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Original Communications.

[A CASE OF PROGRESSIVE MUSCULAR
SCLEROSIS, WITH A PAPER ON
THE SAME.

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The Children's Hospital, Boston, and S. G. WEBBER,
M.D., Boston. Read before the Suffolk District Medi-
cal Society, Sept. 24th, 1870.

J. S., of Irish parentage, five years and two months old, was admitted into "The Children's Hospital" on the 2d September, 1870. To the age of three years he was quite a healthy child, but at about that period he began to move and act as though he had less strength than usual, and by degrees his mother came to acknowledge that such was really the case; this condition increased and the spinal column became very weak, and the "inward crook" of it was noticed by her.

Two weeks before admission he had a whitlow upon the fore-finger of his left hand, and one week before, he had a fall while attempting to run upon a sidewalk; from the first event the mother dated the special failing of his health, which she thought was increased by the second, and the evident decrease of his vital powers induced her to seek for him the benefits of the Hospital.

When first seen by the writer, the child was sitting in a corner of his bed, a soft pillow being behind him, he being in such a position that if a line had been carried from the end of the spine, over it, to the back of his head and continued on the same curve, it would have formed a circle, or nearly so.

On the day after admission, Dr. Webber saw the patient with me, and at once recognized the disease. We caused him to stand upon the floor, and he walked a few steps in a tottling or shambling manner. The spine presented a regular and exaggerated curve inward, from the third or fourth dorsal vertebra to the sacrum. This shape comes well under the name given by Duchenne—"saddle-back." A perfect pic-

ture of this child may be seen in Duchenne's book, *électrisation localisée*, 2nd edition, 1861, p. 355.

The head, while he was standing, and indeed while he was in any position which might have been called upright, gave to the spectator the idea that it was large and heavy, for the chin rested upon the sternum, and was inclined more to the right shoulder than to the left. A copy from Duchenne is presented.

The muscles of the calves were largely developed, and the nates seemed to be so.

Upon attempting to take food, or even water, there was always a great choking and inability to swallow, so that it may be said he took no nourishment. He died on the fifth day after admission, having had convulsions on the day of his death. There was no post-mortem examination, but he undoubtedly died of pneumonia.

The want of precision and completeness in the history of this case is owing to the incapacity of the mother, and not to any want of diligence on the part of the interrogator; such as it is, it seems to introduce a valuable paper on the subject of the disease, by Dr. S. G. Webber.

The name placed at the commencement of this article is that used by Jaccoud, and is expressive of the change characteristic of this disease. Other names proposed are: progressive paralysis with apparent hypertrophy; pseudo-hypertrophic paralysis; progressive myosclerosis; and by Heller

VOL. VI.—No. 20

[WHOLE No. 2233.]

the long name "lipomatosis luxurians musculorum progressiva."

The first case of this disease was noticed only about twelve years ago. We have been able to find the records of only 41 cases; 21 in German, 13 in French, and 7 in English medical journals. Among the American journals which we have seen, no case has been recorded, though the disease has been described by Dr. Clymer. We have been able to find also the references to a few German cases.

As this disease has attracted considerable attention from foreign observers, and its true pathology is not settled, and as, so far as we know, no case has yet been reported in American Journals, the present opportunity is taken to call the attention of the profession to it, that other cases may be reported, and if possible some light may be thrown on its etiology and pathology. Careful autopsies are especially desirable.

Duchenne noticed a case of this disease in 1858, and during the next three years saw several others, so that when he published his work on electricity in 1861, he narrated one of them, and spoke of the disease under the name "hypertrophic paraplegia of childhood from cerebral cause." This name shows the theory which he then held in regard to the cause of the disease. This theory was subsequently abandoned for another. The accompanying wood-cut, which is copied from Duchenne's case, might have been drawn from our patient, so nearly does it resemble the peculiar attitude assumed while he was standing.

Duchenne continued to collect cases of the same affection, and in 1868 published a memoir in the Archives Générales, entitled, "Investigations in regard to pseudo-hypertrophic muscular paralysis or myosclerotic paralysis."

During the time between the appearance of these two articles, several cases were observed in Germany, and advance had been made by Griesinger, Wernich, Heller and Cohnheim, in the study of the pathology of the disease.

It cannot be said that this is a new disease, for cases were seen before Duchenne separated this from the other forms of paralysis which occur in children. But such cases were referred to some other head, or considered anomalies and no attempt made to classify them. The most celebrated observations before Duchenne's were four cases noticed by Edward Meryon. On referring to these there would seem to be room to doubt whether they were all cases of this disease; two seem to have been.

Duchenne, having examined twelve additional cases, published his memoir, founding his description of the disease chiefly upon his 13 cases. Some of his cases had terminated fatally, only two had improved, one of these is said to have recovered.

To Duchenne, then, belongs the merit of having first recognized this as a distinct form of paralytic disease.

The accompanying table will give an idea of how much has been done and how many cases have been recorded, and will give an opportunity of comparing the symptoms which were present.

From these cases, and from the account given by Duchenne, the symptoms may be described as follows:

The disease frequently commences in early infancy, before it is time for the child to walk. Of 41 cases where a tolerably full account of the origin is given, the statement is made in 11 that the child did not walk until late, from 17 months to 3 years, and then its walk was unsteady, accompanied with frequent stumbling and falls. If the child has begun to walk at the usual time, the disease commences "without pain and without previous fever, sometimes after convulsions; the child quickly becomes tired while walking or standing; soon he falls frequently; he can run with difficulty, or not at all, and finally becomes disinclined to walking, but desires to be carried." Soon the legs are separated from each other even while the patient is standing; the walk becomes peculiar, "at each step the body is inclined to the side whose foot is on the ground, hence they have a waddling gait." In cases where this gait is seen in healthy children it quickly disappears. "A very constant symptom is the presence, only while standing, or walking, of a lumbo-sacral curve." "A plumb-line falling from the most posterior spinous process, passed at a greater or less distance posterior to the sacrum." Duchenne considers this attitude due to the weakness of the principal extensors of the vertebræ. Later in the disease occurs bilateral equinus, which gradually increases in extent, and becomes equino-varus. The weakness and inability to walk increase until the patient becomes helpless.

The marked feature of this disease, which has given it its title, does not appear until some time after the weakness is observed. This is the pseudo-hypertrophy of certain muscles. Once only, case 35, is it stated to have been noticed with the other early symptoms, and in that case it is possible that the earliest symptoms had been over-

PROGRESSIVE MUSCULAR SCLEROSIS.

No.	Author and Reference.	Patient.	Age at time of observation.	Age at commencement of Disease.	Time of beginning of hypertrophy.	Time during which hypertrophy increased.	Family History.	Electromuscular contractility.	Deformity.	Results and Remarks.
1	Duchenne. Arch. Gen. 1868. I. Case I.	M.	7 years.	Early. Did not walk till 2½ years old.	Noticed at about 3 years.	During 2½ years.	No one similarly affected.	Perfect.	Muscles of the legs and lumbar region enlarged. Arms small. Equinovarus.	At 13 years of age paralysis was complete. Died at 15 years of phthisis.
2	Ibid. Case II.	M.	5 or 6 y.	B'n with large limbs. Never walked.	Noticed in infancy.		None obtained.		Legs large. Arms & body emaciated.	Sensation perfect. Intelligence weak.
3	Ibid. Case III.	M.	8 years.	Did not walk till 2½ years.		3-4 years.	Two brothers died of granular meningitis.	Unaffected	Gastrocnemii, glutei, lumbar, temporal masseter muscles large. Double equinus.	No pain, nor disturbance of sensation. Intelligence weak. At 11 yrs, arms affected, without hypertrophy and paralysis complete in legs. At 14 yrs, died of pleuro-pneumon.
4	Ibid. Case IV.	M.	9 years.	6 years.	5-6 mos. later.	About one year.	No similar disease in family.		Calves, buttocks and lumbar region enlarged.	At 13 years had lost all power over arms as well as legs.
5	Ibid. Case V.	M.	7½ years.	Did not walk till 3½ years.		Till 4 yrs. old.			Calves, thighs and lumbar region enlarged.	Intelligence weak.
6	Ibid. Case VI.	M.	10-11 y.	At 2½ years.	Noticed at 3-4 yrs. of age.	About two years.	No one affected.	Normal.	Calves, thighs and buttocks enlarged.	No pain, nor disturbance of sensation. At 14, paralysis became general.
7	Ibid. Case VII.	M.	8 years.	At 5 years, with convulsions.	A few mos. later.	About one year.		Diminished in paralyzed muscles.	Calves, buttocks and lumbar region enlarged. Also temporal muscle.	At 6 years, weakness of arms began. At 11 years, arms and legs entirely paralyzed.
8	Ibid. Case VIII.	M.		At 2½ years, with convulsions.	Some mos. later.			In 1862 not affected. In 1864 diminished or abolished.	Calves, buttocks and thighs enlarged.	In 1865, paralysis complete. Intelligence normal. Died of phthisis in 1866.
9	Ibid. Case IX.	M.	8 years.	At 7½ years, without fever or convulsions.	3-4 mos. later.				Gastrocnemii slightly enlarged.	Cured in six months.
10	Ibid. Case X.	M.	8 years.	Began to walk at 17 months, but fell often, and rose with difficulty.	At about 4 years of age.	One year.		Normal.	Calves, buttocks and lumbar region enlarged. Arms small. Equinovarus.	Large head. Intelligence good. At 13 yrs., almost deprived of movement. Volume of hypertrophied muscles diminished.
11	Ibid. Case XI.	F.		Never strong; walked well till, at 10 years, she became weak.	Some time later.			Diminished in lower extremity.	Double equinus. Calves enlarged.	
12	Ibid. Case XII.	M.	10 yrs.	Did not walk till 26 months old.			Nothing similar known. Parents and mother's children healthy.	Enfeebled.	All the muscles, except the pectorals, greatly enlarged.	Could walk to school, 1 kilometre, till 8 years old. Intelligence less than normal.

MEDICAL AND SURGICAL JOURNAL.

No.	Author and Reference.	Patient.	Age at time of Observation.	Age at commencement of Disease.	Time of beginning of hypertrophy increased.	Family History.	Electromuscular contractility.	Deformity.	Results and Remarks.
13	Ibid. Case XIII.	F.	6½ yrs.	Did not walk till 2 years, but walked well. At 4½ to 5 yrs. fell frequently.	About 4½ to 5 years old.		Normal.	Legs and thighs enlarged slightly.	Precocious. No disturbance of sensation. Nearly recovered.
14	Heller. Arch. f. Klin. Med., i. 6, p. 616, 1866. Sch. Jahrb., 1866, p. 286.	M.	11 yrs.	6 years.		Two analogous cases in the same family on the mother's side—brother and half-brother.	Rather diminished.	Lower extremities, with gluteal, abdominal and spinal muscles, enlarged. Rest of body rather small.	Skin of lower extremities purple, marbled. Sensibility normal.
15	Ibid.	M.	16 yrs.	9 years.		Same as in No. 14. Brother to 14.	Abolished.	Same, in greater intensity. Some muscles in upper extremities enlarged.	Same, and also loss of intelligence. All 4 (14 & 15) dwelt in a damp, cold dwelling, unfavorable to health.
16	Wernich. Den. Arch. f. klin. Med., ii. 2, p. 232, 1866. Schmidt's Jahrb., 1866, 4, p. 295.	M.	11 yrs.	Did not walk till 3 years old.	Began in 7th year.	Brother 5 yrs old showed beginning of same disease. Half-brother healthy.	By nerve irritation normal. By direct diminished.	Calves, tensor fasciae latae, sartorius, rhomboidens, infra-spinatus and deltoid enlarged; other muscles wasted.	Sensation normal. Skin of forearm and leg marbled. Increase of fatty tissue between fibres of muscle.
17	Stoffella. Wien. Zeitschr., xxi., 1, p. 85, 1865. Schmidt's Jahrb., 1865, 4, p. 179.	M.	13 yrs.	Not long before first paralytic symptoms appeared after variola and measles.	A short time later.		Dimin. in aff. mus. & others. E-mus. sens. normal or inc. in leg.	Calves, extensors of thighs, deltoid, tricipit, sacro-lumbalis, and longissimi dorsi, were hypertrophied.	No material change after several months' treatment by electricity.
18	Griesinger. Arch. d. heilkund, vi., 1, p. 1, 1865. Sch. Jahrb., 1865, 4.	M.	13 yrs.	Weak, with slight thickening of legs in earliest childhood. At ten, became worse.	About 10 years.		Decreased.	Deltoid & the scapular muscles, those of arm, quadratus, lumbar and rect. abdom. affected.	Sudden twitchings in thighs; marbled surface in legs and arms, and cool.
19	Hoffmann. Ueber die sogenannte Muskelhypertrophie. Berlin, 1867. Canstatt's Jahrb., '67, 2, 1, p. 294.	F.	17 yrs.	In 11th year measles. In 12th year gradually unsteadiness, and inability to run.	In 13th year.	Parents and 1 brother healthy; 7 sisters dead, several from convulsions during teething.	In arms moderate. In legs diminished. In thighs good. Sensibility normal.	Muscles of calves enlarged. Atrophy of ant. edge of trapezius, more so on the right; so of deltoid and arm, of the subscapularis and latissimi dorsi and under part of trapez. Bones normal size. Saddle back.	At 12 years, lost power in right arm, and then in left arm. Muscles of lumbo-sacral region became weak at the same time. Skin of hand and leg mottled; leg cool. Atrophied and hypertrophied muscles weakened.
20	Seidel. Die Atrophie Musculorum Lipomatosa (soge-	M.	16 yrs.	At 9 years pain in walking fast and jumping. Chills.	14 years.	This boy and the next two were brothers. There were 6 other children, who were not affected, though the youngest did	Dimin. in all the muscles.	Upper arm and thigh atrophied, leg and forearm hypertrophied. Equinus.	Scrofulous in early life; measles & scarlat. With loss of power in feet, arms weak. Intell. good; little impaired by induc. cur.

PROGRESSIVE MUSCULAR SOLEROSIS.

21	manlike muscular hypertrophy. Jena, 1867. Can. tralblatt, 1867, p. 666. Can. stat's Jahr., 1867, 2, 1, p. 295. 3 cases.	M.	14 yrs.	At 11th or 12th year growth less than normal. At 13 yrs. weakness.	12 years.	not enjoy good health. The parents were healthy.	Dimin. in legs and biceps.	(Deltoid, biceps and thigh atrophied. Triceps, brach. and calves hypertro.	No spontaneous twitching. Did not improve. For ½ yr. frontal headache. Temp. rather dim. in axilla. No change in sens.
22		M.	12 yrs.			Ditto.	Tib. ant. & flex. digit. com. long. dimin.	Left calf slightly enlarged.	This child was examined in the earliest stage. These 3 lived well, but eat little meat.
23	Roquette. Ueber die sogenannte Muskulaturhypertrophie. Inaug. Diss. Berlin. 1868. Can. stat's Jahr., '68, 2, 1, p. 268.	F.	20 yrs.	At ten could not run so well as before.	Next year	No hereditary disease.	Dimin. in affected muscles.	Pectorals, latiss. dorsi, serrati, and erector spinae atrophied. Neck, thighs normal. Calves hypertrophied. Varo-equinus. Saddle-back.	Good dwelling; well nourished. No mental trouble. Catamenia in 15th year. At first anæsthesia. Hands weak for several years. Skin of legs marbled. Skin on the feet ichthyosis.
24	Benedikt. Elektrotherap. Wien. 1868. p. 188. Case 188.	M.	8 yrs.			Younger brother had the same.	Dimin. in legs and thigh. In outer part of deltoid almost gone.	Both calves, flexor of thigh, sacro-lumbal, bar left, outer part of both deltoids, most of muscles of leg hypertrophied.	Improvement in power of walking and running by galvanizing sympathetic. Hypertrophy not diminished.
25	Ibid. Case 194.	F.	8 yrs.				Glutei, elec. mus. cont. dim. Sens. increased.	Both sacro-lumbal. atrophied. Both glutei hypertrophied. Saddle-back.	Improvement by same means as previous case.
26	Ibid. Case 190.	M.	11 yrs.	After a fright at three years.				Most of those of the leg and pelvis, longissimi dorsi, especially left, hypertrophied. Club-foot.	Intelligence normal.
27	Ibid. Case 191.	M.	About 40 yrs.	Probably a few years sooner.			Dimin. in hypertro. and some mus. Elec. sens. increased.	Thigh and pelvic muscles, anconeus left, both sacro-lumbals, pect. maj. serratus ant. maj., especially right and abdominal muscles hypertrophied.	Formerly melancholia and epileptic imbecility. Hypertrophied muscles are paralytic. Sensation diminished in many places.
28	Ibid. Case 192.	M.	30 yrs.	Perhaps two years earlier.				Deltoid right, pect. maj., teres maj. & min., serrat. ant. maj. right, glutei, add. of thigh & mus. of leg hyper. In l. yr. trap., supra-spin. right, hypertrophied.	Pain and weakness in right shoulder, seemingly in the bones. Right side face redder and sweats more than left. Right pupil dilated. Sympathetic tender on pressure. Improved on galvanization and hypertrophy diminished.

MEDICAL AND SURGICAL JOURNAL.

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29	Eulenber. Berlin klin. Wochen., ii, 50, 1865. Schmidt's Jahr. 1866, i, p. 291.	M.	13 yrs.	At about five years began to walk unsteadily.			Healthy.	Retained.	Parts of legs, arms & body enlarg. Neck normal. Double varo-equinus.	Intell. & sens. norm.; at 10 or 11 yrs. saddle back; impr. by Faradiza. and gymn. Died at 13 of broncho-pneumonia.
30	Sigm. in Tab. Arch. f. klin. med., i, 6, p. 630, 1866. Schmidt's Jahr., 1866, 2, p. 285.	M.	14 yrs.	Began to walk late & always unsteady.			Nothing similar.	Some of aff. mus. lost it. oth's w'k.	Lower limbs as high as glutæi.	Skin of lower extremity mottled, purple and cool. No improvement.
31 & 32	Coste & Gioja. See Schmidt's Jahr., vol. 24, referred to in same, vol. 130, 1866, p. 283.	Two bro's.		At 10 years.					All four limbs.	One at 18, tongue and muscles of jaw hypertroph. Genitals not devel., and bones atroph. Other died of heart disease.
33	Lutz. Arch. f. klin. med., iii, 4, p. 358, 1867. Schmidt's Jahr., 1868, i, p. 171.	F.	28 yrs.	In childhood, age not stated.			Uncle & aunt, mother's side, had do.; died at 42 & 43 yrs. Half-sister f'm mother's letmar. had it; died at 6 yrs., of scarlatina.		Calves, and to back enlarged; arms and up. part body ena. face & neck normal.	Voluntary movement in lower extremity gone.
34	Ibid.	Sister to above.	22 yrs.	At an early period.					Only lower extrem. enlarg. Upper norm.	Parents of these two were retarded.
35	James Russell. Med. Times & Gaz., May 29, 1869, p. 571.	M.	11 yrs.	10½ years.	At once, in calves.		Grandmother had hemiplegia. Elder brother had progressive paralysis. Otherwise good.	Not all muscles respond.	Erector spine, hips & calves enlarged.	Legs ached, hands & feet numb. Legs mottled. Brother, mus. same appearance by microsc.
36	Ibid.	M.	10 yrs.	3-4 years legs failed.	Early in disease.	Until 6 years old.	Great uncle on mother's side paralyzed. 2 uncles walk'd till 9, then paralyzed; died at 16 & 17 years. Rest of 8 living child. healthy; 3 died yng. Patient's mother had 8 liv. child., healthy at birth; 1 boy lost power at 4, died at 16. Another lost power at 9, died at 13; no hypertroph., but limbs wasted. 3 girls 4 to 8 yrs. old, healthy.		Calves enlarged.	No pains; never lost power of walking. Right hand grasps more feebly than left. Not dull.
37	B. W. Foster. Lancet, May 8, 1869, p. 629.	M.	9 yrs.	Weak in legs, walked at two years, stumbled much.	6 years.		Eight children, one died scarlatina; two boys older, two girls younger, healthy.	Unaffected.	Calves, erector spine, enl. Thigh firm, not enl. Talipes equinus.	Dull intell. Arms always weak. Legs and arms flabby. Muscles of arms flabby.
38	Wm. Adams. Trans. Path. Soc. London, vol. xix., 1868.	M.	7½ yrs.	About six years old.			An elder brother 16 years old is unable to walk, helpless.		Calves, delt. & several of fore'm enl. thigh & arm small. Equi.	Dull intellectually, mischievous, dirty.
39	Ibid.	M.	11 yrs.						Calves enlarged.	Unable to stand.
40	Dr. Hillier. Trans. Path. Soc. London, vol. xix., 1868.	M.	nearly 11 yrs.	Stood at 21 months old, & then walked imperfectly.	3 years.		Nothing similar.	No loss.	Erector spin. and quad. lumb. bulge.	No fits; no pain. 6 to 7 years, stumbled. At 10½ yrs. c'd not stand. Arms wasted after 5 yrs.
41	D. Dyce Brown. Ed. Med. Jour., June, 1870, vol. xv., p. 1079.	M.	26 yrs.	26 years.	3 months later.				Thighs, calves, oblique, abdom., triiceps, large. Also, later, glutæi. Fore-arm and hand thin.	At first rheumatic pains in thighs. In eight months, gained five pounds.

looked. It is this apparent hypertrophy which tends to deceive the friends of the youngest patients, and causes wonder that a child with such well developed legs cannot walk sooner.

The hypertrophy usually attacks the muscles of the calves, sometimes the glutæi, and the lumbar muscles, less frequently those of the upper extremities, and once only was it almost general. Any muscle may be hypertrophied, but those of the lower extremities are more frequently so. While many muscles are thus enlarged, others are atrophied, and others partially paralyzed. Duchenne says, "In this disease all the muscles paralyzed are not always attacked with apparent hypertrophy, and the degree of paralysis is not in direct ratio to this hypertrophy."

The disease may remain stationary at this stage for many years, after which the weakness increases; the advancing paralysis is accompanied with emaciation, and, "finally, after having lived still one or two years in a state of marasmus and helplessness, these children are carried off by some intercurrent disease."

Accompanying the other symptoms, there is often a loss of intellectual power, chiefly when the disease commences in earliest infancy. Again, when beginning later, it is occasionally preceded by a high degree of intelligence.

The circulation is frequently materially affected, especially in the lower extremities. Duchenne did not notice this, but several other observers, German and English, have observed it.

Boys have been more frequently the subjects of this disease than girls. Of 45 cases, 7 only were girls.

The duration of the disease is rather indefinite. It is a chronic affection, extending over years, and rarely terminating until after, at least, five or six years. In two cases, 33 and 34, the patients were seen at the ages of 22 and 28 years. An uncle and aunt of theirs were said to have had the same disease, and died at 42 and 43 years. The cases which terminated fatally show that there is a tendency to die from some lung complication. Once heart disease was the cause of death, and a half-sister of one of the patients, whose case is only alluded to, died of scarlatina. The natural termination would seem to be by weakening the respiratory muscles to give rise to a predisposition to fatal lung affection.

Little is known as to cause. In only two cases is locality, damp and cold dwell-

ing, mentioned as a possible cause. Duchenne states that in 20 years he had never seen any similar affection among adults, and, in the cases recorded by others, the beginning is generally referred to early childhood. In the two cases in adults mentioned by Benedikt, the origin is not directly mentioned, but it would seem to have been within a few years. The case reported by Dr. Dyce Brown, concerned an adult. These three are the only cases in which the commencement is not referred to childhood or infancy.

Duchenne found no proof that hereditary taint had any part in causing the disease. It will be seen, however, by the cases mentioned by others that there has been, in several instances, a tendency in certain families to be affected. In eleven cases, the disease existed in other members of the family. Two of these cases were brothers, and two were sisters; three others were brothers. In two cases there were nervous affections in other members of the family; in one of these a brother was paralyzed without hypertrophy, but with increase of fibrous tissue between the muscular fibres, as is found in this disease.

It is curious to notice that, although boys are more likely to be affected, when the same disease has occurred in the family it was on the mother's side rather than the father's. Cases 14 and 15, mother's brother and half-brother were similarly affected; cases 33 and 34, uncle and aunt on mother's side had the same, half-sister from mother's first marriage also affected; case 36, great uncle on mother's side had paralysis, perhaps the same, mother's two brothers were paralyzed at 9, and died at 16 and 17. These are the only cases where distinction is made between the parents.

In some cases the disease is probably congenital. Case 2 was born with large limbs.

Neither rheumatism, syphilis, scrofula, nor any other disease can be traced as the cause of this paralysis; generally, the patients, when attacked after the first dentition, had been healthy up to the time when the weakness of the legs was first noticed.

The condition of the hypertrophied muscles was ascertained in one case after death, in other cases by removing small portions, either by cutting down upon them, or by the "*emporte-pièce histologique*." There has been found an increase of the fibrous tissue between the muscular fibres; the latter have, in many cases, lost their striæ, though not becoming granular nor fatty. The fibrous tissue between the muscular

fibres is unnaturally adherent to the sarcolemma, and cannot be entirely separated from it. Some German observers have seen great increase of fatty tissue between the muscular fibres. The latter condition is generally considered only a later stage of the disease. The hypertrophy is dependent on this increase of intermuscular tissue and not on any increase in the muscular fibres, which have sometimes been found diminished in diameter; only once have I noticed the statement that they were increased in size. The atrophied muscles may also show similar increase of fibrous tissue.

Only once was the central nervous system examined with care by Cohnheim, and nothing was found.

Perhaps it is premature to say much yet in regard to the nature of this affection. Only one autopsy has been recorded, and that with negative results. Eulenburg, Duchenne and others, are inclined to refer it to an affection of the sympathetic, and considering the mottled condition which is frequently seen, it seems not unreasonable to do so. Also, that tissue is hypertrophied which is considered as most likely to be increased by a change in the flow of blood through a part from paralysis of the vaso-motor.

It would be of interest to study this disease in its pathological relations in connection with increase of fibrous and connective tissue in other parts, as sclerosis of the nervous centres, also in connection with one-sided hypertrophy and local atrophies.

So far as relates to treatment there is but little to be said. Duchenne relates the recovery of one, and the probable recovery of another patient, under the use of the induced current, shampooing of the muscles and hydrotherapeutics. Both these cases were treated in the earlier stages. Benedikt used the constant current, applying the copper pole to the lower cervical ganglion, and the zinc pole to the lumbar region. Two cases improved under this, one of them also having the separate muscles faradized. In another case the current was applied only to the cervical portion of the sympathetic. There was also marked improvement in this case, amounting almost to a cure. All other measures prove unavailing.

Note.—After reading the above case before the society, it was stated, by some of those present, that the boy had been seen at the Boston Dispensary several times, and the case was there supposed to be tetanus. Dr. O'Connell, who saw the boy, made a record

of his case, from which the following items are taken:

"The boy was first seen August 1st, with a superficial ulceration of the middle finger of the left hand, arising from a splinter. About August 20, there was noticed a stiffness of the neck, apparently an ordinary case of torticollis." About August 24th, he fell three times, and at about the same time, though subsequently to the falls, the mother noticed a peculiarity in his walk, and stiffening of the body. This stiffening continued to increase without any spasmodic action, and at the date when the notes were made, August 31st, there was "a constant state of opisthotonos," the torticollis being aggravated, "the chin being drawn down to the chest, and any attempt on his part to raise the head resulting only in a bending of his back still more. Now, it is only by a very struggling effort that he can balance himself on his legs. When he tries to stand, the opisthotonos is so decided that it almost pulls him over on to the ground. His abdomen is thrown forward; his legs are spread in his effort to sustain himself, and he has not, within the last few days, had sufficient command of himself to be able to walk at all. The muscles on the back of the neck and along the back are rigid and tense, those on the right side of the neck forming a hard swelling behind." "There is not any spasmodic action. The muscles involved persist in their action without any relaxation."

This is a graphic description of the appearance of a patient attacked with muscular sclerosis. The gradual advance of the inability to stand, the falling, the peculiar stiffness of carriage, the hypertrophy, and tension of the muscles of the back and neck, which, though apparently powerful, and seemingly strongly contracted, are in reality too weak, and hence the bending of the body, as if in a state of opisthotonos; the projection of the abdomen, upon the muscles of which devolve the duty of sustaining the patient, the bending forward of the head for preserving the centre of gravity, and the increase of the lumbo-sacral curve on raising the head. This unnatural posture disappears when the patient lies down. Though not mentioned in the notes, Dr. O'Connell told me that it was so with this boy. The position of the head, drawn to one side, was due probably to the muscles on one side of the neck being affected more than those on the other side. Dr. O'Connell considered it, not a case of tetanus, but a condition of constant spasm,

due to the injury to the finger. Whether the finger had any part in accelerating the progress of the disease it is of course not possible to state, for the pathology of the disease is too little known to decide as to what would be the effect of such a condition. There is, however, no doubt, in our minds, judging from the above history and from the condition of the child when seen by us, that this was a case of muscular sclerosis.

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VOL. VI.—No. 20A

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A CASE OF DELIRIUM TREMENS SUCCESSFULLY TREATED BY HYDRATE OF CHLORAL.

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MICHAEL BURKE, seaman extra, æt. 27, was received in the U. S. Naval Hospital at Chelsea, Mass., from the Navy Yard, Sept. 13th, 1870, suffering from a severe attack of mania a potu. He had been on a debauch for over a week, and for six days had not closed his eyes in sleep. Physical prostration comparatively slight, but he had constant hallucinations of various kinds, amounting at times to maniacal delirium, so that he required constant watching to prevent him from jumping through the nearest window. Bowels constipated, great nausea, and total loss of appetite. Had been under treatment for three days previous to his entry in the Hospital, during which time opium, in its different preparations, digitalis, capsicum and infus. humulis, had been ineffectually used. Immediately after admittance, the patient was placed in a warm bath, at 98° F., and at 1, P.M., hydrate chloral gr. xxx. was given in solution, and an hour later gr. xx. more, as the first dose had no apparent effect. At 4½, P.M., patient expressed a desire for nourishment, but his stomach was unable to retain a pint of strong beef tea, seasoned with capsicum, that was given. At 8½, P.M., he became more excited and the hallucinations increased. Hydrate chloral 3i., in water flavored with ol. menth. pip., was administered. Five minutes after, he was seized with convulsions similar to those of a person partly under the influence of ether or chloroform, and he unconsciously passed a large quantity of urine; this was followed by profound sleep, from which he did not awaken until 7½ o'clock the next morning, when he immediately expressed a wish for his breakfast, and was able to retain a hearty meal. He then had several hours more sleep, and the next day was free from all disturbance, save severe pains in the limbs, caused by wrenches in his previous struggle with the nurses. The hallucinations and gastric irritability disappeared after the first period of sleep. This,