Coccygeus. Is sometimes inserted wholly into the side of the sacrum (Quain).

Sacrococcygeus Anticus (curvator coccygis). This is the name given to a few fleshy and tendinous fibres passing from the lower part of the anterior portion of the sacrum and coccyx. It is well developed in animals with tails.

Transversus Perinæi. This muscle is a very variable one. It is occasionally absent, or so small as to be with difficulty dissected out. It is sometimes inserted either partly or wholly into the accelerator urinæ (bulbocavernosus) muscle or sphincter ani. This muscle is not infrequently fan-shaped, covering the triangular space formed by the three perineal muscles. In these cases the ischiocavernosus forms one edge of the fan. The fibres are inserted into the accelerator urinæ, central tendinous point, and sphincter ani. The muscle is occasionally double, the extra slip joining the accelerator urinæ or levator ani. Henle describes a muscular slip springing from the fascia at the lower border of the gluteus maximus, and inserted into the lower surface of the triangular ligament. In one case of absence of this muscle, the writer found the deep transverse muscle of large size.

Ischiocavernosus (erector penis). Houston has described a variety of this muscle under the name *compressor venæ dorsalis penis.* It is a slip arising in front of the ischiocavernosus and crus penis, which passes upward and forward, and is inserted with its fellow into an aponeurosis above the dorsal vein. The writer once saw this extremely well developed. In the dog and some other animals it is quite a strong muscle.

Bulbocavernosus (accelerator urinæ). This muscle is occasionally joined by the transversus perinæi. Kobelt describes the fibres which cover the most prominent part of the bulb, and which are separated from the others by a more or less distinct interspace, as the *compressor hemisphericum bulbi.* *Francis J. Shepherd.*

MUSCULAR ATROPHIES, PROGRESSIVE.—The presence of muscular atrophy at once suggests to the clinician one of two possibilities, namely:

1. The atrophy is a *symptom.* As such it may indicate injury, hemorrhage, inflammation, or new growth, affecting more or less acutely the oblongata, the spinal cord, or a peripheral nerve; or it may be one expression of joint disease and then due to reflex trophic disturbance in the cord.

2. The atrophy is a *disease.* In other words, it is sufficiently regular in its evolution and constant in its associated symptoms to merit a definite place of its own in our nosology. The scope of the present article is limited to this second group of muscular atrophies, those of the first group being treated under appropriate headings elsewhere in this work.

Our knowledge of muscular atrophy as a clinical entity dates from 1850, when Aran[1] published the first account of what we now recognize as progressive spinal muscular atrophy, although he considered it a disease of the muscles primarily. The disease was elaborated upon by Duchenne a few years later, whence the name "Aran-Duchenne Disease."[10]

The muscular atrophies which are classed as distinct diseases are divisible pathologically into two types, namely:

1. The myopathies or progressive muscular dystrophies; also known as "idiopathic" muscular atrophies, which are characterized by slow premature dissolution of muscle fibres from inherent vital defect. This is a long-recognized tissue condition for which Gowers has recently given us the convenient and expressive term "abiotrophy."[2]

Abiotrophy of the myon would be a concise statement of the pathologico-anatomic status of this group.

2. The myelopathies or "spinal muscular atrophies," characterized pathologically by the same process (premature dissolution) in the spinal motor nerve elements (anterior horn cells or lower motor neurons). Since, however, these same changes often occur also in the cerebral motor neurons (pyramidal motor cells) or in other cases are apparently limited to the peripheral nerves, a more comprehensive designation would be *neuronic muscular atrophy. Abiotrophy of the motor neurons,* therefore, would express the pathologico-anatomic nature of this group.

While this classification serves to define the great majority of cases, a series of mixed forms or so-called "connecting links" between the two main groups is becoming numerous in the literature as experience in their recognition increases, *e.g.*, cases which present symptoms of myopathy and myelopathy combined. These serve to illustrate the anatomical and physiological fact now well recognized, that the entire motor tract from the cortex cerebri to cord and from cord to muscle fibre constitutes a continuous functionating unit, and cannot suffer long in one part without in some degree impairing others.

There seems no good reason, however, to the writer for the use of the term "connecting link" for these cases. The coincident or consecutive involvement of one more segment of the motor tract is all that is necessary to the evolution of these mixed forms, and this may be reasonably postulated in any given case.

Accepting the pathological grouping into myopathic and neuronic, therefore, as the best at present available, we proceed in the order mentioned to consider the individual diseases in each group. The accompanying diagram shows at a glance the anatomical location of the pathological process in the several clinical types (Fig. 3460).

The myopathies or primary atrophies are divided clinically into several "types," somewhat arbitrarily perhaps, since there are good reasons for the view held by many that they are all due to the same pathological processes, differing mainly in location. An exception to this statement, however, must be made in the case of the "pseudo-hypertrophy," which is a prominent feature in one form.

They are all characterized, moreover, by certain clinical features in common, of which the chief are:

1. Hereditary or familial tendency.
2. Onset before puberty.
3. Preponderance in the male sex.
4. Loss of myotatic irritability, and in consequence loss of "tendon reflexes."
5. Electrical changes of reaction of *quantitative* character (diminished response to galvanism and faradism), and absence of typical R. D.

The recognized types of myopathy are:

A. Pseudo-hypertrophic Muscular Atrophy.—*Causation.* The disease begins in childhood; in two-thirds of the cases before the sixth year (Gowers). Heredity is traceable in three-fifths of the cases (Dana). The hereditary influence is strongest through the mother's side, though the *male members* of the family are more frequently affected. Church explains the transmission by the female members of affected families by the fact that the disease renders the males impotent.

The disease is frequently preceded by some acute infection—diphtheria for instance, which probably favors its onset in those already predisposed.

Symptoms.—Weakness in the legs of gradual onset, accompanied by a "waddling" gait and frequent stumbling without evident cause are the earliest symptoms. These are usually noticed about the fifth year and are often attributed to carelessness or stupidity on the part of the child. Later, a noticeable enlargement (pseudo-hypertrophy) of the leg muscles, especially of those of the calves, appears. This may extend to the thighs and gluteal muscles; and the infraspinati are also frequently enlarged. The enlargement of muscles may be slight in some cases, but even in these an undue firmness with lack of elasticity is noticeable on palpation. The shoulder girdle muscles are affected later, while those of the face, forearms, hands, and feet escape for a long time, but are probably affected eventually in most cases that survive a sufficient length of time. Thus in two cases, brothers, aged four-

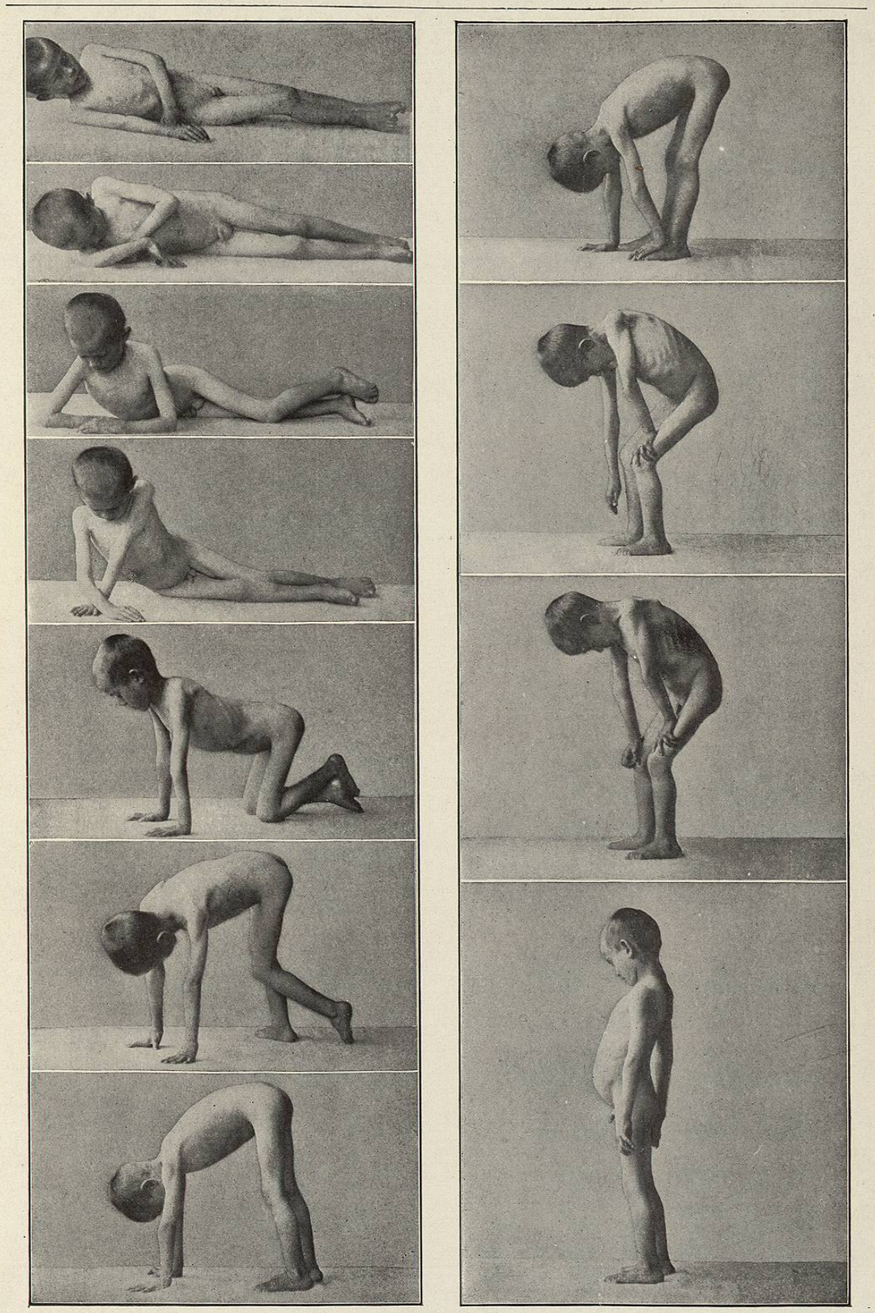
EXPLANATION OF
PLATE D.

EXPLANATION OF PLATE D.

Serial kinetographs illustrating the characteristic method of arising from the recumbent position in progressive muscular dystrophy, the patient "climbing up his own legs." (From Curschmann.)

Beginning at the top of left-hand column the figures are to be read downward; then begin at top of right-hand column. The successive utilization of the muscles of the upper extremities to assist the more atrophied ones of the legs and back is evident.

The weakness of the back muscles is also well indicated by the drooping head, counterbalanced by the lordosis. The figures represent a case of myopathy with little if any pseudo-hypertrophy.



PROGRESSIVE MUSCULAR DYSTROPHY
(CURSCHMANN)

teen and nineteen, seen recently by the writer, through the kindness of Dr. W. E. Lewis of Cincinnati, one presented marked weakness of facial muscles, and the occipitofrontalis could not be made to act at all, though the patient could frown at command. In this same patient as well as his brother, aged nineteen, the hands were markedly involved, the grasp registering by the dynamometer only nine (right hand) and ten (left) in the younger; and nineteen (right) and fifteen (left) in the elder, both showing a reduction in power estimated by me at seventy-five per cent. or more. Moreover, in the elder of these brothers there was marked wasting of the thenar group of right hand, as well as *fibrillary twitching*, though both cases were typical myopathies in their development and most other respects.

Peculiarities of posture and gait due to the muscular weakness are developed in time. Lordosis is commonly present when the patient stands. The gait is waddling and clumsy. Frequent falls result from the lack of muscular power and irregular control of movements, so that contusions and abrasions are frequent accompaniments of the disease. Ascending stairs is particularly difficult, and the patient arises from the prone or supine position in a characteristic manner by pushing with his hands upon the front of each thigh, to steady the legs. Thus he is said to "climb up his own legs." (See Plate D.)

While the statement is commonly made that the face is unaffected, nevertheless the facies of most of these patients, according to the writer's observation, is characteristic in some degree of defective muscular action. A blank, expressionless countenance is the rule, or again a marked senile aspect is present after the disease has progressed for a few years. In one case this facial involvement notably affected the smile, the mouth extending horizontally in a straight line, without the usual curves, a pathological "*risus sardonicus*."

In addition to lordosis, which is common, other forms of spinal curvature appear late in the disease. Contractures are also a feature in the extremities, talipes equinus often resulting therefrom.

Apparent lengthening of the neck, due to the drooping of the shoulders consequent upon weakening of the trapezii (Brissaud) is noted.

Fibrillary twitching in the affected muscles is commonly absent, but there are numerous exceptions to this rule. Notably in the two brothers just cited, the elder presents marked fibrillation in the lower portions of the pectoralis major, as well as in the thenar muscles of the right hand.

Sensation is usually unaffected. The writer has noted one case in which a constant "prickly" sensation was complained of throughout the trunk and limbs, but most accentuated on the anterior chest wall.

The tendon reflexes are progressively diminished and finally lost, as the muscular structure disappears.

The cases without pseudo-hypertrophy are separated from the preceding form by that circumstance and from each other mainly by differences in distribution of the atrophy. Two forms appear to require recognition, clinically speaking.

B. The Scapulo-humeral Form, Erb's "Juvenile Type" (Fig. 3462).—This begins as a rule between the ages of fifteen and thirty-five, though a range of from two to sixty years in ages of patients is recorded (Gowers).

The shoulder, scapular, and upper-arm muscles are first affected, though the deltoid often escapes (Gowers). The pectoralis and latissimus dorsi commonly waste in their lower portions. The supinator longus is commonly involved. As the disease progresses, the muscles generally are invaded.

Fibrillary contractions and R. D. are said to be absent.

C. The Facio-scapulo-Humeral Form, or Landouzy-Dejerine Type.—Also called the "infantile variety." In this the face is the part first attacked, but the ocular muscles and those of mastication escape. The shoulder and upper arm are invaded later.

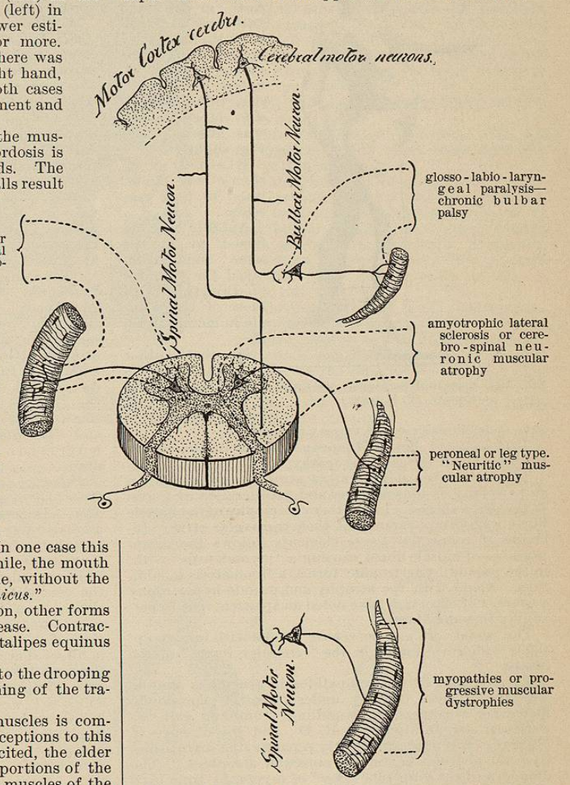


FIG. 3460.—Diagram of Motor Tract from Cortex Cerebri to Muscles, Showing Sites of Initial Degenerations in the Progressive Muscular Atrophies and Dystrophies.

The disease begins earlier as a rule than the two preceding forms, though marked exceptions to this statement are recorded.

For instance, in two cases reported by Hoppe,[3] one began at twenty-three, and was alive at fifty-five. In the other, a daughter of the first patient, the disease began at about the twenty-eighth year. These cases are also anomalous in the fact of R. D. being present as follows: In the mother's case, in the thenar and hypothenar muscles of both hands. In the daughter, in the *opponens pollicis and flexor brevis pollicis* only.

Fibrillation was not present in either case, and the mode of onset and distribution were regular in all respects.

D. Distal Form of Myopathy.—Gowers[9] applies this term to a case recently described by him, which dif-

fers from the other myopathies chiefly in the fact of the atrophy beginning in the hands and feet, although the sterno-mastoids and tongue were also involved.

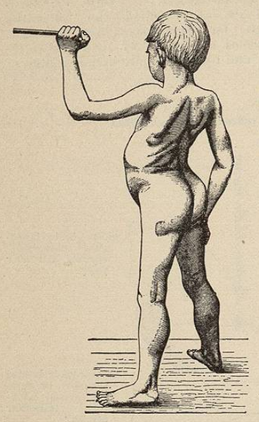


FIG. 3461.—Pseudo-Hypertrophic Muscular Atrophy. (C. L. Dana.)

The disease began at ten or twelve, and at eighteen the patient still had good power in the peroneal groups and calf muscles as well as in the thighs.

The trunk muscles apparently escaped; of the shoulder-girdle muscles the trapezius only was affected, and that slightly.

Electrical response was diminished to both currents.

Another case referred to at the same time combined the features of this type with those of the scapulo-humeral (form B).

As that author remarks, this is only another link in the chain of evidence which favors the essential unity of all types of myopathy.

Pathology and Morbid Anatomy.—These are practically the same in all forms of myopathy.

The process is a degenerative atrophy, with irritation and swelling of muscle fibres and proliferation of nuclei in the early stages. Later, there is atrophy of the muscle fibres with disappearance of their transverse striæ. Increase of connective-tissue elements follows the disappearance of muscle fibres, causing a "myosclerosis" with, in the pseudo-hypertrophic forms, a lipomatosis in addition. Along with the atrophy and pseudo-hypertrophy a number of observers have noted an apparent *true* hypertrophy in some fibres.

The spinal cord and nerves are normal with rare exceptions, which are found in the "irregular forms" of the disease.

The *diagnosis* of the myopathies in general is simple. The age of onset (usually under puberty), the slowly increasing weakness, the waddling, stumbling gait, the muscular atrophy without R. D., and the absence of fibrillary twitching sufficiently separate the myopathies from the neuronic or "spinal muscular atrophies." The diagnosis of the different "*types*" of myopathy from each other is mainly a question of the place of beginning and order of distribution of the muscular atrophy, as already noted. (See Fig. 3464.)

Prognosis and Treatment.—The disease being dependent on inherent defect in tissue vitality, the most that can be hoped for from treatment is to retard its progress and promote the comfort and general well-being of the patient. As already seen from some of the cases cited, the disease is not always incompatible with a moderately prolonged life. From ten to twenty-five years, however, expresses the usual range of duration in ordinary cases.

The usual treatment recommended consists of nutritional and tonic measures. Strychnine is spoken highly of by Gowers.

The glycerino-phosphates of lime and soda and other phosphorous compounds would seem indicated in young and developing patients.

Exercise, massage, and electricity have been recommended by most authors. Overuse of these agents may do harm. Orthopedic measures, such as division of tendons, are useful in the contractural disabilities.

The Spinal Progressive Muscular Atrophies or Myelopathies.

These are the atrophies of neuronic origin, and imply primary degenerative disease of motor neurons of the cord, or cord and brain combined.

A. Progressive Muscular Atrophy Proper, or Aran-Duchenne Disease, "Wasting Palsy."—*Definition.*—A chronic progressive disease of the spinal motor neurons (ventral horn cells), characterized by fibrillary contractions, slow atrophy, and gradually increasing weakness of voluntary muscles, with R. D.

Causation.—Direct heredity is rare. The neuropathic diathesis is presumably the chief predisposing factor. Adult age (twenty-five to forty-five), the male sex, exposure and fatigue, typhoid fever, rheumatism, gout, syphilis, and lead poisoning are credited as additional causes. The actual exciting cause, if such exists, is unknown. Why certain "systems" of neurons should die in some persons and not in others, after the operation of the preceding causes, is a problem which can be solved at present only by assuming a "potential insufficiency" in the nerve elements, an "abiotrophy" (Gowers), only fully developed by the action of some profound and persistent nutritional drain.

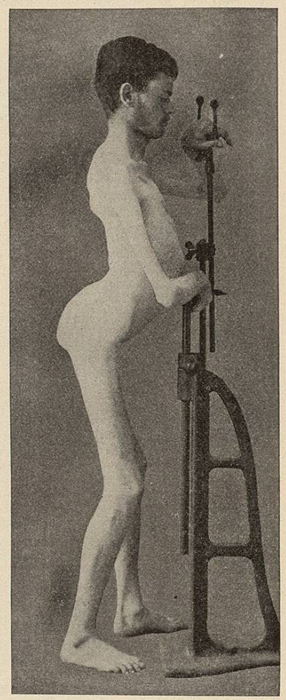


FIG. 3462.—Juvenile Type of Scapulo-Humeral Muscular Atrophy. (C. L. Dana.)

Pathology and Morbid Anatomy.—Degenerative atrophy of peripheral motor neurons (ventral horn cells) is the essential lesion. As a result of this, sections of the cord in affected areas show shrinkage and disappearance of cell bodies, with corresponding degeneration and diminution in their processes (dendrites and anterior root fibres). These changes are naturally most marked in the cervical enlargement, since the disease begins in the upper extremity in typical cases.

The posterior horns, together with the columns of Goll and Burdach, as well as the direct cerebellar tracts are normal, excepting in the rare cases of muscular atrophy supervening upon tabes dorsalis, of which the writer has seen two well-marked instances. In this case two diseases are present, according to our present nosology. It is probable that the first actual destructive changes occur in the neuron endings (muscle plates) of the affected muscles (see diagram, Fig. 3460). Following closely upon this impairment of their neurotrophic organs the muscles degenerate, fibre by fibre, into granular and fatty material, the transverse striæ disappear, while the adjacent connective tissue at first proliferates and later undergoes fibroid changes and contraction. Distention of blood-vessels is also described.

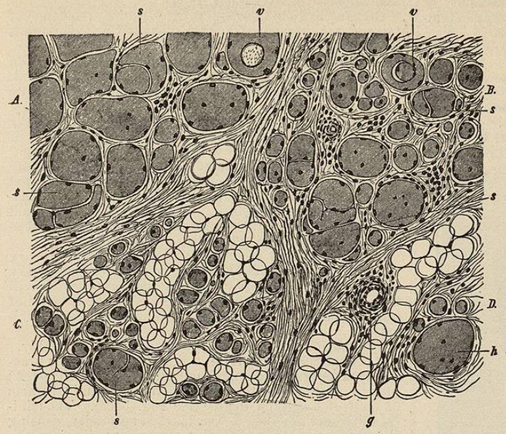


FIG. 3463. — Pathological Histology of Pseudo-Hypertrophic Muscular Dystrophy. (Partly diagrammatic; after Erb.) *A*, Hypertrophied fibres; *B*, mixture of hypertrophy and atrophy; *C*, *D*, atrophy and fatty deposit: *v*, vacuolization; *s*, splitting of fibres; *h*, hypertrophy of fibres; *g*, thickened blood-vessel.

The pyramidal tracts of the cord are usually affected in some degree. In fact, so experienced an observer as Gowers[4] remarks that he has not seen a single case in which they were unaffected.

This, as the same writer aptly puts it, is "the visible expression of a tendency to decay of the whole motor path from the cortex of the brain to the muscles."

Clinical History.—The disease begins with weakness and wasting of the thenar and hypothenar muscles of one hand, usually the right. This may be preceded by dull aching pains in the corresponding limb and cervical region. Otherwise sensory changes are absent. The nutrition of the bones and skin is not affected. The wasting advances slowly to other muscle groups, notably the interossei and lumbricales, producing the characteristic longitudinal furrows between the metacarpals, especially noticeable on the back of the hand. The wrist flexors and extensors may go next, or the deltoid and biceps may be affected before the forearm. In this case, as the writer has seen, the patient may present the anomaly of a powerful grasp, with inability to abduct the arm to a right angle.

The spinal extensors are commonly affected early, producing lordosis and allowing the head to droop forward in extreme cases.

The upper portion of the trapezius escapes, which may be a diagnostic sign of importance, as between this disease and the myopathies.

The disease extending downward involves the hips and thighs, impairing locomotion. Marked weakness of the legs proper also exists, often without atrophy, but with more or less spasm and heightened muscle jerks. The explanation of this is the involvement of the upper neuron for the legs, with escape of the lower. In those cases in which the lower leg is atrophied, however, the peroneal group suffers most, thus corresponding to the same process in the "peroneal" type (to be considered later); and also to the atrophy in multiple neuritis.

This distribution contrasts with the myopathies, which affect more markedly the calf muscles.

If the disease extends upward, the upper cervical segments and the oblongata may be invaded, giving rise to respiratory and bulbar symptoms of grave import, such as dyspnœa, dysphagia, dysphonia, dysarthria, irregular heart action, glycosuria, etc. The facial muscles usually escape, but not always.

Fibrillary tremor is a characteristic symptom and is almost continuous in the affected muscles so long as any contractile substance remains. The mechanical irritability of the muscles is increased, light taps producing marked contractions.

The deep reflexes diminish gradually and finally disappear.

Electrical changes in the affected muscles are marked and constant. Early a quantitative reduction (diminished response) to both faradism and galvanism may be noted. Later, reaction of degeneration in varying degrees from simple sluggish response to complete polar reversal, *i.e.*, the muscle contracts more readily (or to a weaker current) with the anodal (positive) closure than with the cathodal (negative) closure.

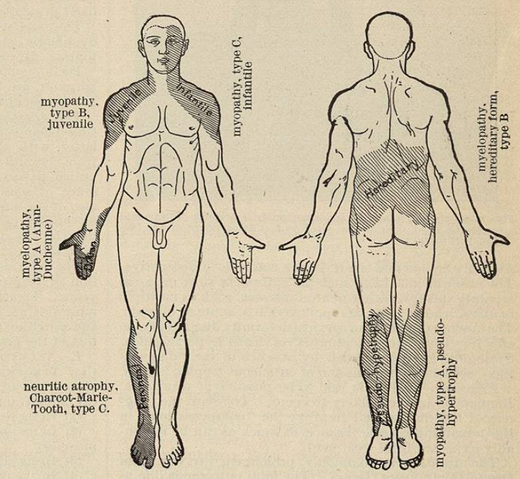


FIG. 3464.—Clinical Chart of Distribution of Muscular Atrophies. The shaded portions indicate the initial locations of muscular wasting in the different forms. (From C. L. Dana.)

The presence of this R. D. is an important diagnostic sign in excluding the myopathies or "idiopathic" atrophies.

Contractions and deformities due to unequal wasting of opposing muscles are common. The "claw-hand" or "main en griffe," lordosis, and talipes equinus are examples.

The sphincters escape as a rule.

Sexual power is commonly lost.

Eventually the patient presents an extreme degree of generalized emaciation, the "living skeleton" type.

Pulmonary complications, bulbar palsies, bedsores, and septic infection are of serious import.

Diagnosis.—The myopathies, the age (adult) at time of onset, the initial affection of the hands, the presence of

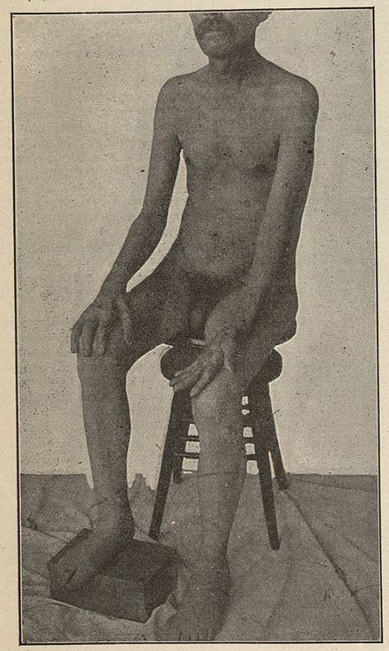


FIG. 3465.—Case of Spinal Progressive Muscular Atrophy of the Aran-Duchenne Type. (From the Neurological Department of the Cincinnati Hospital.)

fibrillary tremor and the R. D. are sufficiently distinctive. The onset in one hand and the extremely slow progress separate this from other neuron diseases, such as multiple neuritis, lead neuritis, and poliomyelitis, acute or chronic. The absence of pains and cervical deformity distinguish the affection from pachymeningitis cervicalis hypertrophica. Syringomyelia is excluded by absence of the "dissociation" sensory symptoms and of cutaneous trophic lesions.

Prognosis.—Arrest is said to be possible in rare cases. Remissions are more likely to occur. To retard the progress of the disease and improve the "compensation" in the remaining muscle tissue is as much as can be reasonably expected from treatment.

The duration is commonly prolonged, varying from two to thirty years (Dana). The fatal outcome is due to complications, pulmonary or laryngeal, resulting from involvement of the respiratory muscles.

Treatment.—Strychnine nitrate, administered hypodermically in doses gradually increased from gr. $\frac{1}{60}$ to gr. $\frac{1}{15}$, is highly commended by Gowers, who has seen the disease apparently arrested by its use.

General hygienic measures, careful diet, avoidance of fatigue are important. The phosphorus compounds should be tried.

Electricity, massage, and gymnastics should be used with caution, and any excess or fatigue should be avoided.

Mercurials and iodides are *never* indicated and may do harm.

B. HEREDITARY OR FAMILIAL TYPE OF PROGRESSIVE SPINAL MUSCULAR ATROPHY.—This is a rare form of neuronic atrophy described by Werding[5] and Hoffman.[6] It is characterized by heredity, by early onset (under two years), beginning in the hips and back; by rapid progress to complete helplessness and a fatal termination at or before the sixth year. R. D. is present, and wasting of the ventral horn cells has been found. Beevor[7] records one case which began in utero.

Diagnosis.—This is distinguished from the ordinary spinal atrophies (Aran-Duchenne type) by the hereditary element, early age, rapid progress, and absence of fibrillary twitching; from the myopathies, by the absence of hypertrophy, the presence of R. D. and the early termination.

Treatment has not influenced these cases appreciably.

C. AMYOTROPHIC LATERAL SCLEROSIS.—This is practically a progressive spinal muscular atrophy (type A) plus involvement of the upper neuron (pyramidal tracts of cord) to a marked degree (see Fig. 3460). The clinical difference consists in the marked spasticity, causing a stiff-legged gait, with toes tending to dig into the ground. There is also a rather more marked tendency to bulbar involvement, and the lower half of the face may be affected. The affection does not differ materially from type A as regards the prognosis and treatment.

D. GLOSSO-LABIO-LARYNGEAL PARALYSIS. — *Chronic Progressive Bulbar Palsy.*—This may occur primarily, or may indicate an extension upward of ordinary spinal muscular atrophy (type A). As a primary affection the degenerative process is limited to the bulbar neurons.

Patients are usually at the degenerative period of life. In many respects the disease appears to indicate a localized presenile change.

The chief *symptoms* are hoarseness, aphonia, dysarthria, dysphagia, sialorrhœa, atrophy, and fibrillation of the tongue, glycosuria, cardiac arrhythmia, and dyspnœa.

Pseudo-emotional symptoms, as causeless weeping or laughter, are frequent accompaniments. The palate reflex is absent in advanced cases.

The *diagnosis* must be made from bulbar apoplexy (small hemorrhage, thrombosis) by the sudden onset of the latter, with sensory defects of face frequently present; from cerebral lesions in the bulbar motor path (pseudo-bulbar palsy) by the sudden onset of the latter, with absence of sensory defects, of atrophy of the tongue, and also of accompanying hemiplegic symptoms; from asthenic bulbar palsy (bulbar palsy without anatomical findings) by the absence in the latter of muscular atrophy and twitching and by the frequent remissions which occur.

The progress is toward a fatal termination from inanition or respiratory complications in from one to seven years. Treatment is similar to that of other neuronic muscular atrophies—viz., rest and attention to hygiene and nutrition. Electrical treatment may add to the comfort of the patient and prolong life.

E. NEURITIC OR LEG TYPE OF PROGRESSIVE MUSCULAR ATROPHY, CHARCOT-MARIE-TOOTH DISEASE.—This variety of the "neuronic atrophies" differs from the typical (spinal) form (type A):

(1) In its hereditary character.

(2) In its onset in the legs and feet (peroneal group of muscles).

(3) In its slower course.

(4) In the circumstance that the prospects of prolonged life are better.

It is a rare disease in this country, Burr[8] recording but

five known cases up to 1897, including one reported by himself.

Pathologically a degenerative neuritis has been found. The disease therefore affects the distal extremities of the lower motor neurons.

Treatment is similar to that of the other neuronic forms. *F. W. Langdon.*

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[9] Gowers, Sir W. R.: A Lecture on Myopathy and a Distal Form. Brit. Med. Journal, July 12th, 1902, p. 89.

[10] Charcot, J. B.: Contribution à l'étude de l'atrophie musculaire Progressive—Type Duchehne-Aran. Paris, 1895.

This work contains a digest of the French literature on the subject, with especial reference to the clinical and pathological distinctness of the Duchenne-Aran form (type A), and the amyotrophic lateral sclerosis form (type C), which Charcot (the younger) considers well established. The work is beautifully illustrated.

MUSK.—MOSCHUS. "The dried secretion from the preputial follicles of *Moschus moschiferus* L.; Order, *Ruminantia*," U. S. P. This little animal, the so-called musk-deer, is a slender-limbed, active, and very timid inhabitant of the mountain regions of Central and Northern Asia. In its general shape it resembles a young deer. It is 60 or 80 cm. long (from two to three feet), from 40 to 60 cm. in height, with the rump higher than the shoulders, of a grayish or blackish-brown color, with long, slender ears, large dark eyes, and two long, curved tusks projecting downward from the upper jaw. The secreting organ containing musk is a large, simple sac, situated in the median line just beneath the skin of the abdomen, in front of the preputial canal (of the male; it does not exist in the female), with which its posterior wall is connected. It is of a round or oval, plano-convex shape, 6 or 7 cm. in its longest diameter, and 1 or 2 cm. or more thick. Internally, it is incompletely divided by folds and partial partitions reflected from the wall. The orifice, often double, is situated over the middle portion of the sac, and is 1 or 2 mm. in diameter. Its position is easily determined from the outside, because the hairs converge toward it and fairly grow within it. In the living animal the secretion contained in the gland is a soft solid, not much thicker than honey, of a brownish-red color, and of a most intense, offensive smell. As it dries it darkens, becomes greasily brittle, and less odorous.

The musk-deer is laboriously hunted in the central and northern provinces of China, in the countries to the north of India, and in Siberia,—in short, over a very extensive portion of Central Asia,—for its skin and musk glands. These "pods," as they are called, are cut out as soon as the animal is killed, and dried, often with the aid of artificial heat, when they are ready for the market. In this condition they are flattish, shrivelled pouches, covered on one side with hairy skin, in the centre of which is the orifice; the other side has the black appearance of dried meat. As now usually marketed, they are prepared so as quite closely to resemble a large fowl's gizzard. The contents are dryish, crumbly, breaking as they are turned out into coarse fragments (called grains), of an almost black color and penetrating odor. The very high cost of musk has led to numerous ingenious methods of adulteration. Thus, stones and other heavy minerals, hair, dried blood, fæces, etc., are sometimes introduced through the natural opening; occasionally, also, the sac is split and emptied, and then filled and dextrously sewed up again, with a mixture containing but little musk, or that of an inferior quality—Siberian musk, for example, instead of that from Tonquin. Or the musk itself, when sold in grains, may be tampered with in similar ways. Sometimes artificial pods are made and filled—the scrotum of some animal is used, or a bag is made entirely *de novo* from skin, with a bladder, or some other membrane, for the back. The geographical source is important in determining the quality of musk; that of China and its tributaries is the most fragrant and best. Its pods are rounder, of more uniform shape, and its grains more odorous and larger. That from the north of India comes in irregular sacs, and that from Siberia in larger and longer ones. The following are the characters of musk as given by the Pharmacopœia: "In irregular, crumbly, somewhat unctuous grains, dark reddish-brown, of a peculiar, penetrating, and persistent odor, and bitterish taste. It is contained in oval or roundish sacs, about one and a half to two inches (4 to 5 cm.) in diameter, on one side invested with a smoothish membrane, on the other side covered with stiff, appressed, grayish hairs, concentrically arranged around two orifices near the centre. About ten per cent. of musk is soluble in alcohol, the tincture being light brownish-yellow, and on the addition of water becoming slightly turbid. About fifty per cent. of musk is soluble in water, the solution being deep brown, faintly acid, and strongly odorous.

"When ignited with free access of air, musk gives off a peculiar, somewhat urinous odor, and leaves behind not more than eight per cent. of a grayish ash."

COMPOSITION.—Musk is a very complex substance, and consists chiefly of common animal tissues and secretions. The following synopsis is from Hager's "Pharmaceutische Praxis": *Fats, cholesterin, wax, biliary matters, mucus, albumin;* besides traces of *lactic, butyric, phosphatic, sulphuric,* etc., *acids;* traces also of *ammonia* and a *volatile oil.* The odorous principle has never been separated. It is supposed to be formed by the gradual decomposition of some of the other constituents.

ACTION AND USE.—This substance was for centuries the leading antispasmodic, and was, during most of this time, much more highly esteemed in medicine than it is at present, and it was given for numerous diseases involving disturbance of the nervous or mental faculties—*e.g.*, hysteria, epilepsy, mania, etc. In a similar manner several other odorous animal secretions—civet, castor, etc.—have been employed, but of them all musk is by far the least disgusting. Its high cost and impressive odor made it well adapted to hysterical outbreaks, for the relief of which it undoubtedly has some real value. As it is, however, it acts in these cases merely as a palliative, and as there are now a dozen or more remedies which are more certain and far cheaper (a single dose of musk, which might be repeated several times a day, costs two dollars or more), musk as a medicine may be considered as fairly out of use. It can, however, be given, if desired, in doses of about 1 gm. (gr. xv.) in pill or emulsion. A tincture (*Tinctura Moschi*, U. S. P., strength one-tenth) is official and may be used, but is mostly employed as a basis of perfumery, where the persistent or "staying" qualities of musk make it of the greatest value.

The odor of musk is considerably diminished by fennel, ergot, tannin, sour fruit juices, and some other things. Mixed with sugar it keeps very well.

W. P. Bolles.

MUSTARD.—BLACK MUSTARD, or SINAPIS NIGRA, "the seed of *Brassica nigra* (L.) Koch," and WHITE MUSTARD, or SINAPIS ALBA, "the seed of *Brassica alba* (L.) Hooker fil. (fam. *Cruciferæ*)," are thus separately official in our Pharmacopœia. Since they agree in most respects, they may be discussed together, and their differences pointed out in passing. Both are natives of Europe and Western Asia, cultivated and naturalized in most countries. Both are slender, tall, and widely spreading, weedy-looking herbs, with coarsely pinnatifid or pinnate leaves, the upper entire, and yellow flowers. The two are best distinguishable by the habit of their pods. In the black mustard these are erect on spreading