within twelve hours of perforation and found a small opening where the oesophagus comes through the diaphragm. As it was impossible to apply a suture in that situation I determined to pack with iodoform gauze for thirty-six hours, and recovery was uninterrupted, and three years afterwards she was in perfect health.

I think it is quite possible that in this case Nature could have effected a cure if no operation had been done, because the opening was very small and in a position which would be in favour of only a small leakage of the gastric contents taking place. It is in the lower dependent positions along the greater curvature of the stomach that we get large quantities of the gastric contents rapidly effused into the general peritoneal cavity.

HERNIA OF THE BLADDER AS A COMPLICATION OF INGUINAL HERNIA.

By G. B. FERGUSON, M.Ch.Oxon., F.R.C.S.Eng.,
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MR. H. J. CURTIS's paper published in the *BRITISH MEDICAL JOURNAL* of July 11th, on a case in the practice of Mr. Bilton Pollard, induces me to publish another example of the condition illustrated by him, and referred to above. Though uncommon, it is of sufficiently frequent occurrence to put all operating surgeons on their guard, with a view to its recognition. I must confess that I was not aware of such a condition, as of practical importance, before I was recently introduced to it in the case which I will now briefly narrate.

It is that of a young man who sought relief for a large irreducible left inguinal hernia, recurrent after an operation for radical cure. This hernia proved to be mainly omental, together with a small knuckle of the transverse colon. The operation done by me was made difficult by adhesions, though it was satisfactorily accomplished at last. Some pounds' weight of omentum was removed, and the neck of the sac was closed, though its total extirpation was impracticable. At the end of the operation a thin, blue, and distended cyst, about the size of a pigeon's egg, was noticed in the immediate proximity of the spermatic cord, and adherent to the remains of the sac. It was mistaken for a hydrocele of the cord, and was punctured, when a considerable amount of clear fluid escaped, and the finger introduced into the cyst passed, at a deeper level, through a constricted neck, into the bladder. The cyst was of extreme thinness, consisting of little more than attenuated mucous membrane. Regrettable as such a mishap was, I am glad to add that no harm followed it. The mucous membrane was stitched together continuously with fine silk, which I used in preference to catgut, feeling more confidence in its asepticity, and trusting that from its fineness it would not project into the bladder to originate incrustations therein; and this continuous suture was completely covered in by a layer of interrupted Lembert's. *Cæteris paribus*, catgut would, of course, have been safer for the mucous suture. A catheter was tied into the bladder for a few days, and I am pleased to add that uneventful healing by first intention ensued. Still and nevertheless the puncturing of vesical cysts is clearly a practice to be avoided, and I am grateful to Mr. Pollard for the simple suggestion that the diagnosis can be quickly cleared up by a sound passed through the urethra.

In the case treated by me the bladder hernia was extraperitoneal, and its mechanism was very evident. After the first operation there was inflammation with suppuration, and an adhesion resulted between the remains of the sac and the bladder, the former of which in its re-descent drew down with it a diverticulum of the mucous lining of the latter.

I suspect that there have been many more cases of vesical hernia observed than have found their way into medical literature, and I am permitted to refer to another case in the practice of my friend, Mr. E. Dykes Bower, Surgeon to the Gloucester Infirmary. Here an innocent-looking little cyst, projecting, as in Mr. Pollard's case, into the hernial sac, was removed, after ligaturing its neck. As in my case, the man had previously undergone an operation for radical cure, also on the left side, and fortunately here again no harm resulted from the injury to the bladder, and the patient made a good recovery, though during the next day, and for some time afterwards, there was a copious flow of

urine through the wound. Clearly, however, it must be the rule hereafter to leave all such suspicious cysts untouched, and surgeons are much indebted both to Mr. Pollard for indicating the proper treatment of such cases, and to Mr. Curtis for his clear and helpful diagrams and descriptions.

FURTHER OBSERVATIONS ON ALCOHOLIC AND ARSENICAL NEURITIS.

By ERNEST SEPTIMUS REYNOLDS, M.D.Lond., F.R.C.P.,
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It will probably be remembered that in 1900 there was an outbreak of arsenical neuritis amongst beer drinkers, especially in the North of England and in the Midland Counties. This was due to the accidental contamination of glucose with arsenic. In various papers which I wrote in connexion with this epidemic, and in my evidence given before the Royal Commission on Arsenical Poisoning, I contended that by far the greater number of cases of so-called alcoholic paralysis, at any rate in the Manchester district, were in reality due to chronic arsenical poisoning, and were not due to alcohol, and that this had been the case for years, probably apart altogether from the use of contaminated glucose. Subsequent investigations showed clearly that malt, especially when prepared by the use of certain cokes, was largely contaminated with arsenic, and that beer prepared from this malt also contained heavy traces of arsenic. Indeed, a small epidemic of marked arsenical poisoning occurred in beer drinkers at Halifax last year quite apart from arsenicated glucose, and was probably due to the malt having been prepared with gas coke.

In evidence given before the Royal Commission in 1902 I stated that I had seen cases of peripheral neuritis in pure spirit drinkers where there was no evidence of arsenical contamination. These cases, however, are very different from the cases of so-called alcoholic paralysis with which we have been so familiar for many years. They show some loss of power in the legs and arms, absent knee-jerk, paraesthesiae of the feet and hands, only slight tenderness on pressure of the muscular masses, very little atrophy, no loss of memory, and no oedema. Such cases are, moreover, considering the large amounts of alcohol consumed in this country, very rare, and during the last two years I have certainly not seen half a dozen in my private practice, in a large out-patient practice at the Manchester Royal Infirmary, and at the Manchester Workhouse Infirmary, where I have 800 medical beds.

By the old so-called alcoholic paralysis I mean those cases of peripheral neuritis, with much paralysis and atrophy of the legs and arms, loss of knee-jerks, great tenderness on pressure of the muscles, and peculiar and typical loss of memory for time and place, and often oedema of the legs—sometimes general oedema—from dilatation of the heart. This was the type found so commonly during the arsenical poisoning epidemic, when with it there were also found various skin lesions—notably pigmentation, keratosis, various erythematata, and herpes.

These cases (even apart from the epidemic), I venture to think, were in reality due to arsenical poisoning and not to alcohol, but many observers have thrown doubt on this view. It is interesting, therefore, to be able to send a short report of what has happened in Manchester during the last two years, in which period Manchester beers have contained perhaps not $\frac{1}{10}$ gr. arsenic per gallon. A close look-out has shown that these cases have practically disappeared (up to about three months ago, about which more presently), and in this statement I am confirmed by the observations of several other Manchester physicians. The "alcoholic" heart has also practically disappeared. Now I think I may take it for granted that the people in Manchester and district are still taking alcohol in large quantities. If these cases are really due to alcohol, why have they disappeared? This is the negative evidence.

Now take the positive evidence. During the last two months I have seen six cases of the old so-called "alcoholic" paralysis type, five of whom are at present under my care.

CASE I.

Edward R., aged 40; has taken ten glasses of beer nightly, more at week ends. Has pressure pains well marked, loss of knee-jerks, some atrophy, paralysis of the legs, slight paralysis of the arms, no loss of memory, burning pains of the soles of the feet, slight general pigmentation.

CASE II.

Albert B., aged 42; very marked paralysis of legs and arms, marked pressure pains, marked wasting, purpura on legs, marked loss of memory, peculiar hoarse voice, marked keratosis of the soles of the feet; has been a heavy beer drinker.

CASE III.

Mary B., aged 49; well-marked paralysis and atrophy of legs and arms, pain on pressure, marked loss of memory, very slight keratosis; has taken much beer.

CASE IV.

Emily D., aged 53; paralysis and atrophy specially marked in the legs, pain on pressure, loss of memory, rapid heart, fairly marked keratosis; husband says she has taken much beer, no spirits.

CASE V.

Hessie T., aged 32; very marked paralysis and atrophy of legs and arms, great pain on pressure, slight oedema, slight keratosis, very rapid heart. This is a most interesting case. She is the wife of a beerhouse keeper, and both her husband and the doctor told me that she drank practically nothing but whisky—in fact, had got drunk on whisky every day for years. But the symptoms have only come on during the last two months. If the cause were alcohol *per se*, why had the symptoms not come on before? I therefore cross-examined the husband very carefully, and he said that during the last three months his wife had also taken a glass of beer occasionally, but he reiterated his statement that whisky was her particular and practically her only beverage. I asked if she had been taking as much as six glasses of beer daily, and he said: "Oh, yes; perhaps eight or ten glasses." This is only another example of what I have pointed out before—namely, that a spirit drinker does not count the amount of beer consumed; he or she looks upon beer more or less as a temperance beverage.

With these five cases under my care I thought it was time to have the beer examined again; and two samples from different breweries, both of which had supplied more than one of the five patients with their beer were obtained for me and analysed, and both were again found to contain arsenic in quantities certainly greater than $\frac{1}{10}$ gr. per gallon, possibly about $\frac{1}{2}$ gr. per gallon.

How this arsenic is again getting into certain beers (even obtained from different breweries) I cannot at present say; but the matter is being investigated for me, as for various reasons it is not an inquiry which I can personally conduct.

I contend that the negative and positive evidences above put forward strongly support, if they do not prove, my statement that, at any rate in this district, what we have been calling for years alcoholic paralysis was really in the vast majority of cases arsenical in origin.

There are no acute symptoms of arsenical poisoning in the cases I have narrated because there is not the enormous amount of arsenic present in the beer which there was when it was manufactured from arsenical glucose; indeed, the only skin affection is the keratosis, to which I would again draw attention as a most valuable sign of arsenic poisoning even if slightly marked. I would again suggest, as I did in a paper read at the Cheltenham meeting of the British Medical Association in 1901, that just as minute traces of lead in drinking water may cause marked paralysis in some persons without any other symptoms of lead poisoning, so also minute traces of arsenic may in a few susceptible people cause peripheral neuritis, loss of memory, and heart failure. It is, of course, only a very small percentage of those taking the arsenic who suffer, for it must be remembered that although there were some hundreds of people affected during the late epidemic in 1900, yet there were tens of thousands drinking the highly contaminated beer who suffered hardly or not at all.

Another practical point, I think, to be noted from the above remarks is that it is not advisable that beer should contain more than $\frac{1}{10}$ gr. of arsenic per gallon or symptoms of poisoning may occur.

A CASE OF ANKYLOSTOMIASIS IN SCOTLAND.

By RALPH STOCKMAN, M.D.,

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THE widespread prevalence of ankylostomiasis in Central Europe and the recent occurrence of a severe outbreak among the men employed in the Dolcoath Mine, Cornwall, arouse the fear that it is only a question of time for the parasitic worm to become widely distributed throughout the mines and brickfields of the whole country. Each person who harbours the intestinal worms and passes ova becomes a centre of infection for others, and owing to the constant migrations of workmen the disease is apt to be spread easily and rapidly. It is very prevalent in Egypt, India, and other

tropical and subtropical countries, and since 1854 the ankylostoma duodenale has been generally recognized as the cause of "miners' anaemia," "tropical anaemia," and "Egyptian chlorosis." As a consequence its life-history has been very carefully studied and is now well-known. In temperate climates the ova develop into the infective larval form most readily under the conditions of temperature and damp which are present in mines, tunnels, and brickfields, hence, in Europe, men working in these places are most frequently, but by no means exclusively, affected. In warm climates the ova flourish in the moist earth anywhere, and hence the agricultural classes and the rural population generally may be greatly afflicted by the parasite and the resulting anaemia. The number of worms in the intestine may amount to several hundreds, and they cause the anaemia by direct abstraction of blood from punctures made in the mucous membrane. A fatal result is by no means uncommon if the case is left untreated. Where the number of worms is small, the host may suffer little inconvenience, and, if he be not further infected by swallowing fresh larvae, the original worms die in about six years, and a natural cure results. The mature worms are very seldom passed in the faeces, and a diagnosis is made by finding the characteristic ova on microscopic examination. The worms are readily killed by large doses of thymol or oil of male fern. The blood presents the usual characters of slight or severe anaemia, but, as in several other diseases, there is a more or less marked absolute and relative increase of the eosinophile leucocytes.

The following case is important from a public-health standpoint, and is interesting as being probably the first which has been observed in Scotland.

The patient was a man, aged 25, a coal-miner by occupation, and working in a Lanarkshire mine. He was admitted to the Western Infirmary on May 15th, 1903, complaining of general weakness and shortness of breath of over a year and a-half's duration. He dates his present illness from an attack of "enteritis," which he contracted in India about two years ago while serving as a soldier, and for which he was treated in hospital at Lucknow for two months. On his discharge from hospital he noticed that he was pale, and very breathless on going upstairs or making any unusual exertion. After being three months longer off duty he was invalided home to Netley Hospital, where he was treated for anaemia with iron, arsenic, and tonics. He did not improve, and six months ago was discharged from the army as suffering from leukaemia.

About the beginning of this year he returned to his former employment as a miner, but has never been really fit for work, and a fortnight before admission had to give it up on account of weakness and breathlessness. He was accustomed to defaecate daily in any convenient part of the mine. He has had no serious illnesses except two attacks of malaria in India; he is a moderate smoker and drinker, and of good physique.

On admission the patient presented the outward appearances of pretty severe anaemia. The cardiac impulse was feeble, the heart was slightly dilated; there were soft systolic murmurs in the mitral and pulmonary areas, and a venous hum in the neck. The pulse varied from 62 to 96 per minute, but was full and of fair tension. He was subject to palpitation. There was no enlargement of the spleen or of any of the lymphatic glands. The lungs and abdominal organs were all healthy, the urine contained no abnormal constituents; the appetite and digestion were good, but the bowels rather constipated. The red blood corpuscles formed rouleaux normally; they were pale in colour, and showed some degree of poikilocytosis. They numbered 3,200,000 per c.mm., the leucocytes were 9,000 per c.mm., and the haemoglobin 28 per cent. of the normal. On several examinations the eosinophile cells were counted as equal to from 12 to 43 per cent. of the total number of leucocytes (the normal being about 2 to 4 per cent.).

On examining the faeces numerous ova of ankylostoma duodenale were found, most of them in the four-celled state, with others more advanced in segmentation. On May 21st (before treatment) the leucocytes numbered 7,850 per c.mm. and their proportions were: polynuclear cells 62 per cent., eosinophiles 12 per cent., large lymphocytes 10 per cent., small lymphocytes 16 per cent.

The patient was put on six Bland's pills daily, and on May 26th he received 20 gr. of thymol in powder, preceded and followed at an interval of several hours by 2 drachms of castor oil. Owing to a misunderstanding this stool was not properly examined for the worms, but none were found on subsequent occasions, although ova were present in the faeces up to June 10th but no later. On May 29th and June 5th the thymol was repeated, but no worms were discharged.

On June 10th the red blood corpuscles equalled 5,000,000 per c.mm., the white 5,000, and the haemoglobin 70 per cent. of the normal; the polynuclear leucocytes were 64 per cent., the eosinophiles 13 per cent., the large lymphocytes 6 per cent., and the small lymphocytes 17 per cent. His colour had become good, and the subjective symptoms of anaemia had passed off. He was sent to the convalescent home on June 12th, and on his return a fortnight afterwards he reported himself as being in perfect health. The red blood corpuscles then numbered 5,700,000 and were well formed, the white 8,420, and the haemoglobin equalled 90 per cent.; the polynuclear leucocytes numbered 42 per cent. of the whole, the eosinophiles 43 per cent., the small lymphocytes 10 per cent., and the large lymphocytes 5 per cent.