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FURTHER OBSERVATIONS ON ALCOHOLIC PARALYSIS.

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SINCE my last communication¹ the subject of Alcoholic Paralysis has received a great deal of attention from different observers. In this country Hadden² has described two fatal cases, in one of which a complete autopsy was made :—in France a thesis by Oettinger³ has been published, treating fully of the history, symptoms and pathological anatomy of alcoholic paralysis ; and another thesis, treating more of those cases which simulate locomotor ataxy, is about to be published. In Germany several papers on this subject have appeared. The more important are by Löwenfeld,⁴ Strümpell,⁵ Knicke,⁶ Lilienfeld,⁷ and Schulz,⁸ who gives an almost full analysis of the hitherto published fatal cases. Of American neurologists, Hun⁹ has contributed one paper containing the report of two cases, one of which ended fatally.

¹ 'BRAIN,' July, 1884.

² 'Path. Trans.' 1885, p. 49.

³ 'Étude sur les paralysies alcooliques,' Paris, 1885.

⁴ 'Arch. f. Psychiatrie,' xv. p. 438.

⁵ 'Berl. Klin. Wochenschr.,' 1885, No. 32.

⁶ 'Deutsch. Medicinal-Zeitung,' 1884, No. 72.

⁷ 'Berl. Klin. Wochenschr.' No. 45, 1885.

⁸ 'Neurol. Centralb.' 1885, Nos. 19, 20 and 21.

⁹ 'American Journal of Med. Science,' April, 1885.

Within the last twelve months I have seen quite an unusually large number of cases of alcoholic paralysis, and several others have come under the notice of my colleagues at the Manchester Infirmary. Considering the widespread attention which this subject has attracted, it may not be out of place to briefly relate some of these cases, and to touch upon some points relating to the history, symptomatology, and prognosis of alcoholic paralysis.

Concerning the history of alcoholic paralysis I find that Magnus Huss¹ has been credited with having given the first description of a paralytic form of chronic alcoholism. On looking up the literature of the subject, I found, however, that an American writer, Dr. James Jackson, had as early as 1822-3 given a very succinct and at the same time very accurate and complete account of alcoholic paralysis.² The affection itself, the author names from the most prominent symptom arthrodynia; he states that he chiefly observed the disease among females, and that it is produced by "ardent spirits."

Dr. Jackson not only describes the pain, the paralysis and atrophy, but also gives an account of the "glossy skin"—a trophic change well marked in some of the cases recently seen by myself, and which has also been observed by others.³ Dr. Jackson's description of the disease is worth quoting, especially as the original is not very accessible. "This arthrodynia comes on gradually. It commences with pain in the lower limbs, but especially in the feet; and afterwards extends to the hands and arms. The hands may be affected first in some instances; and in all cases in an advanced state, the pain is more severe in the feet and hands, than in the upper parts of the limbs. The pain is excruciating, but varies in degree at different times. It is accompanied by a distressing feeling of *numbness*. After the disease has continued a short time, there take place some *contractions* of the *fingers and toes* and *inability to use these parts freely*. At length the hands and

¹ 'Alcoholismus chronicus,' 1852.

² The paper is contained in the 'New England Journal of Medicine and Surgery;' Boston, vol. xi. (third series: vol. i. No. 4,) 1822, p. 351 "On a peculiar Disease resulting from the use of Ardent Spirits."

³ Oettinger, p. 37.

feet become nearly useless, the *flexor muscles manifesting*, as in other diseases, *greater power than the extensors*. The whole body diminishes in size, unless it be the abdomen, but the face does not exhibit the appearance of emaciation common to many visceral diseases. The *diminution is especially observable in the feet and hands*; and at some time *the skin of these parts acquires a peculiar appearance*. The same appearance is noticed, in a slighter degree, in the skin of other parts. This appearance consists in a *great smoothness and shining*, with a sort of fineness of the skin. The integuments look as if tight and stretched without rugæ or wrinkles; somewhat as when the subjacent parts are swollen, but the skin is not discoloured. Yet in this disease there is not any effusion under the skin, and the character which this assumes, arises from some change in the organ itself." . . . (The *italics* are my own.)

The pains, present in the pseudo-ataxic form, have likewise been noticed by this accurate observer, for he says, "The most characteristic symptoms are manifested in the limbs; but the pain is not limited to these, and other symptoms are exhibited in other parts. The pain sometimes *shoots suddenly up one or both legs*, and in one case it frequently passed up the back and then forward to the pit of the stomach, taking the course of the diaphragm."

According to the more prominent symptoms, I have divided clinically this peculiar affection into two groups: the alcoholic ataxia, and the alcoholic paralysis. This classification has been adopted also by several authors—Löwenfeld, Moeli, Schulz, Strümpell and Krücke—who speaks of it as "Pseudo-tabes of alcoholics."

In considering the ataxic form, we have to distinguish between those cases where there is marked inco-ordination without much paralysis, and those where the gait resembles the ataxic gait, but is in some measure due to the paralysis of muscles of the lower extremity. The ataxic form represents a milder type; pathologically, however, as is evident from the cases reported by Déjérine,¹ Moeli,² and the case given below it is the same affection, namely, a peripheric neuritis.

¹ 'Arch. de Phys. norm. et path.,' 1884, No. 2.

² 'Charité Annalen,' 1884.

Of the purely ataxic form I have observed three cases, two of which have quite recovered, whilst the third died from uræmia, due to a contracted, gouty kidney.

The *first* case was a man, æt. 32, whom I saw with Mr. Fitzmaurice, of Bury. The patient had been for some years a heavy drinker, and had also indulged, to an extraordinary and almost morbid degree, in venereal excesses, but has never had syphilis. He complained of severe, lancinating and shooting pains in the lower extremities, sometimes in the upper extremities; both lower extremities showed some spots of anæsthesia and of retarded sensibility; the muscles of the calf were painful on pressure; there was no atrophy or paralysis of either upper or lower extremity, but there was marked inco-ordination. With his eyes open the patient could walk fairly well, and lifted the feet well from the ground, the heels coming down first; with his eyes shut, he showed marked ataxia; there was also distinct inco-ordination for finer movements in the upper extremities; the tendon reflexes were absent; no Argyll-Robertson symptom; no ophthalmoscopic changes. The patient gave up the use of alcohol, and completely recovered; and Mr. Fitzmaurice informs me that the tendon-reflexes, which remained absent for some time after the recovery seemed complete, have now returned.

The *second* case is a patient who is a boarder at the Cheadle Asylum, suffering from Dipsomania, and whom, in my capacity as one of the visiting physicians to that Institution, I had an opportunity of seeing frequently. For the notes, I am indebted to Mr. Reynolds, M.B., one of the assistant medical officers.

The patient, æt. 33, single, had an attack of sunstroke at Ceylon when 26 years old, and after that became very nervous and took to alcohol. He came to England, stayed some time, and drank very heavily; went back to Ceylon, and finally returned in 1882. He went to a Home for Inebriates, then to Loxley Hall, and finally came to Cheadle. He has never had syphilis.

His brothers are heavy drinkers, and one of them suffers from epileptic mania. In May 1884, he commenced to complain of pains in both legs and feet, especially on the dorsal surface of the feet near the toes, which were swollen; the pains were

shooting in character. There was hyperæsthesia of the skin over these parts, and some spots of anæsthesia; he had also pains in his fingers. He became very irritable in temper, lost his memory for recent events, and refused his food. There was loss of tendon-reflex, some slight atrophy of both legs, and marked ataxic gait. There were no bladder or rectum symptoms; the atrophy increased, the pains, however, became less, and the patient walked better. Towards the end of September 1884, the pains ceased altogether; the atrophy, however, remained, yet the patient walked much better. At the beginning of March 1885, he had pains over the distribution of the left supra-orbital nerve; he again complained of pains in his legs, burning sensations in the soles of his feet, and cramp in his legs.

In May 1885, I saw the patient myself. I found him somewhat irritable; otherwise but little affected mentally, except that he had lost his memory for recent events.

The gait was feeble and slow; he walked with the help of one stick, but his gait was distinctly ataxic, and became more so when he attempted to walk with his eyes shut. There was some emaciation of the lower extremities, but no marked paralysis of the muscles; the movements of the extensors of the toes were, however, sluggish. There was some slight atrophy of the muscles of the back and also of the arms, but the patient could flex and extend both fingers and wrists very well.

The patient complained of shooting-pains down the legs to the toes, occurring in paroxysms, and leaving a numb feeling behind. Firm pressure on the soles of the feet and on the legs was very painful. The patient also noticed a cold feeling when touched by any object. (This symptom Mr. Reynolds had noticed on several occasions previously.)

The limbs showed no vaso-motor disturbances.

The tendon-reflexes were absent.

The galvanic reactions were taken by Mr. Reynolds.

The contractions were slow, and followed by fibrillar tremors.

Extensors of the foot . . .	ACC and AOC, with 10 cells.
	KCC, with 15 cells.

A very strong magneto-electric current was necessary to get contractions of the extensors of the toes and calf-muscles.

The muscles of the upper extremities reacted well.

There were no eye or bladder symptoms.

There was loss of appetite, craving for drink ; the tongue was furred ; there was morning vomiting and occasional hæmatemesis.

The bowels somewhat constipated. The liver and spleen were not found to be enlarged.

The pulse was feeble and irregular.

As the patient refused to eat unless he had some drink, he was allowed beer. For the pains he had morphia injections at night, which gave him great relief.

The patient soon began to improve, and again went to Loxley Hall. He has returned from there recently, and is now quite well. He has gained flesh, he has no pains, there is no ataxia, and he shows no abnormality whatever in walking. The tendon-reflexes are normal, and the patient is now only kept as a boarder at his own request. He has never discontinued to drink beer, but avoids all other stimulants.

The *third* case was a female patient, æt. 53, who was admitted into the Infirmary, complaining of pains of shooting character in her lower extremities. Three years ago she suffered from gout in her hands. For some time she has been troubled with paroxysms of pain in her legs, without any swelling of the toes or any of the joints ; she has also noticed that her gait was awkward, and that she could not walk well in the dark. On admission, it was found that she had a typical alcoholic appearance ; the skin was dry, there was a nodular swelling on the metacarpo-phalangeal joint of the left index-finger. The patient looked thin ; there was no œdema, and there was no marked paralysis of any of the muscles. She could walk, but felt weak on her legs ; her walk was ataxic, and it was impossible for her to stand or walk with her eyes shut. She could move her limbs freely when in bed ; the tendon-reflexes were absent ; there was some anæsthesia of the skin of the lower extremities ; but very marked hyperæsthesia of the muscles of the calf and of the muscles of the forearm ; pressure on these parts caused most excruciating pain. The heart was found hypertrophied ; the urine of sp. gr. 1010, profuse in quantity, contained albumen and granular casts ; there was anorexia with

vomiting. The pupils reacted well, the fundus of the eye was normal.

Some time after admission the patient became delirious; there was incessant vomiting for twelve hours, and then the patient had a convulsion and died.

At the post-mortem examination made by Dr Harris, Pathologist to the Infirmary, the kidneys were found small and granular; the left weighed $1\frac{1}{2}$ oz., the right 1 oz.; on section, the cortical substance was found very much diminished, and streaks of urate of sodium were seen passing to the medullary part. Microscopically, marked interstitial nephritis was seen, with extensive deposits of urate of sodium crystals in the renal tubes. The heart was very much hypertrophied, and weighed $13\frac{1}{2}$ oz.; the myocardium was healthy, the valves normal. The liver weighed 3 lbs., and microscopic examination showed marked amyloid changes, together with monolobular cirrhosis, the fibrous tissue being still of very embryonic type; the spleen weighed 11 oz. The brain was anæmic; the ventricles contained more fluid than usual; the membranes of the brain were healthy; the pia mater, however, slightly opaque. The pons, medulla, and spinal cord had a perfectly healthy appearance, and were of firm consistence. The muscles of the leg and forearm were thin and pale. The sciatic, the ant. crural, and the musculo-spiral nerves were preserved for microscopic examination, along with the cord, and some muscles from the back of the leg.

The spinal cord, examined carefully after having been hardened, was found perfectly normal in all its parts.

The sciatic nerve appeared thin, grayish, and was surrounded by a great deal of adipose tissue. Vertical sections showed, when treated with perosmic acid, and stained afterwards with picrocarmin, a moniliform appearance of the nerve tubes, due to breaking up of the myelin; the nuclei were increased, and there was also some interstitial cell infiltration. Transverse sections showed in some few places an increase in diameter of the axis-cylinder, and again the interstitial infiltration.

The muscles showed chiefly increase of the muscle nuclei and an interstitial deposit of small round cells, and in some few places the striation was not well marked.

This case, both in its clinical history and in the post-mortem appearances, resembles very closely the first of the two cases described by Déjérine¹ as peripheric nerve-tabes, and there can be no doubt that both the cases described by this author are cases of alcoholic ataxia. We omitted in our case to remove portions of the skin, and I regret very much not to be able to state anything about the condition of the subcutaneous nerves, which were found markedly affected in Déjérine's cases.

The following case, which was admitted into the Infirmary only a few days ago, shows the combination of alcoholic ataxia with alcoholic paralysis.

W. B., æt. 41, was admitted into the Infirmary Nov. 10, 1884. The patient had been very intemperate and has a distinctly alcoholic appearance. He has had rheumatic fever, and has lately been very much troubled with pains in his limbs. He stated that he has had three attacks like the present, but not so bad, from which he recovered, after rest and abstention from drink.

The patient looks strong and stout; some of the muscles feel flabby, but there is no marked atrophy, though paralysis of some of the muscles is distinct.

In the upper extremity there is marked paralysis of the extensors of the fingers and of the wrists on both sides; some of the other muscles (triceps, deltoid) are slightly affected; the flexors act very well. Such movements as the patient is able to carry out show some inco-ordination.

In the lower extremity the extensors of the toes and of the big toe are but slightly affected; the peronei on both sides, however, are considerably paralysed; the arch of the foot is flattened, and the inner border is raised, whilst abduction is impossible.

The patient is able to walk with some assistance, but his walk is ataxic; he keeps his legs apart, and has to look to the ground for fear of falling; he cannot walk with his eyes shut. Isolated movements with either of the lower extremities show equally marked inco-ordination. With the eyes shut the patient does not know exactly the position into which his limb is put.

¹ 'Arch. de Phys.,' 1884, p. 231.

The sensory phenomena are those of alcoholic paralysis. The patient complains of shooting pains in his legs. There is cutaneous anæsthesia in both upper and lower extremity, irregularly distributed, with extreme muscular hyperæsthesia. There is also great pain if the skin apart from the muscles is firmly grasped. The sense for temperature is affected in both legs; the patient rightly distinguishes a cold body, but contact with a hot body gives him a sensation which he compares to that of an electric shock. There is some analgesia, and the prick of a pin is felt only after some time.

The tendon-reflexes are absent; the superficial reflexes fairly normal.

The pupils are normal, and react to light and accommodation; the special sense-organs are normal.

The mental condition of the patient shows symptoms which are often found in cases of alcoholic paralysis, and which have been noticed by other observers, without, however, laying great stress on them. The patient answers questions rationally; his memory, however, is very defective, and he suffers from delusions. Thus he tells you that he gets up every day, goes into the next ward, and converses with the other patients, though, as a fact, he has never left the ward since his admission. When further pressed, he even gives a description of the ward, details his conversation with the patients with a minuteness and readiness which is astonishing. I have noticed exactly the same peculiarity of the mental state in some of the other cases to be presently described.

The condition of the other organs calls for no special notice. The heart-sounds are normal, but weak; the pulse 120, compressible; appetite fairly good; the liver is normal; urine free from albumen.

We have so far been able to take the electric reactions to the galvanic currents only once, and muscles showed degenerative changes.

Peronei of right side	KCC, with 30 cells.
	ACC „ 25 cells.

The other cases which I have as yet to describe belong to the typical class of alcoholic paralysis, characterised by

well-marked paralysis with atrophy, affecting chiefly the extensors of the fingers and toes.

The paralysis and atrophy sometimes come on very acutely, at others more slowly. When these cases come under observation, the patients are as a rule unable to stand or walk, and it is therefore not easy to make out whether the paralytic stage is here preceded by an ataxic stage. As the sensory phenomena in these cases are the same as in the first group, it is highly probable that where the paralysis comes on slowly, pseudo-ataxic symptoms, as in the case just given, precede it. Of this group I have within the last twelve months seen four cases, all females, two of which have quite recovered, one is beginning to improve, whilst the fourth, when last heard of, had as yet shown no signs of improvement. As the symptomatology of these cases is now sufficiently well known, I will very briefly relate the cases, only drawing attention to particular points.

CASE I.—A lady, æt. 25, whom I saw with Mr. Renshaw, of Altrincham, in August 1884.

The patient had freely partaken of spirits, and had a constant craving for stimulants.

She had complained of some pain for some time; the pains were soon followed by paralysis and atrophy of the extensors of both fingers and toes, with paresis of the other muscles, so that the patient could neither stand nor walk, and could raise herself only with difficulty from the recumbent posture. Cutaneous anæsthesia and muscular hyperæsthesia were well marked. Tendon-reflexes absent, superficial reflexes much diminished.

The patient exhibited the same peculiar delusions just referred to. When I saw her she told me that she could walk very well, that she had paid several visits that morning, and minutely described all she had seen and done when visiting her friends. When her helpless condition was pointed out to her, she became highly emotional, and burst into tears.

The patient was removed to a small cottage, and attended by a reliable nurse, so that she had no access to drink; she completely recovered in three months. Mr. Renshaw informs

me that she now walks very well, and that the tendon-reflexes have reappeared.

CASE II.—A lady, æt. 42, whom I saw with Mr. Kitchen in September, 1884. Her symptoms resembled those of Case I. The paralysis had come on rather more slowly, and the atrophy was much more marked, and affected the greater part of the body. This patient also had the same peculiar delusions. Though entirely confined to bed, she told us that she went out every afternoon for a walk to the seaside; that she sits on the sand at the seashore, watching the waves and the passing steamers. (Her residence was at least twenty miles away from the sea.)

It was astonishing to listen to the account she gave of her seaside rambles, which had every semblance of truth, and yet was entirely imaginary.

We advised the patient's removal, if possible, as we were sure that the patient, unknown to her husband, was still supplied with stimulants. Our suggestion was, however, not acted upon, and when last I heard of the patient, she was still in the same helpless state, and rather more emaciated.¹

CASE III.—E. C., æt. 33, seamstress, married, was admitted into the Infirmary, Dec. 3, 1884, suffering from typical alcoholic paralysis.

She had suffered from "rheumatic" pains at different times. Three months before admission, the pains in her arms and leg became much worse; her legs often "gave way" when she attempted to walk, and a corresponding weakness was noticed in her arms. The weakness increased to such an extent, that she was obliged to keep to her bed, and had to be fed, not being able to use her hands. She admitted having partaken of alcohol in large quantities.

The patient had a characteristic alcoholic appearance. The muscles of the upper and lower extremity felt flabby; the extensors of the fingers and toes were quite paralysed; the

¹ Since the above was written, I have heard from Mr. Kitchen that the patient has lately much improved, and is now able to walk with the help of sticks. She has, however, the same delusions still. She has entirely abstained from spirits, but takes several glasses of beer every day.

hands presented the characteristic drop-wrist; the feet, the paralytic club-foot; the flexor muscles of the fingers and toes were paretic; the arm and shoulder muscles were fairly strong. The tendon-reflexes were absent, the superficial reflexes normal. There was also cutaneous anæsthesia in the legs and fore-arms, with marked muscular hyperæsthesia on firm pressure. The sense of temperature was affected on the outer side of each leg; the patient was able to distinguish cold, but not heat. The paralysed muscles were atrophied to a much greater extent than the paretic muscles.

The patient complained of loss of memory, but otherwise her mental condition was normal. The right pupil was irregular from an anterior synechia.

The application of the electric current was very painful.

A very strong faradic current produced no contraction in the extensors of the hand; the flexors responded to a strong current, but not to a weak current. With the galvanic current directly applied to the muscles, there was found to be degenerative reaction in the extensors of the fingers, the extensor carpi ulnaris, the extensor communis digitorum, and extensor longus pollicis. Some of the flexor muscles also showed degenerative reaction.

The patient remained in the hospital till March 1885, when she left us somewhat improved; she has since then completely recovered. She has lately presented herself in the out-patients' department of the Infirmary, and no trace of any paralysis or atrophy could be discerned. The tendon-reflexes had reappeared.

CASE IV.—M. E., æt. 43, was admitted into the Infirmary on July 13, 1885, under Dr. Leech, who kindly transferred her to me. The patient admitted having partaken of stimulants (whiskey and gin), and gave the usual history of rheumatic pains, with which she had been troubled for some time, followed by weakness which had increased to such an extent that she was rendered quite helpless. On admission, her condition resembled very much that of Case III., except that the paralysis was much more extensive. The patient was very stout; the muscles flabby; she was unable to raise herself from the recum-

bent posture; hands and feet were flexed; there was marked wrist-drop, the elbows were bent, the knees drawn up. The shoulders and hips could be moved, but the movements were sluggish, and when passive movement was attempted, the patient screamed with pain. The tendon-reflexes were absent, the superficial reflexes not well marked.

There was again cutaneous anæsthesia for tactile impressions, with well-marked muscular hyperæsthesia, affecting upper and lower extremities, and to some extent also the muscles of the abdomen. The sense for temperature was normal.

The skin on the dorsal surface of the fingers of both hands had a marked glossy appearance.

The patient's mental condition is affected; she answers questions rationally, and shows no incoherency in her answers, but she has delusions. She fancies she is at home, &c.; she is very restless at nights, and sleeps but little; she is also very emotional.

The tongue is covered with thick white fur; there is anorexia, occasional vomiting, and constipation. The liver and spleen are normal in size.

To the faradic current some of the muscles (triceps and extensors of the fingers and toes) do not respond at all; some, *e.g.* the biceps, supinator, the interossei, contract with a current of medium strength.

The application of the electric current is so painful, that the galvanic reaction could only be tried in the most affected muscles, and here marked degenerative reaction was found, thus:—

Extensors of fingers (left)	ACC = 15 cells.
	KCC = 25 "
	AOC = 20 "

The flexors also showed degenerative reaction, thus:—

Flexors of fingers (left)	ACC = 30 cells.
	KCC = 40 "
	AOC = 45 "

The patient has, up to now, made some slight progress; the extensors of the fingers on the left side show some power, likewise the extensors of the toes on both sides. There are

now, however, very marked contractures of the elbows, shoulders and knees, with absence of deep reflexes. The contractures are more pronounced on the right than on the left side. There is also some slight swelling over the back of the right hand (the tenosynovitis of Gubler, as in lead palsy).

The muscular hyperæsthesia is better.

The mental condition of the patient has undergone marked improvement; she has now no more delusions, and has no recollection of her former delusions.

I have no wish to prolong this paper by any further remarks. The symptoms of the disease are so clear, that there is no difficulty in diagnosing the affection.

The peculiar form of delirium seems almost as characteristic, though by no means as constant, as the muscular hyperæsthesia. Glynn¹ has already drawn attention to it. It occurs in chronic alcoholism without paralysis; and Mr. Mould, Superintendent of the Cheadle Asylum, informs me that he has constantly met with it amongst dipsomaniacs.

As regards the pathology, we have now abundant evidence in the post-mortem appearances that alcoholic paralysis is a multiple peripheral neuritis. This view is again supported by the third case of alcoholic ataxia given above. For an analysis of the results of the post-mortem examinations in alcoholic paralysis, I must refer to Schulz.²

¹ 'Liverpool Medico-Chirurgical Journal,' July, 1883, p. 374.

² 'Neurol. Centralb.,' 1885, No. 21.