create a condition of lung so distinctly microbic as that observed in tuberculosis. Where pleurisy has followed injury and become tuberculous, it is just possible the following has taken place: Knowing the high percentage of pleurisies which are tuberculous from the first or become so, it would appear as if a limited patch of tubercle situated under the visceral layer of the pleura had, as a result of injury to the chest wall, become activated, and the germs from their proximity had infected the pleura, or, as a result of shock to the system by the injury, the resistance of the body generally and of the lung locally had become reduced, the entrance of the tubercle bacillus into the body favoured, and the way made easier for the development and multiplication of the microbe, whose presence therein would therefore be not a direct sequence of the injury, but simply incidental to it.

Tubercle bacilli have been found in the blood of persons who have suffered or who have died from tuberculosis. It is thus we seek to explain the disseminated miliary lesions in acute tuberculosis. The presence of tubercle bacilli in the blood has been shown experimentally and the blood of affected persons when injected into guinea-pigs has induced in them tuberculosis. Kurashige, a Japanese, found tubercle bacilli not only in tuberculous patients, but taking thirty-four apparently healthy persons he found the micro-organism present in twenty. Within eight months afterwards three of these developed tuberculosis. If we admit that tubercle bacillaemia is a more frequent condition than is generally supposed the consequence of injury to the chest wall can be readily imagined.

On this point, however, we must not be carried away too much by microscopical findings, for acid-fast resisting bacilli, closely resembling the specific micro-organisms in question, may be found in other conditions than tubercle. They are occasionally found, for example, in distilled water. To remove doubt Dr. Slade, bacteriologist to the Royal Victoria Infirmary, Newcastle, and myself carried out a series of experiments. We took blood from several patients in all stages of pulmonary consumption, whose expectoration contained tubercle bacilli in large numbers; and after following out rigidly the technique recommended, in none of the films did we detect well-formed tubercle bacilli. Now and again we found what might have been bacillary debris. Nor was the injection of the blood of affected patients followed by tuberculosis in the guinea-pigs who received the blood. These circumstances indicated to us that if tubercle bacilli are present in the blood they are extremely few and therefore difficult to find; in a word, tubercle bacillaemia is not of frequent occurrence.

The relationship of traumatism and tuberculosis may be expressed thus: Tuberculosis develops in a person who, until the accident, was believed to be quite healthy; a latent tuberculosis is roused into activity by the accident; a tuberculous lesion becomes localized at the site of injury, the micro-organisms having been carried thither from a focus elsewhere in the body, or injury has simply aggravated a localized lesion which was previously present. Although there is no tuberculosis without the tubercle bacillus, the part played by the condition of the body, as exemplified in general and local resistance, cannot be ignored, and it is here that injury, by reducing resistance, comes in as a possible factor in the development of the disease.

REFERENCE.

¹ *La Tuberculose pleuro-pulmonaire traumatique*, p. 8. Paris: J. B. Baillière et fils. 1915.

ACCORDING to the *Journal of the American Medical Association*, in the new building of the Berlin Medical Society and the German Surgical Society (Virchow-Langenbeck House) which was opened on April 1st, there was already installed the library of the Berlin Medical Society. It consists, according to the latest information, of not less than 113,163 numbers, which are distributed in the following groups: 12,958 volumes of journals, 5,958 books, 6,682 dissertations and 3,226 reprints. In addition to this, there is the legacy of the Virchow library with 12,689 and the Lassar library with 1,793 volumes, and 149 bound maps with 5,000 volumes—altogether 48,501 pieces. Further, with the society are associated the libraries of some of the medical societies of Berlin with 20,757 volumes, and the library of the German Surgical Society with 44,105 volumes.

THE CARRIER OF YELLOW FEVER (*STEGOMYIA CALOPUS*).

BY

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*... et nova februm
Terris incubuit cohors.*—HORACE.

LIKE other branches of human activity disease has its romantic and its unromantic side. Nobody can regard mumps or measles as romantic. On the other hand, yellow fever calls up all the romance of slave-trading, pirates and the Spanish Main, buccaneers, maroonings and other grisly horrors, whose sole redeeming feature was a touch of romance. Lovers of pirate stories—and who are not?—will always remember the graphic description of Yellow Jack in the West Indies.

We have probably always had disease with us since the creation of the world, that act of "*impardonnable imprudence*," as Anatole France calls it, but the first description of yellow fever only dates back to 1647, when an outbreak occurred in the Barbados. Then, as now, it devastated the shipping of the port, and was soon introduced by ships into St. Christopher and later into Guadeloupe. The following year it was in Cuba and in 1655 in Jamaica, and it gradually spread throughout the whole of the West Indies until a century or more later it reached St. Thomas.

One of the peculiarities of the disease is that it frequently disappears from a given locality for long periods of time. For instance, it was absent in the Barbados after the first outbreak until 1690, and when it recurred it was at first not recognized as being the same disease which devastated the islands forty-three years before. In the eighteenth century there was another break of fifty-four years, and similar breaks can be recorded in most of the West Indian islands.

Besides the West Indies, it is at present endemic in Brazil and on the West Coast of Africa, and is common in Mexico. Whether the disease arose primarily in Africa and is part of the toll the American continent has had to pay for the slave trade, or whether it was brought to the West Coast of Africa from the other side of the Atlantic, is not certain. It apparently appeared as a regular disease in Brazil in the year 1849, and from that time onwards, with the exception of one year, has been a permanent trouble at Rio. From time to time the disease has been carried to neighbouring parts of America, especially to the Gulf, Central America, and the northern coast of South America. It has been introduced more than once into Monte Video and Buenos Aires, and has even penetrated up the La Plata as far as Asunción. Every few years it extends into the Southern States, and has even reached Philadelphia and Boston. With the exception of an outbreak in Leghorn in 1804, European epidemics have been confined to Portugal, Spain, and the Balearic Islands.

It will have been noticed that most of these outbreaks occur on the coast and then pass up the rivers. It is thus most probable that the disease is one which is brought mainly by ships. It is also obviously a disease which must be guarded against by our troops fighting near the coast in West Africa, as well as such troops as are left in the West Indies. But, above all, it must be guarded against in relation to our shipping fleet and our navy, whose chief naval engagements have hitherto been off the South American coasts.

Yellow fever is a disease which requires a winter temperature of at least 63° F., for it is a mosquito-borne disease, and the mosquito flourishes best at about this temperature. It can be introduced into a new locality by the arrival of an infected mosquito, or by the arrival of an infected human being. In the former case the disease breaks out within a few days; in the latter at least ten or twelve days elapse before new cases arise, for, as we shall see later, the organism, whatever it is, that causes the fever is not capable of passing from the mosquito until it has been in its body for ten or twelve days.

Thirty-six years ago Finlay of Havana suggested that the virus of yellow fever was inoculated by mosquitoes; but it was not until the publication of the discoveries by Sir Ronald Ross and others that malaria is transferred by *Anopheles* that a thorough investigation

was made in yellow fever. In the last year of last century an American Commission, consisting of Drs. Walter Reed, Carroll, Agramonte and Lazear, investigated the whole subject, and taking extraordinary risks were able to prove that the infection was not conveyed by contact or through the air, or from bedding or clothes soiled by the dejecta of yellow fever patients, but by a mosquito of the genus *Stegomyia*. Whatever the virus is, it is invisible even under the highest powers of the microscope. It can be filtered through a Berkefeld filter. It is destroyed by

Fig. 1.—*Stegomyia fasciata*. Fly, lateral view, magnified. Note humpbacked outline, and also the position of the posterior pair of legs.

heating to 55° C. If the blood of a yellow fever patient during the first three days be inoculated into a healthy man he gets yellow fever, and it is only during the first three days that the blood is infective. On the other hand, the mosquito is incapable of transferring the disease until the unknown organism has been in its own body for at least ten or twelve days.

The mosquito in question belongs to the species *Stegomyia calopus* (Blanchard), or, as it is more often called in English textbooks, *Stegomyia fasciata* (Fabricius). The genus *Stegomyia* differs from other *Culicidae* in having a dark grey or black colour, whilst the *Culicidae* are as a rule brown. They also have silver-white spots and silver glistening scales, especially on the back of the legs and the abdomen. The grown-up mosquito is comparatively small, and very elegant. Its length is some 3 to 4 mm., but if the mouth parts be added is some 6 to 6½ mm. long. As is usual, the male is smaller and feebler than the female. When settled, as, for instance, whilst sucking the blood of its host, it rests upon its first four legs only, the two hindmost being stretched out abaft like pennants waving in the air, but in general it has the humpbacked appearance of the *Culex* and not the straight outline of *Anopheles*. The colour is greyish-black modified by numerous white spots and rings. There is a white rim round the eyes, and a very characteristic lyre-like pattern on the dorsal surface of the thorax. The structure of the mouth parts is much the same as that of any other

Fig. 2.—*Stegomyia fasciata*: above, the larvae; below, the eggs. Both natural size.

Fig. 3.—Egg of *Stegomyia fasciata*, highly magnified. Notice the air "cells."

Culicidae. The antennae have fourteen joints, the last two of which in the male are longer than the others. As is again usual, the antennae of the male have long brush-like hairs, organs by means of which they scent the female. The legs are banded alternately white and black rings. It is this character, indeed, which has given the mosquito the name of the "tiger-gnat." The rings are very iridescent.

The pupa of *Stegomyia* is darker and blacker than that of *Culex*, and seen from the side the head and the thorax are somewhat more triangular than the same parts in *Culex*. As the pupa grows older it grows darker. The length of the larva is 4 to 6 mm., somewhat larger than that of the gnat. But like that it has a respiratory tube

stretching out from the last segment of the abdomen, almost at right angles to the rest of the body. This respiratory tube is much shorter than that of the *Culex*, but is long enough to enable the larva to hang obliquely down into the water. The eggs are very big. They are covered by a mass of small "cells" containing air, and they never tend to form a conglomerate like those of *Culex*, but are laid singly and remain isolated until the larvae hatch. After floating a certain time they usually sink to the bottom of the water. Their length may be about a millimetre and their colour is almost black. When the egg hatches the anterior third of the shell splits off and the larva at once emerges.

As is so often the case with mosquitos, it is the female alone which bites. The larva nourishes itself on plant juices, saps, and so on, especially they like sugary secretions, and in the absence of blood the female is reduced to a similar diet. Hence the *Stegomyia* is comparatively common in dwellings where sweetstuffs are—bakeries, sugar refineries, and so on.

These mosquitos are, like the cockroach, the fly, and the bed-bug, inhabitants of human dwellings. They are indeed domesticated, and are always to be found in the neighbourhood of human houses or buildings or ships, and are very rarely indeed found far away from the sphere of man's activities.

They are very apt to bite one in the neck, creeping along the darker parts of the clothing until an unprotected region of the body is reached. Unless one has very thick socks they frequently bite the ankle, and they are as tireless in their pesterings as ever Mrs. Pardiggle was—no sooner are they driven away than they return to the attack. The bite is painful, and in many people raises a considerable swelling.

The *Stegomyia* bite not only during the night but also during the day. According to R. O. and O. Neumann, in Brazil, at any rate, they are capable of biting not only during the twilight, but at any time. The bite lasts twenty to thirty seconds, after which the mosquito rests a bit, waving its third pair of legs in the sun. After a considerable rest she flies away to some sheltered spot, and whilst blood is being digested the mosquito takes nothing but water—a very proper dietetic measure. After three or four days the female is ready for another meal.

In the absence of man these mosquitos will suck blood from other animals, and in confinement they are generally fed on rats or canaries, and they will even suck up a drop of blood presented on a piece of cotton-wool.

If the female mosquito has been fertilized before the sucking of blood she will commence egg-laying two or three days later, and two or three days later again the larva will emerge. The larval stage lasts from nine to twelve days, and the pupa stage three to four, so that the whole metamorphosis takes from twenty to twenty-four days. Hence during warm weather many generations succeed each other, but one must have a temperature of at least 20° to 27° C. Below that temperature the processes tend to slow down, and under a temperature near freezing point the regular development is definitely interrupted. But the interruption is only a suspense, and living activities are resumed should the temperature rise again.

It is a disputed point whether mosquitos must have a meal of blood before they can lay eggs, and on this point the evidence is not yet sufficient to make a dogmatic statement. Most mosquitos are very indifferent where their eggs are laid. The smallest collection of water in an empty sardine-tin, a broken tumbler, a puddle in the street, a gutter pipe, is good enough for *Stegomyia calopus*. She will even lay her eggs on moist cotton-wool.

Although *Stegomyia* bites freely during the day-time, it as a rule avoids the light and seeks some dark shelter. Contrary to the habits of *Anopheles*, it prefers a light ground to rest upon, and exposed to light in a dark vessel it will seek the light furthest from it. The larvae live on algae or vegetable matter, or plant detritus, or in

Fig. 4.—Larva of *Stegomyia fasciata* breathing on the surface of the water. Highly magnified.

captivity on white bread or Indian corn. They can remain for a considerable time without food, and this without materially diminishing the rate of their development. *Stegomyia* breed well in ships, and are occasionally found in one part only of the ship, such as the engine-room or cook's galley, where the conditions seem to be most favourable to their development. Thus it comes about that at times certain quarters of a ship provide the greatest percentage of yellow fever cases.

A CASE OF BANTI'S DISEASE WITH ENLARGED HAEMOLYMPH GLANDS.

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CONSIDERING the paucity of the literature on haemolymph glands we have thought it worth while to record the clinical and pathological details of the case here described.

S. C., aged 57, had felt fit and able to perform his ordinary work as an agricultural labourer up to November 6th, 1914. On the following day he was attacked for the first time with aching and swelling of the feet and legs. The swelling extended above the knees, and he had to keep in bed on account of the aching, which he described as fearful. He was very positive as to the abrupt commencement of his illness. His sister stated, however, that he had been getting thinner during the past year, and that twelve months previously he had fallen off a ladder on to his stomach. On account of this he was a fortnight in bed, but did not see a doctor. He was a habitual beer drinker, taking at times up to ten pints in the day. The only previous illness he could remember was an attack of influenza in 1884.

Condition on Admission.

The patient is a small man of spare build and, from the looseness of his skin, has evidently lost flesh recently. His present weight is 8 st. 10 lb. His normal weight, according to himself, was 10 st. The skin of the face is slightly bronzed, there is a well marked malar flush, and the venules of the face are dilated. There is no general pigmentation of the skin, but the forearms and the inner sides of the legs below the knees show some brownish discoloration. There is no conjunctival icterus and the skin is not jaundiced. There is slight oedema of both legs. The muscles of the limbs and trunk are wasted to such a degree that it is surprising how he could have so recently been able to perform hard physical work. The appetite is very poor, the tongue is furred, and the bowels are constipated. The teeth are few in number and are free from pyorrhoëa. There has been no vomiting. The abdomen is tense, but moves well with respiration. The left side bulges forward. The veins on both sides of the abdomen and thorax are distended, and the skin of the abdomen has a stretched appearance.

A large mass can be felt occupying nearly the whole of the left side of the abdomen. It extends from the left costal margin downwards, forwards and to the right. Just to the left of the umbilicus it makes an angle forwards, reaching to a point 1 in. to the right of the middle line and then passes down to Poupart's ligament. The tumour feels smooth and hard, it moves well with respiration, and is quite dull on percussion. No friction rub is audible over it. There is no tenderness of the abdomen except at the notch felt in the region of the umbilicus. This point is acutely tender. Ascites is indicated by the presence in the right flank of dullness, which shifts with change of position. The liver dullness extends from the third rib to 1 in. below the costal margin.

No irregularities can be felt on the surface of the liver. The heart appears normal and the lungs yield the ordinary signs of emphysema. There is nothing else to note in the thorax. The condition of the blood on November 20th was: Red blood corpuscles 2,740,000, haemoglobin 60 per cent., leucocytes 6,000. There was some poikilocytosis, but no nucleated red corpuscles nor marrow cells were found. Differential leucocyte count showed a slight relative lymphocytosis.

The Wassermann reaction was negative, and there was no reaction to von Pirquet's skin test for tuberculosis.

After-History.

The patient steadily lost weight and became weaker. He was always very cheery, and his only complaint was of a constant thirst. On December 7th, when he got up to be weighed, he knocked the back of his wrist lightly against a button; but the injury was so trivial that he did not allude to it at the time. On the 9th there was a tender red swelling over the right wrist, oozing pus. Cellulitis spread up the arm; the patient rapidly sank, became comatose, and died on December 12th. The condition of his blood the day before his death was: Red blood corpuscles 2,368,000, haemoglobin 55 per cent., leucocytes 4,400. It is of interest that the leucopenia increased in spite of the acute infection of the arm. No bile appeared in the urine at any time.

Necropsy.

A post-mortem examination was made twenty-two hours after death. The body was wasted. There was very little subcutaneous fat present, and the muscles were very small. The right arm was swollen from the distal ends of the metacarpals to half-way up the upper arm. The back of the arm was purple and had blebs on it. The legs below the knees were oedematous. The abdomen was not distended. The peritoneal cavity contained 33 oz. of blood-stained fluid. The transverse mesocolon was stained a purple red from extravasated blood. The spleen was seen occupying the left half of the abdomen. There were only some slight adhesions between it and the left abdominal wall. Its lower pole extended below the anterior superior spine of the ilium. The great omentum was very thin, containing very little fat, and was wrapped round the anterior surface of the spleen enveloping and adherent to its lower pole, forming a very definite hammock of support to the huge viscus. The spleen weighed 4 lb. 14½ oz. It measured 15 in. from pole to pole, 5 in. broad and 14½ in. in circumference at the notch. One vein on the posterior surface of the spleen was thrombosed. The capsule of the spleen was ½ in. in thickness and in places almost as hard as cartilage. When cut the spleen pulp was quite firm and did not squeeze out on pressure. It was the usual deep red colour and the Malpighian corpuscles did not stand out clearly. The liver was smooth and weighed 4 lb. 12½ oz. On its anterior surface there was a slightly depressed purple colour patch which proved later on microscopical examination to be an angioma.

The gall bladder was filled with yellow bile and showed no sign of disease. There was no sign of thrombosis in the portal vein nor in its tributaries, with the exception of the one vein on the dorsum of the spleen already mentioned. Macroscopically the portal vein and its tributaries appeared quite healthy. The right kidney weighed 4½ oz.; its capsule was thin and stripped easily. The left kidney weighed 3½ oz. Its cortex was atrophied, and the capsule was also thin and stripped easily. The two suprarenals appeared normal. The stomach and intestines exhibited no abnormality. The pancreas appeared healthy. There was a chain of enlarged lymphatic glands running in front of the lumbar vertebrae on either side of the vena cava. These glands varied in size up to 1½ in. in length and ½ in. across; they had the shape and appearance of lymphatic glands, but for the most part resembled the spleen in colour and firmness when cut across. Some of the glands were a deeper red than others; a few, including one of the largest, were a cream colour; and some showed when cut across, deep red streaks and mottling on a cream ground. Haemolymph glands were also present along the greater curvature of the stomach and near the hilum of the spleen. The pleural cavity was free from adhesions. The lungs were emphysematous, but otherwise showed no disease, and there was no evidence of old or recent tubercle. The left lung weighed 17 oz., the right 20 oz. The pericardial sac contained 1½ oz. of straw-coloured fluid. The heart weighed 8 oz., and was flabby and thin-walled. All the cardiac valves were normal. The aorta had small patches of atheroma above the aortic valve and in the descending aorta.

Microscopic Examination.

The capsule of the spleen is very thick and dense. Large bands of fibrous tissue pass from the capsule into the substance of the spleen, and there is also a great deal of intercellular fibrous tissue. This fibrosis is more advanced in the pulp than in the Malpighian bodies, and involves the periphery of the latter more than the centre. The large bands of fibrous tissue have the appearance of normal organized fibrous tissue and stain red with v. Gieson's stain, but a large part of the intercellular tissue has a peculiar homogeneous glassy appearance, and stains yellow with v. Gieson, suggesting the possibility of effete erythrocytes being contributory to its origin. The endothelial cells and splenic cells are increased in number, and the lymphoid tissue relatively diminished, but the fibrosis is the chief feature of the splenic tissue. There is no excess of eosinophiles, and only a few cells show phagocytosis of red corpuscles.

Haemolymph Glands.

The capsule is composed of the usual fibro-muscular tissue present in normal lymph glands with septa of the same material passing into the substance of the gland. The differentiation between lymph channels and lymph nodes is not distinct, as in normal lymph glands, and what corresponds to the lymph paths are filled with blood corpuscles. There is a great hyperplasia of the endothelial cells, and there is present in small numbers a large cell with a very large single nucleus, almost filling the cell, probably a myelocyte. Numerous cells show phagocytosis of erythrocytes, contrasting in this respect with what obtains in the spleen. There is no excess of eosinophiles. The fibrous tissue is increased, but not to such a degree as in the spleen; there is also hyperplasia of the lymphoid tissue.

It has already been noted that nearly all these enlarged glands, when cut across, were red in colour, like the spleen; a few, including the largest, were a dirty cream colour, and some, on cross section, showed a whitish-cream ground, with red streaks and mottling. Microscopically, the difference between these types lay in the number of red corpuscles present in the lymph paths. This is a very important point. In all there was hyperplasia of the endothelial cells, lymphoid, and fibrous tissues.

Sections of the spleen and lymphatic glands were