with a cold and cough for a fortnight. When admitted he was evidently very ill, and his face was pinched. Respiration was hurried, and evidently gave him pain, as did a slight hacking cough. Temperature 100° F., pulse 108, respirations 44. The abdomen did not move well on respiration, and respiration was mainly thoracic in type and shallow. The upper abdomen was resistant and very tender to pressure, but no definite spot of tenderness was localized. No lump could be felt, and he was not tender per rectum. The abdomen was tympanitic all over, except slight dullness over the stomach, and there was very slight reduction of liver dullness, almost imperceptible. On the right side of the chest, about the fourth and fifth ribs, there was marked bronchial breathing with crepitations and dullness. A diagnosis of perforated gastric ulcer was made and the abdomen opened. A perforation was found in the stomach at its junction with the oesophagus; this was closed by an omental flap.

The interesting points about this case were that he had definite lung signs and that the abdominal signs were not very marked; the diagnosis of perforated gastric ulcer was made owing to the history of indigestion, sudden pain, resistant abdomen, and reduction of liver dullness. The lung signs were due to an old-standing phthisical cavity which had recently become active.

CASE III.

A boy, J. M., aged 18, was admitted to hospital with a diagnosis of appendix abscess, and operated on after this diagnosis had been confirmed. The abscess was opened and a considerable quantity of pus was evacuated and free drainage introduced. Some days after operation the patient had a sudden pain in the right side of the chest. This was attributed to burrowing of the abscess into the pleural cavity, but no definite signs could be elicited except a few crepitations. Temperature, pulse, and respirations rose, and his face became flushed. Commencing pneumonia was diagnosed. The attack seemed to abort, but the patient died, and a *post-mortem* examination was made. Pus was found between the liver and diaphragm, but not in the pleural cavity. In the lower lobe of the right lung were three recent infarcts.

This case was particularly interesting in its after course, and it was very puzzling to localize correctly the signs.

I am indebted to the surgeons of Bury Infirmary for permission to publish these cases.

THE MECHANISM OF TRANSMISSION OF TRYPANOSOMA LEWISI FROM RAT TO RAT BY THE RAT FLEA.

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ANY study of the common rat trypanosome, *T. lewisi*, is important in that it is akin to the trypanosome of sleeping sickness. It was demonstrated by Nuttall (1908) and others that this trypanosome is carried from rat to rat by fleas. Nuttall transferred fleas (*Ceratophyllus fasciatus*) from infected to healthy rats, and found that the latter generally became infected.

Minchin and Thomson (1910) proved that the rat flea does not immediately carry the trypanosome from the infected to the healthy animal unless about a fortnight has elapsed from the time the flea first fed on infected blood—that is to say, the trypanosome must live or develop in the flea for about a fortnight before the flea which carries it can infect a healthy rat.

It has been the general idea that the mechanism of this infection is by the flea biting the healthy animal, and inoculating trypanosomes through its mouth-parts. My object was to discover, if possible, if this were so; or in what other manner the fleas infected the rats.

The following procedure was adopted. The infective fleas were always used in batches of ten for each rat. They were first starved for about three days, so as to ensure their feeding on the rats on which they were to be placed. They were then put on the rats and taken off in about half an hour, by which time they had fed. It was intended by this method to see if the fleas infected the rats either by biting them or, possibly, by contaminating them. In the next place, either these or other fleas were squeezed between fine forceps and then put in a bolus

of bread-sop which was given to hungry rats to eat. In this way it was intended to imitate the rats' natural habit of chewing up and eating the fleas which infest them, and to ascertain if infection was caused thereby.

Result of the Experiment.

A. Fleas placed on rats for half an hour, and then picked off. Of 9 rats none became infected.

B. Crushed fleas fed to rats with bread. Of 9 rats 6 became infected.

C. Fleas placed on rats and allowed to stay thereon, as in Nature. Of 7 rats 2 became infected.

It will be seen (A) that none of those rats on which fleas had been left for a limited time became infected, whereas (B) six out of nine became infected after they had fed on the infective fleas. We can thus say, contrary to previous preconceived ideas, that rats become infected with *T. lewisi* by the process of eating the infective fleas, and not by being bitten, or contaminated by them in a short time.

The result of putting fleas on rats and leaving them as in Nature (C) was that two out of seven became infected, which shows, when compared with the results under (B) that in Nature many infective fleas do not cause infection, probably because the rat only catches a limited number of them.

It has usually been assumed that the trypanosomes, after undergoing a "development" in the flea, are inoculated into the blood of the rat by the mouth-parts of the flea. We see, however, that they must pass through the wall of the alimentary tract of the rat, and thus reach the blood. The forms which do this are probably the "small trypanosomes" described by Swellengrebel and Strickland (1910).

It is interesting to find blood parasites behaving thus, and it is possible that other trypanosomiasis are transmitted by more devious means than by the simple bite of blood-sucking insects. It may be found that non-blood-sucking creatures are the intermediate hosts in some cases.

SUMMARY AND CONCLUSIONS.

1. Infection of rats with *T. lewisi* is caused by the rats' act of eating infective fleas.
 2. It is not caused by contamination of the rats during a short time, nor by their being bitten by the fleas.
 3. Many more fleas are infective than cause infection in Nature.
 4. The infective form of the trypanosome, which is probably the "small trypanosome" of Swellengrebel and Strickland, must reach the blood through the gut wall.
 5. It is possible that other trypanosomiasis may be transmitted by perhaps even non-blood-sucking creatures.
- I would like here to take the opportunity of thanking Professor Nuttall for his kind advice in this work.

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THE Town Council of Macclesfield has adopted recently a proposal put forward by Dr. Hedley Marsh, M.O.H., through the Health Committee, for making provision for the treatment of cases of phthisis selected from among the dwellers in the more crowded and poorer parts of the town. The treatment in view is primarily educational but also curative. From six to eight patients are to be kept in the at present disused small-pox hospital for periods of about six weeks each; it is believed that during this time it will be possible materially to improve their condition as well as educate them in the care of their own health, imbue them with a sense of the value of fresh air, and drill them thoroughly in methods of preventing the spread of infection to others. It is considered that in this way it will be possible to pass through the hospital about 50 cases a year at an annual cost of not more than £337. The food allowance included in the estimate is on a liberal scale as compared with the outlay on the same item at the fever hospital of the town. At the latter the cost is 8½d. a day per head, while 1s. a day is allowed for each phthisis patient.