ON ALCOHOLIC PARALYSIS.

BY J. DRESCHFELD, M.D., F.R.C.P.,

*Professor of Pathology, Victoria University; Physician, Royal Infirmary,
Manchester.*

THE effects of alcohol on the nervous system manifest themselves in various ways, chiefly according to the particular part of the nervous sphere attacked.

Whilst in many forms of chronic alcoholism the brain seems to be the chief organ to suffer, there is one affection, alcoholic paralysis or alcoholic paraplegia, in which cerebral symptoms are either altogether absent, or play a mere subordinate part, while most of the symptoms are referable to the spinal cord or to the peripheral nerves. The symptomatology of this affection has been well described in this country by Wilks ('Diseases of the Nervous System'), Lockhart Clarke ('Lancet,' 1872), Reginald Thompson, ('Med. Chir. Trans.' 1868), and more recently by Glynn, ('Liverpool Medico-Chir. Journal'), and Broadbent ('Lancet,' Feb. 16, 1884). On the Continent, the principal workers at this subject have been Leudet ('Arch. Gén. de Méd.' 1867), Lancereaux ('Gaz. Hebd.' 1865 and 1881), Moëli (Berl. Klin. Wochenschr. No. 14, 1884), and Charcot (see article by Féré in 'Prog. Méd.' June 14, 1884).

Our knowledge of the pathological anatomy of this affection is, however, still meagre, although the observations of Lancereaux, Moëli, and Broadbent, tend to show that the lesions concern chiefly, if not entirely, the peripheral nerves. As a small contribution to this interesting subject, I may be permitted to describe the lesions found in one case which was recently under my care, and to allude to a few cases which I have from time to time observed.

The disease runs by no means the same course in every case, nor does it invariably present the same grouping of symptoms, though certain of these symptoms are always, or almost always, present. Considering, however, the more prominent characteristics, we may, I think, divide alcoholic paralysis into certain types.

The first may be fitly designated *Alcoholic Ataxia*. Of this form I have seen several cases, and other observers have also alluded to it; thus Wilks ('Dis. of the Nervous System,' p. 225) mentions the case of a young woman "whose symptoms very much resembled those of locomotor ataxy, both in the character of the pains, which were like electric shocks, and also in the mode of progression." Leudet (loc. cit.) alludes to the difficulty of distinguishing such cases from true ataxia, and Lancereaux and Charcot also mention the ataxic symptoms. The cases of this description which I have seen were men, from 30 to 40 years old; the symptoms had come on more or less gradually, and consisted chiefly of marked inco-ordination in the lower extremities, absence of tendon-reflex, and lancinating pains. There was neither atrophy nor paralysis.

A patient whom I saw recently with Dr. Roe, of Patricroft, and who had been greatly addicted to alcohol for years, had already had one attack of paralysis, from which he completely recovered, after giving up alcohol. Having again fallen into bad habits, the old symptoms recurred (inco-ordination, lancinating pain, absence of tendon-reflex and slight loss of control over the bladder at night). Dr. Roe informs me that the patient's habits are still very intemperate, that his ataxic symptoms have remained unaltered, but that the patient is now suffering from rapidly advancing phthisis.

A case of very similar description I saw with my colleague, Mr. Jones: here also the symptoms came on after a series of alcoholic excesses, and I am informed have now quite subsided. The patient's family history was free from any neurotic taint, and he had not had syphilis at the time I saw him, though he has contracted it since then.

Another case I had an opportunity of watching for some time in the out-patients' department of the Manchester

Infirmity. A man, æt. 35, an excessive drinker, suffered from the symptoms described above, which came on in the course of three weeks, but subsided completely in three months. The recovery in his case was complete, the tendon-reflex being again established some months after all the other symptoms had disappeared.

These cases differ from those of the second type to be described below, in the absence of hyperæsthesia, hyperalgesia, vaso-motor disturbances, paralysis, atrophies and mental disturbances. The individuals attacked are chiefly men, and the symptoms disappear with the discontinuance of alcohol. In the diagnosis of such cases of pure alcoholic ataxia, one has of course to bear in mind how far alcohol may simply play the part of an etiological factor, and how far alcoholism and ataxia may be mere coincidences; moreover in the ordinary cases of ataxia, the symptoms, especially if they come on somewhat acutely, often show for a time a marked improvement. In the above cases, however, the ataxia followed so soon after the excesses, and so completely disappeared, that I have no hesitation in pronouncing them as alcoholic. This view is still further supported by the fact, that symptoms of ataxia are again seen in the second type to be described (especially the absence of tendon-reflex and the lancinating pains); moreover, some of the symptoms of ordinary locomotor ataxy, such as the arthropathies, the oculo-motor pareses, spinal myosis, &c., symptoms which we may consider spinal, are absent in the alcoholic ataxia. The symptoms observed harmonise well with the pathological anatomy which underlies the alcoholic paraplegia,—for as the changes observed chiefly concern peripheral nerves, we can understand the symptoms if we suppose that in the ataxic type we have an affection, a multiple neuritis, especially of the sensory nerves.

The second type of *alcoholic paralysis*, which deserves better the name of *paralysis*, is far more common, and is that which most observers have described.

This affection chiefly attacks females, who, though they deny the abuse of alcohol, have suffered from symptoms of chronic alcoholism (morning vomiting, chronic gastric catarrh, hæmatemesis, &c.), previous to the appearance of the nervous

troubles. These commence more or less acutely, and consist of sensory, motor, vaso-motor, and trophic disturbances.

The sensory disturbances are, as a rule, well marked and very characteristic, and consist of extreme hyperæsthesia of the feet and other parts of the lower extremities, with or without hyperalgesia; perversion of the sense for temperature (all objects when brought in contact with the skin feel cold); there are often present, in addition, lancinating pains in the lower extremities, and pain when pressure is applied along the vertebral column. The hyperæsthesia is often followed by anæsthesia or retarded sensibility, the hyperalgesia by analgesia.

The motor troubles consist of more or less marked paralysis, attacking the lower, and sometimes the upper extremities; in many cases the extensor muscles are chiefly affected, while in not a few cases the paralysis is more general. The superficial and mechanical reflexes are often diminished, the tendon-reflexes are nearly always absent; contractures have only been noticed by a few observers (Wilks, Buzzard). The vaso-motor affections consist chiefly of redness of the feet or hands, occasionally also of other parts of the body, and œdema, especially seen at the ankle, the dorsum of the foot, and the back of the hand. The nerves and muscles show marked degenerative reaction, though we have but few detailed statements on this subject.

Cerebral symptoms, such as insomnia, restlessness, more or less delirium and hebetude, though absent at first, often make their appearance during the course of the disease, and are frequently the cause of death, unless other complications should occur, such as phthisis, fatty degeneration of liver, &c.

Disorders of the circulatory, digestive and respiratory organs, are often seen during the progress of the disease, but are rather complications, or the primary effects of alcohol on these organs, than secondary manifestations of the nervous affection under consideration.

The prognosis varies very much. Many cases recover when they come early under the care of the physician, and when uncomplicated; some recover for a time, and afterwards relapse; whilst in others the disease pursues a steady down-

ward course, death ensuing from the cerebral symptoms, from exhaustion, or from some intercurrent affection, as is so often seen in other forms of chronic alcoholism.

The following case which was recently under my care, and which I was able to follow to its fatal issue, may be taken as a fairly typical example of this form of alcoholic paralysis, though it differs from some of the reported cases in the extent of the paralysis, which affected the lower extremities more uniformly than is ordinarily described.

The notes of the case were taken by one of my clinical clerks, Mr. E. E. Bowden.

Mary P., æt. 49, married, was admitted into the Infirmary on April 1, 1884.

Previous History.—The patient has resided in Manchester for 30 years; has had six children, all of whom have died, and has had one miscarriage. She has worked in a laundry, and admits having taken stimulants (though the patient denied any excesses, inquiries made of her neighbours satisfied us that she had been a chronic alcoholic; moreover, in her delirium she continually called out for beer). She has been in somewhat straitened circumstances, but enjoyed very good health up to ten years. She had small-pox some years ago, but has never had any other illness; the catamenia ceased two years ago, before which time they were very profuse.

History of Attack.—Two years ago the patient began to feel weak, especially in her knees, and she staggered then slightly in her walk; she also noticed that in dressing, when putting her garments over her head, she had to steady herself; she gradually grew weaker, and during the last few weeks before admission the weakness has so much increased, that she is now unable to walk.

State on Admission.—The patient lies on her back, and is unable to rise or turn over without great difficulty; her face has a haggard, almost demented expression; she is somewhat emaciated, and has a large bed-sore on her back.

The patient is fairly intelligent, and complains much of insomnia and slight headache; there are no other cerebral symptoms.

There is a considerable amount of paresis of the upper and

lower extremities, and also of the muscles of the back. The patient is scarcely able to walk or stand by herself, and with her eyes shut would fall immediately. She lies with her legs flexed, and extends them with great difficulty; can flex and extend her foot; can abduct and adduct the legs, but does so with difficulty and pain. The grasp of her hands is weak; can flex and extend her wrists and elbow; the effort of rising in bed causes great pain. There is great pain in both lower extremities, and patient shrieks when the legs are touched; the left knee is specially painful, the skin over it is reddened, and the joint is slightly swollen; there is also great pain in the back and loins, and along the whole of the vertebral column; there are no spots of either anæsthesia or analgesia; all the muscles seem equally atrophied and flabby; the superficial reflexes are fairly normal; the tendon-reflexes are absent; there is some diminution of the muscular sense in the upper and lower extremities.

The pupils are contracted, react very little to accommodation, and not at all to light. The tongue is furred and glazed; there is loss of appetite; the patient suffers from thirst and diarrhoea; the thoracic organs are found normal on physical examination; there is no cough and no expectoration; the liver and spleen appear normal on percussion; there is retention of urine, and the catheter has to be used; the urine is alkaline, has a sp. gr. of 1011, contains a large quantity of pus and a considerable amount of albumen.

The heart is found normal; the pulse regular, but quick, feeble, and easily compressible.

The temperature was in the morning 99, in the evening 100·4.

April 3rd.—The patient's condition is much the same as on admission.

Faradic Contractility.—Direct application to muscles; no contraction of the dorsal flexors of the right foot, even with the strongest current; those of the left foot react slightly to strong current.

Quadriceps femoris does not respond to any current.

Hamstring muscles and biceps react slightly.

Flexors and extensors of fingers react but slightly with the strongest current.

Biceps and triceps react somewhat better.

Erb's point scarcely any reaction.

Galvanic reactions.

LOWER EXTREMITIES.

	Kat. Cl.	ACl.	A.O.
Left Ext. brev. Dig. . . .	45	40	..
" Exten. Prop. pol. } . .	45	40	45
" Tib. Antic. } . .			
Right Tib. Antic. } . .	45	40	..
" Exten. Prop. pol. } . .			
Left Rect. fem.	45	40	..
Right " "	50	40	..
Left Hamstring	50	35	..

UPPER EXTREMITIES.

	Kat. Cl.	ACl.	A.O.
Left Ext. digit.	30	20	20
" Triceps	20 (slight)	20	25
		25 produces tetanus	
Right Ext. dig.	30 (slight)	25	25
" Triceps	35	30	..
Left Flex. digit.	35 (slight)	20	35
" Erb's point	40	45	..
Right Erb's point	40	..	45

Diagnosis.—Alcoholic paralysis. This diagnosis was arrived at without any great difficulty. The history of the case pointed strongly to a chronic alcoholic intoxication, and the symptoms on admission were well marked and typical. One might have thought of ordinary ataxia; but the extreme sensibility to pain and touch, and the degenerative reaction of the muscles, were against such an assumption. Against sub-acute or chronic poliomyelitis the sensory symptoms told with equal force. Against chronic meningo-myelitis pointed the fact that the disease commenced without pain, that paresis and inco-ordination were the first symptoms observed, and that the tendon-reflexes were absent. Against paraplegia dolorosa, such as is observed in cancer of the vertebræ, pointed the absence of any tumor, the absence of cachexia, the chronicity of the case, and the painless implication of the upper extremities.

There remained, therefore, scarcely any other disease but multiple peripheral neuritis, and with the history before us,

and the absence of any evidence showing that any other toxic agent, such as lead, arsenic, &c., was the cause of the symptoms (though such poisons would scarcely produce the sensory disturbances observed), we had no difficulty in arriving at the diagnosis stated.

Progress.—(I extract a few statements from short-hand notes.)

April 7.—The patient is much worse. She is delirious at night; her speech is slow and muttering; she lies with her legs flexed, and can only extend them with great difficulty.

April 9.—The patient is much worse; the delirium is now constant; the temperature 95; the extremities cold and cyanotic; pulse, very feeble, 80 per minute; the paralysis more extensive.

April 10.—Last night the patient became comatose, and died early this morning.

Autopsy, by Dr. Maguire, Pathologist to the Royal Infirmary. —Some fluid in left pleural cavity, a little fluid in pericardium; aortic valves showed some chronic thickening; the lungs, slightly emphysematous, contained a few caseous nodules in both apices. The liver enlarged, pale and soft. The kidneys showed several patches of caseation in the cortex; the pelvis full of pus; the capsule thickened, but peeled off easily, and showed the surface of the kidney affected by tubercular deposit. The bladder greatly inflamed, its inner surface covered with muco-pus, its walls thickened and infiltrated with tubercular deposit.

The brain was found normal, the pia slightly thickened; the dura mater showed numerous Pacchionian bodies.

The spinal cord and its membranes, when examined in the fresh condition, appeared perfectly normal.

Some peripheral nerves, which were reserved for microscopic examination, also appeared normal.

The muscles were pale, but otherwise normal.

After hardening the cord, I examined portions of it microscopically, without finding any changes: examined by Weigert's method ('*Fortschritte der Med.*' 1884), numerous small nerve fibres were found in Clarke's columns. The ganglion cells in all parts of the spinal cord were perfectly healthy.

Several posterior roots of the lumbar region were examined and found perfectly healthy.

Of the peripheral nerves, there were examined the two sciatics, the musculo-spiral and the anterior crural. These nerves showed the characteristic changes described by Lancereaux in a very marked fashion. In the sciatic, longitudinal and transverse sections showed most markedly the breaking up of the myeline, and in many places the breaking up of the axis cylinder also. Longitudinal sections showed the segmental changes described by Gombault, and seen by me in a case of lead paralysis (see Dr. Moritz's paper in the 'Journal of Anat. and Phys.' 1881). The muscles showed no peculiar changes when microscopically examined. The post-mortem changes showed then a multiple peripheral neuritis, chronic tuberculosis of the lungs, and tuberculosis of the kidney and bladder of more recent date. The diagnosis of alcoholic paralysis was thus confirmed by the post-mortem appearances, which were exactly those described by Lancereaux and Moëli.

Though hyperæsthesia and hyperalgesia are prominent symptoms in this form of alcoholic paralysis, yet I think that both these symptoms may be occasionally absent, and the motor nerves may be principally affected. Thus, not long since, I observed the case of a man, æt. 38, who suffered from far-advanced alcoholic cirrhosis of the liver, and who in the course of a few days lost the use of his arms and legs. When admitted into the Infirmary, March 22nd, 1884, about three weeks after the onset of the paralysis, there was noticed, besides the ascites and enlarged spleen, marked atrophy and paralysis in the upper extremity, affecting chiefly the muscles supplied by the radial, the posterior interosseous and median nerves, and more pronounced on the left than on the right side. The back of the hand was oedematous. There was also observed some paralysis of the lower extremities, affecting chiefly the extensors of the toes and ankle. In the sensory system there was found a perversion of the sense for temperature, namely, the patient felt, as intensely cold, anything that touched his arms or his legs. (I find this symptom stated in several of the recorded cases of alcoholic paralysis.) There was absence of tendon and superficial reflexes.

The electric reactions of the muscles, carefully taken by Mr. A. Mumford, one of my clinical clerks, showed marked degenerative reaction. The special sense-organs were normal.

The cerebral symptoms consisted chiefly of sleeplessness.

The patient was in the hospital some time, without showing any improvement. He left, and died at home, and we had no opportunity of making an autopsy.

The nervous symptoms in this case resembled more those seen in chronic poliomyelitis; yet the history, the onset, the perversion of the sense for temperature, the marked degenerative electric reaction and the associated cerebral symptoms (though these might have been due to the cirrhosis), induced me to look upon this case as one of peripheral multiple neuritis, affecting chiefly the motor nerves and caused by alcohol. The combination of cirrhosis with alcoholic paralysis has already been noticed by Lancereaux ('Gaz. Hebdomadaire' 1865).

The *Anatomical lesions* then which underlie alcoholic paralysis, to judge from the post-mortem results of Lancereaux and Moëli, and those given in the case above, consist of a peripheral multiple progressive neuritis. Broadbent, in his case, found the cord normal, but was not allowed to examine any other part of the body. In one of Lancereaux's older observations the antero-lateral columns of the cord were found slightly sclerosed; in all the later observations the cord was found healthy.

The post-mortem lesions explain the symptoms very well, and also account for the diversity which we observe, according as the one or the other set of nerves are chiefly involved.

As regards *differential diagnosis*, the disease resembles in many respects the progressive multiple neuritis described by Leyden, Strümpell, Pierson and others, of which indeed it forms simply a particular species. It differs, however, from this non-alcoholic multiple neuritis in several particulars; it runs a less acute course, is less progressive, the paralysis is more confined to the extensor groups of muscles, and though there is a considerable amount of pain in the non-alcoholic form, there is not usually the very characteristic hyperæsthesia and hyperalgesia described above; the cerebral symptoms also are absent in the non-alcoholic form.

It further resembles other intoxications, notably those produced by lead.¹ Lancereaux has long ago drawn attention to this, and, pathologically considered, the lesions are the same in both; just as in alcoholic paralysis, so in lead paralysis, we have to do chiefly with peripheral nerve changes; though lead seems to select the motor nerves *par excellence*. The similarity of the action of lead and alcohol is further evident when we consider other diseases in which both play the important etiological element, as in gout, in the true cirrhotic kidney, and, I may perhaps also add, chronic endocarditis and some brain affections.

The *treatment* of alcoholic paralysis, which is often successful in the less advanced and uncomplicated cases, must have for its prime object the withdrawal of the alcoholic stimulant. Of other remedies, the opiates, cinchona (recommended by Leudet and Wilks), strychnia, hydropathy and the application of electricity seem indicated.

The iodide of potassium, which I have tried in some cases, has not given me any very encouraging results.

I wish now to draw attention to certain *visceral neuralgias* which, either alone or in combination with other nervous symptoms, are found in chronic alcoholism, and which I believe are caused by the alcohol. Thus, I was asked to see a lady who was an alcoholic, and who suffered from very intense colicky pains; these attacks would last for days; they were unaccompanied by vomiting or any other gastric disturbance. There was marked constipation. There was no reason to suppose lead-poisoning, nor did the gums show any blue line—yet iodide of potassium was freely given without benefit. Opium gave some temporary relief; the pains, however, recurred from time to time, and only disappeared when the patient was put under restraint, in her own home.

Another lady, aged 27, who was suspected to be an alcoholic, suffered from attacks which resembled very much angina pectoris; there was excessive cardiac dyspnoea; the pain radiated into the left arm, slightly also into the right, and there was also some epigastric pain. Physical examination of

¹ Atrophies produced by arsenic have been noticed by Gerhardt, 'Obl. f. Klin. Med.' 1882.

the chest-organs showed nothing abnormal, and beyond a very visibly pulsating abdominal aorta, nothing abnormal could be detected in the abdominal cavity. The cause of these symptoms was very doubtful to me; but when they disappeared, along with some other neuralgic troubles (chiefly affecting the trigeminus) after the patient was put under restraint in a home for dipsomaniacs, I strongly suspected that they had been due to alcohol; in this view I was confirmed, for the symptoms reappeared, when the patient left the home and recommenced her old habits. She was placed a second time into the home, and again improved; but died subsequently from pneumonia. I had no opportunity of making a post-mortem examination.

These visceral neuralgias again resemble those produced by lead. Lead colic is common enough, and in a small epidemic of acute lead-poisoning produced by the inhalation of particles of lead-chromate, I have seen two cases, where, after the acute symptoms had passed off, symptoms very much like angina pectoris came on. These symptoms resemble most a vaso-motor disturbance, and as such we can easily understand their appearance in chronic alcoholism.