surely on the ground after the second stage, the knee straightens before the limb has finished swinging, and this so suddenly that all the body is shaken by it, and then the foot falls heavily and noisily on the ground. At other times when he wishes to swing the limb forward he cannot flex its segments; the extensors may act instead of the flexors and contract with so much force that the limb remains extended while it is brought forward. If he succeeds in flexing the three segments of the lower limb to make it swing forward, the muscles, which this time have obeyed him, contract in so exaggerated a manner that the rhythm of the walk is interfered with, or the limb is thrown irregularly out or in, or swings sideways instead of moving forward and following the line of the resultant of the component forces which flex the hip in the natural state. As in this stage of the disease the harmony of antagonists is completely lost, these disorderly movements are made without any moderation. Then the forcibly projected limbs break or overturn the objects against which they knock. Finally, the anarchy of the movements reaches such a pitch that standing or walking become absolutely impossible.

The difficulty of remaining standing without swaying, or the impossibility of standing, even when the patient looks at his feet, is perfectly explained by the loss of the instinct and will to unite and balance the powers which keep all those parts of the body which have a tendency to fold on each other extended and within the line of gravity.

A few words on the functional troubles of the hand. I have shown that in most uses of the hand the near phalanges are extended on the metacarpal bones by the extensor communis digitorum, while the two end phalanges are flexed on the first by

the simultaneous contraction of the superficial and deep flexors of the fingers. The opposite movement is brought about by the interossei and lumbricales. Nothing like this is seen when locomotor ataxy has invaded the upper limbs. Then the movements of the fingers are most strange. If the patient wishes to seize an object, the fingers (some extended and stiff, others bent) are seen to move at first in different directions, then to converge with difficulty towards the object, which they have a trouble in taking hold of. The separation of the straightened fingers is also peculiar. This movement is the result of the contraction together of the interrossei and extensor of the fingers. The first produce abduction or adduction, extending the two end phalanges and flexing the near, so that in order to keep the fingers extended on the metacarpal bones, the extensors of the fingers must contract together so as to neutralise the flexion of the near phalanges. One sees how complex are the muscular combinations which are executed, as a rule, so easily, and that the patient stricken with ataxy performs them in the strangest manner, even though he use his eyes. All this is indeed difficult to describe, and I should, I fear, waste the time of my readers by analysing the other pathological movements of the upper limb. For the rest it is enough to have once well observed these disordered movements to be no more deceived by them, and to be able to recognise, as it were, at a distance the disease to which they belong.

Let us now compare the troubles caused by the loss of muscular and articular sense with those of locomotor ataxy.

The preceding physiological considerations, and those which I have already made in reference to loss of muscular and articular sensibility, abridge and throw light upon what I have to say.

Patients who have lost muscular and articular sense generally lose at the same time the sense of touch in the extremities, especially on the soles and palms. Without looking, they are unable to walk or stand, because they do not perceive the resistance of the ground, and take no heed of the movements of their limbs. All other muscular functions are also badly performed in the dark.

But during the day, and with the help of their eyes and by sustained attention, the patients perform these functions well

enough, and *they do not show any of those troubles of motor co-ordination* which characterise locomotor ataxy. Facts of this kind are very common; they are seen in hysterical patients, in rheumatic affections, and in certain neuroses. I could report many cases from my own practice, but I will be content to borrow a case like the preceding from a memoir of Landry (*Arch. Gén. Méd.*, Juillet, 1852), which seems to me to have been perfectly and minutely observed, although wrongly interpreted by its author.

Case No. 90.—Alphonse B., æt. 41, admitted to the Beaujon Hospital, July 2nd, 1851, for paraplegia with incomplete loss of sense of touch and pain. On November 20th, 1851, at the visit of M. Sandras, the patient says that when he tries to walk without looking at his feet, he does not know where he puts them, and is unable to measure their movement, as is evident to all present. I examined this patient afresh, and found his condition to be as follows:—

When the patient was not looking he had no knowledge of any passive movements impressed by me upon his legs. I made him walk supported by two others, and he placed his feet easily enough while he looked at them. When lying down he could bring his feet to any point indicated while he looked at them. But, on the contrary, if he ceased to look, the movements made were in the right direction, but so miscalculated as to overshoot the mark. The patient states that he has no knowledge of the extent of his movements. Electricity causes strong contractions, and he feels the pain of it on the sensitive parts of the limbs, but not the pain of muscular contraction. He has no idea of the extensive movements of the feet produced by electricity. He is able to oppose movements which it is attempted to impress upon his limbs while he is looking at them, but he has no knowledge of the force used. When his eyes are shut, on the contrary, and if he be not warned, the limbs readily yield to any movement impressed upon them, and he has no knowledge of the touch or of the movement of the limb, nor of the effort which I put forth, nor of the pressure of a considerable weight (about 33 lbs.). I should add that the patient, belonging to the middle class of society, is a man of culture, with a sound, well-developed intellect, and that he expresses perfectly all that he feels. This case is one of three reported

by Landry, and is perfectly identical with those published by Charles Bell.

. . . . . .

[As a rule such cases are not to be confounded with cases of locomotor ataxy, but I acknowledge that when troubles of co-ordination exist at the same time with loss of muscular and articular sense, error is possible if the symptoms be not carefully analysed. But when ataxy exists alone, the diagnosis is simple and easy.] . . . . In short it is shown by the facts and considerations given in this section, that the co-ordinative troubles due to want of harmony and unity of motion seen in locomotor ataxy cannot be confounded with the functional troubles caused merely by the loss of muscular and articular sense.

Paralysis of muscular consciousness, or of the aptitude for moving independent of vision (*paralysie de la conscience musculaire, ou de l'aptitude motrice indépendante de la vue*) . . . .

Patients who have only lost skin sensibility have lost the sense of touch, and cannot feel any kind of pain, pricking, burning, or electrisation of the skin; but they feel pressure or pinching of their muscles, and blows on the limbs; they are conscious of passive movements, of the extent of their voluntary movements, and of the resistance opposed to their movements; and they can appreciate weight. The voluntary contractile power of their muscles is normal.

If to the loss of skin sensibility is added the loss of sensibility of the organs covered by the skin (muscles, bones, joints, and nerves) the following additional phenomena are observed. Blows on the limbs are not felt. If the patient cannot see he does not perceive the position of his limbs or the most sudden passive movements, and electric stimulation of his muscles and nerves is not felt, no matter how intense it may be.

But there is another series of signs which seem to me to point to the existence of another muscular faculty necessary for voluntary movement, and which I shall call muscular consciousness (*conscience musculaire*). These signs have an important bearing on physiology. [Duchenne guarded against the possibility of some of his patients having simulated insensibility by submitting them to the proof of strong faradisation, "a test which one can

make more painful than the cautery if necessary," and by making his observations publicly and in the presence of learned *confrères.*] . . . .

Case No. 91.—Towards the end of 1848, a patient, aged about 40, consulted me for pains in his legs of long standing. He had also felt them in his upper limbs, neck, and back. The skin of these regions had been, he said, very sensitive to touch or the rubbing of his shirt. For many months these pains had disappeared, and had been replaced by complete insensibility of his upper limbs. In the feet as well as in the hands he had completely lost the sense of touch. He was able to move, although he walked with great difficulty owing to the insensibility of his feet. *But at night,* he said, *his right arm was paralysed.*

Feeling of every kind was lost in the right arm, and in the left arm and foot sensibility was diminished, but not quite lost. His gait was hesitating, although he was strong and could walk long distances. The power of the upper limbs was preserved and equal on the two sides, but with the right limb he could not distinguish between light and heavy objects. *He gesticulated much when talking to me, but the right arm remained motionless by his side.* When I told him of this he set himself to move it by looking at it, but his gestures were not natural. Then I asked him to give me his right hand while he looked in the opposite direction. To my great astonishment the limb did not move, although the patient thought he had given me his hand; but when he looked at it he gave it to me briskly, and shook my hand violently.

[I was much struck with this case, but lost sight of it, and had almost forgotten it, when a similar one recalled it to my mind.]

Case No. 92.—A woman who suffered from paraplegia in the darkness, and whose legs were completely insensible. Thus she said that "*if she was surprised by night she could not rise from her chair.*" In bed she could not move her legs. But nevertheless she walked well enough in the day-time, and could stand for a long time without being fatigued. I established the fact that she could not move her lower limbs without looking at them.

Electric stimulation of the skin and muscles caused these motor and sensory troubles to disappear quickly enough.

Although I did not at first appreciate the importance of the two cases just mentioned, and which I observed only cursorily, it seemed to me that there must be a condition in which voluntary

movement could not be effected without the aid of sight, and that this condition was perhaps complete anæsthesia of the limbs. I set myself accordingly to observe all the anæsthetic patients which I saw in the hospitals. *I saw a great many in whom sensibility was completely abolished, but who, nevertheless, could move their limbs without looking at them.*

Case No. 93.—Desirée Peletier, æt. 18, of good constitution and lymphatic temperament. Her mother died of paraplegia, aged 40. When 11 years old she had an hysterical attack, which lasted twenty-four hours. At 14, when she first menstruated, she had a second hysterical attack, and from that time she had frequent hysterical fits. In 1852 (aged 15) she was admitted to the Pitié for hysteria and loss of sensation in the upper limbs, and for sixteen months she showed no improvement, but from that time the fits became fewer and milder, and, having had no attack for two months, she was discharged from the hospital on 17th November, 1853. A week later she began a new series of attacks, very severe and long, and recurring every two or three days. *She was very clumsy with the hands, and could not walk without looking at her feet.* One night, during a very severe fit, she fell out of bed on the floor, and was unable to move from her very painful position till the return of daylight enabled her to do so. On December 2, 1853, I examined her in the Charité, where she had been admitted under the care of M. Briquet. I found that she had lost the sense of pain and touch over the whole surface of her body, and that the subcutaneous tissues were equally insensible, except at a spot on the left side of the thorax, where pressure could be felt, and where she sometimes suffered spontaneous pain. The face was as insensible as the rest of the body. Smell was lost, and the sight of the left eye was weak. Her movements were in the same state as those of other patients stricken with absolute insensibility. She had a fair amount of power, but could not moderate her movements, nor judge of weight, nor of the resistance opposed to her. As she could not feel her bed, she often had great frights at the moment of waking, believing herself to be falling, and was not quite happy till she actually saw herself lying in bed.

This patient presented in a high degree the phenomenon which is the subject of this article. *Having so managed that she could*

not see her hand at the moment when I asked her to shut it, the hand remained motionless, although she believed it shut; then, while she was extending and flexing her forearm, she looked another way, and immediately the movement stopped, and the limb remained fixed as if tetanised.

I should add that this patient, whom I was seeing for the first time, was ignorant of what I wanted to prove. I then resolved to submit these phenomena, which I had so often seen of late years, to a public examination, in order that I might be sure of their reality.

I caused what I had seen in Desirée to be established to the satisfaction of those present, and I tried to make the phenomena more striking by varying the experiments.

1. The patient being blindfolded, was asked to flex or extend the elbow, and to open or shut the hand. The limb remained motionless, or there were only a few irregular and limited movements, indicative of the efforts she was making to obey orders. She believed that she had obeyed them, and was astonished and annoyed to find her hand had not moved. Similar experiments were tried with the lower limbs, until it was well established that *in this patient voluntary contraction could not be made without the aid of sight, a symptom indicative of the impotence of a function which I have called muscular consciousness* (*conscience musculaire*).

2. . . .—The following experiment showed that *it was not only necessary for the patient to see, but that, in order to execute a movement, she was obliged to fix her attention on the limb she wished to move.* Having placed the patient's hands so close together that she could see them equally, I asked her to open and shut them both at the same time. The fingers were flexed, but alternately on either side, and it was the same with extension. In spite of every effort she could not contract the same muscles on both sides at once. During the contractions she fixed her gaze alternately upon the hand in movement. . . .

3. Was this condition dependent upon central or peripheral change? [By faradising the skin Duchenne restored the cutaneous sensibility, but the motor power remained unaltered, and therefore he concluded that "*skin sensibility was not able, in this condition, to take the place of sight for the production of voluntary movements.*"]

Next I directed the electric stimulus to the muscles of the forearm and hand, commencing with the right dorsal interossei. The contractility was normal, but Desirée was not conscious of any movement till after three or four minutes. The sensation, which she compared at first to throbbings, soon became almost painful. I then ceased, and asked her to make some movement of her hand (without, be it understood, her seeing it), and *I distinctly saw the fingers execute those movements which belong solely to the interossei.* She made the same movements many times in succession, after I had placed the fingers in different positions. (If it is urged that I was the dupe of her trickery, I would merely reply by calling attention to this experiment, for nobody could suppose that Desirée knew the proper action of these muscles.) I localised the electricity successively on the muscles of the thumb, and then on the extensors and flexors of the fingers and wrist, so that she recovered by turns, and as I willed, all the movements of the hand independently of vision.

I took care to thoroughly moisten the skin, so as not to excite it. In this way the skin remained as insensible as before the muscular stimulation. Thus I could pinch, prick, or burn it without the knowledge of the patient. But if I squeezed the arm or beat it, she had a deep feeling which I could carry to the degree of pain.

I must add that the patient often lost in a fresh attack the ground she had gained on the preceding day, so that I could repeat my experiments daily. These experiments were made in the presence of many observers. I will add that while I was bringing back the sensibility of the left arm, M. Briquet did the same for the left leg with mustard plasters and croton oil. The return of skin sensibility in the lower limbs had no more effect on the power of movement than in the upper, but the stimulation of the muscles had the same effect in the arm and leg.]

Although in this last experiment faradising the muscles restored the power of voluntary contraction independent of sight, and although the recovery of this power was limited to each of the muscles which had been so treated, we may conclude that this faculty of motion, independent of sight, is situated in the muscles, unless we allow that the faradisation, although limited to the muscle, produced, through its nerve, an effect on some

point in the nerve centres. Of these two hypotheses the first seems to me the most probable.

It has been seen that muscular faradisation restored at once the muscular sensibility and the power of motion, independent of sight. It would then at first seem rational to conclude that muscular sensibility (Bell's "muscular sense") and the power possessed by each muscle to contract at will, and independently of sight (*conscience musculaire*, muscular consciousness) are identical, and that the loss of the second is merely due to an extreme degree of the loss of the first. This objection has been made to me, but it is no real objection, as can easily be shown. We must not forget that patients deprived of sensibility (in whom neither blows, fire, faradisation, nor violent passive movements cause the least sensation, and who, with the eyes shut, have not the least knowledge of what is done to them) can nevertheless perform, even with vigour, every kind of movement in the dark. They have merely lost every kind of sensibility (of joints, muscles, and skin). Therefore if their limbs should become paralysed by the mere fact of suspension of sight, it would be irrational, not to say absurd, to attribute this to a greater degree of paralysis of the muscular sensibility.

We must then simply conclude from what precedes that faradisation of the muscles which modified the state of paralysis of muscular and articular sensibility modified at the same time the state of paralysis of that kind of aptitude for movement which I have called muscular consciousness (*conscience musculaire*).

What bitter criticism this name has brought upon me! Let us see if it was necessary or just.

1. Every new fact must have a new name. If not, we can only describe it by definition or circumlocution.

[The existence of the condition of which we are speaking is incontestable. Since my first case, published in 1856, I have seen another at the Charité, published in 1859; a third has been published by Dr. Martin Magron; this case occurred at the Hôtel Dieu, under M. Jobert (de Lamballe) (*Gazette Médicale*, 1859). Two cases have been published by Ch. Lasègue ("De l'anesthésie et de l'ataxie hysterique," *Arch. Gén. de Méd.*, April, 1864, pp. 384, 402). "I have also seen several typical cases at the Hospital du Midi; and M. Fournier has exhibited

these singular functional troubles in three women under his care.]

2. Is the name which I have chosen (*conscience musculaire*) a suitable one? . . . .

To have given the name of "muscular sense" (Bell), or "feeling of muscular activity" to these phenomena would have been to sustain, contrary to all logic, *the identity of those pathological conditions in which movement is quite abolished by loss of sight with those in which movement is always possible without sight.*

If I had called this phenomenon "*motor power independent of sight*" (*aptitude motrice indépendante de la vue*) the name would have been irreproachable. But it was too long, and I preferred that which recalled a theory of the phenomenon. . . .

This faculty then, which appears to reside in the muscle (because when lost it can be recalled by faradising the muscle); which is not the muscular sensibility; which establishes an intimate relation between the muscle and the brain; and which allows the latter to produce movement without the intervention of sight, I believe to be well expressed by the name "*conscience musculaire.*"

But this name is based merely on a theory. If it be not true (which time will show) another must be sought. I hold my own cheaply enough. I merely defend the physiological and pathological fact which I believe I have gained for science.

Conclusions.—1. There seems to exist a kind of sense which resides in the muscle, and serves for the accomplishment of voluntary muscular contraction, and which, in the absence of sight, enlightens the brain as it were, before the latter, stimulated by the will, provokes muscular contraction.

2. We must not confound *muscular consciousness* (*conscience musculaire*), which in the act of voluntary movement seems to precede and determine contraction, with the sensation given by the sense of weight and resistance, which was called "muscular sense" by Charles Bell, and "feeling of muscular activity" by Gerdy. This last is the result or product of muscular contraction; and clinical observation has recently shown me that this sensation is a phenomenon which depends more on articular sensibility than on muscular sensibility.

3. *Muscular consciousness* can exist independently of the sensation of muscular activity.

4. It is necessary for voluntary muscular contraction, and for the cessation of this contraction. Nevertheless the sense of sight is a help to muscular consciousness, and may take its place.

5. The loss of muscular consciousness and of sight must be followed by loss of voluntary movement.

Paralysis of electro-muscular contractility, or the mutual independence of voluntary contractility and electro-muscular contractility.

[This short chapter is devoted to an exposition of the fact that in some cases of paralysis from lead poisoning or damage to a nerve, the power of voluntary contraction may re-appear in a muscle before there is the slightest response of the muscle to the stimulus of faradisation.]

CHAPTER XXIV.

MUSCULAR SPASM AND IMPOTENCE.*

I GIVE the name of functional muscular spasm and impotence to troubles marked either by continued painful or painless contraction, or by clonic contraction or trembling, or lastly by impotence which shows itself only in certain voluntary or instinctive movements, and which is localised in some of the muscles helping in the said movement.

1. *Functional spasms of the head, trunk, and upper limbs. Functional spasm may occur anywhere, or affect a great number of voluntary and instinctive movements.*—Spasm of certain muscles of the hand which has been caused by the abuse of writing, or of a muscular function by efforts made in writing, and which shows itself during the exercise of that function, first received in Germany the name of "Schreibekrampf," which means "writers' cramp." In France it is called "*crampe des écrivains.*" These names, used by all the authors who have written on the subject, prove that the trouble is often localised in the hand, and especially in the muscles used in writing.

Sometimes, in fact, it is one of the muscles presiding over the partial movements of the phalanges which is affected.

Cases No. 94 *and* 95.—I have seen a stockbroker in whom the mid and far phalanges of the index were flexed, while the near phalanx became extended after he had written a few words.

On the other hand, I have seen a War Office clerk in whom the near phalanges of the first and second fingers were flexed, while the mid and far phalanges were extended. My electro-muscular researches teach me that in the first case the trouble was in the flexors and extensor of the index, while in the second case it was situated in the interossei of the index and medius. At other times the supinators and pronators are the muscles affected, as happened in the following case:—

I have seen two cases in which the hand became supinated as

* From *L'Electrisation Localisée*, 3rd ed., pp. 1021—1034.

soon as the patient had written a word, so that the point of the pen looked upwards. Functional spasm does not merely affect the hands of scriveners. It may show itself anywhere, affecting those movements which have been abused. I have seen many examples, of which I will quote a few.

Case No. 96.—In 1855 I treated a tailor whose arm turned violently inwards, by contracture of the subscapularis, as soon as he had made a few stitches. This trouble did not occur with any other exercise.

Case No. 97.—A fencing-master whose humerus of the sword arm rotated inwards, and whose elbow became strongly extended, as soon as he put himself "on guard."

Case No. 98.—A tinman in whom the deltoid and biceps of the hammer-arm contracted painfully as soon as he tried to arrange a slab of metal. This spasm only occurred with this kind of work.

Case No. 99.—A turner, the flexors of whose ankle contracted as soon as the foot was placed on the treadle of the lathe. No difficulty in walking or in other movements.

Case No. 100.—M. Andral was consulted in 1855 by a gentleman from Rouen, whose head turned to the right by the action of the rotators whenever he read, so that he had been obliged to give up his books; he was also troubled with writers' cramp. He had been a great book-worm all his life.

Case No. 101.—A cobbler who suffered from contracture of the right splenius and deltoid, and some of the facial muscles, as soon as he began to work. He was cured by faradisation of the muscles antagonistic to those which were the seat of spasm.

Case No. 102.—In 1855 I was consulted by a scholar who for many years had tired his eyes by deciphering MSS. For the past six months, as soon as he tried to read or look steadily at anything, he suffered as follows: a few seconds after his eyes had rested on any object he saw double, although his sight was good enough as long as his eyes were wandering. It was easy to establish the fact that this trouble was due to spasm of the internal rectus of his left eye, a spasm which subsided as soon as he stared less intently.

Case No. 103.—A student of Strasbourg, preparing for his degree, had worked inordinately. The efforts which he made to conquer sleep caused, he said, a painful tightness across the

temples, forehead, and eyes, so that his work had to be discontinued. Directly he began to read these troubles came upon him, and I noticed that at these times his brows were raised by the contracture of the occipito-frontalis, and his eyelids were closed by contracture of the orbiculares, while his face became flushed and his temporal veins distended. This state of things lasted many years, and was only caused by reading. This young man, whom I was unable to cure, committed suicide in despair.

Case No. 104.—A pavior in whom both sterno-mastoids became contractured during the instinctive contraction of the muscles which keep the head balanced between flexion and extension. The head was flexed by the contracture with extreme force, and the contracture ceased as soon as the head was supported. If he were lying on his back or face, or if the head were supported on the back of an arm-chair, the spasm never occurred.

I have seen a similar condition in a young girl, and Professor Verneuil sent me a lady in 1869 in whom the sterno-mastoid contracted as soon as she stood up. The spasm ceased if the head was supported.

Case No. 105.—The following is a case of expiratory spasm (the most curious I have seen), which seems to me analogous to functional spasm. I give merely a summary of it :—

A country priest, whom I saw in 1859, suffered as follows :—At each inspiration all the right side of his belly was depressed, while the left epigastrium bulged naturally.

. . . . . .

This respiratory trouble was due entirely to spasmodic contraction of the muscles of the right half of the belly, especially the external oblique (*grand oblique*). With each inspiration this muscle was felt to contract, the direction of the contracted fibres being perceptible through the thin skin. The spasm was so violent that the trunk was twisted from right to left with each inspiration. It was a true painful cramp, which continued during the whole period of inspiration. This conflict between the inspiratory and expiratory muscles prevented the enlargement of the epigastrium and base of the thorax, and consequently the distension of the lung. Hence the patient was always in want of breath. There was no fever. It was a neurosis which had resisted treatment for two years. . . .

This clergyman had a mania for playing on the hautboy (*serpent*). He had abused this exercise, and to this he attributed (and I think rightly) his trouble. When he played on his instrument his spasm caused him to emit sounds which amused his congregation, and it was thus that he detected the onset of his disease.

Hereafter we shall see that functional spasm is sometimes situated in the legs.

The facts given above show that functional spasm may affect many regions, and may implicate either voluntary or instinctive movements.

Functional spasm is not always a cramp.—A painful visible contraction of one or more muscles lasting a short time we call a *cramp*. It is true that functional spasm is often a local contracture, which lasts till the patient gives over that movement during which alone it is produced. I have given many examples of this, but often enough there are tremors or momentary clonic contractions more or less strong, which interfere with writing, and only occur during the exercise of that function. Space compels me to give only one example of this.

Case No. 106.—Writers' tremor. M. X., barrister, æt. 26, of nervous, irritable temperament, who wrote about three hours a day, was seized in January, 1860, with tremor of the right hand and difficulty in forming the letters. After persevering for a little time the act of writing became easier, and the letters more legible. The tremor increased so that in seven or eight months he could not write at all. He then had an irresistible tremor in the ring and little finger. He felt the movement begin in the flexors of the forearm, and then invade the hand, so that writing became impossible, and the tremor increased with his efforts. The very thought of writing seemed to favour the tremor, which increased also when he was watched by others. The hand and forearm were the seat of spasmodic movement, and he was obliged to cease working. It reached such a point that he could not write a word, and the attempt caused him the greatest excitement. All means for steadying the hand, compression, friction with chloroform, morphia hypodermically, produced no effect. Absolute rest for four weeks brought no change, and this young man was obliged to learn to write with his left hand. The tremor was caused by no other act than writing.

These spasms are not always painful. Sometimes the pain provoked by function is a temporary neuralgia.

A. Many patients have told me that they suffer no pain and no feeling akin to cramp. But all have felt fatigue in the affected limb when they have tried to continue the movements which brought on the trouble.

B. The following is a case of temporary neuralgia caused by the exercise of function :—

Case No. 107.—Mdlle. R., a pianist of great talent (first prize of the Conservatoire), cannot play for many minutes without feeling acute pain in the right arm and forearm. The pain develops thus: the power of the right hand lessens, then a gradually increasing pain occurs, which apparently follows the course of the median and rises to the shoulder. This compels Mdlle. R. to cease playing, when the pain soon ceases, and does not recur till she begins to play again. This pain is not in the muscles, and I have not seen a single one of them "cramped." Mdlle. R. had tried, she said, to strengthen her hand by playing a piano with a very hard touch. It was after this exercise that her troubles began. They have lasted many years, and have resisted all treatment.

FUNCTIONAL PALSY.

The abuse of certain voluntary movements sometimes causes temporary palsy of one or more of the muscles concerned. The muscles affected are the same as in functional spasm.

Case No. 108.—Canon X., secretary to the Bishop of Nevers, consulted me in 1852 about a writing trouble which interfered with his duties as secretary. He had had no previous trouble with the hand, when in 1851 he began to feel fatigue in the right shoulder, mainly round the scapula, when he was compelled to write for long. The fatigue increased. It was now induced by writing a few lines, and put an end to his correspondence. "The fingers," he said, "guided the pen perfectly, but when the fatigue came on, the hand and forearm, after I had written a few words, seemed nailed to the desk." A little rest enabled Canon X. to write a few lines, and then the functional trouble recurred. I saw these troubles many times when Canon X. wrote in my presence, and I noticed that after writing a few lines it was

impossible for him to make his arm turn from within out, and consequently to move his flexed forearm in the same direction. He was also obliged after every word to drag the paper to the left with his left hand.

Following this case in the last edition were these words, "M. X. was suffering from a form of 'writers' cramp,' which might more properly be called 'functional impotence of writers,' and which in this case was situated in the infra-spinatus muscle."

I have been consulted by many others who after writing a few words suddenly stopped, being unable to move the fingers. They nevertheless suffered from no muscular spasm.

Case No. 109.—A book-keeper, whose adductor pollicis became impotent after having written a few lines, so that the pen fell from his hand. He could only write by holding the pen, American fashion, between the index and medius. Nevertheless the adductor was very useful for everything but writing. After very careful observation of this case I can certainly affirm that there was no spasm.

The existence of functional impotence is then incontestable. It is strange that authors who have specially studied the functional impotence called "writers' cramp" should have misunderstood it. There are other impotences which occur during the performance of other functions, *e.g.*, of the peroneus longus during standing or walking.

Pathology.—Is this trouble peripheral, *i.e.*, limited to the muscles? Do the nerve centres send their normal stimulus to the muscles? Is the excitability of the muscles sometimes increased and sometimes lost or lessened during certain voluntary or instinctive movements?

Or rather is there a point in the nerve centres which, excited or exhausted by constantly repeated movements, sometimes over discharges causing spasm, sometimes sends the nervous stimulus to the muscles irregularly so as to cause tremor and clonic spasm, and sometimes ceases to distribute its nervous force, and this only during the performance of certain functions? The mechanism of this functional trouble which I am trying to explain ought to be found in one of the two latter hypotheses. I admit that I am not actually able to solve this problem, but I lean towards the second hypothesis (that which makes the trouble depend upon some derangement of the nerve centres). How

can we admit that a muscle should be too excitable and become cramped, or act convulsively, or that it should lose its power of responding to nerve stimulation when filling certain functions, while it contracts normally for all other functions? On the other hand there is no difficulty in supposing that the trouble is in the nerve centres. This hypothesis seems to me almost justified by clinical observation. In fact I have seen, among others, two patients, who, not being able to write with the right hand by reason of contracture of the pronator radii teres in the one, and of the muscles of the ball of the thumb in the other, learnt to write with the left hand with considerable ease, but after a certain time they were troubled with spasmodic contracture on the left side. In one of them the pronator radii teres contracted on the left side as it did on the right.

Do not these facts prove that voluntary stimulation often repeated in this or that function not only caused at length a diseased state in a certain point of the cord, but had also extended its action to the near corresponding point on the opposite side of the cord?

We must further admit for the development of this disease, as for all others, a particular predisposition. Numbers of people, in fact, over-write without suffering from "writers' cramp." . . .

Diagnosis.—When an individual has trouble in writing it is easy to diagnose "writers' cramp." But is it spasm or impotence? What muscles are affected? These are the important points to investigate.

We may assert, not only that the form of temporary impotence has not been recognised, but that the exact diagnosis of the finger-muscles involved has not been made. It could not be, because the physiology of phalangeal movements was not known before my electro-physiological researches on the hand.

By the help of the notions which flow from my researches, local diagnosis will be easy enough for the future, and the importance of it will be better appreciated when treatment of the affected muscles is indicated. When functions other than writing are affected the diagnosis is less easy, and the disease has been confounded with troubles of another kind. I shall give presently examples of functional spasm of the sterno-mastoids on both sides and on one. The external characters are the same as those of ordinary wry neck. These troubles, so different

in their nature, may easily be confounded, as happened in these cases. We shall see how diagnosis affects prognosis and treatment. The only distinctive sign which will prevent error is that functional muscular spasm and palsy only occur, as the name which I have given shows, during the exercise of functions.

Prognosis and Treatment.—The prognosis of this muscular trouble is bad, for functional spasm of the hand usually resists every kind of treatment, even faradisation and continuous currents, no matter how applied. In fact, with the exception of functional impotence of the peroneus longus, which I have nearly always cured, I have only had two successful cases out of fifty which I have submitted to localised faradisation in the last twelve years.

Case No. 110 (abstract).—*Functional spasm of the extensor indicis of four or five months standing in a flower girl. Anæsthesia of the hand. Cure by a few faradisations of the damaged muscles.*

It will be noted that in the above case where faradisation triumphed the trouble was complicated by anæsthesia, which I have not seen in any other case. This would seem to place the case in rather a distinct category. . . The want of success of localised faradisation goes to confirm the theory of functional muscular spasm which I have given above, and which attributes the trouble to a central lesion. We can understand then that this stimulation, entirely peripheral and localised, should be incapable of modifying or curing a functional trouble symptomatic of a central lesion.

If, on the other hand, the disease began in the affected muscles it is probable that localised faradisation would have more power over it, and would act as in idiopathic contracture. I have shown indeed that this last affection is usually cured by faradising the antagonists. I reported in the previous edition a case cured in this way, in which a contracture of the left splenius complicated a case of writers' cramp, or which rather had been caused by the efforts which the patient made to write. The faradisation which cured the secondary contracture had no effect on the primary functional spasm of the hand. He tried once to write with the right hand after the cure of the idiopathic contracture of the splenius. The first lines were easy and well enough done, but soon he felt the old stiffness of the hand return

and then the cramp. If he tried to struggle a moment against the difficulty the muscular spasm of the neck began to re-appear. He had the wisdom to give up in time this imprudent attempt, which would infallibly have brought back the secondary contracture of which he is happily cured. He is now engaged in work which only requires an occasional signature.

I have also cured a functional spasm of the sterno-mastoid by provoking and keeping up voluntary contraction of the antagonist muscle by means of an elastic resistance, a kind of gymnastic which I have long thought of trying in functional spasm. The trouble had lasted three years, and had not yielded to six months medication and faradisation of the antagonist muscles. The following is the case :—

Case No. 111.—A young lady, æt. 24, a pianist of Orleans, consulted me in 1855. She had suffered for three years from a torticollis, for which the best known specialists had prescribed in vain. Faradisation of the antagonists combined with mechanical means had been used for six months by M. Bouvier, but without success. M. Bouvier was all the more surprised at his failure because of the success of similar measures with other patients.

I soon saw that this was a *functional* spasm. It only occurred when the patient was erect, and ceased as soon as the head was supported, whether in the recumbent or reclining position. The failure of treatment was then comprehensible.

I experimented with this patient in the same way as I had done with others. When the spasm occurred while standing I supported the nape of the neck with my hand and instantly the spasm ceased. There was this further peculiarity in the case. When erect she could not turn her head to the right, and during the time of her sojourn in M. Bouvier's establishment movement to the right side was very limited, and was brought about only by great efforts. When, however, she tried to overcome the resistance which I offered to the extension of the head she performed the lateral movements of the head with the greatest ease. This was all the more surprising to the patient, because she had been told that the real obstacle to the lateral movement of the head was the deformity of certain joints and their annexes. Our experiment showed not only that the joints were sound, but that voluntary contraction of the antagonists of the contracted muscles was enough to cause the cessation of the spasm.

I made use of my discovery by advising the young lady to practise gymnastic exercises like those which I had prescribed for M. B., and at the same time I employed, as a satisfaction to the patient and her doctor, faradisation of the antagonists of the contracted muscle. A notable improvement occurred in a fortnight. I do not attribute the improvement to the faradisation, which, although properly used, had previously failed. I believe it to have been due to the kind of derivative gymnastic which I prescribed, or perhaps to the two means combined. A few months later M. Bouvier testified to the cure of his old patient.*

I do not pretend that this single case is enough to establish the value of this kind of gymnastic in cases of functional spasm. I give it in order to encourage practitioners to try it in cases of functional spasm of the muscles of the neck.

I should add, finally, that functional spasm of the hand (writers' cramp) has failed to yield to every kind of gymnastic, as well as to localised faradisation. We owe to M. Cazenave, of Bordeaux, the employment of the very clever prothetic apparatus which lessens the trouble caused by writers' cramp. M. Debout—in a work called, *Coup d'œil sur quelques appareils destinés aux malades affectés de paralysies partielles des membres* (*Bulletin Général de Thérapeutique*, 1860)—has given a collection of prothetic apparatus advised, or used, for writers' cramp. Among these will be found figures and descriptions of many machines invented by me for some kinds of functional spasm of the hand.

General Résumé.

Often repeated muscular functions may cause spasm or impotence of one or more of the muscles helping in the function.

Functional spasm at one time (and usually) is marked by continued contraction (contracture), at another time by tremor or clonic spasm. It ceases with the muscular act which causes it. Sometimes painless, often painful. It often comes in the hand muscles, when it has been called "writers' cramp," because it is usually continued and painful, and specially affects writers. But I have seen it in pianists, tailors, cobblers, florists, fencers, &c.

And, further, it may occur in other regions than the hand. Thus I have seen it in the rotators of the humerus, deltoid,

* It is to be regretted that M. Duchenne was not more explicit as to the means employed in this case.

rotators of the head, flexors of the head during standing, muscles of the face, muscles of the eye when reading or staring (causing squinting and double vision), flexors of the ankle (in a turner), in the expiratory muscles during each respiratory movement, and lastly, in the peroneus longus during walking. [Functional spasm and impotence of the peroneus longus is so common, and so misunderstood, that I have given a description of it in a memoir which I have just published in the *Archives Générales de Médecine* (April 1872)]. Muscular impotence is far less common than functional spasm; but (like it) only occurs during the exercise which causes it.

Functional spasm occurs also under the form of tremor during certain movements.

They are usually accompanied by pain, numbness, or a feeling of fatigue in the affected limb.

Faradisation has been of no use for this trouble up to the present time. It has failed, except in functional impotence of the peroneus longus, which has almost always been cured by it. Functional spasm of the muscles of the head may get well under the influence of a gymnastic exercise, which consists in keeping the muscles which antagonise those which are the seat of spasm in a state of continued voluntary contraction, by means of a resisting elastic apparatus.

CHAPTER XXV.

ON FUNCTIONAL IMPOTENCE AND FUNCTIONAL SPASM OF THE PERONEUS LONGUS (*long péronier latéral*).*

THE novelty of the title of this pathological study is doubtless startling. It treats of an affection of the foot analogous to another which I have described as *painful valgus flat foot*, which ought also sometimes to be called *painful valgus hollow foot*, and which was till then misunderstood by pathologists.

The existence of these two kinds of painful valgus was revealed to me by experiment and clinical observation. I discovered that the production of the one or the other depended on opposite states of the peroneus longus, paralysis or contracture. I showed this in my paper before the Surgical Society (1863).

But what, excluding injuries and inflammations of the tarsal joints, were the causes of these two kinds of valgus?

I believe now that I have solved this problem. Thus I had noticed, 1. Youths who had, some time after walking and standing, all the symptoms, sometimes of flat valgus from paralysis, sometimes of hollow valgus from spasm of this muscle; at first painless, but afterwards painful, and with secondary reflex contractures of some other muscles. 2. That all these troubles rapidly ceased during rest, to appear again quickly during exercise. Struck by the resemblance of this functional foot trouble to those professional functional troubles of the hand of which "writers' cramp" is the type, and which at one time take the form of impotence and at another time the form of contracture, I called it *functional impotence and functional spasm of the peroneus longus*, in a paper read before the British Medical Association at Oxford in 1868.

Functional Impotence of the Peroneus Longus.

The peroneus longus is one of the most useful muscles in

* From a pamphlet of 52 8vo. pages, published in 1872.

standing and walking, but is also the first to get tired. Therefore under certain circumstances when called upon to fill these functions it is quickly exhausted, and seems paralysed.

Hence result—1. A special deformity of the foot; 2. Reflex pains and contractures; 3. Retractions which cause a painful valgus flat foot. . . .

The beginning of the difficulty is very insidious, for it causes neither pain nor fatigue in walking.

[There is a change of attitude, and the inner edge of the sole gets worn away; then the feet twist out and the toes turn outwards more than formerly. This change in attitude is very slight at first, but increases and becomes most ungraceful. It seldom causes any anxiety, and only once have I been consulted in this stage since I have known the mechanism of the disease.]

Case No. 112.—In 1862 I was consulted by a little girl, aged 11, who was thought to have a *double flat valgus* foot. It had come on gradually during the past two years. She grew very rapidly when about 9, and a few months later her mother noticed that in standing and walking she wore out the inner part of her shoe-soles only, and that the feet turned out so as to form a kind of tumour on the inside on a level with the ankles. The patient suffered no pain or fatigue in walking or standing, but as her attitude was very ungraceful, she was made to wear shoes with very solid side supports (*contre forts*).

Soon after she was made to wear orthopædic apparatus, which kept her feet straight it is true, but these returned to their old position as soon as the apparatus was removed. Was the deformity a *valgus flat foot?*

On seeing her walk I took it at first sight for a very marked case of valgus flat foot, but on looking at the naked feet when they were off the ground I found their shape and position normal. The plantar arch was well marked, and there was no contracture of any muscle. All the muscles responded normally to electricity. As soon as the child stood, however, the feet resumed their faulty attitude, which became more marked after she had walked a few steps. I then noticed that, seen from behind, the calcaneum was outside the axis of the limb, and that the tendo achillis slanted from above down, and from within out, while at the level of the internal malleolus there was a bulging, and the foot as it rested on the ground was flat. I

thought at the time that the trouble was due to a general muscular atony and laxity of the ligaments, for I had not then the idea of functional impotence of peroneus longus. . . .

The above is a faithful picture of the first stage of the disorder.

The duration of this first stage varies much. It may pass unnoticed, for in most of my cases which were seen in the second or third stage, the parents or the patients recognised the onset of pain as the beginning of the disease, and it is only by questioning that one is able to establish the fact that the pains caused by walking and standing were preceded by the symptoms detailed in the above case.

The *second stage* is marked by tarsal and plantar pain, by reflex contracture of certain muscles, and by difficulty in walking, superadded to the symptoms proper to the first stage. The tarsal pains are at first felt in front of and below the outer malleolus, and later below and in front of the inner malleolus. Sometimes they come in both places at once; they are slight at first, but after long standing or walking they get worse, and soon make walking and standing difficult or impossible. These pains are worst on the outside of the calcaneo-astragaloid joint, whence they radiate forwards and reach the back of the foot. They are lost or lessened during rest, and most quickly when the limb is horizontal. But yet they sometimes come back of themselves, and even during rest, especially if the patient, in spite of the pain, has walked or stood a long time. Pressure over the joints above named increases the pain, and the skin over the painful part is sometimes red and hot, but never swollen or œdematous.

There is sometimes pain in the sole and over the heads of the third and fourth metatarsal bones, increased by the corns which at length develop at these points. These pains are provoked whenever the foot rests on the ground in standing or walking.

Reflex contractures usually occur with the pains in the muscles whose tendons run near the most painful joints, generally in the peroneus brevis and extensor longus digitorum, extending sometimes to the tibialis anticus.

They are due to reflex action, like the contractures caused by inflammation of certain joints (coxalgia, cervical arthritis, and tarsal arthritis, of late wrongly called *tarsalgia*). These

contractures, which are brought on by standing and walking, disappear during rest. Later they increase, as do the pains, and become almost continuous. Under the influence of these contractures the valgus flat foot, which was *passively* caused by the planting of the foot, becomes *active*, continuous, and more and more pronounced.

The contractures increase the pain by causing pressure on certain joints and by stretching certain ligaments, and they often cause a feeling of fatigue in the leg and foot. They do not seem painful in themselves, but the least stretching of the muscle in an opposite direction awakens a very lively pain along the contractured muscle.

By gradually increased stretching the contractures may be overcome, and the movements then given to the foot, in a direction opposed to the contracted muscle, cease to be painful.

When the foot has been long kept (for many weeks or months) in the position of valgus by these contractures, the surfaces of the joints get worn, and while some ligaments are lengthened others shorten, so that the foot keeps its valgus attitude in spite of the cessation of the contractures.

Stage of retraction.—Since the contractures disappear during rest there is complete muscular relaxation in the intervals of contracture. This is seen in the second stage, which I have just described. But muscles which are constantly contracted are not slow to shorten, and they only partly yield when they are sharply stretched under chloroform, and the tightness of the tendons shows that it is the retraction of the muscles which alone opposes the reduction of the valgus. This muscular shortening gradually increases, and seems to replace the reflex contractures. The contractures are doubtless still produced by the joint pains, which continue to increase, but when the retracted muscles reach their maximum of shortening, so that the joint-surfaces will not allow them to shorten any more, the contractures can no longer occur.

Usually the shortening is limited to the peroneus brevis and the extensor longus digitorum; occasionally the tibialis anticus is also retracted.

Causation . . . The causes of functional impotence of the peroneus longus are found either in feebleness of the muscle during the developmental period of youth or childhood, or in too

prolonged walking or standing. . . . Professions which necessitate much standing or walking may give rise to it. I have most often met with it in young apprentices (pastrycooks, pork butchers, shop-servers, laundresses, &c.) who are obliged to be always standing. . . . At first sight the symptoms seem difficult to explain. When we find that a person can contract his peronei perfectly, when his feet are not resting on the ground, how does it happen that in standing or walking one or both feet take a wrong position and become painful, as in the *painful valgus flat foot*, which follows general paralysis of the peroneus longus? It is, nevertheless, this muscle which, in the case we are considering, becomes for a time powerless, and that when it ought to be helping in the act of standing or walking. At first I mistrusted my observation, and it was only by the frequent repetition of this pathological phenomenon that I became convinced of its reality.

My electro-physiological investigations of the muscles of the foot have taught me—

1. That the peroneus longus powerfully depresses the first metatarsal bone on the internal (premier) cuneiform, this on the scaphoid, and this last on the astragalus. 2. That by virtue of the movement given to this line of bones it must be considered as the "*key*" of the plantar arch. 3. That it alone during walking and standing firmly applies the sub-metatarsal prominence to the ground, which at a given moment becomes a "*front heel,*" and supports the weight of the body. This muscle, which is so slender in proportion to the power which is required of it for its important functions, easily tires and becomes impotent during standing and walking, in a youth in the dynamic conditions indicated above, or even in an adult when he is overdone.

The name *functional impotence of the peroneus longus* perfectly expresses the pathological condition, the cause of which I have been investigating. It further recalls other *functional impotences,* which I have described elsewhere along with *functional spasms,* and of which the hand affords a well known instance in "writers' cramp." It will be seen soon that there is also a functional spasm of the *peroneus longus,* which justifies the relationship which I have indicated between these similar troubles of the hand and foot.

If still further proof were necessary of the implication of the peroneus longus I would say, in anticipation, that faradisation of this muscle causes the disappearance of the irregularities which its impotence occasions.

This rapid cure further shows us that the pains observed in functional impotence of the peroneus longus need not make us suspect the existence of inflammation or change in the surfaces of the tarsal joints.

. . . The following is a *résumé* of the mechanism of this condition :—

1. In walking and standing the weight of the body often rests on the *back heel* (the hinder and under part of the calcaneum) and on the *front heel* (formed by the head of the first and partly of the second metatarsal bone, which are protected by a bursa).

2. At a given moment in the first stage of walking the back heel is raised from the ground and the whole weight of the body is thrown on the front heel. Then while the after foot (*arrière-pied*), powerfully extended at the tibio-tarsal joint, drags—depressing, as a single piece, the outer edge of the fore foot (*avant-pied*)—the three last metatarsal bones, to which it is strongly bound by the lower calcaneo-cuboid ligament, the peroneus longus, by virtue of its attachment to the under side of the near end of first and second metatarsal bones, powerfully depresses the front heel, through which passes the line of gravity of the body.

3. The peroneus longus is alone able to fulfil this important function, because it is the sole direct depressor of the front heel. On the other hand, the ligaments uniting the joints of the first metatarsal bones, the internal cuneiform and the scaphoid, being very slack (so as to allow sufficient vertical movement to these bones), voluntary extension of the foot on the leg, if this muscle be not able to help, is solely effected by the triceps of the calf, which, I repeat, extends the after-foot at the tibio-tarsal joint, and at the same time drags down the two last metatarsal bones, which are tied to the calcaneum by the lower calcaneo-cuboid ligament. From this it results that in walking and standing the front heel can no longer work with the back heel in forming the support of the body. Then in place of the sub-metatarsal prominence it is the far end of the two last metatarsal

bones which is firmly applied to the ground, especially when at the end of the first stage of walking, the heel is separated from the ground, the foot being extended to propel the body forward.

4. Then, when the weight of the body is resting on the calcaneo-astragaloid joint, and the resistance of the ground is pushing the far ends of the last metatarsal bones in the opposite direction, the foot tends powerfully to move at the calcaneo-astragaloid joint in the direction of valgus. The tibialis posticus at this moment contracts vigorously to prevent this motion, but, as it acts at a great disadvantage, it quickly tires and becomes useless; then at each step the foot is placed in the valgus position passively, at the time when it is placed on the ground during the pathological rotation at the calcaneo-astragaloid joint, the surfaces of this joint being squeezed together towards the outside, while on the inside the ligaments of the scapho-astragaloid joint are stretched. These incessant strains end by making these parts very painful in the points of election indicated above.

5. The reflex contractures caused by these pains are in the abductors of the foot (peroneus brevis and extensor longus digitorum) and in the tibialis anticus (the antagonist of the peroneus longus), which, by raising the submetatarsal prominence, destroy the plantar arch, and thus convert the *passive valgus flat foot* into an *active valgus flat foot.* This valgus at last becomes irreducible owing to the shortening of the muscles.

. . . . . .

Functional Spasm of the Peroneus Longus.

I discovered the existence of spasm of the peroneus longus almost at the same time as its impotence. When I showed by electro-muscular experiment that the peroneus longus increased the arch of the foot, and at the same time caused valgus, I foresaw the existence of a kind of valgus not hitherto described, and due to contracture of the peroneus longus. The clinical case was not long delayed, for I described the form of valgus in 1863 under the name of *hollow valgus by contracture of the peroneus longus.* . . . .

It was only in 1865 that I recognised how this contracture

may be provoked solely by standing and walking, and vanish when at rest. . . . The causation of this trouble had escaped me when, in 1865, M. Nelaton drew my attention to this class of case in an interesting clinical lecture on some cases in his ward. When they were standing, one or both the feet was valgus, and when they had walked a little time painful contracture came on in the muscles of the affected foot. After rest in bed for some time the valgus and painful contractures disappeared. M. Nelaton being struck with the likeness of this trouble to "writers' cramp," called it "*cramp of the foot.*"

At the request of M. Nelaton, I examined these patients, and showed by experiment that their valgus was caused by two distinct conditions—in the one case a spasm of the peroneus longus, and in the other an impotence of that muscle masked by contracture of other muscles.

The following are the signs of hollow valgus from contracture of the peroneus longus, which any one may produce by faradising that muscle.

1. Depression of the metatarsal prominence (front heel) and increase of the plantar arch, caused by a series of downward movements in the line of the first metatarsal, internal cuneiform, scaphoid, and astragalus.

2. Lessened breadth of the foot across the heads of the metatarsal bones by the squeezing together of the cuneiforms, and the twisting of the front part of the foot on the hinder part at the mid-tarsal joint, causing slanting folds of skin on the sole.

3. Active valgus by abduction at the calcaneo-astragaloid joint, which turns the foot out and rotates its antero-posterior axis on the axis of the leg.

4. Prominence of the tendon of the peroneus longus.

[In this functional trouble the arch of the foot is apt to become flattened during standing, because the peroneus longus, though the seat of spasm, is weak and soon gets fatigued. The contracture of the peroneus longus is probably only an evidence of its weakness.] . . . .

In short, functional spasm of the peroneus longus is only a form or variety of functional impotence of this muscle.

[After a long discussion of the treatment of these troubles

by means of electricity, orthopœdic apparatus, and tenotomy, Duchenne finishes his article with the following]

GENERAL CONCLUSIONS.

1. There is a kind of valgus caused by the fatigue of too prolonged standing or walking, which only appears during the exercise of function, and is similar in this respect to "writers' cramp." I have called it "*functional valgus.*"

2. In these cases the peroneus longus being stricken with impotence from fatigue, no longer depresses the sub-metatarsal prominence with sufficient power to support the weight of the body. Then the foot, whose outer edge alone rests firmly on the ground, tends to twist in valgus at the calcaneo-astragaloid joint, and becomes a *flat valgus.*

3. The functional flat valgus from impotence of the peroneus longus is at first painless, but in the second stage the abnormal pressure of the calcaneo-astragaloid joint-surfaces causes pain there, and secondary reflex contracture of some of the muscles which move this joint (generally the extensor longus digitorum and the peroneus brevis). These contractures vanish during rest.

4. When the reflex contractures extend to the peroneus longus, the flat valgus is turned into a hollow valgus, although the muscle is still impotent during walking and standing. The hollow valgus is only recognisable when the foot is not resting on the ground, for in walking it again becomes flat owing to the impotence of the peroneus longus.

5. This form of flat foot is usually cured at once by faradising the peroneus longus. The hollow form is more obstinate, but this mode of treatment is nevertheless the best for it. In some obstinate cases division of the peronei has been successful.

6. The adhesion of the peroneal tendons to their fibrous sheath, as a consequence of tenotomy, destroys their mutual independence and the power of the peroneus longus to depress the first metatarsal bone.

7. Cutting these tendons in their fibrous sheath behind and above the outer malleolus makes this form of flat valgus more serious by rendering its cure by faradising the peroneus longus difficult or impossible.

8. Division of the peroneus brevis should be done close to its attachment to the fifth metatarsal bone, and that of the peroneus longus above the fibrous sheath.

9. In spite of the reduction of the valgus by tenotomy or mechanical means, the functional troubles due to impotence of the peroneus longus nevertheless persist.

10. In spite of the most marked and persistent valgus from the shortening of the muscles, the pains and troubles caused by functional impotence of the peroneus longus do not reappear when the functional impotence of the muscle has been once cured by localised faradisation.

CHAPTER XXVI.

ON THE REFLEX EFFECTS OF ELECTRISATION.*

. . . . . .

Electrisation by reflex action takes place when the rheophores are widely separated, so that the currents run lengthwise through the limb or its nerves. This method of electrisation affects the sensibility of a peripheral zone which corresponds with certain cells in the posterior horn, whence the effect is propagated to the corresponding cells in the front horn, and causes in the muscles supplied by them, irregular contractions bearing but a small proportion to the pain provoked.

. . . . . .

Reflex electrisation is often effected by placing the extremities of the patient in basins of water connected with the rheophores of an induction apparatus . . . or the hands may be placed in one basin and the feet in another. . . . The electric bath is a form of reflex electrisation.

. . . . . .

Excitation of the nerve centres is sometimes indicated. Usually, in such cases, recourse is had to medicine, but electrisation of the nerve endings acts in an analogous way, and I have applied it to the treatment of some cases of paralysis where it could be used without danger.

I have not always had reason to congratulate myself on the employment of this mode of treatment. The patients faradised in this way have sometimes got better, but after the treatment some of them have felt deep seated pains along the course of the nerves, the extremities of which have been stimulated, and these pains have often persisted as neuralgia. The energetic excitation of numerous nerve twigs especially intended for sensibility explains the frequency of these accidents.

Sometimes this mode of faradisation by reflex action has

* From *L'Electrisation Localisée*, 3rd ed., pp. 116—143.

exposed the patient to very serious trouble. The following case is an example of this:—

Case No. 113.—A medical student, aged 22, was stricken with hemiplegia in 1851, the result of a cerebral hæmorrhage. He progressively improved for a year, but there remained a contracture of the flexors of the hand and forearm which hindered his voluntary movements, and which extended to other muscles when certain actions were attempted, or when certain impressions were experienced.

These phenomena are not merely the result of a secondary sclerosis of the cord, as modern researches would seem to show, but they are sometimes a sign of hyperæmia, which one ought to guard against exciting, lest serious accidents be caused thereby. Ignorant of this danger, our student, who had wrongly interpreted one of my publications, thought that faradisation might be used without danger in the treatment of cerebral hemiplegia within four or six months of the attack. He did not understand the difference between limiting the application to the muscles with the avoidance of all painful impressions, and the production of their contraction by means of strong currents acting in a reflex manner.

Having put in action a powerful induction machine with rapid intermissions he took a cylindrical metallic rheophore in each hand, so that the current ran from hand to hand through the brachial nerves and the cord. His muscles immediately contracted, and his hands closed tight on the cylinders, and feeling great pain in his paralysed side he had the presence of mind to kick over the Bunsen's battery which supplied the coil, and thus stopped the current. He was too late, for the current had done its mischief. The head was very painful; the contracture had spread to the whole paralysed side, and an hour later he was found on the floor of his room rolling in convulsions. He was moved to the Charité, where for many days his life was in danger, and in spite of repeated bleedings, purgatives, and a blister to the nape of the neck, the painful contractures, semi-tetanic in character, continued for many weeks. Finally, after many weeks he left with his contractures far more extensive and far stronger than when he conceived the unfortunate idea of trying the reflex effects of faradisation.

If, in this case, the electric stimulation had been limited to

the paralysed muscles these serious accidents would not have happened. The hyperæmia of the brain, which was developed at the point where secondary sclerosis was established, is, I think, however, a counter-indication for even localised faradisation. I can affirm this, for I have learnt it by long experience. Many a time have I used localised faradisation in cases just like the above without any ill resulting. I have come to the conclusion that such local applications are often useless, but not dangerous, if carefully applied. In my early days I have often faradised by reflex action the antagonists of the contracted muscles, and have sometimes repented of it. One cannot be too careful when using so dangerous a treatment.

. . . . . .

[In 1853, Duchenne repeated the experiments of Leroy d'Etiolles of passing mild induction currents from the mouth to the anus in horses and rabbits. A mild current applied in this way to a horse produced general tetaniform muscular contractions, but without stopping the respiration or the heart.] I made similar experiments on rabbits, many of them in the laboratory of my friend M. Liégeois. With a very mild current there was no appreciable result except a very slight contraction of the tongue and facial muscles. Beginning with a minimum current, and gradually increasing the strength, which was regulated by a sliding tube and a water rheostat, we next produced some tetaniform contraction of the muscles of the trunk and limbs. Neither respiration nor the heart were stopped; they were merely suspended for one or two seconds. This experiment was twice repeated (for twenty or thirty seconds) on the same rabbit, and with the same result on each occasion; but when the current became so intense as to tetanise and fix the thoracic walls then a needle plunged into the heart ceased to move. . . . . These facts seem to show that this mode of general faradisation does not very powerfully excite the reflex activity of the medulla oblongata.

[This method of electrisation, *used with the greatest possible care*, may be employed in cases of asphyxia.]

Long clinical experience has taught me that stimulation of the sensibility of the skin of the thorax, especially of the præcordial region, is one of the best means of treating by reflex action, asphyxia, and certain grave troubles affecting the innerva-

tion of the heart and respiratory organs. The accuracy of this conclusion I shall support by many clinical facts.

Case No. 114.—*Asphyxia from carbonic oxide. Absence of respiration. Heart's action very feeble, and perceptible only by auscultation. Cure by faradisation of the skin over the heart.*

An eccentric young pastry-cook, aged 15, one day tried to drown some fancied sorrow in drink. In his drunkenness he tried to asphyxiate himself by crawling into a recess above his master's oven, where he fell asleep. The next morning he was dragged out apparently dead. Happening to live in the same house I was able within a few minutes to try the effects of faradisation. He was blue in the face, breathing had ceased, he was pulseless, and the heart's action could not be felt, although feeble valvular sound could be heard on careful ausculation.

My son had got my induction apparatus ready while I was examining him. I had intended practising faradisation of the phrenic nerve, but I found that the rheophores had been forgotten.

The case being urgent I applied the end of one metal conductor over the left nipple, while the other was moved about over the apex of the heart. The machine was at its maximum strength and the intermissions rapid. In a few seconds weak and slow respiratory movements were observed, and the pulse could be felt after about a minute. Soon his face wore an expression of pain, then he groaned, next moved his arms and then his trunk, and tried to remove the rheophores from his chest. Then he shrieked and kicked and answered questions. In about six minutes the pulse and breathing were re-established, his colour returned, and intelligence was natural.

We thought him saved, but nevertheless I ordered him to have mustard applied to the limbs and præcordia, and warned his master that his symptoms might recur. In a few minutes after the faradisation had been left off, he no longer answered questions, his breathing got slow, and at times he seemed to forget to breathe. His face grew livid and his lips pale, then his breathing became snoring, and he no longer felt a pinch; in short he was again asphyxiated. He was soon roused from this state by a renewal of the faradisation applied less strongly, and continued by my son for ten minutes, until he could stand up and walk about.

Doubting the permanence of his recovery, I had him removed to M. Trousseau's ward in the Hôtel Dieu, and warned the person who went with him not to let him go to sleep. An hour later I found him in M. Trousseau's ward comatose, having fallen into that state during his removal to the hospital, and in spite of repeated cuffings on the part of his companion, who tried hard to keep him awake. Faradisation of the skin of the præcordial region was repeated at intervals throughout the day, and mustard was applied to the surface of the body, and in the end his tendency to coma was quite overcome.

. . . . . .

The sole and undoubted cause of the disappearance of the symptoms of asphyxia in this case was the faradisation of the skin of the præcordia, and the proof of this assertion is found in the relation of the facts, and it is needless to insist upon it.

The following summary shows the benefit of faradising the skin of the chest, in carbonic oxide poisoning, and at the same time demonstrates the necessity of continuing the application for a long time lest the asphyxia return and prove fatal :—

Case No. 115.—A woman under the care of M. Andral, at the Charité, whither she had been brought the night before stifled by carbonic oxide. I very soon brought back the pulse and breathing, and caused the coma to disappear. She recovered enough to tell me how the accident had arisen, and the nature of the first symptoms. We thought her saved, but a few hours later she again fell asleep, and in spite of mustard and blisters died the following night.

If in this case faradisation had been continued, she would probably have been saved. From this case, supported by others, I have concluded that patients asphyxiated by carbonic oxide are threatened with a recurrence of their symptoms for some hours after these have been made to disappear. It is therefore necessary to persevere with the treatment after apparent recovery has been established.

It is important to add that this secondary coma is due to carbonic acid poisoning caused by the asphyxia, and the danger is greater in proportion to the length of the asphyxia. This aggravates the danger of asphyxia from whatever cause it may arise.

[A case is given of a new born child who was roused several times from asphyxia by faradisation, but ultimately died.]

TREATMENT OF GRAVE FUNCTIONAL TROUBLES OF CIRCULATION AND RESPIRATION, SYMPTOMATIC OF A CENTRAL DYNAMIC NERVOUS LESION.

Reflex stimulation of the medulla by faradisation of certain zones of the skin of the chest can modify or cure—

1. The grave troubles of circulation indicative of paralysis of the vagus, and causing sometimes fatal syncope.

2. Breathing troubles due to weakness or palsy of the expiratory bronchial muscles of Reissessen (paralysis of the lung).

3. Simple asphyxia, or comatose asphyxia, caused by accumulation of phlegm.

The following case is a proof of the two first assertions :—

Case No. 116.—*Grave expiratory and cardiac troubles due to diphtherial poisoning. Summary : diphtheria invading a large surface in a lady, aged* 21. *On the sixteenth day a paralysis of the pharynx and soft palate, and on the twenty-seventh day after a miscarriage (at three and a-half months), other paralytic troubles indicative of a lesion of the medulla, viz.,* (1) *cardiac troubles caused by paralysis of the vagus, and cured by slight faradisation by the "electric hand" of the skin of the præcordia ;* (2) *two days later temporary double vision for an hour followed immediately by motor and sensory left hemiplegia, cured in an hour by the faradisation of the regions invaded by the paralysis ;* (3) *two or three hours later a return of the troubles of circulation, again cured in two days by faradisation of the præcordia at frequent intervals ;* (4) *grave breathing difficulty, symptomatic of palsy of the intrinsic expiratory muscles (bronchial muscles of Reissessen), or in other words paralysis of the lung dissipated at once (for half an hour or an hour) and on several occasions by faradising the skin of the back of the chest ;* (5) *collection of bronchial phlegm from expiratory weakness. Death on the thirty-eighth day at* 2 *a.m.*

. . . . . .

This case is interesting from many points of view, but I shall limit my attention to the influence of the faradisation on the cardiac troubles, and the paralysis of the intrinsic expiratory muscles of Reissessen, (the palsy of the lung,) which ultimately caused death by asphyxia.

The results obtained by faradising the præcordia so far surpassed my expectations that I should doubt them still, had they not been witnessed by the learned *confrères* who called me to the case (MM. Barth, Ricord, H. Roger, Campbell, and Descroizilles).

Let us first examine these results from the point of view of physiology.

The serious circulatory troubles which had first to be met in the case of Madame X., *i.e*, extreme frequency, smallness, and irregularity of the pulse, with syncope, or threatenings of syncope, præcordial anxiety, and the sense of suffocation, were, in my eyes, symptomatic of the paralysing influence of the diphtheria poison on the nuclear origin of the vagus. These are the symptoms seen in an animal when the vagus is cut (except the præcordial anxiety and the stifling, which they do not manifest). I have seen the same symptoms also in glosso-labio laryngeal paralysis at a time when the damage must have reached the nucleus of the vagus. . . .

Now I have to remark that on each occasion when these cardiac troubles appeared in Madame X., they were at once dispelled as by enchantment by faradising the skin of the præcordia, and when my hand, which served as a rheophore, wandered from this region to other parts of the chest, the patient ceased to feel relief, and pointed with her own hand to the præcordia, in order to show me the point which I ought to stimulate.

Since, in this kind of therapeutic experiment, the stimulation of the skin of the præcordia alone had the power of changing or dissipating by its reflex action the serious troubles of the cardiac circulation caused by a morbid state of the innervation of the pneumogastric, it is rational to conclude from it that there exists an intimate connection between the sensibility of the skin of the præcordial zone and the origin of the pneumogastric, or in other words *the cutaneous præcordial zone is reflexogenic of the pneumogastric.*

Another therapeutic result shown in the preceding case confirms the special reflex action exercised on this region of the medulla by the sensory nerves of the præcordia, by showing that certain troubles of breathing are only relieved by faradising another part of the chest. We have seen, in fact, that faradisa-

tion of the præcordia was powerless against the expiratory weakness (palsy of the lung or palsy of the bronchial muscles) which followed the palsy of the pneumogastric, while faradising the skin of the back of the chest always triumphed over it, and on several occasions so as to lessen or dispel for a time the sufferings of the patient, and procure her some rest, and postpone for some days the fatal ending.

One might also conclude from this last therapeutic result that the sensibility of the cutaneous zone of the thoracic region, which corresponds to the posterior aspect of the lungs, has special nervous connection with the point of origin of those nerve fibres which animate the intrinsic expiratory muscles (the bronchial muscles of Reissessen).

Every one can see the practical bearing of the preceding considerations :—1. Electrical stimulation of the skin round the heart's apex is one of the best ways of overcoming syncope and the other functional heart-troubles due to paralysis of the vagus without organic damage. 2. Electric stimulation of the skin of the back of the chest has a special therapeutic action in paralysis of the intrinsic expiratory muscles.

The following is briefly the physiological explanation of the preceding facts. In Madame X. the slight electric stimulation of the præcordia acting in a reflex manner on the nucleus of the pneumogastric gave this nerve the power, which its paralysis had taken away, of moderating the action of the heart. In other words, it re-established the check on the heart, regulating its action, and causing the disappearance of the nervous troubles symptomatic of palsy of the vagus. A like theory can be used for the effect of electric stimulation of the skin of the back of the chest on the expiratory paralysis of the lung.

[Another point of importance in these cases is not to use too strong a current. The general excitability of the patient is often increased, and one must proceed gradually, and proportion the intensity of the current to what the patient can bear, or we may expose him to serious accidents. It will be noted that in the above case the mildest form of electrisation, that by the "*electric hand*," was employed.]

The above case, and the reflections consequent upon it, shows how important it is in this stage of diphtherial poisoning to diagnose exactly the kind of palsy which indicates a diseased

state of the medulla, since a special indication for treatment arises therefrom. We have seen that the heart troubles should be met by faradising the præcordia, reflexogenic (*reflexogène*) of the origin of the vagi; and intrinsic expiratory paralysis by faradising the back of the chest, reflexogenic of the nerves which animate the bronchial muscles of Reissessen. In 1869 I saw, with MM. Millard and Aug. Ollivier, another case of diphtheritic poisoning, which was confirmatory of this assertion, and showed its importance.

Case No. 117.—Without entering into the details of this case, I will merely say that it was that of a father who caught diphtheria by kissing his child, who was dying of this disease. After passing through the stage of formation of false membrane, which threatened his life by obstructing the bronchi, he became convalescent, but towards the twenty-fifth day was affected by paralyses which showed the invasion of diphtheritic poisoning (paralysis of the soft palate and pharynx, of the seventh, hypoglossal and N. laryngis inferior, double vision, and paralysis of the fifth on one side), when suddenly on the twenty-eighth day the intrinsic expiratory muscles were stricken with paralysis. At the same time accumulated mucus obstructed his bronchi and threatened suffocation. This paralysis came on without fever, and we feared sudden death.

This time, again, electric stimulation of the skin of the back of the chest quickly re-established the expiratory force and caused the immediate expulsion of the bronchial mucus. It had to be continued many days in order to completely triumph over the paralysis of the lung. The patient is now out of danger, although he has been under treatment for other troubles (lessened sensibility of the hands and feet, with weakness of the limbs). This important case will probably be published in all its details by M. Millard.

If in the preceding cases the diaphragm had been paralysed I should have faradised the skin of the base of the chest, although I had no proof that this cutaneous zone is "reflexogenetic" of the origin of the phrenic. This doubt arises from the failure of stimulation of this region in a case of diphtheritic paralysis of the diaphragm, which faradisation of the phrenic was alone able to cure.

Case No. 118.—A little child, aged 4 months, living in the

Rue de Lille, had had diarrhœa for some days, followed by a membranous ulceration of the umbilicus. Although there had been no croup nor sore throat MM. Barthez and Trousseau considered it diphtheritic. In a few days the child was stricken with a general paralysis, which lasted about forty-eight hours, and was followed by complete loss of voice, and a difficulty in swallowing and breathing. As soon as the child tried to suck it was seized with cough and suffocation. In these serious circumstances I was called in. In addition to the foregoing symptoms I established a paralysis of the diaphragm. After faradising the skin of the base of the chest with no result, I faradised the phrenics, and quickly diaphragmatic breathing was naturally performed. The palsy returned many times, but was soon overcome by faradisation of the phrenic nerves. After faradising the palate, pharynx, and front of the neck on a level with the larynx, the child sucked better, and voice came back a little. He was completely cured in a few sittings.

. . . . . .

I now come to the therapeutic use of reflex stimulation of the medulla by faradising the skin of the præcordia in a kind of *apnœa*, a neurosis marked by an absence of the sense of the need to breathe (*besoin de respirer*) which makes the respiratory movements infrequent, or suspends them for a time. . . . The following case is an example of this.

Case No. 119.—*Neurosis marked by a kind of apnœa. Cured by faradising the skin of the præcordia, and by faradising the phrenic nerve.* A young man, æt. 17, of nervous temperament, suffered after what was apparently pneumonia (?) from a peculiar breathing difficulty. At intervals he stopped breathing for from thirty to sixty seconds without being troubled by it, although there was observed a slight blueness of the lips and face. After this arrest of breathing there followed a loud deep sighing inspiration, and this curious quasi-spasmodic inspiration was usually repeated many times in succession. At the outset of the disease this kind of attack happened only five or six times a day, but they became more frequent so as to be almost continuous, and, which is very remarkable, they were most exaggerated when the patient thought of his trouble and fixed his attention on his breathing. Then the sighs succeeded each other without ceasing. This trouble had lasted for about six

weeks without affecting the general health, when the patient was sent to me by M. Barthez. I thought that we had to do in this case with a neurosis consisting of *a passing abolition of the instinctive want to breathe.* On this hypothesis the sighs which followed the non-breathing period could be nothing but the satisfying of the instinctive need of breath, which only made itself felt when the defective aeration of the blood was sufficient to cause cyanosis. But beside this there was a spasm of the diaphragm, as shown by the sighs and the rising of the epigastrium and hypochondria, which was repeated whenever the patient thought of his trouble. There were then in these queer attacks two periods—one marked by stoppage of breathing and cyanosis, the other by spasm of the diaphragm and other inspiratory muscles.

The daily stimulation of the diaphragm by faradisation practised every day for five or ten minutes caused, as an immediate effect, an increase of the functional trouble, but after a little time the sighs became less frequent. This treatment causing only slight improvement I added to it the faradisation of the skin of the præcordia, and from this time his troubles rapidly lessened, and at the end of a month he was well.

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CHAPTER XXVII.

DYSURIA.*

Dysuria may be caused by palsy of the muscles of the walls of the belly. I have often seen it in cases of paraplegia. It is proved by the fact that when a catheter is introduced the urine is forcibly expelled, that the bladder has sometimes no part in causing the difficulty; but a still better proof is that faradisation of the abdominal muscles renders micturition easy. We must not therefore always diagnose a paralysis of the bladder from the fact of impossibility or difficulty in passing water. Before applying electric stimulation to the inside of the bladder in these patients it is well to stimulate each of the abdominal muscles for a few sittings. In this way the patient is spared an operation, and one which would be useless unless the cause of the dysuria were due to inertia of the muscular coat of the bladder. I have noted dysuria also in cases of palsy of the diaphragm, but in these cases it is much less pronounced than after paralysis of the abdominal wall. The patients are merely obliged to make long efforts to overcome the resistance offered by the sphincter of the neck of the bladder. The only way in these cases to cure the dysuria is, it will be understood, to cure the diaphragm.

[When the bladder itself is paralysed electric stimulation may be directly applied to the organ either by introducing one rheophore in the bladder, and a second in the rectum or on the abdominal wall, or else by means of a double current rheophore introduced solely into the bladder. Occasionally it is impossible to introduce a rheophore into the bladder, and then good may be done by placing one rheophore in the rectum, and another on the front of the abdomen.] . . .

[Constipation, like dysuria, is often in part attributable to palsy of the abdominal muscles, and great good may be got in some cases by electrifying these muscles as well as the rectum itself.] . . .

* From *L'Electrisation Localisée*, 3rd ed., pp. 921—932.

Prolapsus of the Rectum from Atony of the Sphincter.

The following remarks are only applicable to prolapsus caused by long constipation and dysentery, or occurring in sickly children. I do not refer to prolapsus due to surgical causes.

If the finger be inserted into the anus of a patient affected with prolapsus it is felt to penetrate with great ease, the sphincter dilating before the pressure, so that three or four fingers can be inserted without difficulty. The sphincter is not paralysed in these cases, for we see that the patient can contract it, and the motions are not passed involuntarily. The laxity of the anus seems to depend on want of tonic power. I was thus led to think that this atony of the sphincter might be the chief cause of the prolapse of the rectal mucous membrane (previously detached from the other tunics).

If this opinion were well founded, faradisation of the rectum ought to restore tonic power to the sphincter and prevent the prolapsus, and the success of this treatment, as shown by cases given in previous editions, proves the justness of my views.

I have seen one chronic case in a man aged 35, which dated from infancy, and had been aggravated by dysentery. In this case there was great laxity of the sphincter, which contracted immediately after the application of faradism, so that one finger went with difficulty where formerly four or five had penetrated with ease. From this time the rectum no more protruded excepting when the patient made great efforts at stool, although formerly a cough had been sufficient to produce the prolapsus. . . .

CHAPTER XXVIII.

REFLEX ASCENDING CONTRACTURE FROM INJURY TO A JOINT.*

This form of contracture has not, as far as I know at least, been yet described. It generally comes on after injuries to certain joints (chiefly the wrist from falling on the back or palm of the hand), which cause slight arthritis, or merely a short attack of pain in the joint. The contracture, which usually appears a little time after the joint has ceased to be painful and seems quite well, affects more or less of the muscles which move it, ultimately spreading to the muscles of other joints of the limb. The pain is moderate, and at first limited to the contracted muscles. It then affects other muscles, but is always worse in those first affected. It next affects the nerve trunks which animate the muscles, and finally the origin of the brachial plexus. Often, indeed, after the contracture has disappeared the patient suffers (sometimes for years) from continued pain near the source of origin of the nerves supplying the affected limb. These pains seem to indicate a morbid state of the spinal cord at this level. The power of the affected limb is generally lessened.

I have cured many of these reflex ascending contractures by painful faradisation with a rapidly interrupted "extra-current." I shall only give one case here, which is remarkable because it had resisted for more than a year a variety of treatment, including a long application of the constant current according to Remak's method (with a centrifugal current for the first two-thirds and centripetal for the final third of the treatment), and was nevertheless cured in a few sittings by strong faradisation (with rapid interruptions) of the muscles antagonistic to those contracted. I give the case as typical of this form of contracture.

Case No. 120.—Mdlle. X., of Saint-Brieux, æt. 16, not hysterical, and of good constitution. When 11 years old (in 1861) she fell on the back of her right hand, and this was followed by pain in the wrist, with swelling and weakness. She carried the

* From *L'Electrisation Localisée*, 3rd ed., pp. 199—204.

arm in a sling for two months, at the end of which time there was merely a little weakness of the limb. When she wrote the weakness rapidly got worse, and the pains in the wrist returned at irregular intervals, and lasted one or two hours.

When about 13½, after dancing one evening with great animation, the pain in the right wrist (which had been rather violently pulled upon) returned with such intensity that all manual work became impossible. There was neither swelling nor redness of the wrist at this time. As the pain continued, Jobert de Lamballe, who was consulted two months later, advised flying blisters to the seat of pain, but without any result. In December, 1864, the pain continued, and although limited to the wrist it prevented all use of the hand and kept her awake. At this time M. Nelaton advised the use of an immovable and compressing apparatus, which was renewed every ten days, combined with frictions and tincture of iodine.

This apparatus was worn for four months, but without result; on the contrary, the pain got worse, and after using it for two months, in March, 1864, a contracture appeared in the extensores carpi radiales and the pronators of the affected side. During 1865, by the advice of Dr. Regnault, of Rennes, recourse was had to six flying blisters in succession, to sea baths, to hot sand, to aromatic baths, and finally to firing (*cautérisation transcurrente*) over the contracted muscles, but without altering the pain or contracture.

In 1866 Mdlle. X. was brought to M. Nelaton, who sent her to me. I found the following phenomena: permanent flexion of the hand on the forearm with pronation; inability to extend the wrist or supinate the hand. I managed with some effort, which made the patient cry loudly, to straighten the limb, which returned, however, to its former position when left to itself. The hand was useless; movement of shoulder and elbow normal, no wasting of the limb; general health satisfactory; appetite good, no circulatory trouble, no anæmia. I advised her to take iron and use the continuous current. The use of the current was deferred in consequence of the cholera which then raged at Paris. Mdlle. X. had scarcely reached home when the contracture spread to other muscles. The flexors of the fingers kept the hand constantly shut, so that the nails were driven through the skin; deep-seated pains were felt at the origin of

the brachial plexus, descending to the dorsal region, and then there was fever and sleeplessness. She was cupped three or four times over the painful spot, and flying blisters were applied, but without any relief.

Mdlle. X. returned to Paris on April 18th, 1866. There was then contracture of the *flexor sublimis digitorum, flexor profundus digitorum, biceps brachii* and most of the muscles of the shoulder-joint. These contractures caused pain in the limb and cervico-dorsal region of the spine, which was aggravated by trying to straighten the limb. The nails were so driven into the palm that it was inflamed and threatened to ulcerate. The thumb, tightly enclosed in the middle of the fist, seemed wasted by the squeezing, and was more difficult to extend than the fingers. The size of the contracted limb was normal; she had now neither headache nor fever; appetite good; menstruation regular.

The continuous galvanic current was now applied to the limb in a variety of ways, and after about ten sittings the patient could straighten the wrist a little while the current was passing. Then the hand opened a little, and it would remain open, at first for a few minutes and then for a few hours, after the operation. Next the patient had some power of opening and shutting the hand voluntarily, but she relapsed into her former state at the time of her menstruation.

On renewing the application the contracture of the digits disappeared, but the spasm increased in the biceps brachii, and in the extensores carpi radiales (*les radiaux*), so that although Mdlle. X. could open and shut the hand freely enough it remained supinated and forcibly extended, so that it was of no use to her.

The continuous current used for twelve days more did her no further good.

I then strongly faradised the antagonists of the contracted muscles (pronator radii teres, triceps brachii, interossei, extensor communis digitorum), and to my great surprise in less than a few minutes all the contracture had completely disappeared. This result, so quickly obtained, was permanent, and in half an hour after Mdlle. X. wrote in my presence and played some scales on the piano, which she had been unable to do for more than two years. The faradisation was continued for ten days

more, in order to consolidate the cure and increase the power of the hand. Mdlle. X. continued well till the first frost, when the forearm became supinated (by the contracture of the supinator brevis), although the hand retained its power of movement. The pain in the cervico-dorsal region of the spine came back, and was not relieved by blisters, cupping, or narcotics. On September 29th, 1867, Mdlle. X. came back to me, and in a few sittings I overcame the supination by strongly faradising the pronators. I notably lessened the spinal pain by faradising the skin over the painful spot. Since then the contractures have not returned, but in a few weeks the spinal pain came back worse than ever, with exacerbations at night. This still persisted in 1869 in spite of all treatment.

CHAPTER XXIX.

THE MECHANISM OF EXPRESSION.*

[After some general remarks, and a brief allusion to previous workers on this subject, Duchenne points out that the only way of studying the action of individual muscles is by the application of localised electrisation.]

It was to the face that I first applied the method of electrisation which I had invented. . . . The contraction of muscles shows their line and position better than the scalpel of the anatomist. It is certainly so in the face, where, in making anatomical preparations, the fibres going to the skin must be spoilt. This, then, is a new sort of anatomy, to which might be applied the two words which Haller wished to give to physiology—"living anatomy," *anatome animata;* Sæmmering doubtless would have called it *contemplatio musculi vivi.*

. . . . . .

In order to judge of the part played by the muscles of the face in giving expression, I made them contract by means of electric currents when the face was at rest (bespeaking a quiet mind) and the gaze of the patient fixed and direct.

I took the muscles first singly on one or both sides, and then going from simple to compound, I tried to combine their actions by twos, by threes, &c.

In the following paragraphs I shall set forth the conclusions which were reached.

This method of study shows that the expressiveness of the muscles of the face is, *complete, incomplete, complementary,* or *nil.*

There are muscles which, by their single contraction, have the power of complete expression. This may seem paradoxical, for, although it is the privilege of a few only, it has nevertheless been said that every expression wants the help of many muscles.

* *Archives Gén. de Méd.*, 1862, pp. 44.

I shared this opinion, and even thought that my experiments confirmed it. From the first I had noted that partial contraction of the corrugator supercilii caused a complete expression of suffering.

But as soon as I had caused its contraction, the other features (*traits du visage*), chiefly the mouth and the nose-lip line, seemed to undergo a change, in order to agree with the brow in showing sorrow. In this experiment the brow alone had plainly contracted, and I could not see any contraction elsewhere, but I was forced to conclude that this general change in the features was due to muscular contraction. This was the opinion also of others before whom I made this experiment. What was its mechanism? Was it reflex? Whatever might be the explanation the fact seemed to show that localised electrisation was not attainable on the face. I expected no results from these experiments, but a happy accident showed me that I had been deceived. One day when I was exciting this muscle of suffering, and the other features seemed to be contracted in sorrowful agreement, the veil of the patient accidentally fell and hid the upper part of the face, and I was surprised to find that the lower part of the face showed not the least sign of contraction. I repeated the experiment many times and on many persons, even on the still irritable corpse, and always with the same result, *i.e.*, complete stillness of the features of the lower part of the face, till the brow and forehead were uncovered so as to show the whole countenance, when the lines of expression of the lower part of the face instantly seemed animated by suffering. This threw light on the subject (*trait de lumière*), showing that the apparent general contraction of the face was caused by the effect of the ridge of the brow on the other features. One could not help being deceived by this illusion, which is a kind of mirage, had not experiment scattered our doubts. . . .

To what law is this illusion amenable?

M. Chevreul, director of the Gobelins factory, has published a work of great merit and utility on the deception of the eye by the near approach of colours. He shows that colours and shades of colour, when placed alongside of each other, are so changed as to deceive the eye. If *orange* be placed by the side of *grey*, then if the grey be bluish it will seem pale blue, and if yellowish it will take a greeny hue. This form of illusion is not to be

explained, and it is an illusion of the same sort when the brow works an apparent change in the other features.

Whatever may be the nature of the illusion, its usefulness cannot be misunderstood. The following are, I believe, its uses:—1. If to show each passion and feeling it had been necessary to put all the muscles in action at once to change the features, the nervous action would have been far too complicated.

2. The features which depict a passion being limited to a single muscle, or a small number, and to a small spot of the face, their meaning is easier to seize.

3. These features, though circumscribed, ought to make a greater impression by exercising a general influence; but the number of passions being great, and the number of muscles limited, it would not have done to increase the amount of muscular contraction for depicting passion.

Among the muscles lying below the brow, there are some which, like the foregoing, have their own expression, and react in a general way on the countenance; but still their expression is incomplete. These muscles are very expressive. Acting alone, they tell the working of the mind; each of them is the sole representative of an emotion. Let them act by turns, and by turns will appear the lines expressive of joy from simple contentment to mad laughter; of sorrow from disappointment to weeping. This is the first impression on seeing these partial contractions, but nevertheless one feels that the expression is artificial and not natural, and that something is wanting. What is wanting? This is not always easy to say, if I may judge by the opinions I have heard from those who have watched my experiments. These experiments have taught me that the help of many muscles is wanted to complete an expression. I shall return anon to this important matter.

There are other muscles below the brow which, acting singly, are inexpressive, although with others they have the power of showing passion; they are meant to help certain expressions, either to complete them, or to lend them passion or some other character. I will quote an example of this. There is a muscle which drags the whole lower part of the face downwards and outwards, and causes the front of the neck to swell, and this without showing any expression; it merely deforms the features.

But directly the action of another muscle is added to it, there comes over the face a strikingly truthful picture of the most violent passions—dread, scare, fright, torture, &c.

There is not one of the muscles of the face that is not acted on by passion; but some among them, in very small number, do not cause any line of expression, although their movement is very plain. These must be looked upon as *inexpressive*. . . .

The original expressions of the face (such as are caused by the contraction of muscles which are completely, or by the union of muscles partly expressive) are primordial, and they can, by combining, cause harmony, and give rise to expressions with more meaning and more complex.

For example, the "attention" caused by the partial contraction of the frontal muscle, and the "joy" caused by the *orbicularis palpebrarum*, helped by the *zygomaticus major*, are *primordial* expressions. If they be combined the face expresses the lively impression of good news or good luck. This is a complex expression. If to the primordial expression be added that of lust or lechery we get the expression of attention excited by an object of lust, and we shall have a striking likeness of the elders excited by the chaste Susanna. Thus we see that the combination of primordial expressions causes complex expressions.

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[When muscles whose contractions show passions or emotions of opposite kinds are made to contract together there results an inexpressive condition, or a grimace such as is seen in muscular tic. Occasionally, however, we may get great expression from the union of opposite emotions, such as joy and sorrow.]

The above facts call for the remark that the co-operative muscular actions of the limbs and trunk are in no way comparable to those of the face. This proposition needs to be developed.

[All movements of the trunk and limbs are made with the help of many muscles. Thus, for example, in raising the arm we may only feel the deltoid contract, but we know that the scapula has to be fixed by the contraction of many other muscles in order that the deltoid may have a fixed point to work upon. I might give other examples. Muscular movement in the face is more or less independent of mechanical considerations, so that it is more possible for muscles to act singly and in small groups.]

[Duchenne claims to have proved by numerous and repeated experiment that the movements which he produced by faradising the face were not caused, by (*a*) the pain of the application, and that they were not (*b*) sympathetic, nor (*c*) reflex.]

[No one will contest the novelty of these experiments. I shall show that they are useful also.

Anatomy shows us that the muscles of the face are almost continuous one with another. If this continuity were real, independence of action would be very difficult. Anatomy seems to lead us astray in this matter, and it was reserved for electro-muscular exploration to show that fibrillary continuity is only apparent. By this means I have discovered the limits of certain muscles (such as the *pyramidalis nasi*) which the scalpel had taught us to think were continuous with others.

Electro-physiology shows the existence, in the face, of muscles which are neither classed nor named. I can give many examples of this. A rheophore placed on the ala nasi dilates the nostril, as happens under strong emotions. Anatomy has hitherto denied the existence of muscular fibres in the ala nasi. I hope to be able to show that this muscle has been confounded with the depressor alæ nasi (*myrtiformis*), which is itself composed of many muscles whose actions are opposed.

Anatomy has given but one name to groups of muscles having independent action, as is shown by electric stimulation, or voluntary or instinctive movements. Thus in the *orbicularis palpebrarum,* which has been called a single muscle, are found four muscles presiding over different expressions.]

It is clear that physiology must be the master of anatomy, and that it would be wrong, under the pretext of making simple, to continue thus to confuse the science of life and the study of expression. My experiments will correct those errors which have been made as to the actions of muscles and the parts they play in expression. Thus the *zygomaticus minor* was said to help in the movement of joy, whereas my experiment shows that it expresses vexation or moderate weeping. It is thus that the platysma myoides (*peaucier*), which till now has been forgotten or wrongly studied, is shown to help in expressing with striking truth the most violent emotions—tears, anger, torture, &c. I could say as much of some other muscles which have been mistaken, especially those which move the brow.

The study of the muscular physiology of the human face is closely connected with psychology.

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The following is a table of the muscles of expression, ranged in order of their degree of expressiveness:—

GENERAL TABLE.

Completely Expressive Muscles.

Anterior belly of occipito-frontalis (frontal).	Muscle of	attention.
Orbicularis palpebrarum (orbiculaire)	,,	reflexion.
Corrugator supercilii (sourcilier)	,,	grief.
Pyramidalis nasi (pyramidal du nez)	,,	aggression.

Incomplete and Complementary Muscles of Expression.

Zygomaticus major (grand zygomatique)	Joy.
Zygomaticus minor (petit zygomatique).....	Moderate grief, or vexation.
Levator labii superioris alæque nasi (élevateur de l'aile du nez et de la lèvre supérieure)	Bitter weeping.
Transversalis nasi (transverse du nez)	Lechery.
Buccinator.....	Irony.
Depressor anguli oris (triangulaire des lèvres).....	Sadness; complementary to aggression.
Levator menti (muscle de la houppe de menton).....	Disdain and doubt.
Platysma myoides (peaucier)..	Fright, scare; complementary to anger.
Depressor labii inferioris(Carré)	Complementary to irony and aggression.
Levator proprius ala nasi (dilatateur des narines).....	Complementary to violent passions.
Masseter.....	Complementary to anger and rage.

Levator palpebræ saperioris? (palpebraux)	Contempt; complementary to weeping.
Lower part of orbicularis palpebrarum (orbiculaire palpebral inférieur).....	Benevolence; complementary to open joy.
Orbicularis oris (outer part) (fibres excentriques de l'orbiculaire des lèvres)	Complementary to doubt and disdain.
Orbicularis oris (inner part) (fibres concentriques de l'orbiculaire des lèvres)	Complementary to aggressive and wicked passions.
Upward glance (regard en haut)	Complementary to memory.
Glance slanting upwards and to one side	Complementary to ecstacy and sensual delirium.
Glance slanting down and to one side	Complementary to mistrust or fear.
Glance downwards.....	Complementary to sadness and humility.

Another class might be formed of the muscles which are undoubtedly put in action by certain emotions, but which cause no expressive line to appear on the face. These are the muscles of the ears. I should put in this class also one muscle, the levator anguli oris (*le canin*) which I have not been able to make contract partially, and whose action I cannot exactly describe.

It results from the facts above given—

1. That expressions can be divided into two classes—*primordial* and *complex*.

2. That primordial expressions are caused by the partial contraction of completely expressive muscles, or by the combination of incompletely expressive muscles with others whose expression is complementary.

3. Complex expressions result from the association of primordial expressions.

I will now enumerate the primordial and complex expressions which I have been able to get by electro-physiological experiment.

GENERAL TABLE.

Primordial expressions from partial or combined contraction.	*Muscles producing them—* 1. *By partial contraction of completely expressive muscles.*
Attention	Anterior belly of occipito-frontalis (frontal).
Reflexion	Orbicularis superior (part of the so-called orbicularis palpebrarum), moderate contraction.
Meditation	The same muscle, strong contraction.
Contention	Same muscle, very strong contraction.
Grief	Corrugator supercilii.
Aggression (wickedness)	Pyramidalis nasi.
	2. *By the contraction of incompletely expressive, combined with complementary expressive muscles.*
Joy, smiling, benevolence	Zygomaticus major and orbicularis palpebrarum inferior (moderate contraction).
Laughter	The same muscles and levator palpebræ.
False joy—a lying smile	Zygomaticus major alone.
Irony	Buccinator, depressor labii inferioris (Carré).
Bitter weeping (pleurer à chaudes larmes)	Levator labii superioris alæque nasi, levator palpebræ and eccentric fibres of the orbicularis oris (orbiculaire des lèvres).

Primordial expressions from partial or combined contraction.	*Muscles producing them—* 2. *By the contraction of incompletely expressive, combined with complementary expressive muscles.*
Moderate grief	Zygomaticus minor and levator palpebræ.
Sadness, dejection	Depressor anguli oris (triangulaire des lèvres), compressor naris (constricteur des narines), and a downward gaze.
Disdain	Levator labii inferioris (houppe du menton), depressor anguli oris (triangulaire des lèvres), and levator palpebræ.
Doubt.....	Levator labii inferioris, eccentric fibres of the orbicularis oris (des lèvres), either of the lower half or of both halves together, and the anterior belly of the occipito-frontalis (frontal).
Contempt	Levator palpebræ depressor labii inferioris (Carré), compressor naris, and levator labii superoris alæque nasi.
Surprise	Frontalis, depressors of lower jaw (moderate).
Astonishment.....	The same, with considerable depression of lower jaw.
Stupefaction	The same, with maximum depression of the lower jaw.
Admiration, agreeable surprise	Combination of astonishment and joy.
Fear	Frontal and platysma (peaucier)
Fright.....	Frontal, platysma, and maximum depression of lower jaw.

Primordial expressions from partial or combined contraction.	*Muscles producing them—* *2. By the contraction of incompletely expressive, combined with complementary expressive muscles.*
Fright with pain, torture	Corrugator supercilii, platysma, and depressors of the lower jaw.
Concentrated anger	Orbicularis palpebrarum superior, masseter, buccinator, depressor labii inferioris (Carré), and platysma.
Mad rage.....	Pyramidalis nasi, platysma, and maximum depression of the lower jaw.
Sad reflexion	Orbicularis superior and depressor anguli oris (triangulaire des lèvres).
Agreeable reflexion.....	Orbicularis superior and zygomaticus major.
Wild joy.....	Pyramidalis nasi, zygomaticus major, and depressor labii inferioris (Carré).
Lecherous pleasure	Compressor naris and zygomaticus major.
Sensual delirium (délire sensuel)	Same muscles, gaze upwards and to one side, spasm of the eyelids, the upper covering part of the iris.
Ecstacy	Same muscles, but without the compressor naris.
Great grief, with tears	Corrugator supercilii, zygomaticus minor.
Grief, with dejection and despair	Corrugator supercilii and depressor anguli oris (triangulaire des lèvres).

It will be noted in the above table that, as a rule, the higher a muscle is situated the greater is its power of expression by

partial contraction. It will be noted, also, that these muscles are not only able to show the passions and emotions, but that some acts of intelligence betray themselves in the countenance. Reflection and meditation, the noblest work of which the mind is capable, is shown by a simple contraction of one of the muscles of the brow.

I have only included in the above table those expressions which may be produced artificially and recorded by photography. I could doubtless increase the number by causing varying degrees of the chief expressions.

Be that as it may, it will be found that the expressions which I have analysed are numerous enough.

Man cannot express all his passions in his face, especially if all the passions which have been enumerated by philosophers are to be considered separate ones.

"Nothing," says M. Lélut, "can be more difficult, varied, multiple, and complex, than the names given to the passions." "One has but to refer," he says, "to the following list, borrowed from the best sources (Plato, Aristotle, Cicero, Descartes, Hobbes, &c.), which might have been longer, and which we have left in alphabetical order." (*i.e.*, in the French, but not in the English. —Ed.):—

Wonder (the first passion, according to Descartes).	Prying.	Dulness.
	Charity.	Envy.
	Trust.	Emulation.
Conceit.	Despair.	Transport.
Love.	Woe.	Panic.
Greediness.	Mistrust.	Favour.
Anguish.	Firmness.	Fury.
Covetousness.	Discord.	Kindness.
Idleness.	Grief.	Glory.
Boldness.	Longing.	High-mindedness.
Baseness.	Disdain.	Gluttony.
Meanness.	Loneliness.	Gormandising.
Anger.	Disgust.	Lowliness.
Desire.	Esteem.	Hatred.
Vexation.	Hope.	Pluck.
Fear.	Enthusiasm.	Shame.
Courage.	Fright.	Jealousy.

Joy.	Pride.	Self-satisfaction.
Indignation.	Pity.	Shock.
Irresolution.	Tears.	Care.
Enmity.	Faint-heartedness.	Sensuality.
Drunkenness.	Remorse.	Rashness.
Cowardice.	Repentance.	Sadness.
Luxury.	Gratitude.	Timidity.
Lamentation.	Regret.	Veneration.
Contempt.	Laughter.	Vanity.
Mocking.	Security.	Vengeance.
Malice.		

The study of the moving countenance is that part of psychology which treats of the manner in which man shows his emotions by the movements of his face.

If man has the gift of making known his passions by this sort of transfiguration of the mind, ought he not equally to be able to read the very varied expressions which flit across the faces of his fellow-men? What would be the use of a language which could not be understood? To express and understand the signs of facial movement seems to me to be born with man. Education and civilisation merely serve to develop or moderate them. By the union of these two faculties the play of the face becomes an universal language. But to be universal this language must always be composed of the same signs, or, in other words, must depend on muscular contractions always identical. Reason and experiment are here in agreement. I have established the fact that it is always a single muscle which executes the fundamental movement which represents a given mental operation. This law is so rigorous that man cannot change or modify it. Had it been otherwise it must have happened that the language of the face would have shared the fate of speech; each country, each province would have painted the passions in the face after its own way, and possibly caprice would have caused the facial expression to vary infinitely in each city, or in every individual.

This facial language must needs be unalterable as a condition of its universality. For that reason the Creator has placed the countenance under the dependence of instinctive or reflex movements. All instinctive movements are made with great regularity, as is known. Take walking as an example, during

which a little infant solves the most difficult problems of mechanics with an ease and exactness which the will could never reach. One understands then how each passion is figured on the face always by the same muscles, so that neither fashion nor caprice can alter them.

"Usually," says Descartes (*Les Passions de l'âme*, 2nd part, Art. 113), "all the movements of the face and eyes can be changed by the mind. When wishing to conceal its passion it thinks strongly on an opposite one; so that it can be used to dissimulate as well as to declare."

It is true that some persons, especially actors, have the power of simulating passions marvellously with the face and lips. By creating an imaginary situation they are able by means of this special aptitude to call up artificial emotions. Nevertheless I can show that there are certain passions which it is not given to man to simulate, and that the attentive observer is always able, for example, to detect and confound a deceitful smile.

Be that as it may, the characters of facial expression, whether pretended or really caused by the working of the mind, cannot be changed; they are the same for all people, savages and civilised, not differing among the latter except by their moderation and the distinctness of the lineaments (*distinction des traits*).

The face at rest.—When at rest the muscles have still a force which never sleeps, and which they lose only at death. This force is called tone. It is by reason of this force that the ends of a living muscle draw back when cut across. The muscles are like springs, which are more or less balanced in the intervals of contraction. It is thus, in the face, that the tissues, especially the skin, are dragged in the direction of the greatest force.

In the new-born child the mind is barren of emotions; the face at rest is quite negative, expressive of complete emotional barrenness; but when the child begins to feel and to be influenced by passions, the facial muscles begin to act and to paint them on the face.

Those muscles which are most often exercised by this kind of mental gymnastics develop most and have most tonic power.

Need it be said that the face at rest is necessarily influenced by the changes of muscular tone, or, to follow the trivial comparison already made, by the force of the springs which keep it balanced. Thus is formed the countenance at rest, the expres-

sion of the individual, which should be a reflection of our habitual thoughts, a portrait of our passions. (This is but a scientific development of a well-known and generally-admitted fact.)

Nevertheless the great philosopher Diderot seems to have placed great restrictions on this theory. "Sometimes," says he, "we make our own countenance. The face accustomed to take the expression of the master passion keeps it; sometimes also we get our expression from nature, and such as it is we must keep it. *It may have pleased her to make us good and give us wicked faces, or to make us wicked and endow us with a countenance of goodness.*" If it were true that goodness could be masked by the externals of wickedness, we should have to lessen our admiration for that masterpiece of nature—expression. Diderot's assertion is, happily, not accurate. The expression of a new-born child is always negative. It is time alone which develops individual expression, good or wicked, as good or wicked passions have the mastery. Even if we admit that a good man may be born with a wicked face, the workings of a noble mind would soon blot out this monstrosity.

[The concluding pages of this essay are occupied by arguments which tend to show the importance in the fine arts of a scientific study of the expressions of the face.]

CHAPTER XXX.*

MUSCULAR PROTHESIS.

Physiological muscular prothesis, deduced from my electro-physiological and pathological researches on the movements of the hand, foot, and trunk.

My object is to introduce into practice a system of apparatus destined, 1. To supply as far as possible the individual voluntary power of wasted or palsied muscles by re-establishing or helping natural movements; 2. To prevent or overcome deformities of joints by equalising the tonic forces which naturally control the relations of the articular surfaces. This method is physiological, because it imitates nature in disposing its artificial muscles according to the exact facts of anatomy and physiology. This is its only novelty.

General considerations.—Our objects are—

1. *To supply the individual voluntary action of the palsied or wasted muscles.*

[For this end an exact knowledge of the true action of the muscles is necessary, and also of the movements produced by combinations of muscles. It is necessary also in artificial arrangements to imitate as closely as possible the muscular arrangements found in nature.]

2. *To restore or facilitate natural movements.*

The commonest orthopœdic apparatus is so constructed that the movements of the joints are generally made impossible. The inconvenience of this is evident. I have never used anything but elastic apparatus fixed to artificial tendons.

Elastic force was used by Delacroix, who applied it to an apparatus *intended to replace the extensor of the fingers.* This apparatus is described (*Dictionnaire des Sciences Médicales*, Paris, 1819) by Fournier-Pescay and Begin, and by Gerdy (*Traité des Bandages*, 1837), who also advised this system for *palsy of the flexors of the elbow and ankle.* Mellet (*Manuel*

* From *L'Electrisation Localisée* 3rd ed., pp. 1034—1096.

d'Orthopédie, 1844) advised the use of caoutchouc instead of metal springs. Rigal also used bands of vulcanised india-rubber for orthopœdic purposes. . . . . In spite of these attempts, elastic apparatus has not become general in orthopœdic practice, notwithstanding that this system acts not merely in a prothetic manner, but also as a gymnastic tending to cure the muscular lesion.

In order to obtain regulated movement we need not only a force sufficient to cause it, but a thorough knowledge of the mechanism. But with the views of Galen still prevalent (who taught that during voluntary motion the antagonistic muscles were inactive) it is impossible to explain the movements which should result from the action of springs intended to replace muscles.

If Galen's doctrine were true, elastic apparatus would be inapplicable. In fact, if it were true that during voluntary movement the antagonistic muscles were inactive, every elastic meant to replace a palsied muscle could only produce a jerking movement always the same, unless the will put in action the antagonist muscles (a mental process not to be confounded with the ordinary action of the antagonist muscles during voluntary motion). Suppose the flexors of the elbow are palsied while the triceps is sound, and that a spring with its ends fixed to the arm and forearm keeps the elbow bent. When in this case the patient wants to bend the elbow after extending it, the triceps would become completely inactive, and the spring would completely bend the elbow with a sudden jerk. Of what use would such apparatus be, especially in big movements? If we wished to get an artificial extension of the knee by this means, the motion would be jerking, as in locomotor ataxy when co-ordination is abolished. Flexion of the elbow produced in this way would not allow the hand to seize any object with certainty, and we should get similar effects whenever we replaced muscles by elastic force.

But as a matter of fact, patients with elastic muscular makeshifts, provided the antagonists of the lost muscles are preserved, can flex or extend the damaged limb either gradually, or more or less suddenly, or partially, so that they attain their end with as much certainty as if they had all their muscles.

This is not the result of a sort of education by which the antagonist muscle is relaxed after being voluntarily contracted;

for these patients, children as well as adults, easily exercise this function the first time the apparatus is applied. In this case it is the artificial elastic force which causes the movement, but the resistance of the antagonising muscles helps to moderate it. Here is the proof. In these patients with palsy of the flexors of the elbow I passively extended the elbow without the intervention of the patient's will, and then suddenly leaving it to itself, it was flexed with a jerk, showing that the power of the spring was greater than the tonic force of the triceps. Having straightened the elbow afresh, I asked the patient to half flex it quickly, or slowly and gradually, and they were able to do this at once. It is evident that in this case the triceps acts at the time of the voluntary movement, *i.e.*, that it suddenly contracts (which, by the way, I could feel), and then relaxes so as to moderate the elastic force in accordance with the wishes of the patient (*au gré de la volonté du sujet*). . . .

These facts show that voluntary motion results from a double nervous action, which I have called the *harmony of antagonists*, which on the one hand causes the contraction of the muscles producing the movement, and on the other, contraction of the antagonists, immediately followed by a relaxation, proportionate and parallel to the movement produced, which moderates the movement and makes it more certain.

In presence of these facts the theory of the alternative repose of muscles during voluntary movement breaks down. We can no longer say, for example, that during walking the flexors and extensors rest by turns. Without going into the history of the various opinions which have been held with regard to the mechanism of muscular action since Galen, I must say that Winslow was the first to attribute to the antagonist muscles, which he happily called *moderators*, an active part in the voluntary movements caused by the muscles which bring about certain positions and attitudes, and which he called the *principal motors* (Winslow, *Traité des Muscles*, p. 160). This doctrine of Winslow had need of demonstration, for although it has been professed by some physiologists since his time, it took no place in science and was soon forgotten, so that no traces of it are found in modern classical works. But Winslow only accorded to the antagonist muscles a secondary place in voluntary movements. I give them a more important part, for I think the power of their

action is parallel with that of the muscles which cause the movement.

A knowledge of these facts is needed by those who use elastic apparatus in orthopœdy in order that they may not only know *how* but *when* to apply them. I will endeavour to explain.

There are some palsies in which these apparatus cannot produce good results. These are chiefly palsies complicated by reflex contractures. In cerebral hemiplegia it is known that contracture occurs after a certain time, especially in the flexors of the wrist and fingers. These contractures increase or occur spasmodically during voluntary exercises or under the influence of some impression, even when the movement of the extensors is partly restored. Thus, if the patient wants to open the hand or extend the wrist, the flexors are seen to contract violently, so as to neutralise the action of the extensors. Many muscles will sometimes behave in this way when the patient attempts certain movements. These spasmodic contractions were attributed by Marshall Hall to the reflex action of the cord, which had become very excitable. The cause of this extreme excitability of the cord in cerebral hemiplegia is the irritative action of secondary fasciculated sclerosis, which Marshall Hall knew nothing of.

Further, the brain has lost the power of localising the excitations by means of which this or that function can be accomplished. The theory is of little importance, for the facts are real enough and spoil the play of elastic apparatus. . . . . These spasmodic contractions do not, happily, occur in infantile palsy, nor in palsy from nerve-damage, nor lead palsy, nor rheumatic palsy, in all of which elastic apparatus may be used with advantage.

Need I say that these apparatus are of no use in cases where there is a muscular retraction? This must first of all be cured or overcome by special means.

[Having tried springs of metal and springs of india-rubber in his elastic apparatus, Duchenne has abandoned the latter because their power is too much influenced by the weather, and they are apt to break and to need frequent renewal. Although the springs are usually placed in the position of the belly of the muscle to be replaced, this rule is liable to exceptions, as will be seen when details are entered upon.]

It would be illusory to suppose that an artificial elastic muscle

can act like a living muscle in the production of voluntary movement. The living muscle, when worked by the will, puts forth a very variable power, gradually or suddenly as needs require, according as it acts as motor or moderator, or helps in the work of others. The elastic makeshift, on the other hand, to which a fixed and determinate power can alone be given, cannot fulfil such many-sided functions. Experience has taught me what reason would have led me to expect. There are some cases in which elastic force is quite inadequate to replace the lost functions. An example will show this. Suppose the gastrocnemius and soleus is palsied or wasted; extension of the ankle is abolished and it remains always flexed. If we only wanted to restore the power of extending the ankle when the foot is not resting on the ground a spring with a power of a few kilos would surely be enough. But if we want to support the weight of the body, or give it a forward impulse, as in the first stage of walking, this limited power would not be sufficient, because in walking the gastrocnemius and soleus contracts with a force at least equal to the weight of the body. But if we gave such a power to our makeshift spring the foot would be for ever extended, and walking would be hindered, as it is in cases of palsy of the flexors of the ankle. In such cases I have been obliged to combine rigid apparatus with a spring arrangement.

3. *To prevent or overcome deformities of joints by balancing the tonic forces which control the normal relations of the articular surfaces.*

[In cases of palsy not only do the sound muscles, which are no longer antagonised, contract, but the joints are apt to get deformed and dislocated; the ligaments contract in one direction and stretch in another, so that false ankyloses are produced; the limb is deformed, and its use interfered with. The key to these troubles is the exaggerated action of the non-paralysed muscles.]

Oppose to this exaggerated action of healthy muscles the action of artificial muscles which imitate nature and you will prevent, cure, or improve the deformities of joints which follow on local palsies.

. . . . Palsy of the peroneus longus may be taken as an example. Knowing the proper action of this muscle, and that of its antagonist the tibialis anticus, one foresees as a result of

its palsy, in addition to an immediate varus, an increased width of the foot from separation of the cuneiforms on their plantar aspect, an upward movement of the bones along the inner edge of the foot, and finally disappearance of the plantar arch and flat-foot. An artificial peroneus longus closely imitating nature is clearly the only means of overcoming this deformity. Nature has been so clear-sighted in the selection of points of attachment for the tendons that any departure from them hinders movement and causes deformity. For example, I have fixed an artificial peroneus longus at a spot more or less close to the front end of the first metatarsal bone in order to depress it more powerfully. The first metatarsal then became dislocated on the internal cuneiform, so that the front edge of the latter caused a prominence on the instep, on which the shoe pressed painfully. Nature, by fixing the tendon to the hinder under part of the first metatarsal, has had the intention of depressing equally and at once the first metatarsal, the internal cuneiform, and the scaphoid; by fixing the tendon too near the front end of the first metatarsal the action became too strong on this bone and too weak on the others.

[Duchenne is of opinion that in cases of infantile paralysis, prothetic apparatus cannot be applied too soon in order to prevent deformity.]

Prothetic apparatus sometimes cures or benefits stiff joints and false ankyloses caused by the immobility of a limb from local palsy or other cause.

One might, as in the usual apparatus for overcoming muscular contracture, use a fixed power; but experience, as I shall show, has taught me that a continued elastic force, especially during sleep, acts like the gentle power of never ceasing muscular tone; that it conquers the resistance of ligaments more certainly than the brute force of rigid machines, without causing pain, and without fixing the joint.

MUSCULAR PROTHESIS OF THE HAND.

[The application of these apparatus requires a knowledge of anatomy and physiology. One must determine exactly the seat and action of the lost muscles and put upon the apparatus a correspondingly greater or less number of artificial muscles. . . .]

[In cases where the extensor communis digitorum is paralysed Duchenne recommends the apparatus invented by himself. It consists of the following parts: (*a*) a glove, the fingers of which reach to the upper thirds of the mid phalanges, upon which they should fit firmly; (*b*) artificial tendons fixed to the far ends of the near phalanges, and running in sheaths sewn to the back of each phalanx, and converging towards the wrist, where they end in little rings; (*c*) a stiff leather sleeve, laced or buckled on the forearm and joined to an armlet of stuff fixed above the elbow to prevent the leather sleeve from slipping down. Lastly, two spiral springs, covered with leather, fixed to the back of the sleeve and having a power of three or four kilos, and joined to the extensor tendons by the rings.]*

[In cases where the above apparatus cannot be used, as in the instance of a patient who was wounded by a ball in the lower part of the arm, and could not bear the pressure of the armlet, M. Duchenne recommends a machine which consists of a steel plate fixed to the front of the forearm by a stiff leather sleeve laced at the side; another steel plate is modelled to the palm of the hand, and jointed with the first at the wrist so as to allow side movements. This palm plate, which reaches forward to the metacarpo-phalangeal joints, and outward to the fold which bounds the ball of the thumb, is fastened with a strap to the metacarpus. A digital plate is applied to the palmar surface of the first phalanges, and is jointed to the palm plate. A spring fixed to the palmar portion keeps the finger-plate extended so that the first phalanges are always in extension. The whole is lined and finished so as not to cause bruising.

When the flexors of the fingers (superficial and deep) are lost M. Duchenne recommends an apparatus which consists of a glove, sleeve, and armlet, as in the machine for replacing the extensors. The tendons run in sheaths on the palmar surface, and reach to the end phalanges, and they are joined to springs fixed on the sleeve. The fingers of the glove are slashed across the back of the knuckles so as not to restrain flexion.

The springs should be strong enough to flex the mid and far phalanges, but should have no action on the near phalanges.]

* Figures of these apparatus are given in the third edition of the *Electrisation Localisée*, p. 1048, *et seq.*

Substitutes for the Thumb Muscles.

. . . . . .

[The muscles of the ball of the thumb very frequently are wasted. Duchenne has invented a machine intended to replace the muscles which cause opposition of the thumb. The sleeve and armlet are the same as in the apparatus previously described. It consists of a glove from which all the fingers have been removed, the thumb alone remaining, and artificial tendons working in grooves and attached by rings to the springs, which accurately replace the abductor and opponens pollicis. This machine extends the far phalanx, moves the near phalanx laterally outwards on the first metacarpal bone and opposes it, as in nature.]

. . . . . .

SUBSTITUTES FOR THE MUSCLES OF THE LOWER LIMBS.

These are of more practical importance than those for the upper limb, because they may prevent deformities which, once developed, become incurable. With this object they should always be used along with other modes of treatment, especially faradisation, the object of which is to cure the palsy or wasting, the prime cause of the deformity. Further, the cases to which these means are applicable are very numerous.

My researches have been largely made on youths and children. My object was as much to prevent deformity while the paralyses were being treated, as to restore movement or make it easier.

. . . . . .

All my efforts to get the complete movement of the foot were fruitless until I imitated nature by fixing my false muscles to their proper anatomical points.

. . . . . .

The apparatus is composed of a sock or gaiter, false muscles, and a system of bands, with or without metal protectors, which serve as a point of support for the false muscles.

The "sock" is of ticking, and extends from above the ankle to the roots of the toes, so as to allow of the free movement of the latter. The sock is cut like a boot, with the seams (made smooth so as not to hurt the skin) encircling the foot. It is

usually laced in front, but, under certain circumstances, on the inside or the outside. It must fit well, without folds, and be not too tight. A silk sock should be worn beneath it. . . .

The tendons of the false muscles for moving the foot, as regards their attachment and direction, are made to closely imitate nature. They are made of silk laces. . . .

The motors are covered spiral springs. Their extension is checked so as not to exceed a third of their length, so that further pulling upon them causes them to act like a rigid force. These motors end below in hooks which fasten to the rings of the false tendons, and above in straps which fasten to the contrivance to be described.

I have tried in these attempts to get rid of metal as much as possible. At first I made a calf-piece (*molletière*) of thick leather lacing on the outside, which could be tightened by the garter so as to fix it firmly. This proved insufficient, for when the calf was wasted it was pulled down by the power of the false muscles, and when the calf was well developed the constant pressure of the garter caused it to waste.

I then tried to support the calf-piece by a thigh-piece taking its point of resistance above the condyles of the femur. . . . . This machine, closely resembling the one used for the hand, is not applicable when great power is required, because the tight-lacing of the thigh-piece, which then becomes necessary, causes wasting of the thigh-muscles, especially the vastus internus and vastus externus. . . .

The thigh-piece is further very disagreeable in hot weather. . .

The apparatus, as ultimately contrived, fulfilled all the indications. It consists of two metal props, to which are fixed two circular metal bands, the fronts of which are jointed and can be opened; and of a stirrup, to which a leather sole is fixed, sometimes strengthened by a plate of metal, all being covered with leather.

The sock being put on the foot the limb is placed in the apparatus, and a boot holds all firm. Then the false muscles are stretched and fixed to the upper ring by means of buttons, which are riveted to it.

. . . . . .

Substitute for the Gastrocnemius and Solens.

[As soon as these muscles are palsied we must prevent the talipes calcaneus which inevitably results. The lost muscle must be replaced by a spring and tendon fixed to the sock at the level of the attachment of the tendo achillis. The apparatus must be worn at night, and the false muscle must have power enough to keep the foot extended when at rest.

. . . . . .

The artificial elastic muscle cannot in any case assist in walking or standing. A power at least equal to the weight of the body would then be needed to keep the foot always extended. . . . . In cases where the weight of the body is considerable I always use a rigid apparatus, in which the movement of the stirrup is checked so as not to exceed a right angle.]

A False Peroneus Longus. Apparatus for the night.

The physiology and pathology of the peroneus longus having been completely ignored till now, the substitution of this muscle has also been neglected. Not that nothing has hitherto been done to counteract flat foot caused by palsy or congenital weakness; but since this was always looked upon as a derangement of the bones, attempts were made to form a plantar arch by pressing against it by means of a convex sole. Such an arrangement, worn for a year or more, must make the troubles of flat foot worse by compressing the plantar nerves and causing numbness, creepings, and pain in the foot and sole. And when this suffering has been borne long enough to produce a result it is not a true plantar arch that has been formed, *i.e.*, the depressing of the sub-metatarsal prominence by a series of little movements of the joints of the inner edge of the fore-foot, but a true hollow foot by bending the fore-foot on the hind foot at the medio-tarsal joint. Hence the outer edge of the foot, which is naturally level, makes a curve with a downward concavity just like the inner edge. For one deformity of the foot another is substituted. As we know that a single force (the peroneus longus), acting from without in and from above down on a definite spot (the hinder lower part of the first metatarsal and adjoining part of the internal cuneiform bones), causes the combined movements which help to form the plantar arch, nothing

is easier than to imitate nature by fixing to the gaiter-sock, at the point of junction of the first metatarsal and internal cuneiform, an artificial tendon, which follows the line of the peroneus longus in the sole, passing behind the external malleolus to be attached either to the outer part of the calf-piece, or to the apparatus of metal bands.

The false peroneus longus just described quickens considerably the restoration of the plantar arch, which is a long time being re-established by the faradisation of a palsied or weak peroneus longus. . . . It is especially in the treatment of congenital flat foot, when the patient is not very young, that the help of the false peroneus longus is useful if not necessary. In these cases the dorsal ligaments of the inner edge of the foot are so shortened as to allow very little play to the joints, and offer a great resistance to the action of the peroneus longus, whether true or false.

I have faradised many painful congenital flat feet for nearly two years, and the pains and fatigue caused by walking and standing have disappeared. But the curve of the plantar arch was developed with despairing slowness because of the resistance of the ligaments. If I had not wished to judge of the actual value of faradisation of the peroneus as the sole treatment for flat foot, I should have tried long since to show that in these cases the plantar arch may be developed solely by the influence of a false peroneus longus.

. . . . . .

The false peroneus longus is only of use to preserve the normal shape of the foot, or to form a plantar arch when the foot is flat. It is of no help in walking because for this, as in the case of the false gastrocnemius and soleus, a force sufficient to lift the entire body would be necessary. When, therefore, this muscle is incurable a rigid apparatus is needed to prevent the foot from turning outwards. This will prevent the sprains and twists which are caused in walking owing to the outer edge of the foot taking wrong points of support.

A False Tibialis Anticus.

. . . . . .

[Palsy of the tibialis anticus is inevitably followed by contracture of its antagonists, the gastrocnemius, soleus and

peroneus longus. In such cases the foot on the paralysed side is more extended than that of the healthy side during sleep; the plantar arch is exaggerated by the action of the peroneus longus, and the tendo achillis retracts so as to necessitate orthopœdic appliances or tenotomy. It is necessary, therefore, to keep the foot semi-flexed and adducted at night, in order to prevent these troubles. A false tibialis anticus fulfils these indications, but it is insufficient when the equinism has passed its earliest stage. It is then necessary to use a rigid apparatus, or to cut the tendo achillis. Tenotomy ought not to be too long delayed, lest an incurable hollow foot be formed.

When the contracted tendo achillis offers no obstacle, flexion of the foot is accompanied by abduction. Hence results at length a valgus from deformity of the calcaneo-astragaloid joint, so that in extreme cases the internal malleolus will rest on the ground during standing and walking. To prevent this I put the foot, covered with a gaiter (*guêtre*) and sock, into a machine with metal rods, the flexion movement of which is limited to a right angle. Then, when the boot covers the stirrup and sock, I stretch the false tibialis anticus, so that the foot is kept at a right angle during rest. This machine causes the foot to bend to a right angle during walking, and helps, with the apparatus worn at night, to prevent deformity of the foot.]

False Extensor Longus Digitorum.

. . . . . .

[This muscle is a flexor-abductor of the ankle, and in replacing it I have neglected its action on the toes, but have fixed the tendons to the sock at the level of the far ends of the four inner metatarsal bones and of the near end of the fifth metatarsal bone (in imitation of the peroneus tertius, which ought to be considered as accessory to the extensor longus digitorum). These tendons run in a groove fixed to the lower and outer part of the leg, and unite in a ring which is fixed to the false spring muscle, which itself is fixed to the calf-piece or to the machine with metal guards.]

False Peroneus Brevis and Tibialis Posticus.

[When these muscles are palsied rather a stiff boot is necessary, in order to prevent the foot from twisting in or out.]

[It is needless to add that in practice the muscles of the foot are seldom paralysed singly. Many are usually affected at the same time, and the making good of muscular defects by means of false muscles is then by no means a simple or an easy matter.]

I have said before, and I repeat it, that the loss of one or two muscles is often more serious than a total palsy of the foot, as in the former cases the deformity is more severe. The orthopœdy is most simple when the palsy is complete. I could give instances of partial palsy in which every appliance has failed, for although the foot could be restrained when at rest, the morbid instinctive movements of the sound muscles during walking were such as to cause compressions and lacerations which prevented the apparatus from being worn. On the other hand, it is easy to give firm support during walking by means of orthopœdic apparatus when all the foot muscles are palsied. When, therefore, certain muscles are irreparably lost, would it not be rational in some cases to destroy the power of the sound ones by means of surgical interference, seeing that these latter cause morbid movements which hinder standing and walking? Such an operation, it is well understood, should be done before the joints become deformed.

A child with complete palsy of the foot ought not to be allowed to walk till the lateral movements of the foot have been prevented by a suitable apparatus. I employ for this purpose an apparatus with metal rods, of which the stirrup is allowed only a limited amount of flexion and extension, and the foot is kept midway between flexion and extension. False muscles are added to this machine in some cases. It is very light, and keeps the foot firmly flexed at a right angle. The boot or shoe merely covers the gaiter-sock, and is completely independent.

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Artificial Spinal Muscles.

The physiological prothesis of the spine cannot be understood or rationally applied without a knowledge of the individual actions and functions of the muscles, without an exact diagnosis, or without a knowledge of the mechanism of the curves in health and disease. . . .

The following is a *résumé* of investigations which are set forth at length in my *Physiologie des Mouvements* (1868, Part II., chap. xi., p. 701) :—

1. The muscular bundles which form the *sacro-lumbalis* and the *longissimus dorsi*, and which are fixed above to the four or five lower ribs, and the transverse processes of the lumbar vertebræ have the same action on these vertebræ, giving them an oblique movement of extension and flexion to the same side; in other words, they are extensors and lateral flexors of the lumbar and lower dorsal vertebræ. Physiologically they form a single muscle, which might be called the *superficial spino-lumbalis*. By the combined action of the muscles of the two sides direct extension of the lumbar and lower dorsal vertebræ is produced.

2. Diemerbroeck and Winslow have shown that under the name sacro-lumbalis four distinct muscles have been confounded.

Electrical experiment shows that these muscles, which form the superficial layer of the spinal muscles, ought to be divided, physiologically, into the superficial lumbar and superficial dorsal, because they can be made to contract independently of each other, and in certain attitudes of the trunk they are seen to contract, producing lumbar and dorsal curves in opposite directions (a false physiological scoliosis), and because clinical observation affords frequent examples of atrophy on one side or both, sometimes of the dorsal muscle and sometimes of the lumbar.

Without this independence of the lumbar and dorsal spinal muscles the S curve of scoliosis could not be produced.

3. Contrary to the opinion of anatomists in general, none of the bundles of the sacro-lumbalis or longissimus dorsi have any appreciable rotatory action on the vertebræ under normal circumstances.

4. The essential rotator of the vertebræ is the *semi-spinalis* (*transversaires epineux*), which I shall call, to distinguish from the preceding, the deep spinal muscle. Acting unilaterally it extends feebly, but when both sides contract together they extend forcibly.

5. The degree of obliquity of the pelvis, and consequently of the sacro-lumbar curve, depends upon the balance between the power of the extensors of the lumbar and last dorsal vertebræ and the flexors.

This statement rests on the following facts :—

(*a*) When the flexors of the lumbar vertebræ (abdominal muscles) are wasted, the pelvis bends forward during standing and walking, so as to throw all the weight of the trunk on the extensors of the lumbar vertebræ, which, in order to bring the centre of gravity of the trunk within the basis of support, pull up the spinal column in its lumbar section, causing a *lordosis* (lumbo-sacral curve). This curve is the more marked, the buttocks are the more prominent, and a vertical line let fall from the most prominent dorsal spine falls far in front of the most backward part of the sacrum, in proportion to the amount of wasting of the abdominal muscles.

(*b*) When the extensors of the spine are wasted while the abdominal muscles remain sound, the pelvis is placed in the greatest extension possible, so as to throw the trunk back and cause its weight to be supported by the abdominal muscles. Hence results a kind of lordosis very different from the last, for the buttocks are flat, and a plummet let fall from the most backward dorsal spine falls far behind the sacrum (see figs. 12 and 13, p. 55).

6. A physiological saddle-back, or lumbo-sacral curvature, with very pronounced inclination of the pelvis, far from being rare or exceptional, are ethnological characteristics of some races.

They give a graceful form to the body, as in the Iberian races, or they cause a sort of deformity, as in some African and Indian races.

The opposite characters, *i.e.*, a straight stiff spine without apparent lumbo-sacral curve, is seen in the Anglo-Saxon race and other races of Northern Europe.

7. The above clinical facts show that physiological saddle-back is due to relative weakness of the abdominal walls. This is confirmed by the development of the belly during pregnancy, and the looseness and weakness of its walls after delivery; so that in women with slight lumbo-sacral curve, the abdominal wall by its force offers more resistance to the development of pregnancy, and it recovers and remains firm and tense even after many pregnancies.

Women with natural saddle-backs are liable to deformity of the belly, and sometimes other troubles, after pregnancy. The

cause of "saddle-back" being understood, it may be possible to moderate it by gymnastics.

8. Between the extremes of pelvic inclination mentioned above are many intermediate grades. What is the proper amount? We cannot fetter beauty with absolute rules.

9. We gather from the preceding facts that the flexors and extensors of the lumbar vertebræ work together to maintain the erect position. This is well shown by the return to the natural position of the trunk when standing, under the influence of the gradual recovery of the power of the lumbar extensors, when it has been bent back owing to their wasting.

10. The lumbar spinal muscles are far more important than the dorsal in relation to the vertical position of the trunk in standing and walking, and its shape. This is shown by clinical observation. When the lumbar spinal (*spinaux lombaires*) muscles are palsied on both sides, there results *lordosis* (lumbar curve with forward convexity), and a difficulty of balancing during walking and standing. When one side is affected there is gradually developed a *scoliosis* (*i.e.*, a deformity of the vertebral column and thoracic wall), while bilateral palsy of the spinal dorsal muscles causes only a *cyphosis* (dorsal curve with convexity backwards) without appreciable difficulty in standing or walking, and a one-sided palsy causes very slight spinal deformity.

11. Excessive action of the dorsal spinal muscles may change the *normal* cyphosis into a straight line, or even a curve with forward convexity (dorsal lordosis), whence results a flattening of the thorax, and a lessening of its diameter from front to back. This excessive action of the dorsal spinal muscles is caused by an effort of compensation when the cervical part of the column is kept continuously flexed owing to palsy of the cervical extensors or caries of one of the last cervical or first dorsal vertebræ.

My experiments have, as a rule, confirmed the opinions of authors; nevertheless I have to call attention in this *résumé* to the two following facts:—

12. In the erect posture the flexors and extensors of the head help to keep it straight on the neck. In persons in whom the extensors of the head are weak or palsied the head is kept bent back in order to throw its weight on the flexors. When the flexors are palsied the head and neck is kept bent forward, so as to throw its weight on the extensors.

13. Local faradisation can cause contraction of the sternal or clavicular part of the sterno-cleido mastoid.

Either of these parts may be affected with spasm. The sternal part rotates the head more than the clavicular. It is the opposite with regard to flexion. Therefore I think that these two parts may act independently under certain circumstances.

These facts justify the old division of the sterno-cleido mastoid into two muscles—the sterno-mastoid and the cleido-mastoid.

Origin and Relief of Spinal Curvatures.

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1. Lordosis. . . .

I have often noted in my practice among the Boulognese, especially the women belonging to the maritime families near Boulogne, that the abdominal walls of women in whom the saddle-back was very great had been greatly distended, and that after their first pregnancies they remained loose and flaccid. On the other hand, when the lumbo-sacral curve was slight, the abdominal walls completely retracted after many pregnancies, and the belly was as flat and the skin as tense as in a young girl. I have found but few exceptions to this rule.

This looseness and flabbiness of the belly after pregnancy in women with pronounced saddle-back seems to me to be explained by the failure of tonic power in the muscles.

This condition of things is often very trying. After the first or second pregnancy the belly gets so big from the bulging of the intestines that the trunk and figure get deformed. Walking and standing soon provoke fatigue and lumbar pain, and at length internal pain, and a deviation or lowering of the womb obliges them to wear a girdle to assist the abdominal-wall, which is too weak to support the bowels. . . . .

From what has been said it is plain that by increasing or lessening the tonic force of the abdominal muscles the lumbo-sacral curve will be lessened or increased.

[An elegant "fall" to the back, which may be a subject of admiration in the young, is often the precursor of a state of the belly after pregnancy and in advanced life which is the reverse of elegant.]

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2. Scoliosis is usually caused by the predominance or one-sided action of the extensors of the lumbar vertebræ. . . I have seen a scoliosis begin and develop in a child after wasting of the muscular mass on the right of the lumbar vertebræ. The child was brought to me when a year and a-half old, and I saw it again when six. The lumbar spine had gradually bent to the left, forming a curve the convexity of which was towards the wasted muscles, and here the sides of the vertebræ could be felt; and they were felt to have undergone a rotation on their axis. The trunk was warped to the right, and the dorsal spine had a compensatory curve in the opposite direction. The scoliosis vanished to a great extent when the trunk was bent forward, and the spine presented a lumbo-dorsal curve with backward convexity, which showed that the sound lumbo-spinal muscles had undergone no contraction. There was no sign of rickets, no inequality of the legs, and no cause other than that specified to account for the scoliosis.

I have seen many similar cases.

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[When these lateral curvatures are due to failure of muscular action alone, they are slight in amount, but, "being continued, the vertebræ get deformed by degrees in the direction of the curving and twisting of the two parts of the spine, and thus is explained the gradual increase of this scoliosis."]

Need it be said that scoliosis does not occur when the lumbar and dorsal muscles of the same side are affected at once? In such cases I have seen the formation of a long lumbo-dorsal lateral curve.

Since scoliosis originates in the extensors of the lumbar spine on one side, treatment is naturally directed to these muscles.

Faradisation, galvanisation, and gymnastics, combined with a suitable corset, have given me good results in the early stage.

Usually I localised the electricity in the muscular masses on the side of the convexity of the lumbar curve, and the gymnastics have been designed to exercise the muscles which bend the body outwards and backwards, and to cause a rotation in the opposite direction to that caused by the scoliosis.

[We know that in the early stage of scoliosis the scapula on the side corresponding to the dorsal (compensatory) convexity projects, and that its spinal border is displaced. This has been

wrongly attributed to palsy of the serratus or contracture of the deltoid.]

The habit of holding the body (*hanche*) so as to give a side twist to the pelvis may be an occasional cause of scoliosis. We may act on the pelvis in an opposite direction by making the patient sit on a chair with one half of the seat higher than the other ; the buttock opposite to the lumbar curve resting on the highest half, the lumbar spine necessarily bends to that side, and the scoliosis is replaced by an artificial curve in the opposite direction. I have used this means, more potent than the best corsets, whenever the patient sits, and it has seemed to me to be very useful in the treatment of scoliosis.

THE END.

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