in these cases a double curve—a primary wave, due to the inoculation; and one or more secondary curves, due to the incidence of autoinoculations. Fortunately, these reactions are not fraught with so much anxiety as formerly, as it is possible to bring them to an end by a dose of bovine tuberculin.

This question of the bovine and the human strain is at the moment agitating the medical world, and whether one should use a homologous vaccine, or whether it is an advantage, as is claimed by Nathan Raw and by Camac Wilkinson, though on widely differing grounds, that the appropriate tuberculin to combat infection caused by the tubercle bacillus *typus humanus* is that manufactured from the *typus bovinus*, remains still unsettled. According to some searchers there is no difference between the supposed varieties, but these are now in a minority, and opinion is, I think, overwhelmingly in favour of the view that the bovine and the human tubercle bacilli belong to two separate and distinct varieties. The method I have used in the cases I am now bringing forward is that advocated in the *Lancet* of April 23rd, 1910. I would like, however, if I may be permitted, to advance the suggestion that larger doses of tuberculin than these should be more efficacious if they could be given without acute reactions. Perhaps a way may be found to control these reactions, or rather to prevent them, but until that time comes I can recommend my method with complete confidence to any medical man, whether he has experience of inoculation work or no, as being reasonably safe and most unlikely to result in harm.

Before closing my remarks there are two points I should like to touch upon.

The first is that I do not think it can be too often insisted upon, notwithstanding all that Spengler and Wilkinson may say to the contrary, that in the hands of the ordinary medical man there is grave danger associated with the exhibition of tuberculin in phthisis whilst patients are walking about and pursuing their ordinary avocations. It is only a matter of common sense after all. Tuberculin causes hyperæmia, and the more hyperæmic the lesion the more easily will a dangerous autoinoculation be induced by exercise. True the cure may be through autoinoculations; but if autoinoculations be allowed to become excessive, as they will become excessive if tuberculin and exercise are combined, then inevitably will the disease extend.

The second point is as to the length of time that it is necessary to continue treatment. Too little is known of the diagnostic tests of Calmette, Wolff-Eisner, Moro, and v. Pirquet, to adjudge their value as trustworthy guides on this question. Two excellent indications, however, remain to us, and both should be utilized before giving a definite opinion:

1. The patient should be able to take 1000 T.R. without any effect noticeable, either in the temperature or in the opsonic index.

2. After sufficient exercise to cause thorough exhaustion there should be no sign of an autoinoculation; that is to say, that the opsonic index, a far more reliable test than any subjective sensations and many times more delicate than the temperature—the opsonic index should not fluctuate.

One example of this and I have finished. A boy, whom I had considered cured, remained well for three months, then had an attack of influenza. After recovery the temperature remained above normal. The parents, afraid of a recurrence of lung trouble, kept the lad in bed. I ordered him to get up, to walk about for such time as might cause him to be fairly tired, and to send me specimens of blood taken before and at varying intervals after the exercise. I found no alteration in the index to tubercle, and I ordered him to disregard his temperature and to get up every day. The result justified this decision, for the temperature came down to normal after a few days, and in a short time he was able to return to school. The other day I heard that he had just won a quarter of a mile race.

THE next meeting of the German Psychiatric Association will be held at Stuttgart on April 21st and 22nd, 1911. Among the communications promised is one of methods of psychological investigation by Professors Kraepelin of Munich and Sommer of Giessen.

TYPHOID INFECTION.

BY

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TYPHOID fever is more common in autumn, and in consequence has been called "autumnal fever." There must be a reason for its greater prevalence at this season.

The rains of this time of year, as favouring the passage of the typhoid bacilli into sources of drinking water, have been assigned as a cause. But heavy and continued rainfall is common at other periods without a greater incidence of this disease. Such an explanation cannot, therefore, in itself be correct. Again, it may be said that the temperature in autumn is more favourable for the growth of the bacilli in media such as water, milk, and other everyday foods. This is not so, for typhoid bacilli grow best at 37° C., or about blood heat, which is a temperature belonging to the summer months. (Typhoid bacilli do not grow below 9° C. nor above 42° C.)

The fall of the leaf and fruit, it is conceivable, might produce decompositions favourable to bacillary growth; but here again the temperature would not be suitable. Flies, it is known, can and do convey typhoid; their number, however, decreases at the end of summer, and as a rule they are few and far between in October and November.

Another possible factor may be suggested: It is in autumn, when the grain has been gathered and as winter approaches, that rats and mice leave the fields and make for human habitations. One means by which they obtain entrance—as a matter of common observation—is by drain and sewage passages, especially if these be defective. (Rats themselves are apt to cause leakages and defects in improperly constructed sewage passages.)

Granted the existence of people with actual typhoid, or of human typhoid carriers whose excreta is among the sewage, where the rats live, and which it is possible they devour, it is easy to see how millions of bacilli may be present on the body, feet, jaws, and tails of these rodents, whereby food and drink may become directly as well as indirectly contaminated.

In addition typhoid bacilli may exist in the intestine of mice and rats, and their excreta in turn be a source of infection. (Proof of the latter assumption might be sought in bacteriological examination of the droppings and intestinal contents of animals in a typhoid area, and experimentally by feeding rats with typhoid germs.)

In this connexion it is interesting to note that the virulence of a great number of organisms can be increased by the passage from one animal to another, so that an attenuated and comparatively harmless growth of typhoid might, through the agency of a rat for instance, become at any time virulent and highly infective. Conveyance to fingers is probable, nor is the distance from there to the milk pail far.

Dr. J. Clements, Medical Officer of Health for Beckenham, Kent, is responsible for the statement:

Typhoid is a pure filth disease, and wherever there are facilities for human excreta to be carried to food and drink there cases of enteric will occur.

He further mentions in one of his reports that Dr. Robertson, of Birmingham, has grown typhoid bacilli from one season to another in the soil of a field.

Mr. Anderson, clerk of works at Durham County Asylum, has several times seen rats go up and down the pans of closets in the lavatories, and the workmen, on questioning them, have seen the same. There have been sporadic outbreaks of typhoid at that asylum for several years. Since 1896 the number of male cases recognized and notified has been 22. Fifteen of these were not over 30 years of age. Similarly the number of female cases has been 17, of whom 8 were not 30 years of age. The females do no outside work. Some of the above are traceable to workers on the sewage garden; some are traceable to direct infection from one patient to another; several have this in common that they slept in a large upstairs dormitory accommodating seventy patients; it is more recently built than most of the other parts of the asylum, it is well provided with closets, and is and has been free from rats.

Explanations of this dormitory being a focus of infection are:

1. Some of the sewage garden workers have been accustomed to sleep in it.
2. Most of the recent admissions sleep here, among whom there might be at times an undiagnosed case of enteric whose delirium was mistaken for insanity.
3. The presence of typhoid carrier or carriers. Five isolated outbreaks have occurred in patients or attendants connected with it since January, 1905, the last of which was an attendant who died from typhoid in October, 1910. One of these cases, however, worked in the sewage garden.

Several of the remaining outbreaks show an association with rats. The Winterton division, where there are between 500 and 600 male and female patients, is about five minutes' walk from the main asylum. It is surprisingly free from flies at all times. The same cannot be said of rats. No. 3 female ward in particular being infested; recently there were two rat-holes leading into it. The nurses have on many occasions seen rats run into the drain-pipe from the lavatory. The ward is a large one, with accommodation for 80 patients, about half of whom are epileptics. About 10 per cent. only are females under 30 years of age. Three separate outbreaks have occurred here since 1896—A. S., aged 23, notified October 1st, 1903; 3 cases in the summer and autumn of 1905; 1 case, S. A. S., aged 24, who died October 30th, 1910. With the exception of a small, recently-built infirmary, which is free from rats, No. 3 female ward is the nearest to the sewage garden—the distance being about 500 yards—where there are pipes discharging the sewage from it and the other wards of this division of the asylum. The sewage garden appears to be a good breeding place for rats, as many as eighty being killed in it within a week. There are numerous rat-holes in it and the adjoining fields at the present moment.

In No. 2 female ward, at the main asylum, a considerable distance from the above, a nurse aged 26 years was notified on November 26th, 1909. Following on her at intervals 4 new cases occurred in the same ward, the last in October, 1910.

During this period rats have been frequently seen in the ward, especially in the scullery. The lavatory accommodation here is insufficient, but this is being remedied. There may be a typhoid carrier among the patients in this ward.

The wife of an attendant living in the village of Sedgefield, a mile from the asylum, took typhoid in the autumn of 1902. There were 3 cases of typhoid at the asylum then. Her husband had a terrier which was killing rats every day about the institution. The dog stayed in his house at night. This appears to have been an isolated case in the village.

Her husband, T. D., aged 35, was himself notified December 9th, 1907, five years afterwards. He had now two dogs which were ratting every day in the neighbourhood of the Isolation Hospital, outside which he had been employed for some time, and where there had been previously cases of typhoid. Both dogs were kept inside the house he lived in at night.

There were 4 cases of typhoid in 1906 on the male side of the main building, the first being an attendant stationed in No. 10 ward, the infirmary, where he and others were nursed. Over 200 rats were killed here in the year 1906-7.

There were 5 cases in connexion with No. 7 female receiving ward and No. 8 female adjoining it, both at the main asylum, in the summer and autumn of 1901. Rats were present in these wards at the time, and are still seen in both.

Concurrent with these female cases, W. A., a male, aged 50 years, was notified. He was employed at the time in one of the houses on the estate where there was a dog with which he killed rats daily.

It would be instructive to learn if an association with rats can be traced in other asylums and institutions where sporadic outbreaks occur not otherwise explainable; also, if unexplained cases are common among people possessing dogs who go ratting.

UNDER the will of the late Karl Beit of Hamburg, the Heidelberg Institute of Cancer Research will benefit to the extent of £5,000. During the last twelve months the Institute has received donations amounting to over £500.

MYASTHENIA WITH ENLARGEMENT OF THE THYMUS GLAND.

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The following are notes of a case of myasthenia with enlargement of the thymus gland:

A governess, 21 years of age, was sent to the Bristol General Hospital under my care, by Dr. Manning of Wells, on November 25th, 1909. Until November 15th, 1909, patient had enjoyed uniform good health, and there was no unfavourable family history.

On November 15th she complained of pain in the neck and headache, and was thought to be suffering from a cold. On the 18th she was confined to bed with weakness in the legs. The left leg was affected before the right, and the weakness was such that she could not turn herself in bed or stand upright. There was difficulty in swallowing, and on November 22nd a tingling sensation in the hands, and pain and loss of power in both arms, her friends stating that "she was able to move them slowly, but there was loss of strength in them." They also state that at this time "she did not feel a touch or a pinch." The pulse was rapid, and there were occasional attacks of heart failure.

On admission to the hospital the patient was much exhausted. When she had somewhat recovered, her manner was hysterical, she giggled and laughed, and said that she could not control herself. For these reasons a detailed examination of her nervous system was postponed. The temperature on admission was 97° F., pulse 106, respirations 24. There was no fever throughout her stay in hospital. She was menstruating.

There was loss of power in the trunk, arms, and legs. The wrists, elbows, knees, and ankles could be flexed and extended, and both upper and lower limbs could be voluntarily raised from the bed. They could only be raised for a short period, and the patient could not lift any object from the bed. She could not stand even with a moderate amount of support, the knees giving way beneath her. The muscles were not wasted. Occasionally there was twitching of the fingers. The facial expression was peculiar, there being ptosis of both lids, most marked on the left side, and inability to completely close the eyes, even during sleep. There was no squint, but the ball of the eye rolled up under the upper lid, so that only the sclerotic showed between the lids. This was constant during sleep, but only happened occasionally during the day. Mastication and swallowing were difficult, and saliva collecting in the mouth and mucus in the trachea gave rise to distress. It was difficult to get the patient to talk, but no defect of speech was present. There was no stridor and no enlargement of tonsils. The patient complained of a sensation of coldness in the legs, and a feeling of constriction behind the manubrium sterni, especially on swallowing. Sensation to touch and pain was normal. There was tenderness over the left ovary. The deep reflexes in the arms were normal, the left knee-jerk was not obtained, the right was present, but feeble. A feeble flexor plantar response was present on both sides. The sphincters were unaffected; the pupils reacted to light and accommodation. The heart and lungs were normal, and the pulse rapid. The urine contained no albumen or sugar, specific gravity 1030, acid. The striking feature of the case was the extreme lassitude and muscular weakness without wasting. Thirty hours after admission, at 1 a.m., the patient became restless, dyspnoeic, and cyanosed. Strychnine and oxygen were administered, but she died suddenly at 1.45.

The post-mortem examination was performed by Dr. Emrys-Roberts, who reported that the body was well developed and covered with fat. The palate was high and arched, teeth absent, no lobes to ears.

The thymus gland showed both lobes to be enlarged, each being 4½ in. long by 1½ in. broad. No cysts. The thyroid gland was also somewhat enlarged. The heart and lungs were normal. The stomach was normal. The duodenum showed a number of dilated capillaries, and the same condition, together with several haemorrhagic areas, was present in the ileum. The spleen was enlarged. The liver and pancreas showed capillary dilatations and minute haemorrhages.

Microscopic examination of the organs showed a considerable amount of altered blood pigment or debris in the liver and thymus gland, and more recent haemorrhages and dilatation of the capillaries, such as were also present in the intestinal walls. The thymus gland was normal, except that no Hassell's corpuscles could be found.

When first seen, this case suggested a diagnosis of hysteria. The mental condition, the complaint of tiredness and weakness, the ptosis, difficulty of swallowing, and breathlessness, were all compatible with such a diagnosis. The sudden fatal termination and the post-mortem appearances, however, taken in conjunction with the facial appearance, the peculiar nature of the muscular weakness, and the absence of sensory phenomena and the unaltered reflexes, all negated this diagnosis. Bulbar paralysis would not account for the ptosis, and the ordinary symptoms of a poliomyelitis were not present. A toxic polyneuritis or paralysis of Landry's