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BY

LANDON CARTER GRAY, M. D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE NEW YORK POLYCLINIC.

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By LANDON CARTER GRAY, M. D.,

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The muscles, the motor nerves, and the anterior column of gray matter, whence the motor nerves originate, constitute, anatomically and pathologically, a system apart, which may be termed the neuro-muscular apparatus. Disease of any one of the three component parts of this neuro-muscular apparatus—muscles, motor nerves, anterior column of gray matter—is so apt to extend into the two others, or be associated with them, that the close anatomical connection between the three is evidently the main material reason for as intimate a pathological relationship. It is, therefore, necessary to have a clear conception of the anatomy of these structures.

The anterior column of gray matter extends throughout the spinal cord and the upper enlarged intracranial end of the spinal cord, which is known as the oblong cord or medulla (medulla oblongata), and into the third ventricle. It is placed in different positions in these portions of the nervous system. In the spinal cord it is in the center, in the shape of so-called "horns," or "cornua." In the intracranial portion of the spinal cord—the medulla oblongata,

the floor of the third ventricle, and the aqueduct of Sylvius —it is diffused into separate masses, so-called "nuclei."

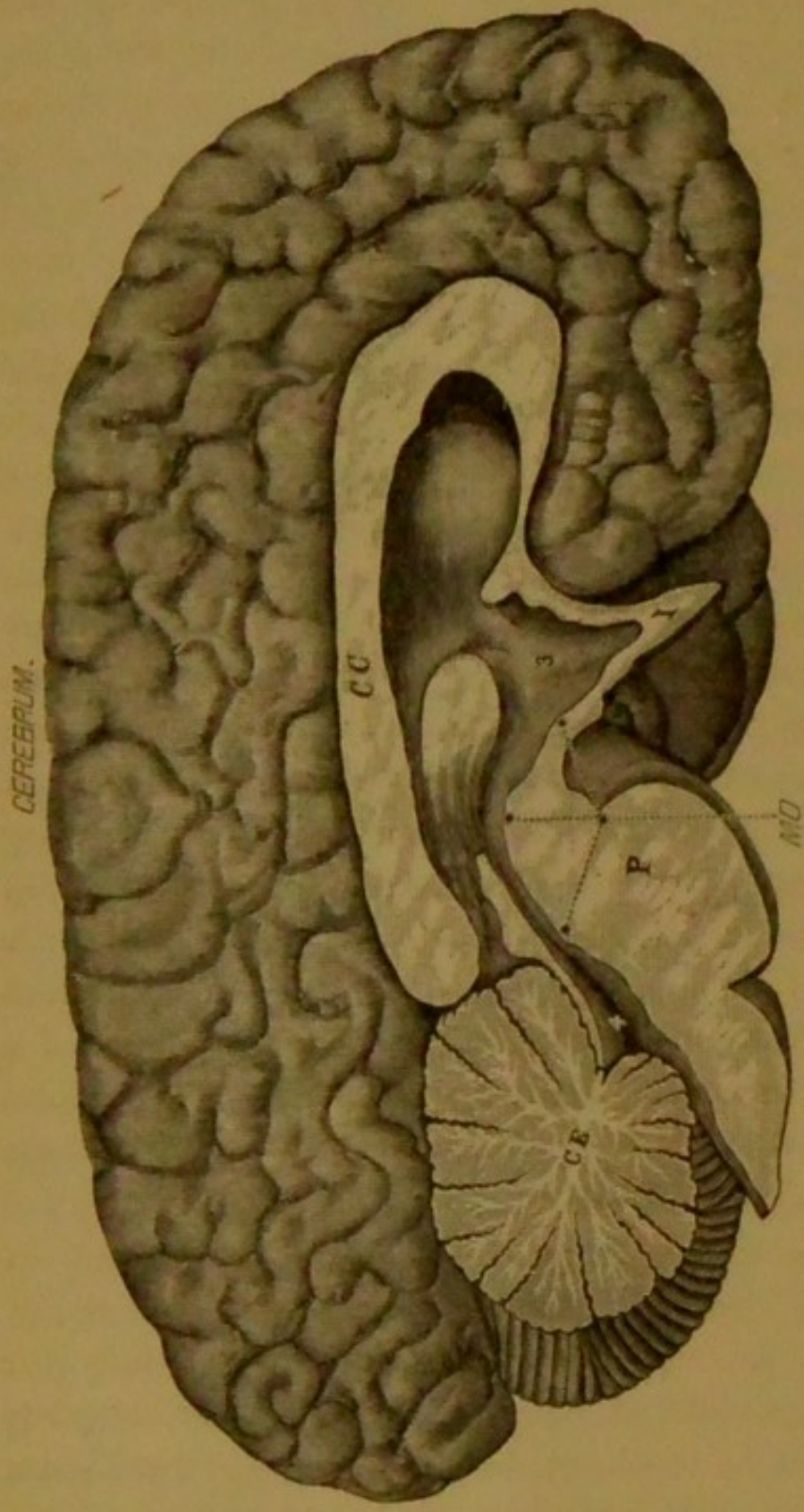


FIG. 1.—CC, the corpus callosum; P, the pons Varolii; 1, the infundibulum, or funnel-shaped prolongation of the third ventricle; 3, the third ventricle; 4, the fourth ventricle, the floor of which is formed by the upper surface of the medulla oblongata; CE, the cerebellum. From the fourth to the third ventricle is seen a channel, which is the aqueduct of Sylvius. The three diverging lines MO point to the surface whence arise the fibers of the motor oculi, the anterior line indicating the origin of the fibers to the iris, and the tensor of the chorioid; and in the floor of the third ventricle, the aqueduct of Sylvius, the fourth ventricle, and below, are the nuclei of the other cranial nerves.

This anterior column of gray matter extends, therefore, from the cauda equina up into the anterior and lowermost

portion of the third ventricle. It is always, without a known fact to the contrary, motor in its function, and gives origin only to the motor nerves. At the present day our knowledge is very exact concerning the levels in which the different motor nerves thus have their origin, commencing above with the third pair, or motor oculi, and passing down to the nerves of the lower extremities. Fig. 1 will show the intracranial portion of this gray matter, and should be carefully studied. It represents a longitudinal section through the center of the intracranial mass, showing the ventricular cavities and their relations. The nerve cells within this anterior column of gray matter are known as the ganglion cells. They are of considerable size and endowed with a distinct nucleus and nucleolus and with a number of protoplasmic prolongations running off from the body of the cell, known as processes. These processes are of two kinds. One branches and rebranches as it passes away from the cellular body, growing smaller as it goes, and is called the "protoplasmic process." The other does not branch as it passes away from the cell, but grows slightly larger, and has been seen by a great number of competent observers to take on a covering of myelin and pass to the periphery; hence, it is extremely probable that it represents the axis-cylinder of a peripheral nerve, and it is called the "axis-cylinder process." The termination in the muscles of the nerve-fibers thus originating has been carefully investigated by many of the best-known histologists of the day; most recently by Gessler in the green Italian lizard and the guinea-pig. Gessler states that the motor end-plate is essentially the same in the *Mammalia* and reptiles. This motor end-plate consists of a fine nerve-plexus, between or under which—for authors differ on this very immaterial point—lies a finely granulated mass, containing two varieties of nuclei—the one kind, the smaller, being granulated and of irregular shape;

the other kind being larger, oval in shape, with double contours and a nucleolus. The former are known as the *Scheiden-* or *Geästkerne*, or, as Ranvier calls them, the *noyaux vaginaux* or *de l'arboration*, while the latter are the *Grund-* or *Sohlenkerne*, or *noyaux fondamentaux* of Ranvier. To this motor end-plate, lying upon the muscle, come the medullated nerve-fibers at a right angle, losing their medullary sheath as they pass into the nerve-plexus just described.

There are reasons for supposing that these ganglion cells in the anterior column of gray matter are of two kinds in regard to their function; for these anterior columns are subject, on the one hand, to disease in which motor paralysis is the first and predominant symptom and, on the other hand, to other disease in which wasting of muscular fiber is the first and predominant symptom. It would, therefore, seem as if in the first case there were a motor cell, whose disease gave rise to paralysis, while in the latter case it seems impossible to deny that it is disease of a nutritive or trophic cell which has produced the primary wasting of muscular fiber. To be sure, in every case of disease of the cells of the anterior gray column there is sooner or later certain to be both motor paralysis and muscular atrophy, without regard to which came first; and the two kinds of cells are thus always affected, although not simultaneously.

Disease in any one of the three component parts of this neuro-muscular apparatus — ganglion cells, motor nerves, muscles—will produce a motor paralysis and atrophy. This should be very easily understood. If the ganglion cells are diseased, the motor nerve will degenerate and the muscle will be paralyzed and will atrophy. If the motor nerve is diseased, the portion of nerve below the site of disease will also degenerate and the muscle will be paralyzed and will atrophy as before. If the muscle itself is diseased, it

may be paralyzed, of course, and may atrophy. The three symptoms, therefore, of disease of the neuro-muscular apparatus are: 1, motor paralysis; 2, atrophy of muscles; 3, electrical changes brought about by degeneration of nerve and muscle.

We can now readily understand why there has been such dispute, when these three symptoms were present, as to which one was diseased of the three component parts of the neuro-muscular apparatus—ganglion cells, motor nerves, or muscles. The real objective point is the muscle. It is very dependent on the nervous system, and the nervous system is very dependent on it. Neither can do without the other. The muscle can not contract intelligently except by means of the nervous system. The nervous system can not express itself except by means of certain muscles, and can not exist except by means of certain others. The brain, sending down its mandates to the cells in the anterior column of gray matter, and thence through the motor nerves, can only communicate its various thoughts and emotions to the outside world through the muscles, and can only act through them; and the nervous centers that, in their mysterious way, supply nervous force to the muscles of the heart, respiratory structures, and arteries, can not maintain life without muscular action, as of the vascular apparatus. On the other hand, of what use would a leg or a heart be unless each was in intimate connection with the nervous system? So, after all, it is the muscle that is to be studied in disease of this neuro-muscular apparatus. Muscular—*i. e.*, *motor*—paralysis and muscular atrophy, then, with or without electrical changes, are the signs of disease in the neuro-muscular apparatus, *i. e.*, of disease either in the anterior gray column, in the motor nerves, or in the muscles.

A priori it is difficult to understand why disease attack-

ing one portion of this anterior gray column, say that of the lumbar enlargement, should not travel up throughout the whole column, implicating all the centers of origin of the muscles of the trunk, neck, heart, respiratory structures, face, and head; or why disease should not always extend inward through the muscles as well as outward to them. Laws of disease, however, as yet unknown to us are at play here, as in other structures of the body; and we can only study the workings of these laws without attempting an explanation of them. We can certainly say that, during the first three years of life, the anterior column of gray matter in the dorsal cord is specially prone to one-sided disease. We can certainly say that that portion of the anterior gray column containing the nuclei of the facial, hypoglossal, and spinal accessory nerves is specially prone to disease that does not extend upward or downward. We can certainly say the same thing of the nuclei of the different nerves of the eyeball, the third, fourth, and sixth. We can certainly say that in certain individuals the muscles alone are diseased. But certain as we are in saying all these things, we can not attempt to explain why it should be so. It must, moreover, be remembered that these different results were seen before the pathology was known; so that the nomenclature, being representative of the purely clinical or locally pathological standpoint, is by no means consistent.

The diseases of the neuro-muscular apparatus are known as—

1. Myelitis of the anterior horns, or cornua.
2. Glosso-labio-laryngeal paralysis, or bulbar* paralysis.
3. Progressive ophthalmoplegia.
4. Muscular pseudo-hypertrophy.
5. Progressive muscular atrophy.

* An adjective bestowed upon the medulla oblongata because of its bulbous shape.

Of these, the first three—myelitis of the anterior horn, bulbar paralysis, and ophthalmoplegia—are due to lesions in the anterior gray column; the fourth, pseudo-hypertrophy, is indubitably of muscular origin; while the fifth, progressive muscular atrophy, is sometimes due to lesions in the anterior gray matter, sometimes to muscular lesion, sometimes to both neural and muscular lesions. Each of the three due to central lesion has an individual localization in the anterior gray matter. The first, myelitis of the anterior horn, arises from implication of the anterior gray matter of the spinal cord; the second, glosso-labio-laryngeal paralysis, arises from implication of the nuclei in the medulla oblongata of the hypoglossal, facial, and spinal accessory nerves; while the third, progressive ophthalmoplegia, is due to affection of the nuclei of the ocular nerves in the floor of the aqueduct of Sylvius and the third ventricle.

MYELITIS * OF THE ANTERIOR HORNS, OR CORNUA, is a motor paralysis affecting muscles of the extremities that are associated in function, and followed by atrophy and electrical alterations in the paralyzed muscles. Although it may occur at any time of life, cases having been observed at the extremes of three months and sixty-seven years, there are yet two periods of life which seem especially predisposed to it—viz., the three first years of infancy and the period between the eighteenth and fortieth years. It may be acute, subacute, or chronic. In all cases the paralysis is the first symptom, and is followed by atrophy in the course of a few weeks in the acute cases, or a longer time in the chronic ones. It is usually confined to one extremity in children, generally to a lower limb, while in adults paraplegia is the most usual form of paralysis.

* Synonyms are anterior polio-myelitis, essential paralysis of children, infantile spinal paralysis, atrophic paralysis of children.

Glosso-labio-laryngeal paralysis * is a motor paralysis with muscular atrophy, affecting the motor cranial nerves and their muscles, especially the hypoglossal, certain filaments of the facial, and certain filaments of the spinal accessory. Usually the atrophy is the first symptom that is noticed, although some authorities contend that the paralysis may be primary. The atrophy first shows itself in the muscles

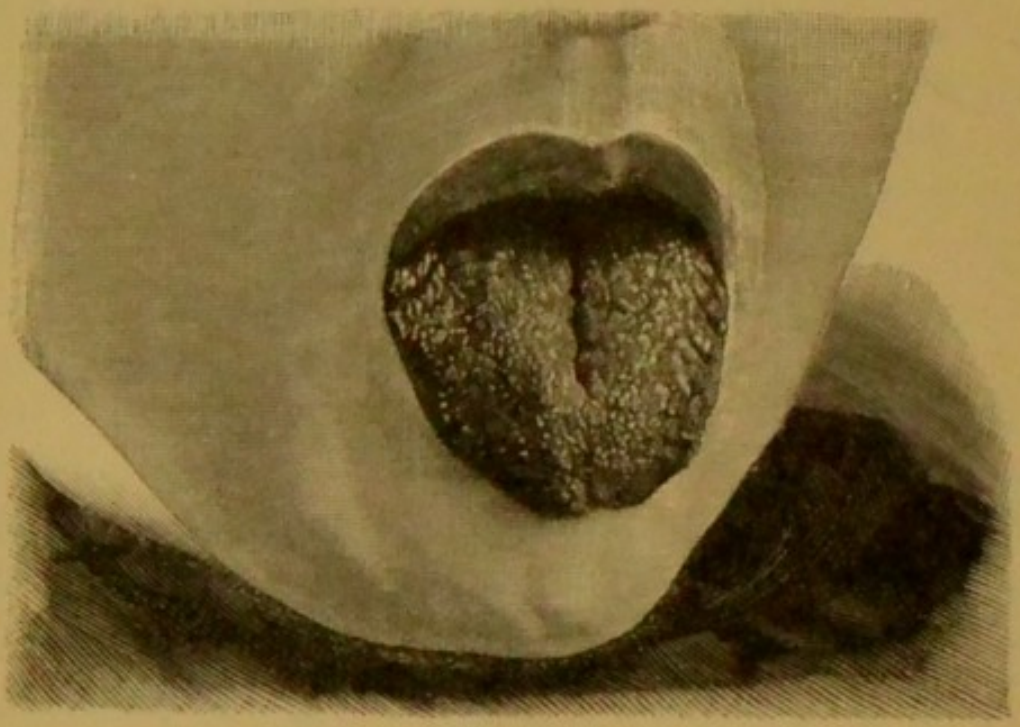


Fig. 2.

of the tongue, then in the muscles of the lips and the lower face (seldom in the eyelids), and, finally, in the muscles of the larynx, pharynx, œsophagus, and heart. The lingual difficulty is evidenced, as has been said, by the atrophy of the tongue, giving the tongue a fissured and crenulated appearance, as if its substance had been chipped out in places and trimmed on the edges, as is seen in Fig. 2, in which the thinning of the organ causes it to curl over and have a fallacious increase in size. Furthermore, the increasing paralysis causes difficulty in the pronunciation of the letters

* The synonym is bulbar paralysis.

which are mainly pronounced by the tongue, and some difficulty in deglutition. The affection of the lower face and lips is denoted by a wasting of their muscles, by difficulty in the pronunciation of the lip-sounds, and by alteration in the expression of the face.

PROGRESSIVE OPHTHALMOPLEGIA is manifested by a paralysis of the muscles of the eye, which are of two varieties —the exterior and the interior. The interior muscles are the sphincter of the pupil and the tensor of the chorioid, while the exterior muscles are the six well-known ones attached to the globe. The third pair, the motor oculi, supplies these interior muscles and four of the six external ones, as well as the levator palpebræ superioris. The four thus supplied are the internal rectus, turning the eye inward; the superior rectus and the inferior oblique, the two elevators of the eye; and the inferior rectus, one of the two depressors of the eye. Of the two others of these six exterior muscles, one, the superior oblique, is supplied by the fourth or trochlear nerve, while the other, the external rectus, is supplied by the sixth nerve, the abducens. It is the nuclei of these different ocular nerves which are affected in the so-called ophthalmoplegia, and the symptoms are thus the symptoms of paralysis of these exterior and interior muscles, together with the levator palpebræ superioris. As might be expected by reasoning beforehand, the paralysis may sometimes affect the exterior muscles, sometimes the interior muscles, and sometimes partially both.

MUSCULAR PSEUDO-HYPERTROPHY is a disease of early childhood, characterized by a deposit of fat around the muscular fiber, more especially in the muscles of the lower extremities, which come to have a hypertrophied appearance. The upper extremities are usually affected simultaneously with an atrophy, and the contrast between the seemingly sturdy legs and the wasted arms and shoulders is not likely to be

forgotten when once seen. Attention is at first directed to the disease by the stumblings and falls and increasing weakness, which seem the more inexplicable because of the simulated robustness of the members.

Progressive muscular atrophy is a disease about which there has been much dispute and great confusion. The early writers were strongly of the opinion that it was always due to disease of the anterior gray column; Friedreich wrote a great tome upon the subject in 1873 to prove that it was always of muscular origin; and in the last few years facts have accumulated to prove that it has sometimes a muscular and sometimes a central origin. The great strife waged in regard to the pathology led to an attempt to throw light upon this subject by the study of so-called "types," or, in other words, to say that, when certain groups of muscles were affected, the disease must be of central origin, and that, when certain other muscular groups were affected, the disease must be of peripheral origin. This type-making business has been carried to such ridiculous extremes that there are fully a dozen different groups which the pretended discoverers would have us know by their own names; nevertheless, we are warranted, both from a clinical and from a pathological standpoint, in accepting four types:

1. The hand type.
2. The juvenile type (Erb).
3. The infantile facial type (Landouzy and Déjérine).
4. The peroneal type (Charcot, Marie, Tooth).

1. *The Hand Type.*—In this the onset is generally in the right hand, occasionally in both hands. The short muscles of the thumb and the ball of the little finger are generally first affected—the so-called *thenar* and *hypothenar* eminences. It is usual for the atrophy to begin in the abductor pollicis brevis, and then be observable in the opponens and adductor. The complete atrophy of these thumb muscles

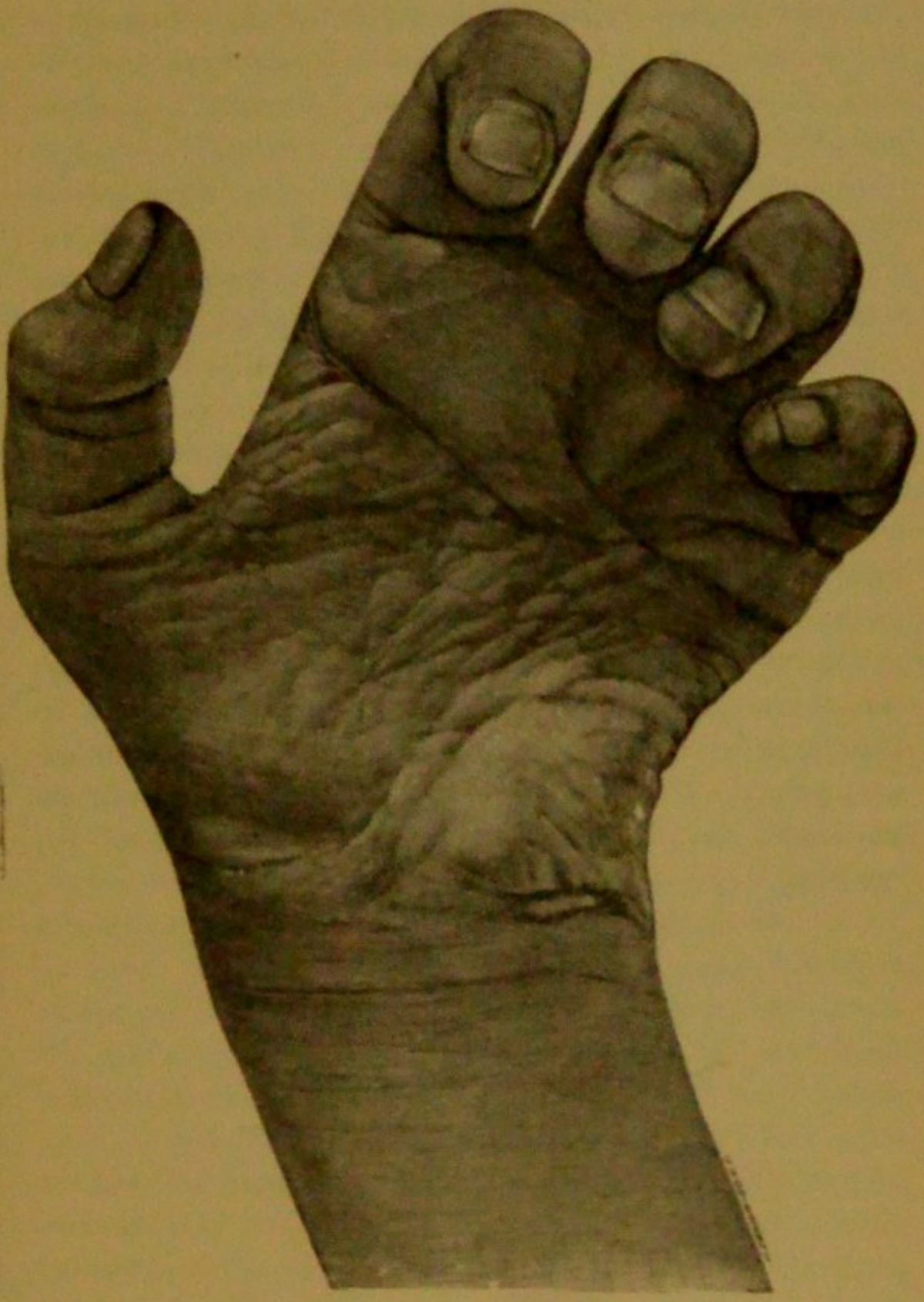


Fig. 3.

produces a condition to which the name has been given of "ape-hand" (*Affenhand*). Fig. 3 shows this beautifully, besides the droop of the wrist due to ulnar paralysis.

At about the same time, sometimes a little sooner, sometimes a little later, the interossei are implicated, as is to be seen by a sinking of the spaces on the back of the hand; when this atrophy has attained to a certain grade, it produces, together with a contraction of the antagonist muscles, the so-called "claw-hand" (*main-en-griffe*, *Klauenhand*).

Consecutively to the hand muscles, often after a long time, sometimes even after years, the forearm or upper-arm muscles are usually attacked. In the forearm it is apt to be the abductor and extensor pollicis longus, the supinators, and the flexors. In the upper arm the deltoid is almost invariably the first to waste, then the biceps, and, finally, the triceps last of all. But, sooner or later, the muscles of the trunk may be implicated, especially the trapezius, the pectorals, the rhomboids, and the latissimus dorsi. Atrophy is infrequent of the cervical or respiratory muscles, or of the diaphragm. After many years of duration the nuclei in the medulla oblongata may be affected, and then may ensue atrophy of the tongue and difficulty of deglutition, and death may result from inanition or respiratory disturbance.

2. *The Juvenile Type* (Erb).—This form was first described by Erb, in 1884, and differs essentially from the hand type in the location of its onset, which is almost always in the muscles of the shoulder and upper arm, far less often in the muscles of the pelvis and lower extremities, while, as has just been explained, the hand type primarily affects the hand muscles; and only secondarily, often after a long time, are the shoulder and arm muscles involved. The shoulder and arm muscles often atrophied are the pectorals, trapezius, rhomboids, serrati, latissimus dorsi, longissimus, sacro-lumbalis, forearm flexors, supinator longus, and triceps; and, in the lower extremities, the glutæi, quadriceps, peronei, and tibialis anticus.

3. *The Infantile Facial Type* (Landouzy and Déjérine).—It generally begins with an atrophy of the muscles of expression, which is thus described by Landouzy and Déjérine: "The face is in repose, the lips protruding, the brow like ivory, a seeming protrusion of the eyes; in efforts at mimicry the smile is sad (*rire en travers*), the movements of the lips are incomplete, and the immobility of the lips is in marked contrast to the animation of the eyes." When the face is wasted, it is the rule that the muscles of the shoulder and arms are involved next; but certain muscles usually remain intact—such as the supra-spinalis, the infra-spinalis, the infra-scapularis, and the flexors of the hand and fingers.

4. *The Peroneal Type* (Charcot, Marie, Tooth).—The French writers described this form in 1886, while Dr. Tooth states that he also called attention to it at the same time in his graduation thesis. As is indicated by the name I have given it, it first shows itself in the muscles of the leg, invading the hand, and then the forearm several years afterward.

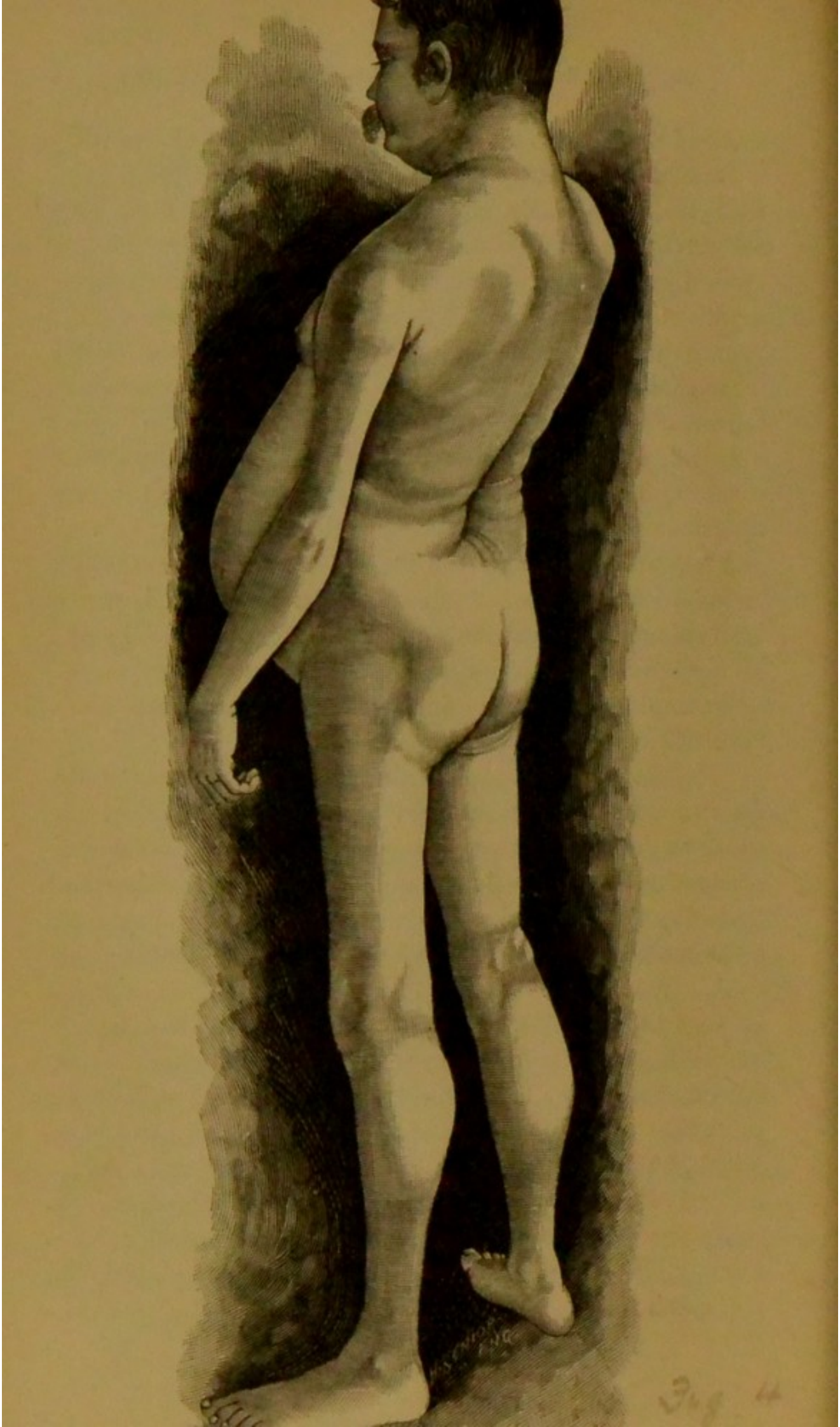
The accompanying table shows, side by side, the location of the atrophy in each of these four types:

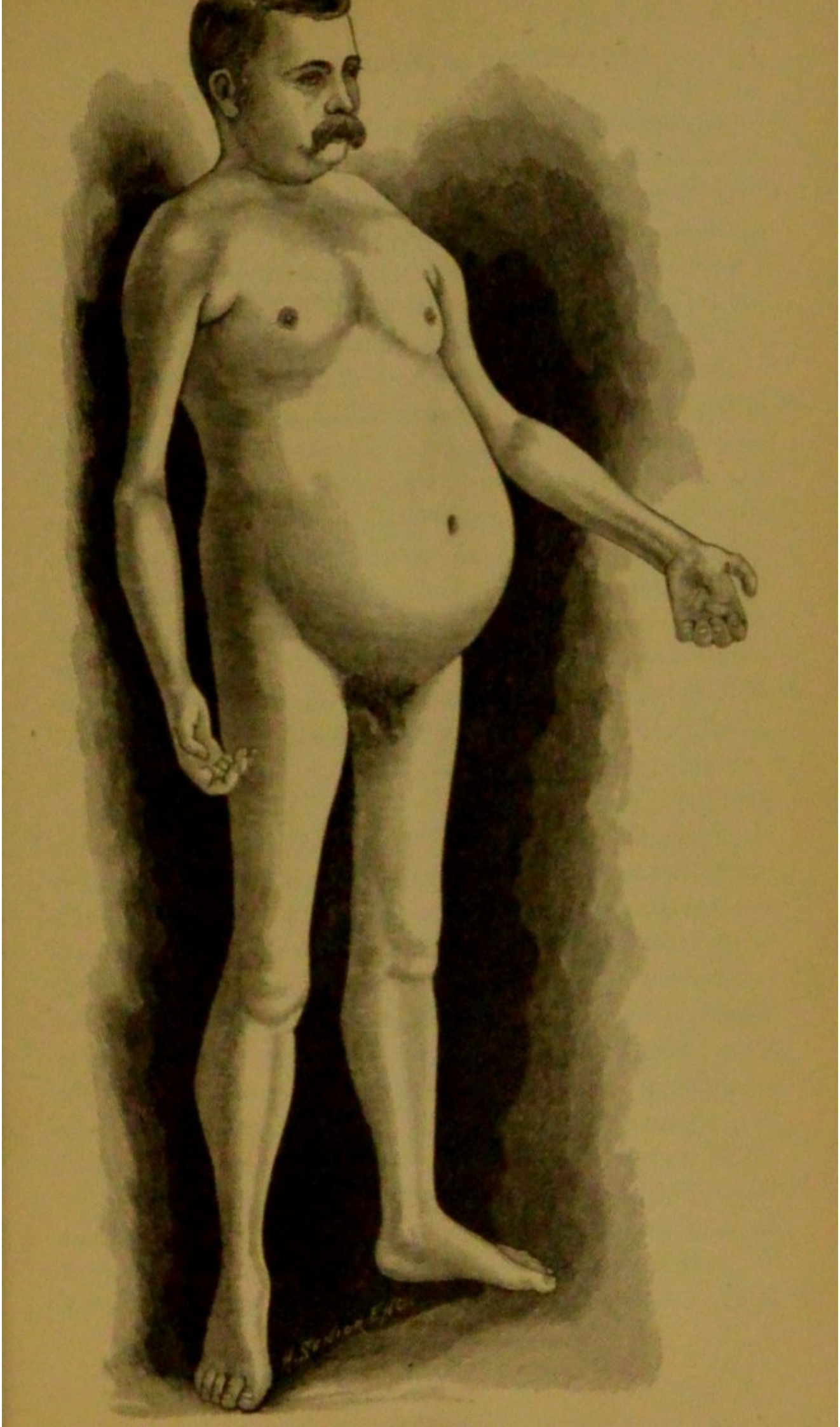
Hand type.	Juvenile type.	Inf. facial type.	Peroneal type.
Muscles of the thumb and fingers: Abductor pollicis brevis, Opponens, Adductor, Interossei, Lumbricales. Ape-hand. Claw-hand. Arm muscles: Abductor and extensor longus pollicis, Supinators, Flexors, Deltoid, Biceps, Triceps.	Muscles of the shoulder and upper arm: Pectorals, Trapezius, Rhomboids, Serrati, Latissimus, Longissimus, Sacro-lumbalis, Flexors of the forearm, Supinator longus, Triceps. Muscles of the lower extremity: Glutæi, Quadriceps, Peronei, Tibialis anticus.	Facial muscles of expression. In the shoulder and arm muscles these are intact: Supraspinalis, Infraspinalis, Flexors of the hand and fingers.	Muscles of the leg, then the hand and forearm.

The course of progressive muscular atrophy is gradual, and the duration is from five to thirty years. The chronic and fibrillary atrophy may not be detected for some time, until enough of the muscle has been affected to alter the contour and consistence, which latter is then found upon palpation to be softer and less resilient than in the healthy muscle. The paralysis is usually proportionate to the atrophy. It not infrequently happens that the loss of muscular strength first attracts the attention of the patient, and an examination then brings the atrophy to view. So-called "fibrillary contractions" are frequently present in all the types, and consist of minute movements of the muscular fibers, varying in extent from an occasional whipcord-like movement under the skin, likened by the patient to a pulse-beat, to a wide set of fibrillary movements that may set a whole group of muscles in tremulous movement. In many

cases of progressive muscular atrophy the muscular atrophy is curiously conjoined with real or pseudo-hypertrophy—*i. e.*, an actual increase in the size of the muscular fiber, or a substitution of fat in the place of the muscular fiber or around it. This is well seen in Figs. 4 and 5, which are of a patient who has been in my hospital wards for some eight years, and whose case began with atrophy in the thumb muscles, as is shown in Fig. 3. Robust as this man seems, he can not raise himself from a sitting posture without great aid, and, when erect, can be literally pushed over by a light touch with the finger. Especially noteworthy are the contrasts between the size of his shoulders and his upper arms and between his forearm and his upper arm; the great ridge in his dorsal and lumbar spine from curvature of the vertebral column anteriorly to compensate for the atrophy and paralysis of the anterior abdominal muscles; the pseudo-hypertrophic outgrowth on the left calf; and the many alternating atrophies and hypertrophies.

The electrical changes in these different diseases of the neuro-muscular apparatus are often very considerable. In order to understand them it is necessary to have some comprehension of the mode of reaction of a healthy nerve and muscle to the galvanic and faradaic current. For diagnostic purposes two currents, the galvanic and the faradaic, are used. When the faradaic current is applied to a nerve leading to a muscle, or to the muscle itself, a contraction of the muscle results, dependent upon the strength of the current used, very little difference being observed between the effect of the positive and that of the negative pole. When, however, a galvanic current is applied to a motor nerve leading to a muscle, or to the muscle itself, the resulting contraction of the muscle will be very different accordingly as the positive or negative pole is employed. If we search for the weakest galvanic current that will cause a





muscular contraction, we shall obtain it first with the negative pole, before we can obtain any contraction whatsoever with the positive. Nor is this all. This contraction thus obtained with the negative pole is only seen when we close the circuit, and is not at all present when we open the circuit. Let the galvanic current now be made stronger, and we shall obtain an intensification of the contraction thus produced with the negative pole at the closing; and after a while we shall obtain a contraction with the positive pole, sometimes first at the closing of the circuit, sometimes at the opening of the circuit. Then, making the galvanic current still stronger, we shall obtain an intensification of what we have already obtained—viz., a contraction with the negative pole at the closing, and with the positive both at the closing and the opening. It will be noticed that so far we have obtained no contraction at all with the negative pole at the opening of the circuit. It is very important to remember that this will not be obtained in a healthy nerve or muscle until the current becomes so strong as to be painful.

From this it will be observed that the healthy muscle reacts first to the negative pole at the closing, and then to the positive pole either at the closing or the opening, and finally that the negative pole at the opening does not produce a contraction unless the current is so strong as to be painful. Suppose, then, that the negative pole at the opening gives us a muscular contraction in a certain case with the same strength of current that is sufficient for a contraction with the negative pole at the closing, is it not evident that the reaction of the healthy nerve and muscle is entirely altered? Or suppose that exactly the same strength of current gives us equally strong contractions with the negative closing and the positive opening and closing, is it not again evident that there is some alteration? In other words, any marked de-

parture from this healthy formula of nerve and muscle is evidence of disease. At the same time that we get these changes with the galvanic current we should also have others with the faradaic, consisting simply of decreasing reaction to the current, so that larger and larger quantities would be required to produce a muscular contraction. But there are other electrical symptoms in these cases, which any one can understand who will take the trouble to study them in any case of neuritis in the human being, or in an artificial neuritis in the frog, rabbit, or guinea-pig that can be produced by section of the sciatic nerve. It will then be found that during the first ten days or two weeks there will be, in the motor nerve and in the attached muscle, decreasing reaction to both the galvanic and the faradaic currents. About the third week the nerve and muscle will begin to behave differently to each of the two currents. To the faradaic the nerve and muscle will display a constantly decreasing reaction, so that larger and larger quantities of electricity will be required, until it may happen that no contraction whatsoever can be produced. But, singular to say, at this period—*i. e.*, about the third week—the nerve begins to show an increasing reaction to galvanism, so that lesser and lesser quantities are required to produce a muscular contraction. At the same time a reversal in the healthy polar formula, such as has been above described, is apt to ensue, so that the negative opening may equal the positive closure, or the positive opening and closure may equal a negative closure. In the course of time this increased reaction to galvanism and the reversal of the polar formula may pass away, and the nerve and muscle may again decrease in its reaction to this current. During this whole period of altered electrical reactions the muscular contraction is more sluggish than in the healthy muscle. All these alterations in the reaction of the healthy nerve and muscle, either togeth-

er or separately, are known as the reaction of degeneration. They consist, as will have been seen, of—

1. Decreased reaction to faradaism and galvanism.
2. Increased reaction to galvanism.
3. Reversal of the healthy polar formula.
4. Sluggish muscular contraction.

These should be studied with the same care and patience that are given to auscultation and percussion.

The pathology of these different diseases of the neuro-muscular apparatus is very well understood at the present day. In three of the five diseases which have been enumerated—myelitis of the anterior horn, glosso-labio-laryngeal paralysis, and progressive ophthalmoplegia—the lesion consists of death or injury of the ganglion cells in the anterior gray horns of the spinal cord or in the motor nuclei of the medulla oblongata, the aqueduct of Sylvius, and the third ventricle. The changes in these ganglion cells vary somewhat according to the extent and kind of the disease. In some cases there is not a vestige left of the cells. In others they are more or less disintegrated, some processes being lost, others being altered in shape and size, while the nuclei or nucleoli may be destroyed, although these latter almost invariably persist when any vestige of the cell is left. In myelitis of the anterior horns and in glosso-labio-laryngeal paralysis the affection is generally confined to the cells, or other changes are evidently consecutive to these, such as increase in the size of the vessels, increased growth of the connective tissue, and shrinking of the cord or medulla oblongata on the affected side. But in progressive ophthalmoplegia the affection of the ganglion cells seems to be only part of a somewhat diffused myelitic process. When the ganglion cells are affected, the motor nerves and the attached muscles will be found to have undergone serious alterations. The nerves are apt to be thinner, grayish look-

ing, and more transparent, their axis-cylinder and medullary sheaths will be found more or less disintegrated, and the connective-tissue sheath thickened and its nuclei increased in size and number. The muscles usually undergo an atrophy, which may be of two kinds: the so-called simple atrophy, in which the muscular fiber is simply decreased in size but its transverse striæ are preserved, while in the degenerative atrophy the transverse striæ are lost. But there may also be hypertrophy or pseudo-hypertrophy of muscle, the first consisting of actual hypertrophy of the muscle fiber, the latter being a conversion of the muscle fiber into fat, as well as a surrounding deposit of fat. In the fourth of the five diseases which have been enumerated, muscular pseudo-hypertrophy, the disease is purely of muscular origin, cases having been collected by Middleton and Schultze in demonstration of this fact. In progressive muscular atrophy there can be no doubt but that some cases are of central origin, others of muscular origin, while it would seem, from the history of some few cases, as if a third class were both of central and of peripheral origin. I have been able to collect thirteen cases in which the disease was central, as was proved post mortem, while there have been but two autopsies of cases of purely muscular origin.

The diagnosis of these various affections of the neuro-muscular apparatus is made, in the vast majority of cases, with ease and certainty. In every instance the symptoms are purely motor; sensory, cerebral, vesical, and rectal symptoms being either temporary in duration or entirely absent. Of course, if the anterior gray column should be implicated in diffused disease extending into it from other parts of the nervous system, the diagnosis may become a very complicated one; but this may be met by bearing in mind the usual complications. These purely motor symptoms consist, as has been said, of motor paralysis,

muscular atrophy or hypertrophy, and altered electrical reactions.

Whenever these three symptoms are present, disease of the neuro-muscular apparatus may be positively diagnosticated. It then becomes a question as to whether the lesion is in the anterior gray column, in the motor nerve, or in the muscles.

The symptoms upon which lesion of the anterior gray column may be affirmed are a sudden onset of paralysis, followed in the course of a few weeks by atrophy and reaction of degeneration; or the group of symptoms described under the head of glosso-labio-laryngeal paralysis; or the gradual paralysis of the different interior and exterior muscles of the eyeball, described under the heading of progressive ophthalmoplegia.

The symptoms upon which a diagnosis may be positively made of disease of the muscles alone are the group of phenomena which have been described under the heading of muscular pseudo-hypertrophy, and the infantile facial type of progressive muscular atrophy described by Landouzy and Déjérine.

Undoubtedly there are many other symptoms that have been vaunted by different authors as of importance in the differential diagnosis, but these are, in my opinion, the only ones upon which we can place absolute reliance. For instance, it has been contended that the form of progressive muscular atrophy which I have described above as the "hand type" always points to a spinal origin, yet cases of purely muscular disease have begun in the hands. And Erb would maintain a purely muscular origin for his cases of the so-called juvenile type, but, as Schultze very clearly points out, Erb's cases are of several kinds, some being evidently pseudo-hypertrophies, others belonging to the Landouzy and Déjérine type, etc., while not a single autopsy has been made in this juvenile form of Erb.

The altered electrical reactions of the so-called reaction of degeneration are only indicative of muscular change. This is indicated very clearly, it seems to me, by the fact that this reaction of degeneration has been found in cases of both peripheral and central origin, as well as by the researches of Gessler upon guinea-pigs and lizards that demonstrated the dependence of this reaction of degeneration upon muscular atrophy alone. Fibrillary contractions, supposed to be of so much diagnostic importance in the days of old, are found in cases of both central and peripheral origin, and are, therefore, of no importance in the differential diagnosis.

Causation.—The most frequent causes of the diseases of the neuro-muscular apparatus are infection, heredity, muscular strain, trauma, acute diseases, exposure to continuous cold, and warm weather. The later German authors are displaying a tendency to ascribe the acute form of myelitis of the anterior horn to infection, because of the simultaneous manifestation of the disease in two or three children of one family or one locality. There is no definite proof of this, however, and it is possible that it may be explained by the fact to which Dr. Weir Mitchell called attention several years ago—viz., the greater frequency of myelitis of the anterior horn during the warmer months of the year. Heredity is usually found in the infantile facial type of Landouzy and Déjérine, and plays a large part in all the types. Some authors would maintain that heredity is a diagnostic mark of the purely muscular forms, but the scientific evidence at our command does not warrant this assertion. Friedreich points out the relation of muscular strain to the resulting atrophy in case after case, and it is a very familiar clinical fact to me.

Progressive muscular atrophy has often been known to follow typhus, diphtheria, measles, acute rheumatism, par-

turition, and cholera; so that these acute diseases must have some unfavorable influence upon the neuro-muscular apparatus.

Diagnosis.—In making a diagnosis it must not be for gotten that other diseases of the spinal cord and its sur rounding membranes or osseous structures may extend into the anterior horns, in which case the symptoms of motor paralysis and muscular atrophy may be superadded to the original symptoms of the invading disease. Such complicating diseases may be spinal hæmorrhage, transverse myelitis, syringo-myelitis, or locomotor ataxia. All of these, besides the characteristic features of each of them, will have marked sensory, vesical, and rectal symptoms. Should a spinal hæmorrhage take place to such a limited extent as only to involve one anterior horn, it might be impossible to make the differential diagnosis; but I know of no such case on record. In the early stages of syringo-myelitis—*i. e.*, formation of a gliomatous tumor and cavity within the substance of the cord—the affection might be limited to one anterior horn, and thus cause confusion, but the later history of the case will make matters plain.

A differential diagnosis must also be made from the acute exanthemata; meningitis, traumatic, from ear disease, or cerebro-spinal; meningo-encephalitis of children; neuritis, simple or multiple; lead palsy; joint atrophies; and hysteria.

It is possible to confound the early stage of myelitis of the anterior horn with meningitis. But in the latter the mental disturbance is much more marked and of longer duration; the cranial nerves may be seriously implicated, the sensory as well as the motor, which never happens in myelitis of the anterior horn; there is usually also the retraction of the head characteristic of meningitis; and the paralysis is almost invariably hemiplegic in distribution,

while hemiplegia is extremely infrequent in myelitis of the anterior horn, and this hemiplegic paralysis is never succeeded by atrophy, except from years of disease.

The ordinary neuritis of a mixed nerve generally causes considerable and continuous pain, œdema, and hot and glossy skin; and the chronic form is distinguishable by the disproportion between the pain and the paralysis and atrophy, as well as often by the fact that it is confined to one nerve-trunk, or even one branch of a nerve-trunk. In multiple neuritis, differentiation is made easy by the infectious history, in some cases the malarious neighborhood, the continuous pains in both lower extremities, the steady progress within a week or two of the bilateral paralysis and atrophy, at the same time that the painful symptoms persist.

The extensor paralysis of lead palsy, conjoined with the lead line on the gums, colic, and history of exposures to lead, ought never to permit of confusion in diagnosis.

Atrophy from joint disease is too easily recognized to need more than a mere mention.

It is only necessary to call attention to the possibility of hysteria simulating a myelitis of the anterior horn, because the lack of the atrophy and of the electrical alterations and the presence of an unimpaired tendon reflex will demonstrate that the anterior cornua are not affected, even though there may be some doubt as to the existence of a motor paralysis.

Prognosis.—The prognosis of these different forms of disease of the neuro-muscular apparatus varies. In myelitis of the anterior horn the rule is that there is always a certain incurable residue of paralysis. In the acute forms the amount of this can not be determined for several weeks, and not for months in the chronic forms. Glosso-labio-laryngeal paralysis is always fatal. Progressive ophthalmoplegia is sometimes recovered from, though the majority of

cases are fatal. Muscular pseudo-hypertrophy is incurable. Progressive muscular atrophy usually runs a chronic course, and ends in death after years of duration; but there is a doubt at the present day as to whether the purely muscular forms are not amenable to treatment. Schultze details a remarkable cure, and I have seen great improvement in several cases in which the muscular pain was so great as to seemingly warrant the diagnosis of diffused neuritis.

Treatment.—The treatment of all the different forms of disease of the neuro-muscular apparatus is essentially the same in its general principles, except that the acute forms of myelitis of the anterior horn may be accompanied by reflex disturbances calling for special treatment—such as fever, coma, convulsions, etc. In the main, however, the therapeutic items are these: Rest, electricity, massage, and drugs.

Rest is of prime importance to a muscle or nerve degeneration from any cause whatsoever. I have never yet failed to obtain some improvement by rest in any case of muscular atrophy if there was any muscular tissue remaining. The rest should be proportioned to the extent and acuteness of the disease. In wide-spread or acute atrophy rest should be absolute in bed for weeks, or a month. In more localized disease the rest need not be so radical. In every case, however, and at every stage, it should be borne in mind that fatigue is to be avoided.

The electrical treatment should be by means of galvanic, faradaic, and static currents, and should be addressed to the spinal cord, the motor nerves, and the muscles themselves. Galvanism of the spine should be by means of large electrodes, one being placed over the upper cervical region and the other in the lower dorsal, and the duration of the application should be from five to ten minutes, and the current should vary from five to thirty milliampères. It will be

observed that I am no believer in the short applications and feeble currents that are so much vaunted by the German authorities. Patients become used to electricity as they do to any drug, with this difference—that the larger doses of electricity, unlike the larger doses of many drugs, will cause no ill effects. But the idiosyncrasy of each patient will vary in regard to the quantity of electricity that can be taken, and this must be ascertained. Usually, however, the strength will vary between the figures I have mentioned. Galvanism and faradaism should be applied to the affected nerves and muscles at what are known as the "motor points," a description of which can be found in any text-book of electricity. The motor points will indicate the nerve-trunks and the motor filaments going into the muscles, and it is precisely at these spots that galvanic and faradaic currents must be applied. Here the quantity of galvanism should be much smaller, and should vary from two to ten milliampères, care being taken not to cause any painful sensation. A gentle faradaic current will be quite sufficient, and the application should be from five to ten minutes. In some cases I have derived considerable benefit from an application of the gentle faradaic current from thirty to sixty minutes, flat sponge electrodes being fastened on to the affected muscles by means of elastic bands. Static electricity will be found of use in certain cases of greatly atrophied muscle, and, as Charcot has pointed out, contractions can sometimes be obtained by this form of electricity when it is impossible to obtain them by galvanism or faradaism.

Massage can be used with great advantage in the chronic forms, or in the chronic or later stages of the acute forms. It should always be limited in duration from five to twenty minutes, and should be very gentle, special care being taken not to gripe the muscle or irritate it mechanically in any

way. Massage is, however, a very uncertain agent, in spite of all the praise that it is nowadays the fashion to bestow upon it, and it should be carefully determined in each individual case as to whether the manipulation of the muscle is beneficial or not. In some cases it is positively harmful, and in these it should be abandoned.

Except in the acute or subacute forms of myelitis of the anterior horn and progressive ophthalmoplegia, drugs are usually of little use, and we can only employ them empirically. Iodide of potassium, ergot (either in the form of the fluid extract or in that of ergotine), and strychnine may be used, but I am bound to say that I have never seen any tangible effects from them.

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