An essay on wasting palsy (Cruveilhier's atrophy) / by William Roberts.

ON

WASTING PALSY.

"And, behold, there was a man which had his hand withered."

MATT. xii. 10.

"La libre vérité fut toute mon étude."

BOILEAU. Epit. v.

AN ESSAY

ON

WASTING PALSY,

(CRUVEILHIER'S ATROPHY.)

BY

WILLIAM ROBERTS, B.A., M.D., LOND.

SCHOLAR OF THE UNIVERSITY OF LONDON: PHYSICIAN TO THE MANCHESTER ROYAL INFIRMARY, FEVER WARDS, AND LUNATIC HOSPITAL; LECTURER ON GENERAL AND MORBID ANATOMY AND PHYSIOLOGY AT THE MANCHESTER ROYAL SCHOOL OF MEDICINE.

WITH FOUR LITHOGRAPHIC PLATES.

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PREFACE.

THE purpose of this little volume is to bring together, within a convenient compass, and in a convenient form, all the information the writer could lay hands on concerning a peculiar species of palsy, of which the most striking characteristic is a remarkable wasting of the disabled muscles, but which is independent of lead impregnation. Hitherto, English medical literature has been wanting in a systematic treatise on the subject; it is the ambition of this Essay to fill up that gap.

On the Continent, since 1850, this subject has attracted considerable attention, and Cruveilhier, Aran, Duchenne de Boulogne, Oppenheimer, Wachsmuth, and others, have made it the object of special study; but, among us, the existence of the disease has scarcely been recognised, and the papers of Cruveilhier seem the only ones which have elicited even the faintest echo in this country.

It was while seeking aid to the better understanding of the examples which had presented themselves to my observation, that the thought came to me, that I might render some service to my countrymen, who should be embarrassed similarly to myself, by placing within their reach the facts which had cost me so much labour to trace out and collate.

I cheerfully acknowledge my obligations to those who have preceded me in this enquiry, and especially to the studies of Aran, Wachsmuth, and Duchenne; but I have sought throughout a sounder basis for my conclusions than mere authority; and all the deductions rest entirely on the facts adduced, which I have analysed without any desire to uphold a preconceived theory. I present the facts to the reader; let him judge whether I have advocated aught which their evidence does not warrant.

While the latter portion of these sheets was passing through the press, several very interesting cases of Wasting Palsy have been published. (See Canstatt's "Jahresbericht" for 1857, band iii. p. 78; "Medical Times and Gazette," April, 1858;

"British Medical Journal," May 1st, 1858). They fully support the views taken of the nature of the complaint in Chapter VII. In two of them autopsies were obtained, and the most careful examinations—one of them by Traube—failed to detect any disease in the nervous system.

It is also my pleasing duty to acknowledge the assistance I have received from professional friends. To Dr. Wilkinson and Dr. Browne I am indebted for some valuable suggestions. Dr. Parkes, among whose pupils I have the honour to rank myself, gave me the benefit of his ripe erudition and experience; he went over the entire manuscript, and it is mainly through his encouragement that I have ventured before the public. My friends Dr. Manley and Mr. Smart have assisted me most materially in revising the manuscript, and in the irksome task of correcting the proofs.

If this Essay serve to attract the attention of medical practitioners in this country to this class of cases, and induce the publication of illustrative examples, and especially of post-mortem examinations, I shall be abundantly rewarded for my labour.

Care must be taken, however, to ensure a correct diagnosis. Several examples have already been published as wasting palsy, which are unquestionably of different nature, and either depend on metallic poisoning or belong to a hitherto undescribed class of cases.

With these considerations I consign this imperfect effort to the kindness of my professional brethren.

W. R.

Oxford Road, Manchester,
May, 1858.

Fig 1

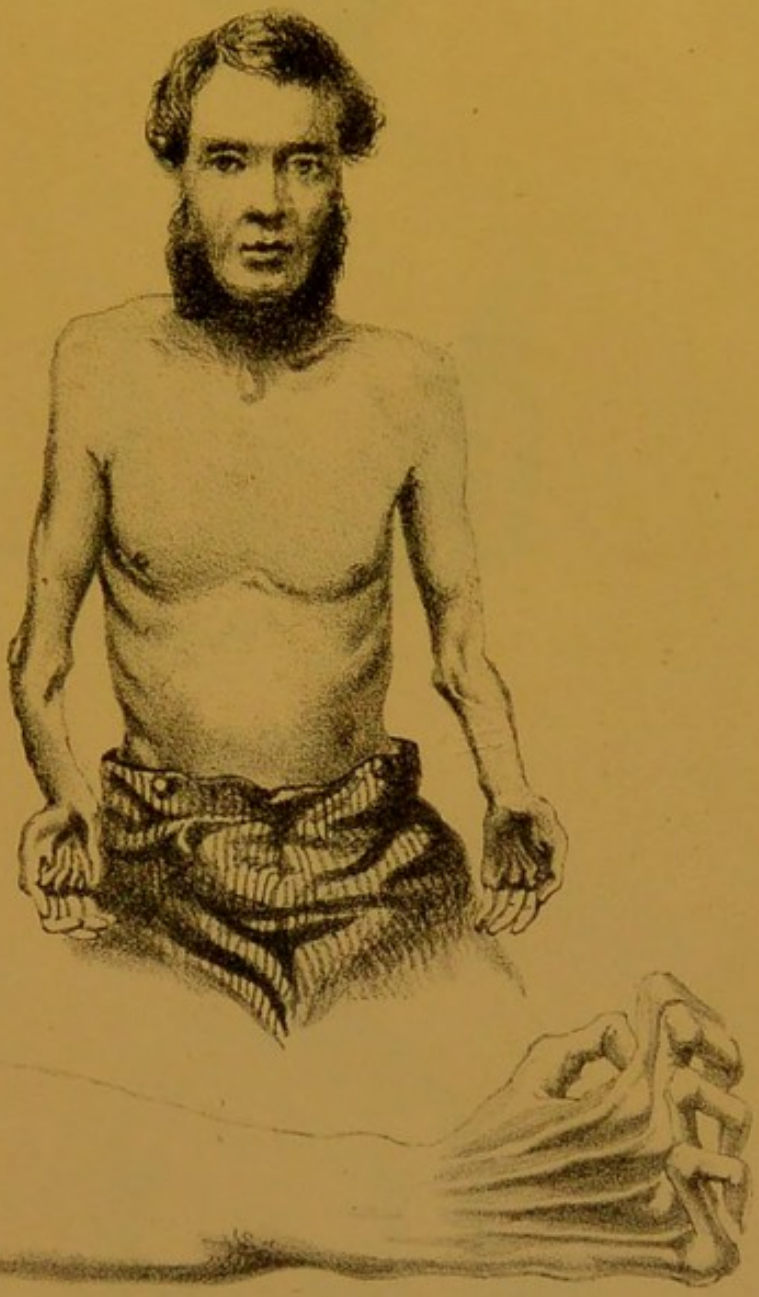


Fig 2

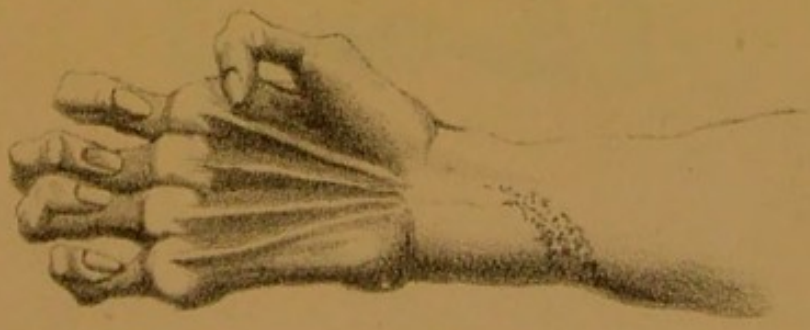


Fig 1

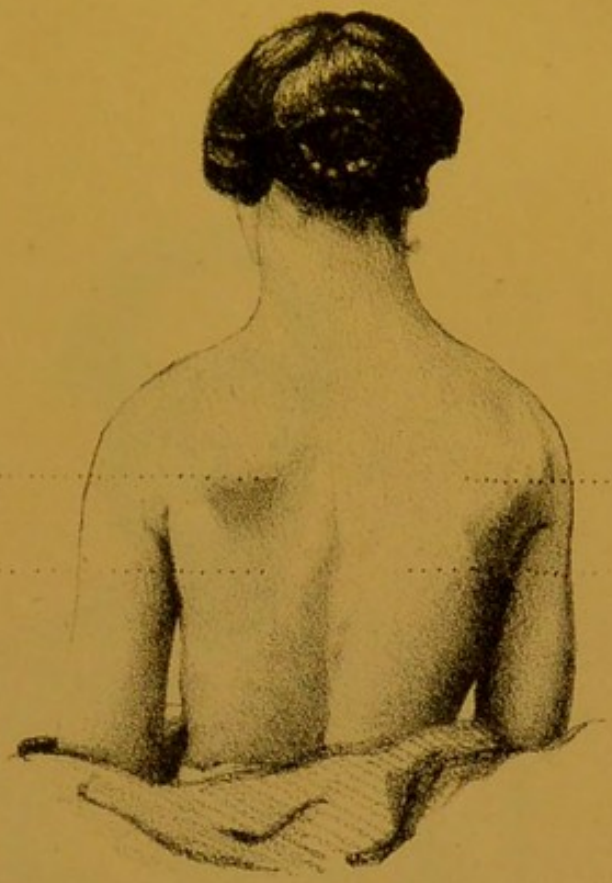
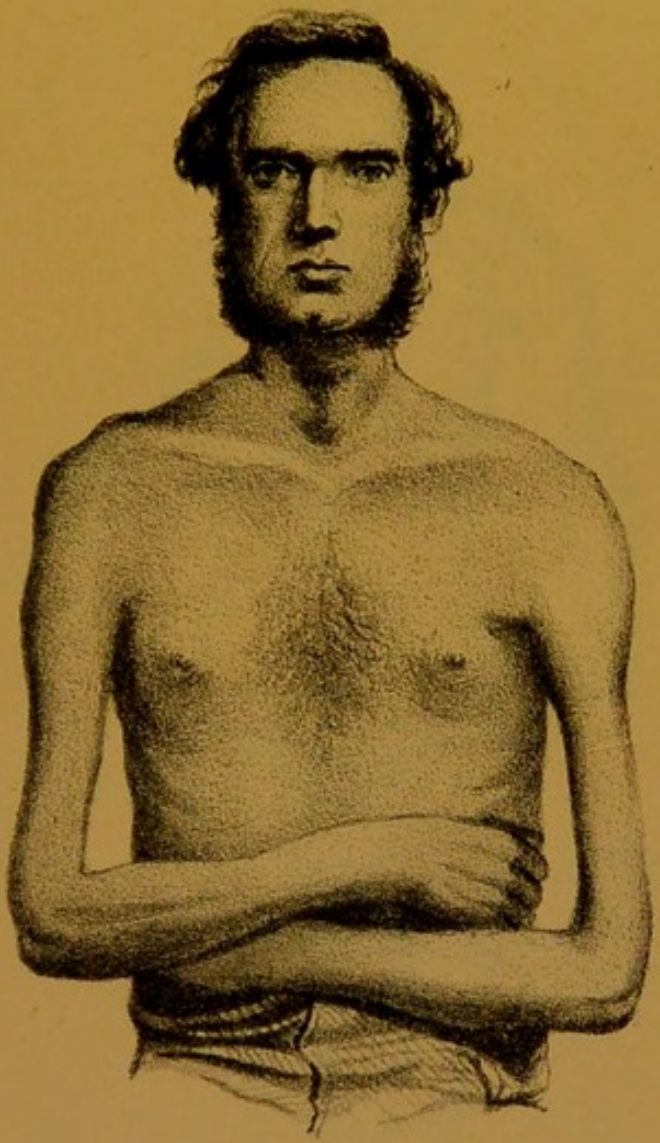
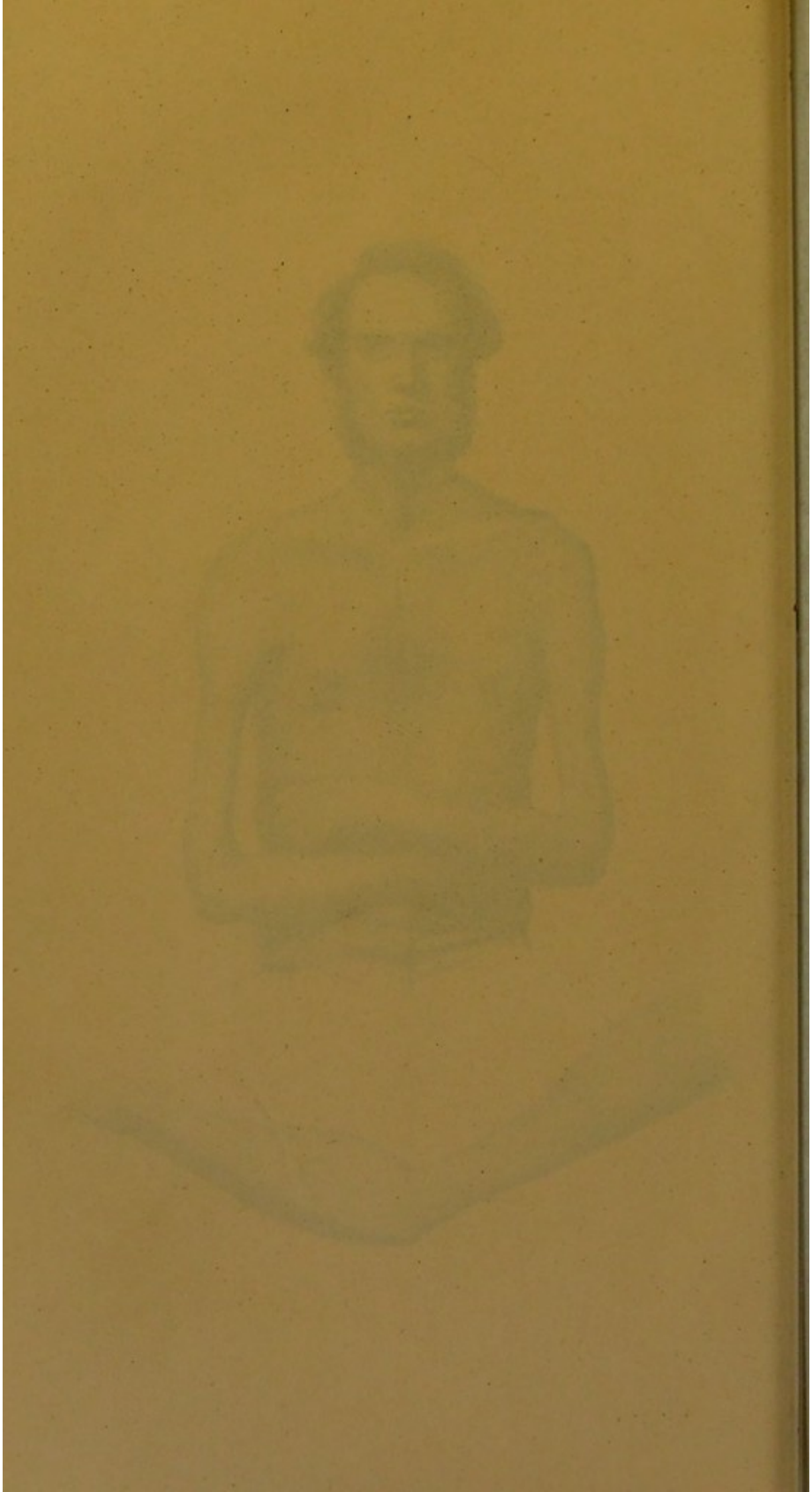
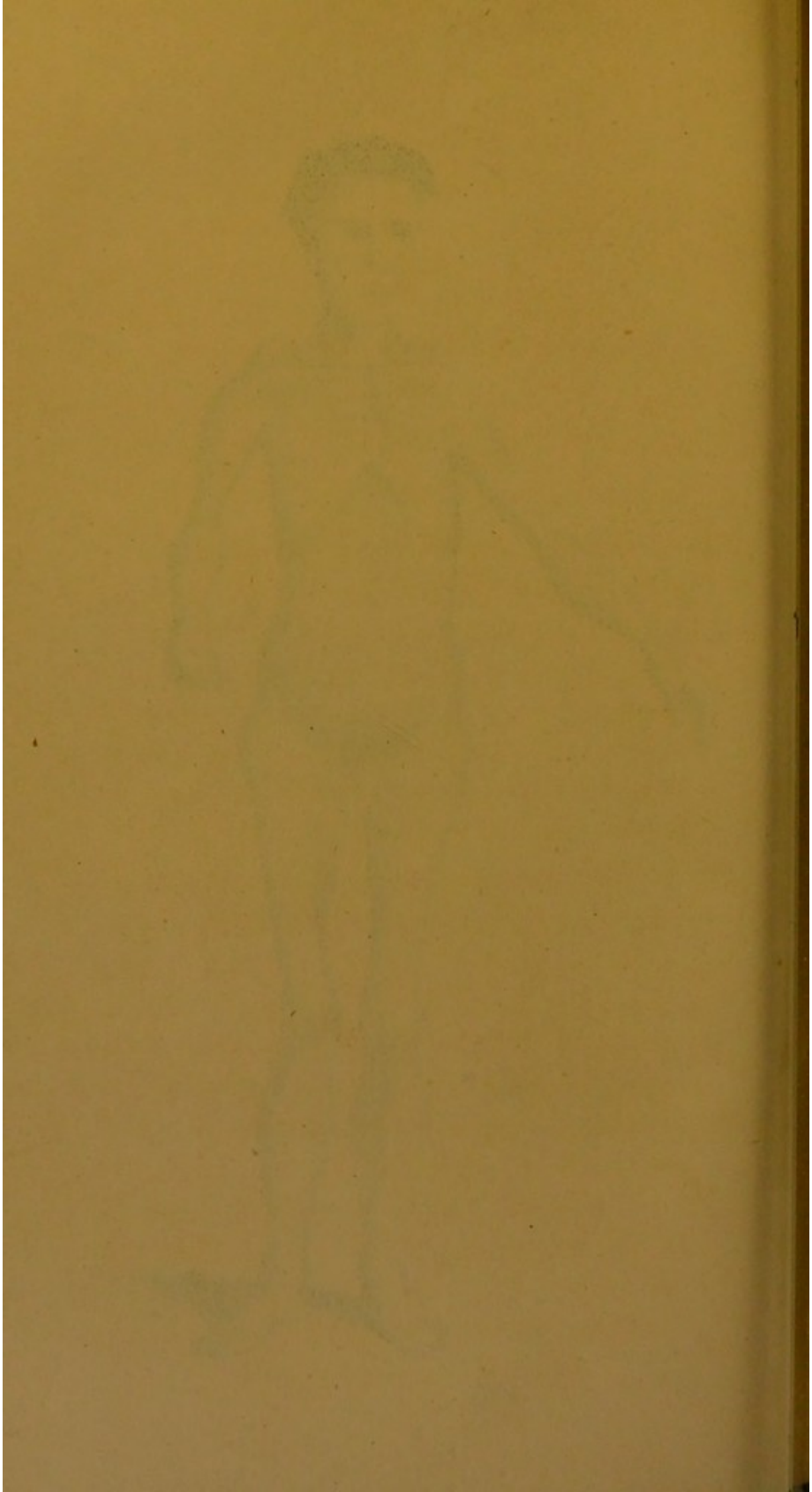


Fig 2







EXPLANATION OF PLATES.

PLATE I.

FIG. 1.—Portrait of Richard Rogers, whose case is detailed at p. 26. It exhibits complete atrophy of the muscles of the forearms, with the exception of the Pronator Teres. The hands are deprived of the fleshy masses constituting the thenar and hypothenar eminences; and the track of the flexor tendons is distinctly seen in the hollow of the palms. These hands are examples of the "main en griffe."—See p. 103. Drawn from a photograph.

FIG. 2.—Dorsal and palmar aspects of the "main en griffe"—exaggerated. (After Duchenne de Boulogne).

PLATE II.

FIG. 1.—Portrait of Emma Walker, whose case is detailed at p. 40. It illustrates the dislocated condition of the scapulæ from wasting of the muscles which brace it to the trunk.

a. Projection produced by the inferior angle of the scapula, which is seen to be raised very nearly to level of the top of the shoulder.

b. Natural position of the lower angle of the scapula when it lies evenly against the ribs.

FIG. 2.—Front view of the same. The pectoral muscles, and the clavicular parts of the deltoids, especially the right, are completely atrophied. The infra-clavicular regions are deeply hollowed, while the forearms and the lower part of the upper arms are very athletic.

Both taken from photographs, which only imperfectly convey an idea of the striking appearance presented by the patient.

PLATE III.

Portrait of Dr. Reade's patient. The pectoral, deltoid, biceps, and triceps muscles are greatly wasted, while the forearms and hands are normal. From a photograph. See "Dublin Quarterly Journal," Nov., 1856. Case 52 in the Analytical Table.

PLATE IV.

Copy of a wood-cut exhibiting general wasting palsy of the trunk and extremities. The thorax and upper arms are quite devoid of muscles, but the forearms and hands are free. Above the shoulders are seen the projecting tops of the displaced shoulder-blades. The front of the thighs are also seen hollowed from atrophy of the Quadriceps femoris. The legs are athletic. See Duchenne. "De l'électrisation localisée," p. 580. Case 105 in the Analytical Table.

ADVERTISEMENT.

I very much regret that I had not determined to introduce Plates for the Illustration of this Volume until after the printing of the Sixth Chapter. This will account for the want of reference to them in the text.

W. R.

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CHAPTER I.

HISTORICAL INTRODUCTION.

Cases of excessive wasting of the muscles of one or more limbs, independent of any well-defined cause, have from time to time been observed, and records of them are found scattered in various published works. They are usually introduced as *extraordinary* or *anomalous* cases, and are referred to in systematic treatises as instances of *creeping palsy*, *lead palsy without lead*, *peripheric paralysis*, and *partial or local palsy*.

Dr. Cooke relates the earliest example known to me. He calls it a case of *anomalous hemiplegia*. "An officer of high rank in the army, who is now about sixty years of age, was in the year 1795 affected with a diminution of power in the right hand. This complaint increased, notwithstanding a variety of modes of treatment, till the year 1800; when, after a course of mercury recommended by Mr. Cline, its further progress was stopped, since which time the disease has remained stationary. The peculiar circumstances of this case are the following:—The muscles of the left arm, from the shoulder to the elbow, are much wasted and greatly diminished in power,

while the muscles of the forearm are not at all lessened in size, and but little in power. The state of the right side is just the reverse, the muscles of the upper arm being of their natural size and possessing their full power, while those of the forearm are much wasted, and their motion, especially that of the fingers, is almost entirely abolished. In all other respects this gentleman appears to be perfectly well. No cause for this disease can be assigned, nor did any method of treatment afford the slightest relief till the mercurial course was adopted, when the progress of the disorder was arrested in the year above mentioned; and since that time no attempts have been made to remove the complaint,—yet it does not increase."*

In the Collected Works of Caleb H. Parry † the case of a man of fifty years of age is reported, in whom, after he had suffered eleven months from diarrhœa, the muscles of the two legs and hands wasted away. There was, in addition to loss of power corresponding to the atrophy, a great sensitiveness to cold.

Sir Charles Bell ‡ met with several cases in his

* Dr. Cooke, On Palsy, p. 31. Lond. 1822. Dr. Graves cites this case in his Clinical Lectures on the Practice of Medicine, Lect. XXXIII.

† Collected Works of Caleb H. Parry, p. 523. Lond. 1825.

‡ The Nervous System of the Human Body, by Sir Charles Bell, third ed. Lond. 1830. Appendix, p. clxi.; and Nervous System with Additions, p. 432.

own practice; and his acute understanding did not fail to perceive how widely they differed from local palsies dependent upon injury to muscular nerves or on lesion of the nervous centres. He has left a record of four of these:—

In the first case, an ironmonger, aged twenty-two, the ball of the right thumb was totally atrophied, and the long extensors of the thumb in the forearm were equally destroyed, while the rest of the hand and forearm, and all the other parts of the body, were in full vigour.

In the second case, that of a young gentleman of fifteen, we are told—"The muscles of the thumb are not wasted, and the flexor muscles of the wrist and fingers are powerful: he can grasp his father's hand so as to make him cry out. Yet the extensors of the wrists and fingers are weak, so that the hand remains generally bent and at an angle with the arm. Whilst the forearm is firm to the feeling as you grasp it, the muscles of the arm are wasted and loose, so that you can feel all the processes of the humerus from its upper to its lower end; the deltoid muscle is also quite gone. The rotation and motion of the arm are very curiously performed by the muscles inserted into the scapula, which are firm and strong, so that the arm is thrown about by the rotation of the scapula upon the chest. The muscles which come down from the neck to the shoulder are particularly strong, and it is by them that the scapula

is heaved up and a secondary kind of motion given to the arm. All the muscles which are for bracing down the scapula to the chest and drawing them backwards, are wasted, and the inferior angles of both the scapulæ start out three inches from the ribs. It is astonishing with what energy he can fling his arms about, by those muscles alone which come from the neck to the shoulder; for example, he jerks on his coat and draws it upon his back solely by the action of the muscles of the neck. When undressed, he can swing his arms round and round; but it is by adjusting the action of raising the scapulæ with the gravitation of the lower extremity that he contrives to do this, and seems to possess a more extensive influence on the muscles of the arm than he actually does." Some time after—"I was again visited by his mother, when she reported that his muscular strength had declined, particularly in the right leg, and that it required two men to put him into the carriage. His spirits are excellent, his remarks shrewd, and his education is proceeding. He has grown considerably."

The third instance concerns the lower extremities, and occurred in "a young gentleman about eighteen. All the muscles of the lower extremities, hips, abdomen, are debilitated and wasted. The extensor quadriceps femoris of both limbs is wasted, and yet the vasti externi have

not suffered in an equal degree. A firm ball, remarkably prominent, just above the knee joint, marks the place of the vastus externus, while the rectus is quite wasted and gone. The upper part of the body, the shoulders and arms, are strong. There is no defect perceptible in the evacuation of the bladder or of the bowels."

A coal-heaver, aged forty, was the subject of the fourth case. In the course of three years the flexors of the left hand and fingers and the ball of the thumb, together with the extensors of the thumb, had almost completely vanished, and the ball of the thumb was replaced by a hollow between the metacarpal bones. The power of flexion was lost to the hand, but that of extension remained perfect. The upper arm and shoulder were sound. On the right side it was the upper arm and shoulder that were affected, while the forearm and hand preserved their vigour. "The trapezius and latissimus dorsi and the other muscles uniting the scapula to the trunk were vigorous and plump, but the supra-spinatus, the infra-spinatus, and the deltoid appeared atrophied to the last degree. Owing to the atrophy of the deltoid, the acromion, the coracoid process, and the caput humeri were sharply defined under the skin. The entire biceps humeri was very much reduced, and felt like a thin cord from the shoulder to the elbow when the skin was thrown into loose folds. The triceps,

on the other hand, was as strong and full as in a robust man. The patient was unable to raise his arm, and could only swing it by moving the whole body. He was equally unable to bend his elbow joint, though he could extend it with full force. * * * General health unimpaired."

In none of these cases was there any loss of sensation or any symptoms referrible to the brain and spinal cord. These are by far the most important instances of this disease published before 1850, and they illustrate in a striking manner some of its most remarkable features. It is seen to attack isolated groups of muscles, reducing them to mere membranes, while those around are plump and vigorous; falling capriciously in one case on the muscles of the thumb, in another on the two shoulders, in a third on the forearm and hand on one side, and the shoulder and upper arm on the other; according extraordinary immunity to one or a few muscles amid the general ruin; and, lastly, not disturbing the even course of the vegetative functions, nor impairing in the least the operations of the intelligence. Pain, of a neuralgic character, was observed only in one of the patients.

Introductory to the history of these cases, Sir Charles Bell observes—"This is an obscure subject. The paralysis does not extend to a part of the arm or leg, nor is it a defect reaching so far up the limb, nor so far down the limb, but it is

an affection of the muscles naturally combined in action; although these muscles lie in different parts of the extremity, and are supplied by different nerves, as they are by different arteries. For example, the muscles of the thumb may be affected, but then the wasting will not be confined to the short muscles of the ball of the thumb but will extend to those muscles of the thumb which lie upon the forearm, and these wasted muscles are lying in contact with others which are plump and powerful; or, sometimes, all the extensor muscles will lose their power while their opponents will preserve it, producing a characteristic position of the limb. It will sometimes happen that one class of muscles having suffered, another class will come into play and be developed by unusual exercise. I have found the action for writing gone, or the motion so irregular as to make the letters be written zigzag, whilst the power of strongly moving the arm or fencing remained." Of their nature he remarks—"These affections imply a very partial disorder of the nerves. A disease of the brain, or a disease in the course of the nerves, must influence the whole limb or that portion of it to which the nerve or nerves are distributed. But in these cases particular subdivisions of the nerves, included in the same sheaths, or running the same course, are affected. I am inclined to attribute such partial defects to visceral irritation. In that

case it must still be the influence of the sympathetic nerve which produces it; and yet it seems, on the other hand, impossible to account for such entire loss of motion without the intermediate influence of the brain."

From these quotations the reader may apprehend that Bell had distinct and well-defined ideas about these cases. He correctly grouped them together, and apart from other local palsies proceeding from injury to the spinal cord or a nervous trunk, and from lead palsy. He went some way towards furnishing the clinical history of a new disease, and speculated with much ingenuity about the initial lesion on which the muscular wasting depended. His merit has not been fully acknowledged by continental writers. If he did not explicitly announce the discovery of a new disease, he so far foreshadowed that discovery that a portion of the honour must attach to his memory.

The foundation thus so ably laid by Bell was not built upon by his countrymen; they failed to see the significance of his observations and to follow up his speculations, and it remained for a distinguished Frenchman, M. Cruveilhier, to bring forth into full day what had dawned on the mind of Bell.

Writers contemporaneous with, or subsequent to Bell, had far less correct ideas on the nature and symptoms of *wasting palsy*. Abercrombie*

* Abercrombie, On the Brain & Spinal Cord, p. 419. Edin. 1828.

published a most marked case, in which both upper arms were greatly wasted, while the forearms and hands were sound and strong; but with it he groups a case of infantile paralysis with arrested development, and two cases of intermittent anæsthesia.

In the seventh volume of the "London Medical Gazette" * is an account of three very striking cases by Dr. Darwall. Two of them were women, in whom the atrophy was confined to the muscles of the shoulder and upper arm; the forearm and hand being sound. It involved both sides in one, but only the left side in the other. In the third case the entire upper extremities had been reduced to complete uselessness by atrophy of the muscles, and hung helplessly by the side. With these he reports three other cases of palsy; one of which appears to have been hysterical, and the other two rheumatic. The determining cause could be traced with some confidence to overwork; and Dr. Darwall based his view of their pathology on this supposition. He thought the nervous trunks in the axilla were injured by the straining necessary in lifting or carrying heavy weights; and that the muscles wasted from a withdrawal of the nervous supply, on which their nutrition and functional activity depended. The emaciation was further precipitated, in his opinion, by the immobility of the muscles. He recommended electricity as a ra-

* "Lond. Med. Gazette," p. 201, vol. vii.

tional remedy; though he did not succeed with it in the three undoubted cases.

In treating of muscular atrophy, Dr. Herbert Mayo * relates two easily recognized instances of wasting palsy. He also describes fatty degeneration of the muscles, and quotes a case from Vicq-d'Azyr in which the psoas, iliacus, gluteus medius and minimus, adductors and deep posterior muscles of the leg and plantar muscles were completely changed into cellular fat, without traces of remaining muscular fibre.

This completes the first era in the history of wasting palsy. It can scarcely yet be said to have a name or place in the nosological scheme. In England alone were there any well-marked cases recorded; and, even here, no progress, but rather the reverse, had been made in its study since the time of Bell.

We hear nothing more of it until 1849, when M. Duchenne de Boulogne presented a Memoir to the Institute of France entitled *Atrophie musculaire avec transformation graisseuse*. And in the following year M. Aran published a more extended Essay in the Archives Générales, under the title *Recherches sur une maladie non encore décrite du système musculaire (atrophie musculaire progressive)*, in which the clinical history of

* "Outlines of Human Pathology," p. 117. Lond., 1836. Rokitansky quotes one of these cases in his "Lehrbuch der Pathol. Anat.," p. 215, vol. ii., third ed.; and Dr. Reade, of Belfast, extracts it as an original observation. Its real derivation is as stated in the text.

the disease is fully exposed by the analysis of eleven cases. A year later (1851) M. Thouvenet made it the subject of his inaugural dissertation. But these three productions were inspired by the observations and teaching of Prof. Cruveilhier,* which were not given to the public until 1853. I shall postpone, therefore, what I have to say about the three former until justice has been done to the important labours of the latter. He alone can claim the merit of having established the existence of a new species of paralysis, totally different in its course, symptoms, and nature from those dependent on lesions of the cerebro-spinal centres or on metallic poisoning. It is to him, also, that we owe the demonstration, by post-mortem examination, that the disease is seated mainly in the muscular system, which is progressively destroyed, while the brain and spinal marrow may remain, apparently at least, perfectly sound.

In the cases which had attracted M. Cruveilhier's attention, the atrophy was not confined to a limb or a part of a limb, as in the examples related by Bell and Darwall, but had become general, to the extent of involving nearly all the voluntary muscles throughout the body. In the Memoir which he read before the Académie de Médecine he gives the following account of the

* Cuveilhier read his Memoir to the Académie de Médecine in March, 1853. It was published in the "Archives Générales," p. 561, May, 1853.

observations which had enabled him "to establish clinically, since 1848, the existence of this remarkable form of paralysis, under the title of *Gradual paralysis of movement from muscular atrophy, (Paralysie graduelle du mouvement par atrophie musculaire.)*"

In 1832 he had attended a general's widow of forty years of age, in whom there existed a widespread atrophy of the muscular system. The upper extremities were first affected, and then the face, which lost all expression from destruction of its subcutaneous muscles. The articulation of sounds was slow, monotonous, and incomplete. Sensation was perfect in every part, and the nutritive functions were in a state of the highest efficiency. The intelligence was unimpaired. A lesion of the spinal cord was diagnosticated with confidence. As the disease advanced the atrophy involved the muscles of the lower extremities, and progression became impossible. In the course of a year it had invaded nearly every voluntary muscle, and finally reached the muscles of deglutition and respiration, necessarily threatening speedy dissolution. In the midst of this complete destruction of muscular power—without expression in the face, and incapable of uttering an intelligible sentence—this lady preserved the most exquisite sensibility in the skin, and her judgment was sound to the last moment. Digestion was vigorous, and defecation and micturition were performed normally, and with the utmost

regularity. The muscles of mastication and those moving the eye-ball had been alone spared; and, by means of the latter, she was able to express, in some imperfect manner, the workings of her mind. It was clear that the least impediment to respiration would prove fatal; and one morning she was found dead in bed.

"We obtained an autopsy. I had, with all the other medical men who had seen the case, diagnosticated a profound injury to the spinal cord. What was my amazement to find the spinal cord perfectly intact, as well as the constituent parts of the encephalic mass, cerebrum, cerebellum, and pons Varolii!" He thus gives expression to his astonishment and perplexity,—"Quoi! me disais-je, tout un système locomoteur est paralysé, depuis les muscles des membres jusqu' aux muscles de la respiration, de la phonation, de l' articulation des sons, et rien, absolument rien au centre nerveux céphalo-rachidien!" The muscles and nerves were not examined.

The case made a deep impression on his mind and he continued to look-out for others; but, during the eight years that he was physician to the Salpêtrière, he met with none similar.* Subsequently at the "Charité" several cases fell under his notice, which, however, remained barren of results from want of autopsies; and it was not

* This is accounted for by the advanced age of the inmates in the Salpêtrière, for this disease is almost confined to early and middle age.

until 1848 that a new opportunity offered, of solving the long pending problem.

The subject of this second observation was a young shepherd, eighteen years of age, named Legrand. He was afflicted with precisely the same symptoms as the general's widow. He died of smallpox before the atrophy had completed its work of destruction. As before, the brain and spinal marrow were found perfectly healthy. It was clear the source of the disease must be sought-for either in the nervous trunks or in the muscles themselves. On examination, the muscles were found wasted in different degrees; some yet preserved their natural colour, and these had been observed clinically to have retained their contractility; others were reduced to a pale rose, others to a pale yellow or buff colour; but a very great number were the subjects of a fatty degeneration. It was noted as remarkable, that not only were muscles functionally connected and contiguous in position unequally affected, but that individual bundles in the same muscle had not suffered to a like degree. Thus in the same muscle red fasciculi were found side by side with rose-coloured and even fatty ones—demonstrating the independence as relates to nutrition and action not only of different muscles but of the different fasciculi of the same muscle.

From this period (the summer of 1848) Cruveilhier taught in his lectures the existence of a new species of palsy, and here dates the commencement of its separate history.

M.M. Duchenne de Boulogne, Aran, and Thouvenet were aware of these observations.

M. Duchenne,* who had then commenced those studies on the local application of electricity which have since borne such important fruits, occupied himself chiefly, in the memoir above alluded to, with the anatomical characteristics of the disease. He insisted that it was "solely a lesion of nutrition characterised by atrophy and fatty transformation of the muscular fibre," and proposed the name *Atrophie musculaire avec transformation graisseuse.*

The Essay of M. Aran† is a most important one, and it entitles him to a place second only to Cruveilhier in the history of wasting palsy. It is based upon the facts of eleven cases, from the analysis of which he deduces, with rare skill, the course, symptoms, diagnosis, prognosis, and treatment—in short, the entire clinical history of the disease. He divides his cases into two classes, according as the atrophy is confined to the extremities (eight cases); or threatens the muscles of the entire body (three cases.) He has the merit of having shown that the partial form passes by insensible gradations into the general, and that they are really one and the same disease.

* The Memoir of M. Duchenne was presented in 1849. An analysis of it may be found at p. 622 of his work "De l' électrisation localisée et de son application à la physiologie à la pathologie et à la Thérapeutique." 8vo., Paris, 1855.

† "Arch. Gén.," Sept. 1850, p. 5, tome xxiv.; continued at p. 172.

Two important symptoms, likewise, were first pointed out by him, namely, cramps in the affected limbs and muscular vibrations—little rapid twitching movements of the individual fasciculi, spreading in swift undulations beneath the skin, but not causing a contraction of the entire muscle nor any motion of the limb.

Like Darwall, M. Aran insists on the importance of over fatigue of the muscles as a cause of the complaint. To his thinking "it is a disease of the muscular system, and the morbid action is primitively and exclusively in this system.*"

At the commencement of his Essay, M. Aran gives a short account of the cases reported by Bell, Abercrombie, and Darwall; but he by no means does justice to the observations of the first, while he gives undue prominence to those of the last. He failed to find allusion to this complaint in the more ancient writers, but in Van Swieten's Commentaries he discovered the following passage:—"Crebram occasionem habui hunc morbum videndi et tractandi, et summa cum commiseratione vidi, deltoides musculos in utroque brachio evanuisse quasi, ita ut sola cute tectum humeri articulum tangere potuerim. Torosam illam carnem musculosam, quæ primæ phalangi pollicis in parte manum spectante apponitur, & pollicem magna vi, dum agit, versus manus palmam adducit, pariter adeo emarcuisse vidi, ut ne vestigium ejus fere superesse videretur, et miseri illi nullam vim ma-

* "Loc. Cit.," p. 210.

nubus exercere potuerint."* This quotation refers, however, unquestionably to the palsy following Painters' Colic, for in the preceding sentence he observes,—"In illa paralysi, quæ colicam Pictonum dictam sequi solet, omnium frequentissime illa atrophia partium paralyticarum observatur," &c. Nevertheless, when we see in p. 357 that he regards *Colicam Pictonum (dictam)* as capable of being produced by other causes than lead,† it may be conceded that Van Swieten had seen instances of this wasting palsy, and that he had them in view in writing the passage above quoted; but it is very clear that he did not distinguish them from those produced by lead.

M. Thouvenet's thesis is founded on some cases collected in the "Charité," and chiefly on the case of the mountebank Lecompte, whose history is related in the next chapter. He contends, in opposition to Aran, that the disease is primarily located in the peripheral nerves, and that it must be classed with rheumatic affections.‡

On the 9th of December, 1851, Dr. E. Meryon§ read a paper before the Medico-chirurgical Society on *Granular and fatty degeneration of the volun-*

* Van Swieten, "Com. in Boerhaave Aphorism," p. 370, t. iii.

† "Frequentes habui occasiones mirabilem hunc morbum videndi, et licet non negem, illum ab aliis causis nasci posse tamen frequenter," &c.—"Loc. Cit.," p. 357.

‡ M. Thouvenet was an interne of M. Cruveilhier. His Thesis appeared in 1851. A complete analysis of it occurs in "Gaz. des Hop.," Nos. 143 and 145. 1851.

§ "Med. Chir. Trans.," p. 73, vol. xxxv.

tary muscles. He was apparently unaware of the labours of Aran and Duchenne, and his observations were made quite independently of any foregoing researches. He gives an account of a gentleman's family in which three boys were the subjects of general muscular degeneration. The eldest died at sixteen, after having suffered between eight and nine years from gradually increasing failure of the muscular power. The disease commenced in the lower limbs, and gradually involved nearly all the voluntary muscles. After death Dr. Meryon found the brain and cord healthy, but the muscles extensively destroyed and partly converted into fat and granular matter. Two brothers of the deceased were attacked in a similar way; but the sisters—six in number—remained quite healthy. He refers, in addition, to two other families, in which the disease appeared to be hereditary. In all of them it assumed the general—not the partial—form; and its manifestation was further singular in confining itself to the males and sparing the females. The paper is illustrated by excellent drawings of the appearance of the muscles under the microscope. He repudiates the idea that the disease depends on a defalcation of nervous energy, and argues that the primary morbid change is a default of nutrition in the muscular fibres.

The Memoir published by Cruveilhier in 1853 embraced, as I have already explained, observa-

tions made prior to 1848. These have been discussed in their proper place. But it contained, in addition, the history of a third* case, which terminated fatally in January, 1853. This example, which is related at length in the next chapter, was more thoroughly studied than the others; and the post-mortem examination revealed, in addition to fatty and granular muscular degeneration, a most marked atrophy of the anterior roots of the spinal nerves, and of some of the motor trunks in their course to the muscles. This discovery led Cruveilhier to conclude that the cause of the disease "resided, not in the muscles themselves, but in the anterior roots of the spinal nerves."†

The *Académie* devoted two sittings to the discussion of Cruveilhier's Memoir. M. Bouvier contended that the disease was a primary atrophy of the muscles. M. Parchappe supported the opinion of the author of the Memoir; while M. Guerin‡ regarded it as "paralysie rhumatismale périphérique du mouvement!"

To complete this account of M. Cruveilhier's labours, I will anticipate a little. In December,

* This case—the Saltimbanque Lecompte—is the most famous of all the examples of wasting palsy. It is related by Aran, "Archiv. Gén.," p. 30, t. xxiv.; twice by Cruveilhier, in two separate Memoirs; by Duchenne, "Electrisation Localisée," pp. 481 and 554; and by Wachsmuth in Henle and Pfeufer's "Zeitschrift," p. 35, heft. 1 and 2, band. vij.

† "Arch. Gén.," Mai, 1853, p. 602.

‡ "Loc. Cit.," p. 626."

1855, he published a fourth case,* which coincided in almost every respect, with the third example; but his opinion had undergone a little change with respect to the location of the primary lesion. This he now placed in the cord—the grey matter, where, he insists, the anterior roots take their rise.

Since 1853, several cases have been published in France and Germany, and especially four fatal ones by Valentiner, Schneevoogt, Landry, and Virchow. These do not all bear out Cruveilhier's doctrine of the origination of the disease in the anterior roots. In some of them there was distinct softening of the spinal cord.

Two separate Essays have appeared lately in Germany, in which the subject is discussed with great learning and labour. One is an inaugural dissertation by G. Oppenheimer,† and the other an Essay by A. Wachsmuth,‡ in Henle and Pfeufer's "Zeitschrift" for 1855.

Oppenheimer, after having given the details of three new cases, of which one was fatal, discusses at length the question—What is the nature of progressive muscular atrophy? He arrives at

* This case is published in an Appendix to a second Memoir read at the Institute of France (Académie des Sciences), Dec., 1855.—See "Arch. Gén.," Jan., 1856, where it is fully reported.

† "Ueber progressive fettige Muskel-entartung," von Dr. G. Oppenheimer, Habilitations-schrift, Heidelberg, 1855.

‡ "Ueber Muskel-atrophie," von Adolph Wachsmuth, zu Gottingen. Henle and Pfeufer's "Zeitschrift für rationelle Medicin," p. 1, heft. 1 and 2, band. vij.

precisely the same conclusion as Meryon; that the primary evil is malnutrition of the muscles.

Wachsmuth, in addition to a case published by himself, analyses fifty-nine others which he had collected from various sources, and enters fully and systematically into the entire pathology of the disease. His Essay shows great research, great labour, and is by far the most comprehensive, as well as the most severely critical account of the disease yet published.

In 1855, Duchenne published his large work (8vo. pp. 926,) on the "Local application of Electricity." In this work, although written without special reference to wasting palsy, there are a very great number of facts bearing on its diagnosis, symptomatology, and therapeutics; and I am indebted to it for many valuable and interesting observations.

In Canstatt's Jahresbericht for 1856, Dr. Eisenmann gives abstracts of several new cases, and examines into the nature and seat of the disease—which he denominates *Paralysis Atrophica*—by the aid of some newly published autopsies, where the spinal cord had been found softened and otherwise changed in structure; and he deduces the belief that the original departure from health is a dynamic alteration in the spinal cord similar to that which causes tetanus.

In addition, a few cases have been published at home. One is from the pen of Dr. Chambers,*

* "Med. Chir. Trans.," p. 19, vol. xxxvii,

and is remarkable from being associated with mollities ossium. Dr. Brittan,* of Bristol, relates the history of two more; and to Dr. Reade,† of Belfast, we owe the description (with portrait) of a fourth example.—Abstracts of these are given in the table of cases at the end of the succeeding chapter.

From all these sources, and others which will be mentioned in their proper place, I have gathered together a hundred cases; and from the facts supplied by them with five examples that have fallen under my own observation, I propose, in the ensuing pages, to consider the clinical history, nature, and treatment of wasting palsy.

Before proceeding, however, I have some considerations to offer on the nomenclature. Short as is the time since wasting palsy has taken rank among recognised diseases, it is rich in names. M. Aran called it *Atrophie musculaire progressive*, and this is the name by which it is most widely known. Exception has, however, been taken to this designation on two accounts. In the first place, all the cases are not *progressive;* indeed, the larger number become stationary after a while, and others recover. Again, it is asserted by Cruveilhier, Eisenmann, and others, that the nerves or nervous centres are at fault anterior the muscles, and that the atrophy of the latter is a

* "Assoc. Med. Jour.," March 7, 1857.

† "Dublin Quarterly Journal of Medical Science," Nov., 1856, p. 394.

secondary process. Accordingly, they introduce the word *paralysis* into the name, and Cruveilhier designates it in his last Memoir *Paralysie musculaire atrophique*, and Eisenmann, expunging all mention of muscle, calls it simply *Paralysis atrophica;* which is not a bad name did it not equally apply to lead palsy, and palsy following injury to a motor nerve. A writer in the "Gazette des Hospitaux"* suggests *Atrophie musculaire paralytique*, and Duchenne insists that it should be named *Atrophie musculaire avec transformation graisseuse*. Each writer endeavours to square his nomenclature to the opinion he holds of its essential nature.

Were I inclined to follow the same rule, I should call it *Idiopathic degeneration of the voluntary muscles.*† But this is too cumbrous to be advantageous, and it may not prove correct in the end, so I have substituted the more homely name of *Wasting Palsy;* which, if not a hidden mine of pathological signification, has the advantage of being plain English, and convenient for tongue and pen.

One more suggestion. Diseases have been sometimes named after their discoverers, or after those who have taken the most prominent part in elucidating their histories. Our own countrymen

* "Gaz. des Hop.," Dec. 25 and 27, 1856.

† See Chap. VII., where the objections to the preceding names are made apparent in the discussion of the nature of the complaint.

have been especially favoured in this respect. We still speak of *Pott's fracture* and *Pott's gangrene.* Dr. Bright's name is immortalised in connection with dropsy and albuminuria; and France and Germany have made *Maladie de Bright* and *Bright'sche Krankheit* household words. And, what is odd, these designations have outlived the more ambitious names—*Granular disease of the kidney*, *Néphrite albumineuse*, and others—which contained some opinion as to the pathology of the disease. Still more recently, Prof. Trousseau has rendered famous another English name; and *Maladie d'Addison* has taken its place in the nosological series. May not we, then, pay a graceful and not inappropriate tribute, to the distinguished and now venerable professor of pathology to the Paris faculty, and attach his name to the lesion which his labours have so much served to make known. I shall occasionally venture to do so; using the term *Cruveilhier's paralysis* or *Cruveilhier's atrophy*,—leaving it to my countrymen to sanction or repudiate its employment.

CHAPTER II.

GENERAL PHYSIOGNOMY.

For the correct and confident diagnosis of any disease nothing is so essential as a familiarity with its general physiognomy. No array of symptoms present to the mind, no number of anatomical facts remembered, will give such certainty to the conviction as having witnessed the disease in the living subject. Failing this, the next best thing is the perusal of a succession of individual histories of the disease; so that by a repetition of characteristic examples a general idea may be conveyed to the mind, which shall serve as a guide and reference during the subsequent analysis of its pathology.

I shall, therefore, take this opportunity of relating the five cases which I have myself observed. They illustrate the partial form of the disease. Four concern the upper extremities, and offer good examples of its more common manifestation; while the fifth is an instance of a rarer variety, in which the atrophy is confined to the lower limbs. Of that more fatal type in which the voluntary muscles of the entire body—both trunk and extremities—are involved in one common destruc-

tion, I have seen but one illustration; and of that I have not preserved notes. Its subject was a young woman under the care of the late M. Valleix. She died of smallpox before the wasting of the muscles had reached its term. To fill up this hiatus I have borrowed two well-marked cases of the general disease which had run to a fatal issue, and in which careful post-mortem examinations were made. The first is from the hand of Cruveilhier, and the second from that of Dr. Th. Valentiner, of Kiel.

Case I.*

The earliest observed of my cases came under my notice while holding the office of Physician's assistant at University College Hospital, London. The subject of it had suffered complete atrophy of the forearms and hands.

Richard Rogers, aged thirty-nine, a shoemaker by trade, was admitted into the hospital under the care of Dr. Garrod, Sept. 14th, 1852, when the following particulars of his previous history were obtained:—

He is a native and inhabitant of Caernarvon. Of medium stature, and rather spare habit of body, his general health has been remarkably good; he states that he has never been a day ill in his life. His circumstances are easy; and enquiry fails to detect any hereditary taint. None

* I have to thank Dr. Garrod's kindness for the notes from which the above account is drawn up.

of his family or neighbours were ever afflicted similarly to himself. He has never lived near lead works, nor has he in any way been concerned with occupations likely to cause saturnine poisoning. His work has not been hard, and he has never been taxed to carry heavy weights. Three years before the present affection came on, he contracted syphilis; for this mercury was administered, and he is inclined to connect the origin of his complaint with the untoward operation of the metal on his system.

It is now two years (Autumn, 1850,) since the hands commenced to fail. He then, for the first time, perceived a weakness of the right thumb, and an inability fully to extend its first phalanx. This did not disable him from pursuing his occupation. Five or six weeks afterwards, he found that the index finger of the same hand was getting weak; and, subsequently, the remaining fingers became gradually affected in the same way. He had lost the power of extending them; but the power of flexion still remained in tolerable vigour. Before the right hand had been thus far injured, the left began to follow, and in a few weeks it was nearly as helpless as the right. The disease now began to travel upwards, the wrists gave way, and he lost the power of straightening them. His forearms fell off in volume and continued to waste progressively, until about twelve months ago, when he was compelled to give up working altogether. For three months after this, the fleshy mass of

the forearm and hand continued to shrink until these parts had reached an excessive degree of attenuation. The morbid action now ceased, after having been in progress fifteen months. For the last three quarters of a year, the condition of his arms has not undergone any change.

Rogers experienced no pain while the wasting proceeded; and sensation remained unaffected throughout.

On the 25th of Sept., 1852, he was found in the following condition:—Somewhat, but not very thin; complexion moderately clear. The muscular development for a man of his make is everywhere good, except in the upper extremities. The upper arms are only a little smaller and flabbier than natural, but below the elbow the members are withered and shrunk to an inordinate degree. The forearms and hands seem almost devoid of muscular tissue, and to be literally reduced to skin and bone. The course of the tendons is distinctly visible, however, beneath the integument. The diminution of size is greater at the lower than the upper part of the forearms, owing to the immunity enjoyed by the pronator radii teres on each side. This muscle seems nearly of its natural size, and stands out prominently on the front of the forearm.

On the right side the circumference, an inch below the condyles, is $8\frac{1}{4}$ inches; three inches below the condyles 7 inches; five inches below the same point, $6\frac{3}{4}$; and seven inches below the

condyles, or three and a half above the wrist, only $5\frac{3}{4}$ inches.

The lower third of the forearm is excessively thin in the antero-posterior direction, not being more than half an inch thick in the interosseous space. The pronator radii teres, as already stated, is nearly of natural size, and some remains of the supinator longus can be detected. The muscles of the posterior part of the limb are exceedingly attenuated, but the amount of cellular tissue and subcutaneous fat is quite as great in the fore– as in the upper arm, and the skin itself seems in no respect to have suffered in its nutrition. The muscles of the ball of the thumb, as well as those of the palm, have almost entirely disappeared; so that the outline of the metacarpal bone of the thumb is easily traced in the palm of the hand, and there is a hollow between its extremities, in place of the prominent convexity of the thenar eminence. The flexor tendons are seen to pass through the shrunk palm, which is entirely robbed of the soft cushiony feel natural to it. As he lies in bed with the hand supine, the fingers and thumb are slightly flexed, and the little finger more than the rest.

Some of the movements are quite abolished, and some greatly enfeebled, in the wasted parts. When the forearm is flexed on the arm, the hand falls helplessly backward; the back of the hand forming almost a right angle with the back of the forearm. He is quite unable to exercise the

flexors so as to bring the palm to a plane level with the forearm. He can bring the tip of the little finger into contact with the end of the thumb, but he is quite unable to flex the fingers on the palm, or, indeed, to bring the ends of the other fingers into contact with the thumb. When he attempts to close the fist the fingers are arrested midway. The distal phalanges remain in a state of continued semiflexion, but the proximal phalanx is slightly extended, giving the whole hand the form of a bird's claw. The movements of pronation and supination can be performed without much difficulty; the contraction of the pronator teres and the supinator longus being distinctly perceptible to the hand applied during these efforts.

He is unable to grasp or seize anything with the hand. The bent fingers can be readily straightened (except the first phalanx of the little finger) by artificial help; but they slowly return to their original position when left alone.

An exactly similar description applies to the left forearm and hand, except that on this side the attenuation is still greater and the hollow of the palm still deeper. There is also less power in the thumb, and the patient is unable to adduct it farther than to touch the middle phalanx of the index finger. There is the same absolute powerlessness of the wrist and fingers as on the right side. The power of pronation and supination remains.

Rogers has never been troubled with cramps or twitches in the decaying muscles. There is no febrile movement; the intelligence is perfect, and all the organic functions exhibit the highest efficiency.

This man remained in hospital nearly two months, and he was treated first with strychnia, which was pushed to the verge of its physiological action; then by galvanism, which was applied three times a day to the arms and forearms; and, last of all, blistered surfaces were opened, one on the front and the other at the back of the forearm. A plate of platinum was applied to the one and a plate of zinc to the other, and the two plates were connected by a copper wire, so that a slow and continuous current of electricity was made to traverse the member. But all was in vain—or almost in vain. He went out with a little increased power, but still totally helpless in the hands and forearms.

The urine was examined for several days in succession. The average daily quantity was 44 ounces, and the mean specific gravity, 1021°. It deposited uric acid on standing, but not to such a degree as might indicate any departure from the healthy standard.

On leaving London, he returned to his native town, where he has since been actively employed, as postman, in carrying letters between Anglesea and Caernarvon. By the kindness of Dr. Watkyn Roberts, of that town, I have learned

that, at the present time, (Nov., 1857,) the forearms continue much in the same state; if anything, a slight improvement has taken place.

Case II.

Mr. B., aged forty-seven, a book-keeper and collector, called my attention to his hands, about eighteen months ago. He suffers from atrophy of the muscles and loss of power in both members.

On the 12th of May, 1857, I had an opportunity of examining him. He is a strong muscular man of somewhat florid, congested countenance; hair turning grey. As he walks, a slight defect is perceptible in the right leg, which is a little inclined to drag; and he states, that when irritated or excited by passion, the right foot does actually trail along the ground.

I obtained the following particulars of his antecedent history. The general health has been exceedingly good, and a serious illness is a thing unknown to him. He mentions, that from boyhood he has been remarkable for absence of mind, but in all other respects his intelligence is acute, and he holds the character of a thoroughly sensible and responsible man.

His father was subject to fits (probably epileptic) and died of apoplexy. He himself, has never had any fit or seizure of any kind. No relation was ever afflicted in the same way as himself.

He is a man of cultivated tastes, and in ear-

lier life (when he followed another occupation) he enjoyed the goods of fortune moderately; was fond of music, played often on the piano and organ; and rode much on horseback; in short, led a life not badly calculated to nourish a robust health.

With plumbers, painters, or white lead manufacturers he never had concern, and the water of the cistern from which he drank, was found, on analysis, to contain no lead. Unaccustomed to any excess, he has been temperate in all his habits. At the time of the invasion of the complaint in the hands, he had to work hard, lift heavy weights, and his mother was ill at the time; so that altogether he was in considerable mental trouble. Besides this, no possible cause presents itself to his mind, and he has sought long and anxiously for the root of his malady. It was in buttoning his straps that he first detected the incapacity of the right thumb; near the same time his mother pointed out to him the flattening of the ball of the thumb. He was then thirty years of age, (seventeen years ago,) and he thinks now that the power of the thumb had been failing gradually some time before it arrested his attention. From this time onward the weakness and wasting gradually increased, until he lost all the strength of the thumb, and the power of extending the fingers of the right hand. Before the disease had completed its ravages in the right hand, it seized on the left, and gradually took away the power—and

with the power the volume—of the muscles of the thumb.

The disease continued to advance for about two years, then it stopped; and since, it has never sensibly increased, so that for fifteen years it has remained stationary.

During six months of the two years during which the atrophy continued to advance, he travelled about for his health, visited Brighton, and other watering places, and consulted a great number of medical men, who, he complains, gave most conflicting directions for treatment. Some recommended wine, spirits, ale, &c.; others forbad alcoholic liquors altogether. Some advised cold baths, others condemned them. He could not endure this last remedy; the baths left an abiding chilliness that was most distressing to him.

Present state.—Well nourished and very muscular; he states that he can walk from morning till night except when irritated, and then the right leg hangs like a dead weight, scarcely to be dragged along. The right thigh is somewhat smaller in circumference than the left, but when in favourable condition, he is sensible of no weakness in it.

Hands.—The right hand is always closed unless opened by extraneous aid. Not the least movement of the closed fingers takes place when voluntary efforts are made to stretch them; and when they are opened out for him—which can

readily be done, to the full extent, and without pain—they return forthwith, but gradually, to their original position in the palm. The thumb can be slightly and very feebly flexed and inclined to the palm, but not so as to be of the least use. The power of extension remains, he can draw it back forcibly and quickly. The flexors of the fingers are in full vigour, and when the fingers are pulled back they can be drawn in again forcibly; his grip is remarkably powerful.

All the short muscles of the thumb and the abductor indicis have suffered the most extreme atrophy. The ball of the thumb no longer exists; in its place is a flattened hollow. The two first metacarpal bones appear in plain relief beneath the skin, and have no covering but the integument; while the middle of the palm is plump, and the hypothenar eminence possesses its usual volume. The back of the hand looks emaciated, and the interosseous spaces are slightly hollowed. When the forearm is bared, the anterior aspect is seen brawny with muscle, but the back is a sad contrast; it is quite flat, and the bones and interosseous membrane are very thinly clad. Along each border stand out prominently the uninjured extensors of the wrist, while a deep hollow stretches between. The contrast with the left forearm comes out strikingly on comparison. The common extensor of the fingers, the extensor indicis and extensor minimi digiti have completely vanished. The extensor ossis metacarpi and ex-

tensor primi internodii pollicis are similarly absent, but strange to say, the extensor of the second phalanx of the thumb remains, and at the call of the will—or still better, under the stimulus of galvanism—contracts very actively. The others respond not in the least to strong shocks—shocks that make the last remaining extensor of the thumb start violently. The extensor carpi radialis longior and brevior, and extensor carpi ulnaris retain their bulk and power, and answer promptly to the electric excitant.*

When shocks are passed through the short muscles of the thumb, no movement occurs in the abductor and opponens, but a very faint response is obtained from the short flexor and adductor, and the thumb feebly bends into the palm. Yet this hand, thus mutilated, is by no means a useless one. By the wonderful power of adaptation to new circumstances latent in the animal frame, this gentleman can write, and not only with ease, but with marvellous rapidity and beauty. For this, he places the pen, with the aid of his left hand, between the index and middle fingers, which tightly grasp it; then half bending the fist on the forearm, he guides the pen from the wrist with the most admirable dexterity. The thumb is of no use at all, and to put it out of the way he

* The galvanic exploration was conducted on the method of Duchenne; the extremities of the conductors, covered with wet sponges, were applied directly over the muscle, in close proximity to each other.

thrusts it, with his left hand, into the hollow of the fist. When he wishes to seize anything with this hand, he jerks the member violently backward from the wrist; the fingers fly open by their own weight; he then darts upon the object with open palm, and closing the fingers, grasps it securely.

The left hand is not so extensively injured as the right; the wasting is confined to the ball of the thumb and the abductor indicis. These are as completely destroyed as on the right side, and the thumb is almost reduced to total immobilty; it is capable only of a feeble oscillation from side to side, and is of no use whatsoever. The fingers, however, can be flexed and extended powerfully; indeed, all the movements of the hand are perfect except those of the thumb, and abduction of the index. The atrophied muscles yield no response to galvanic shocks.

Sensation, tactile and ordinary, is as delicate as in a perfect hand. There is no pain whatever, but both hands are very sensitive to cold; he never washes them in cold water, even in summer, for did he do so, a cold numbness would cling to them for several hours.

The organic functions are vigorously performed. The only ailment he complains of is a neuralgia of the right sciatic nerve, which now and then torments him; and to relieve it, he is in the habit of taking warm baths occasionally. This explains the odd behaviour of the right leg.

The disease having accomplished its work of destruction so long ago as fifteen years, it is no wonder that the atrophy has reached so excessive degree; and for the same reason, treatment can scarcely be pursued with any chance of success at this advanced period.

Mr. B. never experienced cramps or twitches in the arms or hands, and he never perceived any quivering movements in the muscles. At the present moment, I need hardly say, there are no fibrillary tremors in any part.

Case III.

Thomas Holland, aged sixty-seven, was registered as an out-patient of the Manchester Royal Infirmary, under my care, July 19th, 1856. He sought advice for a neuralgic affection of the face, and for a failing of strength in the right hand. He is a tailor by trade, and has worked moderately hard; his habits have been very temperate; well clothed and fed, he has usually enjoyed excellent health, and is now a hale old man.

The weakness of the right hand dates three months back. About that time he found that the member failed him in its usual service; he could not grasp the needle with sufficient force to use it at his trade, and he was compelled, in consequence, to leave off work. The neuralgia affected the subcutaneous malar and the inferior dental nerves.

On a cursory examination of the hand, it would

have been difficult to imagine that it was the seat of so serious an injury—an injury that rendered it as useless to the owner, in the practice of his handicraft, as if it had been altogether cut off. When asked to do so, he flexed and extended the fingers and wrist with great vigour, and when I placed my hand in his, he grasped it with inconvenient force. Yet this man assured me he could do nothing with his needle; it turned between his thumb and fingers, or fell away entirely from his hold. Perhaps, I thought, the sensation is at fault; no: he could feel as well with the right as with the left hand. Considerably puzzled, I examined the hand more attentively, and, warned by previous experience, I was not long in tracing out the source of the defect, which took away from this poor man the power of gaining his own bread.

The web of skin between the thumb and index finger, which contains, in the natural state, the fleshy bellies of the abductor indicis and the adductor pollicis, was remarkably thin; so thin, indeed, that it felt like a simple duplication of the integument without any intervening muscular substance. The figure of the metacarpal bone of the index finger had lost its rounded contour, and it stood out square and unnatural, as if unclothed by other covering than the bare skin. It was clear that the abductor indicis had wasted away—barely a trace of it could be detected. The adductor pollicis was likewise exceedingly attenu-

ated. The other muscles of the fingers and thumb were quite sound, and served by contrast, to bring out more prominently the diminution of bulk in the atrophied part. The loss of power corresponded accurately with the anatomical changes. The delicate movements which the thumb and index perform in concert, such as managing the pen, or handling the needle in sewing, were altogether abolished; while flexion and extension of these, as well as of the rest of the fingers, remained unimpaired. No other part of the body was similarly affected. Sensation was everywhere perfect, and the wasted muscles had never been the seat of pain; the intelligence was acute, and the organic functions in the best order.

The neuralgia of the face having speedily yielded to morphia-dressed blisters, he soon ceased to attend, and I was unable to see the full effects of treatment on the atrophied muscles. The only application employed was camphorated liniment, which was directed to be rubbed-in twice a day. Notwithstanding the shortness of the time, the patient reported a decided, though slight improvement. In this case no muscular vibrations were observed; but, as I was not aware, at the time, of the significance of that symptom, I cannot be quite sure of their absence.

Case IV.

Emma Walker, a tall athletic young woman, twenty years of age, and unmarried, presented her-

self among my out-patients on the 16th of August, 1856, stating that she had lost the use of her arms. As she stood before me in her ordinary servant's attire, it would have been difficult to associate with a frame apparently so well-developed and strong, and a complexion so rosy, any ideas save those of robust health; but, when her upper clothing was removed, a simple glance revealed a state of things, sufficient to explain the incapacity of which she complained. The muscles of the shoulders were the seat of extensive and far-advanced atrophy. She gave the following account of herself:—From infancy she had enjoyed uninterrupted health, and she could not call to mind any circumstance, that might have acted deleteriously upon her muscular system. Engaged as a domestic servant, she had always been well fed and lodged; and the work apportioned to her had not been more than what she could easily accomplish. The closest enquiry failed to detect any unusual labour or fatigue occurring about the time her arms began to decline in strength. She had never been in the way of any plumbeous emanations. The catamenia made their first appearance at fifteen, and their recurrence has continued tolerably regular ever since. She has had interruptions five or six times, but they never extended beyond a single period.

It is now eighteen months since she perceived a weakness in the right shoulder. It was first brought under her notice by inability to reach

objects placed on a shelf above her head. The weakness continued to increase, and a few weeks later, affected the left shoulder in the same way. She found that while the hand and forearm retained their usefulness and strength, the power of lifting the arm from the side, and especially of raising it above her head, was passing away from her. Wandering pains, not severe or long continued, or such as prevented her going on with her work, or disturbed her sleep, were felt between the shoulders and in the back during the progress of the complaint.

The increasing failure of strength in her shoulders compelled her to leave her place; and, for several months, she went about from one situation to another, giving no satisfaction, and being soon discharged for incapacity. Quite unable to account for her lamentable condition, she was at length forced to apply at the Royal Infirmary for medical aid.

The foregoing history was obtained at the above-mentioned date. Some medicine was given her, and she was ordered to be galvanised twice a week. To my disappointment, she did not make her appearance at the next visit, nor on subsequent days, and I lost sight of her for above twelve months. Being interested in the case, I sought out her mother, and from her I learned that she had been so severely "punished" by the galvanism, that she had refused to return; and being unable to go to service, she had, for many months,

sought retreat in the workhouse; but that, quite recently, she had come out again, and was now at work as a domestic servant.

I made a request that she might be sent for and brought to me, and on the 5th of September, (1857,) she appeared a second time among my out-patients. In reply to my enquiries, she stated that she had not been able to keep her place, and that she intended returning to the workhouse, as her arms were no better. I persuaded her, however, to enter the house, which she did, under my care, on the 19th of September, 1857.

Her general appearance has not changed since the above notes were taken. She is in perfect health, and strong in every part except the shoulders. When stripped to the waist the whole of the diseased region is exposed, and by contrast with the healthy parts around, is brought into striking prominence. Below a stout bull-neck, along which thick muscular bolsters may be seen to pass from the clavicles to the head, lies a lean and shrunk chest, hollowed beneath the clavicles, narrowed at the shoulders, with a deep broad furrow between the scapulæ behind. On closer examination, the scapulæ are found very considerably displaced; their lower angles, when the arms hang by the sides, being tilted upwards, so that they point toward the vertebral column, and, instead of lying smoothly against the ribs, start out in unseemly projections, giving the back an odd, deformed look. The superior angles project

forward into the anterior region of the neck, and overhang the clavicles; the whole bone seems twisted on its axis in such sort, that, instead of lying vertically on the chest, it takes a direction across it, stretching its lower angle upward and toward the spine, while the upper angle and articulating portion incline forward and downward.

The pectoral muscles seem to have totally disappeared, and the anterior fold of the axilla feels like a mere doubling of skin. The ribs in front are felt in strong relief through the integument, as in the advanced stages of phthisis, yet the skin contains the usual charge of fat, and the mammæ are large and firm. The deltoids on both sides are greatly wasted, but not nearly to the extreme degree of the pectorals. The supra- and infra-spinati are tolerably marked; but of the latissimi dorsi, the teretes majores, and minores, the serrati magni, and the rhomboidei, there is scarcely any trace. The portion of the chest covered by them looks miserably emaciated, and contrasts strangely with the fleshy masses about the pelvis. The trapezius and levator anguli scapulæ, on both sides, are remarkably strong and firm; apparently developed beyond their usual volume. The rest of the upper arms, the fore-arms, and hands, together with the pelvic members, are round, full, and vigorous. The two sides are nearly equally affected; the right being a little worse than the left.

The loss of power corresponds exactly to the

atrophy. The movements of the hand are all perfect; she can grasp powerfully, pronate and supinate, extend and flex the fingers, at will. The forearm can be strongly bent on the arm, and stretched out again without let or hindrance; but the movements of the shoulders are greatly weakened and abridged. Few are altogether abolished; for, it is evident, that the muscles which remain, undertake supplemental duties, and, in some imperfect manner, perform the functions of those which have disappeared. The arm can be raised from the side, on the left, to the horizontal position; and on the right, nearly so; but when she is told to lift the hand above the head, (which, in the natural state, is performed by a rotation on its axis of the shoulder blade,) she makes a vain attempt, and is quite unable to effect the movement. The arm is brought to the horizontal level, chiefly through the instrumentality of the supra- and infra-spinati, which are felt to contract vigorously. The deltoid helps, too, but its efforts are greatly enfeebled; while, on the contrary, the elevator of the angle of the scapula is felt to tug strongly at the top of the shoulder in concert with the trapezius. The arms can be carried to a considerable distance across the chest; but the pectorals cannot be felt to contract during the effort; the movement being, in reality, effected by the clavicular half of the deltoid. The backward motion of the arms is very feeble and limited. When she is asked to pull the shoulder blades

together behind, they move upwards, and toward each other, by the force of the trapezii muscles. Many of the movements, here enumerated as capable of being performed, are so feeble as to be of little service. She cannot even raise a small can-full of water to her mouth. "Under the elbow," however, she "can do anything."

When the condition of the muscles is explored by galvanism, the deltoids are found to respond pretty actively, and the supra- and infra-spinati still more so; while the pectorals, latissimi dorsi, the serrati magni, and rhomboidei refuse altogether to contract. It must be owned, however, that only very mild shocks could be employed, in consequence of the patient's extreme sensitiveness. The muscles of the neck and forearm spring into lively contractions when the electric current is passed through them.

There are no muscular vibrations seen in any part, although efforts were made to provoke them; and the patient maintains that such a phenomenon never did take place in the wasting muscles. Cramps and twitches have been noticed, from time to time, in the muscles of the shoulders. They have never been severe, and are now absent. For the last twelve months, the disease appears to have been almost stationary. The intelligence is unimpaired; her answers to questions are prompt, lucid, and pertinent; the emotions a little excitable, especially, as her mother informed me, at the catamenial periods. All the organic func-

tions are in the best order. There is no deficiency of sensation in any part, nor any unusual or abnormal feelings in the diseased regions.

Before admission, she had not been subjected to any medical treatment. She was now galvanised daily, for about ten minutes each time, over the atrophied muscles. For the reason above stated, only feeble currents could be employed; but some amendment took place in a period of five weeks, during which she continued an inmate of the Infirmary. At her departure, she could lift the left hand at least two inches higher than on admission, and the right could be brought to the horizontal level. She never overcame her dislike to the galvanism, and left the house to avoid the torment of it.

Case V.

Elizabeth Forster, aged thirty-eight, was admitted into the Infirmary June 22nd, 1857, for a loss of power in the right lower limb. She is a healthy-looking, but not a very robust woman. The muscular development and subcutaneous fat are moderate. She is unmarried, and has never borne children. The following includes the main points of her previous history:—Born in the country, she went out to nurse at thirteen, and two years afterwards entered a cotton-mill, where she worked for several years. Subsequently, she became a domestic servant; and has continued to follow that employment until her present com-

plaint forced her to abandon all work. In her last place she was servant of all work, and had to undergo very considerable fatigue; though not more, she says, than she could comfortably bear, if her health had been good. She is subject to occasional dyspeptic symptoms—sick headaches, and cardialgia; otherwise the general health has been good.

It is now two years since she perceived a weakness in the right thigh. She first noticed that it grew tired sooner than the left, on long standing or walking, and that she tripped in going up stairs, especially in mounting quickly. From this small beginning the evil grew; the limb became less and less able to bear its share of the daily labour. She was obliged to sit the greater part of the day; then she went about with a stick, and, last of all, had recourse to crutches. The lameness came on gradually, and increased by degrees. Sometimes there was pain in the hip and knee, and along the course of the anterior crural nerve; but it was never very serious, and was often absent. There never were any cramps or twitches in the affected limb.

On examining the lower limbs, it is easily perceived that the right thigh is less voluminous than the left. The legs appear about the same size. On the 27th of July, the circumference of the thigh was taken at three different points, and compared with the dimensions of the left thigh at

the same levels. The following table shows the results obtained:—

No.	Right thigh.	Left thigh.
1.— 5 in. below the great trochanter.	14½ in.	16½ in.
2.— 7½ " " "	13½ "	15⅝ "
3.—13 " " "	12¼ "	13¾ "

The right leg, at the thickest part, was a quarter of an inch smaller in circumference than the left. The knee joints were found of equal girth. Both thighs were encased in a thick padding of fat; and, on careful exploration with the hand, it was evident that the fatty cushion was quite as thick—if anything a little thicker—on the right as on the left; so that the diminution in bulk must have depended on a falling away of the muscles. The knee and hip were quite supple, and free from any traces of organic disease.

The muscles of the right thigh had lost their natural elastic feel, and seemed dead and resistent beneath the hand. The thick layer of fat prevented a very accurate examination of their condition; but, as far as could be made out, the atrophy had fallen chiefly on the front and back of the limb, and much less severely on the adductors at the inner side. When the patient was told to extend the leg, she succeeded in doing so, but very slowly and feebly; flexion was more vigor-

ous; but, as compared with the normal state, it was exceedingly imperfect. Every movement of which the thigh is capable, could be performed; but it was with so great an effort, and so very slowly, as to remind one of the torpid motions of a limb benumbed with cold.

Exploration with the volta-magnetic apparatus, after the manner of Duchenne, yielded the following results:—The rectus femoris, the vastus externus and internus, contract feebly; the sartorius responds more actively. The muscles at the back of the limb are very difficult to excite, even with the strongest currents of a very active battery; scarcely a trace of contraction is observed in the biceps, and none at all in the semitendinosus and semimembranosus. The adductors, on the contrary, contract powerfully, and seem scarcely less active than those of the opposite limb. The glutei also have preserved their bulk and electric contractility intact. The muscles of the right leg spring into active motion under the stimulus, and seem not a whit behind those of the left leg; they have also retained the elastic feel and voluntary mobility natural to them in the healthy state. When the current is made to pass through the great sciatic nerve, as it lies in the hollow between the right trochanter and the tuber ischii, the muscles of the calf are thrown into lively contractions; but those of the thigh remain perfectly unmoved. All the muscles of the left limb respond readily to the galvanic stimulus.

No fibrillary tremors have ever been observed in the thigh, or any other part; and, if they existed, the thick investment of fat would, I imagine, effectually prevent their being perceived.

Galvanism was the remedy on which I was disposed to place chief reliance, and it was applied daily for five to fifteen minutes; but, from the proper apparatus not being at first ready, it was not regularly employed until after July 27th. She was likewise ordered to rub the thigh, morning and evening, with Lin. camph. comp.

At subsequent visits, she declared that she felt stronger after each application. Progression was certainly improved, and the limb was pulled up more actively; but the amendment was very slow. After the treatment had been followed for above six weeks, the measurements of the right thigh had notably increased, as the following table will prove; and it shows the degree of progress at three separate dates. The points at which the measurements were taken previously, had beeen marked with lunar caustic to ensure accuracy.

No.	July 27th.	Aug. 7th.	Aug. 21st.	Sept. 8th.
1	14½ in.	14⅞ in.	15 in.	15⅛ in.
2	13½ "	13⅝ "	14⅛ "	14 "
3	12¼ "	12½ "	12⅞ "	13 "

The difficulty experienced in lifting the limb from the ground, and in flexing it on the pelvis,

seems to indicate that the psoas and iliacus are deeply implicated; but, being beyond the reach of accurate palpation, it is impossible directly to ascertain whether they, like the other muscles of the thigh, have suffered a diminution of bulk and a loss of their electric contractility.

In one important particular the condition of the thigh differs from the atrophied limbs in the foregoing cases. In them the loss of power accorded with the grade of atrophy, so that it might be predicated from the anatomical changes apparent to eye and hand, to what degree the voluntary power had declined; but here, while the diminution of volume is not greater than has been occasionally seen in hemiplegia, the loss of power has reached a limit beyond which it seldom advances in wasting palsy; and had the diminution of bulk followed the same progression as the declension of strength, the muscles would have been reduced to mere vestiges, whereas they still retain a volume not incompatible with vigorous movement. How is this? We must look for the explanation in the direction sometimes taken by the atrophy. The researches of Cruveilhier, Dr. Meryon, and Mr. Partridge show, that in some instances, the wasting muscular fibres are replaced by fat, so that the bulk of the muscle is preserved, even when its contractile powers are well-nigh annihilated. I have no doubt but that the muscles of this woman's thigh are in a state of advanced fatty transformation; and the dead,

inelastic impression conveyed to the fingers, is in perfect accordance with such a conclusion.*

The movements of the leg are as strong and vigorous as in health. The foot can be flexed and extended; the toes bent in, and stretched out again, as actively as on the left side.

Sensation is quite natural in every part of the limb. There is no undue sensitiveness to cold; and, since her admission into the Infirmary, there has been no pain in the failing member, nor elsewhere. The organic functions, the intelligence, and the special senses are in perfect order.

This woman remained in the house over three months; and at her departure, she could lift the foot and carry it forward as in natural progression. The improvement, however, I am sorry to add, was not such as to encourage any hopes of final recovery.

Case VI.

(Reported by Prof. Cruveilhier, Archiv. Gén. 1853.)†

Lecompte, (Prosper,) aged thirty-two, a showman, came under my care in July, 1850, suffering from atrophic muscular paralysis, already in great part become general. He traces the origin of his complaint two years back, to a night in

* See Chapter VI.

† For the sake of brevity, the report here adopted is the one annexed to Cruveilhier's second Memoir to the Académie, read in December, 1855. Where ampler details are required, I have interpolated portions of the more lengthy account published by him in 1853.—See note p. 19.

September, 1848, passed in the open air on a muddy pavement. He awoke quite benumbed in the right side, upon which he had lain; he went to warm himself in a tavern, and soon recovered both sensation and motion.

Three weeks after, he perceived a weakness in the right hand, a great difficulty in seizing objects, and in drawing his handkerchief from his pocket; and from this time forward it became impossible for him to play the cornet à piston. He is certain, that at this period, all the movements of the upper extremity, except those of the hand, as well as those of the rest of the body, were perfectly free. For a year, the weakness was confined to the muscles of the hand; at the end of which time, in consequence of another cold wet night passed in the open air, Lecompte was sensible for the second time of a great weakness in the lower limbs. From this period, the muscular paralysis progressed with great rapidity. In July, 1850, when he entered my wards, the larger number of the muscles had been attacked in diverse degrees; including the facial muscles, the muscles concerned in the articulation of sounds, the muscles of deglutition, and those of respiration. The patient could yet dress himself, and walk without assistance, though slowly, and with effort. He could feed himself, and articulate intelligibly. I was struck with a remarkable phenomenon, namely, that all the muscles were agitated with a fibrillary quivering or tremor—a species of twitches of lightning

rapidity, or rather resembling those which would be produced by an uninterrupted succession of little electric shocks. I have observed these fibrillary movements nearly constantly in this disease, but never have I seen them in so marked a degree.

In opposition to this progressive decadency of the active organs of locomotion, the tactile sensibility is developed to its highest degree; the organs of special sense are remarkably delicate; the nutritive functions are (except the muscular nutrition) performed in the most regular manner. The intelligence and emotions, are in a state of the highest integrity. I have seldom encountered a patient who has given me so precise an account of his most trivial impressions; and, while he could yet make himself understood, he was accustomed to sum up his condition in such words as these—"I am not ill; but my strength is gone; I have no complaint but weakness, which increases every day, and a feeling of great lassitude in my limbs, which torments me every hour, but especially at the moment of awaking from sleep."

The condition of the limbs was as follows:—

Upper extremities.—The *right upper extremity* in which the disease first appeared, is atrophied in all its parts, but to an unequal degree; the wasting is especially marked in the hand; the thenar and hypothenar eminences are gone; it seemed in these regions as if the skin was in con-

tact with the corresponding bones; the inter-osseous muscles also appear equally deficient in the inter-metacarpal spaces. All the fingers except the index, which is deprived of all movement, can yet be flexed and extended; but very imperfectly. The extension and abduction of the thumb are very distinct; but, the contractions of the extensor and flexor muscles of the hand are so feeble, that the hand falls, by its own weight, either in the sense of extension or flexion, in spite of the contraction of the antagonistic muscles. The movement of supination is nearly impossible; and the patient states that this was the first movement to fail him. The muscles which move the arm on the shoulder, are singularly enfeebled; especially the deltoid, which is very slender, and cannot carry the arm from the side.

The *left upper extremity*, which is a little less atrophied than the right, retains all its movements; but in a debilitated degree.

The *lower limbs* are not sensibly atrophied, and enjoy all their movements; but they are notably weakened. The patient can support himself in the upright posture; and he often tells me that when he is standing, without moving, he feels strong and firm; and that he experiences no sense of lassitude in the legs; and were it not for a feeling of fatigue in the loins, he could stand several hours on his legs; and, on one occasion he stood, with his back to a stove, for four consecutive hours, without complaining of any weariness.

His progression was curious. He walked slowly, the body strongly bent forward, the arms hanging in front of him, with the hands clasped to balance himself; nevertheless, he staggered, and threatened every minute to lose his equilibrium. He often fell on the back; sometimes on the side: and he tells me that when he falls, it is not from weakness, but from losing his balance.*

From July 1850 to January, 1853, the atrophy and paralysis continued to increase. At the time of his admission, Lecompte could walk, as I have said, without assistance, feed and dress himself; but, by the end of 1851, he could not walk at all. The unhappy man was condemned to lie continually on his back; he could not, without help, change his position. His meat and drink were given him, and he was put to bed like a little child. The muscles of deglutition, of articulation, of vocalisation, and respiration, had become more and more invaded. The patient

* To prevent misconception as to the cause of this staggering gait, I will anticipate the revelations of the post-mortem examination. This condition was produced not by any mischief in the spinal cord, but simply by the destruction of the intrinsic muscles of the foot. In the report of the autopsy, "all the muscles of the pelvis and thigh" are declared to be "perfectly sound. In the leg the three muscles of the anterior region are thin and pale in all their length, and have undergone fatty change in their upper part. The posterior muscles are sound, but thin. * * * In the foot, the fleshy body of the short flexor of the toes seems to have completely disappeared. The muscles of the plantar region, the interossei, are wasted, a little decolorised, but sound."

can now, in no wise, swallow his saliva, which runs continually from his mouth. The buccal portion of the act of deglutition can no longer be effected, owing to the almost complete paralysis of the tongue. Twice he was nearly choked, once by a carrot and another time by a potatoe lodged in the pharynx, which the surgeon on guard, who was, happily, in the ward at the time, extracted with his fingers. I was, therefore, compelled to reduce the unfortunate man's diet to thick soups and finely hashed meats, made up into a mess, with sauce and bread. To render swallowing possible, the intelligent attendant charged with his case, thrusts down the food in a spoon deeply into the pharynx; then the patient bites the spoon with his teeth, and that so strongly, that, from motives of economy, an iron spoon had to be used instead of a tin one, which was rendered unserviceable in a few days. The spoon being thus kept fixed at the back of the mouth, the patient makes considerable efforts at swallowing on the spoon and its contents, which latter he gulps down; the more readily the larger the morsel is. The spoon being withdrawn, the patient makes repeated efforts at deglutition, and succeeds sometimes in swallowing the whole of the morsel; but more commonly a portion regurgitates, and falls on a bib of taffeta placed beneath the chin to protect the chest. The patient has a voracious appetite, and swallows four enormous basins of soup each day, besides the hashed meat.

Liquids are still more difficult to swallow, and the greater part always returns, and is received into a basin placed under the chin for the purpose. It is very clear that the first or buccal act of deglutition is not performed at all, on account of the palsy of the tongue and lips, and that the pharyngeal act is only enfeebled.

The articulation of sounds, which had become more and more difficult, is now completely lost. The patient makes his wants known by a slight nod of the head; by the eyes, whose proper movements are fully preserved; by the contraction of the great zygomatic muscle, which still persists; and by a sound of voice, extremely feeble, inarticulate, guttural, and nasal.

The respiration, very incomplete, seems to be accomplished by the diaphragm alone; and its contraction is notably diminished, being only manifested by the elevation and separation of the five lower ribs. The patient, whose intelligence continues quite unimpaired, tries in vain to take, at my request, a deep inspiration; never have I been able to obtain the least elevation, or depression of the thoracic cage *en masse*. All the expiratory play occurs at the base of the chest, at the expense of the diaphragm and the muscles of the abdominal walls.

It was evident that the unhappy man was menaced every moment with asphyxia, either during deglutition or on the occasion of the least bronchitis, seeing that it would be impossible for

him to free the air passages from the mucus which would be thrown out; and so it was. On the 15th of January, 1853, he was seized with the prevailing influenza—the air passages became clogged with mucus—the respiratory sounds were drowned in loud rhonchi—no effort, spontaneous or provoked, at expectoration, was possible; and one morning they found him dead in his bed.

Autopsy.—Nearly all the muscles and nervous centres were dissected with the utmost care. The principal nerves were dissected, in their course to the muscles, and into the substance of the muscles themselves; then the nerves were subjected to the action of dilute nitric acid.

Muscular System.—Several muscles had escaped the atrophy; among these were the muscles of the pelvis and of the thigh. The elevators of the lower jaw, the muscles of the pharynx, those of the supra- and infra-hyoidean regions, the platysma on both sides, and the zygomatics had suffered simple atrophy or emaciation. Other muscles were emaciated and pale; others had undergone atrophy with fatty transformation. Several seemed, at first sight, to have disappeared, so thin and slender were they; but a remnant of them could always be found. There remained not a single muscle of the upper extremity unaffected; but all were not equally destroyed. In every region some muscles were found especially attacked; and, lastly, not only were the several fasciculi of each muscle unequally affected, but even the dif-

ferent fibres of each fasciculus had suffered in an unequal degree.

In classifying the muscles according to their degree of atrophy, the first rank must be accorded to the intrinsic muscles of the hand; second to them range the muscles of the shoulder; next in order the muscles of the forearm; and after them those of the upper arm. The tongue, of which the palsy was so complete in the later years of life, engaged my special attention. It was changed into a fatty mass, in the midst of which appeared a great number of vertical muscular bundles. The antero-posterior and transverse fibres of the organ had degenerated entirely into fat.

Let us examine the condition of the *Nervous System*. The encephalic mass is in the most perfect condition; its weight is one kilogramme and 300 grains,* which is very near the ordinary weight. The spinal cord is quite sound, and of the usual bulk, colour, and consistence. The antero-lateral columns are in their normal condition. I examined them with peculiar care, inasmuch as they are considered to preside over the movements of the body. The same could be said of the posterior columns; but the *anterior roots* of the spinal nerves are remarkably small compared with the posterior; and this inferiority of size is particularly great in the cervical region. The proportion between the two roots had become greatly changed. According to my observations, in the

* In Avoirdupois weight 36 ounces and 163 grains.

normal state, the posterior roots compare with the anterior, in the cervical region, as 3 to 1; in the dorsal region as $1\frac{1}{2}$ to 1; and in the lumbar region as 2 to 1. But here the proportion was as 10 to 1 in the cervical, and 5 to 1 in the dorsal and lumbar regions. Further, by plunging the cord into dilute nitric acid, I was enabled to observe that a very large number of the anterior cervical rootlets had been completely reduced to their neurilemma, and appeared as grey filaments, which, searched with a strong lens, presented no trace of nervous tissue; while, on the other hand, the anterior roots in the dorsal and lumbar regions, had only suffered atrophy by emaciation. I was unable to trace the grey nervous filaments, or those simply atrophied, beyond the point where the anterior root joins the posterior; but I have been able to establish the existence of atrophy of the nerves as they are about to penetrate the muscles; and the contrast is most striking on comparing the slenderness of the muscular branches with the full development of those destined for the skin. These observations received their highest corroboration from what was found on the tongue. It is well known that the hypoglossal is the exclusive motor nerve of the tongue. Before immersion, in dilute nitric acid, the hypoglossal appeared reduced to a third of its ordinary size; but when deprived of its neurilemma, it measured no more than one-sixth of the diameter of the healthy nerve, and its colour was observed to

be tarnished and grey. But what rendered the wasting of the hypoglossal still more striking, was the comparison with the gustatory nerve; the white and well-nourished fibres of which contrasted strongly with the lean filaments, and grey, dim colour of the trunk and branches of the hypoglossal.*

Case VII.

By Dr. Th. Valentiner.†

A. B., a gentleman, forty-five years of age, with a well-knit frame and athletic muscular development, remarkable for bodily strength and unusual mental capabilities, had, with a few insignificant exceptions, always enjoyed sound health.

A. B. was a great eater; and could tolerate a very large quantity of alcoholic drinks. Formerly expert in gymnastic exercises, he amused himself in later years, now and again, in the same way; and on one of these occasions, about ten years ago, he fell, a height of eight or ten feet, on his back; but this accident left behind no noticeable consequences.

According to the patient's statement, his health began to waver in the years 1846 and 1847 (soon after the above-mentioned accident); for, the pre-

* For the results of the microscopic examination of the muscles see Chap. VI.

† "Prager Vierteljahres-Schrift, bd. ii., 1855;" quoted in "Schmidt's Jahrb.," p. 182, bd. lxxviii., 1855; and in "Canstatt's Jahresb.," p. 80, bd. iii., 1856.

viously blooming hue of his countenance faded, and the whole skin assumed a yellowish tint; while the hair, previously brown, began to turn grey. Nevertheless, in the beginning of 1852, his strength had suffered little; for he could lift with as much ease as ever two fifty pound weights in each hand.

In the spring of 1852 he undertook a long journey through Germany into Italy; after his return, his great strength was betokened by his appearance. In the winter 1852–3 he detected a weakness in the hands, and in April, 1853, he first called the attention of his medical attendant to them. Examination disclosed the following:— Both hands were greatly enfeebled, so that the patient could make but a very faint pressure with them. The right hand was weaker than the left. All the fingers of the right hand, together with the middle, ring, and little fingers of the left were incapable of extension; and maintained, in spite of the strongest efforts to stretch them, a slightly bent position. The left thumb and index, on the contrary, were much stronger; they could be fully extended, and were still able to grasp pretty strongly. At this time there was no perceptible wasting of the hands, but the forearms had lost considerably in bulk; and the once powerful muscles had, to a considerable extent, disappeared, and were become withered and soft. All the movements of the arm could be performed, though much more weakly than formerly. The patient

could still feed himself, although even this fatigued him somewhat. Progression was altogether unhindered; yet he grew weary sooner than formerly. With the exception of the upper extremities, there was no emaciation, although the frame had become something lax and flabby, and the above-mentioned yellow tint of the skin was distinctly cognizable. There was neither tenderness over, nor curvature of, the spine; nor any symptom referrible to the head. The expression of the features was unchanged, and the intellectual power altogether undisturbed; the organs of sense were normal, and there were no hallucinations; sensation was perfect in every part, and digestion in the best order.

The patient was sent to Franzensbad, which he reached in June, 1853. He drank at first the saline, and then the Franzen water,* and used mud baths; but without any results. Not long after his arrival in Franzensbad, he had to be fed; his hands being no longer able to carry the food to his mouth. Progression continued still tolerably free. In the beginning of September, the patient left Franzensbad with the intention of spending the winter in a southern city, and with that view he came to Nancy. Here the disease made rapid progress. A medical man learning that, several years before, the patient had con-

* Both springs are saline; but the latter contains, in addition, a small proportion of iron.

tracted a chancre—which, however, was speedily healed by mercury—conceived that syphilis lay at the bottom of the complaint; and prescribed Iodide of Potassium, which was persevered in for four or five weeks, without the least benefit; on the contrary, the patient became rapidly worse, and very soon he could not walk unassisted.

While he sojourned in Nancy, he noticed in the debilitated muscles, now here, now there, rapid twitches. These were not present every hour, nor every day, but only at long intervals; and after the occurrence of these twitches (fibrillary tremors) the palsy invariably extended. By the end of November, the state of the patient had become so alarming that he determined to return home. Examined on his arrival, a great change was found in his condition. Considerable emaciation had taken place, and he looked much older than he really was. The arms hung lifeless along the trunk; they could still be moved at the shoulder and elbow joints, but in a limited degree, and with very feeble force. The hands were in a state of almost complete immobility; the fingers were fixed in the flexed position, and when they were stretched by another, (which could be easily done) they forthwith returned to their old posture. The thumbs alone still enjoyed a slight mobility. The muscles of the hands had entirely vanished. In the lower extremities, the wasting had not advanced nearly so far as in the upper. The

knees could no longer be kept fully extended in the standing posture; they were always bent at an obtuse angle. In walking, the feet were somewhat turned in; often striking against the ground, and not unfrequently they caught behind each other. While standing, the trunk fell heavily forward; still the patient could, with an effort, straighten the back to a certain extent, and draw the shoulders backward. To get up from his seat without help, was already an impossibility. The head could be moved in every direction with tolerable ease. The muscles about the mouth showed, in speaking, unmistakeable signs of palsy; so also the muscles of the tongue, which caused the speech to falter somewhat. The muscles of the forehead, of the eyelids, of the eyeball, and of mastication were quite free; whereas, those of deglutition, were considerably affected. Sensibility was perfect in every part; occasionally, pricking sensations were felt in different spots of the face, and in the external auditory meatus. The activity of the organs of sense and of the intellectual faculties was undisturbed, and remained so till death. He was remarkably sensitive to low temperatures, and often he would be seized with a severe shivering fit if the room was not particularly warm. During the three months that the patient yet lived, the paralysis continued to increase. The speech became less distinct, from day to day, and at length the patient lost

the power of speaking altogether; and swallowing was greatly impeded. Digestion continued good; but it became often necessary to administer glysters, or purgative medicines, to overcome the sluggishness of the bowels (evidently a consequence of loss of power in the extrinsic muscles of defecation). Micturition was normal. In the last year a pollution occurred. His sleep, long since very short, became more and more uneasy; for, in the horizontal posture, he was speedily seized with oppressive sensations, and was unable to shift himself except in a very slight degree. The pulse was changeable; sometimes very slow (50–60); sometimes faster, (80–90). On the 18th of March, a slight bronchitis set in, with infiltration of the lower lobes of the lungs, which, on the 19th, brought about a fatal result.

Autopsy.—There was extreme emaciation, and the lower limbs were slightly œdematous. The upper parts of the body were much more wasted than the lower. The face had not fallen-in to any marked degree; the neck was lean, while the upper extremities presented the highest degree of emaciation. The deltoid, biceps, triceps, and the muscles of the forearm, on both sides, had almost entirely vanished. The muscles covering the thorax were also in the last stage of atrophy; on the contrary, in the lower limbs, tolerably thick bolsters of muscular flesh could be felt.

When the muscles of the back were uncovered,

a remarkable change in their colour struck the eye. All the regions, however, were not changed in an equal degree; in the neck the muscles had a very pale red hue; in the lumbar region they were a darker red; while in the upper dorsal region the muscles were pale red, with peculiar yellow streaks. Microscopic examination showed the presence of fatty degeneration in different stages. In the neighbourhood of healthy fibres were found others transformed into a beaded shape; in others the cross striæ had completely vanished, and had given place to a finely granular appearance, with fat molecules intermixed; in yet other places, a number of fibres had been destroyed, and their places occupied by fat-drops and cells.

When the spinal dura mater was opened, a considerable number (about 100) of small white bodies came into view; they were irregularly scattered over the posterior surface of the cord; the cervical portion only was quite free from them. They varied in size from a pin's head to a lentil, and were imbedded in the tissue of the arachnoid. Smooth on the outer aspect, and rough on the inner, they craunched beneath the knife in many places; and appeared, under the microscope, to consist of an intricate net of fibres; they also contained some masses of pigment; and, in four places, there was presented an appearance strikingly similar to imperfect Haversian canals of bone.

The anterior roots of the spinal nerves were much thinner, and more withered than the posterior. The latter had preserved their striated and satiny look, while the former looked remarkably faded and flabby; they also seemed as if infiltrated with a reddish serum, and a lens showed a very rich plexus of large vascular ramifications. The contrast between the anterior and posterior roots prevailed everywhere, but was by far most prominent in the lower and upper dorsal regions. Here the anterior roots were to the posterior as 1 to 6. The microscope showed that the posterior roots were normal in respect of their primitive fibres, within the well-filled sheaths of which, the contents had already coagulated. In the anterior roots there were found also many sound fibres; but beside these, were others, of which the only trace were empty sheaths; and some had suffered evident fatty transformation.

The cord was at first thought healthy, even on section, but closer enquiry showed that it was considerably diseased. The part included between the origins of the fifth cervical and the fourth dorsal nerves felt less firm; and, on section, the central portion was found softened, so that the line of demarcation between the white and grey matter had, in certain places, disappeared. A large number of granular cells were found in the softened parts, both white and grey.

The skull presented nothing unusual. The dura

mater was attached to the bone; the transparency of the arachnoid was a little troubled, and it was slightly œdematous. The Pacchionian bodies were strongly developed. The brain substance was healthy—somewhat rich in blood; and large sandy concretions were found in the pineal gland.

There was commencing fatty degeneration of the heart and liver: in the remaining organs, there was nothing found worthy of note.

The muscles of the chest and abdomen laid bare in the examination, and the diaphragm, appeared atrophied, pale, and streaked with yellow lines. A portion of the biceps (which had a very pale red colour), examined microscopically, showed commencing fatty degeneration. The nerves of the brachial plexus showed nothing abnormal, either to the naked eye or the microscope.

In the following Table is a summary of the chief points in the history of all the undoubted cases I have been able to collect. Altogether they number 105. This includes the cases already detailed at length, which are here introduced for the sake of uniformity. Some of them are extracted from records of a date anterior to the publication of Aran's paper; and others, although occurring after that epoch, have been reported in

evident ignorance of that essay, and naturally, so very imperfectly, that they are but of small service in the study of the pathology of wasting palsy. On some points—the age, sex, occupation of the patients—the parts most liable to be assailed—the starting place—the duration and the issue of the disease—this Table affords most valuable information; and it may be confidently looked-to as furnishing data, for laying a firm foundation for the entire pathology of this curious malady. The cases are arranged in two divisions—according as they are *partial* or *general*.

Aran first proposed this arrangement, and I heartily endorse it. Practically, it is of great importance to keep the two groups distinct in the mind; and throughout this Essay they are separately regarded. The gravity of wasting palsy as a disease, so far as the part is concerned, is commensurate with its extent; but, so far as life is concerned, it depends on the location. With the exception of 6 cases out of the 94 in which the starting point of the disease was ascertained, all had commenced in the extremities;* and while the atrophy was confined to those parts, it was impossible to say whether the malady would remain thus circumscribed, or push onward to invade the head and trunk; but it is, nevertheless, true, as may be gathered in the course of the succeeding chapters, that the impli-

* See Chap. IV.

cation of the muscles of the face, mouth, pharynx, and thorax, is, in a large number of instances, associated with peculiar conditions of origin, and peculiarities of course, which would, almost of themselves, have warranted this classification, but which the totally different aspect of the prognosis renders altogether imperative. The first 10 cases of the second Group exhibit some examples of an intermediate complexion, in which the chief morbid manifestations are in the extremities, but in which also the muscles of the head or trunk are, not obscurely, threatened.*

* It is necessary to understand that the *ages* recorded in the succeeding table are not the ages when the cases came under observation, but the ages at the time of the invasion of the disease. It was desirable to make this distinction, inasmuch as the disease, in some instances, continued to progress for five or six years; while in others, after having reached a certain limit, it ceased to extend, and remained stationary to the end of life. This will explain some apparent mis-statements of age, when these abstracts are compared with the fully recorded histories.

GROUP I.—*In which the Disease was confined to the Extremities and the Muscles thereto pertaining.*

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
1	Dr. Cooke, On Palsy, page 31, Lond. 1822.	M. 27	An officer in the army.	———	Right hand.	Right forearm and hand, completely atrophied: upper arm unaffected. Left upper arm wasted; forearm normal. General health excellent.	Loss of power.	None.	Extended gradually for 5 years in spite of various remedies until arrested by a course of mercury. Unchanged 22 years afterwards.	Arrest.
2	C. H. Parry, Coll'd Works, p. 523. Lond. 1825.	M. 50	Tall, stout, accustomed to much exercise; temperate.	Diarrhœa for the preceding 11 months.	Left leg.	Extended to the right leg, then to both hands which became greatly wasted. General health good.	Loss of power.	No pain; great sensibility to cold.	Arrested by Bath waters.	Arrest.
3	Sir Chas. Bell, Nervous System, Append., p. clxi.	M. 21	Served in an ironmonger's shop.	———	Ball of the right thumb.	Ball of thumb wasted away. Extensors also wasted but not destroyed. It never extended to any other parts.	Loss of power.	No pain.	———	Arrest.
4	Ditto.	M. 10	Young gentleman.	———	Thighs.	All the muscles of the lower extremities wasted, except the vastus ext.; rectus quite gone.	Loss of power. Curious twisting and jerking of the body in rising from his seat.	None.	Has progressed for 8 years.	Progressing.
5	Ditto.	M. 15	Young gentleman.	After Scarlet Fever.	Right arm.	Gradual loss of power and wasting of both arms, the scapulo-humeral muscles and those bracing down the scapula to the thorax. Forearms, except extensors of wrists powerful. Extended	Loss of power.	None. No pain.	Had progressed for 2 years.	Progressing.

						ral health good.				
6	Sir Chas. Bell, Nervous System, 3rd. ed., with Additions, p. 432.	M. 37	Coalheaver.	———	Left thumb.	Great atrophy of flexors of left hand and fingers, also of ball of thumb and of the three extensors of thumb. Right side, supra- and infra-spinati, biceps and deltoids wasted away. Triceps strong; forearm and hand normal. General health good.	Loss of power.	Pain at the outset in left arm; also constant and often violent pain in the wasting muscles.	The right shoulder not affected until a year and a half after the left hand. History extends over three years.	?
7	Abercrombie, On the Brain, p. 419. Lond. 1828.	M. 14	———	———	Shoulders.	The deltoid and biceps reduced to mere membranes. Muscles of scapula affected, but in a less degree. Forearms full and vigorous. Came on gradually. General health good.	Loss of power.	None.	———	?
8	Dr. Darwall, Med. Gazette, p. 201, vol. vii.	F. —	Washer-woman.	Carrying heavy weights.	Deltoid.	Confined to the upper arms. Forearms and hands strong. General health perfect.	Loss of power.	No pain.	———	———
9	Ditto.	M. —	Labourer in a Corn Factor's Warehouse.	Carrying heavy weights.	Shoulders.	At first only upper arms affected; but gradually it involved the entire of both upper extremities.	Loss of power.	No pain.	Continued progressing in spite of seton in back of neck, for eight yrs.	Arrest.
10	Ditto.	F. —	A servant.	Carrying heavy weights.	Left shoulder.	The wasting of the muscles had gradually extended down the left arm. Forearm and hand unaffected.	Loss of power.	Pain over the deltoid and in the shoulder joint.	Under observation four months. Acupuncturation without effect. Arrested by electricity.	Arrest.
11	Med. Gazette, p. 607, vol. vii. (Doubtful case.)	M. 40	Sailor.	Cold taken while salivated for Typhus Fever.	Upper extremities	Commenced with loss of power, and also partly of sensation, in arms and legs. The legs soon recovered motion and sensation; but the arms	Loss of power.	Feeling of numbness and coldness.	Improved very slowly by the persevering use of acupuncturation.	Amendment.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
						never recovered properly, and when seen eight months afterwards the muscles were found much wasted, especially the adductors of the thumb. Fingers flexed on the palms. Hands livid. General health good.				
12	Dr. Bright, Reports of Medical Cases, p. 391, vol. ii.	F. 31	Soldier's wife. Subject to constipation.	———	———	The hand wasted to the condition of a person suffering from lead palsy. General health good.	Loss of power.	———	History extends over seven months.	?
13	Herbt. Mayo, Outlines of Pathology, p. 117. Lond. 1836.	M. 23	Carver and gilder.	Keeping the hand for an hour in cold water when heated by exercise.	Right arm.	Disease came on with rheumatic pains, followed by wasting, and at length great attenuation of muscles of arm and forearm. Lat. dorsi and Serrat. magn. quite gone.	Loss of power. Trembling of arm and hand.	Rheumatic pains at onset.	Treated without success with stimulating lotions and warm flannel. History extends over four years.	?
14	Do.	M. 45	Farm labourer.	Exposure to wet and cold.	Left shoulder.	Started with violent pain in left shoulder, which continued 6 weeks, and then ceased. Then shoulder began to waste rapidly, during and after the pain, so that in 4 months the deltoid, infra- and supra-spinati, and teretes muscles were completely gone. Forearm and hand not affected.	Loss of power.	Violent pains.	Course very rapid. The shoulder muscles totally destroyed in 4 months. Complicated with neuralgia of the circumflex nerve.	?
15	Dubois, Gaz. Méd. de Paris, 1847.	M. 15	Labourer.	Carrying heavy weights.	Hands.	The left arm was first attacked, and soon after the right. In 2 years the atrophy	Loss of power.	Tactile sensibility perfect. A year after	History extends over 3 yrs. No tendency to extension beyond shoulders	Arrest.

					joint, as well as those which bind the scapula to the trunk. Forearms unaffected. General health good.		joints of upper extremities and along the nerves.	counterirritants to spine, strychnia, & electro-puncture.	
16	Aran. Arch. Gén. p. 1., 1850.	M. 50	A strong healthy workman.	Excessive work.	Hands.	Atrophy of both hands and forearms. Other parts not affected. Thenar and hypothenar eminences totally gone; also the interossei, front and back. Front and back of forearms quite wasted. General health good.	Loss of power. Cramps in the forearms, which disappear as the atrophy advances.	For several months deep-seated pains in lower limbs; sometimes lancinating.	History extends over 1½ years. The disease progressed at first more rapidly after he came into the hospital than before; but, after a while its progress was stayed.	? Arrest.
17	Do.	M. 37	A healthy mason.	Hard labour.	Right shoulder.	Two months after the right, the left arm became enfeebled. Disease extended to all the muscles of both shoulders, and, in a less degree, to the arms, forearms, and hands. After repose, the power of the affected muscles is much greater than after fatigue. General health excellent.	Loss of power. Cramps in the wasting muscles. Fibrillary tremors.	None. No pain.	In 2 years quite unable to work; continued to get worse for another year. Treated with galvanism, iron, sulphur baths, but not with the effect of arresting the disease.	Progressing.
18	Do.	M. 33	Farm labourer. Tall, good constitution.	———	Right hand.	In 18 months, the two upper arms (except the triceps) completely wasted. Forearm and hand, especially the right, affected, but unequally. The least touched were the muscles attached to the epicondyle and epitrochlea. The triceps maintained its volume throughout in strong contrast with those around. General health excellent.	Loss of power; cramps—no permanent contractions. A few fibrillary tremors in the muscles of the forearm.	None.	The two arms rendered nearly useless in 9 months. A year subsequently, the disease had advanced. Galvanism caused a temporary amendment. History extends over 2 years.	Amendment.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
19	Aran. Arch. Gén. p. 1, 1850.	F. 28	Milliner; good constitution.	Over work with the needle.	Right hand.	Commencing with the interosseous muscles, it extended to the thenar and hypothenar eminences, and gradually to the forearm, and slightly even to the upper arm. The grade of atrophy diminished in the order of the parts mentioned. Left hand attacked 2 years after the right, but the disease had not mounted higher on this side. No other part affected. Health excellent.	Loss of power; cramps and twitches in the affected muscles; talon-shaped hand. Fibrillary tremors.	No pain.	At the end of 12 months obliged to give up the needle. Performed domestic service for another year; but, during the third year, incapable of any work. A very bad attack of typhoid accelerated its progress; treated with galvanism with slight success.	Slight Amendment.
20	Do.	F. 40	Strong and robust. Had when a child a previous attack of muscular atrophy in right hand, from which she recovered completely at the age of 12.	———	Right hand.	Progress very gradual. Hollowing of the interosseous spaces, then of the ball of the thumb, which was completely destroyed. Hypothenar eminence less wasted. No farther extension. General health perfect.	Loss of power. No cramps or twitches. No fibrillary tremors. Claw-shaped hand.	No pain in part affected. Neuralgia in left sciatic. Great sensibility to cold.	Very slow in its course. History extends over about 5 years. Galvanism tried, but no results obtained as yet.	?
21	Do.	M. 36	Shoemaker. Middling constitution. Usual health good.	Over work.	Left upper arm.	Commenced somewhat suddenly in the biceps and brach. ant. In the course of a year the disease had extended to the deltoid and the muscles connected with the epitrochlea. Some weakness also of the muscles of the hand. On	Loss of power. No cramps or subsultus. Fibrillary tremors.	No pain. Sensitiveness to cold.	History extends over 1 year. No treatment mentioned.	Progressing.

						touched is the deltoid. Triceps of left side strong and large.				
22	Do.	M. 38	Lapidary. Good constitution. This man is the subject of saturnine intoxication.	Over work.	Right hand.	In the course of a year the ball of the thumb and that of the little finger had become quite wasted, also two of the dorsal interossei. Slight affection of the tongue and muscles of the face, indicated by slight stammering and twitches of some facial muscles. No atrophy except in the right hand.	Loss of power. Tremblings of the tongue and twitches of the face. Cramps in the lower limbs. No fibrillary tremors.	Pain in the lower limbs; none in the affected part.	History extends over about 18 months. No treatment mentioned. The diagnosis of this case from lead palsy, depended on the persistence of the electric contractility in the decaying muscles when the atrophy had not reached its term; and on the perfect freedom of the extensors of the wrist.	?
23	Do. Gaz. des. Hop. No. 74, 1855.	M. 18	Agriculturist. Strong constitution. Two aunts died of general muscular atrophy.	Hereditary taint.	Right leg.	Right leg affected with weakness, and then slight wasting; 8 years afterwards, left leg became similarly attacked; then, in 6 months, the right arm. The disease never proceeded far in the lower limbs; but in the right upper, the deltoid, biceps, and triceps were completely destroyed. The trapezius and great pectorals of both sides were wasted. Forearms and hands free.	Unsteady gait. No fibrillary tremors. Loss of power in the shoulder.	None.	For 8 years confined to slight weakness of right leg; then more rapid progress for 2 years. Great amendment by electricity and sulphur baths.	Amendment.
*24	Rayer. Union Méd. 1850.	M. —	Mason.	———	Left thigh.	Front part greatly wasted.	Loss of power. No fibrillary tremors.	Pains in the knee at onset.	———	———

* The only authority I have been able to consult about the following cases has been Dr. Wachsmuth's Table: Nos. 24, 25, 26, 29, 30, 31, 62, 87, 88, 92, 96. For the rest I have had access to the original narratives.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
25	Froriep. Rheum. Schwiele, (Henle and Pfeuf.)	F. 20	Always healthy.	———	Left hand.	Muscles of the forearms except the pronators and supinators wasted, likewise the entire hand; upper arm sound. General health perfect. Lividity of affected parts.	Loss of power. Claw-shaped hand.	Dull pains in right shoulder. Sense of Coldness.	Progressed slowly for 3 years, then became stationary. Treated with strychnia and blisters, without effect. Gymnastics and electricity, followed by better results.	Amendment.
26	Romberg. Klin. Ergeb. I. and II. heft. (Henle and Pfeuf.)	M. 37	Tailor.	Hard work. Cold.	Right shoulder.	It commenced with hemiplegia, but the palsy left the lower extremity completely, and the loss of power in the arm corresponded only to the wasting of the muscles. The deltoid, supra- and infraspinati only affected.	Loss of power. Fibrillary tremors.	None.	Russian steam baths, cold douche, friction, two months galvanizing, and nux vomica, without effect.	Progressing.
27	Do. Nervous Dis. Sydenham Soc. transl. vol. ii, p. 372.	M. 50	Coachman.	———	?	Right side—complete atrophy of forearm and hand. Left side—entire upper extremity and shoulder.	Loss of power.	None.	Complicated with pulmonary phthisis; which caused death ten years after supervention of the paralysis.	Death.
28	Do. p. 373.	M. 52	A gouty Merchant.	———	Left thumb.	The muscles of left thumb became atrophied; and, a few months later, those of the right also.	Loss of power.	A year before the atrophy set in, severe pains in right shoulder running down the arm.	Complicated with violent neuralgia of the arm opposite to that where the atrophy commenced.	?
29	Richter.	M.	Engineer.	Plunging	Right	Slight atrophy of the fore-	Loss of power.	None.	Stationary for	Arrest.

30	Bouvier. Gaz. des Hop. 1851.	M. 39	Joiner.	snow. Hard labour.	Left hand.	Interosseous muscles of hand atrophied.	2nd and 3rd phalanges constantly semi-flexed. 4th finger flexed on the palm.	Benumbed feeling. Sense of weight and coldness in the fingers. Afterwards pain in them.	Treated with sulphur baths, frictions, & electricity, with benefit; and, after 6 months, almost complete recovery.	Recovery (almost complete.)
31	Valleix. Journ. de Méd. et de Chirurg. 1852.	M. —	———	———	Left arm.	The atrophy extended to the muscles of the breast, and to the left lower extremity.	General fibrillary tremors.	———	———	———
32	Gros. Gaz. Méd. Oct., 1854.	M. 28	Chasseur.	Contusion 6 weeks previously.	Left shoulder.	Entire of the left upper extremity atrophied.	Fibrillary tremors very distinct.	No pain.	Arrested by galvanism.	Arrest.
33	Betz. Prag. Vierteljahrssch. 1854.	M. 47	Workman.	Hard work.	Right hand.	Wasting of the muscles at the back of the hand, and the extensors of the forearm.	Loss of power.	Sensitiveness to cold.	Had been stationary for 3 years.	Arrest.
34	Do.	M. 36	Smith.	Wound of elbow long while ago. Uln. nerve notinjured.	Right hand.	Atrophy of the muscles between the thumb and index finger, and of the interossei.	Loss of power.	No pain.	Stationary for 14 years.	Arrest.
35	Do.	M. 48	Smith.	Hard work.	Right thumb.	Atrophy confined to the thumb and fore finger.	Loss of power.	No pain except in the elbow.	Leeches and an ammoniacal liniment have almost removed the disease. No relapse after 3 months.	Recovery.
36	Wachsmuth. Henle & Pfeuf. Zeitsch. 1855.	F. 56	Washer-woman.	Hard work.	Right thumb.	As yet not extended beyond the thumb.	Loss of power. Fibrillary tremors.	———	Progressed gradually for a year.	?
37	Do.	M. 59	Shoemaker. Healthy, sober.	Hard work.	Ball of left thumb.	It gradually extended up the left arm to the deltoid. In about 1 year the pectorals,	Loss of power. Fibrillary tremors in the	No pain; but the fibrillary tremors could	History of the case extends over 15 months, and	Progressing.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point,	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
						shoulder and neck muscles began to waste. Finally, it extended to rhomboid of the same side, and began to threaten the muscles of the right shoulder. Lower extremities free. General health good. Back of left hand livid; became œdematous when allowed to hang.	wasting muscles and those about to waste. Artificial warming greatly improved the power of the wasted limb for a time.	be felt on patient's attention being drawn to them. Sensibility perfect, but objects feel flattened.	shows a gradual progression in spite of frictions, steel tonics, and elcctricity.	
38	Oppenheimer. Ueber prog. fett.muskel-at. Heidelberg. 1855.	M. 38	Professor.	———	Left arm.	The left arm, forearm, and hand, were successively atrophied; then the right arm and forearm were seized. It spread afterwards to the muscles of both shoulders. General health perfect.	Loss of power. Claw-shaped hand.	No pain.	Under observation 2 years. Cold water treatment and baths, without effect. Galvanism caused at first considerable amendment; but afterwards the disease progressed in spite of it.	Progressing.
39	Schneevoogt. Niederlandsch. Lancet. 1854.	M. —	Sailor.	Pumping several successive days in a leaky ship.	Right arm.	The muscles of the hand and the biceps and deltoid were chiefly affected. The atrophy does not seem to have reached a high degree.	Loss of power.	?	Cured in 2 months by frictions, acupuncture and electricity.	Recovery.
40	Do.	F. 24	———	Suckling and carrying the child on the left arm.	Left shoulder.	Involved the deltoid, the supra- and infra-spinatus; also slightly the teres major and minor.	Loss of power.	Pain in the wasting muscles.	After continuing about 9 months, it gave way in about 4 weeks, to galvanism.	Recovery.

	Gunzburg's Zeitsch.— (Canstatt, 1855.)	17	to venereal excesses.	and venereal excess.		slowly for 7 years; in which time the serrat. ant., deltoid and biceps, on both sides, were almost annihilated. Muscles of forearm and hand less wasted. The thighs strikingly atrophied.	The ability to walk varies greatly; sometimes he can walk several hours, at other times scarcely at all. No fibrillary tremors.	No pain.	After advancing for about 7 years, a doubtful arrest was obtained by repeated hydropathic courses, gymnastics, and fresh air.	Doubtful. Arrest.
42	Dr. Eulenburg Canstatt's Jahresb. 1855.	M. —	Of mature age.	———	Right hand.	In the course of several years it extended to the forearm, arm, and shoulder.	———	———	———	———
43	Do.	M. —	Ditto.	———	Right hand.	Invaded the whole of the right upper limb, and then attacked the left hand.	———	———	———	———
44	Do.	M. 24	———	Hereditary taint.	Legs.	Both lower extremities were involved in the highest degree of atrophy. It affected no other part.	———	———	———	———
45	Do.	M. 24	———	Hereditary taint. Brother to preceding.	Legs.	Same as preceding.	———	———	———	———
46	Dr. Meyer. Wiener Wochensch. No. 41,	M. 22	Of good family which is inclined to mental and nervous complaints.	———	Right hand.	The hand became gradually weaker and more wasted, and the forearm also, but only slightly.	Loss of power. No fibrillary tremors.	No pain.	History extends over 2 years. The local use of electricity produced some improvement.	Amendment.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
47	Do.	M. 30	Merchant.	———	———	The right arm and the left leg were the only parts affected.	———	———	———	———
48	Do.	M. 20	Pharmaceutist. Very strong and muscular.	———	Right hand.	The disease involved the right hand and forearm in the most complete atrophy; but the rest of the arm and body in perfect health and great vigour.	Loss of power; electric contractility still remained.	———	The disease has been thoroughly arrested. It began 10 years ago.	Arrest.
49	Dr. Duchenne de Boulogne. De l'élect. local. p. 816.	M. —	Old soldier. Subject to lumbago.	———	Right shoulder.	It spread to all the scapulo-thoracic muscles on both sides; especially the serrat. magn. and rhomb. on the right side; lat. dorsi deeply affected. Forearms and hands untouched. Health excellent.	Loss of power. Fibrillary tremors.	None. No pain.	Three months from the onset he was assiduously galvanized for 5 months, with complete recovery of the affected muscles, except the trapez. and rhomb. The cure has already lasted 2 years.	Recovery.
50	Do. p. 578.	M. 15	A strong tall young man.	———	Right arm.	Right side—The biceps, trapez., rhomb., serrat. magn. and lat. dorsi have disappeared. Triceps untouched. Rest of muscles of this side sound. Left side—Triceps vanished; biceps not much wasted. Lat. dorsi, serrat. magn. rhomb. and supinat. long. gone. The forearms [illegible]	Loss of power.	None. No pain.	Continued to progress 7 years.	?

					of thigh much wasted.					
51	Ditto.	M. 38	Professor of Physiology. Good constitution.	None.	Left arm.	When the loss of power was first noted in the left arm it had already diminished in volume one half. This had been observed previous to the weakness. After destroying the muscles of the left arm, and attacking the forearm, it passed to the right upper limb, which it involved from below upward to the shoulder. The erectors of the spine greatly wasted. Rest of body sound.	Loss of power. A few fibrillary contractions.	None.	Has progressed for about 2 years.	?
52	Dr. Reade. Dublin Quart. Journal, Nov., 1856.	M. 18	Tall, healthy, young gentleman.	——	Shoulders.	Total atrophy of pectorals, muscles of anterior, and posterior regions of the neck, scapular muscles and muscles of upper arm. From elbows downwards muscles full and vigorous. After 6 years the muscles of neck and scapula recovered almost completely. Deltoids improved; rest of muscles of upper arms unchanged. Forearms and hands weaker than at first report; probably from inaction. Perfectly symmetrical on the two sides. General health unimpaired.	Loss of power.	Came on with pain.	History extends over 7½ years. Treated with seton in the back of the neck. Methodical exercise. Mercury and galvanism, with partial success.	Amendment.
53	Dr. Brittan. Brit. Med. Jl. Mar. 7, 1857.	M. 23	Shoemaker. Soldier. Had an attack of	Cold & wet.	Right upper arm.	The wasting was first perceived in the right arm, then in the left, soon after an at-	Loss of power. Flexion of forearm on arm	Came on with pain.	History extends over 6 months. Iodide of Potas-	Arrest.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
			acute rheumatism; also syphilis.			tack of acute rheumatism. In 2 months both arms no thicker than the wrist. All the shoulder and humeral muscles (except the biceps) much wasted. Triceps is a mere band. Forearms untouched.	strong; extension feeble.		sium given for a month, brought about a doubtful improvement.	
54	Moussons. Union Méd. de la Gironde. Gaz. des Hop. No. 108. 1857.	M. 26	Cork cutter; strong and healthy.	Typhoid fever.	Arms.	The two shoulders and arms greatly wasted, especially on the right, where skin seems only to cover the bones. Forearms very slightly touched. Ball of right thumb atrophied. Left normal. Lower limbs have suffered, especially the left. The thighs are greatly more affected than the legs.	Loss of power.	None.	Sulphur baths and regulated exercise employed with success; but electricity accelerated the amendment. History extends over about 2 years.	Great amendment.
55	Do.	F. 14	A woman in hospital, in perfect health.	Painful chaps in hand preventing movement.	Right hand.	The atrophy was confined to the thenar and hypothenar eminences of the right hand.	Loss of power.	Pain.	Had existed 10 years. No treatment. Hand affected with Chronic Eczema.	Arrest.
56	Author.	M. 33	Gentleman in robust health.	Mental irritation. Hard work.	Balls of thumbs.	On right side—Ex. digit. comm., interrosei, and all the muscles of thumb wasted away except ext. secund. internod. Rest of arm highly developed. Left side—The muscles of the thumb are the sole sufferers; these have	Loss of power.	Sensitiveness to cold.	Disease continued to progress for 2 years. For last 14 years perfectly stationary.	Arrest.

		37	Remarkably healthy. Had syphilis.	at his trade.	thumb.	disease involved the whole right hand, and a few weeks afterwards invaded the left. It travelled upwards, and in 15 months had reduced the two forearms to the most abject emaciation, with the exception of the pronator teres on each side. A remnant of the supinat. long. also existed.			for 15 months it became stationary, and had continued so for 5 years. Treatment—Strychnia, without avail. Slight amendment with electricity.	
58	Do.	F. 21	Domestic servant, strong and healthy.	——	Right shoulder.	It involves both shoulders and the top of the upper arms. Forearms and hands powerful.	Loss of power.	Slight pains between the shoulders.	Has lasted 18 months, and is now stationary.	Arrest. Slight amendment.
59	Do.	M. 67	Healthy tailor.	Working at his trade.	Right thumb.	Involved the muscles of the ball of the thumb and abductor indicis only.	Loss of power.	None.	Has lasted about 6 months.	Arrest.
60	Do.	F. 38	Domestic servant.	——	Right thigh.	It attacked the muscles of the thigh only. Those of the leg untouched.	Loss of power.	Occasional pain along the nerves and in the knee joint.	Has existed about 2 years. Galvanism used assiduously. Slow amendment.	Slight amendment. Arrest.

GROUP II.—*In which the Disease involved, more or less extensively, the Muscles of the Head, Neck, and Trunk.*

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
61	Niepce. Archiv. Gén. p. 626. 1853.	M. 42	Medical practitioner, of bilious temperament but good constitution.	Excessive exercise. ? Syphilis.	Back of neck.	Commenced with rheumatic pains in neck, back, and arms, with ague; in 6 weeks these disappeared; then there was noticed a wasting and weakness in the back of the neck and left upper extremity. Left side completely atrophied at the end of 3 years. The st. mast. and muscles binding scapula to trunk, and those of the entire upper limb (left) wasted. Right side—Deltoid enfeebled; remainder sound, as well as rest of body. General health good.	Chin rested permanently on chest.	Rheumatic pains.	History extends over 3 years. Remedies—Baths, moxas to the spine, without success. The sulphuretted and ioduretted thermal baths of Allevard and vapour baths arrested the atrophy, and a further use of these, with Iodide of Potass. brought about a very considerable amendment in 12 months.	Amendment.
62	Burcq. Canstatt's Jahresb .1853.	M. 45	Had delirium tremens.	Fall into cold water while perspiring.	Right hand.	The atrophy spread extensively; most marked in the right hand and toes. Muscles of face slightly affected. Tongue free.	Muscular twitches in the lower limbs and forearm. Fibrillary tremors.	Tactile sensibility a little duller, otherwise sensibility normal.	Rapid improvement by means of Burcq's metallic armour.	Recovery.
63	Gros. Gaz. Méd. Oct., 1854.	M. 20	Labourer.	Contusion 3 months previously,	Both shoulders.	Pect. maj. and min., serrat. magn., lat. dorsi, trapez. and rhomboid atrophied on both	Fibrillary tremors.	———	———	———

				completely recovered.		later, the muscles of the back wasted. Conical deformity of the thorax.				
64	Aran. Gaz. des Hop. No. 74. 1855.	M. 28	———	A month before invasion, fever with severe headache and general enfeeblement of all the members, which passed away.	Upper extremities	The upper extremities remained weak and the left arm began to waste, especially the muscles of the shoulder. Deltoid almost destroyed. Muscles of the thorax also attacked.	Loss of power; cramps.	Pains in the wasting muscles.	History extends over 18 months. Not yet subjected to treatment.	———
65	Do.	—	Workman.	———	Left lower extremity.	The atrophy of the left lower limb was spontaneously arrested and it recovered completely; but, 16 years afterwards, the upper extremities were seized, especially the left, which in 4 years had wasted away. The right was affected similarly, but in a minor degree. Several muscles of the trunk were attacked. Fattening of the individual, especially over the wasted muscles. Tendency to cephalic congestions.	Fibrillary tremors.	Sensitiveness to cold and slight numbness at first. Electric sensibility of muscles universally present. Persistent pains.	Continued altogether for twenty years; arrested for 16 yrs., it went on more rapidly for 4 years; but was again arrested by electricity.	Arrest.
66	Dr. Meyer. Wiener Wochensch. No. 41.	M. 54	A Clergyman. Always healthy up to 4 years ago.	Mental distress and a wetting.	Legs.	He first noticed a weakness in the legs, which continued 2 years without much change for the worse; then the arms began to waste, from the	Electric contractility diminished out of proportion to the atrophy.	Loss of power. Sense of weight.	History extends over 4 years, in the 2 last of which the disease was	Progressing.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
	(Canstatt, 1855.)					shoulders downward, so that in a year he could not lift the arms into the horizontal position. In the 4th year all the muscles of the trunk and extremities were to some extent wasted. Those of the shoulder and upper arm more than those of the forearm and hand.	Fibrillary tremors.		making rapid advance.	
67	Oppenheimer. Uebr. progr. fett. Muskel-at Heidelberg. 1855.	M. 18	Peasant. Always weakly	Onanism? At 17 crushed slightly in the thigh; at 18 fell from a waggon.	Lower extremities	The disease gradually invaded the muscles of the extremities and of the trunk, so that both arms and legs were useless. Some muscles completely annihilated. Skin blueish.	Loss of power. No fibrillary vibrations. Loss of electric contractility.	A little cold feeling.	History extends over 8 years of gradual progress. Localized electricity used for 6 months, without the least success.	Progressing.
68	Gros. Gaz. des Hop. No. 50. 1855.	M. 22	Carpenter. Had typhoid fever.	———	Extremities.	The disease spread in the course of 3 months over the upper and lower limbs, so as to deprive them of all movement except at the shoulders and hips. Atrophy spreading to the muscles of the trunk. Lividity of hands and feet. General health good.	Fibrillary tremors.	Remarkable decrease of the electric sensibility; pains.	Case came under treatment 3 mths. from date of invasion; and by daily use of electricity for 8 months almost complete recovery.	Recovery.
69	Do.	M. 51	Italian figure vender. Healthy; strong constitution.	Cold.	Thenar eminences.	During a period of nine months the disease attacked successively the hands, forearms, arms, shoulders, and buttocks; and when the case	Fibrillary tremors very marked, preceding the wasting and	Pain. The electric sensibility of the muscles faded in proportion	History extends over 18 months. Gradual progress in spite of baths, friction, iron, and	Progressing.

					full career of atrophy. General health good.	until the muscles were destroyed.	wasted. Headache.	cines; galvanism was also used daily for 5 months.		
70	Dr. Brittan. Brit. Med. Journ. Mar. 7, 1857.	M. 23	Cotton spinner also soldier, discharged on account of rheumatism of joints.	Rheumatism.	Left shoulder.	Came on gradually with aching pains in the shoulder, and stiffness and wasting of its muscles; later it involved nearly the whole body. General health good. The disabled limbs perspire very freely.	Loss of power. Can move the arms better at night, especially after exertion during the day.	Came on with pain.	Disease had lasted 12 months. Galvanism used, but he left hospital scarcely at all improved.	Arrest.
71	Cruveilhier. Archiv. Gén. 1853.	F. 40	General's widow.	———	? Upper limbs.	There was advanced atrophy of the muscles of upper extremities. Lower extremities less affected. Face almost devoid of expression. Gradual increase and extension of the atrophy to all the voluntary muscles, including those of respiration. General health unimpaired.	Loss of power. Monotonous voice.	None.	The wasting having reached the respiratory muscles, soon caused death. Treated by cauterisation over the spine.	Death.
72	Do.	M. 18	Shepherd.	? Masturbation. Cold.	Upper extremities	General atrophy of the voluntary muscles—some complete, others incomplete. All the muscles of the face implicated except orbicularis palpebrarum.	Loss of power and of facial expression.	None.	History extends over 5 years. Died of smallpox.	Death.
73	Do.	M. 30	Showman.	Cold.	Right hand.	For a year disease strictly confined to the right hand. Then it invaded the lower extremities, and spread gradually to nearly every region. General health perfect.	Loss of power; tottering gait; monotonous voice; fibrillary tremors.	None.	History extends over 4½ years. Treated by galvanism and every conceivable remedy without effect.	Death.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
74	Do. Archiv. Gén. Jan., 1856.	M. 40	Smith.	———	Upper extremities	From upper limbs the disease invaded the muscles of deglutition, and finally the feet. Shoulders especially wasted. The disease became general. General health perfect.	Loss of power; fibrillary tremors; monotonous voice.	None.	Died after remaining in hospital 8 months; treated by galvanism.	Death.
75 & 76	Do. Arch. Gén. p. 601. 1853.	—	Two children of same family.	Hereditary taint.	Muscles of the face.	No further particulars.	———	———	———	———
77	Do.	M.	Merchant. Strong constitution.	———	Tongue.	Tongue chiefly affected. The muscles of deglutition also touched.	Fibrillary tremors.	———	———	———
78	Aran. Archiv. Gén. p. 28. 1850.	M. 45	Sea captain. Two uncles and one sister had died of muscular atrophy.	Hereditary taint.	Upper limbs.	Commenced with cramps and twitches in the arms. From the arms the disease spread to all parts of the body, invading lastly the muscles of respiration. Although the disease was almost universal, few of the muscles had lost their contractility altogether, and nearly all the movements could be performed, though feebly.	Loss of power. Cramps and twitches always preceded the invasion of a new part.	Deep pains in the muscles about to waste.	Had run a course of two years. It proved fatal in spite of cauteries to the spine. 12 applications of galvanism brought about a visible amendment; but patient took cold, and was attacked with bronchitis.	Death.
79	Dr. Meryon. Med. Chir. trans. vol. xxxv. p. 73.	M. 8	Gentleman's son. Always inactive and weak on his legs. Could only walk 1 mile without resting, at 8	Hereditary taint. 4 brothers similarly affected. 6 sisters healthy.	Lower limbs.	Weakness of lower extremities gradually increased. At 11 years of age he could not walk a step—scarcely stand. Three years later the upper limbs became involved. The pharyngeal muscles were involved.	Gradual loss of power.	None.	Died at 16, after 8 yrs. of progress, from an attack of fever. Treated by tonics & mercury. Tenotomy practised.	Death.

		9	preceding.		limbs.	going, the child was observed to have no spring in the nurse's arms, but fell heavily when tossed up. The progress of this case was much more rapid. In 1 year he could neither walk nor stand, and in 5 years the upper limbs were assailed.	Gradual loss of power.	None.	This case was observed for three years. During the last year the disease has not advanced. Treated by electricity.	Arrest.
81	Do.	M. 5	Brother to preceding.	Hereditary	Right foot.	Not observed to proceed beyond weakness and dragging of right leg and foot.	Gradual loss of power.	None.	In the course of a year considerable amendment with electricity.	Amendment.
82	Do.	M. 2	Brother to preceding.	Hereditary	Lower extremities	Had not yet proceeded beyond the initial stage.	———	———	———	———
83	Do. and for the autopsy see Partridge Med.Gaz.1847	M. 9	Clergyman's son. Always conspicuously weak.	Hereditary Two brothers attacked.	Upper extremities	Both the upper and lower limbs were gradually involved so as to produce complete helplessness. No diminution of volume. Calves rather larger than usual, but quite impuissant.	Gradual loss of power.	None.	Temporary improvement by residence at Interlacken; but, afterwards, fatal advance. Patient died of Pneumonia in an attack of measles.	Death.
84	Do.	M. 9	Brother to preceding.	Hereditary	Upper limb.	Same morbid phenomena as preceding.	———	———	———	Progressing.
85	Do.	M. 12	Ill fed.	Hereditary A brother died of the same complaint.	Lower limbs.	The loss of power was gradually increased until at 20 the legs were useless. The arms then began to fail, and at 25 patient quite helpless.	Loss of power.	None.	———	Progressing.
86	Do.	M. 12	Brother to preceding.	Hereditary	Lower limbs.	Same history as the foregoing.	———	———	———	Progressing.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
87	Romberg. Klin. Ergeb. heft. i. & ii. Henle & Pfeuf. loc. cit.	M. 33	A waiter.	Cold taken in a damp cellar while sweating.	Ball of right thumb.	Commencing in the right thumb it extended to the fingers, and successively to the fore and upper arm and pectoral muscles; then it passed to left arm, and affected it in the same way. Later, the lower extremities, the muscles of the back, abdomen, and tongue were seized; and, lastly, it reached the respiratory muscles. General health good. Sexual powers active.	Fibrillary tremors very marked preceding & pointing out the track of the atrophy. Painless contraction of the left middle finger. Cramps in calves of legs.	None.	Complaint lasted 5 yrs., and terminated by attacking the respiratory muscles. It proceeded very gradually.	Death.
88	Do. Klin. Wahrnehm. heft. i. p. 52. Henle & Pfeuf. loc. cit.	F. 69	———	———	Tongue.	Tongue quite atrophied, and almost completely palsied. Muscles of deglutition and respiration afterwards attacked; also sterno-cleid. mast., and trapez.	Fibrillary tremors in the tongue; difficulty of breathing; impediment of speech.	Began with severe pains in back of neck, and later in the face. Sensation and taste perfect.	Gradual extension.	?
89	Landry. Gaz. Méd. No. 17. 1853.	M. —	———	Cold in a damp bed.	Left arm and hand.	Left upper extremity atrophied. Abscess in left foot, then in knee. Twice amputated. No pulse in left arm.	Loss of power came on brusquely.	None.	Lasted 9 months. Death in consequence of the amputations.	Death.
90	Oppenheimer. loc. cit.	M. 11	Two brothers, two cousins (males), & two uncles affected in the same way in early years. The uncles and 1	Hereditary	Lower extremities	After the lower limbs had wasted and lost power the upper were attacked; and, finally, the disease involved the muscles of the trunk, especially those of the back. Extensors of knee, & flexors of foot annihilated. Hands	Cramps in calves. The flexors of leg and muscles of calf became permanently contracted. Fibrillary	No pain. Sensation perfect.	After a course of 4 years the disease reached a fatal issue from bronchitis. There was tubercle in right lung. Treated with moxas, cauteria-	Death.

			dead. 2 other brothers, 3 sisters, and parents sound.			Health good.			medicines, without the least effect.	
91	Do.	M. 11	Brother to preceding.	Hereditary	Lower limbs.	Learnt to run only in his third year. At 10 years feebleness of lower extremities, with wasting of the muscles.	Loss of power.	———	———	———
92	Béraud. Canstatt's Jahresb. 1852.	F. 40	———	———	Trunk.	General destruction of the muscles of trunk. Face, eyes, neck, hands and feet free.	———	———	———	Death.
93	Dr. Meyer. Wiener Wochensch. No. 41. Canstatt 1855.	M. 24	Secretary.	Fatigue from militia exercise and a wetting.	The shoulder.	Began with a sense of fatigue in the arm while holding the musket; then, after a wetting, pains in back of neck came on, which soon gave place to a stiffness in the movement, and weakness, so that in a month the head fell forward unless supported. The mischief also spread in the shoulders and down the arms and forearms to the hands. In six months marked atrophy of the left side of the neck and thorax, and of the entire of both upper limbs.	Loss of power. The electric contractility weakened in proportion to the atrophy. No fibrillary tremors.	None. Electric sensibility of muscles diminished in the proportion of the atrophy.	History extends over about 6 mths. Electricity, applied for a month, improved the arms; but the legs showed signs of giving way.	Progressing.
94	Virchow. Virchow's Archiv. Band viii.	M. 21	Father died of same disease.	Hereditary	Lower extremities	This was a very chronic case. The disease first involved the lower, then the upper limbs; and, lastly, it became general. A temporary improvement took place 6 years from the onset.	Loss of power.	———	History extends over 23 years. It advanced to a fatal issue in spite of every remedy, including galvanism. It was complicated	Death.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
									with phthisis and albuminuria.	
95	Laboulbène. Union Méd. Dec. 15, 1855.	M. 44	Joiner. Strong constitution.	Hard work.	Arms.	Health very precarious for several years previous to invasion. Several attacks of temporary palsy, with loss of memory; then symptoms of myelitis (without pain, however); and, after this, complete atrophy of the upper extremities, followed gradually by wasting of the lower limbs. Later, the disease invaded the respiratory muscles.	Cramps; tonic spasms preceding the wasting.	Pain; sensitiveness to cold.	History extends over 1½ yrs. Temporary amendment by galvanism. He left hospital for some mineral baths in the Pyrenees, where he rapidly got worse. He then returned to hospital, and died in spite of diligent galvanisation.	Death.
96	Frerichs Wiener Wochensch. 1854.	M. 33	Sailor.	Wetting.	Hands.	Involved upper and lower limbs, especially the hand, forearm, and lower part of thigh. Facial muscles attacked. Lastly, it became general, and seized on the muscles of respiration.	Cramps. Fibrillary tremors.	Commenced with severe pains in the limbs; & this kept pace with the atrophy.	Continued steadily advancing for about a year. Treated with strychnia, with temporary benefit. Left hospital with death impending.	Impending death.
97	Schneevoogt. Niederlandsch. Lancet. 1854. (Schmidt's Jahrb. 1855.)	M. 58	Shoemaker. Subject to asthma.	Hard labour.	Left thumb.	After attacking the right thumb, both forearms were involved. The speech became indistinct; and, afterwards, the gait became uncertain. This gradually increased to palsy of the lower limbs, without apparent atrophy	Contracted pupils. Loss of power.	No pain. Oppression beneath the sternum.	Complicated with asthma, and evidently with true central paraplegia at the last. Treated with electricity 10 minutes each day. Blisters.	Death.

					Mention is made of involuntary discharge of urine. The disease finally reached the respiratory muscles. A tightness of the chest was a very early symptom.			nux vomica. The arms improved a little, but the legs got worse.	
98	Valentiner. Prag. Vierteljahrsschrift. 1855. (Schmidt, 1855.)	M. 45	Gentleman. Healthy.	Excessive exercise; fall.	Both hands.	Became general; for particulars, see p. 63.	Fibrillary tremors.	Sensitiveness to cold.	Lasted 1½ years.	Death.
99	Dr. Chambers. Med. Chir. trans. vol. xxxvii.	F. 26	Had always been very weakly.	———	Lower limbs.	The disease was marked in its early stage by difficulty in ascending stairs, and general feebleness of the lower limbs. She continued much the same for several years. About a year before death, she suffered spontaneous fracture of both femora; and, subsequently, it became evident that she was the subject of universal mollities ossium. The disease invaded all the bones, and lastly those of the chest; and she died suffocated.	Loss of power.	No pain except in the right arm.	Precise time of invasion not ascertainable. During the last 12 m'nths, steady progress. Complicated with mollities ossium. Treated with steel tonics, without effect.	Death.
100	Dr. Diemer. Günzburg's Zeitsch.— (Canstatt, 1855.)	M. 45	A forest ranger Healthy until 6 years previously, when he was troubled with rheumatic pains.	Cold in forest.	Right arm and leg.	The right arm and leg became gradually enfeebled, with proportionate muscular atrophy, so that in 10 months he was almost impotent on the right side. The wasting progressed to a fatal issue.	Loss of power. Fibrillary tremors.	Much pain; complained of deafness and abnormal sensations.	The disease lasted 1½ years, & issued fatally in spite of all remedies.	Death.

No.	Observer.	Sex and age.	Antecedents.	Alleged cause.	Starting point.	General Course and Extent.	Lesions of Motility.	Lesions of Sensibility.	Duration, Treatment and Complications.	Result.
101	Duchenne. De l'élect. local. p. 822.	F. 23	Servant.	Compelled at 11 years of age by a schoolmaster to carry a heavy stone in each upraised hand several hours a day for a twelvemonth.	Shoulders.	It extended to all the scapulo-thoracic muscles, those of the abdomen, some muscles of the lower limbs and of the face. The pectorals and deltoids full and vigorous. General health good.	Loss of power. No fibrillary tremors, tho' the atrophy seemed extending.	None. No pain.	Had been coming on 11 years.	Progressing.
102	Do. p. 824.	M. 31	Porter.	Carrying a basket on the head with the hands held up to keep it steady.	Arms.	The atrophy spread to the muscles of the neck, causing the head to fall forward, and to the erector spinæ, so that the back became curved. It extended also to the limbs. The other muscles vigorous.	Loss of power. Very abundant and widespread fibrillary tremors.	No pain.	Great amendment in a month by electricity. Two relapses from going to work too soon; and final cure.	Recovery.
103	Do. p. 614.	M. —	———	———	———	The atrophy had become general. Some of the muscles of the extremities had disappeared altogether. The muscles of the tongue and lips had been reached.	Abundant fibrillary tremors. Loss of power.	———	———	———
104	Do. p. 818.	M. 25	Mechanic. Tall, good constitution. Left handed.	Excessive labour.	Left arm.	In the course of 2 years the atrophy spread to the scapulo-thoracic, and scapulo-humeral groups of muscles on both sides; also to the dia-	Loss of power. Fibrillary tremors.	No pain.	History extends over 2½ years. After 6 months treatment the muscles of the arm	Recovery.

						phragm, causing much difficulty of breathing.			were restored completely; but the scapulo-thoracic muscles remained atrophied. The breathing was relieved.	
105	Do. p. 580.	M. —	Young man.	Heredit'ry. A sister 12 years of age had lost the muscles of the face.	——	The disease became general, and involved the face, a great part of the trunk, upper arms and thighs. The forearms, hands, and legs, excessively muscular and athletic.	Loss of power.	None.	——	——

CHAPTER III.

ANATOMICAL DISTRIBUTION AND SYMPTOMATOLOGY.

Out of the 105 cases which I have collected in the foregoing table, 60 belong to the partial and 45 to the general form. These numbers by no means represent the proportionate frequency in which the two groups occur; there is no question but that the partial form is relatively far more common than these numbers indicate; indeed, cases of atrophy of a limited group of muscles are within the experience of nearly every practitioner, and they are generally spoken of as *paralysis of the deltoid*, or *paralysis of the serratus magnus ect.*, according to the part disabled. In many of these, I doubt not, other muscles of the shoulder would have been found atrophied if search had been made for them, and they would have proved to be examples of the malady in question. Such a case is adverted to by Dr. Barton, in the *Dublin Hospital Gazette*,* in a paper "On Displacement of the Scapula from Paralysis of the Serratus Magnus." It occurred to Mr. Banner of Liverpool. The two scapulæ suffered displacement one after the other; their inferior angles projecting two inches from the surface of the ribs and pointing

* June 15, 1857.

toward the spine, precisely as in my fourth case. And a further clue to the true interpretation of Mr. Banner's case is supplied in the fact, that the lower extremities began to be affected, and the gait became unsteady. In fine, it was clearly an instance of wasting palsy, first attacking the scapulo-thoracic muscles, and then involving the lower limbs. Very often such cases have been confounded with palsy of the muscle from injury to its nerve—a condition altogether different in pathological signification, as well as in the more practical aspect of prognosis and treatment. *General* wasting palsy is, unquestionably, a rare disease; at least, if we are to judge by the small number of observations published since Aran awakened the interest of medical men in the subject, and the almost universal absence of them in the older records of practical medicine.*

* I am strongly inclined to believe that the following obscure cases were instances of general wasting palsy; but, from the curtailed account of the symptoms, and morbid appearances, it is impossible to speak with absolute confidence. Case 188, in Bright's "Reports," page 400, part 1, vol. ii., concluded, with an account of the post-mortem examination in page 631, part 2., vol. ii.; Case 67, in Bell's "Nervous System," p. cxxxii., with autopsy; also Bell's 57th Case, p. cxvii.; a Case published by Sir B. Brodie, in the "Lancet" for December, 1843, also with autopsy; and Case 157, in Abercrombie's treatise "On Diseases of the Brain," p. 400. In all these cases there was an unimpaired intelligence, perfect performance of digestion, urination, defecation, &c.; natural sensation, with almost universal loss of power in the muscles; and, after death, no lesion of the brain or spinal cord could be detected.

Anatomical Distribution.—Sir Charles Bell was correct in stating that this disease "is not a defect reaching so far up the limb, or so far down the limb;" but he was in error in supposing that it is universally "an affection of the muscles combined in action." As a general rule it is so; but many exceptions occur. In several instances, the triceps retained its full vigour when all the other muscles about the elbow joint had suffered destruction; and in my second case, the extensor secundi internodii pollicis continued active 14 years after the other muscles of the thumb had vanished. Commonly, however, when the destroyer falls upon a muscle, all its comrades are speedily involved.

In no case yet published have *all* the muscles of the body been found implicated in the same individual; and there are a few which seem altogether exempted. The muscles of mastication, and those of the eyeball, including the levator palpebræ, have not yet been known to be affected.

This disease confines its ravages, strictly to the muscles of the life of relation—those that are under the control of the will—leaving the involuntary muscular structures altogether untouched. Hence its career in the economy is easily traced, either by the changes produced in the external conformation—by the absorption of the muscular masses, the displacement of bones, and the abnormal position of joints from loss of their muscular supports—or, by the failure of certain move-

ments which contribute to outward expression or inward function, such as the facial physiognomy, deglutition, vocalisation, or respiration; all of which depend on the operation of striped muscles under voluntary sway.

When the disease is seated in the outer muscular layers of the trunk, or in the extremities, its features are very remarkable; and it is worth the trouble to make a short survey of them as they vary in the different regions. The simple *disappearance of the muscles* causes very notable changes of configuration. The symmetrical rounded contour gives place to a lean and withered aspect; the bony levers stand out in unaccustomed distinctness, and impart to the limb the appearance of a skeleton clothed in skin; but the skin itself, and the subcutaneous cellular tissue, have undergone no change, and cannot be distinguished from the integument of healthy parts. But loss of substance is not the only anatomical change observed. The natural equilibrium of the muscular forces is overset by the *unequal wasting* of the muscles; those less atrophied overcoming the resistance of those more deeply diseased. Hence arise very considerable, and sometimes peculiar, distortions of the head, trunk, and extremities.

Beginning with the hand, we find it frequently the seat of a very singular deformity. I allude to the "claw-shaped" hand, or "main en griffe" of the French writers; and my first

case, Rogers, offers a marked example of it. Imagine the plump, well-padded palm shorn of its cushions; ugly hollows occupying the sites of the thenar and hypothenar eminences; the palmar area traversed by the prominent, diverging lines of the flexor tendons, which are stretched between the wrist and the bulging bases of the fingers. The proximal phalanges are bent backwards, away from the hollow of the hand; while the middle and distal ones, inclined in an opposite direction, are in a state of continual semiflexion. At the back, the hand is correspondingly changed; the interosseous spaces are hollowed into long furrows, and the first joints of the fingers are pulled in a reverse direction, giving the hand a broken-backed appearance. This "main en griffe," was, I thought, produced by the overbalancing power of the common digital flexors, which, both in this disease and in lead palsy, exhibit a greater power of resistance to the atrophic changes than their antagonists. I conceived that the second and third phalanges being constrained into a continued flexion, the extensors would be considerably lengthened; but having lost their natural elasticity in the process of degeneration, and being reduced to mere membranous strings, they were unable to yield to the elongation, and, consequently, held back the proximal phalanx in a posture of permanent extension. Duchenne, however, regards the deformity as the direct effect of atrophy of the lumbrical and interosseous

muscles; which he considers "the sole antagonists of the extensors, for the first phalanx, and of the superficial and deep flexors, for the two last." *

Passing up the limb, the forearm is found flattened, or even hollowed, on its anterior and posterior surfaces; the arm reduced in circumference, and the muscular remnants, running down from the shoulder toward the elbow, appear like strings beneath the integuments. The whole limb hangs lifeless at the side; the roundness of the shoulder has given place to a flattening, and the head of the humerus, the acromion, and the coracoid process, are plainly designed through the thin covering of skin. If the serratus magnus be among the destroyed, the angle of the scapula stands out from the ribs and projects upwards towards its fellow. The wasting of the supra- and infra-spinati causes the spine of the scapula to start into unwonted prominence. Descending to the lower extremities, corresponding deformities have been observed. The foot is distorted by the unequal involvement of its intrinsic and extrinsic muscles, and there result contractions of the toes on the sole, deflections of the foot inward, or of the heel upward,—talipes varus and equinus—putting a complete bar to progression.

But, perhaps, the most remarkable of all the anatomical changes are seen in the face, when the muscles of expression are destroyed. The intelligent countenance is veiled, as it were, by an

* Op. Cit. p. 210.

impenetrable mask; no emotion changes its unvarying aspect—always solemn, stolid, and unmoved. The eyes, however, are spared, and by their movements alone (in the last periods) the immortal part holds some imperfect communion with the world around. The muscles about the mouth are the earliest disabled, and the saliva dribbles over the helpless lips.

Sometimes the muscles that support the head give way, and the head, no longer properly supported, falls forward, and has to be held up with the hand, otherwise it sinks downward, and the chin rests on the top of the chest (Nos. 61 and 93). Duchenne has described and figured an odd deformity resulting from the implication of the muscles of the abdominal wall. The lumbar curve is enormously exaggerated by the unopposed action of the erector spinæ, and the belly projects in front, while the upper chest is thrown back as a counterpoise.* When, on the other hand, the supporters of the back are damaged, the body is bent in a contrary direction; and the standing posture can only be maintained with great difficulty (Case 102, see Chap. x.)

But wasting palsy is frequently not content with ravaging the extremities and the external muscles of the head and trunk, it sometimes invades the interior of the body; and a new class of morbid phenomena engage the attention and interest of the observer. The tongue is often attacked, and a slight falter in the

* Op. Cit. 312.

speech is one of the earliest indications that the disease is about to assume the general character; indeed, in two instances, this was its starting point. Difficulty in masticating and swallowing food follows; the organ can no longer move the morsel in the mouth, in chewing; the patient fails to protrude it properly, and it has a soft, limp feel, and, not unfrequently, is the seat of violent tremblings. The involvement of the laryngeal muscles is known by a change in the voice, which loses its register, and is, finally, reduced to a single note. When the diaphragm and intercostals are reached, there are violent and suffocative fits of coughing; the play of the chest is reduced to a slight movement in the lower ribs, and the least impediment to respiration is now necessarily fatal. Dissolution is usually brought about by a bronchitic seizure; the air tubes are speedily filled with effused mucus, which no efforts can dislodge, and rapid asphyxia closes the painful scene.

When the atrophy is *not general* we observe that certain parts of the body, and certain groups of muscles, are much more obnoxious than others to the inroads of wasting palsy. The muscles of the trunk are less liable than those connected with the extremities; while, of the latter, the muscles of the upper limbs are far more liable than those of the lower. Of 62 cases, in which the atrophy was either undoubtedly partial, or only faintly foreshadowed an extension to the trunk, the upper extremities were alone the seat

of the malady 51 times, while the lower were exclusively affected only 5 times, and the upper and lower together only 7 times. The right arm is much more frequently attacked than the left; and the hands oftener than the shoulders. The following table gives a comprehensive view of the anatomical distribution of the atrophy in the 60 cases bearing the partial type. In the first column is told the total number of times in which the hands, forearms, upper arms, shoulders, and lower extremities were respectively affected; in the second, the number of times in which they were the sole seat of the disease, all other parts being healthy. The third column shows how often the two sides were implicated together; and the fourth and fifth indicate respectively the liability of the right and left sides to exclusive attack.

Table of 60 *cases of the partial form of Wasting Palsy, exhibiting the ratio of liability to attack of the different parts of the extremities.*

	Total No. of times affected.	No. of times exclusively affected.	Both sides together.	Right side only.	Left side only.
Upper extremities.	55	48	31	17	7
Hands.....	39	7	17	17	5
Forearms.....	30	0	15	11	4
Upper arms.....	32	0	18	8	6
Shoulders	33	3	22	6	5
Lower extremities.	12	6	8	1	3

From the figures of this table flow the following additional conclusions:—That, as a general rule, when one limb is attacked, its fellow on the opposite side shares its fate; that when the disease is unilateral, the right side is more likely to be its seat than the left; this is especially the case when it invades the hands, but far less so in the upper arms and shoulders, and the distinction disappears altogether in the lower limbs.

One of the most striking characteristics of this disorder is the capriciousness and uncertainty of its line of attack. Scarcely two instances are exactly alike in the combination of muscles implicated, and the relative degree in which they have suffered; hence an almost endless variety of feature. Yet wasting palsy has its favourite combinations; and there are some that it habitually avoids. I have examined the table of abstracts with a view of searching out this point, and have obtained some interesting results. When the malady strikes the shoulder, it scarcely ever fails to include the upper arm. Out of 13 cases in which these parts were the only ones affected, the shoulders and upper arms were attacked together 10 times; the shoulder alone only 3 times; and the upper arm alone, not once. Another very favourite combination is the forearm and hand. Out of 18 cases in which these alone suffered, there were 11 in which the two were blended together in one common ruin: in 7 instances, the hand stood alone; but the forearm

was not even once the sole part affected. It would seem as if the muscles which move the shoulder, and those which move the elbow joint, had a mutual sympathy in their relation to the progress of Cruveilhier's atrophy; and, likewise, that those which move the wrist and hand pair off in a similar way; whereas, the muscles which move the elbow, exhibit no community of behaviour with those which guide the wrist, and it seems not a little remarkable that not even in one instance hitherto published have the muscles of the upper arm and those of the forearm been associated together as the sole sufferers.

In the upper limbs it may be said that the morbid action radiates from two centres; one in the hand, from which the forearm is invaded, and the other at the shoulder, from which are reached the muscles of the upper arm, and those which brace the shoulder blade to the ribs. When the hand and forearm are destroyed, the evil does not then pass up the arm, but starts away to the shoulder or to the opposite hand. In the same way, when the shoulder is first attacked, the disease does not descend along the upper and forearm to the hand, but, passing over the elbow, it begins afresh in the ball of the thumb, and from that focus spreads up the limb; so that the parts latest reached are those about the elbow, especially the masses that take their rise from the humeral condyles. Whether a corresponding peculiarity in its extension, exists in the lower extremities,

cannot, as yet, be ascertained, owing to the limited number of observations.

Other combinations deserve mention from their being still more strange, although less frequent. In 3 cases the shoulder and upper arm were selected on one side, and the forearm and hand on the other; and in another case the right arm and left leg were the only parts involved.

SYMPTOMATOLOGY.—I propose to consider the symptoms under the separate heads of lesions of motility and lesions of sensibility, and to conclude the chapter with some remarks concerning the complications of wasting palsy.

Lesions of Motility.—The chief of these is loss of power; the secondary ones are fibrillary tremors, cramps, twitches, and diminution of the electric contractility.

The loss of power corresponds in the typical cases to the grade of the atrophy of the muscle; and, inasmuch as the muscle goes on decaying slowly, for months and years, its force declines at the same deliberate pace; so that it is only in extreme cases that any part is reduced to absolute immobility. More commonly the movements are still capable of being executed but with so feeble a force as to be of no service to the individual. The correspondency here claimed between the bulk of the muscle and its contractile power is a distinctive feature of the disease, and serves to separate it in a marked manner from hemiplegia, paraplegia, facial paralysis (of Bell), and,

indeed, from any palsy depending on an injury to the cerebro-spinal centre or a nervous trunk. In these latter, the loss of power is sudden—generally instantaneous, and seldom delayed beyond a few hours or days—and the subsequent wasting of the muscles, if there be any, holds no proportion whatever to the loss of power; but here, as the degeneration advances, *pari passu*, the strength abates, and it is not altogether abolished until every fasciculus and every fibre is destroyed; while, to the last, the muscle remains obedient to the calls of the will. In the uncomplicated cases this appears to hold true to the letter—it applies to my first and second cases, and to a multitude of others; but it is undeniable, that, in certain instances, this exact correspondence between the wasting of the muscles and the loss of power is departed from. The palsy sometimes overshoots the atrophy; and sudden variations of amendment or aggravation have been observed, not at all consonant with our ideas of a defect dependent solely on an equably acting cause, such as muscular degeneration. In the case of Lecompte, after a second night passed in the open air, the weakness of the limbs underwent a sudden increase. A patient of Diemer (No. 39) could walk for some hours, at times, while at others he was unable to stir at all. These exceptional cases will be again noticed in the 7th chapter, and some observations explanatory of their occurrence will there be made.

Muscular vibrations are the most remarkable of the subsidiary or unessential symptoms. They make their appearance as little convulsive twitchings or quiverings of individual muscular bundles; they do not impart any movement to the entire muscle, but spring beneath the skin in quick momentary tremors, undulating over the surface of the muscle. They were first observed in the famous case of Lecompte, and Aran thus expresses himself concerning them:—"In examining the lower limbs of this man we were struck with a very extraordinary phenomenon. The muscular masses on the front of the thigh were overrun at every instant, while in complete repose, by fibrillary contractions, which appeared here and there in wavy oscillation, raising the skin at varying points, and without the patient having the least consciousness of their existence. In fact, there were no contractions of the whole muscle—like those the patient called our attention to in speaking of subsultus of the tendons or cramps—but isolated contractions of the distinct fasciculi which compose the muscle. If the skin was excited, by touching it with a cold substance, for example dashing it with cold water, the contractions became more vivid and frequent. They increased in intensity especially, when the member was raised and the muscle pressed on, as I did in applying the stethescope. There was then felt beneath the instrument a sort of shock—a jerk, then a check—which indicated the com-

mencement and termination of the fibrillary contraction." * On looking for them, they were found elsewhere, in several places, especially when the patient held the limbs in a posture of forced extension.

They have since been found in a great number of cases; indeed they are rarely absent when the atrophy is actively progressing. They are brought out and intensified in several ways besides those above-mentioned. Stripping the patient quickly and exposing the surface to the cold air; passing a galvanic current through the part; filliping or tickling the skin; attempting violent movements; in all these ways their presence may be evoked and their activity quickened.

An examination of the table of abstracts shows that fibrillary tremors are not universally present, even during the active stage. As might be expected, in the older records no mention is made of them; but it does not follow that they were absent, inasmuch as they might easily have escaped detection, unless the observer's attention was awakened to their existence. In most of those recently published they have been noticed when the atrophy was progressing; but some cases reported by Duchenne prove this not to be invariable. Nor are they altogether unknown in other palsies and muscular degenerations. I have under my care, at the present moment

*Archiv. Génerales, p. 33, vol. xxiv.

(August, 1857), in the Royal Infirmary, a man, whose right arm is greatly atrophied and nearly deprived of motion, in consequence of the long continued pressure of the unreduced head of the humerus on the brachial plexus in the axilla. The deltoid muscle, in this man, is the seat of most marked muscular vibrations. Duchenne, also, mentions that he excited the phenomenon, by a galvanic current, in a woman suffering from spinal palsy.* But, abstracting these two instances, fibrillary tremors have not, so far as I know, been observed, except in Cruveilhier's palsy; so that, although they are not absolutely confined to this disease, they are so nearly so, as to constitute a valuable diagnostic sign.

It must be remembered that they are never present in muscles wholly destroyed, nor in those which have ceased to be the seat of active disease; hence they must not be looked for in very old cases, where permanent arrest has taken place. This, probably, is the reason why they were not present in my own observations, in all of which the disease was stationary. The occurrence of fibrillary tremors is a sign, therefore, that the malady is advancing; and it is particularly mentioned, in several instances, that every aggravation of their violence was speedily followed by an increase of debility and extension of the atrophy. Sometimes they serve to indicate the line of march the disease is following; and their

* De l'éléctrisation localisée, p. 617.

appearance in muscles distant from the morbid focus, and which have, as yet, exhibited no symptom of weakness or wasting, is a fatal warning of their impending destruction.

These vibrations are not in continuous activity even where their presence has been ascertained. They come and go, and their violence varies from time to time. In Valentiner's case, related in the preceding chapter, they are said not to have been present every hour or every day, but at long intervals. The patient himself is un-unaware of their existence, unless they are very violent and his attention is especially called to them. Dr. Wachsmuth's patient could *feel* them; and described the sensation "as if a finger lightly passed over or tapped the skin." In one or two cases, they were so violent as to be a source of torment to the sufferer, and a prominent phenomenon to the observer; more frequently they are of moderate intensity; and sometimes are so feeble and delicate as to require attentive searching to detect them. Generally, they prevail spontaneously; but, sometimes, only appear on laying bare the limb to the cool air, or lightly pinching or tickling it.

Cramps and twitches differ from fibrillary tremors in that they shorten the whole muscle, and, therefore, move the limb or part to which it is attached. They are often present; sometimes accompanied with pain, troubling the patient at night and preventing sleep. Generally, they

pass away without leaving any memento of their presence; but, sometimes, they lock the joints in some unnatural position, producing a persistent deformity. They are to be regarded as complications dependent on cold, or some (so called) rheumatic affection of the nerves, and forming no essential part of the symptomatology of the complaint.

The relation of the muscles to the galvanic stimulus has engaged a good deal of attention, especially from Duchenne and Dr. Meyer; and, if the statements of the former had been confirmed in other quarters, a most important aid in the diagnosis would have been secured. Duchenne asserts that the electric contractility of a muscle undergoing Cruveilhier's atrophy gradually dies away with the voluntary power, and does not altogether fail until the muscle is annihilated; and that this is so surely the case, that a palsy accompanied with an elevation or even perfect preservation of the electric muscular contractility, or one in which it is abolished, or depressed out of all proportion to the grade of the atrophy, may at once be confidently pronounced *not* to be an example of wasting palsy. But facts do not warrant so absolute a rule; Dr. Gros found the voluntary power returning in one of his cases while the battery failed to elicit any response. A few other contradictory instances might be mentioned; but I am convinced, both from my own finding, as well as by the recorded

experience of others, that Duchenne's conclusions are, as a rule, correct; and, for undue elevation, or even perfect preservation of the electric contractility, that they are absolutely so. (See Chap. vii.)

Lesions of sensibility.—In the uncomplicated cases there is no interference whatever with the sensibility of the part. Tactile sensation retains its delicacy in the skin over the affected muscles. When the tongue is attacked, neither the gustatory sense nor its common sensibility is in the least blunted; and, according to Duchenne, the special muscular sense survives to the last. There is neither pain, numbness, nor any other abnormal feeling.

But matters do not always pass thus; in a large minority of cases the sensory nerves are untowardly affected; and pain, sensitiveness to cold, and even slight numbness are added to the more constant symptoms.

Out of the 105 tabulated cases no mention is made of *any* lesion of sensibility in 67 — considerably above half the number—and in nearly all of them it is expressly stated that there were none present; and if, in some, silence on this point does not afford a sufficient warranty of their absence; in the greater number, the amplitude of the details in other respects, and the open and striking character of such symptoms, amply justify that inference; so that it may be safely asserted, that, in at least one-half of the cases,

abnormal sensations were wholly unknown in the diseased parts and their neighbourhood.

Tactile and *common sensibility* continued altogether unimpaired in 102 out of the 105 examples. In the remaining three, a little numbness of the finger ends is reported; in one it was temporary, and soon passed away; in another, related by Romberg, although there was a sense of numbness, confined to the index and middle fingers, it is expressly added that "the skin of the finger continued very tender to the touch." To Oppenheimer's patient objects felt flatter than natural—perhaps from curtailment of movement in the tactile organs, rather than any diminished delicacy in the recipient nerves. In only one instance, was the cutaneous sensibility unduly raised.

Pain is by far the commonest of the symptoms at present under discussion. Its presence is notified 25 times, in or about the seat of the disease; and 26 times its absence is expressly mentioned. When present, it varies greatly, in degree and kind, in the parts it affects, and in the period of the disease at which it prevails. Sometimes it is a slight wandering pain in the neighbourhood of the wasting muscles; at other times, it is sharp and lancinating, shooting down in the course of the nerves, having all the characters of *neuralgia;* or it may be of an aching kind, affecting the joints and the substance of the muscles; resembling, and often called, *rheumatic*

pain. In one patient it was so trifling as scarcely to attract attention; in another—as in Mr. Mayo's—so violent that "he felt as if his arm was coming off."

In several instances it marked the onset of the disease, and passed away as the atrophy set-in in good earnest; but more frequently it followed the steps of the morbid agent, and served to point out its presence. It is very seldom persistent throughout, even during the active stage; more commonly it comes and goes at intervals, or continues for a time, and then departs altogether.

Unusual sensitiveness to low temperatures is, occasionally, a prominent and very annoying symptom. It is reported 14 times. In Dr. Valentiner's patient, a violent shivering came on, if the room was not moderately warm. Another felt a very great falling off in his muscular power in the cold; while in Wachsmuth's shoemaker, artificially warming the maimed arm, enabled him to pursue his labour for hours, when without this device, he was quite disabled. In my second case, washing the hands with cold water, even in the summer, left an enduring sense of debility and numbness.

Pains, in parts distant from the seat of disease, are occasionally complained of. In a few instances there was headache, though only in one case, was it severe or persistent; uneasiness in the back and tenderness over the spine are also mentioned in a few cases. But the trifling intensity, or the

the evanescent duration, of these symptoms, deprives them of the importance which might otherwise be attached to them.

The *general health* is not affected. The intelligence is clear, the judgment firm, and the emotions duly restrained. Digestion, circulation, nutrition (except that of the muscles), the evacuation of urine and fæces—in short, all the organic functions are performed with the utmost regularity. Respiration, however, may be very seriously compromised, in an indirect way, by the involvement of the diaphragm and intercostal muscles, as already explained. In long-standing cases, an effusion of serum sometimes takes place into the subcutaneous cellular tissue of the affected extremities; and the skin assumes a livid appearance. Both œdema and cutaneous congestion are uncommon accompaniments of wasting palsy; and are produced by secondary and purely mechanical, causes. (See Chap. vii.)

Complications.—Like every other complaint, Cruveilhier's palsy may be associated with other maladies. The most common complication is neuralgia, either in the nerves of the diseased part, or elsewhere, as in the opposite limb or in the face. So called muscular rheumatism seems also very often the source of the pain. Phthisis (No. 27), tubercle and albuminuria, (No. 94), and mollities ossium (No. 99), have likewise been observed as concurrent affections; but the few-

ness of the exceptions goes to establish the prevalence of the rule, that, putting aside neuralgia, wasting palsy is a disease conspicuous for its infrequent complications.

CHAPTER IV.

INVASION, COURSE, DURATION, AND TERMINATION.

The invasion of wasting palsy is usually slow and insidious; it creeps on unawares, and the victim of its attack only becomes cognisant of the redoubtable enemy that has effected a lodgment in his system, on the occasion of some marked failure in the muscular power. The tailor discovers that he cannot hold his needle; the shoemaker wonders he cannot thrust his awl; the mason finds his hammer, formerly a plaything in his hand, now too heavy for his utmost strength; the gentleman feels an awkwardness in handling his pen, in pulling out his pocket handkerchief, or in putting on his hat. One man discovered his ailment in thrusting on a horse's collar; another, a sportsman, in bringing the fowling-piece to his shoulder. In whatever way the deficiency is discovered, it is next noticed that the weakened member is greatly wasted; its muscles do not possess, perhaps, half or quarter the bulk of those of the opposite side; and it is then often remembered, that this at-

tenuation had been remarked some time previously, but had not attracted any serious attention, because the strength had not materially given way. The failure of power, perhaps, when first detected, slight and little thought of, soon increases; and, although all the movements proper to the limb, still remain, their vigour is lost. The arm may be raised, or the hand opened and closed, but the lifting power is reduced to nothing, the grasp is gone; and so, by little and little, the power and usefulness of the limb are abstracted, until at length it becomes completely palsied.

The gradually diminishing volume and vigour of the affected muscles, is either discovered all at once, in the ways above mentioned, or it is slowly forced on the conviction as the defalcation becomes more and more prominent. As Aran pointed out, the disease is not detected at the outset, partly because the waning power is made up by increased effort, unwittingly put forth by the patient to cover the deficiency; partly, also, because the part, soon wearied, is often rested, and allowed to renew its exhausted strength. Besides, other muscles of kindred function, which have not suffered, take up, to a certain extent, the duties which their maimed comrades are unable to perform. In this manner, the increasing defect is for a period concealed; but, at length, an unusally severe, or prolonged muscular exertion, lays it bare all at once, and the patient, alarmed,

seeks medical aid.* On this account, it is seldom that these cases are seen before the disease has already committed profound ravages, and several months have elapsed from the real *début* of the complaint. These considerations sufficiently explain the apparently sudden invasion in some persons; and why it is, that the advancing decline of force and bulk does not earlier attract attention. Cases there are, no doubt, where another solution must be sought, for the abrupt onset; and this is to be found in the secondary and subsidiary phenomena, chiefly of a nervous nature, which re-act in a powerful manner on the already damaged muscles, and add a new source of weakness to that which already existed.†

The *starting point* of Cruveilhier's atrophy is generally in the upper limb, even when, in its subsequent development, it assumes the guise of the general disease. Of 94 cases, in which the starting point is expressly mentioned, 56 were

* An interesting parallel to this gradually-increasing, yet suddenly-discovered loss of muscular power, is found in Dr. Kane's "Arctic Explorations." "The imperfect diet of the party," he goes on to say, "was showing itself more and more in the decline of their muscular power. They seemed scarcely aware of it themselves, and referred the difficulty they found in dragging and pushing to something uncommon about the ice or sludge, rather than to their own weakness. But, as we endeavoured to renew our labours through the morning fog, belted in on all sides by ice fields, so distorted and rugged as to defy our efforts to cross them, the truth seemed to burst upon every one." Vol. ii. p. 262.

† See Chap. vii.

partial, and 37 general; and the following table shows how often certain parts were selected as the starting point in the two groups :—

Table showing the relative liability of parts to be primarily affected.

	Partial.	General.	Total.
Trunk	0	1	1
Back of neck.....	0	1	1
Muscles of the face	0	2	2
Tongue	0	2	2
Shoulder	12	4	16
Hand.....	25	7	32
Lower extremities.....	7	14	21
Upper extremities.....	48	19	67

It is seen from the table, that, in the partial form, the disease commences in the overwhelming majority of cases, in the upper extremities; but where the disease afterwards became general, it is found to have commenced nearly as frequently in the lower as in the upper limbs. It matters, therefore, very considerably, in the prognosis, to know whether the point of departure was in the arms or in the legs; for, if the latter, the probability is very much increased, that the atrophy will spread to the trunk, and thus bring life into jeopardy.

In more than one-third of the entire number, and in about one-half of the partial cases, the hand was the member originally seized; and the exact spot is nearly always the ball of the thumb, which, of all the parts of the body, is most liable to the incursions of Cruveilhier's atrophy. When the two sides are compared, it is found that the right hand is greatly more obnoxious to its assaults than the left; for, while the latter was selected only 7 times, the former was chosen 20 times; and, in 5 instances, both hands were seized simultaneously.

Next to the hands, the shoulders are the favourite starting point; and it may be remarked, that there is no apparent difference in the liability of the two sides. The right was seized 4 times, the left also 4 times; and the remaining 8 times, the two were attacked concurrently. In the lower limbs, so far as the facts at my disposal enable me to judge, there is no preference for either side.

Wasting palsy is essentially a chronic disease—it runs its course slowly; we measure its advance, not by days and weeks, but by months and years. Its duration is found to vary through a very considerable range. Some cases complete their history in six or eight months; others linger through many years. In calculating the exact duration, I have guarded against including any periods subsequent to the occurrence of permanent arrest; which, as I shall presently explain,

is one of the natural terminations of the disease. Bearing this in mind, I sought out all the cases in the table which had reached some final issue—recovery, arrest, or death—and in which the time of duration was expressly stated. Out of 105, there are only 28 which fulfil these requirements; of these, 4 ended in recovery, 13 in arrest, and 11 in death. The mean duration of the whole was 38 months, and the following table shows how it varied for the three groups of cases :—

Table showing the duration of Wasting Palsy.

Mean duration of	4	cases ending in	recovery..	1 yr.	2 mths.
"	13	"	arrest	2 yrs.	3 mths.
"	11	"	death	5 yrs.	2 mths.
General mean				3 yrs.	2 mths.

The maxima and minima in the numbers from which the table is constructed, are very wide apart, especially in the second and third groups. The greatest duration of a case, ending in recovery, was $2\frac{1}{2}$ years, and the shortest, 8 months. The longest case ending in arrest, continued active for 7 years, and the shortest for 4 months. Fatal cases have not been known to terminate under a twelvemonth, while one lingered for 23 years, another for 8, and four more beyond 4 years. It is important to note that cases which issue in recovery run a much briefer career than those which end in death. The table does not

bring out this difference so saliently as it deserves; for, in the duration of a recovered case, the whole time from onset to issue is included, whereas it is evident that this interval not only embraces the time of morbid activity, but the additional time in which nature, after the cessation of the diseased action, is occupied in restoring the strength and volume of the injured muscles. In order to compare correctly with cases ending in arrest or death, only that time should be counted which intervenes between the invasion and the arrest of the morbid movement. Such a calculation, however, I have failed to make, with any approach to accuracy. I note this fact to bring out more prominently the deduction which flows from the table in respect to the success of treatment: the more recent the case, the greater the probability of success in applying remedial means; and, conversely, the longer the disease has lasted, the more desperate becomes the prospect of amendment.

We may also study the duration of the disease in those cases which, at the time that the observations were recorded, had not reached any final issue. To such cases, in the table of abstracts, are appended, in the column for results, the words "*amendment*," or "*progressing*," according as the atrophy was, at that time, retrograding or advancing. Of the cases which were improving at the date of record, 9 are available for our present purpose; and their mean duration was

2 years and 11 months; while that of the examples marked as still progressing, of which there are 14 to answer the same enquiry, was 3 years and 7 months. From these various data we obtain a tolerably distinct understanding of the period of activity of this disease. It may run its course in 4 or 6 months, or drag its weary length along for 5, 10, 15, and even 20 years; but, generally speaking, it brings its operations to a close in from 2 to 4 years.

Wasting palsy may *terminate* in one of three ways—namely, in recovery, permanent arrest, or death. The second mode of termination occurs when the wasting of the muscles ceases, and the limb continues for an indefinite period in its maimed condition, neither amending nor deteriorating; the muscles which are entirely destroyed do not re-appear, and those which are only half or quarter consumed, continue to exercise their feeble powers under the control of the will, but do not regain their former bulk, or vigour. This stationary condition being once ushered-in by the arrest of the atrophy, the disease may be said to have reached its ultimate term; and the skeleton-like footprints it leaves behind are to be regarded, not as the malady itself, but, like the scar of a healed-up wound, only as the commemoration of a morbid activity which has now altogether passed away.

Generally speaking, when the disease has fairly entered on the stationary phase, and has

continued so a year or two, there is very little danger that it will resume its active career. Individuals have lived 5, 10, 20, and even 30 years, with their crippled limbs unaltered; and of one it is recorded, that life was brought to a close by pulmonary phthisis, 10 years after the atrophy had stood still. But this is not the invariable sequel. After lying torpid for years, the malady may awaken to new and more violent activity, and run an accelerated career of destruction. M. Aran has related three interesting examples. One was a woman, who, when a child, had atrophy of the muscles of the right hand, from which she recovered completely in her 12th year. When 40 years of age, she was attacked again in the same place. In another instance, more recently published, the right leg became the subject of wasting palsy, but gradually recovered; 16 years after, the shoulders were seized, and the disease involved both upper extremities, and several muscles of the trunk. In a third case, the right leg was the seat of debility and atrophy; after remaining quiescent for 8 years, the disease started into fresh activity, in the left leg and right arm. Virchow relates of his patient, that 6 years after the onset, an arrest took place, and the disease continued stationary for a time, and even underwent some amendment; but, subsequently, it resumed its slow march, and, at the end of 23 years, terminated in death. Tempo-

rary lulls in the progress of the atrophy, are common enough, either from the successful employment of remedies, or spontaneously arising. They seldom last long, however; in a few weeks, or months, the work of destruction is recommenced, and pushed on, probably with more vigour than before, to a final issue.

The mode of termination was ascertained in 49 only out of the entire number of cases; 9 ended in recovery, 22 in permanent arrest, and 18 in death. Of the remaining 56, 11 were in process of amendment, 15 were still progressing; and, concerning the 30 residuary cases, it is not clearly stated, whether they were in a state of activity or at a stand still. The following table shows the different terminations in the two forms—partial and general.

Table showing the terminations of Wasting Palsy.

	Partial.	General.	Total.
Final issue in recovery	5	4	9
" arrest	19	3	22
" death	0	18	18
At date of record amending.....	9	2	11
" still progressing ..	6	9	15
" condition not explained.	21	9	30

This table brings forth into prominent light the fatal nature of the disease when it invades

the trunk; three-fourths of the general cases which had reached a final issue, having ended in death, while only 4 had recovered and 3 undergone permanent arrest. The partial form, on the contrary, in no way menaces life; it issues always either in recovery or permanent arrest, unless, indeed, by extension of its operations it passes over into the more fatal type; which event, strictly speaking, ought to be regarded as its third mode of termination. Nearly all the "arrests" and "amendments" took place in the partial form; and although the number of "recoveries" is, proportionally, even somewhat greater among the general cases, it is not to be supposed that the recoveries are really more frequent in that class; quite the contrary, as the overwhelming number of "amendments" in the partial form sufficiently shows.*

* The cases here noted as recoveries are, it should be known, for the most part, incomplete or partial cures; only a few cases having yet been recorded in which the normal state had been perfectly restored; but I have judged it not inadmissible, in a disease so little amenable to treatment, to call those recoveries, in which not only had the morbid action been altogether arrested, but the larger number of the affected muscles restored to their original bulk and strength; and also those cases, in which the amendment had advanced so far, that a perfect cure was counted-on by those who reported them.

CHAPTER V.

CONDITIONS OF ORIGIN—ETIOLOGY.

An examination of the conditions of origin of Cruveilhier's atrophy sheds only a faint, yet, from the general obscurity of the subject, a valuable light, on its pathology. It is a disease of mature years; affecting males much more frequently than females; not confined to any particular kind of occupation, nor to any separate class in the social scale.

The mean *age* in the 88 cases in which it was recorded, was 30 years and 6 months; in the partial atrophies, (49 cases,) the average was 32 years and 4 months; and in the general, (39 cases,) 28 years and 3 months. According to this calculation, the subjects of the latter are attacked somewhat earlier than those of the former; but the discrepancy arises from the fact, that general atrophy favours no particular age. It seeks victims among children, among adults, and in advanced age; whereas the partial form very rarely falls on individuals under adult age, or over 50. In 10 instances of the former, the patients were under 12, and two more are reported as children; on the other hand, one was 69 and

another 54. If these extreme numbers be abstracted, the prevailing ages for the two forms are, as nearly as possible, the same.

Of 99 cases in which we have information as to the *sex*, 84 were males and 15 females—a disproportion so striking and constant that it cannot be attributed to accident. Aran signalised it in his 11 cases; and it has been noted more recently by Wachsmuth and Meryon. It depends in part, probably, on the greater and more sustained muscular exertion which men's occupations demand. Women who engage in needle-work, washing, and household service, are seemingly not much less liable than men employed in kindred occupations; for, I find that of those whose labour did not press excessively on any particular sets of muscles, as domestic servants and cultivators of the soil, females formed a much larger proportion than of those in easier circumstances. Of the former nearly one third were females; whereas of the latter 18 were gentlemen, and only one a lady. Now, there is no comparison whatever between the muscular exercise of a lady, who is waited-on by her domestics, and that of a servant, a washerwoman, or a housewife who is necessitated to undertake her own household duties. Gentlemen, on the contrary, either from pleasure or necessity, usually go through as much and as severe muscular labour as the daily workman. But in addition to harder work, men are much more exposed to wet and cold; and these,

we shall find, are frequently alleged causes of muscular atrophy. This explanation is less satisfactory when applied to cases, which own for a cause a hereditary taint; the males are even still more in excess among these; for they range in the proportion of 7 to 1; and this notwithstanding that in them the disease is very apt to make its appearance at an early age, even in childhood, and long before the sexes are unequally subjected to fatigue and exposure.

In 70 instances we are supplied with information as to the *occupation* and *social standing* of the patient. Nineteen of them were in easy circumstances—merchants, professional men, owners of property, clergymen, book-keepers, etc.; and, as above stated, all were males except one.* Twenty were handicraftsmen—mechanics, masons, shoemakers, tailors, (and one needle woman), smiths, etc.; 34 followed occupations of promiscuous kinds—domestic and farm service, washing, housewifery, etc.; 2 were sailors, and several were labourers. It is worthy of remark that among the handicraftsmen the atrophy had become general only in a fourth of the cases, while among the gentlemen nearly half bore that type, and more than half among those following promiscuous occupations. It should likewise not be overlooked that the artizans constitute over one

* Six of Dr. Meryon's cases were gentlemen's sons; but, being boys under 12, they are altogether excluded from this enumeration.

fourth of those about whose occupation we have any knowledge—a proportion very considerably greater than they make up in the general population of the country.

There is no point connected with the etiology of the complaint, so firmly grounded as the influence of *consanguinity*. We have information of 10 families in which the disease had taken root, and they have supplied 18 cases sufficiently detailed, to earn a place in the analytical table; but mention is made in the narrations of additional relatives, affected with wasting palsy, whose histories it was not possible to write. In 4 of these families, the disease was confined, in each, to two brothers. Dr. Meryon's first-described cases were 4 boys, who had 6 healthy sisters. In another family mentioned by him, all the boys, namely two, were affected, while the two sisters were sound. A sea captain, whose history is related by Aran, had lost two maternal uncles and a sister by the same disease; but two other sisters, and three brothers, continued in perfect health. In another instance, more recently recorded by the same observer, the patient's two aunts had died from general muscular atrophy; and, in a family known to Oppenheimer, two uncles and a cousin were already deceased; while another cousin (both cousins were males) and two brothers, still suffered from wasting palsy. Another had lost his father, by the same complaint. Altogether, these 10 families included

29 individuals struck with this terrible disease; and of these, only 4 were females. It is quite inexplicable why the male sex should be so much more obnoxious to its inroads than the female. The females, do not, however, always escape; and, as in one of Aran's observations, a sister may be attacked while the brothers go free. Cases arising from hereditary taint, present another noteworthy peculiarity — nearly all of them eventually become general; and, in consequence, in the great majority of cases, tend to a fatal termination; in only 3 out of the 18 hereditary cases mentioned in the table, was the disease confined to the extremities — a proportion which is the inverse of that obtaining in the entire series.

To complete this account of the antecedents of Cruveilhier's palsy, it only remains to state, that the patients are usually reported, of excellent constitution, of good physical development, and gifted with ordinary intelligence. Several times it attacked men of remarkable muscular power, glowing health, and unusual psychical endowments. In 9 instances a certain weakness had existed from early youth; but 7 of these owned a hereditary cause; and the 8th was not a simple case, being complicated with mollities ossium. Indeed, it might be said, without exaggeration, that the victims of wasting palsy are the strong, the healthy, and the intelligent; and these it attacks in the flower of their age, cutting them off

altogether, after a long and doubtful struggle, or leaving them, in the midst of their usefulness, helpless and deformed cripples.

Coming now to the *special determining causes*, we are at once struck with the large proportion in which no assignable cause can be adduced; these form over 36 per cent. of the whole. Of the remainder (69 cases), 25 are attributed to hard work, in one guise or other; 16 to cold, or cold conjoined with wet; and 18 to hereditary predisposition. These are the three chief causes alleged to produce wasting palsy. In a few instances, other causes are doubtfully put forth; 1 came on after scarlet fever; 2 after typhoid fever; 5 had been preceded by violence, and 1 by rheumatism. But, in all of them, the original disease, or injury, had been completely recovered from; and, it is to be noted, that the injuries mentioned did not concern the nerves going to the affected muscles; nor, indeed, could they be linked to the atrophy of the muscles in any way, except as antecedent circumstances. Venereal excesses, and onanism, were suspected in several cases, and proved to have been practised in a few; but these were not brought home as the originators of the disease, in a single case, even to a probable degree. As to syphilis, the following is the only one which even *primâ facie* looks as if it had relation to that taint. A medical practitioner (No. 61), affected with wide-spread muscular atrophy of the upper part of the body,

subjected himself to the internal and external use of the thermal, sulphuretted, and strongly iodurettted waters of Allevard. This treatment brought out an abundant syphilitic eruption, after which, the disease ceased to progress, and by subsequent continued use of iodide of potassium, the muscles began to enlarge, and recover strength; finally, by a further use of the same baths, which brought out a new eruption—no longer, however, syphilitic, but simply composed of miliary vesicles—considerable improvement took place. The only significant part of this history, in which certainly, syphilis and wasting palsy were concurrent, is the extrusion of the venereal taint by antisyphilitic remedies, simultaneously with the arrest of the atrophy and the setting-in of recovery. This coincidence loses much of its import, however, when it is remembered that mineral baths have, in several other instances, when no syphilitic taint existed, proved of equal service. Particular enquiries were instituted in this direction, in nearly all the more fully detailed cases, and it clearly comes out that syphilis has no hand in the production of wasting palsy. In the vast majority of instances, it had not been contracted at any time; and, in those who had experienced its presence, it had either quickly disappeared without affecting the system, or the time of its sojourn there had passed by, long before this strange malady had fastened on the frame.

There remain, then, for consideration, three main causes,—*inherited proclivity*, *undue muscular exertion*, and *exposure to cold*. The first must be regarded primarily as a predisposing peculiarity; but, as is usual with hereditary predispositions, when excessively potent, it may alone determine the occurrence of the complaint. It has been already pointed out how it adheres, almost exclusively, to the male sex, passing over, or rather through the female, to the next in descent in the male line; how also, the atrophy it engenders is, in five-sixths of the cases, spread over the entire muscular system, and not confined to one or two regions; and, lastly, how that its pernicious presence is revealed in the notably earlier age at which the disease is declared.

Excessive muscular action was pointed out first by Darwall as a likely cause of wasting palsy; and his opinion has received the support of Aran and Wachsmuth. Darwall asks—"Are we to regard the facts of several of these persons having been accustomed to carry heavy weights as merely accidental, or is there any connection between the exertion thus required and the after loss of power, as cause and effect?" And he proceeds to answer, that "the impression on his mind" is, "that the straining necessary in raising and carrying heavy weights" injured the nerves; and that the muscles which they supplied being deprived of nervous force, wasted from disuse and malnutrition.

Aran directs attention to the additional fact, that the particular muscles which are necessarily in long continued contraction in persons following certain mechanical trades—such as masons, milliners, shoemakers—are those which are first invaded and most deeply involved. He goes on to say:—"The subject of the first observation (No. 15), who was a mason's labourer, and carried a hod on his shoulders, and the mason (No. 17), who chiselled the stone, both continually used the muscles of their shoulders in working, and they presented an atrophy of the muscles of the shoulders and of the adjacent parts of the trunk. So likewise, No. 16 who (after having been engaged in turning the wheel of a mechanical press for 20 hours a day) had been employed in glazing paper and was accustomed to carry in his hands heavy sheets of zinc, the milliner (No. 19) who worked assiduously with her needle, and the lapidary (No. 22) who held the right hand in continuous contraction, * * were first attacked in the muscles of the hand." In the same manner he saw a correspondence between the muscles most heavily taxed and the starting point of the atrophy in the ploughman (No. 18), and in the shoemaker, (No. 21); but avows that in the woman (No. 20), *sans profession*, and in the three cases of *general* atrophy which he recorded, it was impossible to assign an appreciable cause to the early wasting of the muscles of the thumb; and he concludes with these judicious observa-

tions:—"Sustained and excessive labour, prolonged contraction of certain muscles, are therefore circumstances which may play a great part in the production of progressive atrophy; but it is impossible to see in them any thing more than occasional causes. There is an immense number of persons pursuing the same occupations as these patients, and with the same ardour, in whom we observe nothing similar; but if there be a predisposition * * * those members which are the most fatigued will certainly be the first assailed." How do these deductions stand in the face of a more extended acquaintance with the disease? On the whole, they are affirmed. We have seen that women of the better class, to whom muscular exertion is almost unknown, enjoy an extraordinary immunity; and that the *age* at which the disease prevails points to the same conclusion, for it is during the *working day* of a man's life that he is most liable to wasting palsy. In childhood and old age, when the muscular system has not yet reached, or has passed beyond, the full stress of its activity, we hear little of it. Again, more than one-third of the cases in which a cause is assigned are attributed to excessive work; and, if we compare the two hands, the right, the hardest worked, is found to be the original point of seizure 20 times, but the left only 7 times—a disproportion quite inexplicable unless by the greater and more constant use of the right hand, especially by handicrafts-

men, who, as we have seen, are specially obnoxious to wasting palsy.

But though the rule be well established, that active muscular exertion is one of the most important conditions of origin of the disease, it is by no means a necessary one, and exceptions frequently occur, as in the officer's widow, mentioned by Cruveilhier; in the young gentlemen spoken of by Bell and Abercrombie; and many others, where the closest scrutiny failed to elicit any proof of undue muscular fatigue. Aran's second deduction also—that when wasting palsy is about to invade the system, the muscles most strained will be the first attacked—although supported in the main, is not to be understood too literally; for persons practising the same handicraft are not always seized in the same muscles, as, indeed, Aran himself pointed out in more than one example; but the table shows that for the most part, workmen who had borne heavily on the muscular resources of their upper extremities, were first seized either in the shoulders or the hands.

It is not difficult to understand why undue toil should affect the nutrition of the muscles. An overstraining of the bodily powers, whatever class of them be regarded, invariably results in a prostration of their activity. Up to a certain point, increased exercise of a muscle quickens its nutrition and enlarges its volume, as we witness in the oft-quoted blacksmith's arm, or in the jockey's

crural adductors; but if exercise be carried to fatigue, and proper rest be not accorded to the wearied muscles, their force declines, and they become an easy prey to disease.

In 16 cases we are told that the atrophy arose from *cold*. In an engineer, mentioned by Richter, the hand and forearm wasted after plunging the perspiring member into snow; the showman, Lecompte, traced the origin of his complaint to a night passed in the open air on a muddy pavement; and a similar night similarly passed, about twelvemonths after, gave a new impulse to the morbid action. Wearing damp apparel, rapid cooling of the perspiring surface, exposure to storms of rain and cold winds—all these have been distinctly shown to be effective exciting causes of wasting palsy.

Cases arising from cold, are subject to neuralgia and rheumatic pains in the affected parts, either at the onset of the atrophy and ceasing when this has fairly set-in, or continuing throughout its progress, and imparting a special character to the morbid history. I find that in this class of cases (16 in number) pain was present 7 times, and absent 9 times; whereas, in the entire series, pain was present only in a quarter of the number. Wachsmuth separates these from the rest, under the title of "rheumatic form of muscular atrophy." Their invasion is often somewhat sudden, and they are accompanied by cramps and twitches; but these peculiarities by no means warrant their

separation into a distinct division; for, some cases that set in with the so called rheumatic complexion, subside soon after into the ordinary form (cases 13 and 14). In one of Aran's observations, on the other hand, (No. 15) the pains, deep-seated and violent, did not break forth for a twelvemonth after the commencement of the atrophy. Neuralgia and muscular rheumatism are, in reality, superadded symptoms or complications, and should not be suffered to obscure the comprehension of the disease in its simplicity.

In conclusion, it may be mentioned that when cold is the source of the atrophy, it is much more apt to extend to the muscles of the trunk than when it arises from overwork. Of the 25 cases attributed to overwork 18 were partial, and only 7 general; whereas, of the 16 charged to the agency of cold, 6 were local and 10 general. It is a much more serious matter, therefore, for the atrophy to be tracked back to cold, or a wetting, than to over fatigue of the muscles. As might have been expected, in more than one instance the disease is alleged to have risen from *cold combined with fatigue.*

CHAPTER VI.

POST-MORTEM APPEARANCES.

Hitherto all the instances of Cruveilhier's atrophy examined after death, have been of a general type; either cases in which the disease had run on to a fatal issue from its own intensity by compromising the respiratory muscles, or cases in which some intercurrent disorder, as smallpox or measles, had anticipated the result which could not have been long postponed, if the original malady had been allowed to pursue its course undisturbed. The essential mischief has always been looked-for, and found, in the nervo-muscular system of animal life. Other organs have been found occasionally diseased. In 3 cases, there was tubercular deposit in the lungs; in another, the kidneys were degenerated; and, in nearly all, the disturbance immediately preceding death had left its morbid footprints. It has usually been some disorder of the respiratory apparatus, bronchitis or broncho-pneumonia, that has proved the direct cause of dissolution; and the lungs, either by their reddened bronchial mucous membrane, or by hepatisation of their proper substance, have borne

witness to the nature of the closing struggle. But these lesions have had no share in the production of the muscular atrophy; they are independent intercurrent maladies, which, except the last-mentioned, would, in all probability, have assailed the system equally, if there had been no affection of the muscles at all. Even tubercle can claim to be regarded in no special relation to wasting palsy; it is present only in an insignificant minority of cases; and, if the length of time during which this disease may linger, be taken into consideration, is it any wonder that a taint so prevalent as the tuberculous, should now and then be found conjoined with it?

The heart, the liver and other chylo-poietic viscera, the kidneys and spleen, do not partake of the morbid movement. In only one instance, was there commencing fatty degeneration of the heart and liver; and that was in a person near fifty. The examination of the central organ of the circulation, has some interest in the present inquiry, both from the striated character of its fibres, and the special liability of its tissue to fatty degeneration; and, it is with astonishment, that we find that repeated and most minute search only proved its perfect immunity from the anatomical degradation which had befallen the voluntary muscles.

Turning now to the nerves and muscles, we at once perceive that only those of the life of relation exhibit morbid changes; and, in the next

place, that while the lesion of the muscles is constant and nearly identical in all the cases, that of the nervous system is most uncertain in its occurrence, and most dissimilar in its nature.

Let us examine, first, the muscles. To the naked eye they correspond to what clinical observation had already led us to anticipate. They are *wasted* away; some only in a slight degree, others more profoundly, while a few are reduced to thin layers, or are even altogether annihilated, their place being only known by comparing the origins and insertions of certain fibro-cellular or aponeurotic-looking vestiges, which are the scanty remnants of the once contractile bellies of the muscles. Between these extremes may be seen all the intermediate grades of degeneration.

The muscles are also changed in *colour;* they have lost the deep red hue of health, and are faded to a pale red, like the flesh of a frog or fish; others are ochrey red, pale yellow, or buff—some being of one colour, some another. Some, again, present yellow streaks of fat between the redder bundles; pointing out, even to the unassisted eye, fatty degeneration.

All the muscles are not changed in exactly the same way. In addition to the differences of colour just noticed—and these may all exist in the same individual—there are considerable variations as to the degree of change into *adipose matter*. Some muscles appear atrophied and changed in colour, but no fat can be seen in

them; while others are streaked with yellowish lines of fat, and some are converted into masses of pure fat, almost totally devoid of muscular fibres. Cruveilhier regards the primary change as one of simple emaciation *(atrophie par macilence)*, and contends that the fatty metamorphosis is an after degeneration, in which the morbid process attains its maximum. But the sequence here claimed cannot be substantiated—at least, it is not universal. Cruveilhier's own descriptions prove this; for the extreme point of change is not always a conversion into fat; this is certainly the more common termination, but sometimes the muscular elements are gradually removed, without fatty substitution, until at length there remains apparently nothing but the connective tissue which bound together the component fasciculi; and, in the latest case published by this author, there was *no fatty change at all;* even the microscope failed to detect any.*

The fact would seem to be, that the muscular fibre dies, and then begins, as a matter of course, to degenerate; but whether the fat, probably always produced in the morbid histolytic process, accumulate within the precincts of the sarcolemma, or be removed as soon as generated, depends on the general condition of the system. Sometimes the economy is prone to collect adi-

* Of Laboulbène's case also, it is said, "the atrophied muscles contained no fat in the bundles."

pose matter, which is deposited in every convenient spot throughout the body, causing obesity. In other conditions (or in individuals of a different tendency) there is no accumulation of fat. That which is taken in excess with the food is not stored away for future need, but is forthwith expelled. Some such difference of constitution may not improbably give a direction to the regressive changes accompanying wasting palsy. When the tendency is towards obesity, and the elimination of fatty matter proves inordinately difficult, the fat set free by the decaying muscular fibrillæ, remains on the spot permanently, or is so slowly removed, that not until a remote period is the muscle reduced to a mere filamentous vestige; when the contrary tendency prevails, the fat is eliminated as fast as it is produced, and the microscope reveals only albuminous granules, or these mixed with merely a few oily particles. But, although this reasoning may explain the varying intensity of the fatty metamorphosis in different individuals, it is necessary to assume the operation of an adjuvant local cause, to account for the inequality of the transformation observed in the muscles of the same patient.

When there is much fatty change, the characteristic *wasting* is far less conspicuous than when the degeneration is simple; and, in several instances, we are told that the muscles had almost preserved their original volume, having been

transformed, as it were, into corresponding masses of adipose substance. So far as I am aware, this has *only* been observed in the muscles of the lower limbs.

Most remarkable is the mutual independence of the muscles. What was foreshadowed in life in the unequally attenuated condition of the muscular masses, is declared in the dead-house with ten-fold distinctness. Scarcely any two are affected in the same degree—side by side with a soft, pale-red, almost filamentous looking remnant, may be found a muscle of dark red hue and considerable bulk; one muscle may be like a mass of pure fat, a neighbour may only show commencing change. But this is not all; not only are the different muscles of the same neighbourhood unequally affected, but the different parts, and even the separate fasciculi of the same muscle, present diverse degrees of degeneration. One-half or one-third of a muscle may be deeply implicated, while the remaining portion is almost or quite sound; again, healthy red muscular bundles may be found lying in the midst of others, partly or wholly changed into fat.

Dr. Meryon was the first to describe and figure the degenerated muscles as they appear under the microscope; and his description has been endorsed, in almost every particular, by later observers. "The striped elementary primitive fibres were found to be completely destroyed, the sarcous element being diffused, and, in many

places, converted into oil globules and granular matter; whilst the sarcolemma or tunic of the elementary fibre was broken down and destroyed." * Dr. Galliet, who examined the muscles of Lecompte, goes more into detail. "In those parts of the muscle (he examined the deltoid) which had retained a roseate hue, the primitive fibres have preserved their striæ tolerably distinct; but the colour is paler than natural, and between the striæ are seen fine molecules, some grey, others brilliant, rounded and more voluminous (from 0.005 to 0.008 of a millimetre in diameter), calling to mind, by their brightness, molecules of fat. In the completely decolourised parts, those which to the naked eye appear of a straw tint, there can still be recognised long cylinders representing the primitive fibres. The sarcolemma which envelopes each primitive bundle is preserved, but the contained substance has lost its striated character; it is replaced by a uniform granular mass, presenting numberless little grey points (molecular granules) which, by their diminutive volume, elude microscopic research. Other shining granules of fatty nature, are scattered about in considerable numbers, in the molecular mass. In a few points of the preparation, the cylinders still exhibit a striated appearance; but this can only be perceived after a most attentive search, for they are almost completely

* Meryon. Med. Chir. Trans. vol. xxxv. p. 76.

obscured by the granular mass. The alteration does not stop at these granular cylinders, which still retain the form and size of the primitive fibre which they replace. These cylinders are seen to diminish in volume, and to become narrower. The largest have a diameter of 0.02 to 0.03 of a millimetre, which is about the normal diameter of the primitive muscular fibre. The smallest granular cylinders, on the contrary, measure only from 0.008 to 0.009 of a millim. In these latter are seen even interruptions more or less numerous; and their enveloping membrane (sarcolemma) has disappeared, and the muscular substance (of the fibrillæ) seems to lie naked in the cellular tissue. Thus, the primitive bundles of the muscle have vanished, more or less completely, and in the parts most deeply altered, no trace of them can be recognised. The striated substance does not disappear all at once—it passes first into the state of granulo-fatty cylinders; which, at a later period, are completely absorbed, so that there remains of the muscular mass only a few granulo-fatty cylinders, and the cellular skeleton of the muscle."*

In the last case related by Cruveilhier, the wasted flesh had a pale red hue, and resembled the muscular fibre of fishes; it was very easily torn and under the microscope showed no trace of fat, while the fibres seemed to be confounded and mixed up with one another, and distorted.

* Archiv. Gén., 1853, p. 584, 1re vol., 5me serie.

The accurate and minute examinations of Oppenheimer, Virchow, Schneevoogt, Valentiner, and Laboulbène of the degenerated muscles in the fatal cases which fell under their observation, coincide almost textually with the account of Galliet. There would seem to be, in fine, a degeneration of the muscular fibrillæ; these gradually give place to a granular amorphous substance, which, in its turn, gives way to fat molecules; and, finally, these disappear, and the empty sarcolemma, if it have not previously deliquesced, thins away out of being, and nothing is left but the frame-work of connective tissue which held together the muscular bundles. In some instances, the degeneration would seem entirely granular, and at no time fatty; in others, fat is present in larger proportion, and sometimes it is exceedingly abundant; so that Meryon's designation, "fatty and granular degeneration," is the correct one, and Duchenne must give up the nomenclature *(atrophie musculaire graisseuse progressive)* for which he contends so earnestly.*

We have found in the muscular system enough to account for the salient morbid appearances which mark the disease at the bed side. The emaciation and loss of power are the direct consequences of the muscular degeneration — the muscle being gone, its place is empty, and its functions are no longer performed. But the enquiry is not complete until the condition of the

* Loc. cit. p. 583.

nervous system is ascertained—that system which is so intimately interwoven with the contractile tissue, that physiologists yet dispute over the precise allocation of their conjoint functions.

Three portions of the nervous system have engaged the attention of observers—*the central organs*, *the spinal roots*, and *the peripheral distribution*. In two instances, we learn the state of the *sympathetic* in the neck. Altogether, 15 cases were examined *post mortem;* but all were not searched with equal care and minuteness, so that the negative evidence of some of them is only of moderate value.

The brain—cerebrum, cerebellum, crura and pons Varolii—was in every case free from disease.* The medulla oblongata likewise was always healthy; but the spinal marrow, the anterior roots of the spinal nerves, and the peripheral distribution of the same, were far from presenting this uniformly uninjured condition. For the study of these, we have but 13 cases; for Drs. Chambers and Beraud do not advert to the nervous system in the subjects of their observations (Nos. 92 and 99).

In 9 out of these 13 autopsies the *spinal cord* was sound, but in the remaining 4 there was a departure from the normal state. Of the former, two only are expressly said to have been examined

* Schneevoogt found the septum lucidum softened in his case; but this is clearly attributable to putrefaction, which was already far advanced.

microscopically, (those of Oppenheimer and Meryon). Cruveilhier says of the spinal cords in his cases, that they were natural in colour, size, and consistence; and of one of them he adds, that he examined the anterior and posterior columns *with peculiar care;* but there is no mention made of the appearance on section, nor under the microscope, though it is scarcely credible that these means were not employed, considering the exhaustive nature of the enquiry into the rest of the nervous system.

We may be quite certain that there was no *softening* in any of the cords declared healthy; and, in Meryon and Oppenheimer's cases it is positively stated, that the microscope revealed nothing abnormal, and especially that there were no exudation corpuscles.

Of the 4 instances in which the cord was diseased, there was softening to be detected by external manipulation, in three; and, in the fourth (that of Virchow) the morbid change—which was other than softening — struck the naked eye on section. And although it must be confessed, that a thoroughly reliable examination of the spinal cord is a matter of difficulty, the fact, that in all the examples in which profound microscopic investigation *did* reveal disease, there were unmistakeable evidences of its presence appreciable to the unaided senses, inspires confidence that the spinal cords pronounced healthy were in reality such.

The morbid states found in these four cases, differ so remarkably from each other, that it is difficult to classify them. Taking, first, those in which there was softening (3 in number, viz., Schneevoogt's, Laboulbène's, and Valentiner's cases) we find the diminished consistence to extend in each case over the lower cervical and upper dorsal regions; mounting, in one, as high as the origin of the second cervical pair, and falling, in another, to the level of the 6th dorsal. In one (No. 98) it affected exclusively the central part of the cord; in another (No. 95), it was confined to the white substance; and, in Laboulbène's (No. 97), the remainder of the cord, both above and below the softened portion, was hardened; whereas, in the first two, the remainder of the cord was healthy. Under the microscope, there were found in all of them, granular corpuscles—the inflammation or exudation corpuscles of Gluge—in both the white and the grey substances. Fat globules were found abundantly in Laboulbène's and Schneevoogt's cases, but no mention is made of them in Valentiner's. The first-named observer also found a very large quantity of amorphous granular matter, the smallest particles of which exhibited lively molecular movements; in addition, the nervous tubules, which, in the indurated part of the cord, had double contours, few varicosities, and viscid homogeneous contents, were, in the softened portion, fewer in number, more varicose,

and filled with granular contents. The ganglionic cells of the grey matter were still present.

In Virchow's patient, there was no softening; but, on cross section, there was observed a notable departure from the normal state. It was confined to the posterior columns, and increased in intensity from the upper part downward to the lumbar intumescence. The alteration consisted in the deposition of a light grey somewhat transparent matter, close to the posterior median fissure, displacing the white substance. Under the microscope it was ascertained that only the posterior columns were affected, and that even the posterior grey horn was not touched. In the discoloured part, there was extensive disappearance of nerve tubules; these had been reduced to a small number, and between them lay a soft nucleated substance, and numberless scattered corpora amylacea; also, on closer examination, there appeared many granular oval nuclei, in some instances evidently enclosed in an oval cell-membrane. There was, finally, no abnormal presence of fat.

The roots of the spinal nerves assume unusual importance and interest in the history of wasting palsy, from the remarkable observations of Cruveilhier. In his two last-reported cases, the anterior roots, and their tributary rootlets, had suffered a most marked diminution of size, and were changed into grey, lustreless strings, which contained only a few true nerve tubules. The de-

struction was far greater in the cervical than in the dorsal and lumbar regions; indeed, in several rootlets in the cervical region, only the neurilemma was left—all the nervous tubules had disappeared. The posterior roots, on the contrary, were of the usual volume, glistening-white, and contained abundance of full, broad, healthy nerve tubules, contrasting markedly in all these respects, with the anterior. From finding this condition in two cases, Cruveilhier too hastily concluded that he had discovered the essential lesion of the disease. In addition to these two examples, atrophy of the anterior roots has been found in two others (Nos. 97 and 98), in which also the cord was softened. The difference of size was here equally striking, and the microscopic exploration certified the greatness and reality of the change; the nervous tubules had in great part disappeared, only a few remained with their broad outlines and full contents; most of them were mere shadows of their former selves, empty tubes, broken and narrowed in the midst of the filamentous tissue of the neurilemma. The only other support to Cruveilhier's account of the anatomical characters of wasting palsy that I know of, is a reference made by Dr. Reade.* "In the union workhouse," he says, "they have had a post-mortem examination of another case, confirming the pathology of the disease, having found the same morbid changes

* Dublin Quart. Journ. of Med. Science, Nov., 1856, p. 399.

in the roots of the anterior nerves of the spine as Cruveilhier has described."

But this remarkable disproportion between the anterior and posterior roots, has not been found uniformly present. In 5 cases (Nos. 79, 89, 90, 94, and 95), although specially looked for, it was not perceived; on the contrary, the anterior roots possessed their accustomed volume, and manifested no morbid alteration whatever;* their nerve tubules were broad, full, and doubly contoured, just like the posterior. The existence of these five instances is a fact of capital importance in the discussion of the pathology of wasting palsy.

Lastly, let us examine the condition of the *peripheral distribution* of the nerves. The motor nerves, at the junction of the anterior and posterior roots, become so mixed-up and confounded with the sensory, that we are not much surprised to find that the alteration, so prominently conspicuous in the anterior roots, could not be traced into the mixed trunk formed by their union. Cruveilhier confesses that he was unable to detect the least change in the mixed nerve, or in the brachial plexus; but he was more successful in the examination of their exclusively muscular terminal branches, and in the hypoglossal, which is purely a motor nerve. He dissected out the

* I need scarcely state, that the slight varicosity found by Laboulbène in the nerve tubules of the anterior roots, ought not to be ranked as a morbid alteration.

ultimate ramifications of the ulnar nerve, where it supplies the muscles of the hypothenar eminence and the interosseous spaces, and found a marked diminution of its volume, both absolutely and on comparison with the corresponding cutaneous branch, which had preserved its usual calibre. The branches to the wasted muscles appeared to consist almost entirely of the fibrous neurilemma, with very few true nerve tubules. But it was in the tongue especially, that an opportunity was afforded of examining the condition of the motor nerves of the atrophied muscles. The hypoglossal possessed only a third of its usual volume, and when the neurilemma was removed, the proportion of the true nervous matter was reduced to about a sixth, and several of its ultimate branches were completely void of nerve tubules. On the other hand, the gustatory nerve was in the most healthy condition. Such were the alterations discovered by Cruveilhier in the peripheral motor nerves; and, with them accord the results obtained by Virchow and Schneevoogt in the examinations of the same parts. Laboulbène, however, found no change in the peripheral nerves, and Mr. Partridge,* who examined Meryon's 5th case (No. 83), states briefly that the tendons and nerves were unchanged. Besides these six cases, there are none which supply information on this interesting point.

In two examples (Nos. 89 and 97) the *sym-*

* Partridge, Med. Gaz., 1847, p. 944.

pathetic in the neck was searched. Landry states that "nothing abnormal could be discovered. The three cervical ganglia exist. The branches from the middle and lower ones are seen to anastomose with the brachial plexus."* The other instance (Schneevoogt's) was remarkable for the fact, that the destruction of the sympathetic was diagnosticated during life, from the enduring contraction of the pupils. The ganglionic cord in the neck was almost converted into a fatty string; one of the lower ganglia was also fatty, and the same change was observed in the sympathetic in the thorax. This is the sum of our information on the condition of the sympathetic in wasting palsy.

These varying and conflicting results may be summed up as follows: —

The nervous system was examined in 13 cases.

In two of them the spinal centre was sound, but the anterior roots and peripheral distribution of the muscular nerves were extensively diseased.

In three cases, there was inflammatory softening of the cord; accompanied, in two of them, with fatty degeneration and destruction of the anterior roots, and in one of these, of the peripheral muscular branches also. In the third case, the anterior roots and peripheral branches were healthy.

* Gazette Médicale, No. 17, 1853, p. 260.

In one case, there was amyloid degeneration of the cord, confined to the posterior median columns, and a granular degeneration of the peripheral muscular branches, but with entire preservation of the anterior roots.

In two, the nervous centres and anterior roots, were found, on the most rigorous investigation, perfectly sound.

In the remaining four cases, the nervous system was pronounced healthy so far as the examination was conducted.

I have tabulated these results as follows:—

Nos. of the Cases in the Table of Abstracts.	Spinal cord.	Anterior roots.	Peripheral distribution.
71, 72, 87	Healthy.	No mention.	No mention.
73, 74	Healthy.	Atrophied.	Atrophied.
83	Healthy.	No mention.	Healthy.
79, 89, 90	Healthy.	Healthy.	No mention.
94	Amyloid degeneration.	Healthy.	Atrophied.
95	Partly softened; partly indurated.	Healthy.	Healthy.
97	Softened.	Atrophied.	Atrophied.
98	Softened.	Atrophied.	No mention.

CHAPTER VII.

NATURE.

HAVING, in the preceding chapters, gathered together and classified the facts of the clinical history — the symptoms, course, duration, and conditions of origin — and of the post-mortem examinations, we are in a position to enter upon the consideration of the nature and being of this complaint. This is the culminating point of the enquiry, and in so far as we are mere natural historians, its termination; but to us as practitioners of medicine, the three most interesting and important questions concerning it have yet to be raised. These are the differential diagnosis, the special prognosis and the treatment; and to their exposition the concluding chapters will be devoted.

To solve completely the problem of the nature of wasting palsy is beyond the aim of the present discussion; such an attempt could only issue in profitless speculation, so long as the essential nature of all disease is a matter of hypothesis. We must be content to confine the enquiry to two questions—first, what is the precise seat of the initial lesion? secondly, what is the proximate nature of that lesion?

The chief light for our guidance is obtained from the results of the post-mortem examinations. They show that only the muscles and the nervous system have suffered organic changes, and in one or other of these we must look for the point of departure of the malady.

We may at once dismiss the idea of a disease of the arteries, curtailing the supply of arterial blood and inducing atrophy by starving the tissues. There is no evidence in the thirteen autopsies of any morbid alteration in the conduits of the circulation* capable of bringing about such a result; and the healthy condition of the skin and subcutaneous cellular tissue, which receive their supply of blood through the same channels as the muscles, precludes the supposition. The livid hue of the integument and slight œdema observed in two or three cases are owing to exceptional or secondary conditions. The long compulsory rest which the limb is forced to maintain, impairs the nutrition of all

* In Virchow's case the small arteries ramifying in the wasted muscles showed granular fat in their walls; but no mention is made of any change that might have impeded the circulation through them; on the contrary, some of the degenerated muscles after having lost their inherent red colour, were still red from the blood-containing vessels which traversed them. When a muscle is atrophied, its vessels must of necessity dwindle away and degenerate from loss of function; but this a secondary, not a primary change. Cases of paralysis from Arteritis are described by Rostan aud Stokes. In these there was loss of sensation as well as loss of motion.—See a Lecture by Dr. Stokes in Ryan's *London Surgical and Medical Journal*, p. 195, vol. vi.

its tissues; the vessels lose tone and permit a passive congestion of their ramifications, the watery parts of the blood exude through their distended walls, and œdema of the part is produced. The sluggishness of the circulation is also aggravated by the loss of the impulsion received, in the healthy state, by the returning blood-current from the compression of the veins by the contracting muscles. The same thing is observed, not unfrequently, in hemiplegia: and in limbs that have been long kept immovable for the treatment of fractures, it is the usual result; when the bandages are undone on the completion of the cure, the surgeon is nowise alarmed or astonished to see the limb swell more or less for some days after.

Let us turn to the nervo-muscular system. We have seen in the preceding chapter, that while the muscles are invariably the seat of conspicuous anatomical changes, the nervous system, although in some instances deeply diseased, is in others reported quite healthy. This proposition if it could be affirmed without reserve, would settle the question in favour of the muscles; for it is evident, that if a single case of true wasting palsy could be produced in which the entire nervous system was free from disease, it would necessitate the conclusion that the causal lesion is *not* in the nervous system; inasmuch as no effect can own for its essential cause any condition that may be occasionally absent. But here

lies the difficulty; can we be sure that the cases in which the nervous system was reported healthy were in reality free from disease? Is there not a lurking suspicion in the mind, that if the examination had been more searching, and the means of exploration more perfect, a hidden unsoundness would have been revealed, and the seeming health pronounced a delusive appearance? Such a suspicion has arisen, and it must be owned not unreasonably, seeing the extreme delicacy and difficulty of the enquiry, and the darkness which yet veils the minute anatomy of the nervous centres even in the healthy state. Such being the case, we cannot narrow the question to one of simple observation; the facts must go to the jury, and their interpretation canvassed and their worth estimated, according to the general evidence at hand.

Cruveilhier, Schneevoogt, and Eisenmann contend strongly that the disease is of nervous origin, and that the muscles are only secondarily affected. In his last paper, Cruveilhier announces his belief "that this gradual muscular paralysis is the consequence of progressive atrophy of the anterior roots of the spinal nerves," and as the anterior columns with which these are apparently connected are quite sound, he infers that their real origin is in the central grey matter, and *here*, he says, we must seek the cause of the atrophy of the motor roots and of the wasting of the muscles. Dr. Eisenmann believes in a dynamical alteration

of the spinal cord, causing paralysis and wasting of the muscles, and bringing about after it has lasted some time hyperæmia of the cord, and, finally, inflammatory softening of it. He compares this succession of events to what takes place in tetanus; in recent cases of which no change is found in the cord; but if death be delayed beyond the fifth day, congestion and softening of that centre are discovered (sic).

Schneevoogt sees two elements in the complaint; one is a disturbance in the anterior columns of the cord, and the other a degeneration of the sympathetic nerves; and on this latter, he conceives, depends immediately the destruction of the muscles.

All these opinions, and any opinion that traces the phenomena in order of causation downward from the nervous system — whether commencing in the grey matter of the cord, in the anterior roots, or in the sympathetic nerves — to the muscles, must, I think, in the united light of the cases here recorded be given up. Both the post-mortem examinations and the clinical history point the same way — that no cause acting downward can possibly produce the results observed.

In the first place, there are at least two cases in which a very accurate search found no change in any part of the nervous system. Secondly, when the nervous system was diseased, the alterations discovered were so different in their seat and nature, that it is incredible, that they should

be the cause of a muscular change so constant and uniform. Can we believe the anatomical changes discovered in the *spinal cord* to be the primary lesions, when those changes were, in one case, a softening of the central grey substance, in another of the cortical white substance, in a third, softening of one part conjoined with hardening of another part; while in a fourth, there was no softening at all, but an amyloid degeneration confined to the posterior median columns? Further, in Schneevoogt's case, the anterior roots were atrophied and the spinal cord softened *between the fifth cervical and second dorsal* nerves; but it is remarkable, that *above the fifth cervical* nerves the cord was perfectly sound, whereas the anterior roots coming off from this part were atrophied (like those arising from the softened part) to the last degree. Could anything show more conclusively that the atrophy of the anterior roots did not depend on any disease of the cord?

Again, the healthy condition of the *anterior roots* in 5 out of 9 cases, in which they were microscopically examined, proves beyond appeal that the seat of the disease is not in them.

Finally, it is impossible to admit that the *nerves in their traject* to the muscles should be the starting point, inasmuch as the topography of the atrophy is out of all correspondence with the distribution of the nervous trunks. Microscopic exploration of the peripheral nerves disclosed, it is true, atrophic degeneration in 4

cases; in two others, however, they were healthy; and it will not be difficult to explain the diseased state of the former, without regarding it as a primary phenomenon (vide infra). But let us proceed to enquire:—What evidence does the clinical history yield?

In studying the nature of a disease it is very important to avoid confounding the incidental occurrences with the essential phenomena, and to separate the occasional from the constant. As the chemist subjects the substance he is going to analyse, to numerous washings and filterings, to separate extraneous ingredients and secure a pure article as the basis of his operations, so the pathologist should question himself:—Can this or that symptom or anatomical change be ignored and the identity of the disease be preserved? If the answer be affirmative, then that symptom or morbid change does not form part of a true conception of the malady. It is by searching out and studying *simple uncomplicated cases,* and marking what ordinary symptoms may be absent in this and that case, that he eliminates the sources of error, and gradually spells his way to a knowledge of a group of phenomena, which are exclusively essential to the being of the disease.

What are then, the essential phenomena of wasting palsy? Is pain? It may be absent altogether. Muscular vibrations? They also may be wanting, or, if at any time present, may pass away; the same may be said of cramps and

twitches. Morbid sensations and irregular movements may be wholly wanting, they are mere accessories: the never absent phenomena are the *destruction of the muscular substance and the consequent loss of power*. Observe the cases of Emma Walker and Thomas Rogers. In the former the muscles attaching the scapula and humerus to the trunk had gradually disappeared—the loss of power had declined at the same rate. The upper arms, forearms, hands, trunk, and lower extremities were in a most vigorous condition; the skin and subcutaneous fat over the wasted muscles were quite natural both in colour and thickness. What shadow of evidence is there, in her case, of any disease apart from the muscles? The second case is similar: nothing, absolutely nothing, but a progressive atrophy of the muscles of the hands and forearms is offered to the search of the pathologist. Dr. Reade's patient "exhibited neck, chest, and arms down to the elbows, reduced to the most abject degree of emaciation; * * * the muscles of the forearms and hands displayed the full development of a robust and vigorous man. All the muscles outside the pelvis and those of the inferior extremities were full, strong, and well formed."* There was corresponding loss of power, but no further symptoms whatever.

Cases like Emma Walker and Dr. Reade's, and

* Dublin Quarterly Journal of Medical Science, Nov. 1856, p. 349.

they are not infrequent,* in which the proximal segments of the upper or lower extremities are despoiled of their muscles, while the distal ones are full and strong, are very hard to reconcile with a central theory of causation. I am not cognisant of any injury or disease, undoubtedly located in the spinal cord, which affects the shoulders and upper arms and leaves the hands and forearms untouched. If the central connection of the former is so diseased as to involve the total destruction of the correspondent muscles, is it not certain that the nervous centres of the latter, which are in immediate proximity, and connected by continuity of tissue, would be more or less implicated? Even more striking than these is the case of Mr. B. — one muscle only on the back of the forearm had survived; of the rest there was not a vestige. Is this compatible with a central lesion?

What does *experiment* teach? If the rootlets of origin of one of the nerves of the extremities be cut, just as they emerge from the cord, there is produced not a palsy of one or two muscles, but a certain weakening of all the muscles of that extremity. Panizza divided the roots of one of the three nerves that go to form the crural plexus of the frog, but the movements of the limb were only slightly affected; and when he divided two, the effect

* There are 13 cases in the table, in which the shoulders and upper arms were *solely* affected; the forearm and hand being untouched as well as every other part.

was not a complete palsy of any one set of muscles, but all the muscles were debilitated in an equal degree. A muscle therefore is not in exclusive communication with a single rootlet or even a single root, but receives fibres from all the roots that go to form the nerves of the extremity; in the same way, probably, it is related not to a limited spot of the spinal cord, but to a considerable tract — a tract corresponding to the origin of its nerves. If this be true, the operation of a lesion confined to a certain spot in the cord, or to a certain nervous rootlet, would be general over the muscles of the whole limb; a result which is quite at variance with the *dissecting* progress of wasting palsy.*

* Dr. Aug. Waller, (Comptes Rendus 1852. tom. xxxiv. pp. 582, 843, where may be found his memoirs presented to the French Academy,) thinks he has proved that the spinal motor nerves have a centre of nutrition in the cord, because when they are cut off from this connection, they forthwith die and degenerate. If this were true, it might be advanced that the initial lesion of wasting palsy is in the nutritive centres of the motor nerves. Against this all the considerations already offered against *any* spinal lesion equally avail; but it seems repugnant to sound physiology to believe that any tissue has a nervous nutritive centre. I would not deny the influence of the nervous system on nutrition; but that it is *absolutely dependent* on it is at variance with every day facts. May not Dr. Waller's results be explained on another supposition? When he cut a motor nerve, the distal portion — that connected with the muscle — died; and why? Because it no longer received impulses from above; its function had ceased, and it died for the same reason that the kidney dies when the ureter is tied, or an artery dies up to the next collateral branch, when a ligature is thrown round it.

The post-mortem examinations, the clinical facts and the deductions of physiology, then, all go to establish the position claimed at the outset — that no lesion acting downwards from the nervous system to the muscles can produce the result observed.

As to the speculations of Schneevoogt, that the muscular atrophy depends on a degeneration of the sympathetic, it rests on a single fact; and that fact loses its significance when it is remembered, that the spinal cord and its derived nerves were likewise atrophied in that instance. For the sympathetic is so closely united to the spinal system, that any destruction of the latter would unfailingly re-act on the former.

To the muscles themselves, therefore, we must look for the primordial phenomena of wasting palsy. This opinion is held by Duchenne, Aran, Oppenheimer, Wachsmuth, and Dr. Meryon.

It now only remains, to glance at the nature of the morbid action provoked in the muscle, and then to attempt to explain the so frequent occurrence of nervous symptoms, and of organic changes in the nervous system.

Of the nature of the blight that withers the muscles, I am only able to say, with Dr. Meryon, that it is a fatty and granular degeneration of the muscular fibre similar, (often at least) in its anatomical bearings, to what is observed in fatty heart, or in muscles which have degenerated from section of their nerves. There would seem

to be an error of nutrition in the muscular fibre; not dependent, as Dr. Meryon thought, on a general depression of the nutritive functions — these being almost always in their highest perfection—but brought about under the influence of a peculiar constitutional predisposition or diathesis, by the aid of one or more of the special exciting causes mentioned in Chap. V.

That the entirety of the disease is not comprehended in its local manifestations, and that a constitutional predisposition lies behind these, is made evident by the transmissibility of the disease from parent to offspring, and also by the total inadequacy of the exciting causes, acting alone, to produce the series of events which characterise wasting palsy (see p. 143). An additional proof is seen in the bilateral symmetry of its march. This symmetry, although by no means constant or always exact, is quite as remarkable as anything presented by pulmonary tubercle, articular rheumatism, or syphilitic or other eruptions of the skin.*

In considering the rational explication of the nervous phenomena, it must be remembered that the peripheral expansions of the motor nerves are

* Dr. Reade grounds his belief in the spinal origin of wasting palsy on this very fact of its bilateral symmetry; but the absence of *exact* correspondence in nearly all the cases, and its *total absence* in others, render this opinion untenable even on its own ground, leaving out of consideration the objections already advanced to the spinal theory.

constituents of muscle, which it is impossible to ignore; and if we knew something, with certainty, from actual observation, of the connection of the ultimate nerve tubules with the muscular fibre, the question would have to be raised, whether the spring of the morbid stream was in the muscular fibre itself, or in the nervous expansion which directs its contractility; but, inasmuch as we are in ignorance, except that the tubule divides into still finer filaments before it impinges on the primitive fibre, we may be spared the discussion. However, this much may be inferred with confidence, that there is an organic connection—a continuity of tissue—between the ultimate ramuscles of the nerve tubule and the sarcolemma of the muscular fibre, so that the destruction of the latter involves of necessity the ruin of the former.*

That two systems so intimately connected, both anatomically and functionally, as the nervous and the muscular, should sympathise with, and re-act on, each other in diseased states, is what reflection would teach us to anticipate. Proof that the muscle sympathises when any evil has befallen its nerve lies on every hand. Not only does section of the nerve in its course, and injury at its centre, instantly depress or abolish the functional activity

* This admission may be safely made. All the anatomy of the nervous system, as understood by the aid of modern research, leads to the conclusion that a continuity of tissue is essential to the action of nerve tubule, nerve cell, and ganglionic corpuscle.

of the muscle, but even neuralgia in the nervous trunk — which, in some of its manifestations at least, can scarcely, from its passing duration, its sudden onsets, and equally sudden retrocessions, be regarded as bound to an organic cause — brings on an immediate and profound impairment of the muscular movements. The sympathy downwards is ever present to our observation and reflection; and this, perhaps, is the reason why we have not perceived the more occult, and it must be admitted, far less important, but not the less real, re-action taking place in an opposed direction, from the muscle to its nervous expansion, thence to the trunk of the nerve, and so upward to the nervous centre.

As just stated, the destruction of the muscular fibre involves injury to the extreme branches of the motor nerve, and it is quite consonant with what is observed in other diseased textures, that a morbid movement set up here may creep upward — the unbroken continuity of the nervous thread favouring its advance — until at length it reaches the anterior roots, and even the spinal cord. Dr. Graves is clearly of this opinion. "May not" he asks, "the decay and withering of the nervous tree commence occasionally in the extreme branches?" And in another place he speaks of "creeping paralysis commencing in the peripheral extremities and travelling gradually towards the centres of the nervous system." *

* Dr. Graves' Lectures on Clinical Medicine. Second edition, vol. ii. lecture 33.

But there is another reason why the nervous trunks and the portions of the cord corresponding to the wasted muscles, should be prone to morbid changes. It is notorious that every organ, gland, or tissue, speedily decays when its proper function is arrested, and the nerves are no exception to the rule.* When, therefore, a limb is deprived of motion by muscular atrophy, and reduced to a state of continuous rest, motor impulses cease to be transmitted along its nerves; and these, brought to a state of functional inactivity, decline in their nutrition, and begin to waste throughout their length even to their source in the substance of the cord. If all goes on well — as is usually the case — the nervous tracts corresponding to the withered muscles are gradually absorbed,

* Perhaps it has been too much the custom to look at the function of the nervous tubule as one of mere conduction, in the sense that the speaking tube in a warehouse conducts sounds. The nerve tubule is not a passive instrument, but an active agent, and its internunciary operations are carried on by a power of conversion acting along its whole length. If the sciatic nerve of a frog be cut-off from the cord, and the peripheral segment pinched with a pair of forceps, the muscles of the leg spring into active contraction; and why? It is not that the mechanical violence inflicted by the forceps is transmitted as such to the muscles; it is in reality a case of conversion. The mechanical force is changed by the special functional power of the nerve into the nervous motor impulse, which may be — must be — looked-on as a correlated force; this, once produced, speeds on its way to the muscle, just as if it had been derived from its legitimate sources, and obeys the laws of transmission by which nervous force is known to be guided.—See Müller's Physiology, by Baly, p. 635.

leaving the contiguous parts of the cord unimpaired. But it is not difficult to understand, that if this process — which is altogether secondary and only borders on the morbid — be in any way disturbed, a new action may be set up in the delicate structure of the reflexive centre, and softening, or amyloid or other degeneration arise to complicate the phenomena; an accidental cold, a constitutional bias, or an unusual intensity in the process itself, being sufficient to determine such a contingency.

We may look, therefore, for three orders of phenomena in wasting palsy.

1. *Primary or Direct.* These are, destruction of muscle and consequent loss of power. They are necessarily always present, and, in the simple cases, are the sole factors in the problem.

2. *Secondary or Reversed.* These include atrophy and fatty degeneration of the motor nerves, together with softening or other change in the spinal cord. These are not invariably present. Probably some of the neuralgic pains should find a place here; the nervous filaments ministering to the muscular sense, being involved in the general devastation, may be the source of those severe pains sometimes experienced in the substance of the affected muscles; but I am disposed to believe that most of the abnormal sensations and movements are the expression of the next order.

3. *Tertiary or Reflected.* These may be

produced through irradiation of impressions conveyed to the spinal centre by the nerves of the special muscular sense; or they may be the direct consequences of the secondary organic changes just described as going on in the part of the cord where the nerves of the decaying muscles originate, whereby the contiguous sound portions are irritated. In both these ways abnormal movements and feelings are provoked — undue sensitiveness to cold, neuralgic pains along the nervous trunks or in the joints, twitches, cramps, contractions, and fibrillary tremors.

This explanation of the pain and convulsive movements accords perfectly with what occurs in some other morbid states. Convulsions arise in children from an irritation in the primæ viæ — epilepsy has not unfrequently an eccentric cause — caries of a single tooth often inflicts the most terrible neuralgia, extending to all the branches of the trifacial nerve — irritation in the hip in morbus coxarius, is the real source of the pain at the knee. Why, therefore, may not a decaying muscle, as well as a decaying tooth, originate eccentric nervous phenomena? To my mind there is not the least difficulty in understanding that the excruciating pain at the outset of wasting palsy, or afterwards in its course, is the expression of an irritated state of the cord set up by mischief going on in the muscles. That pain or abnormal movement is not *always* present, is not more inexplicable than that irritation of the bowels or

a carious tooth does not *always* produce convulsions or tic.

As a corollary to the foregoing demonstration—as I hope I may call it—stands the explanation of what has been a stumbling-block to Dr. Eisenmann, namely, the want of proportion sometimes observed between the wasting of the muscles and the loss of power, and the somewhat sudden advent of the latter in a few cases. The irritation of the cord, superinduced as above described, re-acts in an untoward manner on the healthy muscles, or on the uninjured fasciculi of the diseased ones. When there is neuralgic pain the lowered muscular power occurs only as its usual accompaniment; and when there is none, the debilitating effect is still produced in the same way, and finds its parallel in the ephemeral palsies produced similarly by intestinal parasites or hysteria: they are all *derived* phenomena, either reflected *viâ* the spinal cord from the decaying muscles, or to be referred to the direct effect of the secondary organic changes taking place in that centre.

CHAPTER VIII.

DIAGNOSIS.

The outward appearance of a fully developed example of Cruveilhier's paralysis is so striking, that the disease is recognised almost at first sight with certainty; but in its early stages, before the atrophy has yet reached a high degree, it is much more difficult to distinguish.

The *partial* form is liable to be confounded with paralysis from injury to a motor nerve, lead palsy, and that palsy which sometimes follows the endemic colic of hot countries.* In all these, there is a marked atrophy of the muscles; and the affection may be confined to a narrow region, around which are healthy muscles offering a strong contrast to the decayed ones.

Atrophy resulting from *section of, or pressure on, a nerve,* is distinguished by the exact limitation of the wasting to the parts supplied by that nerve; also, if the nerve be a mixed one, there is, or was, an accompanying loss of sensation.

Any cause necessitating *a state of continued rest* will bring about wasting of a muscle. A few weeks ago, I examined the deep interosseous muscles in the stump of an amputated leg, which

* See note p. 189.

had to be removed in consequence of its conical shape. I found them profoundly changed, and, under the microscope, they presented all the appearances of fatty and granular degeneration, as described by Drs. Meryon and Galliet in wasting palsy.

My colleague, Dr. Wilkinson, presented to our weekly consultation, some months back, a strong muscular young man of eighteen, who could not move his left shoulder. At first sight, I was sure there stood before me an instance of the disease in question. The muscles uniting the shoulder-blade to the arm were greatly wasted; above and below the spine of the scapula were deep hollows; whereas the right shoulder was plump, and its bony prominences scarcely to be felt. On examining the joint, however, it turned out that complete bony anchylosis had taken place at the scapulo-humeral articulation; and the short muscles of the scapular fossæ had been, in consequence, for many months condemned to unbroken inaction. The cause of the atrophy was now explained, and it was confined strictly to the muscles passing from the scapula to the head of the humerus.

The *real* difficulties of the differential diagnosis lie in the separation of lead paralysis, and that palsy which sometimes follows the so-called vegetable colic (endemic colic of hot countries.)

Lead palsy has strong points of resemblance to Cruveilhier's atrophy. It falls on limited groups

of muscles, leaving the adjacent ones untouched; it generally does not interfere with sensation in the palsied part; the affected muscles speedily waste and the atrophy may reach the extreme degree seen in Cruveilhier's disease. The parts attacked are similar; both prefer the upper extremities; both also have a partial and a general form. It must be admitted that they are closely allied; and, in some old cases, when the atrophy has compassed the entire destruction of the muscles, and marks of saturnine intoxication no longer persist, I know of no means, except the study of the previous history, by which the two affections may be distinguished. In the case supposed, however, it must be remembered that we are not dealing with an existing disease, but with the memorials of one long passed away.

When in actual progress, the diagnosis, notwithstanding these many marks of resemblance, is not difficult. In lead palsy there is a comparatively sudden invasion; and when this is not the case the precursory phenomena, which are quite distinctive, never altogether fail.* These include colic, anæsthesia, tremblings of the limb, neuralgia, pallor, general dyscrasia, undue mobility of the emotions, especially the depressing ones, in fine, all those anomalous symptoms and conditions embraced by the term lead cachexy.

*Falck. "Die klinisch wichtigen Intoxicationen"—"Blei-Lähmungen" in Virchow's "Handbuch der speciellen Pathologie und Therapie." Band. ii. Abtheil. ii. p. 200.

The facts of the previous history and the violet line on the gums complete the proof of saturnine intoxication. But suppose a painter or a plumber, who has had colic and presents the blue line on the gums,* be the subject of a loss of power with atrophy, how may it be ascertained, whether it be an example of lead paralysis or of Cruveilhier's atrophy? The case, it must be allowed, is

* The blue line on the gums in lead impregnation, although frequently a very valuable sign, is sometimes both positively and negatively useless. I have seen a woman labouring under well-marked and altogether undoubted general lead palsy with perfectly rosy gums and no trace of a violet line. The poisoning had taken place nearly a twelvemonth previously, and she had been, in the mean time, entirely removed from the contamination; so that, in all probability, the metal had been completely eliminated; and the palsy, which was rapidly disappearing, was but the effect persisting after the removal of its cause. On the other hand, teeth encrusted with canker, and even teeth simply decayed, are frequently surrounded by a distinct livid blue line undistinguishable from that produced by lead. One morning I examined the teeth of 56 of my out-patients with a view of enquiring into this point. I took them as they came, only excluding children, who have always clear pink gums, and obtained the following results:—35 had no blue line at all; 14 had an imperfect one; and 7 presented a characteristic blue margin around the necks of the teeth. I always found the teeth decayed or clogged with tartar when the gums were thus discoloured. Of course, I took care to ascertain, as far as enquiries could go, that there was no possibility of lead impregnation.

My friend and colleague Dr. Browne has called my attention to the fact that Nitrate of Silver and Trisnitrate of Bismuth may give rise to the production of a blue line on the gums after internal administration.—See Wood's "Therapeutics," Art. Nitrate of Silver; and Dr. Brinton, "On the Pathology, &c., of Ulcer of the Stomach," p. 117.

embarrassing, but the diagnosis is not the less capable of being unravelled. Lead palsy invades the muscles in a certain order. The extensors are its special prey; and unless the affection has become general the loss of power is confined to them, the flexors escaping altogether;* so that the implication of the latter, apart from the contingency just mentioned, precludes at once the idea of lead palsy. Secondly, the loss of power is out of all proportion to the atrophy in recent cases; for the wasting is very distinctly *posterior* to the failure of strength, and very frequently we see a limb altogether disabled by lead while the mass of the muscles is still undiminished. The onset too, although often comparatively gradual, has nothing like the slow event of Cruveilhier's palsy. In a day or two, a week, or a fortnight at the most, lead paralysis has reached its height, and the muscles it affects are reduced to complete immobility; any movement the part may afterwards possess being really due to neighbouring muscles, which take up in an im-

* Dana. "Lead Diseases," p. 200. Duchenne gives the following, as the invariable order in which the muscles of the forearm are attacked in partial lead palsy; common extensors of the fingers, the proper extensors of the index and little fingers, and the long extensor of the thumb. He also goes so far as to say, that wasting of the ball of the thumb is not produced in these cases by the lead; and that, when, in a few instances, the right thumb in painters has been so affected, it is due, not to lead poisoning but to the mechanical pressure of the brush on the muscles of the ball.—*Archiv. Gén.* p. 15, tom. xxii., 1850.

perfect manner the functions of the defaulters.* Very often an attack of colic is immediately followed by a dropped wrist, or by an aggravation of a pre-existing palsy; very often too, lead palsy is evanescent; it quickly disappears and no atrophy takes place—things that never occur in the contrasted disease. Dr. Duchenne† has brought in aid a test which may be of service in doubtful cases. A muscle palsied by lead loses entirely, or in great part, its electric contractility; whereas, in our complaint, it responds to the electric stimulus in a degree proportionate to its bulk.

The palsy which follows the endemic colic of hot countries, (Devonshire colic, dry colic, vegetable colic, &c., &c.,) seems, in so far as it has been studied, very similar to that produced by lead. It attacks the same parts, and Duchenne found the paralysed muscles insensible to the galvanic current. I am aware that Tanquerel des Planches, and after him all writers in this country, and nearly all elsewhere, consider these cases instances of concealed lead poisoning; and the discovery of that metal in the cider of Devonshire settled the question in the conviction of medical men. There is no doubt but that this explanation is a true one in a great number of instances; but there are some facts so difficult to reconcile to such a theory of production, that it

* Tanquerel des Planches. "Lead Diseases," by S. L. Dana, M.D., p. 199. Lowell, 1848.

† Duchenne. "De l'électrisation localisée," p. 510.

is necessary, for the present at least, to admit the existence of a disease marked by obstinate colic, and frequently by consequent palsy, arising apparently from malarious influences, and independent of metallic intoxication. Too little is known of it, to enable us to say how it may be separated from true wasting palsy, except by the previous history, which discloses antecedent colic, brusque invasion of the debility in the muscles, and evidence that the atrophy followed, not accompanied, the palsy.*

* Those who wish for further information on this subject may consult a paper by Valleix in the "Union Medicale," Nos. 6 and 9, May 1848; and the article on *Colique Nerveuse* in his *Guide du Médicin practicien.* Ed. 3me. tom. iii, p. 60. There is also a very elaborate account of the same disease in the *Archives Générales*, 4me. serie, tom. xxix, p. 129, under the title *Memoire pour servir à l'histoire de la colique nerveuse endémique des pays chauds* by Dr. J. B. Fonssagrive. It is based on 9 cases carefully detailed. Segond has described the same disease as occurring in Cayenne, (Essai sur la nevralgie du grande sympathique, Paris, 1837). He considered it a neuralgia of the sympathetic nerve. But it is not confined to tropical countries. Valleix observed it in Paris; and a very characteristic example, which I shall quote, is related by L. Saurel of Montpellier, occuring in a gardener in the south of France (Rev. Ther. du Midi. Juill. 1854, also Schmidt's Jahrb. 1854, Bd. 84, p. 299). The colic, in this case, came on after a severe wetting and left a weakness in the arms. Three more attacks of colic occurred subsequently, and on each occasion the loss of power in the arms increased. Atrophy of them set in, and, in 18 months, reached a high degree. However, under the use of mineral baths and the internal employment of the mineral waters of Balaruc a rapid amendment took place, and in no long while the man resumed his work. After two years, he took cold again and the loss of power returned, when suddenly

Wasting palsy, when extensively diffused, is liable to be confounded with *general lead poisoning* and with the *general paralysis of the insane* under certain circumstances. With the former, confusion is scarcely possible with ordinary care—

he was seized with a violent colic attended by constipation, and biliary vomiting, as in the previous attacks; both arms were completely deprived of the power of motion, and wasted rapidly. By the continued use of the same remedies, however, and electricity, the volume and strength of the arms were again restored.

I am strongly inclined to believe, that a remarkable case published by Dr. Todd in his admirable clinical lectures on Paralysis and Disease of the Brain, p. 338, Case 65, is an instance of the affection in question. It concerned a tavern waiter from Devonshire, who had never drank the cider of that country; he was affected with complete atrophy of the muscles of the forearms, complicated with gout and albuminuria. He was admitted into hospital in a state of epileptic coma, which soon passed off. This man had been paralysed in the forearms for four or five years, and had suffered several times from severe colic, with vomiting; and these paroxysms were usually followed by an attack of gout in the great toe. While in hospital he had a very severe double seizure of colic and gout. He died. No trace of lead could be detected by careful analysis in the nervous centres, nor in the muscles. Dr. Todd believed the man was poisoned with lead, which, however, he confesses was very difficult to account for, and as a *pis aller* he lays the blame on the pewter pots, which the man was in the habit of polishing with his hands, and points to a blue line on the gums, in confirmation. It may be objected that London pot-boys are not subject to lead poisoning, and that a blue line on the gums, when the teeth are incrusted with tartar—and we are told this man's teeth were bad—is not uncommon, where there is no suspicion of plumbeous infection (see note p. 186); and that, conceding the existence of a malarious colic (this seems to me the most appropriate name for it), the whole case is cleared up without the least difficulty.

the physiognomy of the two diseases is altogether different, and the multiform signs and symptoms of lead intoxication can not be mistaken or overlooked. In wasting palsy on the contrary, the economy, as a whole, shows no indication of a blood poisoning, but exhibits unimpaired soundness of all the nutritive functions, with the sole exception of the muscles. In long standing cases of general lead poisoning, the disease involves not only the muscles, but also the adjacent tissues: the subcutaneous cellular fat disappears, "the skin is whitish, pale, of a livid aspect, often yellow, tawny rough, earthy, dry and shrivelled. The epidermis appears thinner than usual, often falls off in scales." *

General paralysis of the insane very frequently† sets in before the unsoundness of mind is declared; and the unsteady gait and faltering utterance conjoined to a perfectly clear intelligence, might mislead. The doubt, of course, can only be passing, for delusions soon make their appearance; in the mean time the diagnosis turns on the following points. In general palsy of the insane, although the muscles waste and lose power, the limbs suffer uniformly; there is none of the *dissecting* character so remarkable in Cruveilhier's atrophy. Duchenne relies on another sign; namely, the thorough preservation of the

* Dana's Tanquerel p. 202.

† See a series of articles in the "Gaz. des Hop." for July 1846, by Dr. Baillarger.

electric contractility in the former, and its depression in the latter.

In other forms of cerebral palsy, their sudden advent, their range, the implication of the sensory nerves, the absence of emaciation in the affected muscles, the lively reflex activity of the latter, and their ready response to the electric stimulus, prevent the possibility of error.

Much more easy is it to mistake a long standing case of the *Essential paralysis of infancy and childhood* for Cruveilhier's atrophy. A little girl of seven years of age was brought to me a few weeks since, with almost total loss of power, and great emaciation of the lower limbs, which were involved from hip to ankle. She could with difficulty stand alone, and in walking, or rather in the attempt at walking, the limbs seemed to serve merely the purpose of a mechanical support, and the knees, especially the left, were deflected backward with her weight; the ligaments, seemingly, having become elongated or relaxed. The limbs were lifted from the ground, and thrown or swung forward by the power of the pelvic muscles, which were strong; and in this way a feeble and limited progression was possible. Sensation was acute in every part. Altogether, to superficial observation, it seemed not unlike the third case reported by Sir Charles Bell (see page 4), but it lacked the dissecting character — all the muscles appeared to have suffered an equal blight; and enquiry into the previous history, removed any lingering doubt

that might have remained in my mind. A year and a half ago, while the little girl was in perfect health, she was seized with a slight fever, accompanied with a loss of power in the lower limbs, which confined her to bed for three days. At the end of this time she was unable to walk or stand; and the inability has never left her since. At the first, there was also loss of sensation in the legs and thighs, but the feeling gradually returned. The muscles, on the other hand, began to waste, and their bulk has continued diminishing almost to the present time. All this showed unequivocally a central origin in the spinal cord.*

Injury or inflammatory softening of the spinal cord can rarely or never produce any perplexity. The loss of power is attended with some modification or loss of sensation. It is confined to, and evenly distributed over, the parts below the seat of injury or disease. It often involves the sphincters of the bladder and rectum; it has come on suddenly, is attended with rigid spasms; the electric contractility is preserved in the muscles; the occurrence of any of these conditions or circumstances is incompatible with the idea of wasting palsy.

I have never been able to satisfy myself about the nature or identity of the so-called *rheumatic*

* On this subject see an interesting paper by Dr. Henry Kennedy in the "Dublin Quarterly Journal of Medical Science," February 1850; also Rilliet and Barthez "Traité Clinique," Tom. ii. p. 335.

palsies. Those which have been pointed out to to me as such, presented no wasting in the disabled muscles, which sprang into active contraction on the application of galvanism. Their general physiognomy also, resembled in no degree that of Cruveilhier's paralysis.

CHAPTER IX.

PROGNOSIS.

THE prospects of the sufferer from wasting palsy, are always serious; but, fortunately, the danger is more imminent to the usefulness of the limb than to life. The early writers on this disease, Aran, Cruveilhier, and Duchenne in his first essay, prophesied a most gloomy future to its victims. The very name which Aran imposed upon it implied the doom he foresaw. The general disease, in all the early observations, both of Aran and Cruveilhier, withstood every remedial effort, and marched deliberately, but with a terrible certainty, to the final catastrophe. In the partial form, the prognosis was declared equally hopeless in regard to the wasted muscles. Once assailed their fate was sealed, and the most favourable issue hoped for, was the arrest of the creeping death, before its grasp had been laid on *all* the muscles of the stricken member. But in the present day, owing to the extraordinary perseverance and almost heroic faith of Duchenne, coupled with a more comprehensive study of its career, the physician can contemplate

more hopefully the advent of this subtle malady; and, although it must still be ranked among the most intractable in the domain of physic, there is, in many cases, a reasonable hope of recovery, if remedial means be timeously applied.

By a reference to the concluding part of Chapter IV., and especially the table p. 132, it will be seen that the gravity of the prognosis, in so far as it is a question of life or death, depends on the disease confining itself to the extremities and the muscles thereto relating, or extending its operations to the muscles of the trunk. When this latter is the case, the chances of life are small; only a fourth of such cases, which had reached a final issue, having been reported as recoveries or arrests, the remainder having died; nearly all of them in spite of the most active and judicious treatment.

The signals of coming dissolution are impressive, and not difficult to interpret. When the destruction of the facial muscles has effaced the expression from the countenance, and the tongue lies motionless in the mouth; when the voice, reduced to a monotonous droning, vainly struggles to translate, in broken sentences, the conceptions of the intelligence that still beams unaltered from the eyes; when the chest no longer heaves with the strongest breathing effort, and painful gusts of coughing only reveal the more distinctly the utter inability of the sufferer to expel the mucus that clogs the bronchial tubes; with the

head sunk on the sternum; incapable of motion in hand, foot, or trunk; devoured by hunger, yet almost choked by swallowing a morsel or a drop; then, indeed, the physician can only lament the imperfection of science, and store in his heart the mournful incidents of the painful scene; trusting that, like a skilful general, he is gathering in the hour of present defeat, the precious seed of future victory.

But when the muscles of the trunk are only partially affected, and the respiratory muscles are not yet reached, there is still a chance of life, and even of recovery, as may be seen in two cases mentioned in the next chapter. The chance is small, but it is of great importance to substantiate its reality, that we may be encouraged to ply our remedies in spite of every discouragement, even to the last.

The signs that the disease is extending itself to the trunk, and that, consequently, the worst results are to be apprehended, are diminished mobility of the lips, a slur in the articulation of words, abridged range of the vocal register, frequent sighing, the appearance of fibrillary tremors on different parts of the chest, abdomen, or face. So long as none of these phenomena can be observed, life may be considered unmenaced.

The likelihood that the partial form will pass into the general depends on various circumstances. If there be a hereditary taint, there is every fear that the disease will involve the whole body, and

run a fatal course. The same danger, though in a greatly inferior degree, is to be apprehended when the lower limbs are the first attacked (see table p. 126); also, when the upper and lower limbs are *both* implicated. On the contrary, when the atrophy starts in the hands, and delays there and in the forearms, expending its force in entirely immolating the muscles of those parts, there is not much ground of apprehension that it will pass higher up. Generally, it would seem that the more limited its operation, and the more tendency there appears in its career, to do its work thoroughly in a narrow tract, rather than incompletely to occupy an extended territory, the less is the peril to life. Whereas, when it passes from one region to another, before the muscular degeneration in the first has proceeded far, there is more cause for alarm.

It may be considered a favourable circumstance, when the cause can be pretty clearly made out to be excessive labour; hence the prognosis is better in artizans and needlewomen than in those moving in easier circumstances, or those following promiscuous employments, servants, labourers, &c. (See p. 136.) It is an ill augury, on the contrary, when the disease has resulted from cold, inasmuch as it is more liable to become general than when arising from hard work. (See p. 146.)

The longer wasting palsy has continued, the less amenable it is to treatment, and the more remote is the prospect of recovery. (See table,

p. 128.) No case which has lasted above two and a half years has hitherto issued in recovery. With regard to recovery from a partial atrophy, which has become stationary, the same remarks hold good. After a year or two, if no amendment has set in, I fear the foregoing histories do not warrant any hope of recovery.

CHAPTER X.

TREATMENT.

The plan of treatment put into practice, by those who have encountered instances of wasting palsy, has varied according to their view of its pathology. Those who place the chief and primary lesion in the spinal cord, apply their remedies over the vertebral column, where they open issues, insert setons, cup, or blister, to effect a favourable diversion in the outer tissues, from the more deeply seated and important part within. There is no evidence that benefit has been derived from this plan, and very often its futility has been most apparent. When improvement has taken place during the operation of such means, as in Dr. Reade's case, other remedies were employed at the same time, which, from other experience, are known to possess a really curative power. Of strychnia and nux vomica it may be said, that, although frequently tried, they have not yielded any beneficial results.

When the entire economy is looked on as deranged, and the atrophy of the muscles is only regarded as an isolated sign of a general dyscrasia or taint, internal remedies have been exhibited which are supposed to exert a restorative influence

on the general nutritive functions; or which are reputed capable of ridding the system of the incubus which oppresses, or the poison which defiles it. On the first of these grounds, Mr. Cline prescribed mercury in the case published by Dr. Cooke, and the arrest of the wasting was coincident with the operation of the metal; on the first and second grounds, steel and other tonics, and cod liver oil, have been employed by Drs. Meryon, Gros, and others; and on the third ground, Dr. Niepce put his patient under the influence of iodide of potassium to expel the syphilitic poison suspected to lurk in the blood, and recovery set in simultaneously with the appearance of an eruption, pronounced venereal. *Mercury*, either to affect the gums or in alterative doses, has been frequently employed on one or other of the above enumerated pleas; with what success can not be asserted with confidence. In the officer described by Dr. Cooke, a result was brought about, which, independent of any medicament, is the commonest issue of the disease, and it may be doubted, until further proof, what share the drug had in determining the arrest. In other instances, mercury and *iodide of potassium* have not had a show of success. And in a third class of cases, other means conjoined with them so embarrass our estimate of their real value, that it is impossible to know what portion of the favourable results may be adjudged to their influence. On the whole, I am disposed to regard mercury and

iodide of potassium as weapons to be kept in view, and their employment to be recommended where there is cause to suspect either a syphilitic or a strumous taint, or where—which is very rarely the case—the general nutrition has become faulty.

Two remedies of unquestionable value, *methodical exercise, and cold mineral and douche baths*, have a double working. They affect directly the muscular system by their topical operation, and they re-act potently on the aggregate of the functions by their general impression. Yet these means have been found, in some instances, acting prejudicially; hastening, instead of staying, the morbid action. Probably, they should not be used indiscriminately. When the disease is in active progress, they are more likely to do injury than to promote recovery. Cold baths are to be especially avoided; for depression of temperature is, on all hands, allowed to be most inimical to the vitality of parts stricken with atrophy; and the same objection lies against exercise of the muscles, except the most moderate, during the active stage; but once the morbid force has spent itself, and there appears no fear of a renewal of its activity; when we have to deal with the remnants of a bygone struggle, and the prostrated vitality is no longer vitiated but dormant, then the bracing stimulant of mineral baths and systematic exercise acts beneficially, rousing the enfeebled nutritive force to a

fresh activity, and staving off that permanent sinking of the muscle into a hopeless marasmus, from which there is no amendment.

Thermal and sulphur baths are frequently mentioned as having produced a favourable result; in some cases only temporarily, in others leading to recovery. Indeed, there is only one remedy, galvanism, which has greater evidence for its efficacy; and, in looking over the table of cases, it may be seen repeatedly that arrest, either temporary or permanent, was obtained by their use. There is not the same objection to their employment during the active periods as to cold baths, and their success is very much more decided.

To those who, with the writer, regard the muscles themselves as the primary seat of the disease, strictly topical remedies assume a place of the highest importance. *Frictions*, with stimulating liniments are favourably mentioned; their effects probably resemble those of galvanism more nearly than any other remedy, but they are much feebler. When galvanism is not to be obtained, or cannot be borne, they are the best substitute. In the case of Elizabeth Foster, the limb was galvanised in the morning, and rubbed with camphor liniment at night; in that of the tailor, Thomas Holland, a stimulating embrocation was the only means employed, and its use was followed by decided improvement; but, as he ceased attending, the further history of the case fails.

The most effective remedy for wasting palsy is *galvanism*, applied locally to the wasting muscles. Its efficacy is placed beyond cavil by the evidence of Duchenne, Gros, Meyer, and others. It has generally, indeed nearly always, been found to yield encouraging results; too often the amendment has been but temporary, but in several instances it has brought about arrest, and, in a few, re-establishment of the bulk and power of the wasted muscles, either wholly or in part.

Duchenne relates some very remarkable examples of the power of localised electricity. The following observation not only shows the value of the remedy, but also, incidentally, the possibility of repeated relapses from too early resumption of muscular exertion:—Goulard, æt. 31, carried fowls to market in a basket, placed on his head. This was kept steady by the uplifted hands, which were thus habitually raised to the vertical position. This occupation soon affected his strength, and wasting palsy set in, involving the muscles of the upper limbs, and those of the vertebral column in the lumbar and cervical regions, so that he could not raise the arms from the side, nor straighten the body. The head fell forward on the chest, and the spine was strongly projected backward; his gait was tottering, and he could with difficulty preserve his equilibrium. "Such was the state of Goulard," says Duchenne, "when I commenced the treatment by localised Faradisation,* practised

* This is a term, invented by Duchenne, to signify the employ-

three times a week in my parlour, for from five to ten minutes each time. At the commencement of this treatment, my apparatus, which is a very powerful one, provoked but feeble sensations, when I directed the excitation on the sacro-spinal mass and the deltoids, although it was graduated to its maximum, and the intermissions were exceedingly rapid. But after a month of Faradisation, all the muscles had recovered a great part of their (muscular) sensibility. The trunk could be erected, and the arms raised to the horizontal position; the muscles grew in volume in a very sensible degree, and the movements of the hands had improved. Goulard then, believing himself half cured, came no more, and returned to his employment, although he had barely strength for it. (This was in June). In the month of August he revisited me in the same condition as the first time. * * * Goulard was again vigorously galvanised, and steel tonics and sulphur baths, two or three times a week, were employed as auxiliaries. Under the influence of these means the muscles enlarged rapidly; the vertebral fossæ filled, the head and trunk were held up, and the elevation of the arm was soon complete; finally, at the end of a month, Goulard was nearly cured. I continued the Faradisation for another month, and then, having recovered the action of all his muscles and his muscular

ment of the electricity of the induced or secondary current in the helix round the magnet, which was discovered by Faraday; and, in his honour, Duchenne constructed the term.

force, he went to work. After this, from time to time, the Faradic excitations were directed to those parts of the body where fibrillary tremors were observed. The cure of Goulard lasted nearly a year; but having abused his strength by carrying heavy weights, 150 or 200 pounds, on his head for months successively, he had a second relapse, and was compelled again to renounce work. The same muscles were affected as heretofore, but in a minor degree. Goulard was much less bent when he assumed the standing posture, and he could raise the head; but the slightest push was sufficient to tumble him over on his face. He was again submitted to localised Faradisation and cured."*

Duchenne gives the following directions for employing this powerful remedy, and here he is entitled to be heard as a master. "Every muscle ought to be Faradised in a special manner, according as it has suffered more or less in its electric contractility and nutrition. * * * Thus, the more a muscle is atrophied and its contractility diminished, the longer it should be submitted to the stimulation, the more intense should be the current, and the more rapid its intermissions. And this strong current and quick intermission are the more necessary, according as the sensibility of the muscle is more benumbed. But, when the sensibility is seen to return, it is prudent to diminish the intermissions

* Duchenne. Loc. Cit. p. 824. slightly abridged.

and abate the intensity of the current, and even to abridge the number of sittings, lest there be provoked unmanageable neuralgia, and, which sometimes has arisen, inflammatory accidents. During the Faradic treatment of the patients mentioned in this work* I have excited the muscular sensibility, as much as possible, by rapid intermissions, inasmuch as I have found this the most effective means of re-acting on the nutrition of the atrophied muscles. Sittings of too long duration fatigue and even exhaust the muscles, just as forced exercise induces atrophy instead, like moderate exercise, of favouring nutrition. I believe that no sitting should be protracted beyond 10 or 15 minutes, at the most. I rarely give more than one minute to each muscle. To prevent weariness, and a bruised feeling, that sometimes follows the application of electricity, I pass rapidly over the muscles, taking care to return to each of them several times during the same sitting, so as to leave a short interval of repose between each excitation." †

Not inferior in interest to the history of the case just related, is one recorded by Dr. Gros.‡ A young carpenter, aged 22, suffered from general muscular atrophy, which had deprived him of

* De l'électrisation localisée.

† Op. cit. p. 702. These remarks are made at the conclusion of his article on the application of electricity to traumatic paralysis of the mixed nerves, but they apply strictly to Cruveilhier's atrophy.

‡ Gaz. des Hop., 1855, No. 50, slightly abridged.

the use of his arms and legs. Muscular vibrations were extensively distributed over the trunk, and the wasting was extreme in several muscles of the legs and arms, with a total loss, in some of them, of their electric contractility. He was admitted into hospital on the 26th of August, 1853, and the report goes on to say, "He was galvanised every morning. After 10 sittings the fibrillary tremors had ceased, and the contraction of the arms and legs was improving. The wrists had begun to recover their movement. In a month he supported himself on his feet, as if on stilts; the muscles of the leg did not, as yet, fix the tibiotarsal articulation. On Oct. 27th, an appearance of voluntary contraction was seen in the anterior muscles of the leg. In the course of November, the foot was solidly joined to the leg; the patient walked without support, but he could not stand still in the same place, his two feet being in a state analogous to two Chopart's amputations. In December, he could raise the wrists freely; but the hand was still a claw. The use of electricity was now stopped for 21 days to observe the consequences. On the 22nd of January he was exactly as I left him. The treatment being resumed, he continued to progress. In March he could stand firmly, and the muscles had regained their tonicity and volume. The interossei of the hand had resumed their play, and the fingers could be stretched out. For some time past he has fed himself. The eminences (thenar and hypothenar) are still atrophied,

but give a "soupçon" of a contraction. A new interruption of 16 days (owing to the illness of Dr. Gros) proves that the patient makes not the least progress without the electricity."

Adopting the view of the pathology of wasting palsy advocated in this essay, the treatment naturally divides itself into two lines of indication. The object of the first is to arrest or cure the atrophy of the muscles, and that of the second to combat the secondary phenomena. In dealing with the primary disease, our first endeavour must be, to seek out the *exciting cause*, and obviate, if that be practicable, its continued operation. The handicraftsman must immediately renounce the practice of his mechanical art; the labourer must take off the strain from the overworked members. If cold and damp be the original excitant, they must be sedulously avoided in future, to escape the risk of a recrudescence or relapse. The *direct* treatment must have for its end, in the active stage of the malady, to restore the nutritive operations from their depraved estate to their original healthy tenour. This is accomplished, by a judicious combination of perfect repose and regulated stimulation. Experience has shown that, for the latter purpose, no remedy approaches galvanism, which should be applied to the muscles daily, or every other day, in the manner above directed. With galvanism may be combined gentle frictions of the parts affected, with some stimulating

liniment; and, if the means be at hand, warm sulphuretted or saline baths may be employed occasionally, as adjuvants to the local applications. When the disease has become stationary, more violent stimulation by electricity and friction must be resorted to, and cold bathing, with enforced exercise of the muscles, may be tried, if other means fail of effect. Remedies should be employed without loss of time, for when the atrophy has extensively affected the muscles of the trunk, or has remained stationary for a lengthened period, the chance of recovery diminishes in an accelerated ratio.

The *secondary* phenomena, neuralgic pains, spasms, etc., are most effectually subdued by warm baths, and inunctions; or, if they obstinately persist, by morphia-dressed blisters over the painful tracks. Any ailment of the general system, or concurrent and independent disease, must be dealt with, in accordance with the recognised canons of therapeutics.

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