Three cases of alcoholic paralysis (multiple neuritis) / by David W. Finlay.

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Alcoholic Paralysis

by

David W. Finlay

[Medico-Chirurgical Trans. 1884]

With the Author's Compliments.

THREE CASES

OF

ALCOHOLIC PARALYSIS

(MULTIPLE NEURITIS).

BY

DAVID W. FINLAY, B.A., M.D., F.R.C.P.,

PHYSICIAN TO THE MIDDLESEX HOSPITAL AND TO THE ROYAL HOSPITAL FOR DISEASES OF THE CHEST.

Read May 24th, 1887.

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Received January 11th—Read May 24th, 1887.

It is only comparatively recently that paralyses, formerly thought to be due to a central lesion, the proof of which could not be found, owing, it was supposed, to defective methods of pathological investigation, have been traced to their true source in the peripheral nervous system. Perhaps the most important factor, in this country at least, in the production of this form of disease, is the abuse of alcohol, and it is to the class of cases which own this ætiology that those belong which form the subject of the following paper. All three were admitted into the Middlesex Hospital during last year (1886), and I may say, to save repetition, that all were married females.

My first case, E. F—, æt. 28, came under observation on January 26th. There was nothing suggestive in her

family history, but she was stated by her former medical attendant to be a woman of loose morals and a chronic drunkard. She had had four children, three of whom were stated to have been stillborn; and she had suffered from two previous attacks of the same nature, apparently, as the present, occurring after confinements, and lasting for from five to six weeks each.

History of present illness.—She has complained of more or less weakness in the knees for nearly a year, the weakness being noticed particularly on going upstairs. In August and September of 1885 she had an attack of what was called "congestion of the liver," and complained then of being very weak. About the beginning of November she experienced some difficulty in walking, and this has gradually increased. About the end of the same month she began to complain of numbness in the fingers, and was said to have no feeling in the forearms when pinched. Soon after this double wrist-drop was noticed. Gradual wasting, most marked in the arms and legs, has been observed during the last three months, and for about four weeks she has been wandering in her mind.

Present condition.—Pulse 108, small, regular, compressible; resp. 24, laboured; temp. 102·4°. The tongue is red at tip and edges, dry and tremulous, coated on the dorsum with a thick, brownish-white fur; the skin is hot, dry, and harsh. She is much emaciated, has an apathetic expression, and lies helplessly almost on her back in bed, with the legs flexed, and resting on their right side. The face is pale, except on the cheeks, where there is an irregularly-shaped pink flush. The pupils are moderately dilated and equal, acting to light and accommodation. The lips are dry, teeth decayed, gums congested, and there is a dark red line on them close to the teeth. The voice is feeble and the speech rather thick. She has an inefficient paroxysmal cough unaccompanied by expectoration. The thorax is large and rounded; the respiratory movements are chiefly upper costal, and are increased in amount as regards elevation but not as regards expansion. During inspira-

tion there is recession of the epigastrium. Over the front of the chest the percussion note is somewhat increased in resonance ; breath-sounds are harsh, expiration prolonged, and abundant large and small râles are heard in the mammary and infraclavicular regions on both sides, chiefly during inspiration. Behind the percussion note is clear, but numerous dry and moist sounds are audible. The heart's maximum impulse is found in the third left interspace an inch within the nipple line ; it is feeble but well defined. The area of dulness extends from the upper border of the third cartilage to a point in the fourth left interspace an inch within the nipple. At both apex and base the sounds are shorter than usual, but there are no murmurs audible.

The abdomen is soft and flaccid, and marked by lineæ atrophicæ. It recedes during inspiration and is bulged during expiration. The liver reaches to about an inch below the costal margin in the nipple line. The area of splenic dulness is normal.

Patient has the delusion that there are several children in the bed with her ; she cries readily, as when she is being examined ; rambles in her talk and is forgetful. Muscular power in both arms is feeble, the muscles of both arm and forearm are wasted, and there is double wrist-drop, the hands hanging like flails. Extension causes pain and there is no power of supination. Superficial and deep reflexes are absent, but sensation is perfect everywhere. The right forearm at its thickest part measures $7\frac{3}{8}$ inches and the left 7. The interossei of the hands and the muscles of the ball of the thumb are greatly wasted. Round the middle of the biceps the right arm measures $7\frac{1}{4}''$, the left $8\frac{1}{8}''$. The legs are also much wasted, but sensation is perfect, and there is no œdema. She can draw the legs up and move them slightly, but there is no movement of the feet, which are " dropped " like the hands. There is no patellar reflex, no plantar reflex, and no ankle clonus, the attempt to produce which seems to cause considerable pain. There is also pain on passive movement

generally, particularly on movements of extension. She has no difficulty in swallowing and has complete control over bladder and rectum. On ophthalmoscopic examination the fundus of each eye appears quite normal. Examination of the throat shows nothing abnormal, and no nodes are found on the bones. There is no facial or ocular paralysis, and the facial muscles are not wasted although those of the neck are.

The urine is opaque and yellow in colour, acid, having a specific gravity of 1028, deposits an abundant sediment of lithates, and is free from albumen and sugar.

Electrical examination of the muscles of leg and arm by Dr. Pasteur, the Medical Registrar, to whom and to Dr. Pringle I am indebted for all the electrical investigations, gave the following results:

Left forearm.—No contraction front and back to *faradic* current.

Galvanism:

Supinator longus	A.C.C. (μ 7)	= C.C.C.
Ext. carpi ulnaris	A.C.C. (μ 5·5)	= C.C.C.
Ext. carpi radialis	A.C.C. (μ 6)	> C.C.C.

The response was immediate and the contraction voluminous and slow.

Left leg.—No contraction to *faradic* current of the following muscles: Tibialis anticus, extensor communis, peroneus longus, peroneus brevis, extensor brevis, gastrocnemius.

Galvanism:

Tibialis anticus	A.C.C. (μ 5) > C.C.C.
Extensor communis	A.C.C. (μ 5) > C.C.C.
Peroneus longus	A.C.C. (μ 8) = C.C.C. (mere flicks).
Peroneus brevis	A.C.C. (μ 8) = C.C.C. (decided).
Extensor brevis	No contraction.
Gastrocnemius	No contraction.

No contraction of intercostals is obtained either with faradism or galvanism.

No change in her condition was recorded until the evening of January 31st, when she had an attack of

syncope, for which a small quantity of brandy was given her. During the night she slept well and in the morning had rallied somewhat. The grasping power of the hands, however, was very feeble, and she passed her motions involuntarily.

On Feb. 9th it was noted that the heart's first sound was murmur-like and rough. Her general appearance was improved although the cough was still troublesome and the breathing laboured and rapid.

Next day (Feb. 10th) she was much worse; the breathing was still rapid (32) and shallow; she had a wild appearance with flushed cheeks, glistening eyes, lips dry, and covered with sordes; the tongue also was dry and fissured and coated with a thick yellow fur; pulse 126, small, feeble, and irregular. Over the chest generally mucous râles were audible both with inspiration and expiration.

During the succeeding night she was restless and sleepless, and at 3.45 a.m. she died rather suddenly from failure of the respiration.

The temperature was febrile throughout, being rarely below 100° F. and often as high as 103° F.; the respirations were double the normal rate, and the pulse averaged about 120.

At the post-mortem examination (made eight hours after death) the only gross pathological appearances were found in the liver and lungs. The former was large and fatty throughout. As to the lungs, the left was adherent to the chest wall; each had cavities at the apex surrounded by areas of caseation which were undergoing rapid softening. The rest of the upper lobes presented small caseous nodules of recent tubercular disease, and in the lower lobes there were recent tubercles extending down to the base. The middle lobe of the right lung also showed recent infiltration and a small cavity.

In the brain and its membranes there were no obvious changes. The membranes of the cord were normal. The cord itself was firm throughout. On section the grey

matter was so inconspicuous as to be scarcely distinguishable from the white; it appeared to be generally shrunken. It may be noted in passing that this appearance has been reported by Dr. Hadden in one of his cases.[1]

Microscopical examination.—The cord together with portions of the left median and musculo-spiral and of the right anterior crural and plantar nerves and extensor carpi radialis longior muscle were hardened in a 2 per cent. solution of bichromate of ammonium, and after being placed in spirit for some months were cut with a freezing microtome and stained with various reagents. The results as regards nerves and muscle are seen in the preparations now shown to the Society (see Plates VII and VIII).

The cord, a section of which is also shown, appears to be normal in all its divisions—cervical, dorsal, and lumbar, excepting, perhaps, that to the naked eye the grey matter does present in certain sections something of the shrunken look which was referred to in the report of the post-mortem examination. Under the microscope, however, there is nothing to distinguish it from a cord free from all suspicion of disease, in particular the large cells in the anterior horns appear everywhere healthy.

Right plantar nerve.—In transverse sections the perineurium is seen to be thickened, and there is an increase in the connective-tissue nuclei. The walls of the vessels are also thickened and infiltrated by numbers of round-cells (leucocytes). The nerve-bundles show an increase in the nuclei of the sheaths, and there is a great excess of fine fibrillated connective tissue in the endoneurium (fig. 1).

Under a high power these appearances are brought out with greater distinctness; the perineurium is infiltrated with round-cells in parts, some being also scattered throughout the endoneurium. There is scarcely a sound nerve-fibre to be seen, the axis-cylinders having for the most part disappeared, and the medullary substances

[1] 'Trans. Path. Soc.,' vol. xxxvi, p. 53.

being granular where the fibres themselves have not become altogether indistinguishable. A few axis-cylinders, however, still remain, some of which are much swollen (fig. 2).

In longitudinal sections, and in specimens where the nerve has been teased out merely in glycerine, the granular degeneration is well seen.

The myelin is segmented and the tubules irregular in their diameter, being empty and fined away to a thread-like prolongation where the myelin has disappeared, and bulged where it has accumulated in degenerate masses (fig. 4).

Right anterior crural nerve.—In this nerve similar conditions exist but the degenerate changes are less extreme. Many apparently sound fibres showing clearly the axis-cylinders occur in the transverse section; and in the longitudinal the segmentation and granular condition of the myelin are less marked; the perineurium is also less thickened.

Left median nerve.—In transverse section increase in the connective tissue of the endoneurium is well seen, and there are many atrophied nerve-fibres. Some of the fibres are rather swollen and granular showing no axis-cylinder. A considerable proportion, however, appear quite normal. The connective-tissue nuclei and leucocytes are abundant.

Left musculo-spiral nerve.—The same description applies to this as to the median, except that there appears in transverse section to be a larger relative number of sound fibres. In the longitudinal section, however, most are degenerated at some point, and the segmentation and aggregation in masses of the myelin are conspicuous.

Right extensor carpi radialis longior.—The section of this muscle shows an enormous increase in the nuclei of the connective tissue and of the sarcolemma, with numerous leucocytes. The muscle-fibres are diminished in number and size, and the bundles are widely separated; here and there the fibres are almost completely hidden by the masses of nuclei (fig. 5).

Areas of fat-cells occur in places between the muscular bundles, in which are embedded vessels with thickened walls ; and in one of the sections are seen two fine nerve-twigs cut across, which present the same degenerative appearances, with thickened perineurium and nuclear proliferation, as are seen in the larger nerves.

Spinal nerve-roots.—These are sound in all regions of the cord.

My second case, M. J. P—, was brought into the hospital on November 19th, 1886, unable to walk or even stand. Her age was stated to be 28, although she looked older. She had had three children, one of them stillborn ; had always previously enjoyed fair health, and had been a teetotaller until four years ago. Since then she had been in the habit of drinking heavily, gin being her favourite beverage. Latterly she had been almost continuously intoxicated. There was nothing of importance in the family history.

History of present illness.—Her illness was stated by her husband to have come on eight weeks before admission. She then began to feel weak, and to suffer from loss of appetite, losing power in her legs, and staggering in walking. A fortnight later, failure of power in the hands and wrist-drop were observed. She had also wandered in her talk and slept badly.

Present state.—On admission she is described as a corpulent woman with florid complexion, tremulous, and coated tongue and offensive breath, complaining of loss of power in legs and hands, with numbness in the legs ; of pains also in the limbs especially on movement. She lies in bed on the right side with the legs flexed, and is unable to turn or to extend the legs without assistance.

The examination of chest and abdomen shows nothing abnormal except that the heart's sounds are very feeble, and there is some enlargement of the liver. Her voice is feeble, her speech thick, and she has delusions. There is no facial or ocular paralysis and the pupils are normal. When

she lies on her back the arms are folded across the chest, and there is well-marked wrist-drop on both sides with some wasting of the muscles of forearm and hand. The fingers are flexed, and she cannot fully open the hand; there is no power of extension of the wrist, but she can move the arms so as to cross them over the chest or put them up to her face. Any manipulation of the arms causes pain. The grasp of the hands is exceedingly feeble. The legs below the knee are wasted and the muscles flabby. She cries out with pain when the muscles or nerve-trunks are pressed upon. She is unable to move either leg to any extent, and all passive movements cause pain. The feet are "dropped;" there is no œdema. Sensation of the skin of feet and legs is much impaired, and there is neither plantar nor patellar reflex, nor ankle clonus. The feet are cold. She has no difficulty in swallowing and no loss of power in the sphincters, or appearance of bedsore. The urine is acid, sp. gr. 1015, free from albumen and sugar.

A note made on November 23rd states that she is in much the same condition as on admission physically, but that her mental state is more confused. She says she wishes to go home, and that she is quite able to get up and walk. She is restless and sleepless at night and very noisy. She requires to be fed, and has occasionally slight difficulty in swallowing. Two days later there were sordes on the lips and teeth. On the same day it is also noted that there are frequent involuntary movements and twitchings. The chief respiratory movements are thoracic, but even these are limited. The movements of the diaphragm are reversed, the epigastrium bulging during expiration and receding during inspiration. She has a short cough, the tongue is dry and coated with a brown fur, and she now passes her urine and motions in bed. She is still restless and noisy at night, crying out for beer. The electrical examination was made on this and the next day, the results being as follow:

Left arm.—Triceps extensor reacts to moderate, and

supinator longus to strong, *faradic* currents. The muscles in front of the forearm react to weaker currents. There is no reaction to strong *faradic* currents in the extensor communis digitorum or in the extensor minimi digiti.

Galvanism :

Extensor communis digitorum A.C.C. (μ 3) > C.C.C.
Extensor minimi digiti C.C.C. (μ 2) > A.C.C.

Legs.—Faradism.—Strong secondary currents produce no contraction of the gastrocnemius, peronei, tibiales, or extensors of foot, either over the muscles or from motor points.

Galvanism.—With a current of five milliamperes there is both anodal and kathodal contraction of the extensor longus digitorum of right leg ; the contraction is slow and wave-like (A.C.C. = C.C.C.), and other factors of modified degenerative reaction are present ; the amount of current required for contraction being subnormal, and contraction produced not being that of health. No other group of muscles was examined on account of the impossibility of moving patient's legs without causing pain, but these reactions probably give a correct index of the condition of other paralysed muscles.

After this date she got rapidly worse, the wrist-drop and foot-drop became more marked, and there was much tenderness in the calves of the legs ; her incoherence continued and she spoke with weak voice and a snuffling tone ; the tongue was dry and protruded with difficulty. The pulse became more rapid and feeble, the extremities cold, and the breathing shallower. She had occasional twitchings of the arms the day before her death, which took place on November 29th, at 3.45 a.m.

The temperature was only on three occasions over 100°, and was below that figure on the evening before her death ; it was generally about normal. The pulse, 72 at first, increased to 160 during the ten days she was in the hospital, and her respirations rose from 20 to 32.

At the post-mortem examination the lungs were found to be congested and extremely œdematous, the liver very

fatty and of a pale fawn colour; the kidneys large, with swollen cortex and showing fatty striæ.

In this case also the brain and its membranes presented no obvious appearance of disease. The spinal cord on section seemed somewhat atrophied in the central grey matter, but no other changes were noted.

The cord, together with portions of several nerves from the right side of the body, and of the right extensor communis digitorum, were hardened in solution of bichromate of ammonium for five weeks, and examined in the same way as were those of the previous case.

Microscopic examination.—The cord here too may be described as normal, although it does not present such a typically healthy appearance as was found in the other. The ganglion cells are fewer in parts, most of them are shrunken, numbers have dropped out altogether, leaving holes in the section; and of those which remain, the branching processes are less clearly defined. These appearances, so far as they deviate from the strictly normal type, are probably due to the preparation of the cord for cutting having been less successful than in the other case. It should also be noted that the blood-vessels are larger and more numerous than usual.

Right anterior crural nerve.—The chief noteworthy appearance in transverse section is the large number of the nuclei of the nerve sheaths, and of large round-cells scattered through the endoneurium, and infiltrating the walls of the vessels. Roughly speaking, about half of the nerve-fibres appear normal; the rest present a cloudy or granular appearance of the nerve substance. There is no increase in the connective tissue.

In longitudinal section some segmentation of the myelin is apparent, but it is not a strongly marked feature.

Right anterior tibial nerve.—Here, on the other hand, the segmentation of the myelin is the most striking feature; there is not a sound fibre to be seen in the specimens examined (fig. 6).

Right musculo-spiral nerve.—In transverse section some

of the nerve-fibres are swollen and granular, others shrunken, a large number apparently normal, showing well the axis-cylinder. In longitudinal section also the fibres appear granular; the myelin in many is segmented, and collected in round or oval masses.

Right median nerve.—The appearances here are similar in kind to the above; in degree they are more marked. In addition the nuclear proliferation before referred to is conspicuous in endo- and perineurium, and round the vessels.

Right ulnar nerve exhibits precisely the same appearances as those found in the last mentioned nerve, and in about the same degree.

Right phrenic nerve shows no appreciable appearance of pathological change.

Right pneumogastric nerve.—A few of the fibres appear degenerated, and there is here and there slight segmentation of the myelin.

The *spinal nerve roots* are sound, both anterior and posterior.

Extensor communis digitorum.—The striation of some of the muscle-fibres is indistinct, and some are decidedly granular. There is, perhaps, slight increase in the nuclei of the sarcolemma.

The *liver* shows early cirrhosis and fatty change.

My third case, E. C—, a cook, æt. 43, is still under treatment, so that the ultimate test in her case cannot be applied; but if regard be had to the history and symptoms it would scarcely be too much to say that her pathological state, in kind if not in degree, could be predicated with tolerable certainty.

She came under my observation on November 5th, 1886. There was a history of rheumatism in two members of her family. She herself had suffered occasionally from winter cough, and two or three years ago had an attack of hæmatemesis. She had had two miscarriages in the first two years after her marriage, and one living child subse-

quently, the latter being now a healthy woman of twenty-two years of age. There was no history of syphilis on the side of either the patient or her husband. Her present illness came on about three weeks before admission with pains about the joints, which were thought to be rheumatic; these were first noticed in the knee-joints, and then in the hands and ankles.

She was observed, on admission, to be a sparely nourished woman, peevish in manner, with dilated facial capillaries, having pain in the joints and a short cough. Her chest showed fair elevation and expansion, and was normally resonant; breathing was weak and accompanied here and there by scattered sonorosibilant sounds. The heart's apex was in the normal position, impulse feeble, sounds fairly normal. In the abdomen the liver extended to near the level of the umbilicus, measuring eight inches in the vertical nipple line. Its margin was sharp, and the surface smooth; it was free from tenderness, and was distinctly felt descending with deep inspiration. There was no dulness in the flanks, and no jaundice. Both knee-joints and ankles were tender, and the former were thought to contain a little fluid; there was no redness, and they did not feel hot.

She had all the appearance of a person of alcoholic habit, but what with the family history of rheumatism, the apparent localisation of pain in the joints, and slight effusion into the knees, I was disposed to pass over her case very lightly as one of slight rheumatism, but on examining her again a few days later (Nov. 10th) I observed that she had double wrist-drop. The hands could be held out in straight line with the forearm but there was no power of further extension. The extensor muscles were wasted, and there was tenderness on pressure over nerve-trunks, and on grasping the arm or forearm. As to the legs, it was found that the ankle-joints were painful on movement, and also the muscles of the calves when handled. The muscles of the legs generally were wasted and flabby, the feet dropped, and without

power of dorsal flexion. Patellar and plantar reflexes were completely absent; the feet and hands were neither shiny nor œdematous.

On renewing inquiry into her history it was elicited from her friends that during the last eight and a half years she had taken spirits and beer very freely, and that on the occurrence of her present illness the joint pains were followed by weakness of the legs which rapidly became more marked, so that she was unable to walk. She had no numbness or tingling anywhere, but for a year had suffered from a feeling of coldness in the feet.

In the hospital she was restless and slept badly at night. After the lapse of little more than a week from the time when her paralytic symptoms were noted she complained of a feeling of numbness in the legs. Power of grasp in both hands was very feeble. Her urine was acid, sp. gr. 1030, depositing lithates but free from albumen and sugar. She took food well and began to sleep better, but she occasionally passed her motions in bed while retaining her urine. She was apathetic and rambled in her talk. The following is a note of the electrical reaction of her muscles.

All the muscles on back of forearm react well to *faradic* current.

Galvanism:

Ext. carpi ulnaris C.C.C. (μ ·5) > A.C.C.
Ext. com. digitorum C.C.C. (μ 1·5) > A.C.C.

Marked wasting of muscles. No tremors, coarse or fine.

Muscles in front of forearm react to weakest faradic current.

Galvanism:

Flex. sublimis digitorum C.C.C. (μ ·5) > A.C.C.
Supinator longus C.C.C. (μ 3·5) > A.C.C.
Flex. carpi ulnaris C.C.C. (μ ·5) > A.C.C.

Difference between A.C.C. and C.C.C. extremely slight, in many cases the two are practically equal.

Legs.—All the muscles of both legs act to a rather

strong, primary *faradic* current, except the peronei of left side, which do not react at all.

Galvanism.—Right leg :

Tibiales, Extensors, Gastrocnemius	A.C.C. (μ 2) = C.C.C. Action slow and wave-like.
Peronei	A.C.C. (μ 6) > C.C.C.

Left leg :

Tibiales, Extensors, Gastrocnemius	A.C.C. (μ 2) = C.C.C.
Peronei	A.C.C. (μ 4) > C.C.C.

A few days later (Nov. 29th) it was noticed that cutaneous sensibility both in arms and legs was much impaired and she was becoming more apathetic and incoherent. She could not grasp anything in order to feed herself. She could move the legs slowly but not the feet, and there was scarcely any sensation on pricking the skin of arms, legs, chest, or abdomen. The respiratory movements were almost wholly thoracic and the breathing shallow ; she had occasional twitching of the arms but not of the legs.

The following notes record the further progress of the case.

Dec. 2nd.—Patient lies on her back with her knees drawn up. She is in a more stupid and drowsy condition, will scarcely answer when spoken to, and does not protrude the tongue. The cheeks are flaccid and are blown out in expiration ; the skin is dry and harsh. Movements of the chest are very limited, and there is scarcely any respiratory movement of the abdomen. There is no jaundice. Resonance of front of chest is fairly good ; breath-sounds weak and accompanied by faint sonoro-sibilant sounds. Posteriorly resonance is normal, breath-sounds scarcely audible ; no adventitious sounds are heard. Heart's maximum impulse well felt in fourth interspace, three quarters of an inch internal to nipple ; sounds normal and well pronounced. Legs and arms are rather

more wasted. Ophthalmoscopic examination of each fundus shows nothing abnormal.

7th.—Patient is much brighter to-day although she rambles in her talk and her speech is rather thick; the tongue is clean and moist; the diaphragm acts in respiration tolerably well, as evidenced by epigastric bulging and descent of the liver during inspiration. There is now but little complaint of pain when the limbs are manipulated, but the dropping of the hands and feet is as well marked as ever. She complains of no discomfort and says she feels better. Urine and fæces are sometimes passed in bed. She has the delusion that she gets up every day and walks in the ward.

11th.—Mentally she is clearer now. Sensation of pain on pinching the skin of the legs is acute. There is some rigidity of the adductor muscles of the thighs; she lies with the knees pressed together. The pupils are moderately dilated and act sluggishly to light.

15th.—The extensors of the forearm are stronger. She is able to bring the hand above the horizontal line of the forearm when the hands are held out, and she can slowly pronate and supinate both hands. She can turn in bed without assistance, and does not now pass urine or motions in bed.

17th.—Electrical examination of right leg made to-day shows that the peroneal and sural groups of muscles all act to moderately strong *faradism*.

Galvanism:

Extensor longus	A.C.C. (μ 4) = C.C.C.
Gastrocnemius	A.C.C. (μ 6) > C.C.C.
Peronei	A.C.C. (μ 5) = C.C.C.

In all cases the contraction is feeble, slow, and wave-like; the degenerative reaction is less marked than on first examination. The examination and necessary manipulation of limbs now cause no pain. There is a red line on the gums close to the margins of the teeth; the tongue is clean and moist, but of a deep red colour, and somewhat glazed.

Up to this point there was evidence of some improvement, but on December 30th and two following days she had a sharp attack of diarrhœa, which pulled her down a little. She still, however, showed the possession of more power in the extensors. Her delusions continued. It should have been mentioned before that she had no spinal pain or tenderness, and that she could distinguish heat and cold perfectly over the feet and legs below the knees. There was some power of dorsal flexion of the foot, and a slight amount of glossiness of the skin of the fingers was noted.

She has lately (January 7th) become more restless at night, and has been trying to get out of bed. The wasting of muscles, especially those of the legs, is still more marked; the interossei of the hands are much wasted also; reflexes remain absent; cutaneous sensibility of feet and legs is perfect, except that she does not feel when a light touch is applied to the outer and upper part of the left ankle.

January 11th.—Electrical examination. *Right arm.*—All the muscles of arm, forearm, and hand react to moderate faradism, and the galvanic reactions are quantitatively and qualitatively normal. *Right leg.*—The muscles act as before to faradism; their action to galvanism requires a more powerful current than formerly (eight to ten milliamperes). A.C.C. still equals C.C.C. About this time the patient was got out of bed, and allowed to sit up for an hour in the evenings. It was noted that the feet did not become œdematous when in the dependent position.

18th.—She gets up daily for an hour, and is decidedly stronger. She is unable yet to stand, however: she is clearer in her mind. There is much impairment of cutaneous sensibility over the backs of the hands and over the outer and posterior aspect of the forearms; the sensation of pain when pricked with a pin is retarded for the most part in hand and arm, where it is not absolutely abolished. As to the legs, the sensation of pain is decidedly retarded, and in the sole of the foot is absent altogether. Reflexes are still absent, and wasting is very marked. She is able

to lift the legs from the bed, and to move them laterally, crossing one over the other.

28th.—Patient complains that she does not feel so well, and this she attributes to going out and falling on the street. (She has not been out of the ward since her admission to the hospital.) She moves her limbs more freely, however, and has no pain.

February 14th.—Yesterday, for the first time, she was able to feed herself; to-day, she complains of abdominal pain, chiefly in the region of the lower border of the liver. There is but little, if any, tenderness, her tongue is clean and moist, and she does not feel sick. She can raise herself in bed, and has much greater power of extension in the arms, and of dorsal flexion of the feet. There is no difference as regards wasting of muscles, and no return of reflexes.

19th.—She is now able to walk a little round the ward, supported on either side, and can stand with very little assistance. In walking she seems to have most difficulty in getting under way, the ankles bending under her when she first stands up, but after being fairly started she moves her legs pretty well. The toes are turned in, however, and she tends to walk on the outside of the foot; the toes are rather scraped along the floor. She walks best when looking down at her feet. When sitting, she can cross one leg over the other with tolerable ease.

28th.—There is a marked improvement now in her mental state. Her memory is clearer, and she has not the delusions about getting up and going out for a walk which she used to have. She can walk now with a little support on one side.

In about ten days after this she could walk with the help of a stick only, and was able to dress herself, and a week later the stick was discarded, and she walked without support of any kind. It was noticed, however, that after being out of bed the greater part of the day her feet and legs became œdematous; but this condition has gradually improved, her progress has latterly been uninterrup-

ted, and she may now be pronounced convalescent. She has steadily gained weight (from 7st. 8¾ lbs. on March 11th, to 9 st. 4 lbs. on May 6th), and she eats and sleeps well. The patellar and plantar reflexes are still absent.

Remarks.—I have brought these cases under the notice of the Society in the hope that they would form a contribution of some interest to the clinical history and pathology of alcoholic paralysis, especially since no cases accompanied by full pathological details are to be found in its published 'Transactions.' In saying this I am not unmindful of the papers contributed by Dr. Reginald Thompson and Dr. Broadbent in 1868 and 1884 respectively, but these dealt with the disease chiefly from the clinical standpoint.

Until a few years ago the symptoms in cases such as those I have narrated were attributed to some change in the spinal cord, and this view was held by both British and foreign writers. Dr. Wilks, for instance, wrote of alcoholic[1] paraplegia as a "spinal affection" from the influence of alcohol exerted on the spinal cord. Dr. Broadbent's communication, also just referred to, bore the title "On a Form of Alcoholic Spinal Paralysis," and in the discussion which followed the reading of his paper at the Society's meeting on February 12th, 1884, the opinion seemed to be generally held that the disease owed its symptoms to some change, either organic or dynamic, in the cord.[2] And it may be said that the writings of both German and French observers pointed for the most part to similar conclusions, till Lancereaux[3] published cases in which the peripheral nerves were described as manifestly altered, a large number of the fibres of the nerves examined showing the myelin to be segmented in rounded granular masses of unequal size. The

[1] 'Lancet,' March 9th, 1872.

[2] Dr. Buzzard, however, referred to the observations of Lancereaux and others as suggesting its peripheral origin.

[3] 'Gazette Hebdomadaire,' No. 8, Feb. 25th, 1881.

same author also published further observations in an article on the general subject of Toxic Paralyses[1], in which he repeated his previous conclusions that Alcoholic Paralysis is a disease of peripheral origin, having definite symptoms, course, and pathology.

Since this direction has been given to pathological investigation many cases have been recorded both in this country and abroad,[2] and there has been a general agreement that inflammatory or degenerative changes have always been present in some part of the peripheral nervous system. The view that the essence of the disease is an inflammation has found expression in the name "multiple neuritis;" but it has been suggested that the morbid condition found in the nerves is rather a degeneration similar to what takes place in the peripheral part of a nerve after division, and that the term "neuritis" is a misnomer. With reference to this, I think it may be said that my fatal cases, especially the first, would seem to favour the former view. The masses of leucocytes seen in the perineurium, as well as the numbers of similar cells scattered through the endoneurium, together with the appearance of the walls of the vessels, induce the conclusion that there at least the lesion is of the nature of an inflammation. May it not be, as suggested by Dr. Ross, of Manchester, in a paper recently published,[3] that different pathological conditions may exist at different levels of a diseased nerve, the degeneration being found below the point at which a perineuritis has been established? It seems to me *a priori* reasonable to expect changes of an inflammatory nature as the result of prolonged alcoholic excess, and I should look upon the condition as comparable to what is found in the liver in cirrhosis.

Regarding the diagnosis of my three cases, I think there can be no doubt. The first case certainly died of

[1] 'Gazette des Hôpitaux,' No. 46, April 21st, 1883.

[2] See Appendix.

[3] 'British Medical Journal,' January 1st, 1887.

acute phthisis, but the symptoms of alcoholic paralysis were even better marked than those referable to the lungs, and the morbid appearances at least equally so. Both fatal cases had cirrhotic and fatty livers, and the liver of the third case appears to be in the same condition. The history in each case pointed entirely in the same direction. There was nothing to favour a suspicion of syphilis, gout, lead-poisoning, or diphtheria, or of any other disease likely to give rise to a nerve-lesion; and in all there was the marked and prolonged history of alcoholic indulgence, as well as the presence of morbid conditions other than those found in the nerves, likely to result from such a habit.

I should say that the paralysis of the diaphragm and intercostal muscles, which was well marked in both the fatal cases, had much to do in determining the fact of death at the particular time it occurred.

This consideration applies most strongly, however, to the second case, since in it there was no efficient and immediate cause of death found except œdema of the lungs.

As to other symptoms, the muscular pains, the mental derangement, the absence of bedsores, and of paralysis of the sphincters, and the electrical reactions of the paralysed muscles, are sufficient to establish the diagnosis.

Tubercular disease of the lungs has very often been associated with alcoholic paralysis, and this is an interesting fact, which has not yet received, so far as I am aware, any special explanation. It seems to me that here, as often elsewhere, the simplest explanation may be the true one, that both the disease of nervous system and of lungs are due to the one common cause. Abuse of alcohol is not unlikely to determine the appearance of phthisis in those who have any hereditary predisposition to the disease, as well as to lead to other insanitary conditions tending in the same direction.

In connection with this point it should be mentioned that M.M. Pitres and Vaillard have lately published[1] the

[1] 'Revue de Médecine,' March, 1886, p. 193.

results of investigations into the condition of the peripheral nerves in the subjects of pulmonary tuberculosis, being led to this by observation of the troubles of sensation and motion, and the trophic changes which they have met with in that disease. The position laid down by them in the paper referred to is that tuberculosis acts like diphtheria, variola, typhus, and enteric fever in occasionally producing a change in the peripheral nervous system, such change consisting of a parenchymatous neuritis, evidenced by segmentation of the myelin, proliferation of the nuclei of the inter-annular segments, disappearance of the axis-cylinders, and atrophy, more or less complete, of the nerve-fibres. They quote a number of cases in detail, collected from various sources, in some of which there were no marked clinical symptoms of nerve disease,—nothing beyond vague and passing pains. In others muscular wasting was a prominent feature; in a third group sensory troubles, such as pain and cutaneous hyperæsthesia or anæsthesia, predominated. In all the cases examined the pathological appearances above enumerated were found in peripheral nerves, and the brain and spinal cord were healthy. In some the clinical features were precisely those of alcoholic paralysis, excepting the mental derangement; but it is worthy of note that in two at least there was a decided history of alcoholism as well. Their conclusions are set forth in the following terms:

"In the course of tuberculosis, just as in the course of other infectious maladies, it is not uncommon for the peripheral nerves to become the seat of parenchymatous alterations, presenting the histological characters of inflammations called degenerative.

"2. These depend on an inflammation in the nerve itself, and not on a pre-existing lesion of the brain or spinal cord, and are found in subjects whose nervous centres and spinal roots are in a condition of complete integrity.

"3. They are indifferently found in sensory, motor, and mixed nerves. They are equally seated in the cranial

nerves (including the optic and oculo-motor nerves) in the pneumogastric, phrenic, &c.

"4. Their symptoms, very complex and very variable, are as yet incompletely understood. However, in comparing the observations published up to the present time, they may be divided into three groups.

"The first comprises those cases where the symptoms of neuritis shown at the autopsy have passed unnoticed amid the severe troubles attendant upon the evolution of the tuberculosis (latent neuritis).

"In the second are placed those in which muscular atrophies, localised or diffuse, have constituted the predominant symptom (amyotrophic neuritis).

"In the third may be included those cases in which the nerve inflammations have produced during life the more or less serious troubles of sensation, hyperæsthesias, anæsthesias, neuralgias, &c. (painful or anæsthetic neuritis.)

"5. The frequency of peripheral neuritis in the subjects of tuberculosis, the variability of its distribution, and consequently of its symptoms, explain the existence and the many clinical forms of most of the nervous troubles which supervene in the course of tubercular disease."

The considerations advanced by these authors open up an important field for observation regarding the changes to be found in the peripheral nerves in the subjects of phthisis, which it may be hoped will soon be more completely occupied. They have at least established a case for further inquiry. It must be said, however, that in none of their cases is alcoholic influence expressly excluded, and this is a fact of some importance when the difficulty of obtaining such a history from patients is borne in mind, and when it is further remembered that varying degrees of alcoholic indulgence produce different effects on different individuals. And, as already mentioned, two of the cases quoted in their paper (one of Strümpell's and one of Müller's) were alcoholic.

As to treatment, my first and second cases were so rapidly fatal as to make any discussion on this point in

relation to them of no value. In the case of the third patient, iodide of potassium did not appear to do any good, and bromide of potassium and chloral, instead of making her quieter and producing sleep, seemed to have the opposite effect. Further than this, she has had strychnia in gradually increasing doses for many weeks, to the extent of ten minims of liquor strychniæ every six hours, and latterly cod-liver oil, with faradism and rubbing to the wasted muscles. Probably the benefit she has received has resulted more from the withdrawal of alcohol than from any more direct element of treatment, and it has been a favorable point in her case that she has always been able to take food well. At the same time, I have no doubt that the faradism and rubbing, as well as the strychnia and cod-liver oil, have played a considerable part in her restoration to health.

APPENDIX.

A very full bibliographic index, brought down to 1885, will be found at the end of the work first mentioned below. Those references which follow complete the list to date, so far as I have been able to find.

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Oppenheim. Beiträge zur Pathologie der Multiplen Neuritis und Alkohol Lähmung, Zeitschrift für Klin. Med., 1886, vol. xi, p. 232. 1 plate.

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Buzzard. On some Forms of Paralysis from Peripheral Neuritis of Gouty, Alcoholic, Diphtheritic, and other Origin (J. & A. Churchill), 1886.

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Kast. Primäre degenerative Neuritis, Deutsches Archiv für Klin. Med., vol. xl, part 1, p. 51.

Bernhardt. Ueber die Multiple Neuritis der Alkoholisten, Zeitschrift für Klin. Med., vol. xi, part 4, 1886.

Ross. On Peripheral Neuritis, Brit. Med. Journ., Jan. 1st, 1887, p. 6.

Ormerod. Illustrations of Peripheral Nerve Disease, St. Barth. Hosp. Rep., vol. xxii, pp. 181—185.

Duckworth. Three Cases of Multiple Peripheral (Alcoholic) Neuritis in Women, ibid., p. 253.

Seguin. Alcoholic Multiple Neuritis, Journ. of Mental and Nervous Disease, New York, March, 1887, p. 206.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 275.)

DESCRIPTION OF PLATES VII AND VIII.

(Three Cases of Alcoholic Paralysis (Multiple Neuritis), by DAVID W. FINLAY, B.A., M.D., F.R.C.P.)

PLATE VII.

(From drawings by Dr. FINLAY.)

Fig. 1 (E. F—, Case 1).—Transverse section of plantar nerve, stained with logwood and mounted in Canada balsam, showing thickening of the perineurium and of the walls of the blood-vessels, with proliferation of nuclei and leucocytal infiltration. × 175 (about).

Fig. 2.—Part of the same section more highly magnified. The appearances of degeneration in the parenchyma of the nerve are better seen; the small dark spots are remaining axis-cylinders, some of the larger are probably swollen axis-cylinders; the others are nuclei of the nerve-sheaths and leucocytes. The comparatively hazy-looking large round bodies are nerve-tubules with granular contents from which the axis-cylinders have disappeared. × 450.

Fig. 3.—Normal nerve, transverse section, for comparison with Fig. 2, and prepared in the same way. × 450.

PLATE VIII.

(From drawings by Dr. FINLAY.)

Fig. 4 (E. F—, Case 1).—Plantar nerve: fibres teased out, treated with osmic acid and mounted in glycerine, showing degeneration and segmentation of the myelin. × 450.

Fig. 5.—Extensor carpi radialis longior. Transverse section stained with logwood and mounted in Canada balsam. Many of the muscle-fibres are atrophied, and the whole section is crowded with nuclei and leucocytes. × 175 (about).

Fig. 6 (M. J. P—, Case 2).—Anterior tibial nerve, longitudinal section, stained with osmic acid and carmine, and mounted in Canada balsam. The appearances are entirely similar to those found in Case 1 (Fig. 4). × 450.

Fig 1.

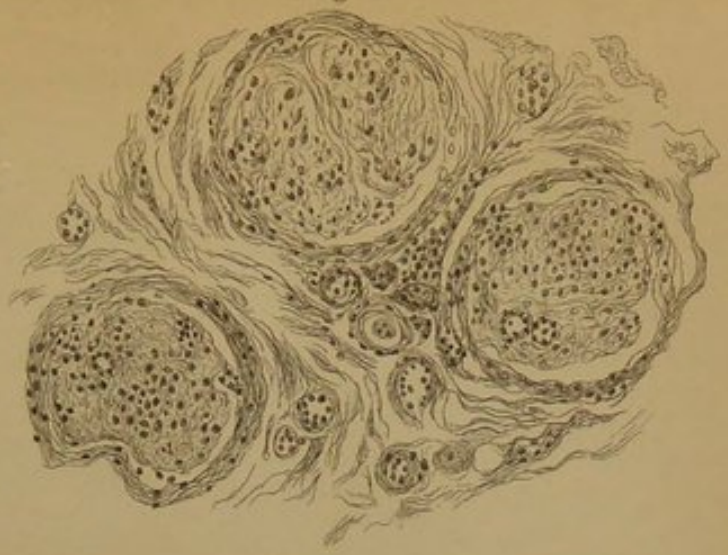

Fig. 2.

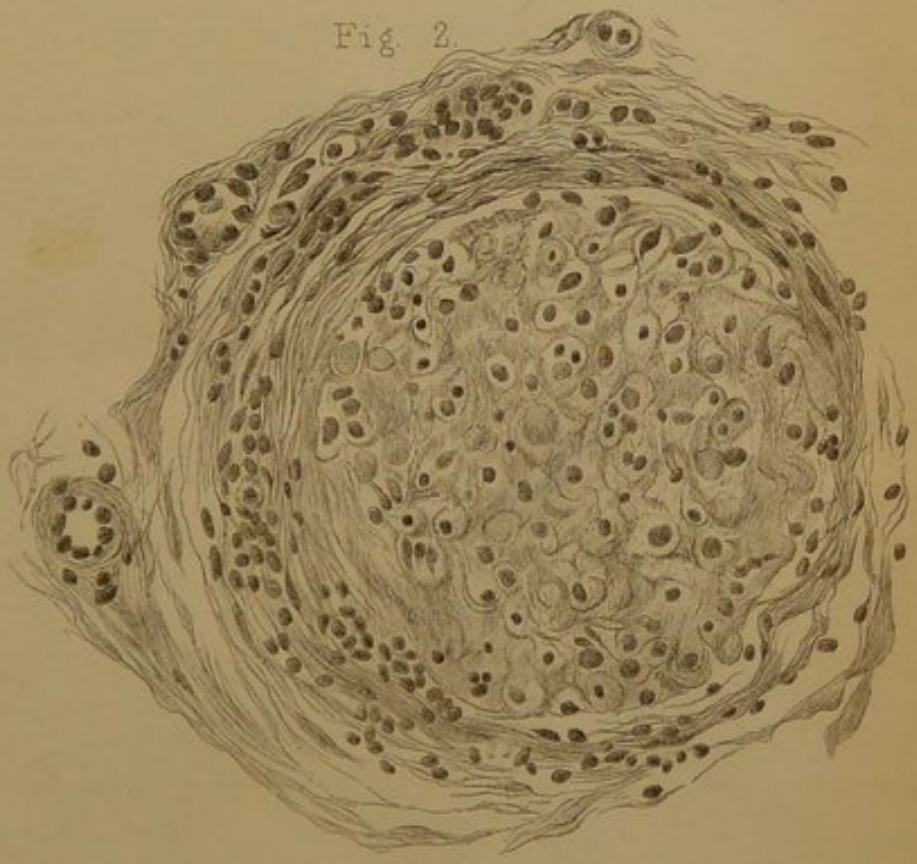

Fig. 3.

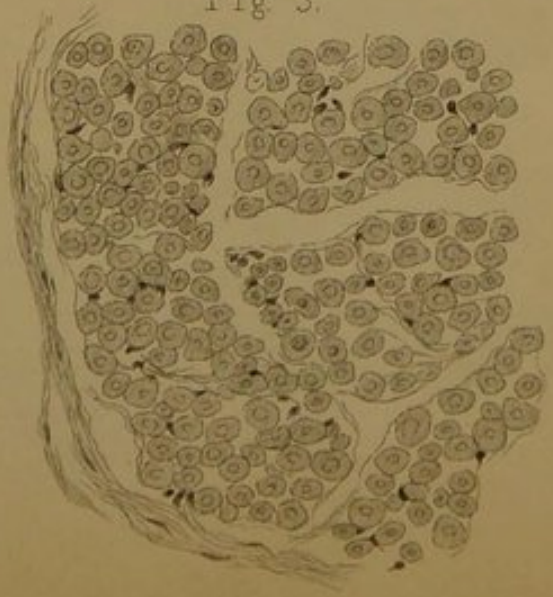

Fig. 4.

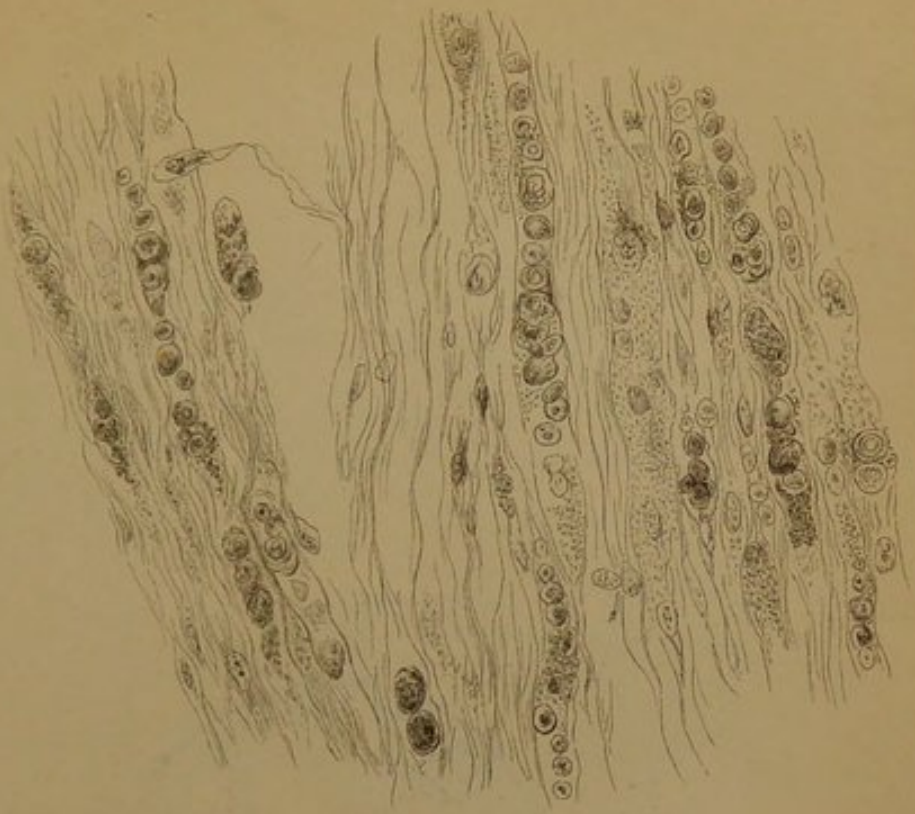

Fig. 5.

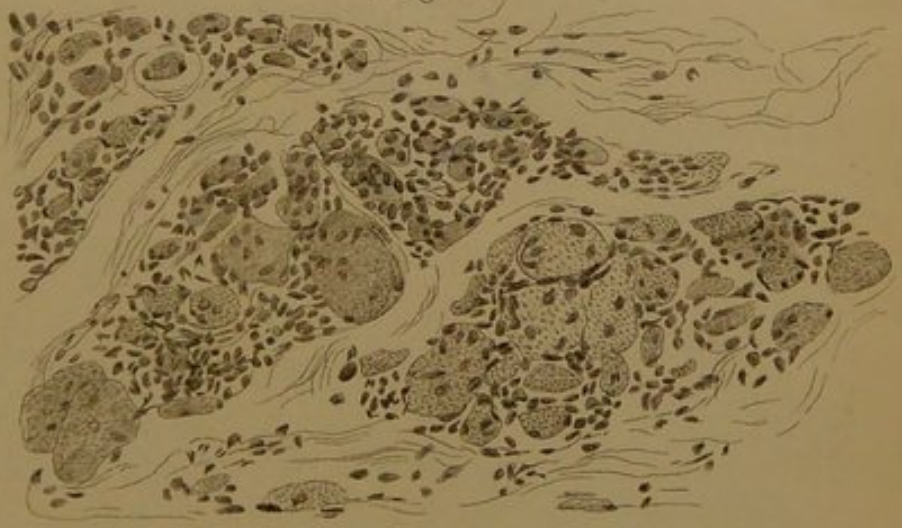

Fig. 6.