were ameliorated. He took the case of the *Impregnable* Training Establishment in 1912-13 (reported by Dudley). The boys, aged 15½, were housed in three old wooden battleships, the air space was continuous, and the hammocks were 16 inches apart. The incidence of tonsillitis was 397 and of rheumatic fever 43.7 per 1,000 respectively—a very high figure. This dropped to normal proportions on transference of the boys to barracks ashore.

In the report of the health of the Royal Air Force for 1928 a similar epidemic of tonsillitis and acute rheumatism is recorded, although the crowding here was not so marked. The incidence of tonsillitis was 175 and of rheumatism 16.5 per 1,000 respectively.

In the instance under discussion it is suggested, in view of the synchronicity of the attacks and the crowded living conditions, that these three children have either infected one another or have been infected by a possible carrier in the person of the fourth child, the boy with chronically infected tonsils. The latter seems the more likely, since the patient in Case 3 had not seen her sisters for a period of six weeks previous to admission to hospital herself, as the sisters had been in hospital and at a convalescent home for that time. This would imply a very long latent period for chorea.

CONCLUSIONS

1. Chorea may assume epidemic proportions in a family where the living conditions show overcrowding.

2. The cases reported support the contention that rheumatism may be contagious in overcrowded conditions.

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A CASE OF BRILL'S DISEASE

BY

H. M. ROYDS-JONES, M.B. B.S.

The following case seems worth reporting in view of the absence of diagnostic symptoms, and the interesting points arising from it.

HISTORY OF THE CASE

A male, aged 49, a vegetable cook working in the galley of a passenger liner with a crew of 400, reported to the ship's surgeon on February 25th, 1931, complaining that he had been feeling dizzy, had pains in the legs, and had been coughing for two days. His temperature was normal and physical examination was negative. He had never lived abroad, but had served in France during the war. At that time there was an epidemic of mild influenza among the crew; his case was diagnosed as influenza and he was given salicylates and alkalis.

On the morning of February 27th his temperature was 103.4° F., and he was moved into the ship's hospital among other influenza patients. He vomited twice that day. On the 28th he complained of nothing but the headache, which had become more intense, and a troublesome cough. That night he was slightly delirious. The temperature began to settle down on March 3rd, and the patient felt much better; but on the 6th it rose again and remained up until the 13th, at which time he felt perfectly well, but weak after being confined to bed. There was no enlargement of the spleen at any time during the illness and no physical signs of disease except a few occasional rales at the bases of the lungs. The bowels, which had been rather free during the first two or three days, were subsequently constipated, and had to be regulated by aperients. A few small pink papules were observed on the back and chest, perhaps a dozen in all; but as these occurred when the ship was in the Indian Ocean, they

were put down to the heat and sweating. No spots occurred on the limbs.

In view of the duration of the pyrexia, slight cough, and irregularity of the bowels, the case was diagnosed as probably one of paratyphoid fever, and on March 18th, on arrival at

Fremantle, 10 c.cm. of blood were sent ashore to the quarantine authorities with a request for a Widal test. The result of the test was a negative Widal but a positive Weil-Felix.

COMMENTARY

The following points arise in connexion with this case.

1. It was found impossible to trace the source of infection. This man had not been ashore at any port since sailing from England; and, if we accept a twelve days' incubation period, this would put the date of exposure to infection a day or two prior to the sailing of the ship from London. No lice were ever found in his bunk, in the hospital bed, or on his body; he denies the possibility of any lice while at his home, and of having slept away from home where lice might be present. No rats have ever been seen in the ship since she was launched, and he says that there have never been any in his home. The medical officer of health for Gravesend was communicated with, and he stated that no similar cases had been reported in the borough "for very many years," and he kindly inquired of the port sanitary medical officer, who stated that no cases of typhus or contacts had come into the port.

2. The difficulty of diagnosis was due to the mildness of the symptoms—namely, the total absence of any distinctive rash, no suffusion of the face or conjunctiva, etc.—and to the fact that there had been no cases of Brill's disease or typhus on board. This would have caused the diagnosis of this case to be missed had not a routine Weil-Felix test been carried out. The infection was apparently contracted in England, where the disease in such a mild case, occurring, as this did, during an influenza epidemic, would probably not be diagnosed, and suggests the routine use of the Weil-Felix reaction in prolonged cases of pyrexia with influenzal symptoms in which other causes are not found.

3. All authorities seem agreed that this disease is not communicated direct from case to case. Nicolle,¹ Anderson and Goldberger,² and more recently the report of the Typhus Research Commission of the League of the Red Cross Societies to Poland,³ all agree in indicating the body louse as the means of spread of infection in typhus. In Brill's disease the louse seems to play either no part or a very minor one, and cases do not occur spreading directly through families and communities. This was noted by Brill⁴ in America, by Fletcher and Lesslar⁵ in the Malay States, and by Hone⁶ in South Australia; the latter put forward evidence to show that the spread is in some way connected with the presence of abnormal numbers, or of abnormal activity, of rats or mice. This leads to a point of some importance to shipping companies and port health authorities. At present there is no official recognition by port health and quarantine departments of any difference between typhus and Brill's disease. Typhus is one of the five quarantinable diseases, and if a case occurs on board it leads to

the regulations for quarantinable diseases being enforced and free pratique at ports being withheld. Whatever view is held as to whether typhus and Brill's disease are varieties of the same infection or are different diseases presenting similarities, there should be no necessity for including the latter among the quarantinable diseases if the evidence regarding the method of its spread is accepted, as there is no likelihood of a virulent epidemic arising. Brill's disease might be made notifiable at ports in a similar way to measles, scarlet fever, venereal disease, etc., when the *Q* and not the *L* flag is flown on entering port.

4. There does not seem to be any authoritative statement as to how long after recovery a patient who has once suffered from typhus or Brill's disease will give a positive Weil-Felix reaction. This point might arise in a case of pyrexia in which the Weil-Felix reaction, done as a routine, is found to be positive, and in which the patient has been abroad and may have had a mild attack of Brill's disease that was undiagnosed at the time.

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Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

TREATMENT IN PERNICIOUS ANAEMIA

The striking success which in recent years has been obtained by feeding patients suffering from pernicious anaemia with liver in some of the many available forms, or more recently by some extract of the stomach mucosa, leaves us still in ignorance of the actual cause of the disease. Some cases, which at first dramatically improve, after a time relapse, and the increased anaemia and asthenia are not then benefited to the same extent—possibly not at all—by liver or stomach feeding. The question, therefore, is, Why is it so?

It appears that in giving liver we are supplying a "something" to the patient which an animal normally has in that wonderful warehouse—its liver. If we assume that the liver itself does not manufacture this "something," but rather *stores* it, this possibly explains why we get better results clinically by varying our methods of administering liver and by altering the brand of the preparation of liver extract; because on this assumption we can recognize that the animal's storehouse (liver) may be poorly stocked with the "something"—even in a normal, healthy animal—at the time of killing to procure the extract. This something is possibly an amino-acid.

Now the gastric mucosa has been found to be of value in replacing liver; this "something" can therefore be probably found in the stomach and stored in the liver. In the American medical press it has been recorded that beef muscle, incubated with normal healthy human gastric juice, has been found to be a substitute for liver treatment, whereas feeding with normal gastric juice alone was found ineffective.

I venture to record the following case of feeding with beef muscle and gastric juice, which succeeded dramatically for a time, so that this procedure may receive trial by those in charge of refractory cases of pernicious anaemia.

CASE HISTORY

A male patient, aged 61, was sent to me last July by Dr. Ainsley Hawes—an advanced case of pernicious anaemia.

The patient had benefited wonderfully on liver and ventriculin, and had received a blood transfusion on eight occasions. Recently, however, he had rapidly relapsed, was extremely weak and ill, with constant vomiting, and was taking little interest in his surroundings. His liver and spleen were both enlarged—the spleen could be felt three inches below the costal margin. He begged for another transfusion. Realizing that it would be of only very temporary benefit alone, I consented on the condition that this temporary improvement should be utilized for an active attempt at treating the disease as well. Stating this to the relations, I had no difficulty in getting the patient's daughter-in-law to offer to provide the gastric juice daily.

Immediately after the transfusion, which picked him up rapidly, he was given glucose and 15 units of insulin; shortly after, he received 2 oz. of pounded beef muscle, which had been incubated for three hours with 2 oz. of gastric juice, aspirated two hours after the donor had taken an egg beaten up in milk. This was repeated daily, and also insulin with glucose twice a day. The improvement was dramatic; he became active in mind, the vomiting entirely ceased, and the spleen got smaller in two days, at the end of the week being hardly perceptible. After ten days he went home to continue the gastric-juice-beef diet and improved continuously for a month. Then he had a bilious attack, disliked the diet, and it was discontinued. Since then I hear he is going downhill again rapidly.

I have urged, as the donor is perfectly willing to resume, that the treatment should be started again surreptitiously, and that the meat should be varied—mutton, chicken, veal, or rabbit muscle—so that the disguise may be complete.

The value of giving glucose and insulin as a general tonic in many grave conditions to tide over a critical period is as yet hardly sufficiently appreciated.

Consideration of this case emphasizes the point of view that pernicious anaemia is a deficiency disease, the "something" being contained and made by the action of gastric juice of a healthy person acting on the beef muscle at body heat. Obviously beef muscle does not contain it. The liver then stores this "something" and supplies it, when necessary, to the blood-forming system.

In conclusion, the young lady rapidly learned to pass the tube on herself, and to aspirate her gastric juice perfectly easily with the aid of a mirror; she is quite ready to continue her daily gift for an indefinite period. I hope to add to this preliminary report at a later date.

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ASTLEY V. CLARKE.

HERPES ZOSTER AND ENCEPHALITIS

The following case is, I think, of sufficient interest to be worth publication.

On August 10th, 1931, a married woman complained of severe pain extending from above the left orbit to the top of the head; there was no pain on the right side. She also had mild conjunctivitis of the left eye. Five days later the typical rash of herpes zoster developed above the left eye, and extended through the hair to the top of the head. The conjunctiva was now very injected; there was ptosis of the left upper eyelid, and typical ophthalmic zoster.

On September 6th she complained of weakness in her left arm, and of inability to grip with the hand, or brush her hair. On examination, the left leg was also found to be weak, but there was no exaggeration of the knee-jerk, and Kernig's sign was absent. There is no keratitis of the left eye so far. She eventually developed a left-sided hemiplegia.

I noticed in the *Epitome* of September 12th (para. 197) that André-Thomas and J. B. Buvat had recorded a very similar case. They stated that among the numerous nervous manifestations in patients with herpes zoster hemiplegia was one of the most frequent, especially in ophthalmic zoster, but that the relation between the two had not always been firmly established.

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