of the depression and instability of the function of conductivity. It is interesting to note how the pulse-frequency increases as conductivity improves and *vice versa*.

In Fig. 2 we record the conductivity as before and compare it with the auricular and ventricular frequency. The auricular and ventricular frequency are calculated from the venous and radial pulse tracings. The administration of thyroid is shown by the two horizontal lines in the upper portion of the chart. This chart demonstrates marked variations in the auricular frequency which at first sight might appear to be the result of the administration of thyroid. This, however, is probably not the case, because we find similar and marked fluctuations in the auricular frequency occurring during the prolonged and uninterrupted administration of thyroid from Jan. 20th to March 7th. On Jan. 19th, the day before the patient began to take thyroid, the auricular frequency was 63, on Jan. 23rd it had fallen to 60, on Jan. 26th to 56, and on Feb. 8th it was recorded as 100. On Feb. 10th it had again fallen to 63, rising to 75 on Feb. 22nd. On March 2nd, the last record before the temporary cessation of the thyroid, the auricle was beating at 66. The auricular frequency appears therefore to vary independently of the administration of thyroid. We mention this because the chart from March 7th to April 10th certainly suggests that the variations in auricular frequency were the outcome of the exhibition of thyroid. For this reason it is unfortunate that the exhibition of thyroid was stopped on March 7th when the fits began and all routine treatment was in abeyance. So far as we know this method of charting the conductivity in relation to auricular and ventricular frequency is new, and we believe that observations carried out over long periods and charted in this manner will do much to help to a better understanding of the determining factor in the seizures.

In this patient the *a.v.* bundle was partially obliterated; it was functionally and organically below normal. As a result of this damage to "the pathway of the stimulus to contraction" a 2:1 rhythm had to our knowledge been present for months and probably, judging from the history, for years. Organic lesions of the *a.v.* bundle are, as a rule, progressive, and this man did not prove an exception. In January, 1906, he entered on what one may term the second stage of Stokes-Adams disease and began to suffer from syncopal and epileptiform seizures. Such seizures usually occurred in groups and were preceded by certain prodromal symptoms which indicated an increased liability to fits. Subsequently to these attacks the patient felt much better and on several occasions quite well. At such times conductivity was normal. Variations also occurred in conductivity, irrespective of the occurrence of convulsive seizures.

In this case two factors at least must be considered of importance: (1) The organic changes in the *a.v.* bundle which were demonstrated post mortem; and (2) nervous influences which either directly affected the conductivity of the remnant of the bundle or which altered the auricular frequency increasing it and so depressing conductivity by diminishing the resting periods of the bundle. It will probably be urged that the variations in auricular frequency were directly due to the action of the thyroid. There can, of course, be no doubt that the tendency of the thyroid would be to increase this frequency, but a careful consideration of the first half of Fig. 2 leads us to hold the view that there were agents other than thyroid affecting the auricular frequency. These agents were of varying intensity, causing great fluctuations in the degree of conductivity: note that serious depressions were followed by periods in which the conducting power of the damaged bundle was far above its normal. This is most suggestive of a vagus influence, and if it were not that the periods of depression were associated with increased auricular frequency we would feel inclined to ascribe these changes to vagal activity. Gaskell has shown that stimulation of the vagus³ first lowers conductivity, and in the case of a 2:1 rhythm may cause complete block; later, as an after-effect of such stimulation, it greatly increases the conducting power and may even transform a 2:1 into a normal rhythm. The stimulation of the vagus required to bring about this change in conductivity need only be slight, and such slight stimulation does not necessarily affect auricular frequency. This is beautifully shown in a tracing published by one of us in the *Manchester Medical Chronicle* for September, 1906. It was obtained from a patient of Dr. James Mackenzie suffering

from early aortic and mitral disease.⁴ The slight stimulation of the vagus produced by swallowing was sufficient to bring about temporary block, and two ventricular systoles dropped out as a consequence.

An analysis of the tracings in this patient revealed a slight but persistent depression of conductivity. The vagus was consequently acting on an already functionally abnormal bundle and accordingly was potent. The increase in the depression of conductivity was not associated with any slowing of auricular frequency, and thus the tracing is a beautiful example of the selective action of the vagus, one function responding to the depressing effect because already damaged, the other escaping. Another example of the fact that a bundle damaged functionally or organically differs in its response from the normal bundle is found in the effect of digitalis on the heart.⁵ Digitalis when pushed has no effect on conductivity unless the function is before the administration of the drug already below the normal. If there be a persistent depression in conductivity the digitalis still further increases this depression and eventually causes the dropping out of ventricular systoles and a tracing is obtained similar to that referred to above.

Summary in conclusion.—In this communication we have recorded a case of Stokes-Adams disease in its second stage—that is, in the stage of syncopal, apoplectiform, or epileptiform seizures. These seizures were of all grades of severity and tended to occur in groups with periods of comparative comfort intervening. The advent of seizures was usually indicated by definite prodromal symptoms. The necropsy revealed partial obliteration of the *a.v.* bundle; this would cause a persistent depression of conductivity. An analysis of numerous tracings revealed marked variations in conductivity, all grades from normal conduction to complete heart block being in evidence at one or another time. This suggests influences in addition to the organic lesion. Such influences were probably nervous in character and affected conductivity either directly through the vagus or indirectly by altering the auricular frequency.

NOTE ON DETERMINATIONS OF THE AMOUNT OF PHYSIOLOGICALLY ACTIVE HYDRO- CHLORIC ACID IN THE STOMACHS OF NORMAL MICE AND OF MICE SUFFERING FROM CANCER EXPERIMENTALLY PRODUCED.

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THE main object of our experiments has been to ascertain whether an increase or decrease of physiologically active HCl could be demonstrated in the stomachs of "cancer mice" with either ulcerated or non-ulcerated tumours, as compared with normal mice.

The experiments, which extended over the period December, 1905, to June, 1906, have involved the examination of over 500 stomachs in seven series, with an average number of 70 in each series, including all three classes—A (normal), B (non-ulcerated tumours), and C (ulcerated tumours). Dr. E. F. Bashford, director of the Imperial Cancer Research Fund, to whom we are indebted for the necessary material, arranged that the mice should be killed one hour after a full meal of bread and milk, and that the stomachs should be sent to us for examination as speedily as possible after death.

The method adopted for determining the physiologically active HCl was that described by Dr. W. H. Willcox, of St. Mary's Hospital, in THE LANCET,¹ with such small modifications as seemed desirable for our special purpose. The *modus operandi* is as follows. The stomachs belonging

⁴ See also Brit. Med. Jour., Oct. 27th, 1906, p. 1110.

⁵ Mackenzie (J.): The Action of Digitalis on the Human Heart, Brit. Med. Jour., March and April, 1905.

¹ THE LANCET, June 10th, 1905, p. 1566.

³ Gaskell: The Contraction of Cardiac Muscle, Text-book of Physiology, vol. ii. Edited by Schäfer.

respectively to Classes A, B, and C are counted, weighed, and macerated in water for about 12 hours, filtered through fine linen, and the filtrate from each series made up to 200 cubic centimetres. The testing of this filtrate is carried out in the following manner. (a) The *total chlorides* (inorganically and organically combined chlorine) is estimated by Volhard's method in 50 cubic centimetres. (b) 50 cubic centimetres are evaporated to dryness on a water-bath and charred at a gentle heat, after which the residual *inorganic chlorides* are determined by the same method. (c) The *total acidity* is determined in 50 cubic centimetres by titration with $\frac{N}{10}$

sodium hydrate solution, phenolphthalein being employed as the indicator. From these determinations are deduced: 1. Physiologically active HCl = $(a - b)$. 2. Organic acids in terms of HCl = $[c - (a - b)]$. Our results have been expressed as percentages of the average weight of the stomachs. Taking into consideration the whole seven series of experiments, we have found, on the average, a decided *increase* of physiologically active HCl in the stomachs of Classes B (non-ulcerated tumours) and C (ulcerated tumours) over that in the case of Class A (normal mice); the increase amounting in some cases to as much as 50 per cent.

It is necessary to direct attention to the fact that in these preliminary experiments, owing to the small amount of the contents of the mouse's stomach, the estimations have been carried out on extracts of stomach contents and stomach tissue, a fact which may possibly account for the interesting divergence between our results and those of a number of previous observers who have worked with material removed, after feeding with test meals, from the stomachs of human beings suffering from malignant disease. Further work dealing with the stomach contents only in normal and in "cancer" mice is in contemplation, and will be carried out as soon as opportunity serves for obtaining simultaneously the necessary number of mice.

ERYTHEMA AUTUMNALE, HARVEST RASH, OR PRURIGO DU ROUGET.

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WHEN investigating the origin of the skin affection ultimately traced to the handling of the caterpillar of the yellow-tailed moth and described in THE LANCET of August 4th of the present year I found that the agricultural labourers in Essex were acquainted with some form of irritating skin affection which more frequently affected their legs than any other part of the body and which they attributed to the "harvest spider," "harvest bug," or "harvesters," since the eruption was most frequently met with when these insects were most prevalent. Further inquiry showed that during this autumn the spider (*Phalangium opilio*) has been very prevalent and that sportsmen not wearing buskins have suffered severely from the pest. The *fons et origo mali*, however, is not the harvest spider but a mite parasitic upon it, the larva of a trombidium. Murray, in his "Economic Entomology," briefly refers to it (though he does not appear to regard it as a trombidium), and says: "This troublesome insect most prevails near the sea-shore where the soil is light. It begins to appear in the early part of July and is very troublesome in August. Their great numbers may be seen crawling upon and over all green things, on turnips, grass, and corn. The larval form is that in which this species is generally encountered; and although it has long been supposed that it was merely the larva of a similar mite, yet it is only a few years since the fact was determined by direct observation." During the present season I have found that ladies were the chief sufferers, probably on account of their possessing more delicate skins. One medical friend who wrote to me for information said that the effect of "harvesters" was well known in Oxfordshire, that it was a most obstinate and distressing malady, and that some lady friends had been made very wretched by it. A lady informed me that she and her mother and sister had been into a remote country district for a holiday and had all returned because they had been attacked with a skin disease which they thought might be

scabies but which I have no doubt was prurigo du rouget. A full description of this malady is given by P. Mégnin in "Les Parasites et les Maladies Parasitaires chez l'Homme, les Animaux Domestiques," &c., 1880. He calls the malady "prurigo du rouget," a term which he prefers to "erythema autumnale," under which it appears to have previously been described. He regards the very minute mite (rouget) which causes the malady as being the hexapod larva of the silky trombidian (*Trombidium holosericeum*). Plate XXV. shows this species in all its stages of development. The larval form is so small as to be barely visible to the naked eye, but being of a brick-red colour masses of them are easily distinguishable. They apparently breed upon plants but when opportunity occurs transfer themselves to man or animals. They chiefly attack persons with delicate skins and seem to prefer the legs and inner side of the thighs, but may reach any part of the body. From the legs they very quickly travel to the upper parts of the body unless their progress is arrested by some tight article of clothing. The base of the downy hairs attracts them and here they attach themselves by their formidable-looking claws and insinuate their suckers under the epidermis or into the glands at the base of the hairs. The abdomen of each larva then gradually distends until it is four or five times its original size and becomes visible as a bright red dot. The itching produced is so intense that the patient cannot rest. The skin swells, becomes red, or sometimes even violet, around the points attacked. These points, which may be a centimetre in diameter, are always discrete and usually have a central projection of appreciable size. Sometimes the eruption is distinctly papular or vesicular and if rubbed may become pustular.

The journal *Sante Publique* for May, 1872, contains the history of an outbreak which led to judicial proceedings. A number of men engaged in unloading certain sacks of corn speedily complained of intense itching on the exposed parts of the body. During the night five of them became covered with an eruption which was associated with a certain amount of fever, insomnia, and ardent thirst. The initial erythema was followed by an eruption of acuminate pimples on the arms, the chest, and the neck. It was at first alleged that they had been poisoned but an expert who was consulted found that the corn swarmed with acari, and from the description given of these mites Mégnin concludes that they were the larva of a Trombidian and that these were the cause of the illness.

The treatment which has been found most beneficial in France is the application of benzene to kill the acari, followed by warm baths, or the use of dilute vinegar to allay the irritation. If a delicate part has been affected, such as the eyelid, the use of a fine needle may dislodge the parasite.
Chelmsford.

THE TREATMENT OF LARYNGEAL TUBERCULOSIS.¹

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THE treatment of laryngeal tuberculosis will always be a difficult problem and the results will never in the main be brilliant. We have here a tuberculous lesion which occurs most often in a patient the subject of advanced consumption, in a part which is difficult of access, which can never be kept thoroughly aseptic, and where complete immobility cannot be obtained. When the disease in the lung is so far advanced or so rapidly progressing that a restoration to some degree of health and comfort cannot be looked for, I hold as strongly as possible that the treatment of the laryngeal affection should be entirely symptomatic; that where there are no severe symptoms it is both useless and cruel to submit the patient to active treatment either with cutting instruments or strong caustic pigments. Such cases make up a large proportion of all the cases of tuberculous laryngitis which come under my care. Nevertheless this is, to my mind, no reason why active treatment should be

¹ A post-graduate lecture delivered at the Mount Vernon Hospital for Consumption.