

made out. Opacity of vitreous is again somewhat increased in both eyes. R. S. 18/25. L. S. 18/25.

Nov. 4: Right eye. The membrane in lower part of vitreous is no longer visible and the vessels that were seen on it are now indistinctly seen at a greater distance from the anterior part of vitreous than formerly. In the place of the membrane there are now many small fixed membranous opacities. In lowest part of vitreous apparently immediately behind lens are seen two small bluish-white glistening bodies of globular form and in outer region closely behind or on posterior capsule of lens are several very dark opacities. A diffuse opacity of vitreous still veils the disc in both eyes. R. S. 18/25. L. S. 18/25.

Feb. 21, 1877: Right eye: the interior portion of the vitreous is again full of floating membranous opacities. The semi-globular protrusion previously noticed, is again visible. The veins on it have changed their course somewhat. They now run downward instead of forward. The left eye is now free from floating opacities. R. S. 18/20. L. S. 18/15. Patient is still taking potassium iodid in 10 grain doses, three times daily.

March 16: Right eye: The floating membranous opacities have largely disappeared. The convex protrusion is now distinctly seen as a grayish membrane, over which vessels pass from behind forward.

Left Eye: A grayish membrane, convex in form, has made its appearance also in this eye. It is situated in lower outer portion of vitreous, apparently not far behind lens, and protrudes toward the axis of the eye. Vessels are seen on it, but no connection between them and the retinal vessels can be made out. It looks very much like a detached retina. The membrane is sufficiently translucent to allow floating opacities to be seen behind it. In both eyes the V. F. is intact. R. S. 18/20. L. S. 18/15.

June 22: In both eyes the convex protrusion has apparently become larger, and its outline is more opaque.

Dec. 14: Right eye: There is an increase in floating membranous opacities in anterior portion of the vitreous. The convex protrusion is hidden by them.

Left Eye: The convex protrusion seems larger than at last visit.

Jan. 26, 1878: Right eye: Vitreous opacities have nearly all disappeared. The protrusion previously noted, is no longer seen. S. 18/20.

Left Eye: The convex membrane seems to have become loose anteriorly, as it now floats slightly on movements of eye. Several bluish-white, brilliant globular bodies are again visible in lower part of vitreous, not far behind lens.

December 1: Right eye: There is still diffuse haziness of vitreous, but no floating opacities are present. The apparent detachment of the retina is no longer visible. S. 18/20.

Left Eye: The same as right; the membranous protrusion is not visible. There are no vessels in vitreous. S. 18/20.

June 7, 1879: Right eye: No change since last entry.

Left Eye: An increase in the number of floating opacities. The semi-globular protrusion in lower outer part of vitreous is again visible and in addition there is now seen a similar protrusion with vessels on it, in lower inner portion, not far behind lens. S. 18/20. V. F. intact.

April 14, 1880: Right eye: Media are perfectly clear; fundus is normal. S. 18/15.

Left Eye: Large floating membranous opacities. The semi-globular protrusion in anterior lower portion of retina remains as before. S. 18/20.

Nov. 22, 1881: Right eye: Diffuse haziness of vitreous, otherwise normal. S. 18/20.

Left Eye: Haziness of vitreous. The protrusions in lower part of vitreous are no longer visible.

Feb. 2, 1882: Right eye: Vitreous not quite clear. S. 18/15.

Left Eye: Membranous floating opacities in vitreous. Two small atrophic patches are now visible in outer periphery of choroid. S. 18/30.

During the following five years she lived in the tropics. She married and had one child. On her return to this country I examined her eyes and found both free from opacities in the vitreous and fundi normal. During the last few years, the lens of this left eye has become cataractous, and more lately the lens of the right eye has become somewhat

opaque at posterior pole. On Dec. 14, 1905, I found her vision as follows: R. Hm. 1.50; S. 6/6; L. Hm. 1.5; D. S. 6/12. With the exception of the opacities of the lenses the eyes are apparently normal.

As to the pathology of this affection I have nothing new to offer. Although I did not see any actual extravasation of blood in the vitreous body in this case, I have seen it in others with similar membranous formation in the anterior parts of the vitreous, and think that in this case also, hemorrhages from the ciliary body were present before I first saw the case. The resemblance of the membranes with the vessels on them to a detached retina, was so great that one of my most experienced colleagues, who saw the case, urged me to make a scleral puncture, but having watched the development of these membranes for months, I felt sure that they were not the retina and, therefore, did not follow his advice. The gradual disappearance of the connective tissue formation was followed by the complete absorption of all the floating opacities in the vitreous. The participation of the choroid in the morbid process was shown by the appearance of an atrophic spot near its periphery.

### SPINAL AMYOTROPHY WITH PUPILLARY INEQUALITY, AND JUVENILE DYSTROPHY.\*

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The following case, seen in consultation with Dr. Alexander C. Wiener, is reported not as such an extraordinary case, for it is one of amyotrophic lateral sclerosis, but on account of the association with it of inequality of the pupils. This association is exceptional.

CASE 1.—Mrs. J. P., aged 49, twice married, has two healthy children by her first husband. During the nine years of married life with her second husband she has borne no children, but has had four or five early abortions.

*Family History.*—Her father died of consumption when the patient was six years old. Her mother died of paralysis of the heart at the age of 56. She has a half brother and a sister in perfect health. Nothing further of interest is in the family history. She had the usual children's diseases, and a severe attack of inflammation of the bowels twelve years ago. Menstrual life began at 13 years of age, is regular and painless. Ten years ago the patient worked hard, so hard that men refused to do the work, kneading bread all day in a bakery. This she continued for about five years. She would use a barrel and a half of flour a day. As a strong, healthful, farmer's daughter, she had worked hard pitching hay in her early life.

*History of Disease.*—Her present trouble commenced about three years ago. The middle finger of the right hand began to be stiff, especially when she was sewing. It seemed like "a steel spring at times, and flew back." There was absolutely no pain, soreness or other sensory manifestation with it. Then the wrist became weak and dropped on the right side. Still there was no pain or soreness whatever, no numbness or tingling either. About four months later the same phenomena took place, in about the same way, in the left hand. When she would pull out the middle finger it flew back like a steel spring. Then the wrist became weak and dropped on the left side as it had done on the right. No sensory phenomena of any sort, however, appeared at this time anywhere. This condition remained more or less stationary until last Christmas, when the trouble seemed to pass to the arms and shoulder.

\* The patients were shown to the Chicago Neurological Society, March 29, 1906.

At present she can raise her left arm to the head, but not the right arm. There is wasting to a considerable degree in the deltoids, and to a lesser degree in the other shoulder and neck muscles. All of this is more pronounced on the right than on the left side. There is a distinct reaction of degeneration on electrical examination of the right deltoid, and partial reaction in the supraspinati and infraspinati, interosseal, and thenar muscles of the right side; and a very slight reaction in the corresponding parts of the left side. There seems to be no wasting of the latissimus dorsi and rhomboids and very slight, if any, in the pectorals. The left arm is somewhat softer and smaller than the right. The thumb muscles are slightly wasted. The hands seem to her swollen and somewhat larger generally than normal. They are feebly rigid and tend to assume the *main en griffe* attitude. The muscles of the neck, "the cords" she calls them, feel a little stiff and "seem to crack." She does not think she is any thinner about the neck. There have been creeping, fibrillary twitchings in the muscles of the right arm at times, but not in those of the left. The collar bone feels sore and slightly depressed.

There are no symptoms whatever in the legs; but the knee jerks are very decidedly exaggerated. No ankle clonus or Babinski could be elicited.

Until last Christmas there were no notable sensory symptoms in any part of the body or extremities; no local tenderness anywhere now or referred to in the past. Under the left shoulder there is now a little soreness, and under the right collar bone she feels a slight degree of soreness. There is absolutely no spinal tenderness on vigorous percussion; no tenderness along the course of any of the spinal nerves. There have been no neuralgic manifestations. There have been no bowel or bladder troubles of any sort.

The right pupil is markedly dilated, but contracts slightly, quickly and sharply on accommodation and light stimulation. This condition is probably due to ciliospinal irritation and not to a sympathetic lesion.

**Differential Diagnosis.**—There are some suggestions of syringomyelia in this case; but the latter disease is ruled out by the absence of the syringomyelic syndrome, for the pain and heat sense, as well as the tactile sense, are normal everywhere. Peripheral neuritis is excluded by the absence of all sensory symptoms; and for the same reason pachymeningitis cervicalis hypertrophica is also ruled out. The latter disease, were there present any sensory symptoms at all, might be well considered, in view of the fact that the patient has had two healthy children with her first husband, but a number of suspicious abortions with her second. The closest inquiry in regard to any specific trouble elicited continued denial.

**Treatment.**—The treatment consists of the daily injection hypodermatically of nitrate of strichnia gm. 0.003. After four weeks of this treatment, without any improvement in the paralysis, there seems to have been a little improvement in the appearance of the atrophied muscles around the right shoulder. The patient and her husband are very positive of this, though imagination may have something to do with it, I suspect.

The case of juvenile myopathy may be diagnosed as such clinically, though there is some question as to its being a pure example of Erb's type. The case is from the Cook County Hospital, where I was asked to see it in consultation, in the service of Dr. H. V. Halbert.

**CASE 2.**—G. H., aged 19, German, unmarried, watchman and laborer. Patient has indulged in beer, whisky and tobacco, but not to excess. Had measles in childhood and says that when young he was "deathly sick" several times, but from what cause or disease he does not know. He has never had typhoid fever or diarrhea prior to Christmas, 1905, when the present trouble seems to have begun. He had an attack of gonorrhea, July, 1905; syphilis is denied.

**History of Disease.**—On Dec. 28, 1905, the patient entered the Cook County Hospital, in the service of Dr. W. S. Harpole, and gave the following history: About two weeks previously he commenced to have diarrheal attacks, obliging him to go to stool three or four times a day and the same number of times at night. He formed no opinion of his own as to the cause of

this trouble. The stools increased to five or six a day and the same number at night. At one time they decreased in frequency and severity, but increased again, for which reason he entered the hospital. The stools, according to the patient's own account, were thin and watery, yellowish, sometimes odorless and at other times "putrid." Prior to coming to the hospital this condition existed for one week, during which time he ate no solid food whatever. He never saw any blood in the discharges. Vomiting began with the onset of diarrhea, and during the week prior to entering the hospital, occurred every day. Whenever he took a bite of food he became nauseated and immediately began to vomit. During the last two or three days of the week he vomited two or three times during the day and as often during the night. He thirsted keenly for cold water, but the drinking of it would set up an attack of emesis.

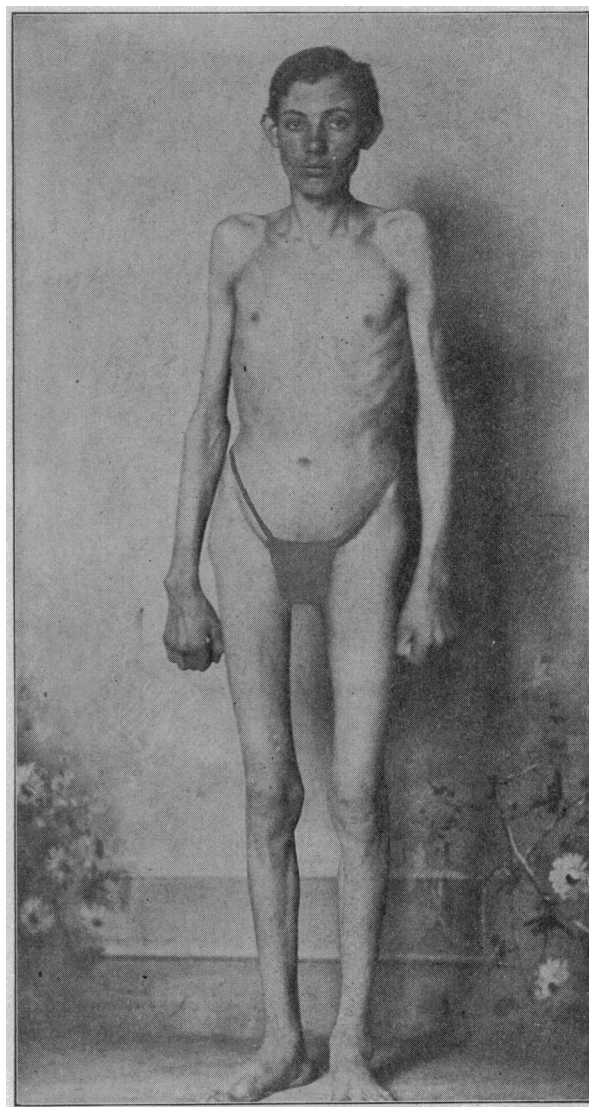


Fig. 1.—Juvenile myopathy.

He had no appetite for food and he believed he had lost ten pounds at least in two weeks. A slight cough was present. There was tenderness in the region of the stomach, and occasionally for a few minutes he would experience a dull pain in the stomach itself. He never had had night sweats; never had had any difficulty in urination.

**Examination.**—The record of the physical examination of the patient, made Dec. 28, 1905, shows no abnormality about the head. There were prominences of the intercostal, supraclavicular and infraclavicular spaces; winged scapulæ; narrow and emaciated thorax; no pulmonary, cardiac or pericardiac signs; no disturbance of the pulse, except a slight retardation and reduced tension; no abdominal tenderness, but a somewhat

scaphoid abdomen. Liver and spleen were impalpable; there were no evidences of fluid or tumor in the abdomen. Nothing of note was discovered in connection with the genitalia, the nervous and glandular systems, the extremities or the skin. Urinalysis revealed; color yellow, cloudy, acid reaction, sp. gr. 1,020, trace of albumin, no casts, no sugar, abundant deposit of urates. Examination of feces was negative.

Examination of the blood showed white corpuscles, 8,200; reds, 4,500,000 or 90 per cent.; hemoglobin, 90 per cent.; color index, 90/90-1. Differential—polymorphonuclears, 67 per cent.; large mononuclears, 20 per cent.; small mononuclears, 11 per cent.; basophiles, 2 per cent.; eosinophiles, 0; enucleated reds, 0; transitional forms, a few. The blood stained well and

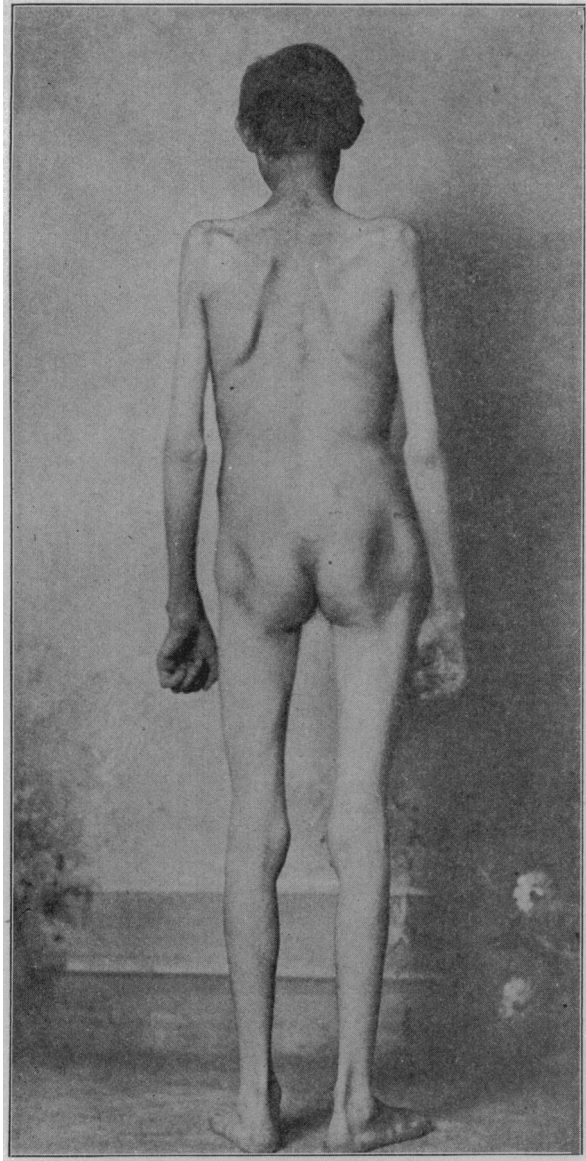


Fig. 2.—Juvenile myopathy.

coagulated rapidly. There was slight crenation and poikilocytosis of red blood corpuscles. Widal reaction, solution, 1-40, was not positive in 35 minutes. The temperature remained at all times practically normal, while the pulse ran a little subnormal. An antityphoid régime was adopted, and the patient left the hospital Jan. 6, 1906, recovered.

On March 13, 1906, the patient returned to the hospital. He says that almost immediately after he had gone out as cured he began to notice some pain and stiffness in his legs. The pain increased on walking. The muscles seemed to swell up and then grow small. Similar phenomena appeared in the thighs. After an interval of about ten days the same thing, or something like it, occurred in the shoulder and advanced down

the arm to the elbow. In the upper limbs these manifestations did not seem so marked to him as in the lower limbs. Finally, the neck muscles became gradually involved, so that he had difficulty in holding up his head. He could walk when he entered the hospital, but he complained of pains of a dull aching character in the right leg when he exerted it. According to the patient's own testimony, the left leg has made an almost complete recovery. He feels that he has lost 20 pounds or more in weight the last three months. The marked atrophy of the shoulder and pelvic girdle muscles is obvious. In the upper extremities, the deltoid, and infraspinati and supraspinati, the pectorals, and the muscles of the arm down to the elbow are decidedly wasted. The forearm is very little wasted, if any, and the hand is practically normal. The glutei muscles, the muscles extending the hip joint, and those of the thigh are wasted. The legs below the knee and the feet are practically normal. The appearance of the face is characteristic. The knee jerks are present but much reduced. There have never been any fibrillary contractions at any time.

On electrical examination the wasted muscles show a quantitative reduction in excitability to both Faradic and Galvanic currents. No reaction of degeneration or inversion of normal formulæ have I ever been able to detect. There are no sensory disturbances, nor any recorded except for the slight dull pains in the beginning. There are no disturbances of the sphincters.

A few words of interest may be said in regard to the etiology of the myopathies, and especially of this case. As we all know, heredity plays a dominant rôle, but cases have been reported by Erb himself, Gowers and others, in which no heredity could be traced, hardly even a neuropathic tendency. In our case certainly no heredity could be elicited. Other cases are on record in which infection, such as diphtheria, rheumatism, measles, scarlatina, played a very possible and even probable rôle. Gowers says, "As a rule no exciting cause can be traced. In a few instances the onset has succeeded some other morbid process, such as chlorosis, acute disease, rheumatic affections due to exposure to cold, and the depression of general health resulting from these may have determined the time of onset." In a case reported by Gowers the patient died from diarrhea. In connection with the history of infection that appears in this case it is to be noted that the winged scapulæ and marked depression about the shoulder girdle were made note of when the patient entered the hospital in December on account of diarrhea. The diarrhea may have been a mere incident, and the muscular wasting, unnoted, may have already begun.

In the next place a polymyositis of mild degree has been reported as occurring before these myopathies, and is suggested, in our case, in the soreness, transient swelling of the muscles of the leg, and other characteristics of the disease. The view has been put forth that some of these myopathies, if not all of them, are due to a transient mild degree of polymyositis. Jacoby<sup>1</sup> of New York reports an interesting case with many features like those of our own, and discusses most interestingly and plausibly the possible relationship between his case and cases like his and primary progressive muscular atrophy. He asks:

In short, is our case, perhaps, a case of progressive muscular atrophy in which the inflammatory stage is more marked clinically than usual, due to the myositis being more of a parenchymatous character? . . . Are not some cases of primary progressive muscular atrophy the resultants of light cases of polymyositis parenchymatous? We believe that there is considerable evidence in favor of these views and we do not stand entirely alone in possessing this opinion.

Wagner insisted, and Unverricht admitted the possibility of their cases being acute cases of primary progressive muscular atrophy. Wagner's cases showed a

1. Jour. of Nervous and Mental Disease, vol. xliii, No. 11.

duration respectively of three and eight weeks; Unverricht's a duration of six weeks. Gowers, Beevor and most authors believe that the pathologic change in the myopathies in the muscles is interstitial, that is, overgrowth of fat, proliferation of nucleated fibrous tissue between muscle fiber, with secondarily changes in the muscle fibers themselves, causing the narrowing and distortion of the fibers and their wasting.

A transient infectious polymyositis may, therefore, in our case, as has been suggested in many if not all cases of primary myopathy, have been present. The evidence, at all events, points away from a primary cord or nerve degeneration, and toward a primary muscular change.

But again, in these cases of myopathy there may be both a neural and a muscular process going on at the same time. In one case<sup>2</sup> the cord was normal except at the last dorsal segment, where there was found an area of granular disintegration in the intermediate substance on each side, but the cells of the anterior cornua were never atrophied, according to Beevor. Barsicow's cases and Zimmerlin's show that different, so-called types, of primary myopathy occur in the same family; and other cases have been reported wherein clinically neural disease was suspected and yet only myopathic changes were discovered and *vice versa*. Such cases lend color to the contention of Erb, who holds in contradistinction to Gowers and others, that the change in the muscle is primarily in the muscular substance itself, and secondarily in the interstitial tissue, and that the whole process is a functional disturbance in the trophic centers of the cord; thus, according to Erb, making of a myopathy both a muscle and a neural disease. These transitional cases, such as Barsicow's and Zimmerlin's, give support to the contention of many of our latter day physiologists that the lower motor neuron, with its associated muscle, constitutes a physiologic unit, a neuro-muscular apparatus. Grasset has even gone so far, in declaring for the solidarity of the lower neuron, as to affirm that an anterior poliomyelitis can not be differentiated from an inferior motor polyneuritis, because they are both but expressions of an inferior motor neuronitis.

The etiology and pathology of our case are such as not to exclude it entirely from the class of juvenile myopathies, though they do not give it the frank, clear-cut position demanded by the average text-book.

The symptomatology, especially in regard to the distribution of the atrophy involving as it does the shoulder and pelvic girdles, favors the diagnosis of juvenile dystrophy for our case.

cord; thus, according to Erb, making of a myopathy

In differentiating in doubtful cases between a dystrophy insists on the great importance of the grouping of the atrophied muscles. Thus, in spinal cases, he finds that the deltoid, infraspinati and supraspinati, clavicular part of the pectoral, biceps, brachialis anticus and supinator longus are in one group involved (fifth cervical root); the latissimus dorsi, sternal part of the pectoralis and triceps being involved in another group (sixth cervical root). In the myopathies, the biceps, triceps and supinator longus are wasted, while the deltoids and spinati muscles escape. In other words, he finds in the spinal atrophies that there is a physiologic grouping of the muscles according to the center of the cord involved; while in the myopathies there is no evidence of such spinal grouping. This, I think, is a little too rigid. Gowers has stated, in his description of the myopathies, that occasionally hardly any muscle of the body escapes.

He reports the case of a man, 27 years of age at the time of his death, with no history of any analogous case in the patient's family. The affection began at the age of 14, when the feet began to turn in so that he walked on the outer side of the foot. He soon noticed a gradual wasting of the legs, which slowly progressed, and at about the age of 24 involved the arms. When the patient was first seen, a few months before his death, the muscular atrophy was universal and the subcutaneous fat had completely disappeared. Even the hands were greatly wasted. There was a hollowing in the position of the thenar eminence, just as one sees in progressive muscular atrophy of the spinal type, and wasting in the interosseal muscles. There had been slight fibrillation in this case. The intercostal muscles were paralyzed. The wasting of the legs was extreme and the patient ultimately died with diarrhea. The muscles were found to be small and pale, the chief microscopic change being intense granular fatty degeneration of the fibers. A very few normal fibers were seen in some muscles. There was no increase of interstitial tissue. Careful microscopic examination revealed no morbid changes whatever in the spinal cord.

I am convinced that the mere distribution of the atrophy is not a sufficient basis whereon to distinguish special types of this disease. If, as it is believed, the primary trouble is some neural effect, the distribution of the atrophy would have to follow along physiologic lines represented in central functions. It is difficult to understand why certain areas for the atrophy should be selected as the prominent manifestation of a disease which is so generalized as an hereditary or infectious etiology would presuppose. On the other hand, if these primary myopathies are nothing but the late results of a transient polymyositis, as has been contended by some, it is also difficult to understand why there should be so much uniformity in the distribution of the atrophy in the so-called types. As a matter of fact, the types, as such, are exceedingly rare. The mixed or obscure cases are much more frequent and give strength to the view that these types, as such, do not really exist, but are the mere accidental clinical illustrations of a much larger pathologic process than they would presuppose or represent. In the myopathies there is partial preservation of the deep reflexes. In our case the reflexes were neither increased nor absolutely lost.

In the differentiation between spinal atrophy and primary dystrophy much has been made of the presence and absence, respectively, of muscular fibrillation. In our case, fibrillation seems to have been totally wanting, and it is usually regarded that absence of fibrillation is a cardinal sign of primary dystrophy. Gowers says that fibrillation is absent and that this is almost the invariable rule; and yet in his own case, referred to above, he mentions that there was slight fibrillation. Another point of value in the differentiation between progressive muscular atrophy of the spinal type and primary dystrophy is the absence in the latter of the reaction of degeneration or qualitative change in the electrical excitability. As in our case, only a quantitative loss of excitability to both faradic and galvanic currents was observed. On this point Gowers says very emphatically that in a primary dystrophy there is no trace of the reaction of degeneration.

In both progressive muscular atrophy of the spinal type and in the primary dystrophies, the sensibility remains practically normal. There may be in both slight transient pains of a dull character, possibly due in the former to irritation of the sensory elements, and in the

2. Clarke and Gowers, Med. Chir. Trans., vol. lvi, p. 247.

latter to the initial transient polymyositis, if, indeed, the dystrophies are at all myositic in origin.

As a result of my own observations, I feel like seconding Gowers when he remarks that "it seems, therefore, undesirable to form a separate variety of juvenile muscular atrophy, as Erb has proposed. Spinal atrophies may also be juvenile." So also may myositic atrophies.

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## PRETUBERCULAR CONDITIONS AND THE TREATMENT OF ASSOCIATED ANEMIA BY HYPODERMIC INJECTIONS OF IRON AND ARSENIC.\*

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DETROIT.

It is a well-recognized fact that pulmonary tuberculosis is the cause of more deaths than any other one disease, except pneumonia. In the state of Michigan our mortality record for some years has averaged above 2,500, while in Detroit our last report shows that 327 individuals died from this cause. We are all familiar with the fact that nearly one-seventh of all persons die of tuberculosis, and more than 50 per cent. of the remainder give postmortem evidences of tuberculous foci that had remained dormant or succumbed entirely to the antitoxic properties of our cells.

If this latter astounding and well-conceded fact is true, seven out of ten individuals that reach late adult life have been infected with tuberculosis.

The profession and the laity are awakened to the stupendous problem at hand. Many of us appreciate our inefficiency in battling with the ravages of this disease. We are especially apt to become therapeutic nihilists in the treatment of tuberculosis and to keep most prominently in mind the phenomena of hopeless advanced cases where all therapy has proved unsuccessful. It is with a hope of re-establishing an enthusiasm along the brighter and more encouraging fields of so-called pretubercular and early tuberculous conditions that I present this subject for your consideration.

Our daily routine work in the examination of the chest will divide our patients into five distinct degrees of disease.

First.—The acute miliary or the advanced chronic cases of pulmonary tuberculosis, generally incurable in any climate.

Second.—The moderately severe tuberculosis, a considerable number of whom will recover under proper treatment.

Third.—Early or incipient phthisis, 70 per cent. of whom it is estimated can be cured under favorable conditions.

Fourth.—Bronchopneumonitis and other non-tubercular lung diseases.

Fifth.—Cases with hereditary or acquired predisposition presenting prominent nutritional or constitutional changes, which we may classify for convenience as pretubercular conditions.

Every consumptive of the moderately or advanced type is putting up a fight for life, and we are helping him as best we can. The symptoms of hemoptysis or the chills and fever of the mixed infections are usually signals for a change of physicians or a consultation.

It is at these times that the relatives and the physicians become thoroughly alarmed, and no financial or physical effort can be too great to satisfy the anxious minds at these crises.

A most conclusive demonstration of the ravages of the disease up to the time of hemoptysis or when the evidences of mixed infection become pronounced is offered us by the *x*-ray. Two facts are at once apparent. First, phthisis pulmonalis progresses to small cavity formation usually before ordinary skill in percussion and auscultation reveals it; second, *x*-ray examination at the period of hemoptysis or at the onset of fever frequently demonstrates extensive tubercular disease all out of proportion to the physical signs. A keen knowledge of the diagnosis and treatment of the so-called pretubercular stage, which antedates outward tubercular manifestations, is exceedingly important from all points of view.

Those who take careful histories of chest cases and carefully analyze a series of them will most certainly be impressed with the great number of persons who report a prevailing impoverishment or morbid state of the whole system for a considerable period before the apparent development of the local lesions. The general practitioner finds a deep problem in the relations of hereditary and acquired predisposition to pulmonary tuberculosis, and his therapeutic indications must be promptly met.

If I were to state that pretubercular phthisis pulmonalis was as distinctly a stage of phthisis as phthisis incipiens, with prominent fixed diagnostic signs and manifestations, I am confident many would assent. How many of us vigorously supply the therapeutic indications in this curable onsetting stage of tuberculosis? Are we justified in terming this condition tuberculosis from postmortem findings in individuals of this type? No, but the patients should have the benefit of our suspicions. It is impossible to overestimate the value to the patient or the state of timely applied hygiene and treatment in the pretubercular stages.

Some very interesting questions arise along this line of thought. First, where does the so-called pretubercular stage of phthisis end and incipient phthisis begin? Second, what methods of diagnosis have we that differentiate the stages and what treatment must be applied? Third, how much tuberculosis of the lungs may exist before ordinary skill in physical diagnosis can detect it? If we can successfully thresh out these problems and throw the brunt of our efforts on patients exhibiting the phenomena of the pretubercular or the incipient stages, the mortality from consumption would certainly be lowered. The maxim of Tyndale is certainly logical: "that localized tuberculosis does not endanger life of itself so long as the general nutrition begets a reasonable resisting power of the pulmonary tissues." And especially the pulmonary gland tissues, I might add. This is demonstrated to us daily in the postmortem findings in accidental deaths.

We may divide our tuberculous cases into two grand classes. First, the infections that have taken place in strong healthy individuals, who have no evidences of predisposition, acquired or hereditary, such as renowned athletes or prize cattle. Second, those cases which present a prodromal period of impaired general condition from all causes. It is this latter class that I wish to consider.

It is my belief that the pretubercular stage of phthisis, as we now classify it, is, in the vast majority of cases, nothing more than a latent unrecognized tuber-

\* Read before the Wayne County (Mich.) Medical Society.