cause of the affection, whether it is due to the alcohol of beer and spirits, or to some impurity, or, in certain cases, to arsenic, which was found by Dr. E. S. Reynolds a few years ago in the beer sold in Manchester and proved by him to be the cause of an epidemic of peripheral neuritis. I wish in this note to refer simply to one sign which I believe to be of some value in the diagnosis of alcoholic failure of the heart from other forms of cardiac muscle failure. In cases in which shortness of breath, oedema, and other signs of cardiac failure are present the diagnosis of alcoholic heart failure is based chiefly on (1) the history and evidences of alcoholism; (2) on the absence of indications of valvular cardiac disease; (3) on the absence of nephritis; and (4) on the absence of indications of other known causes of cardiac muscle failure. Several peculiarities in the symptoms have been pointed out by Dr. Steell. In the diagnosis of alcoholic heart failure with secondary albuminuria from chronic nephritis with secondary cardiac failure I would emphasise the importance of ophthalmoscopic examination, since the detection of albuminuric retinitis would indicate the latter diagnosis. (To the writings of Dr. Steell the reader may be referred for an account of the diagnosis of alcoholic heart failure.)

A definite history of alcoholic excess is often difficult to obtain in private patients and especially in females, and hence other evidences of alcoholism are of value in the diagnosis. The knee-jerks are lost in some cases of alcoholic heart failure, but I soon found that the proportion of such cases was too small for the sign to be of any diagnostic value. Loss of the tendo Achillis reflex is one of the first signs of the injurious action of alcoholic beverages on the peripheral nervous system and this reflex is often lost long before the knee-jerk. As it appeared probable that in this sign we should have an indication of diagnostic value I have carefully examined the condition of the tendo Achillis jerks in 21 cases of alcoholic heart failure and in many cases of heart disease which have not been caused by alcoholism. In making the examination I have used a *heavy* percussion hammer. The following are the results which I have obtained:—

21 Cases of Alcoholic Heart Failure.

	Tendo Achillis jerks.	Knee-jerks.
18 cases with well-marked symptoms ...	Both lost ... 16 cases	Present ... 10 cases. Lost ... 6 "
3 cases with slight symptoms ...	One lost and one present 2 "	One present and one lost 1 " Both present 1 case.
	Both present ...	Both present.

In these cases paralysis was absent and there were no signs of neuritis beyond loss of reflexes, pains in the legs, and muscular tenderness. The two latter symptoms were often very slight or absent.

100 Cases of Cardiac Disease not due to Alcoholism (Valvular diseases, and dilatation or muscle failure not due to alcoholism).

	Tendo Achillis jerks.
100 cases ...	Present ... 98 cases. Lost ... 2 "

In the two cases of non-alcoholic heart disease in which the tendo Achillis jerks were lost the knee-jerks were absent and there was the Argyll-Robertson pupil. Both were cases of aortic valvular disease and there can be little doubt that the loss of the tendo Achillis jerks and knee-jerks and the Argyll-Robertson pupils were all due to commencing tabes dorsalis.

These observations demonstrate, therefore, that in cases of heart disease not due to alcoholism the tendo Achillis jerks were present in all cases except two, and in these two cases the loss of these reflexes was almost certainly due to early tabes. On the other hand, in *alcoholic* heart failure the tendo Achillis jerks were lost on both sides in 16 out of 21 cases, on one side in two other cases; they were both present in only three cases. In these three cases the symptoms were slight and the diagnosis not quite certain. Hence we may say that the tendo Achillis jerks are present in non-alcoholic heart disease unless there is some nervous complication, but are usually absent in alcoholic heart failure if the symptoms are well marked and if oedema and dyspnoea are both present (i.e., absent in alcoholic heart failure on both sides in 76 per cent. of cases, on one or both sides in 85 per cent.).

There are, of course, individuals who can take very large quantities of alcohol without either cardiac failure or loss of the tendo Achillis jerk occurring. Hence it is the absence of this reflex which is of chief value in diagnosis as one indication of alcoholic excess.

The loss of the tendo Achillis jerks in alcoholic heart failure is probably due to very early neuritis. It is not due to the oedema of the leg, since these reflexes may be obtained in non-alcoholic cases when there is marked oedema.

Probably the tendo Achillis reflex is always present in health. Under the age of 50 years it is generally admitted that the reflex is always present in the normal condition, but it was stated a few years ago that after the age of 50 years it was sometimes absent. Some observers have found, however, that both over and under 50 years of age the tendo Achillis jerk is always present in health. This has been my own experience. But in order to obtain evidence on this point I have recently examined the tendo Achillis jerk in 100 individuals over the age of 50 years who were either in good health or not suffering from any affection of the nervous system. The reflexes were present in all of the 100 cases.

It is to be remembered that the tendo Achillis jerk is often lost in diabetes mellitus² and after diphtheria. In both of these conditions cardiac failure may occur, but the history in the latter affection and the condition of the urine in the former are diagnostic. In lesions of the lower part of the spinal cord and of the cauda equina this reflex may disappear on both sides and in severe sciatica on one side (the side of the pain).

From the facts just recorded I think we may conclude that under certain conditions the tendo Achillis reflex may be of service in the differential diagnosis of alcoholic heart failure, but we should first exclude diabetes mellitus, diphtheria, and tabes as causes of the loss of the tendo Achillis jerks. Providing there is no glycosuria, no evidence of a recent attack of diphtheria, no sign of lesion of the spinal cord or cauda equina, and providing the pupils react to light and that there are no lightning pains or other signs of tabes dorsalis, then the loss of the tendo Achillis jerk is of value in diagnosis as a confirmatory sign that the alcoholic beverages taken have had an injurious action on the system—i.e., that they have produced one of the first signs of their toxic action on the peripheral nerves. (It is interesting to note that there were no signs of peripheral neuritis in any of the 21 cases examined beyond the loss of reflexes and in some cases aching pains and tenderness in the legs.)

The Argyll-Robertson pupil is practically never produced by alcoholism, but it is found in most cases of tabes. Hence the pupils should always be tested before attaching importance to the loss of the tendo Achillis jerk.

In a case of heart failure, in which, from the symptoms and signs, alcoholism appears to be a possible cause, the loss of the tendo Achillis reflex would be strongly in favour of this view, if we can exclude the other affection, just mentioned, which produces loss of this reflex. If the symptoms of cardiac failure are well marked the *presence* of the tendo Achillis jerk would be a point against alcoholic heart failure. But if the symptoms of heart failure were slight then the presence of the tendo Achillis jerk would be of little diagnostic value.

Manchester.

A CASE OF SPLENIC ABSCESS.

BY REGINALD H. LUCY, M.B., C.M. EDIN.,
F.R.C.S. ENG.,

SURGEON TO THE SOUTH DEVON HOSPITAL, PLYMOUTH; CONSULTING
SURGEON TO THE KINGSBRIDGE AND LISKEARD
COTTAGE HOSPITAL.

ABSCESS of the spleen seems to be so rare in this country that the following instance may prove of interest.

On Nov. 17th, 1906, I was asked by Mr. S. G. Vinter of Torpoint to see a married woman, aged 23 years. She had been delivered with forceps of her first child, a boy, after an eight hours' labour, on Oct. 14th, 1906. The perineum was not torn, but the placenta being firmly adherent was removed piecemeal, leaving a rough, shreddy surface. A lysol douche was given immediately afterwards and on each succeeding day. On the fifth evening post partum the temperature ran up to 104.4° F., followed on the thirteenth evening by two severe rigors, recurring the two succeeding days, while on the seventeenth morning the temperature fell to 98° and the pulse to 84, which was accompanied by a profound rigor, lasting half an hour. The temperature again rose to 104.6° and oscillated violently.

² R. T. Williamson: Review of Neurology and Psychiatry, October, 1903.

On the evening of the twenty-second day 20 cubic centimetres of antistreptococcic serum were given per rectum, repeated next day, and again on the twenty-seventh, thirtieth, and thirty-fourth to thirty-ninth days. During this time the pulse-rate varied between 100 and 130 per minute with profuse sweating and occasional vomiting. On the thirty-fifth evening following delivery I found the abdomen distended, tympanitic, and tender, more especially in the left hypochondrium, where a hard lump could be felt, moving slightly on respiration and exhibiting the characteristic notch on its anterior border. Being uncertain as to the presence of pus in what was obviously the enlarged spleen and also whether that organ was adherent to the anterior parietes or not I advised the continuance of the antistreptococcic (polyvalent) serum. This was given daily until the forty-first day, when I saw her again with Mr. Vinter and his partner, Mr. R. J. Percy Thomas. The spleen had increased in size; it no longer moved on respiration, but still retained its notched border and the parietes superjacent were slightly reddened and cedematous.

Under chloroform a three-inch vertical incision was made along the centre of the left rectus sheath; the areolar tissue was found to be cedematous and on pushing a pair of sinus forceps through the peritoneum a quantity of horribly foul-smelling pus of a dirty yellow colour, mixed with greyish-purple sloughs of splenic tissue, escaped. A large rubber drain-tube was passed into the cavity of the spleen and stitched to the skin incision. The temperature rapidly fell and in 48 hours was normal and remained so during convalescence, while the pulse-rate soon reached 84. The number and bulk of sloughs that came away were extraordinary, but very little bleeding occurred. The cavity closed at the end of three weeks.

The interesting features of this case seem to be: (1) the spleen was the only organ or part affected; (2) though swollen it never lost its characteristic outline, but ceased to move on respiration; and (3) the large amount of splenic tissue lost in the form of sloughs and yet there are now (May, 1907) no apparent blood changes. I regret that no microscopical or bacteriological examination of the discharge was made, but the stench was so overpowering that a specimen would have nauseated even the most hardened pathologist.

With regard to the pathology of this case it is probable that septic thrombosis of the veins at the placental site took place and from there an embolus was carried through the heart and pulmonary circulation into a branch of the splenic artery. As throwing a light on the reason why the spleen became the "seat of election" for infective embolism and infarction, a history was given that three weeks previously to the birth of her boy the patient fell from a table, striking the left side of her chest so violently across the back of a Windsor chair that the latter was broken by the force of her impact. It is therefore probable that a hæmatoma was caused in or around the spleen which subsequently became infected in the manner above outlined.¹ The patient has never resided abroad nor suffered from enteric or other fever, nor had she valvular disease of the heart. Abscess of the spleen occurs in the tropics during malaria or yellow fever, also in the course of enteric fever, septicæmia, pyæmia, and particularly in ulcerative endocarditis.

The following references in recent medical literature are appended. 1. Nolan,² in describing a case of abscess of the spleen, states that only five successful results are recorded after operation. His case is very similar to the one I have just described. A woman, aged 25 years, six weeks after confinement became ill and feverish. Enteric fever was suspected. Sudden acute pain in the left side was followed by dyspnoea and a pleuritic effusion was found, together with a greatly enlarged but not tender spleen. A vertical incision along the left linea semi-lunaris was made; a cavity was reached from which about a litre of dull brown pus, with a faintly sweet smell, escaped. The peritoneal cavity was not opened. The wound was plugged with gauze and uninterrupted recovery followed. It is doubtful whether this patient suffered from enteric fever or whether the abscess followed puerperal infection; in either case an infective embolus would be the proximate cause. This paper is referred to in the *British Medical Journal Epitome*, April 14th, 1894, para. 295, and also quoted by Mayo Robson in the *Medical*

Annual for 1895, p. 453. 2. Black.³ The patient was a sailor, aged 21 years, who two years previously had contracted yellow fever; the tumour was incised yielding three and a half pints of bluish yellow pus; the cavity was plugged and healed by granulation after secondary suture with complete recovery. 3. Mayo Robson in the *Medical Annual* for 1904, p. 651, refers to a case reported by Dr. G. Riolo in the *Riforma Medica* of April 24th, 1902, of a splenic abscess complicating malaria successfully treated by incision and drainage. 3, 4, and 5. Mr. Robson also refers to a case under his own care and cites a paper by Dr. A. L. Staveley in the *Annals of Surgery* for June, 1903, on Splenic Infection, reporting two successful operations. 6. Harrington⁴ reports two cases both following typhoid fever. 7. Moynihan⁵ refers to seven cases of splenic abscess treated by operation up to 1900 and three cases recorded since by Murphy, Karewski, and Eberhart.

With regard to the treatment it appears to me better to wait until adhesions to the anterior parietes have formed and then perform splenotomy with drainage rather than traverse the unguarded general peritoneal cavity to undertake splenectomy, an operation sufficiently severe in an uninfected patient with its amount of shock and risks of hæmorrhage, immediate and remote.

Plymouth.

RICE AND BERI-BERI:

PRELIMINARY REPORT ON AN EXPERIMENT CONDUCTED AT THE KUALA LUMPUR LUNATIC ASYLUM.

By WILLIAM FLETCHER, B.A., M.B., B.C. CANTAB.,
DISTRICT SURGEON, KUALA LUMPUR, FEDERATED MALAY STATES.

DURING the year 1905 an epidemic of beri-beri broke out in the Kuala Lumpur Lunatic Asylum. Commencing in February, it reached its height in July and August, declining somewhat towards the end of December. Out of 219 lunatics treated in the asylum during the year 94 persons were affected, of whom 27 succumbed to the disease. The chief constituent of the rations supplied to the inmates of the asylum was uncured (Siamese) rice, and in view of the fact pointed out by Dr. Braddon that beri-beri occurs chiefly amongst communities with whom such rice is the staple article of diet it was decided, with the sanction of the Government, to place half the lunatics on cured (Indian) rice. The Government readily gave its consent and the experiment was commenced on Dec. 5th, 1905. The result up to Dec. 31st, 1906 (i.e., one year and 26 days) was that 34 out of 120 persons fed on uncured rice suffered from beri-beri and 18 died, whilst among 123 patients dieted on cured rice there were no deaths from beri-beri and only two cases, both of whom were suffering from the disease on their admission to the asylum.

1. By uncured rice is meant the ordinary white rice, sold in the Federated Malay States as Rangoon or Siam rice, which is eaten by all classes except Indians and Malays. This rice after being harvested is taken to the mills, where it is husked and cleaned before being sold to the rice merchants. Cured rice is brownish in colour and forms the staple of diet of Indians and Ceylonese. The great difference between this rice and the uncured variety is that the former is boiled and dried before being milled. The Malays keep their rice stored unhusked and pound and winnow it when they wish to cook it. A. White rice is stored unboiled after being husked. B. Cured rice is stored after being boiled and then husked. C. Native Malay rice is stored unhusked. Except for the difference in the rice the two parties—those on cured and those on uncured rice—received the same kind and the same amount of rations. Excepting the rice the food-stuffs for all patients were prepared together in the same kitchen and cooked in the same cooking pots.

2. The lunatics are housed in two exactly similar buildings on opposite sides of a quadrangle surrounded by a high wall. On Dec. 5th all the lunatics at that time in the hospital were drawn up in the dining shed and numbered off from the left. The odd numbers were subsequently domiciled in the ward on the east side of the courtyard and no alteration was made in their diet, they were still supplied with the same

¹ Cf. Moynihan's Abdominal Operations, p. 646.

² Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde, March 10th, 1894.

³ Brit. Med. Jour. Oct. 17th, 1896, p. 1116.

⁴ THE LANCET, Nov. 11th, 1905, p. 1393.

⁵ Abdominal Operations, p. 646.