

Pseudo-hypertrophic muscular paralysis : a clinical lecture / by W. R. Gowers.

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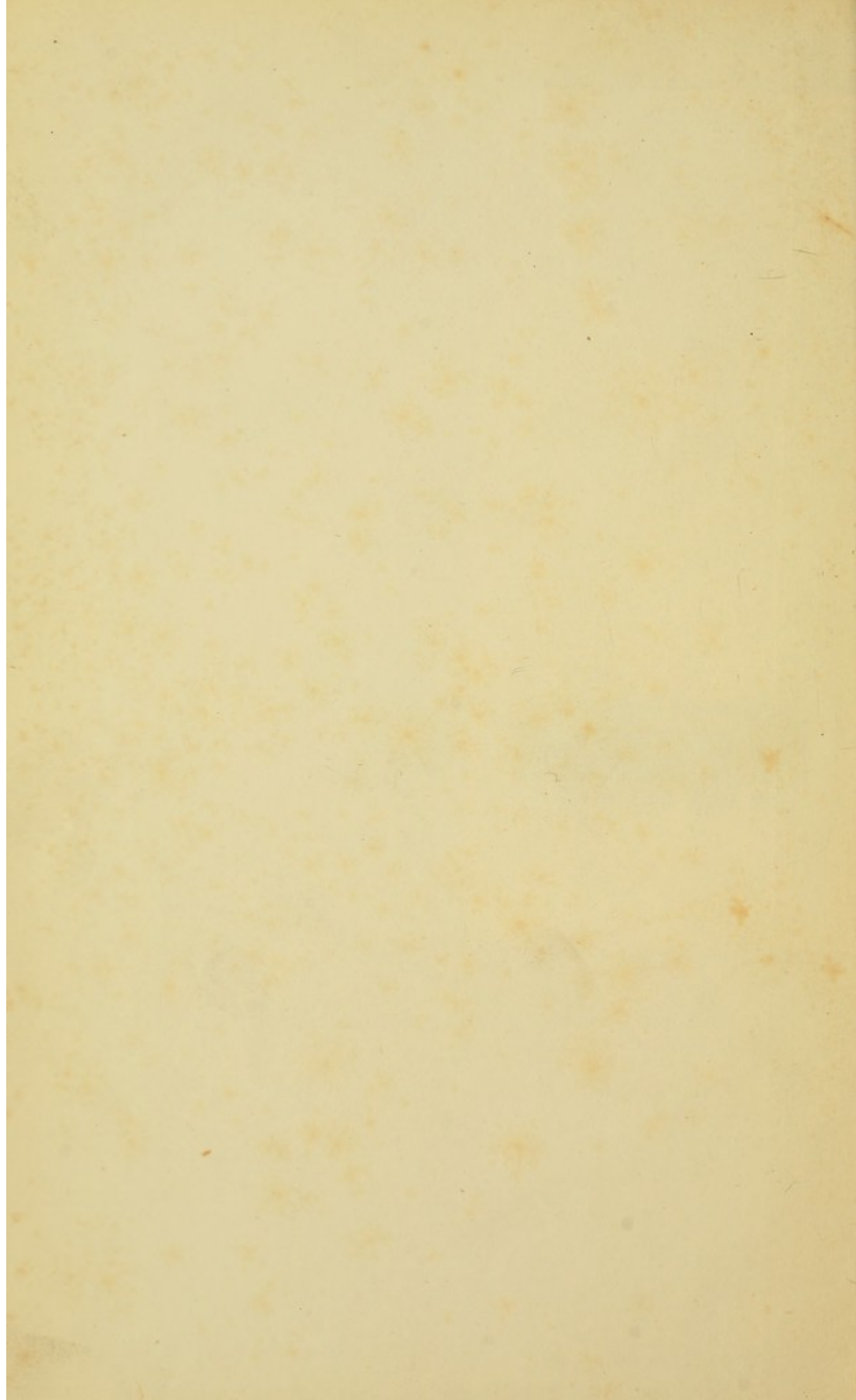
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PSEUDO - HYPERTROPHIC MUSCULAR PARALYSIS

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①
PSEUDO-HYPERTROPHIC MUSCULAR
PARALYSIS

A CLINICAL LECTURE

BY

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FOR THE PARALYSED AND EPILEPTIC



LONDON

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P R E F A C E.

THE following Lecture was delivered at the National Hospital for the Paralysed and Epileptic, to students of University College, and appeared in the "Lancet" for July, 1879. Since its publication, two other cases of the disease have come under my notice. I have described these in the Lecture (Cases 13 and 23), and have added some further details to the descriptions of the other cases. Mr. William Adams has very kindly placed at my disposal his unpublished notes of eighteen cases which have been under his care, and I am indebted to Dr. Clifford Allbutt for a note of two cases which he has seen. These twenty cases are described in an Appendix. I have also found records in medical literature of some cases which had escaped my earlier search, and others have been published since the delivery of this Lecture. These additions raise the number of original cases to forty-four, and of cases analyzed to 220. Bibliographical references to the collected cases included in the analysis are given in the Appendix, together with a brief outline

of some recorded cases of a similar muscular affection in adults, associated with signs of central disease—cases which, for reasons stated, have not been included in the analysis.

I should add that the accounts of some of the cases in the text are condensed from the notes taken by the successive Registrars to the National Hospital for the Paralysed and Epileptic, Dr. Newman, Mr. H. R. O. Sankey, Dr. Allen Sturge, and Mr. A. E. Broster.

QUEEN ANNE STREET,
November, 1879.

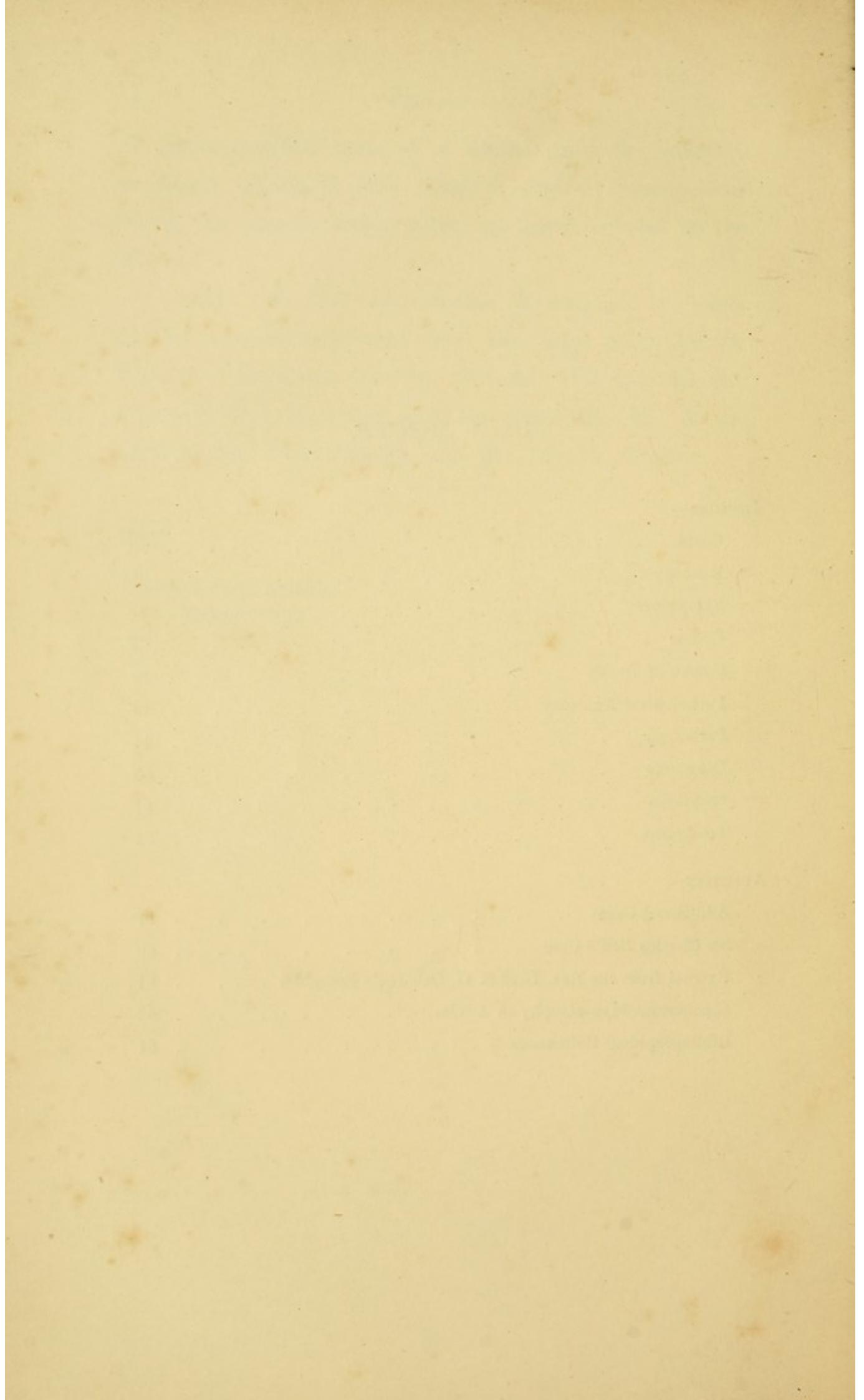
CONTENTS.

LECTURE—

	PAGE
Cases	3
Etiology	21
Symptoms	27
Course	38
Causes of Death	39
Pathological Anatomy	40
Pathology	44
Diagnosis	50
Prognosis	51
Treatment	52

APPENDIX —

Additional Cases	57
Sir Charles Bell's Case	60
Extract from the Rev. Lord S. G. Osborne's Pamphlet	61
Lipomatous Myo-atrophy in Adults	62
Bibliographical References	64



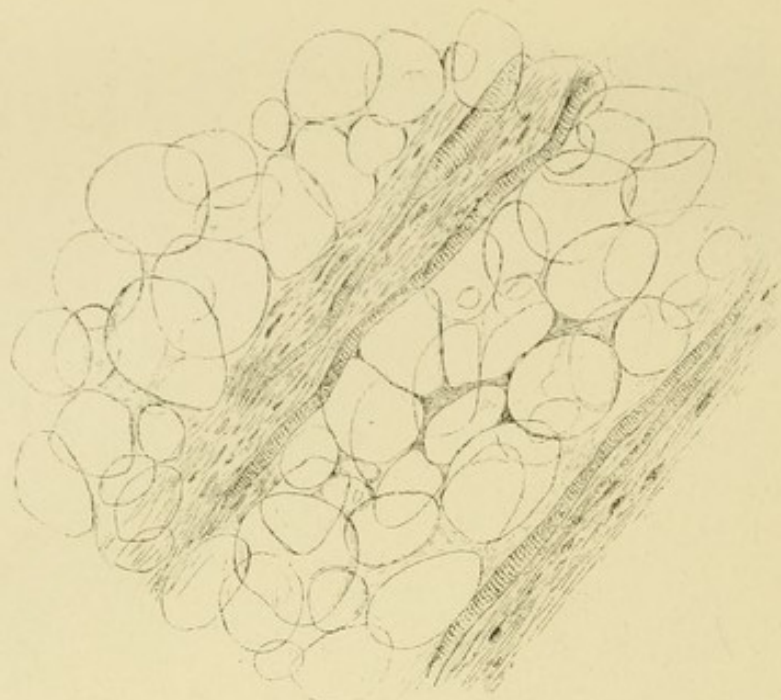


Fig 1

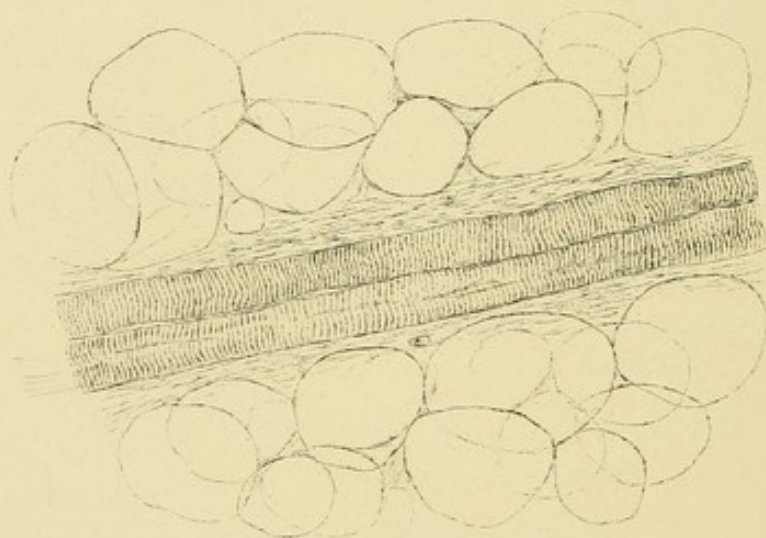


Fig 2.

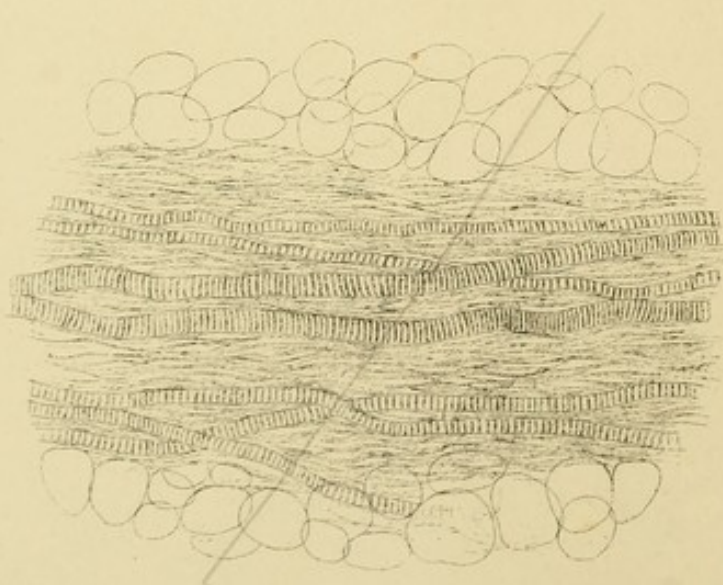


Fig 3.

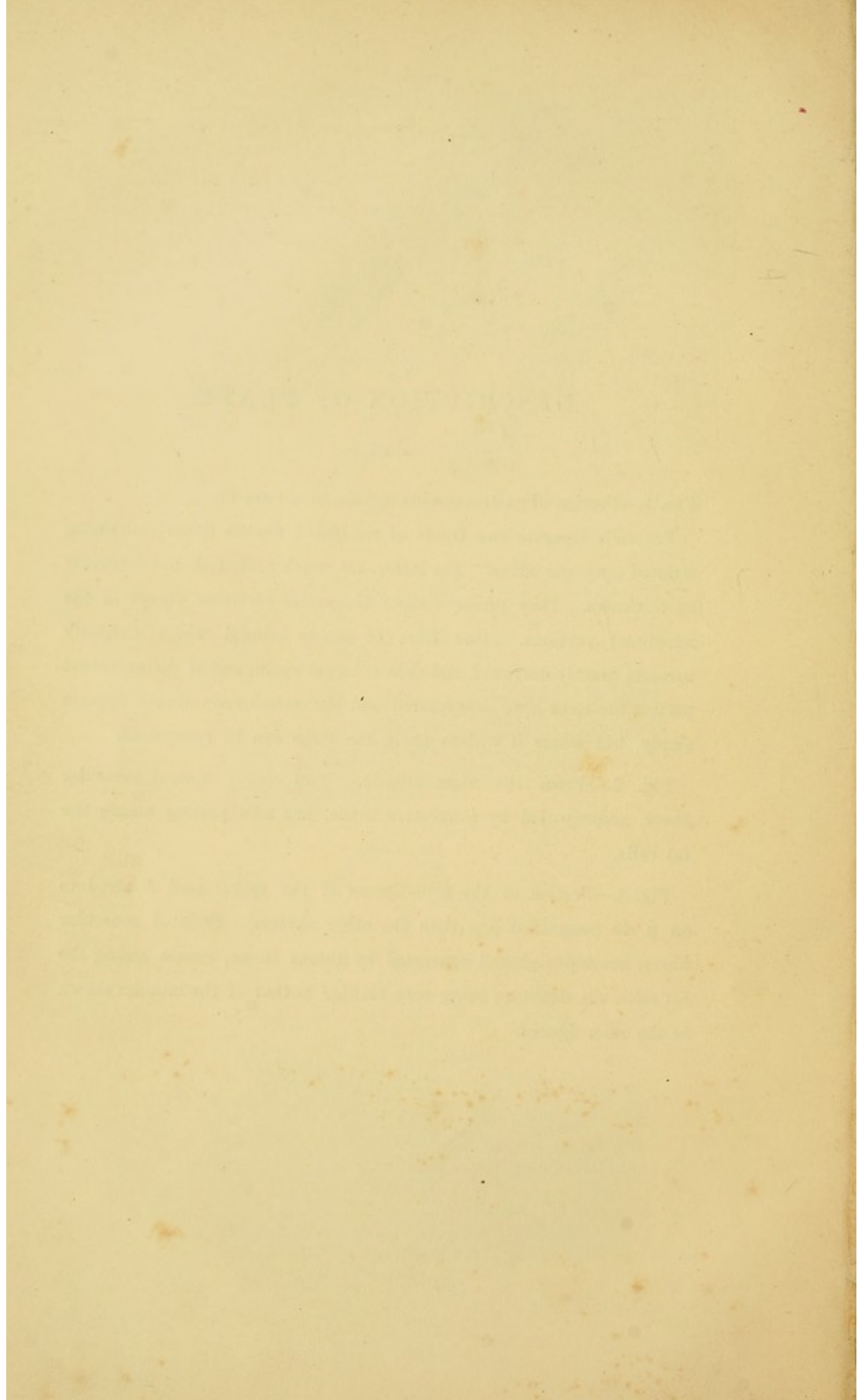
DESCRIPTION OF PLATE.

FIG. 1.—Section of gastrocnemius muscle from Case 17.

Fat cells separate two tracts of nucleated fibrous tissue containing striated muscular fibres. The latter are much narrowed and irregular in thickness. They preserve their transverse striation except in the narrowest portions. One fibre of nearly normal width suddenly becomes greatly narrowed and then enlarges again, and at the narrowest portion the striæ have disappeared and the sarcolemma sheath appears empty, but where it widens again the striæ can be recognized.

FIG. 2.—From the same muscle. Two nearly normal muscular fibres, accompanied by connective tissue, are seen passing among the fat cells.

FIG. 3.—Section of the myo-lipoma of the spinal cord referred to on p. 48 (magnified less than the other figures). Striated muscular fibres, accompanied and separated by fibrous tissue, course among the fat cells, the structure being very similar to that of the muscles shown in the other figures.



CLINICAL LECTURE

ON

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS

GENTLEMEN,—A few months ago I showed to some of you two children who were suffering from the remarkable disease commonly known as “pseudo-hypertrophic muscular paralysis.” Having the opportunity of showing you to-day two other cases of the same disease, I propose to direct your attention to its chief features and clinical history, as exemplified by these four cases and by twenty others which have come, directly or indirectly, under my notice.

The disease is one of the most interesting, and at the same time most sad, of all those with which we have to deal: interesting on account of its peculiar features and mysterious nature; sad on account of our powerlessness to influence its course, except in a very slight degree, and on account of the conditions in which it occurs. It is a disease of early life and of early growth. Manifesting itself commonly at the transition from infancy to childhood, it develops with the child's development, grows with his growth—so that every increase in stature means an increase in weakness, and each year takes him a step further on the road to a helpless infirmity, and in most cases to an early and inevitable death.

These facts do not render its study a matter of less importance. Because we can do little by our treatment, it is not the less necessary to be able to recognize the disease when it occurs, and to know the little, and to know how little,

we can do. Moreover, regarding every disease now incurable we may entertain the hope—faint it may be with respect to some, stronger in the case of others—that our powerlessness may not be permanent, and that we, or those who come after us, may be able to speak in very different terms.

Many of you are probably aware that the discovery of the disease is of comparatively recent date. A remarkable series of cases were published by Dr. Meryon in 1852, and, although a few probable examples had been described before—as by Sir Charles Bell in 1830,* by two Italian observers in 1838,† and a well-marked case by the late Mr. Partridge in 1847,‡—Dr. Meryon was the first to call attention to it as presenting similar features in many cases. He was of opinion, however, that the disease was identical with progressive muscular atrophy (Cruveilhier's atrophy). The same mistake was made by a German observer, Oppenheim, who, in a thesis on progressive muscular atrophy, described, in 1855, a well-marked group of cases. Dr. Meryon only alluded incidentally to the feature, the enlargement of the muscles, which was seized upon as the most salient characteristic of the disease by the distinguished French physician, Duchenne, in an original description published in 1861.§ The enlargement of muscles is, however, in degree and seat, a variable feature of the disease, and this may account for the fact that it received little notice from Dr. Meryon. Duchenne, indeed, at first denied that Meryon's cases were of the same nature as his own, but most other writers on the disease have recognized their identity, and it seems to me undoubted.¶ The same writer, in an able monograph,

* See Appendix.

† For this and other references, see the bibliography appended.

‡ Partridge: "Lond. Med. Gaz." 1847, p. 244; Report of Path. Soc.; not published in the Pathological Transactions. Some writers have referred to a case as recorded by Mr. Stanley in the "Med. Gaz." for Feb. 1840. The case referred to is, however, one of locomotor ataxy.

§ Duchenne states in his "Electrisation Localisée," 2nd edition, that his first case was observed in 1838, and the observation has thus been quoted. A reference, however, to his papers on the disease in the "Archives Générales de Médecine" will show that "1838" is a misprint for "1858."

¶ See Note 2, appended.

described, in 1868, thirteen cases, and up to the present time about 170 unequivocal cases have been described or mentioned by various observers.* In this country cases have been described especially by Mr. Wm. Adams, Dr. Langdon Down, Dr. Ord, Mr. Kesteven, Drs. Russell and Balthazar Foster (of Birmingham), Dr. Barlow (of Manchester), and Dr. Davidson (of Liverpool.)

The name "pseudo-hypertrophic muscular paralysis" has been proposed by Duchenne, and, on account of its rough aptness, it has received general acceptance, although a more exact pathological term was suggested by him in "myo-sclerotic paralysis," and has been sought by others in "progressive muscular sclerosis" (Jaccoud) and "atrophia musculorum lipomatosa" (Seidel), as well as in other names with which I need not trouble you, since their use has remained, and is likely to remain, limited to their authors. Probably the best pathological designation is the last, which may be rendered in English as "lipomatous myo-atrophy."

In the lad before you (David W—), who is nine years of age, the first thing which should attract your attention is his peculiar attitude and gait. He stands, as you see, with his shoulders and head thrown back and legs apart, as if balancing himself, but without unsteadiness, although a slight impulse will throw him down. This backward carriage of the shoulders is apparently due to the remarkable curvature of the spine in the lumbar region, with the concavity backwards—"lordosis," as it has been termed. This curve disappears when he sits down; then, indeed, there is a slight curve in the opposite direction, convexity backwards. The upper limbs are thin, but he can move them freely in all directions. His thighs are thin, in front especially. His calves are

* A collection of eighty cases was published in 1873 by Friedreich ("Prog. Muskel Atrophie"), and recently ninety-four cases have been collected by Möbius ("Ueber Hered. Nervenkrank." Volkmann's Series, 1879). Both of these authors include many cases which I think ought not, on clinical and pathological grounds, to be classed with those which form the subject of the present lecture. (See Appendix, Note 3.)

large, although scarcely larger than would be natural for a well-developed boy of the same age; they are, however, remarkably firm, and the muscles in front of the leg are also large and firm. On looking closely you may see that he has a little difficulty in bringing his heels down upon the ground, and that the Achilles tendons are very tense. Next observe his gait as he walks across the room. The balancing character of his attitude when standing is increased when he walks. He moves his body from side to side, and swings his arms, at each step. When he runs, this character is still more marked. And now we will put him on the ground. You see that he is quite unable to rise without assistance. If a little aid be afforded him he helps himself in a very peculiar way—by putting his hands upon his knees, and then grasping his thighs higher and higher, and so by (as has been said) climbing up his thighs, he apparently pushes his trunk up. I call your attention to this action, placing the hands on the knees in rising, because it is probably pathognomonic. Attention was called to it by Duchenne, and I have never seen it absent in a case so long as the patient possessed the necessary muscular power. I have never seen it in any other disease, and every doubtful case in which it was present ultimately proved to be an example of the affection. Its diagnostic importance is thus very great.

If we examine the power of the legs, as the boy lies, we find that all the muscles, even the calf muscles, which are so firm, are weak. The extensors of the knee and of the hip are especially weak, the former being, as I have said, noticeably wasted. The flexors of the hip are also feeble. The muscles of the back are wasted, the angles of the scapulæ project, and the serrati act imperfectly when the arms are put forward, and the latissimi dorsi and lower parts of the pectorals are almost gone. Other characteristics are chiefly negative. The lad's intellect is clear and sharp. We shall search in vain for any affection of sensibility, general or special. His sphincters are unaffected; there is no tenderness of the

spine and no trace of general rigidity of limbs. If we passively flex the foot we find, however, the movement opposed when the ankle-joint is brought to a right angle by the tightness of the tendo Achillis, which prevents the heel coming well on to the ground when he stands. We cannot obtain by this movement the clonic spasm which, as I have often shown you, is to be obtained in many forms of weakness of the lower limbs. One negative point is, however, worthy of note. If we set the lad on a chair and tap the patellar tendon, we find there is no trace of the jerk of the leg, which, as you know, usually occurs in healthy subjects. The boy's history is this. As a baby he was regarded as healthy, but was remarkably stout, so that his mother did not like to put him on his legs early, and he did not walk till he was eighteen months old. Soon after he began to walk a peculiarity in his gait was noticed, and this increased until, a few years ago, the difficulty in rising from the ground was observed, which has since increased to inability.

A brother of this lad is affected in the same way. I show you a series of photographs of him taken at different ages. In the first we see him as a child of five or six, with legs scarcely larger than other children's. In the second, at eight or nine, the calves are distinctly large, and, as another photograph shows, he placed his hands on his knees for help in rising from the ground. He has now become weaker, is unable to stand, with contracted ankles and wasted limbs. There is no other instance of the disease in the family, which consists of two other boys, aged eight and two, both said to be healthy.

Before showing you the second patient I should like to recall to your recollection the two cases I showed you a few months ago. They were, as you remember, two brothers, named S——, aged respectively four and seven years. I show you now their photographs.

CASE 3. That of the youngest, Harvey S—— (Fig. 1), would not suggest to you the idea of disease. There is no obvious muscular wasting or enlargement, the calves being

—right 8, and left $7\frac{1}{2}$ inches in circumference. They were, however, hard, and, as you may remember, his movements were greatly impaired. He could only just succeed in rising from the floor, getting his toes upon the ground and his hands under him, then placing first one hand upon his knee, then the other, and so working himself up. He only

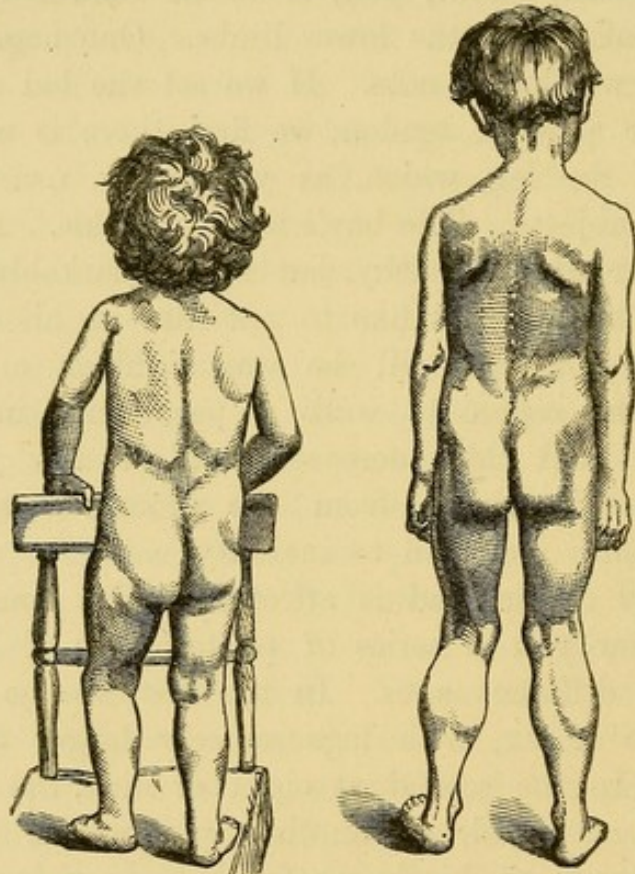


FIG. 1.

FIG. 2.

began to walk at two and a half years, and from the first manifested a difficulty in rising or going up-
 CASE 4. stairs. The other boy, William S—, aged seven (Fig. 2), presents, as his photograph indicates, very distinct enlargement of the calves. The right measured $10\frac{1}{2}$ inches and the left $10\frac{1}{4}$ inches in circumference. His thighs are small, the back thin, hollow in the lumbar region, the angles of the scapulæ prominent, the muscles of the upper limbs thin, except the deltoids which are rather large, and the infra-spinati which, in action, stand out prominently beneath the skin. The pectoral muscles are normal, but

the clavicular part of the sterno-mastoid is scarcely to be detected. You will remember his shuffling gait, and that, although he had less difficulty in rising from the ground than his younger brother, and could do so without putting his hands on his knees, yet his tendency was to do this, unless prevented. I would draw your attention to this fact, that in a slight but undoubted case of the disease, the patient exhibited merely a tendency to this action, being able to rise without it if he tried. The first indication of the disease was noted only at four years of age — a peculiarity in walking, and a tendency to fall.

CASE 5. The eldest brother, Arthur S—, aged twelve, presented, a few years back, the same difficulty in rising, and the same enlargement of the calves. He is now unable to stand, his calves are still large, but they are contracted so that the heels are drawn up and the feet are in a line with the legs. The thighs are very thin, and the hip and knee muscles possess very little power; the flexors of the knees are contracted. The muscles of his arms and back are very thin, and possess but little power, the deltoids only being of fair size. The sternocostal parts of the pectorals are gone. The muscles of the forearms are less affected, and the power of the hands is nearly normal. As soon as he was able to walk the difficulty was noticed. At four or five the enlargement of the calves was observed, and at seven they became smaller, and the weakness rapidly increased.

Besides these three brothers, a fourth, aged three years, presents a difficulty in movement, which reminds
CASE 6. his mother only too forcibly of the early symptoms presented by his brothers who are affected. In the family there are two other brothers — one aged ten, quite healthy, and one aged four, who is also believed to be perfectly healthy, although he is a twin-brother of Harvey, whose symptoms are, for his age, so marked. There are no sisters. This does not, however, exhaust the remarkable history of the family. The father

and mother are healthy. The mother had four sisters and one brother. The latter had large calves, which ultimately wasted, and he died at the age of fifteen. One of the sisters suffered from similar symptoms, and also died at the age of fifteen. The other three sisters were healthy; they married, and their children have presented no sign of the disease. Thus we have evidence of six cases in the family.

The other patient whom I have to show you is a lad (Charles Legg), fifteen years of age, who, as you see, has almost reached man's stature, being about 5 feet 6 inches—in length rather than in height,

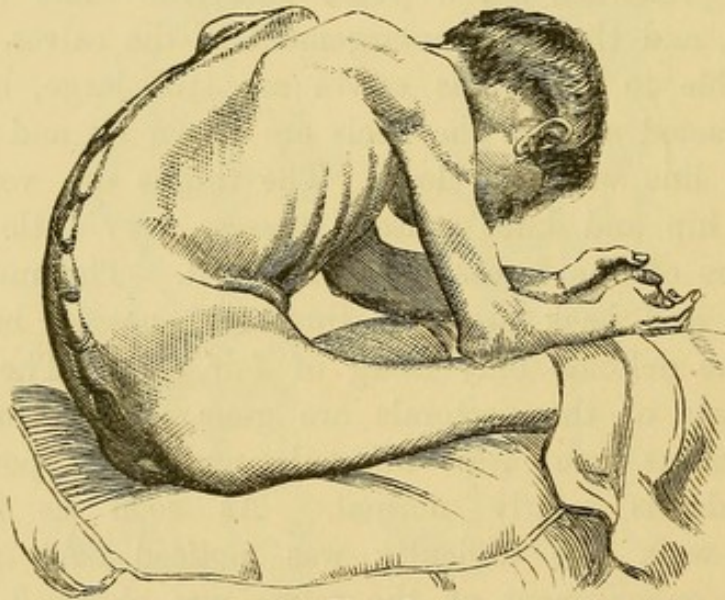


FIG. 3.

for he can no longer stand, and presents a painful extremity of helplessness. Except "weak knees," no symptoms were noticed till he was seven years of age, when difficulty in getting up attracted attention, and this has since steadily increased. Two years ago he was able to stand, but rose from his knees in the characteristic manner. His arms were thin and weak, and there was marked atrophy of the muscles of the back. The calves were unduly large: right $11\frac{5}{8}$ inches, left 11 inches in circumference. He lost the power of standing at Christmas last. Now the extreme wasting and weakness of the back at once arrest attention. As he sits his lumbar spine is

extremely curved (Fig. 3), the convexity being backwards, as in the other patients, in the opposite direction to that observed when standing. There is a slight rotation of the spine, and the wasting of the extensors is so great that you can feel the anterior surfaces of the transverse processes, and even the bodies of the vertebræ on the left side. This curve is solely dependent on the muscular weakness, and disappears if the boy is raised by the arms. His latissimi, rhomboids, and serrati are almost gone, and the consequent mobility of the scapulæ is extreme, so that the weight of the arm rotates them and brings the inferior angles in contact. The trapezii are also much wasted and feeble, and his head readily falls forward. The lower parts of the greater pectorals are gone, and even the upper parts are thin. The deltoids are of moderate size; their power is somewhat impaired. The arm muscles, however, are, although thin, not extremely wasted. Those of the upper arm are weak, the biceps smaller and weaker than the triceps, but those of the hand and forearm possess fair power. The buttocks are thin, and there is very little power of flexing or extending the hip-joints. The right thigh is thin, but in the left the vasti are distinctly enlarged in their lower parts. The knees can be flexed or extended, but with little force. The calves are not now large or hard; they possess moderate power, and are not much contracted; the ankle-joint can be flexed to a right angle. He is unable to stand.* On the left side of the neck and trunk there are some peculiar spots of warty growth following the course of certain nerves.† They are not, however, increasing, and probably cannot be associated with the disease.

The photograph I now show you (Fig. 4) represents
CASE 10. a boy (Edward Gloster) who, at the age of fourteen, was under my care here in 1874, in a still more advanced stage of the affection. He was one of a

* Under treatment, rest, tonics, etc., subsequently to the delivery of this lecture, he again recovered the power of standing and walking.

† A form of local ichthyosis, according to Dr. Radcliffe Crocker, who kindly examined them.

family of eight, of whom no other member is affected. He never walked well after an attack of small-pox at two years of age. At six his limbs were observed to be getting thin, with the exception of the calves, which were large and increased in size, attaining their maximum at nine years of age. At ten each was 12 inches in circumference; he could stand and walk, but a slight touch would throw him down. Soon afterwards he became unable to stand, and gradually passed, in 1870, into the



FIG. 4.

condition shown in the photograph, taken in 1874 (Fig. 4). He is represented in his customary attitude, sitting on a chair, with legs deformed and evidently powerless, and with his trunk distorted, so that on the right side his ribs and iliac spine were in contact. His arms, as you see, appear extremely thin, especially the upper arms; they were, in fact, greatly wasted, and almost powerless, only the hands retaining some power of movement. His thighs were thin, and the muscles greatly atrophied. His calves, on

the other hand, were large (left $11\frac{1}{2}$ inches, right 11 inches in circumference), and appeared as if in a state of extreme contraction, the swelling of the muscle being situated much higher than normal, and being abrupt; it is, as it were, an exaggeration of the condition of normal contraction — such an exaggeration as is seen in no other disease—an extreme shortening of enlarged muscles. The effect of this shortening is seen in the position of the feet, which were in such over-extension that the dorsum of the foot, instead of forming an obtuse angle with the leg, was curved backwards, the front of the leg and foot forming a convex curve; and the semi-luxation caused, as you see in the right foot, the astragalus to project beneath the skin in front of the lower end of the tibia. There was also a marked inversion of the feet. It is an extreme degree of talipes equino-varus, but the resemblance to ordinary cases of talipes equinus is lessened by the circumstance that the boy had for long been unable to rest his feet upon the toes.

You will remember that the case we first examined presented a similar, though slighter, thinness of the upper limbs; the thighs were thin, the muscles weak, and the calves presented slight contraction, rendering it difficult to get the heel to the ground—the commencement of the various changes which have, in the case photographed, led to the extreme distortion of the feet.

In this boy, as you may see in the second photograph, there was an evident and considerable enlargement of the masseters. He was remarkably intelligent. Three months later a slight increase in the size of the calves was found (left $11\frac{1}{2}$ inches, right $11\frac{1}{4}$ inches). His weakness increased, and he died at the age of sixteen of chronic lung disease, but unfortunately a post-mortem examination was refused.

I hoped to have shown you to-day another patient
CASE 11. (Charles Hudson, aged thirteen) in the advanced stage of the disease, but his intellect is impaired, and when he came up to the hospital a week ago, his

mental excitement was so great that his mother took him back into the country. When nine years of age, in 1874, he was in the hospital for a short time under the care of Dr. Radcliffe, and then presented the waddling gait, the tendency to fall, the difficulty in rising, and the large calves, characteristic of the disease (the right calf $10\frac{1}{2}$ inches, the left 11 inches in circumference). The thighs and arms were thin, except the deltoids. He was an only child, and there was no family history of similar affection. Until six years of age, the only point which attracted notice was a heaviness of gait—he would never run—and a slight spinal curve. At seven he had bronchitis and measles, and the doctor who attended him remarked, “What handsome legs!” After this illness the calves increased in size, but the thighs became thinner and the muscular power less. During the last five years the limbs have wasted much; he has now no power over the upper limbs, except the hands; the pectorals and latissimi are almost completely atrophied, and the trapezii and deltoids partially atrophied and quite powerless; he can only maintain the head erect when it is well balanced, and on the least inclination it falls forward upon his chest. The forearms and hands are thin, but resemble rather those of a person generally emaciated than the appearance in muscular atrophy. The extensors of the spine are much wasted, and the spine curved and rotated, an upper dorsal convexity to right, and lumbar convexity to left and backwards. The thighs are thin, the calves large (each 10 inches in circumference) and greatly contracted, just as in the photograph of Case 10.

I show you a photograph of another well-marked example of the disease. It is that of a boy
CASE 12. (John Silvey) aged nine. Two other boys in the family are healthy. The patient did not walk till he was four years of age, and even then never walked properly, soon tiring. His calves are large and hard (right $10\frac{1}{2}$ inches, left $10\frac{3}{4}$ inches in circumference); the muscles in front of the leg are also large and hard; the extensors of

the knee thin and weak. Two years ago these muscles were rather large. There is no knee-reflex. His back muscles are thin, the right trapezius thinner and weaker than the left. The latissimi dorsi are almost gone, and so also is the costo-sternal portion of the pectorals. The deltoid and triceps muscles are rather large, the other muscles of the arms thin. He presents the characteristic swinging gait, and the peculiar difficulty in getting up; to assist himself he has recourse to another expedient, placing himself on "all fours" in a manner which I will describe presently.

The next case is a boy (James C——) in whom the disease commenced late, but has made very rapid progress. No history can be ascertained of nervous or muscular affection among his relations, but the patient is the only boy in the family. No difficulty in movement attracted attention until he was seven (two years before he came under my care). The first symptom noticed was that, although still able to run on level ground, he could only get up-stairs on his hands and knees, and soon afterward it was observed that he could not rise from a chair without putting his hands on his knees. The weakness increased so rapidly, that at eight he became unable to stand, and a year later he was brought here. His mental state was evidently defective, although not so defective as his aspect and taciturn manner suggested. He was unable to stand; the calves moderately large (10 inches each), very hard, and contracted, so that the feet were in talipes equinus. Faradaic irritability in the calf muscles was a little below the normal. The extensors of the knees were unduly large, but very feeble, and the flexors contracted so that complete extension was impossible; the knee-reflex was absent. The flexors of the hips were almost powerless. Sitting, the lumbar spine was curved (convexity) backwards, and the muscles on each side of it were very large and prominent. The extensors of the upper part of the spine and trapezii were thin. The infra-spinati, however, were enormous; at rest, their

most prominent parts projected five-eighths of an inch above the posterior surface of the scapulæ (Fig. 5), and when contracted they stood out like half cricket balls beneath the skin. The supra-spinati were enlarged, but not to so great a degree. The latissimi dorsi and costal portion of the greater pectorals were gone. The deltoids were of fair size, except the posterior portions which arise from the spines of the scapulæ, and these were wasted so that, on abduction of the arms, deep hollows intervened between

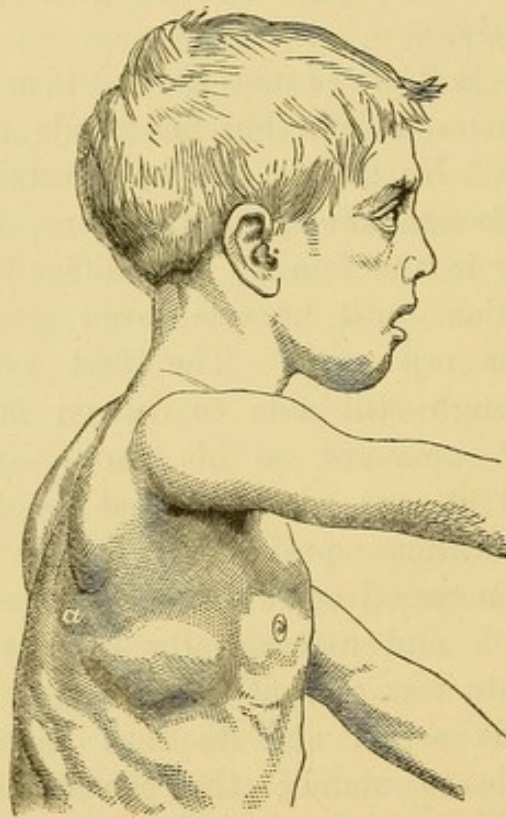


FIG. 5.

the deltoids and the large infra-spinati. The triceps muscles were large, especially the lower portions. The other muscles of the arms were a little below normal size, and feeble. He had slight incontinence of urine.

The next cases I will mention to you very briefly, because they were in their characters very similar to those

which you have seen. One was a boy, William
CASE 14. St——, thirteen years and a half, kindly sent to me in May, 1873, by Mr. William Adams. His parents

are cousins, and his mother's brother suffers from locomotor ataxy. He walked as well as other children until five, after which he gradually lost power. His calves had never attracted attention, but when he was seen, their undue size and firmness were very distinct. The right measured $10\frac{1}{2}$ inches, and the left $10\frac{5}{8}$ inches in circumference. Irritability to faradisation was present, although less than normal. The adductors were distinctly wasted. The thighs, back-muscles, and upper limbs were small, and power below par, without actual atrophy or paralysis, except that the sterno-costal parts of the great pectorals could not be detected. The hollow back was very striking. His gait was shuffling, and his difficulty and manner of rising from his knees were characteristic. Eight months later his condition was nearly the same, but his calves had lessened a little in size, and the left, in which the diminution was the greater (half an inch), had undergone distinct shortening, so that in standing the heel did not touch the ground. The arm muscles were also notably thinner, both upper arms presenting a diminution in circumference of half an inch. This lad has become progressively weaker; his legs are now contracted, the thighs and upper arms are wasted and almost powerless, but his mind is unimpaired. His age is now nineteen years and a half.

It is probable that a younger sister of this boy is the subject of the disease in a slight form. She
CASE 15. now, at the age of nine, exhibits some feebleness in her movements, and in rising from the floor "would put her hands on her knees, unless checked," just as William S——, above mentioned (Case 4).

Another case is that of a boy (Thomas H——), ten years of age, whom I had an opportunity of seeing when
CASE 16. he was a patient here a few years ago, under the care of Dr. Radcliffe. His mother's brother suffered from fits, and the lad had fits in infancy. Weakness in the legs was first noticed at seven; but since infancy his calves had been noticed to be large. At nine and a half he began to walk upon his toes, and his legs had become weaker.

For some time he had had a difficulty in holding his urine during the day: none at night. He was short for age, but plump. Mind intelligent. Calves large ($10\frac{1}{2}$ inches each in circumference), strong, contracted; extensors of knees of fair size, but feeble. His walk was the characteristic rolling gait, the feet being raised high to clear the ground. The back muscles appeared normal: the deltoids were weak, and the left small. There was no marked atrophy of the other muscles of the arms.

The next case is one an account of which has been published in the "Medico-Chirurgical Transactions."*

CASE 17. The patient was an only son; the other child, a girl, presenting no evidence of the affection.

In his early history we have the common account of late walking and a peculiar gait. The large size of the calves attracted notice as early as three years of age, and reached their maximum size at five. When about seven the weakness in walking had increased; he had great difficulty in rising from the floor, and his heels began to be drawn up, so that soon after eight years of age he ceased to be able to walk. At ten and a half his deltoids were observed to be large; the biceps, pectorales, scapular muscles, and extensors of the knee were small. Division of the Achilles tendons (by Mr. Adams) enabled him to walk for a short time with supports. Contraction of the knees came on soon after he again ceased to walk. After an attack of typhoid at fourteen his weakness increased. I saw him first at fourteen and a half. His intelligence was very great. He could not move his shoulder, elbow, hip, or knee-joints. His hands could be used fairly well. The weak muscles were wasted, but not extremely. The elbow and knee-joints were fixed in flexion by muscular contraction. The ankle-joint could be moved a little, but was in extreme talipes equinus, the progressive contraction of the calf muscles having more than compensated for the effect of the tenotomy. The thighs and calves were wasted, the latter being scarcely at all prominent, and the

* Vol. lvii. 1874, p. 247.

earlier enlargement having entirely disappeared. There was a lateral curvature of the spine. He died soon afterwards of broncho-pneumonia. The results of the post-mortem examination I will mention presently.

A well-marked instance of the disease was presented
 CASE 18. by a lad who was sent to me a few months ago by Mr. F. J. Davies, of Newport. No other child in the family or other relative was affected. The boy never walked well, although his legs and calves were much admired on account of their size. His weakness was not regarded as serious until he was seven. When I saw him he was twelve years of age, and his calves were large and firm (right 11 inches, left $11\frac{1}{4}$ inches in circumference), the extensors of the knees small and weak. The flexors of the hip also weak. Faradaic irritability diminished equally in all the affected muscles; no increase in voltaic irritability. No knee-reflex could be obtained. He had the characteristic difficulty and action in rising from the ground. His upper limbs were thin, but the only special muscular defect recognizable was the absence of the clavicular part of the sterno-mastoids, and of that part of the greater pectorals which arises beneath the first rib. The latissimi dorsi were almost gone; and the serrati were also very feeble. His intellect was unusually acute.

The last three cases mentioned were isolated instances of the disease, but the remarkable example of the affection of
 CASES 19-22. many members of the family S—— is almost paralleled by another series of cases which came under my notice very early in my medical work. They were afforded by the children of a near neighbour of the surgeon, Mr. Simpson, of Coggeshall, under whose instruction I had the advantage of commencing the study of medicine. Of a family of ten children, three girls and seven boys, the girls and three boys have been healthy; the remaining four boys, including the eldest-born and the youngest, have shown symptoms of the disease,—difficulty in rising, and enlargement of muscles, first and most in

the calves, but extending until those of the legs, arms, and body became of great size and very hard; succeeded, after a few years, by wasting. The tongue in each was large, but its increase in size was noted later than that of the other muscles. The intellect in all was unaffected. Two have died, each aged sixteen years. A third, whom I saw not long ago, is now in an advanced stage of the disease, with curved spine, wasted limbs, and enlarged tongue. In the youngest child, aged five years, the affection is still in an early stage. There is no indication of the disease in other relatives.

The cases I have mentioned hitherto have been all in early life. A few weeks ago a large, very
CASE 23. fat man, thirty-three years of age, was wheeled into the out-patients' room, complaining of weakness in the legs and inability to stand. His case appeared at first sight very unlike those we have been considering to-day. His history, however, was this: As a young child, as soon as he was able to walk, he was observed to fall frequently down without obvious cause. Subsequently he became able to walk fairly well, but he could never get up from the floor except by taking hold of some object, or else placing his hands on his knees and pushing himself up. This weakness, however, remained stationary for a long time, until about the age of twenty-two. After this it increased, but he was able to stand, and walk a little, until the age of twenty-seven; since then he has been unable to stand, and has become fat; before that he was thin. When seen, his legs were very feeble, almost all power of movement gone, a little flexion of the knee and movement of the ankle alone remaining; they were large and fat, so fat that it was impossible to say what was the state of the muscles, but there was no apparent muscular wasting (right calf $14\frac{1}{4}$ inches, left $14\frac{3}{4}$ inches). There was no contraction of the calves or talipes. No affection of sensibility or of sphincters. All active movements of the shoulder-joint were lost; nevertheless, the deltoids seemed of good size. The greater pectorals and

latissimi dorsi were, however, almost gone. He possessed slight power over the elbow-joints, and a little more over the hands and fingers, so that he was able to use them fairly well. Nevertheless, the intrinsic muscles of the hands were all conspicuously wasted and flat, to a degree, indeed, which I have never observed in any other case of the disease. This fact, together with the man's age, and the absence of any shortening of the calf muscles, suggested a doubt as to whether the case was really one of pseudo-hypertrophic paralysis. But we have seen that, in one indubitable case (Case 9)—even in the most advanced period—contraction in the calves was almost absent, and, on the other hand, this patient's history was very characteristic. In order to obtain further evidence we removed a fragment of one gastrocnemius by means of Duchenne's harpoon, and under the microscope it presented the appearances always seen in this disease—a few broad striated fibres, some undergoing granular degeneration, empty fibre sheaths, much fibrous tissue, and a large quantity of round and oval fat cells. Putting these facts together, I think there can be no doubt of the nature of the case.

You cannot fail to be struck with the fact that of the twenty-three cases I have yet mentioned, eighteen occurred in boys. To this point, as to the tendency of the affection to present itself in many children in the same family, I will return presently. One other case in a girl has, however, come under my notice. Although not a typical case, its nature is scarcely to be doubted, and even in its untypical characters, it is highly significant and instructive.

The patient, when first seen in June, 1875, by the courtesy of Sir James Paget, was fifteen years of age. She

CASE 24. was the eldest of seven children, the others being healthy, and there had been no other instance of the affection in collaterals. She had always seemed to run somewhat differently from other children, and at eleven or twelve years of age a peculiar difficulty in getting upstairs was noticed. When seen, she was a healthy-looking girl, who could walk a good distance on flat ground, but

could not rise from a chair, or from the floor, without putting her hands upon her knees. Her calves were of fair size, but scarcely unduly large (right $12\frac{3}{4}$ inches, left 13 inches). There was, however, a remarkable difference between the upper and lower portions of the muscles in front of the thighs. The upper halves were small, and their faradaic irritability was considerably lessened. The lower portions were enlarged, and on contraction stood out as if a half cricket-ball had been placed above each knee beneath the skin. (Circumference of thighs, $4\frac{1}{2}$ inches above external condyle, right 14, left $14\frac{1}{2}$ inches; 9 inches above, right $15\frac{1}{4}$, left $15\frac{5}{8}$ inches.) The irritability was lowered on the enlarged portions, although it was greater than in the smaller upper parts. The muscles were weak; those below the knee

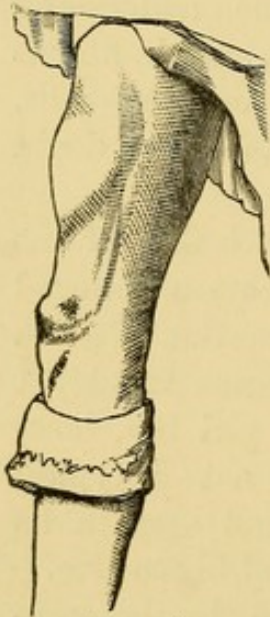


FIG. 6.

having fair power. The flexors of the hip were still weaker. The left extensors of the spine were less irritable than the right. The only condition worthy of note in the upper limbs was that the sterno-costal portions of the pectorals were not to be found. About a year later, the muscular state remaining nearly the same, a peculiar pigmentation of the skin attracted attention, observable in the temples, posterior axillary fold, and most intense on the abdomen, back, and thighs. There was one small spot within the angle of the mouth. It had very much the aspect of Addison's disease,

and Sir William Jenner, who saw the patient with me, had little doubt that such was its nature. On arsenic there was no considerable change, but subsequently, while taking phosphorus, there was a very marked diminution in the tint. Her muscular condition, however, remained nearly the same.

We may now consider in more detail some of the special characters of this remarkable disease. I have described to you nineteen cases which I have seen, and connected with those, we have had a history of five others, making in all twenty-four. In medical literature I have found records of 176 cases, and I am very much indebted to Mr. Wm. Adams for the particulars of a series of eighteen cases which have come under his notice, and to Dr. Clifford Allbutt for some facts relating to two cases under his care.* This gives us in all a series of 220 cases from which to ascertain those points in the disease regarding which statistics can supply information.

Among etiological conditions the first fact which deserves notice is the remarkable relation of the disease to sex. It commonly affects boys, very rarely girls. Of the twenty-four cases I have mentioned to you eighteen were boys, and only three were girls. And of the total of 220 cases 190 were males and 30 females, a proportion of about one female to six males (males 86·4, females 13·6 per cent.).

This remarkable tendency of the disease to affect males rather than females shows itself not only in the relative number of sufferers, but also in the degree in which individuals of each sex are affected. In many cases in girls the symptoms are slight and advance very slowly, sometimes apparently remain stationary. This is seen, for instance, in the girl whose case I have last described, and who at seventeen is still but slightly affected. And it is seen also in Case 30, appended, in which the patient at seventeen was nearly in the same state as the case I have just mentioned. The same fact is shown still more strikingly in Cases 26 and 37 in which the degree of the affection was so slight that it would have passed unnoticed had not attention been called to it by the affection of other members of the family.

The next important fact in the conditions of origin of the disease is its tendency to affect several members of the same family. In four of the examples I have given you

* The particulars are given in Appendix, Note 1.

this was noticeable, and in two the number affected was very striking, in the one four, and in the other six cases having occurred. Lutz has also described an instance of six cases in a family; but the most remarkable instance of the kind is one briefly mentioned by Dr. Meryon, in which eight brothers all died of this disease. In no case has this tendency been better marked than in the group of cases which first drew the same author's attention to the disease. In this every one of four boys of a titled family suffered from it, and died in consequence of the affection, and the title passed in consequence to a distant branch of the family.*

The disease, however, occurs very often in an isolated form, affecting only one member of a family which is otherwise healthy. Of the 220 cases, 102 were apparently isolated, and 118 were grouped in thirty-nine families. It is probable that these statistics underrate the frequency with which more than one member of a family is affected, because, in many, other members of the families were young, and may have subsequently presented symptoms of the disease, and in some instances it is not evident that the point was investigated. Of the forty-four original cases which are here described, in all of which careful inquiries were made regarding other cases, only fourteen were isolated; the remaining thirty cases were grouped in ten families. The number of families was in all twenty-four: in fourteen only one case occurred, in ten more than one. It will probably, therefore, be not far from the truth to say that more than one-half the cases are grouped, and that in these the average number affected per family is about three. The details of these cases, with which in this respect I need not trouble you, seem to show that the sex of the patients has little influence on its isolated or grouped occurrence. The

* An extraordinary series of cases of an analogous malady has been recorded by Barsickow (Inaug. Diss., Halle, 1872). In this, in two connected families, there were twenty-four cases. But the subjects were all of adult age; no children were affected, and there was no enlargement of muscles. The cases have not, therefore, been included in the above statistics.

relationship of the subjects of the disease was as follows: In eighteen families two brothers were affected, in five three brothers, in three four brothers, in one eight brothers, in two families a brother and sister, in two families two brothers and a sister. In the remaining twelve families the members affected were—one boy and his uncle; three brothers and uncle; a son, aunt, and uncle; two brothers, uncle, aunt, and great uncle; two brothers, two cousins, and two uncles; two brothers, sister, and two cousins (also brothers); four brothers, aunt, and uncle; two sisters and brother; three sisters, uncle, aunt, and cousin.

It might naturally be expected that a disease which thus evidently depends on conditions which are congenital, and not acquired, should be distinctly hereditary, and the facts I have just mentioned show that it is so in some cases. How frequently inheritance is traceable is not easy to say, because, in a large number of recorded instances, the point has evidently been imperfectly investigated. It is not enough, as has often been done, merely to ascertain the health of the parents. The parents of affected children rarely themselves present any trace of the disease. This, as Friedreich has remarked, is not surprising, since females are but rarely affected, and in males it commonly leads to death by or soon after the time of puberty. It is in the collaterals of the parents—brothers, sisters, uncles, aunts, or their children—that other cases of the disease must be sought for; or rather, I should say, of one of the parents, for the facts on record show that the disease is almost never to be heard of on the side of the father; when antecedent cases have occurred, they have almost invariably been on the side of the mother. One of the examples I have mentioned to you (the family S——, Cases 6—8) illustrates this. Four brothers were affected; the mother was healthy, but her brother and sister were both affected. Among recorded cases are several striking examples of the same fact. In a family, the history of which has been given by Dr. Russell, of Birmingham,* there were

* "Medical Times and Gazette," May 29th, 1869.

nine children—four boys and five girls. Two of the boys were affected. The mother was healthy, but two of her brothers suffered from the disease. The mother's mother was healthy, but *her* brother was almost certainly affected. Lutz has recorded a case in which a brother and sister were affected, the daughter of a second sister, and three daughters of a third sister, one of them by a different husband from the father of the other two. In a case recorded by Dr. Meryon, of three sisters, two had each one son, and the third had two sons affected with the disease. Other cases, in which a woman's children by different husbands were affected, have been recorded; one by Heller, in which a son of a woman by one husband and two sons by another, were all affected, and the mother's brother was also the subject of the disease; another by Nicolaysen, in which two half-brothers were affected. This unilateral inheritance is a very remarkable fact in the etiological conditions, and has to be taken into consideration in discussing the pathology of the disease.

But although no case has yet been recorded in which members of the father's family suffered from the disease, there is some reason to believe that, in rare cases, the father himself may be the subject of the affection in very slight degree, and may transmit it to his offspring. In one case I have mentioned (Case 23), the patient had reached manhood before the disease made the rapid advance which rendered him helpless, and in Case 36 appended the disease ran a similar course and the patient has actually married.

The father of the family first studied by Dr. Meryon was possibly the subject of the disease. I have been informed by one who knew him well,* that although able to walk well, "he never could get over a hurdle without help." Although a fearless rider he had several bad falls, falling "like a sack," and latterly he was warned to give up driving his team, from the difficulty he had in getting on to the box of the carriage. He often

* The Rev. Lord S. G. Osborne.

said, "for the life of me I could not jump over a hearth rug." *His* uncle (father's brother) also had great difficulty in getting on and off his horse, and "could never, for the life of him, get over a small ditch."

These facts (together with the more doubtful instance mentioned in Appendix, p. 59) merit attention, although they are not sufficiently conclusive to deserve much weight in comparison with the overwhelming evidence that the inheritance of the disease is, as a rule, through the mother.

Are there any indications of ancestral disease of a different character? In a few cases there has been a history of some form of paralysis, of insanity, or of intemperance, but these cases, even taken together, amount to so trifling a proportion of the whole that we are not justified in assuming the existence of any causal relationship. Nor are there any facts to show that consanguinity of the parents exerts any influence; in very few of the cases were the parents related. The age of the parents also seems to have no influence on the occurrence of the disease. Some observers have, on the evidence of a few exceptional cases, attributed the malady to inherited syphilis, but wider observation makes it doubtful whether any influence can be ascribed to this condition. In not one of the cases I have mentioned to you to-day was there any indication of parental syphilis.

The age at which the disease occurs is an etiological fact of great importance. It is a disease of early life, commencing, in the majority of cases, before six years. As a rule (to which there are, however, notable exceptions), the more severe the disease the earlier does it commence. In many cases the date of the first symptoms is difficult to determine on account of their gradual onset. In a large number, slight symptoms, such as enlargement of muscles, or slight weakness, manifesting itself perhaps only as an indisposition to run, or a readiness to fall, existed for some time, often for years, before any actual disease was suspected, and attracted little notice.

Including the cases I have described to-day, the date of the earliest symptoms is given in the records of 139 cases—123 males and 16 females. Of the former, in forty-one, or nearly a third, the first noticed indication of the disease, weakness alone, or with enlargement of muscles, coincided with the first attempts to walk, which in many cases were somewhat later than in healthy children. In one-half the male cases the disease commenced before the sixth year, and in 102, or about 75 per cent., before the tenth year. On the other hand, of the sixteen cases in girls in only one did the first manifestation of the disease coincide with the first walking, and in only three cases did it commence before the sixth year, and in only nine cases before the tenth year. Thus the disease begins in or after the tenth year in only 25 per cent. of the males, and in 50 per cent. of the females, who suffer from it. In some instances the first symptoms have presented themselves at the same age in more than one affected member of the family. This, however, is not always the case; in two or more brothers the symptoms may commence at different ages, as the cases I have described show.

In the vast majority of cases the disease appears to own no other causes than those which exist in, and are born with, the individual. Condition of life, and all that this implies, seem to exert no influence. It has, indeed, been ascribed, in some cases, to bad food, or to damp, dark dwellings. Such conditions may intensify the morbid process, but there is no evidence that they are actual causes of the disease. Indeed, my own observation would suggest that it is less common among the poor than among those who are in comfortable circumstances. Of the cases I have described, only four occurred among the very poor; in the families of the other cases there was no want of the necessaries, and even of the luxuries, of life.

In a very few cases on record the disease has followed some physical injury. It is probable that, as I shall point out presently, some of these ought not to be classed with

the cases I have described to you to-day, and the remainder only show how small an influence is to be ascribed to this cause. In one or two cases, again, the symptoms have first attracted attention after an acute disease. In two cases appended (Cases 31 and 32) the symptoms commenced after attacks of typhoid fever; in one or two other recorded cases the symptoms were first noticed after an attack of measles. More frequently a febrile disease has been followed by a rapid increase in symptoms which before were slight. The remark just made regarding the influence of bad hygienic conditions applies here also. The morbid process may first manifest itself, or may be apparently accelerated, during the weakness of convalescence from acute disease, and the stimulated tissue growth of repair may carry the patient farther on the wrong road, but an acute illness can scarcely be regarded as a cause of the affection.

We may now pass to the consideration of some of the symptoms of the disease. Its characteristic, as you will have gathered from the cases I have shown you, is a progressive alteration in the size and diminution in the power of certain muscles. The alteration in size is of two kinds—an increase and a decrease; and we cannot draw any sharp symptomatic distinction between these two changes, since each may be attended with diminished power; they may be distributed variously in different cases; and the condition of enlargement may pass into, and even be preceded by, diminution. In some rare instances every muscle in the body may be enlarged, as in a striking case figured by Duchenne.* Commonly only a few muscles are increased in size, those most uniformly enlarged being the muscles of the calf, which in the majority of cases are large and firm. Those in front of the leg are less commonly enlarged, as in the case I first showed you. The extensors of the knee are sometimes large, more commonly they are wasted; occasionally the vasti may be greatly enlarged, causing a conspicuous prominence beneath the

* "Electrisation Localisée," 3rd edition, pl. 3.

skin when they contract. In rare cases they may be the only muscles which are large, others being of normal size, or even wasted. An instance of their enlargement is presented by Case 24 (Fig. 6, p. 20). In another case (Fig. 7, Appendix, Case 35) this enlargement is the more conspicuous, because all the other muscles, even the calves, are wasted. In Case 9, just narrated, the vasti of one leg only were enlarged. The glutei are often prominent. Rarely the abdominal muscles are large; more frequently the erectors of the spine are conspicuously enlarged, forming a prominent mass on each side of the spine. The infra-spinate and deltoid muscles are often increased

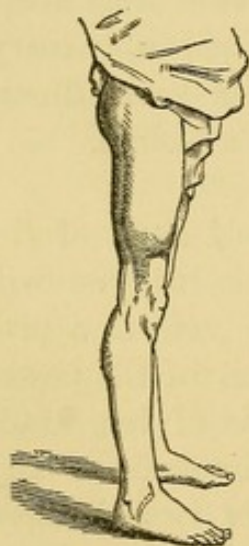


FIG. 7.

in size, the former with especial frequency, as in Case 13 (Fig. 5, p. 14). The latissimus dorsi is commonly much wasted, and so also is the lower (sterno-costal) portion of the pectoralis major, the clavicular part being much less commonly affected. The affection of these two muscles deserves special note, because it is very frequent, occurs often in cases otherwise untypical, and so is of considerable diagnostic importance. These muscles are, it may be noted, associated in action as depressors of the raised arm, and are perhaps, of all muscles of the limbs, the lowest in physiological importance.* The biceps is sometimes enlarged, the triceps much more frequently. Both muscles are, however, often wasted. The forearm muscles are rarely affected, and then only by partial wasting; very rarely by enlargement. The

* The depression of the raised arm can be effected with facility by the weight of the arm, and patients who have lost both these muscles may be unconscious of their loss. Duchenne relates the history of a blacksmith who had lost these muscles, and could nevertheless wield a heavy sledge hammer. The physiological association of the clavicular part of the pectoralis is with the serratus magnus—both concerned in bringing the arm forwards; and, as I have often seen, these two muscles are commonly associated in disease.

small muscles of the hand are scarcely ever affected. The trapezius may atrophy, rarely to a great extent. I have once or twice found the clavicular parts of the sterno-mastoids irre recognizable. The muscles of mastication, temporals and masseters, are sometimes much enlarged, as in Case 10. In a few patients the tongue is enlarged; it was so in the family B—— (Cases 19 — 22). Now and then signs of enlargement of the heart have been observed, and it has been supposed to suffer in the same way as the voluntary muscles, but recorded evidence scarcely at present establishes the fact.

When enlargement occurs it is commonly the first symptom to attract attention. Rarely a condition of diminished size has been observed to precede enlargement.* It is highly probable that the small calves of the patient Harvey S—— (Fig. 1) will become enlarged, and present in a few years the condition seen in his brother. Much more frequently enlargement, already existing, has increased under observation. At a later period the large muscles shrink, and ultimately may become unnaturally small. In one remarkable case, recorded by Dr. Hammond,† the calves and thighs, which were enlarged at six years, wasted until nine, and then presented a very rapid secondary increase in size. In the extensors of the knee, and especially in the latissimi, pectorals, and upper arm muscles, the diminution in size is usually primary. You will readily understand from this that the process which leads to enlargement, and that which causes wasting, may be so proportioned as to counterbalance one another, and muscles may be of normal size, and yet gravely diseased. Such a condition is not rare; it was presented by some of the muscles in the first case I showed you, and has probably in many cases caused the nature of the affection to be overlooked.

As I have said, the enlargement and wasting may be variously distributed. On the one hand we have cases,

* Pepper: "Philadelphia Med. Times," 1871. Mahot: "Thèse de Paris," 1877.

† "Diseases of the Nervous System," 6th edition, p. 493.

such as Duchenne's "miniature Hercules," in which every muscle is enlarged. On the other hand we have rare cases, such as that just mentioned, in which every muscle wasted with the exception of the vasti, which were enlarged. These appear to constitute a transition to still rarer cases, in which all muscles are wasted.

To return to the ordinary form of the disease. The electromotility of the affected muscles is lowered to faradization, but usually only in slight degree, except in the muscles which are greatly wasted. Usually the irritability to voltaism is the same as to faradaism; very rarely greater voltaic irritability has been observed.

The diseased muscles are weak. The amount of weakness varies, and is less in the stage of enlargement than in that of wasting. In the former it may be considerable or slight, so slight that its existence has been doubted, and it has even been said that the muscles are stronger than normal. In no case that I have seen has this been the case, and the recorded evidence does not seem satisfactory. The opinion has arisen from the strength being estimated by the resistance to passive movement—in the case of many powerful muscles a very uncertain test. In the case of the calf muscles, for instance, the force which can be applied thus is trifling compared with that exerted by the healthy muscles, and a test which approximates to their physiological exertion, such as jumping upon tip-toe, will always show them to be weaker than normal. The weakness of the extensors of the knee, and of the flexors of the hip, is usually easily recognizable, and so also, very often, is that of the extensors of the hip, glutei, the extensors of the spine, and of the arm muscles. Ultimately all power over the ankle, knee, hip, shoulder, and elbow-joints may be lost, and even the neck muscles may support the head with difficulty.

It is to the muscular weakness that the peculiar attitude is due. The muscles which are most feeble—the extensors and flexors of the hip and the extensors of the knee—are those on which depends chiefly the maintenance of equili-

brium in the upright posture, when from any cause the balance of the body is suddenly interfered with. Hence the equilibrium is maintained with difficulty; the patients habitually stand with the feet far apart, so as to enlarge the base of support. It is not that this attitude is essential; they are able to stand with the feet near together, but such a posture is insecure; and even with the feet apart, on a very slight disturbance of balance they fall. As the mother of one patient expressed it, "a breath of wind was sufficient to send him down."

The peculiar gait is due to the same cause. The oscillation in walking, by which the body is inclined from side to side, so as to bring the centre of gravity well over the foot which is upon the ground, was shown by Duchenne to depend upon the weakness of the *gluteus medius*.* This muscle normally counteracts the tendency of the pelvis at each step to incline towards the leg which is off the ground; and if the muscle is weak, the weight of the body has to be thrown further over the supporting leg than in health, and hence the oscillating gait.

The lumbar lordosis, the antero-posterior curvature of the spine, which is so conspicuous in these patients when standing, has, by Duchenne and other writers, been attributed to the weakness of the spinal extensor muscles; and Duchenne † described, as a distinction of this form of lordosis from that which results from weakness of the abdominal muscles, that the shoulders are carried so far back that a vertical line from the scapulæ falls behind the sacrum. This is true, however, only of extreme cases. In the first patient you saw, such a vertical line falls well within the sacrum. But it is, I think, doubtful whether the lordosis is mainly due to this cause. Weakness of these muscles may, as Duchenne showed, ‡ cause the shoulders to be carried far back, so as to bring the centre of gravity of the upper part of the trunk as far back

* "Phys. des Mouvements," p. 340.

† "Arch. Gén. de Méd." 1868.

‡ "Phys. des Mouvements," p. 716.

as possible; but, according to his description, the inclination backwards from this cause starts from the pelvis, which is more extended on the thigh-bones than normal. But in pseudo-hypertrophic paralysis, in the erect posture, in which posture alone the lordosis is observed, I have found that the pelvis is much less extended, its inclination forwards is much greater, than normal. This inclination is probably due to the weakness of the extensors of the hip, and the lordosis is apparently connected with it, since the lowest lumbar vertebræ share the direction of the sacrum, the weight of the abdomen falls unduly forwards, and a compensatory backward inclination of the dorsal spine is necessary to keep the centre of gravity in the normal position. When the patient sits, the pelvis rests on the ischial tuberosities, its inclination forwards ceases, and with this the lordosis disappears, and is even replaced by a convex dorso-lumbar curve, due, without doubt, to the weakness of the spinal extensors; slight when these are little affected, as in the first case I showed you; extreme when these are greatly atrophied, as in Fig. 3, p. 8. An unequal distribution of the weakness, and the habitual posture of the patient, lead to the lateral curvature so conspicuous in the later stages of the disease in most patients. In one patient (Case 11) this was associated with such a rotation that the tips of the left transverse processes of the lumbar vertebræ projected backwards as far as the spines. In all the cases I have seen, the lateral spinal curvature has been with the concavity and rotation of the spinous processes towards the right.

The weakness of the muscles of the legs also causes the peculiar difficulty in rising from the floor or a chair, and in going up-stairs, and its distribution determines the expedients to which the patient has recourse to assist himself in the movement. One of the most common of these is the custom of putting the hands on the knees (Fig. 8), apparently to push the trunk up, to help the extension of the hip-joint—an action which, as I have told you, is

practically pathognomonic of the disease. It is met with in, perhaps, no other affection; is never entirely absent in this. I have said that it is *apparently* to help the extension of the hip-joint because, although this is the common explanation, I believe it is only partially correct. We may often distinguish two actions: in one the hands are placed on the knees, and kept there; in the other they are afterwards placed on the thighs, the thigh is grasped, and the hands moved alternately higher and higher. The latter action is that which assists the extension of the

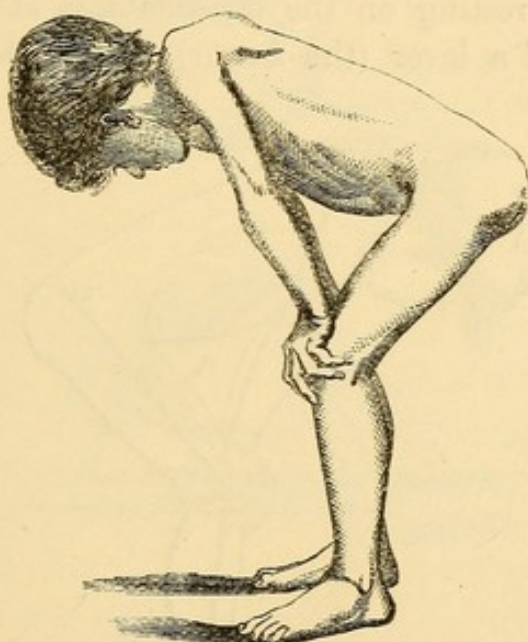


FIG. 8.

hip-joint. The former assists the extension of the knee-joint, and has very little to do with the extension of the hip. You may observe the proof of this in two ways: First, in a patient who performs both these actions it may be observed that whilst the hands are upon the knees there is little extension of the hip-joint, but the knees are being extended; then, when the knees are straight, or nearly so, he works his hands alternately up the thighs, and so gradually extends the hip, and when nearly upright his muscular power is usually enough to complete the extension. Secondly, if a patient merely puts his hands on his knees, and does not work them up the

thighs—*i.e.*, if he helps the extension of the knee, and not that of the hip—you will find, as I have several times observed, that if the knees are kept extended, he can bend his hips so as to touch the ground and rise again, without needing to touch his knees. But if he flexes his knees, he cannot straighten them without putting his hands upon them.

The reason why this action affords such help in extension of the knees is obvious on a little consideration. In rising from the ground with the knees flexed, the weight of the trunk, resting on the hip-joints, is at the extremity (Fig. 9, w) of a lever (the femur) of the third order, the

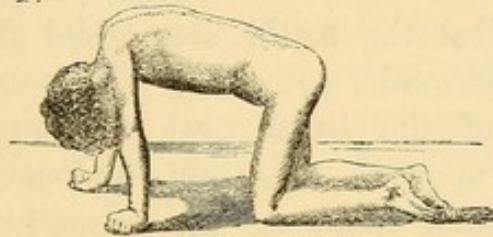


FIG. 9.

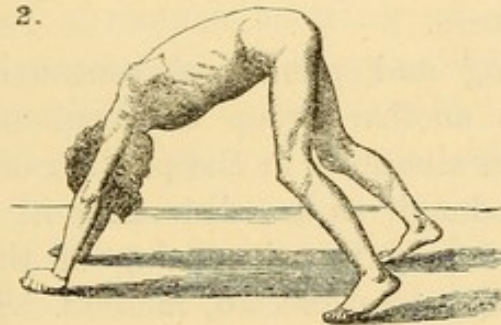
fulcrum (F) being at the knee, and the power, the contraction of the quadriceps extensor, being applied (P) between the weight and the fulcrum—*i.e.*, in the position in which it acts to least advantage. But by placing the hands on the knees—*i.e.*, on the end of the femur—a large part of the weight (the larger the more the patient bends forward) is transferred to the lever (at w) close to the fulcrum; the lever is, in so far, transformed into one of the second order, in which the weight is between the power

and the fulcrum, and the power is economized in the greatest degree. Moreover, if the patient bends down, the centre of gravity may even be carried in front of the knees, and then, if the hands grasp the knees firmly, the weight of the body, instead of being the weight to be moved, becomes a force applied to the upper end of the femur, effecting the extension of the knee without the slightest action of the quadriceps extensor, as any one may

1.



2.



3.

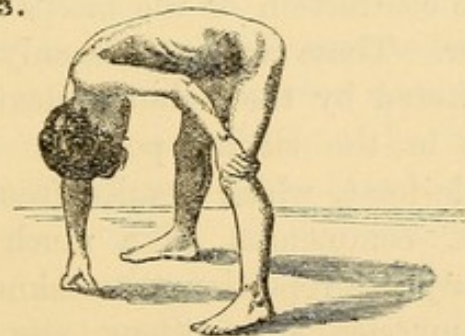


FIG. 10.

ascertain by observing the mobility of the patella in this attitude.*

* It may be observed that the action by which the extension of the hip is assisted, the "climbing up the thighs," is also similar in its mechanical effect. The weight (of the trunk) is brought between the power (applied at the shoulder-joint) and the fulcrum (at the hip) instead of being between the fulcrum and the weight, as when the extensor muscles of the hip alone act.

Some patients, however, with similar muscular weakness, have recourse to another expedient for effecting the extension of the knee-joint (Fig. 10). In getting up they first put the hands on the ground (1), then stretch out the legs behind them far apart, and, the chief weight of the trunk resting on the hands, by keeping the toes on the ground and pushing the body backwards, they manage to get the knees extended, so that the trunk is supported by the hands and feet, all placed as widely apart as possible (2). Next the hands are moved alternately along the ground backwards, so as to bring a larger portion of the weight of the trunk over the legs. Then one hand is placed upon the knee (3), and a push with this and with the other hand on the ground is sufficient to enable the extensors of the hip to bring the trunk into the upright posture.

The shortening and permanent contraction of certain muscles lead to another group of symptoms—distortions due to permanent alteration in the position of joints. Some of these are produced, as are distortions in other forms of muscular weakness, by the shortening of the less affected opponents of other, more affected, muscles. Thus the knee-joints become fixed by the contraction of the flexors, and the elbow by the contraction of the biceps when the triceps has lost all power. These contractions only occur late, and are usually facilitated by the habitual flexion of the knee and elbow-joints in the sitting posture. But the deformity at the ankle-joint, which results from contraction of the calf muscles, commences at a much earlier period, before there is any disproportionate weakness in the opponents of these muscles, and without any assistance from posture. It coincides, commonly, with the diminution in bulk of the muscle, and may be the result of the shrinking occurring in the length as well as in breadth,—a primary contraction. As a consequence of it, the patient cannot get the heels well upon the ground, and the foot cannot be flexed passively beyond a right angle. The gradual increase of the contraction results in a condition

of "talipes equinus," and the patient walks upon the toes in a very characteristic manner. In consequence of the muscular weakness which coincides, the patient is able to walk but little, and the loss of the extension involved in the act of walking permits a rapid increase in the unopposed contraction. The feet, as Fig. 4 (p. 10) shows, soon assume a posture of extreme extension, the dorsum being in a line with the front of the leg, or the two may even form a convex curve. A subluxation of the ankle-joint takes place and the articular surface of the astragalus, its anterior extremity, and that of the os calcis, form three prominences under the skin. When this reversal of the ankle takes place, the tibialis anticus can no longer act as a flexor.—The spinal deformity, as I told you, is solely the result of muscular weakness.

In some cases a marked mottling of the skin of the legs has been observed, an exaggeration of that often seen in healthy children. Attention has been called by Dr. Ord to the temperature of the calves, which has been found to be higher than that of the thighs. But in other cases the change is in the opposite direction. In several cases we have tested this, and failed to find any uniform difference.

Other nervous functions are usually unimpaired. Sensibility is, in all typical cases, unchanged. Occasionally pains are felt on use of the muscles most diseased. Reflex functions are usually unimpaired, with the exception that the patellar-tendon reflex I have found absent in several cases in which the atrophy of the extensors of the knee was considerable. Earlier in the disease it is present. The sphincters are usually unaffected. Rarely, in advanced cases, there may be some difficulty in passing urine. The lad I showed you had such difficulty for two days after a fall, and in a case described by Dr. Byrom Bramwell a similar transient condition occurred, and afterwards there was some incontinence.

In all the cases, except two, which I have mentioned to you to-day, the cerebral functions were unimpaired, and

in several the intellect was remarkably acute. In most recorded cases the mind has been unaffected. In some, however, there has been mental dulness, and even actual idiocy. Several of such cases have been published by Dr. Langdon Down. It appears, therefore, that mental defect is not part of the disease, but that the muscular affection is rather more common among children who have mental defect than it is among others. In a few cases on record there have been epileptic fits (as in Cases 16 and 25), probably, like the mental defect, the result of an associated, not of a related, cerebral disease.

In two or three cases symptoms of spinal disease, spontaneous pain, formication, and paraplegic weakness have been described. These cases occurred in adults, and differed in many important particulars from the common form of the disease, and they ought not, for reasons which I shall tell you presently, to be classed with it. The premonitory stage of the disease, which one writer* has described from these cases, has no existence in the ordinary form.

The course of the disease varies much in different cases. The first symptoms commonly occur in early childhood, the power of standing is lost at ten or twelve years, and death occurs between fourteen and eighteen. In the cases in which the first manifestation of the disease is delayed until five, six, or eight years, the condition attained at puberty is usually less advanced, and life is prolonged until nineteen or twenty years; in some cases even longer. The rule of late development and slight degree is seen conspicuously in some of the cases in girls, as in the last case narrated, and in several others which are on record; as one in which the first symptoms occurred at nine and the patient was seen at thirty-one (Peirson). In boys, exceptions to this rule are not unfrequently met with, in which the disease, appearing late, has made very rapid progress. In a case described by Dr. William Smith, for instance, the first symptoms were not noticed until five

* Berger: "Deutsch Arch. f. Klin. Med." ix. 363.

years of age, and at eight the patient had already lost the power of standing. On the other hand, in a few cases in which the first symptoms coincided with the first attempts to walk, the disease has run a slow course, as in the case of William S—— I described to you, who is now nineteen and a half years of age, and still more strikingly in Case 23. In this patient the first symptom occurred in early childhood, but power of standing was not lost until twenty-seven. Instances of this are also seen in girls, as in a case in which the first symptoms were noted at a year and a half, and the patient when seen was twenty-seven years of age. In adult life general obesity may come on, as in Cases 23 and 36.

Of thirty cases in which I have found the age at death mentioned, in twenty-four the patient died between ten and twenty—viz., in two cases at eleven, in four at thirteen, in three at fourteen, in four at fifteen, in six at sixteen, in two at seventeen, in three at nineteen. In two cases the patient died at six, in one probably, in the other certainly, of an accidental malady. In one case death occurred at twenty-one, in one at thirty, and in two at a little over forty years. (The latter three occurred in families in which other cases occurred and ended in early life.) This list includes only three females, one of whom died (of scarlet fever) at six, one at fifteen, and the other at forty-two.

The disease is not in itself fatal; death is always the result of intercurrent maladies, the occurrence or fatality of which is, however, in most cases the result of the muscular disease. The weakness and wasting of the thoracic muscles, for instance, gradually lessen the respiratory power. The patient is thereby weakened, and is rendered an easy prey for the maladies which lie in wait for the infirm. The most common cause of death, indeed, is the direct interference with the action of the lungs. As the patient becomes weaker, bronchial râles are heard through the chest, dyspnoea comes on, and is rather increased than lessened by the patient's feeble attempts to

cough. Pyrexia may be developed, and the patient dies with the signs of a chronic broncho-pneumonia, which has in many cases been found after death. In some cases a more acute lung affection occurs, a trifling cold developing, under the pulmonary conditions, into a grave disease. In a few recorded cases some other intercurrent malady has been fatal, especially measles, no doubt from the bronchitis which, as you know, often accompanies that disease. Of twenty-one cases in which the cause of death was mentioned, lung disease was the cause in fourteen cases, measles in two, an acute intestinal affection in two, laryngeal croup in one, and scarlet fever in two cases.

We may now pass to the consideration of the pathological anatomy of this remarkable disease. What, in the first place, is the change in the muscles which leads to the striking alteration in their size and power? In the case of the boy on whom I made a necropsy* (Case 17) the appearance of the muscles was very characteristic, and agreed closely with that found by other observers. On cutting into the gastrocnemius it was difficult to believe that the section was that of a muscle. Its appearance was precisely that of a fatty tumour—a yellow greasy mass of fat, in which no trace of muscular redness could be perceived. Under the microscope the resemblance to a fatty tumour was also strikingly close. Cells distended with homogeneous fat covered the field. Passing among the fat-cells, however, were narrow bands, which consisted of fibrous tissue and muscular fibres. In places a group of muscular fibres passed through the fat with little fibrous tissue (Plate Fig. 1), but, for the most part, they were accompanied by, at least, an equal bulk of fibrous tissue, containing many nuclei and elongated cells. As a rule, the muscular fibres presented no granular degeneration. Many were reduced in size, some considerably, and in places these narrow fibres lay beside those which were of normal width. Some fibres varied in diameter at different places (Plate, Fig. 2). In

* "Med.-Chir. Trans." vol. lvii. p. 247.

many of the narrow fibres the transverse striæ were farther apart than in the wider fibres. In a few, both large and small, the striæ seemed to be disappearing, fading away, without becoming granular. Some empty sarcolemma sheaths were seen, evidently such by their continuity with fibres in which the striæ were disappearing. In the deltoid and biceps the tint was red, but much paler than normal. The microscope showed the fibres to be, in places, separated by fat-cells similar to those in the gastrocnemius, and in other parts by much fibrous tissue without fat. The muscular fibres presented still greater variation in size, and a similar separation of the striæ in the narrower fibres. In the triceps there was still less fat, but much fibrous tissue.

This condition corresponds with that found by other observers (*e.g.*, Cohnheim and Charcot) who have had an opportunity of examining the muscles after death. It corresponds also, in the main, with the observations made with the histological harpoon, an ingenious instrument contrived by Duchenne for removing minute fragments of muscle from living subjects; and it shows also the room there is for fallacy in the use of this instrument. In some parts of the deltoid, for instance, the harpoon would have removed a fragment containing little fibrous tissue and much fat, and in other parts one containing much fibrous tissue and little fat. Some observers have concluded, from harpoon examinations, that the fibres may, in the early stage, present actual hypertrophy. The tearing out of a fragment of living muscle must, however, involve mechanical stimulation of the fibres: and as their shortening is unopposed, an extreme contraction of the fibres and proportionate increase in width may have given rise to the appearance which suggested hypertrophy. I cannot think, therefore, that the histological evidence of an initial hypertrophy is less dubious than is the clinical proof of this condition.

The change in the muscle, then, appears to be primarily interstitial—a growth of nucleated fibrous tissue and of fat-

cells. This growth, and especially that of fat, is the cause of the increase in the size of the muscle. The harpoon has been thought to furnish evidence that the increase in size may, in some cases, depend solely upon fibrous tissue, but this is doubtful. The muscular fibres certainly suffer secondarily to the interstitial change. They are apparently narrowed by pressure, and to this the diminution in bulk of the muscles is in part due; in part it is probably the result of the absorption of interstitial fat. The shortening of the muscles may reasonably be ascribed to the contraction of the interstitial fibrous tissue. Everywhere, as you know, fibrous tissue tends to contract in the direction of its fibres. In the muscle these new fibres are all parallel to the direction of the muscular fibres, and hence the effect of their contraction must be a shortening of the whole muscle.

What is the condition of the spinal cord in this disease? Are there changes in it, such as I have often shown you are to be found in progressive muscular atrophy? In the case in which I have just described the condition of the muscles, a careful examination of the spinal cord was made jointly by Dr. Lockhart Clarke and myself, and the conditions found were very instructive.* Changes were scattered through the entire length of the cord. In the cervical region there was "incipient disintegration" in the grey net-work of the lateral columns, adjacent to the grey substance, the white columns being healthy. Here and there, in the grey substance of the anterior and posterior cornua, was some disintegration and wasting of the intercellular matrix, especially marked around blood-vessels, and conspicuous at the bottom of the anterior median fissure, much débris and globules of myelin being accumulated in places. The anterior commissure in parts appeared as if interrupted, but this appearance was partly

* Their significance has not been always correctly estimated by those who have referred to the case. The report of the case stated the facts alone, without any inferences from them. For those given here, I am, of course, alone responsible.

due to the large spaces around the distended vessels, displacing the bundles of nerve-fibres out of the plane of the section. In the lower cervical and dorsal regions there was, in places, an appearance as of disintegration of the myelin of the fibres of the lateral and posterior columns, and there were, in some parts, products of degeneration, globules and masses of fatty matter, at the entrance of the posterior nerve-roots, and, to a much less extent, adjacent to the anterior roots. The most extensive lesion was found in the lowest part of the dorsal region, where in each lateral grey substance was an area of disintegration amounting to an actual cavity outside each posterior vesicular column, which, with the caput cornu posterioris and anterior cornu, was undamaged. A few unaltered nerve-fibres traversed the disintegrated area. In the rest of the lumbar enlargement the changes were slight, the anterior cornua being perfectly normal, except that in the lower part their processes were less distinct than elsewhere in the cord, although the bodies of the cells were normal. In the lowest part of the conus medullaris, one group of nerve-cells had disappeared.

Such, then, were the changes found; the absence of others is not less important. With the exception mentioned, the large nerve-cells of the anterior cornua were conspicuously healthy. In number, size, sharpness of outline, length and distinctness of processes, the cells correspond perfectly with those of the healthy cord, as you may see from the sections I have placed under the microscope. The slight degeneration elsewhere apparent in the anterior cornua affects only the intercellular substance. In the lateral columns, also, although in places there are signs of disintegration, there is no change comparable to the lateral sclerosis, so often present in other diseases.

Compare, now, with these the sections of the cord from cases of progressive muscular atrophy in adults. At first sight you cannot see, in the anterior cornua, a single nerve-cell. In places there are a few shrivelled remains, but practically they are all gone. This change was present

in each of six cases of progressive muscular atrophy in which I have examined the spinal cord, and the result agrees perfectly with the observations of Lockhart Clarke, Charcot, and other observers. But in this case of pseudo-hypertrophic paralysis, although the muscles of the arms were almost as wasted as in progressive muscular atrophy, the corresponding motor nerve-cells are perfectly normal. The characteristic central change of spinal myo-atrophy is, therefore, almost entirely absent in pseudo-hypertrophic paralysis.

What, then, is the significance of the lesions which were found? The degeneration at the lowest part of the dorsal cord is certainly an extensive lesion, and very difficult to explain. Its situation, however, is in a part of the grey matter the function of which is unknown; it affects the matrix rather than the larger nerve elements, and if it has any connection with the muscular disease the links of that connection are entirely unknown. In considering the significance of this and other changes in the cord, we must remember that the patient had been, like all subjects of the disease, for years helpless, with a crooked spine, unable to move, suffering from frequent attacks of pulmonary trouble, with mechanical congestion of the whole venous system, passing through acute illnesses—all conditions not unlikely to lead to accidental degenerations in the cord. Those which we found, in their scattered position and character, and their relation to vessels, were for the most part such as might be expected to arise in this way. Further, the degenerative changes were most marked about the posterior nerve roots. It has lately been discovered by Tschirjew that the sensory nerves do not penetrate the muscular fibres, but end in the interstitial fibrous tissues—*i.e.*, in the tissue primarily changed in this disease. It is conceivable that these nerves suffer; their irritation in the early stage may explain pain on exertion sometimes complained of, and the degeneration of some fibres may ultimately slowly ascend to the cord, and lead to, or set up, some of the adjacent degenerative changes. Moreover,

some change may be expected to result secondarily to that in the muscular fibres, as after amputation of a limb.

How far do other observations confirm these conclusions? Dr. Meryon and Mr. Savory could find no change in the cord. The first minute examination was made by Mr. Kesteven, who found only minute scattered spots of granular degeneration, and the empty spaces around the vessels which are very common in the nerve-centres at almost all ages, and these changes have certainly no more significance than those we found. Mr. Kesteven describes the motor nerve-cells as everywhere normal. In five examinations which have been made upon the Continent by Cohnheim,* Charcot,† Brieger,‡ Bay,§ and Schultze,|| no change could be discovered in the cord except a slight increase of tissue in the lateral columns, which was noted by Bay. The motor cells in every case were normal. In two cases, however, which occurred in adults, extensive changes in the cord were found by Barth and Müller.¶ But the cases differed in many respects from the ordinary form of the disease as it occurs in children, other symptoms of spinal disease being obtrusive and early, and it seems, as Charcot has suggested,** in the highest degree doubtful whether they were examples of the same disease as that which we have been considering. The change in the muscles was, it is true, similar, but we must not attach too much weight to identity of anatomical change in the muscles. It is probable that the same muscular change may occur from more than one cause, and that it may, in some cases, be secondary to changes in the nervous system which are apparently absent in the common form of the disease. A striking illustration of this has been recorded by Hitzig. A man injured the

* Eulenberg and Cohnheim: "Verhandlung der Berliner Med. Gesellschaft." 1866, p. 191.

† "Arch de Physiologie." 1871-2.

‡ "Deut. Arch. f. Klin. Med." xxii. Heft 2.

§ "Hospitals Tidende," 1877, and "Virchow's Jahresb."

|| "Virch. Arch." 1879, vol. lxxv. p. 482.

¶ See Note iv. appended. ** "Leçons sur les Mal. du Syst. Nerveux."

nerves of his arm near the shoulder-joint. A slow increase in the size of the arm resulted, and the condition of the muscles was found, by harpoon examination, to be the same as in pseudo-hypertrophic paralysis. It was an example of the same anatomical change in the muscles, but certainly not of the same disease as that which we are studying to-day. The condition which resulted in Hitzig's case from injury to the nerves may, in other cases, have been a consequence of disease of the spinal cord, and it seems to me most probable that the cases of Barth and Müller were of this character, as well as some other recorded cases in which no autopsy was obtained. I have not, therefore, included these cases among those analyzed.

The conclusion from these facts seems to be that the pseudo-hypertrophic paralysis of early life is not a disease of the spinal cord, is not, as has often been suggested, an infantile spinal myo-atrophy, similar to the common forms of progressive muscular atrophy (Crüveilhier's atrophy) of adults, with a modification in the muscle changes, the result of the peculiar conditions of nutrition in early life. This it apparently is not. Probably there is a very rare form of progressive muscular atrophy of adults which is not due to disease of the spinal cord. A few well-authenticated examples of this are on record. It is possible that this disease, and that which we are considering to-day, may be of similar nature, although it is scarcely probable that they are quite identical, because the latter presents such a remarkable limitation to children in the vast majority of cases. Their close alliance is suggested by such cases as that already described which, although occurring in association with undoubted cases of the disease, yet presented wasting of all muscles with the exception of the vasti.

Another theory of its pathology is that it is a disease of the system of nerves to which it is customary to relegate, perhaps somewhat hastily, all trophic disturbances—the sympathetic. This was the earliest theory, put forward in 1852 by Dr. Meryon. Certain symptoms have been adduced in support of it: the vascular marbling of

the skin observed in some cases, and the alteration in the temperature of the affected limbs. Little weight, however, can be attached to the latter change, for it is neither uniform in existence nor in direction. Nor can the marbled skin be regarded as of much significance; it is merely an exaggeration of that which is present in healthy children, and quite unlike the uniform congestion due to vaso-motor disturbance. As a rule, other symptoms indicative of disease of the sympathetic are absent, even up to the end of the disease. One case, however, which I have described to you, presented a symptom which is believed to be a result of disease of the abdominal sympathetic—pigmentation of the skin, similar to that of Addison's disease. But the case, in this respect, stands alone, and although the fact is worthy of note in this connection, it cannot, by itself, be allowed to have much weight.

Pathological anatomy may soon, it is to be hoped, furnish us with some decisive information on this question, but at present it helps us little. In most cases the sympathetic ganglia and nerves have not been examined. In one case slight degenerative changes were found by Brigidi.* But Cohnheim could find no change in the ganglia in his case, so that it is uncertain what significance is to be attached to those found by Brigidi. It seems, however, on the one hand, scarcely possible that a disease of such wide extent and prolonged course should be due to a primary affection of the sympathetic without, ultimately, obtrusive signs of such affection. On the other hand, if associated or secondary degeneration occurs in the spinal cord, it seems probable that similar degenerations may take place in the sympathetic centres, and may account for the appearances observed.

Another theory is that the disease is not an affection of the sympathetic, but of "trophic nerves." Here, however, we are met with the same difficulty. All other signs of trophic disturbances are "conspicuously absent." The affected limbs grow, the skin remains sound and natural

* "Imparziale," Feb. 1878.

in appearance, the joints present no effusion, and we have seen that there is no primary degeneration of the muscular fibres. It would be very remarkable that an affection of these nerves should change almost all the inter-muscular tissue throughout the body and leave other structures unchanged. But, as you know, the existence of special trophic nerves is still an open question, and it is very doubtful whether there are any trophic nerves to the muscles except the motor nerves, and of an affection of these there is no evidence.

There is yet another theory of the pathology of the disease which deserves attention—viz., that it is a primary disease of the muscular tissue, “a congenital nutritive and formative weakness of the striated muscle substance.”* Pathologists have naturally been loth to entertain this view, because modern research has shown that almost every morbid state of the muscles, once thought to be primary in them, is really due to disease of the nervous system, and perhaps, on this account, the theory has not always received the attention which it deserves.

Some years ago I showed at the Pathological Society a curious specimen, which is, I think, of much interest in connection with this theory of the disease. It was a small fatty tumour, attached to the conus medullaris of the spinal cord. On microscopical examination it appeared, at first sight, to be entirely composed of fat cells; but closer inspection showed that running through it were many striated muscular fibres accompanied by connective tissue. It had, indeed, as you may see from the section of the tumour which I will place under the microscope, and from the drawing I now show you (Plate, Fig. 3), precisely the aspect of a portion of muscle from a case of pseudo-hypertrophic paralysis. This tumour must be regarded as congenital, as a fragment of the germ tissue of voluntary muscle, which had, by some accident of development, become misplaced. But why did it develop into a myo-lipoma, instead of into a piece of pure muscle?

* Friedreich: *Loc cit.* p. 296.

We can scarcely ascribe this effect to a nervous influence, and we must attribute it either to the conditions of its environment, or to a perverted tendency of development possessed by the fragment thus displaced. But whatever was the cause of the peculiar condition here, may be the cause of the development of the similar condition throughout the muscular system in pseudo-hypertrophic paralysis; which, we have seen, must be regarded, in the majority of cases, like this spinal myo-lipoma, as a congenital disease. We cannot regard the position and surroundings of the muscles of the body as a cause of the peculiarity in their structure, and we have only remaining the theory of a perverted tendency of development inherent in the germinal tissue of the muscular system. Such a perverted tendency of development may vary in degree in pseudo-hypertrophic paralysis, both in different situations and in different cases, according to conditions which are at present unknown. The remarkable relation of the disease to sex; its conspicuously congenital nature in many cases; its character as a disease of development, making its chief progress during growth; its remarkable origin from the mother, *i.e.*, from the ovum, almost exclusively—a condition unknown in diseases of the nervous system,—are all facts which, although scarcely constituting proof of this theory, yet harmonize with it in a remarkable way, as they harmonize with no other theory of the disease.*

One other pathological relationship of the disease deserves mention. A few cases on record make it probable that a true muscular hypertrophy may occur as a general disease, having some resemblance to the lipomatous-form. These rare cases have not yet been sufficiently studied to make it profitable to discuss them. They may be the result of an influence, *allied* to that which causes the interstitial growth of fat and fibre in the disease we are discussing to-day. In this connection it is important to note that

* The character of limitation to males and unilateral inheritance from the ovum only, the mother not being affected, but the mother's brothers suffering, is seen also in some cases of hæmophilia, and perhaps in no other diseases.

some observers have described the muscular fibres in lipomatous myo-atrophy as of abnormally large size. The observation rests chiefly, however, upon harpoon examinations, and, as already pointed out, much weight cannot be attached to it until it is confirmed by further post-mortem researches.

The diagnosis of the disease need not detain us long. Its gradual development, the progressive difficulty in movement, especially noticeable in the peculiar gait and in rising from the floor or mounting a step, the action by which the patient aids himself, placing his hands on his knees, and the enlargement and firmness of muscles, constitute easily recognized signs of the disease. Of these characteristics, the muscular enlargement, I would impress upon you, is the least to be relied upon. Pathognomonic when present in conjunction with the other symptoms, its absence is of little significance, since, as we have seen, muscles gravely diseased may be of normal size. The distribution of the muscular disease, enlargement and wasting, furnish, however, important diagnostic information. The largeness and hardness of the calves is the most important; but of scarcely less importance is their tendency to contraction, which, even when slight, is significant. The occasional enlargement of the vasti is also important. Still more so are two conditions of the shoulder muscles, which should always be carefully looked for—first, the increased size of the infra-spinatus, and, secondly, the associated wasting of the latissimus and lower part of the pectoralis major. The latter character is, indeed, in my opinion, scarcely less significant than the condition of the calves.

From spinal infantile paralysis it is distinguished by the sudden onset of the latter, its early wide distribution, and ultimate localization to a limb or certain muscles, the complete loss of faradaic contractility in the most affected muscles, and their rapid wasting. From infantile paraplegia of gradual onset, the distinction is by the

distribution of weakness in pseudo-hypertrophic paralysis, the normal state of the sphincters, and the absence of symptoms of disease of the spinal cord, especially of active spasm. In children with spasmodic paraplegia there may be marked contraction in the calf muscles, simulating that of pseudo-hypertrophic paralysis. There are, however, other signs of spasm, and the knee-reflex is in excess instead of being diminished. Contraction in the calves does not occur very early in pseudo-hypertrophic paralysis—*e.g.*, not under four or five years.

It is rarely that the disease has to be distinguished from the “progressive muscular atrophy” of children, since the latter disease is very seldom met with. In some cases which Duchenne has described, the atrophy commenced in the lips and spread gradually downwards, the power of the legs being unimpaired when the shoulders were greatly wasted, a condition never seen in pseudo-hypertrophic paralysis.

A more frequent difficulty is the distinction of the affection from cases of commencing cerebral or cerebellar disease, in well-developed children who are unsteady on their legs and easily fall. In such cases, however, there is not the peculiar gait and hollow back, and the manner in which the children get up again after falling will always resolve any doubt that may exist.

The fact that the disease sometimes manifests itself, or at any rate becomes obtrusive, after an acute illness, has led some cases to be regarded as “paralysis after fever, measles,” &c. Cases 31 and 32 (Appendix) were diagnosed as “paralysis after typhoid fever.” An acquaintance with the symptoms of the disease, and a knowledge of its occasional development after such acute affections, is all that is necessary to prevent the error.

The prognosis in this disease is, as you will have gathered, extremely grave. It is doubtful whether any case in which the symptoms were well marked has recovered. The progress of the disease appears related in most cases

to the process of growth, and, in severe cases at least, we can no more arrest its progress than we can arrest the growth of the body. But the facts on record enable us to recognize great variations in the degree of the morbid state. The tendency of the disease appears to be, in some cases, much slighter than in others, and in those cases in which it is least it is commonly manifested at a later period, and its degree is still slight at the age at which the more severe cases die. There is some reason to believe that after growth is over the progress of the affection is still less rapid, and that in some cases the disease may then become practically stationary, as in Cases 35 and 36. Sometimes a rapid advance in adult life may occur, as in Case 23. The prognosis, then, must be influenced by the period at which the disease commences, by its observed rate of progress, and by the age of the patient. The later it appears, the more slowly it advances, the older the patient, the better, as a rule, is the prognosis. A few cases, however, are on record in which the symptoms appeared early and the development of the disease was retarded beyond the average. For the reasons I have given, the prognosis is also, on the whole, much less grave in girls than in boys, and, as Case 24, related above, and Cases 26 and 37, appended, show, the disease may remain stationary for many years. The affection of many children in the same family does not appear materially to influence the prognosis in individual cases.

The treatment of the disease has to be directed rather against the effects of the morbid process than against the morbid process itself, which, whatever be its nature, is certainly to a large extent beyond our influence. This is readily intelligible if the theory be correct that it is really a primary error of development in the germinal tissue of the muscular system itself. There are, however, certain remedies which have a profound influence on tissue growth, and these have been employed in cases of pseudo-hypertrophic paralysis, with, it has been thought by some

observers, a distinct effect in retarding the progress of the disease, although in no case could anything like an arrest of the affection be attributed to their employment. Foremost among these is arsenic, which was first used by Dr. Meryon, who, in some of the cases which he published in 1852, thought that distinct benefit was derived from its administration. In the case of the girl with the late symptoms of the disease, which I have mentioned to you, there was a marked improvement in the general nutrition, and certainly no increase in the disease while she was taking it. In the same case, phosphorus, which, in small doses, has an action very similar to that of arsenic, also appeared to be useful, and especially to influence the peculiar pigmentation of the skin which she presented. Strychnine and other nervine remedies have appeared to exert little influence.

Other remedies which have been employed are those which influence in a more direct way the tissue nutrition. Iron effects, however, very little. Cod-liver oil is certainly more useful. It has been proposed by some to limit the ingestion of fat-forming food with a view of lessening the deposit of fat in the affected muscles. I cannot but think, however, that this view is based upon erroneous pathological notions and is detrimental in practice. Fat merely fills up the connective tissue which is formed between the fibres; the latter suffer from the overgrowth of connective tissue as much as, or more than, from the deposit of fat, and we cannot limit the fat-forming elements in the food of a growing child without seriously prejudicing the nutrition of the tissues; the muscular fibres are more likely to suffer in their nutrition, while there is no reason to suppose that the growth of the damaging connective tissue would be hindered. Under the use of cod-liver oil I have observed the enlarged muscles to increase in size, but it has always been in proportion to the general improvement in the patient's nutrition and to the increase in size in other parts, and I have never observed any coincident

diminution in muscular power. In some cases this has been even temporarily improved.

The muscular nutrition may also be influenced by local treatment. Of this one of the most important is faradization, which effects functional stimulation of the fibres. The influence of faradization in this disease has, however, disappointed the expectations which had been formed of it. In pronounced cases, although the weakness has, for a time, not increased under its use, little improvement has been observed. Duchenne, who concurred in this opinion, stated, however, that in two cases treated at a very early period he succeeded in effecting a cure. But his account of these cases is not very satisfactory, since in neither was the subsequent progress of the case ascertained; and in the case of one patient the symptoms were very slight, and, as recorded, not very decisive. His experience as regards treatment in the early stage, moreover, has not been confirmed by any other observers. I think, nevertheless, that faradization is a mode of treatment to be recommended in these cases, but without any exalted expectation of the good which it will effect. There are no facts on record, and I have certainly met with none, to warrant us in anticipating more good from the voltaic current than from faradization, unless the muscles react more readily to it, when it should be employed.

Systematic muscular exercises are of great importance. They constitute the physiological stimulus to the development of muscular tissue, and are often distinctly useful. The patients should be encouraged to the methodical use of the muscles which are weak, as by stepping on to a block of wood which is at first not higher than they can readily mount. The height of it may gradually be raised. Gentle gymnastic exercises for the arms should also be adopted.*

* In the pamphlet referred to in the Appendix, the Rev. Lord S. G. Osborne has given an interesting account of the improvement in one patient by such measures—an improvement lasting several years.

Rubbing, and passive movements of the limbs, assist in improving muscular nutrition, and may be employed with advantage. Passive movements are especially important as a means of counteracting, in some measure, the tendency to contractions and deformities. For the contraction of the calf muscles, division of the Achilles tendons has been employed, but, as a rule, it effects only a transient amelioration in the patient's state. In cases, however, in which the contraction of the calves is excessive, *i.e.*, occurs early and out of proportion to the muscular weakness, the patient's condition may be greatly improved by getting rid for a time of the contraction by division of the tendons. This is the opinion of Mr. Adams, and Case 35 is a striking illustration of its correctness. In this patient, at five years of age, the contraction was such as to threaten, in a short time, to take the patient off his legs, and Mr. Adams divided the tendons. The lad was able to walk well after the operation, and contraction did not return, in troublesome degree, until he was twelve, when tenotomy was again performed, and has not been again needed, the calves now (at twenty years of age) being only slightly contracted, and the patient still able to walk three miles. This case is exceptional; but it illustrates the condition in which tenotomy is useful, and its occasional beneficial influence. Whenever tenotomy promises to preserve for a time the power of walking, it should be employed, not only to preserve to the patient this power, but also because the cessation of the use of the legs means the cessation of muscular exercise—of the physiological stimulus to natural muscular nutrition. In all cases when, from any cause, the patient ceases to walk, the weakness in the muscles rapidly increases.

Mechanical appliances may enable the patient to use his legs for a longer time than he otherwise would, and may increase his comfort. Supports to the spine are also useful. Sayre's plaster of Paris jacket, or the poroplastic jacket, may be tried when the spinal muscles are so

weak that curvature is present in the sitting posture. In the only case in which I have had an opportunity of trying it, however, the patient, who was much emaciated, could not bear it. Crutches fixed to the arms of a chair may enable the patient in the later stages to sit up with greater ease.

In all cases, it must be remembered, death does not occur from the disease itself, but from intercurrent affections, in most cases pulmonary, against the causes of which the patient must be, as far as possible, guarded.

A P P E N D I X.

I.—ADDITIONAL CASES.

THE following are abstracts of the notes of twenty original cases, eighteen of which have been kindly supplied to me by Mr. Wm. Adams and the other two by Dr. Clifford Allbutt. Mr. Adams was a very early observer of the disease, having been, indeed, familiar with its clinical history before attention was called, in this country (by Dr. Lockhart Clarke), to Duchenne's description of it. The accuracy of the diagnosis may, therefore, be relied upon, even in the cases of which the account is very brief.

The first five cases occurred in the family of two sisters, who married two brothers. There was no blood-relation between the husbands and wives. The fathers are both tall, fine men: the elder stout and muscular; the younger spare. The wives are short, and of nervous temperament: the wife of the elder brother is thin and looks feeble; that of the younger looks very strong.

Children of the elder brother.—PHILIP C. W. As an infant, strong and well-developed; calves "large and handsome." After a few years

CASE 25. some difficulty in walking up-hill was observed. Increased difficulty in walking was noted after a fall (spraining his ankles) at nine. At eleven he had two epileptic fits. Six months later the calves began to contract, and at twelve the tendons were divided, after which, with rubbing and galvanism, there was marked improvement for a year. Subsequently the lameness increased: at fifteen, however, he could still stand and play croquet for several hours. The weakness gradually increased, and he died at the age of twenty and three-quarters years from gradual exhaustion, preceded by sickness and irritability of the stomach for two or three months.

A sister, aged eight years, when seen, January 2nd, 1861, presented slight contraction of both calves. The tendons were divided, and the symptoms had not increased when she was seen again at seventeen. Two other daughters were healthy.

The second son, aged ten, was healthy, but the third son, CHARLES W., presented well-marked symptoms of the disease. He was seen at the

CASE 27. age of six years (in 1871), and then presented great difficulty in rising from the floor; walked very badly; could get up-stairs only with great difficulty. The right calf measured $9\frac{3}{4}$ inches, the left $9\frac{1}{2}$ inches in circumference; and the thighs, right $12\frac{1}{2}$ inches, left 12 inches; his height being only 3 feet $4\frac{1}{2}$ inches.

Children of the younger brother, Rev. Jos. W.—The family consists of five children—three boys and two girls—of which the eldest and youngest boys are affected, the intermediate son being healthy. The girls both healthy and well-developed, but run badly and awkwardly, with feet wide apart.

The eldest son, WALTER W., appeared strong until eight years of age, when a peculiarity in gait and feebleness in walking were first observed.

CASE 28. At fifteen he was tall, rather stout; "walks with shoulders thrown back and stomach forward; liable to trip and fall; runs badly, with feet wide apart, and places his hands on his thighs in rising from a chair, although he can rise without. Calves $13\frac{1}{2}$ inches in circumference, very hard, and slightly contracted; feet not flexed beyond a right angle; thighs normal size. Arms feeble, especially at elbow and shoulder joints; deltoids conspicuously large and hard."

The younger brother, BERNARD W., first showed weakness at six, and the large size of the calves was then observed. At ten, he was short and

CASE 29. feeble; "gait similar to elder brother, but worse, with his shoulders thrown back, and stomach forward, and loins incurved. Considerable difficulty in balancing the body; liable to trip and fall; cannot rise from floor or get up-stairs so well as brother. Calves $11\frac{1}{8}$ inches in circumference, contracted, rather more than right angle; thighs flabby; upper arms weak."

Miss. F., aged seventeen. Seen February 22nd, 1870. One of two children, both girls. No family history of any similar complaint. Not

CASE 30. late as an infant; but at three or four used to tumble about more frequently than other children. About eight years of age it was noticed that she could not go up stairs well, and could only rise from the floor in a very awkward manner, by turning the body and assisting herself with the arms. From that time these difficulties steadily increased. When seen she had great difficulty in rising from a chair, or from the floor; she would turn sideways on to her knees, and then help herself up by a chair or table. She could not step into a carriage. The calves were large and contracted, the left three-quarters of an inch smaller than the right.

The next two cases are two brothers, sons of an agricultural labourer: both suffered from typhoid fever six years before being seen, and weakness was in each first noticed after the fever, to which it was supposed to be due.

When seen (March, 1878) the elder, GEORGE H., aged fifteen, was a well-nourished, intelligent lad, in an advanced stage of the disease,

CASE 31. unable to rise from a chair or the floor; both feet contracted in talipes equino-varus. Calves large and prominent, each $9\frac{1}{2}$ inches in circumference; thighs a little below the average size, and muscles weak; knees contracted. Spinal muscles fairly strong. Arms failing in power; deltoids large and tense, fairly strong; biceps on each side very feeble. Pronators and supinators large and firm. Pectorals and latissimi dorsi atrophied; trapezius and scapular muscles well-developed. Intercostals atrophied; and respiration chiefly abdominal.

The younger boy, aged thirteen, was in a less advanced stage, but similar in his general symptoms. Feet contracted in talipes equino-varus; the calves larger and harder than the elder (each $11\frac{1}{2}$ inches in circumference); knees not contracted.

The next two cases were also brothers. JOHN L. C., showed symptoms first at nine: calves became large and hard and feet cold. At thirteen,

CASE 33. when seen (August, 1869), was helpless, but able to feed himself (left calf $12\frac{1}{2}$ inches, right $12\frac{1}{4}$ inches); thighs feeble; no power of adduction; deltoids large and hard; scapular muscles feeble.

ARTHUR C., aged five. A year before began to trip and fall, and would never run with the younger ones. Calves large for age, each

CASE 34. $9\frac{1}{2}$ inches in circumference; gets up from floor in a peculiar way, by rolling over on to his side and then pushing himself up with his hands.

The family consists of three other boys—the eldest eleven—and three girls, all being strong and well.

A clergyman (Rev. S. S.), now about seventy-four years of age, and suffering from symptoms of brain disease, always had conspicuously large calves, but suffered no inconvenience, and was always able to take plenty of exercise. He had seven or eight children, of whom two sons and one girl were affected with this form of paralysis.

Of the sons one (H. S.) exhibited the general features of the disease, in a slight form, his calves presenting marked contraction. Mr.

CASE 35. Adams had divided the tendons in 1863, and again, contraction having returned, in 1871. At the age of seventeen, in 1876, he was able to do some work in the City as a clerk. The calves and limbs generally were thin, with the exception of the vasti externi, which were enormously enlarged (see Fig. 7, p. 28). There is now (Nov., 1879) only a slight tendency to recontraction. He can still walk three miles on level ground, but has great difficulty in going up-stairs, and cannot rise from a chair without putting his hands on his knees. The arms are very thin—"those of a skeleton"—and feeble, but their weakness is not increasing.

Another son (Rev. B. S.) took orders as a clergyman, and, in 1871,

at the age of twenty-five, exhibited many of the phenomena of the disease: large calves, and inability to get up from the floor without

CASE 36. assisting himself with his hands on his knees; ultimately he could not rise without assistance. The disease is making slow progress. His age is now thirty-three. He can still walk on level ground but has extreme difficulty in rising from the ground or going up-stairs. His arms are very feeble, but he has become extremely fat (just as Case 23).

A daughter, now aged about twenty-three, also presents large calves, with distinct tendency to contraction, but her condition has
CASE 37. been stationary for several years.

JOHN L. H., aged seven, seen June 10th, 1869. Intellect good; rises from chair with effort and difficulty; cannot go up-stairs without

CASE 38. pulling himself up by banisters and putting his hands on his knees. Does so also in rising from the floor. Both calves 10 inches; thighs at centre 12 inches; forearms $7\frac{1}{4}$ inches; arms, middle of biceps, 7 inches. A year and a quarter later the calves were a quarter of an inch less ($9\frac{3}{4}$ inches), the thighs $11\frac{1}{4}$ inches, and the middle of the biceps $6\frac{1}{2}$ inches. Otherwise his condition was nearly the same.

EMMANUEL V., aged nine, seen July 19th, 1870. Very weak, large
CASE 39. calves; cannot rise if he falls, nor from a seat; did not walk till four; no history of similar affection in family.

ALEXANDER R., aged fourteen years when seen on December 31st, 1863. Affection commenced at ten; an elder
CASES 40 & 41. brother in more advanced stage of the disease.

ARTHUR W., aged eleven, admitted into Orthopædic Hospital December 3rd, 1867. An only child—other children having died

CASE 42. in infancy—father and mother healthy, no similar affection on either side. A well-developed, healthy-looking boy, well and active until his sixth year, when he began to fall about and gradually developed well-marked symptoms.

The cases communicated by Dr. Clifford Allbutt were two brothers, in whose family no other instance of the disease could be traced.

CASES 43 & 44. The disease presented characteristic features in each. In the elder it was first recognized about eight years of age, but the child had never possessed much muscular power. In the younger it commenced at about four years. Another child died from Hodgkin's disease.

II.

SIR CHARLES BELL'S CASE.—("Nervous System," Second Edition, 1830, p. clxiii.)

"A boy, at eight years of age, began to experience difficulty in rising from a chair. The disease gradually progressed till at eighteen he had to

twist and jerk his body about to get upright. The muscles of the lower extremities, hips, and abdomen were debilitated and wasted. The extensor quadriceps femoris on both sides wasted, but the vasti externi had not suffered so much; a firm body, remarkably prominent, just above the knee-joint, marked the position of the vastus externus. No defect of sensibility or affection of the sphincters. The upper part of the body, shoulders, and arms were strong."

The condition of the thighs was apparently identical with that of the patient whose case is mentioned on p. 59 (Case 35), and this symptom, taken in conjunction with the course of the disease, renders, I think, its identity certain.

III.

The following account of Dr. Meryon's first series of cases is extracted from a pamphlet which was printed for private circulation, in 1859, by the Rev. Lord Sidney Godolphin Osborne, who took great interest in the cases and in the disease, and tended one of the sufferers for several years. I have to thank him for a copy of his pamphlet, which is entitled "The Construction of Voluntary Muscle with Reference to Muscular Degenerative Disease, as seen in Certain Cases, with Observations on that Disease: by a Member of the Microscopical Society." In view of the doubt which has been expressed regarding the nature of these cases, the graphic description has a certain historical interest. The italics are as in the original.

"In the family (in which the four boys were affected) there are eight daughters, all free from any manifestation of the disease. Of the three boys who have died, it may be said that as 'infants,' they were finely formed, large-limbed, healthy children. When very young it was observed that they were deficient in '*spring*,' that they were unusually heavy in the arms of the nurse, *heavy to lift*. Very soon after they had acquired power to walk at all, they showed a disposition *to fall*, falling on the face, making no effort with the hands to save themselves, and, when down, having no power to get up again. They now became subject to an increasing unsteadiness on their legs; the least push or obstruction caused them to fall. * * * A very peculiar gait in walking now became most evident, the back at the loins *curving in*, the chest at the *upper part* thrown back, at the *base of the sternum* projecting forward; the stomach very much protruded. There was extreme difficulty in going up-stairs; on coming down, the tread *noisy and heavy*. If a low chair was sat upon, to rise from it the hands had to be put upon the leg just above the knee. If the kneeling posture was assumed, there was great difficulty in getting up from it; the legs had to be straddled out, and the head and shoulders *butted* forwards. The disease gained ground very rapidly; the walk became an unsteady reeling or

staggering movement, the stomach being pushed out, the shoulders thrown back, the toes turned in, the knees partially bent, and the heels so drawn up that there was at all times a disposition to walk on the toes; where the ground inclined the least upwards *they could only so walk*.

"The disposition to fall now painfully increased; to walk at all became too dangerous and was given up. Sitting still so much, either in the house or when driving, no tension was made against the flexors of the heel and knee; the tendons, unresisted, rapidly and firmly contracted; club-foot and stiff-knee at once supervened. The arms grew weaker; there was a disposition to do everything from the elbow-joint, to leave the upper arm at rest" (pp. 10, 11, and 12). Elsewhere the writer speaks of the enlargement of the calves.

One of the figures of the microscopical condition of the muscles might almost serve for a reduced copy of Fig. 1 in the appended plate. Lord S. G. Osborne's description of the figure is "an ultimate fibre with still some trace of a healthy condition as shown by the striæ on its fibrilla, but it is imbedded in a substance in which no trace of sound fibre can be made out, but in which the oval deposit [fat cells] * * * is abundant. In another portion of the preparation there are good specimens of ultimate fibre, between which is interposed a substance evidently consisting of matter which had been of the same healthy structure; it now, however, is made up of membranous tissue, containing or mixed up with fibrous threads and a good deal of the oval deposits."

IV.—LIPOMATOUS MYO-ATROPHY IN ADULTS.

The following abstracts of recorded cases of lipomatous muscular atrophy in adults may be of interest, as illustrating the conditions in which the symptoms may occur. On account of their wide difference from the form of disease which affects children, they have not been included in the analysis of cases given in the preceding pages:—

Barth ("Archiv der Heilkunde," vol. xii., 1871, p. 121. Case 1).

M., aged forty-four. At forty-one stiffness in left leg, and later, uncertain gait. At forty-two right leg became weak. At forty-three formication in legs, with muscular spasm and pains in right shoulder and hip; weakness in the right arm; wasting of hands and feet. At forty-four electric irritability lost in affected muscles. Death from pneumonia. P.M.—Great increase in subcutaneous fat. Much fat in abdominal and calf muscles, even in their tendinous portions. Extensors of thighs much wasted. Spinal cord: extreme sclerosis of lateral columns; ganglion cells in anterior cornua diminished in number and atrophied.

Müller ("Beitrage zur Path. Anat. des Menschlich. Rückenmark: " Leipzig, 1870).

M., at four, after a fall, wasting of right leg below the knee

and double club foot. At thirty-four erotomania; wasting of right, increased size of left lower leg. Death at thirty-six. Lipomatosis of muscles of both legs below knee; thickening of adventitia of brain; grey degeneration of all parts of the cord at the lower half of the lumbar enlargement; slight degeneration in lateral columns throughout cord.

Eulenberg ("Virchow's Archiv," Bd. xlix., 1870, p. 446).

F., aged forty-four. Weakness in limbs of gradual onset and several years' duration. Right arm as in progressive muscular atrophy; extreme wasting of hand muscles; faradaic irritability very low or absent; left arm, atrophy slighter but similar in distribution. Legs large, especially left muscles below the knee, which were also powerless and hard; with their nerves lost all electrical irritability. Harpoon examination showed interstitial fatty growth.

Dyce Brown ("Edinburgh Medical Journal," 1870, June, p. 1079).

M., aged twenty-six. After two months' stiffness and rheumatic pains in the legs the legs were found to be weak and large, especially the thighs; a month later the calves were enlarged and firm, and also the abdominal muscles. Three months later the arms were weak and the biceps abnormally large, the hands thin and weak. Six months after the onset the man's condition was the same, but the glutei also were large.

Benedikt ("Elektrotherapie:" Vienna, 1868, p. 187. Case 191).

M., aged forty. Suffering from epileptic dementia, formerly melancholic. The muscles of the thighs and pelvis, sacrolumales, pectorales majoris, and serrati, left anconeus, and abdominal muscles large and weak, with diminished electrical contractility. Sensitiveness to pain lessened in various parts of the body.

Dahilup ("Virchow's Jahresb.," 1872, II. 273).

M., aged twenty-nine. At twenty-eight and a half slowly increasing size, and general tension of skin. Three months' later, after an injury, weakness, giddiness, and difficulty in speaking and swallowing. Muscles greatly increased in size; no lordosis; atrophy of optic nerves; dropsy.

Schliesinger ("Wien Med. Presse," 1873).

F., aged 43. Mental derangement; muscles large; of right limbs larger than left, but irritability equal. An excised fragment of the gastrocnemius showed a great increase in the interstitial fibrous tissue.

Cases of muscular enlargement in adults recorded by Auerbach and Berger, although they have been classed with cases of pseudo-hypertrophic paralysis, appear to have little in common with it, since the muscles presented no evidence of interstitial change; in some cases the increase in size was local, and apparently depended on true hypertrophy.

V.

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Barlow, "Liverpool and Manchester Rep."	2
Barth, "Arch. der Heilk." XII. 1871 (Case 2)	1
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* Duchenne's Case 10 (1868), is apparently the same as that published in 1864, although counted as a separate case by Friedreich. Case 12 had been described by Bergeron.

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Foster, Balthazar, "Lancet," May 8, 1869, and April 18, 1874.	1
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INDEX.

- Acton's Reproductive Organs, 14
 Adams (W.) on Clubfoot, 11
 — Contraction of the Fingers, 11
 — Curvature of the Spine, 11
 Allan on Fever Nursing, 7
 Allingham on Diseases of the Rectum, 13
 Anatomical Remembrancer, 4
 Anderson (McC.) on Eczema, 13
 Aveling's Influence of Posture on Women, 6
 Balfour's Diseases of the Heart and Aorta, 9
 Balkwill's Mechanical Dentistry, 13
 Bantock on Rupture of the Female Perineum, 6
 Barclay's Medical Diagnosis, 8
 Barnes on Obstetric Operations, 5
 — on Diseases of Women, 5
 Beale's Microscope in Medicine, 8
 — Slight Ailments, 8
 Bellamy's Surgical Anatomy, 3
 Bennet (J. H.) on the Mediterranean, 10
 — on Pulmonary Consumption, 10
 — on Nutrition, 10
 Bentley and Trimen's Medicinal Plants, 7
 Bentley's Manual of Botany, 7
 Bigg (H. H.) on Orthopraxy, 11
 Bigg (R. H.) on the Orthopragms of Spine, 11
 Binz's Elements of Therapeutics, 7
 Black on the Urinary Organs, 14
 Bose's Rational Therapeutics, 7
 — Recognisant Medicine, 7
 Braune's Topographical Anatomy, 3
 Brodhurst's Anchylosis, 11
 — Orthopædic Surgery, 11
 Bryant's Practice of Surgery, 11
 Bucknill and Tuke's Psychological Medicine, 5
 Bulkley on Eczema, 13
 — on Diseases of the Skin, 13
 Burdett's Cottage Hospitals, 5
 — Pay Hospitals, 5
 Burnett on the Ear, 12
 Burton's Midwifery for Midwives, 5
 Butlin's Sarcoma and Carcinoma, 13
 Buzzard's Diseases of the Nervous System, 9
 Carpenter's Human Physiology, 4
 Carter (H. V.) on Spirillum Fever, 7
 Carter (W.) on Renal and Urinary Diseases, 14
 Cayley's Typhoid Fever, 9
 Charteris' Practice of Medicine, 8
 Clark's Outlines of Surgery, 10
 Clay's (C.) Obstetric Surgery, 6
 Cobbold on Parasites, 14
 Coles' Dental Mechanics, 13
 — Deformities of the Mouth, 13
 Cormack's Clinical Studies, 8
 Coulson on Stone in the Bladder, 14
 — on Syphilis, 14
 — on Diseases of the Bladder, 14
 Cripps' Cancer of the Rectum, 14
 Curling's Diseases of the Testis, 13
 Daguene's Manual of Ophthalmoscopy, 12
 Dalby's Diseases and Injuries of the Ear, 12
 Dalton's Human Physiology, 4
 Day on Diseases of Children, 6
 — on Headaches, 9
 De Chaumont's Sanitary Assurance, 4
 Dobell's Lectures on Winter Cough, 8
 — Loss of Weight, &c., 8
 — Mont Dore Cure, 8
 Domville's Manual for Nurses, 7
 Druitt's Surgeon's Vade-Mecum, 11
 Duncan on the Female Perineum, 5
 — on Diseases of Women, 5
 Dunglison's Medical Dictionary, 14
 Ellis's Manual for Mothers, 6
 — of the Diseases of Children,
 Emmet's Gynæcology, 5
 Eulenburg and Guttmann's System of Nerves, 9
 Fayer's Observations in India, 7
 — Tropical Dysentery and Diarrhœa, 7
 Fenwick's Chronic Atrophy of the Stomach, 8
 — Medical Diagnosis, 8
 — Outlines of Medical Treatment, 8
 Fergusson's Practical Surgery, 10
 Flint on Phthisis, 8
 — on Clinical Medicine, 8
 Flower's Diagrams of the Nerves, 4
 Foster's Clinical Medicine, 8
 Fox's (C. B.) Examinations of Water, Air, and Food, 4
 Fox's (G. H.) Photographs of Cutaneous Syphilis, 14
 — Skin Diseases, 13
 Fox's (T.) Atlas of Skin Diseases, 13
 Frey's Histology and Histo-Chemistry, 4
 Fulton's Text-Book of Physiology, 4
 Galabin's Diseases of Women, 6
 Gamgee's Fractures of the Limbs, 11
 — Treatment of Wounds, 11
 Gant's Diseases of the Bladder, 14
 Gaskoin on Psoriasis or Lepra, 13
 Gay on Hæmorrhoidal Disorder, 13
 Gill on Indigestion, 10
 Glenn's Laws affecting Medical Men, 14
 Godlee's Atlas of Human Anatomy, 3
 Gowers' Diseases of the Spinal Cord, 9
 — Epilepsy, 9
 — Medical Ophthalmoscopy, 9
 — Pseudo-Hypertrophic Muscular Paralysis, 9
 Habershon's Diseases of the Abdomen, 9
 — Stomach, 9
 — Pneumogastric Nerve, 9
 Hamilton's Nervous Diseases, 9
 Hardwicke's Medical Education, 14
 Harris on Lithotomy, 14
 Harrison's Surgical Disorders of the Urinary Organs, 14
 — Prevention of Stricture, 14
 Heath's Injuries and Diseases of the Jaws, 10
 — Minor Surgery and Bandaging, 10
 — Operative Surgery, 10
 — Practical Anatomy, 3
 — Surgical Diagnosis, 10
 Hemming on the Laryngoscope, 12
 Higgens' Ophthalmic Out-patient Practice, 11
 Hillis' Leprosy in British Guiana, 13
 Hogg's Indian Notes, 7
 Holden's Dissections, 3
 — Human Osteology, 3
 — Landmarks, 3
 Holmes' (G.) Guide to Use of Laryngoscope, 12
 — Vocal Physiology and Hygiene, 12
 Hood on Gout, Rheumatism, &c., 9
 Hooper's Physicians' Vade-Mecum, 8
 Horton's Tropical Diseases, 7
 Hutchinson's Clinical Surgery, 11
 — Rare Diseases of the Skin, 13
 Huth's Marriage of Near Kin, 4
 Ireland's Idiocy and Imbecility, 5
 Irvine's Relapse of Typhoid Fever, 8
 James on Sore Throat, 12
 Jones' (C. H.) Functional Nervous Disorders, 9
 Jones (C. H.) and Sieveking's Pathological Anatomy, 4
 Jones' (H. McN.) Aural Surgery, 12
 — Atlas of Diseases of Membrana Tympani, 12
 Jones' (T. W.) Ophthalmic Medicine and Surgery, 11
 Jordan's Surgical Enquiries, 11
 Lancereaux's Atlas of Pathological Anatomy, 4
 Lane's Lectures on Syphilis, 14
 Lee (H.) on Syphilis, 14
 Leared on Imperfect Digestion, 10
 Lewis (Bevan) on the Human Brain, 4
 Liebreich's Atlas of Ophthalmoscopy, 11
 Liveing's Megrin, Sick Headache, &c., 10
 Lucas's Indian Hygiene, 8
 Macdonald's (A.) Chronic Disease of the Heart, 6
 Macdonald's (J. D.) Examination of Water, 4
 Macewen's Osteotomy: Knock-knee, Bow-leg, &c., 11
 Mackenzie on Diphtheria, 12
 — on Diseases of the Throat and Nose, 12
 Maclise's Dislocations and Fractures, 11
 — Surgical Anatomy, 3
 MacMunn's Spectroscope in Medicine, 8
 Macnab's Medical Account Books, 14
 Macnamara's Diseases of Bones and Joints, 11
 — the Eye, 12
 Madden's Principal Health-Resorts, 10
 Martin's Military and State Medicine, 5
 Mason on Hare-Lip and Cleft Palate, 12
 — on Surgery of the Face, 12
 Mayne's Medical Vocabulary, 14
 Mitchell (R.) on Cancer Life, 13
 Mitchell's (S. Weir) Nervous System in Women, 6

[Continued on the next page]

- Moore's Family Medicine for India, 7
 — Health Resorts for Tropical Invalids, 7
 Morris' (H.) Anatomy of the Joints, 3
 Nettleship's Diseases of the Eye, 12
 Nunn's Cancer of the Breast, 13
 Ogston's Medical Jurisprudence, 4
 Osborn on Diseases of the Testis, 13
 — on Hydrocele, 13
 Parkes' Practical Hygiene, 5
 Pavy on Diabetes, 10
 — on Food and Dietetics, 10
 Peacock's Diseases of the Heart, 9
 Pharmacopœia of the London Hospital, 7
 Phillips' Materia Medica and Therapeutics,
 Pirrie's Principles and Practice of Surgery, 11
 Pollock on Rheumatism, 9
 Pridham on Asthma, 8
 Purcell on Cancer, 13
 Radford's Cæsarean Section, 5
 Ralfe's Morbid Condition of the Urine, 14
 Ramsbotham's Obstetrics, 6
 Reynolds' (J. J.) Diseases of Women, 6
 — Notes on Midwifery, 6
 Reynolds' (J. R.) Clinical Electricity, 10
 Roberts' (C.) Manual of Anthropometry, 5
 Roberts' (D. Lloyd) Practice of Midwifery, 5
 Ross's Diseases of the Nervous System, 9
 Roth on Dress: Its Sanitary Aspect, 5
 Rousset's Transfusion of Blood, 8
 Routh's Infant Feeding, 6
 Royle and Harley's Materia Medica, 7
 Sanderson's Physiological Handbook, 4
 Sansom's Diseases of the Heart, 9
 — Antiseptic System, 9
 Savage on the Female Pelvic Organs, 6
 Sayre's Orthopædic Surgery, 11
 Schroeder's Manual of Midwifery, 6
 Sewill's Dental Anatomy, 12
 Sheppard on Madness, 5
 Sibson's Medical Anatomy, 3
 Sieveking's Life Assurance, 14
 Smith's (E.) Wasting Diseases of Children, 6
 — Clinical Studies, 6
 Smith's (Henry) Surgery of the Rectum, 14
 Smith's (Heywood) Dysmenorrhœa, 6
 — Gynæcology, 6
 Smith (Priestley) on Glaucoma, 12
 Sparks on the Riviera, 10
 Squire's Companion to the Pharmacopœia, 7
 Squire's Pharmacopœias of London Hospitals, 7
 Stillé and Maisch's National Dispensatory, 7
 Sullivan's Tropical Diseases, 7
 Swain's Surgical Emergencies, 10
 Swayne's Obstetric Aphorisms, 6
 Taft's Operative Dentistry, 12
 Taylor's Medical Jurisprudence, 4
 — Poisons in relation to Medical Jurisprudence, 4
 Teale's Dangers to Health, 5
 Thomas on Ear and Throat Diseases, 12
 Thompson's (Sir H.) Calculous Disease, 13
 — Diseases of the Urinary Organs, 13
 — Diseases of the Prostate, 13
 — Lithotomy and Lithotripsy, 13
 Thompson's (Dr. H.) Clinical Lectures, 8
 Thorowgood on Asthma, 8
 — on Materia Medica and Therapeutics, 7
 Thudichum's Pathology of the Urine, 14
 Tibbits' Medical and Surgical Electricity, 10
 — Map of Motor Points, 10
 Tidy and Woodman's Forensic Medicine, 4
 Tilt's Change of Life, 6
 — Uterine Therapeutics, 6
 Tomes' (C. S.) Dental Anatomy, 12
 — (J. & C. S.) Dental Surgery, 12
 Van Buren on the Genito-Urinary Organs, 14
 Veitch's Handbook for Nurses, 6
 Virchow's Post-mortem Examinations, 4
 Wagstaffe's Human Osteology, 3
 Walker's Ophthalmology, 12
 Waring's Indian Bazaar Medicines, 7
 — Practical Therapeutics, 7
 Warner's Guide to Medical Case-Taking, 8
 Waters' (A. T. H.) Diseases of the Chest, 8
 Waters (J. H.) on Fits, 9
 Wells (Spencer) on Ovarian and Uterine Tumours, 6
 West and Duncan's Diseases of Women, 5
 Whistler's Syphilis of the Larynx, 12
 Whitehead's (J. L.) Climate of the Undercliff, 10
 Whittaker's Primer on the Urine, 14
 Wilks' Diseases of the Nervous System, 9
 Wilks and Moxon's Pathological Anatomy, 4
 Wilson's (Sir E.) Anatomists' Vade-Mecum, 3
 — Lectures on Dermatology, 13
 Wilson's (G.) Handbook of Hygiene, 5
 — Healthy Life and Dwellings, 5
 Wilson's (W. S.) Ocean as a Health-Resort, 10
 Wise's Davos Platz, 10
 Wolfe's Diseases and Injuries of the Eye, 11

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