Clinical and other Notes.

A CASE OF CYSTICERCOSIS (T. SOLIUM).

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It is unusual to encounter an instance of cysticercosis in which intestinal infestation, somatic invasion and post-mortem appearances are observed within the course of a few months. The following is a summary of the notes of such a case:

Private H., 21 years old, with two years and four months' service in India, was admitted to hospital on March 10, 1934, suffering from slight fever and generalized body pains of three days' duration.

The day after admission his temperature rose to 103°F. and it remained raised for nineteen days from the onset of the illness. Then there was an afebrile period of six days which was followed by a second bout of moderate pyrexia lasting for eight days. The temperature was mostly irregular, but the chart had a certain resemblance to that of enteric.

On and about the tenth day severe occipital headache occurred; its severity and late onset raised a suspicion of intracranial complication, but neck stiffness and Kernig's sign were absent, the optic discs were normal, and the headache soon subsided. At the same time his general condition began to improve.

On the eleventh day one segment of tapeworm was found in the faeces; it was thought to belong to T. saginata, but definite identification was uncertain.

The blood picture was noteworthy: the total white cell count remained between 14,000 and 17,000 per cubic millimetre. The differential count was as follows:——
### Clinical and other Notes

<table>
<thead>
<tr>
<th>Day of disease:</th>
<th>6th</th>
<th>7th</th>
<th>11th</th>
<th>18th</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polynuclears (per cent)</td>
<td>82</td>
<td>60</td>
<td>66</td>
<td>63</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>14</td>
<td>27</td>
<td>30</td>
<td>36</td>
</tr>
<tr>
<td>Mononuclears</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Eosinophiles</td>
<td>3</td>
<td>7</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>Türk cells (number seen)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
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</table>

The clinical state of the patient was still so suggestive of enteric that, in spite of the finding of the tapeworm segment and the blood picture, it was thought advisable to postpone an attempt to expel the parasite until the former disease had been excluded.

On the twentieth day, that on which the primary attack of fever ceased, one tapeworm ovum was found in the faeces; again identification of the species was uncertain.

Throughout the illness, including the period of febrile relapse from the twenty-sixth to the thirty-fourth days, the pulse-rate was slow and there were no physical signs to indicate any definite morbid condition. The usual investigations for enteric and melitensis infections were entirely negative.

On the thirty-fifth day 
filix mas
was administered and a complete tapeworm was passed. The head of the parasite was recovered and identified as that of T. solium.

Special clinical and radiological search for cysticerci was made but none was found.

The patient gradually regained health and was discharged on May 15, after seventy days in hospital, to a month’s light duty in the hills.

On June 2 he was admitted to hospital again complaining of weakness, headache and vomiting. At first the headache was confined to the right frontal region, later it became general and pain extended down the neck, chiefly on the right side. There was intermittent stiffness of the muscles of the neck. Other than this the central nervous system showed nothing abnormal. No ova or cysts were detected in the faeces, the blood picture was normal and the Wassermann reaction was negative.

There was some improvement and vomiting ceased under symptomatic treatment, but by June 28 vision in the right eye was rapidly deteriorating—it was down to hand movements at six inches—and papilloedema was developing. Lumbar puncture produced clear fluid under normal pressure. (Unfortunately further examination of the fluid was precluded by an accident to the messenger taking it to the laboratory.)

On August 6 the papilloedema had reached five dioptres in each eye. Headache again became very severe and vomiting recurred. Cerebration was slow and the mental state apathetic. X-ray examination of the skull showed lack of definition of the posterior clinoid process. There was no sign by which the cause of the raised intracranial pressure could be located.

On August 15 no improvement had resulted from treatment with potassium iodide and mercury by the mouth, and 25 per cent solution of magnesium sulphate by the rectum. In consultation with Captain
S. Smyth, I.M.S., as ophthalmologist, it was considered that, while no prospect of cure could be offered, the only hope of ameliorating the patient's intense suffering, and perhaps obtaining some improvement in vision, was symptomatic cerebral decompression.

On August 16 right sub-temporal decompression was carried out by Lieutenant-Colonel W. L. E. Fretz, R.A.M.C. The brain was found to be under considerable pressure; such inspection and palpation as were possible revealed no cause. The operation was immediately successful in relieving the headache and improving the man's mental condition so that he was able to talk intelligently—a relief which lasted till shortly before his death. There was no improvement in vision.

On August 20 paresis of the left arm and face began to develop and the left plantar reflex became "extensor." By August 25 the paresis involved the left leg and the brain began to bulge at the opening in the skull. On September 4 left hemiplegia and left hemianesthesia were complete. A slough had appeared in the tissues covering the hernia of the brain. Cerebration was slow and the patient spent most of the time sleeping. On September 20 the temperature rose abruptly to 103°F. and unconsciousness supervened.

Death occurred on September 23.

Post-mortem examination by Major A. Mearns, R.A.M.C.—There was a hernia cerebri in which infection had occurred and produced an abscess with localized meningitis. The brain contained about 150 small cysts scattered throughout the grey and white matter of the hemispheres as far down as the cerebral peduncles; one was found in the cerebellum. There was none further down the central nervous system nor in the spinal cord. Two were embedded in the meninges close to the optic chiasma. None was calcified. All the other organs of the body were healthy and contained no cysticerci. In no part of the skin or muscle was any swelling detected; pieces of muscle were excised and examined but no cyst was found.

NOTES.

(1) The period of highest fever (from the eighth to the twelfth day) during the pyrexial attack for which the man was admitted in the first instance was characterized by intense headache and by eosinophilia; the latter was present at no other time while the patient was under observation. Was this the occasion of invasion of the central nervous system?

(2) Somatic infestation was confined entirely to the brain.

(3) Although cysticerci were present in the cerebellum and in almost every part of the cerebral hemispheres there were no focal signs.

(4) The wisdom of operating upon the patient was open to question. The chief points considered were:

Pro: Steadily increasing intracranial pressure of high degree which was unrelieved by simple treatment and which appeared likely to cause death within a few more weeks if still unrelieved;
almost total blindness due to papilloedema; continuous suffering from vomiting and intense headache; danger of sudden death from repeating lumbar puncture to relieve headache.

Con: The great probability that the cause of raised intracranial pressure was cerebral cysticercosis, although there was no definite positive evidence, and hence the likelihood that no permanent benefit would accrue; the risk of the operation and perhaps subsequent cerebral hernia; the alternative of relieving pain by full doses of morphia until the end came.

The last alternative seemed to offer no advantages over decompression and the pros had it. In the result the patient's suffering was relieved and he lived at least as long as he appeared likely to do under "medical" treatment.

I am indebted to Headquarters, Rawalpindi District, for permission to forward these notes for publication.

A SIMPLE FLY TRAP.

BY LIEUTENANT-COLONEL J. F. JAMES,

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In dealing with flies, apart from preventive measures in limiting breeding grounds, the sugar or gur arsenic poison is a most effective one, especially in dry weather.

The wooden trap with rollers usually employed to expose the poison has been found to be somewhat unsatisfactory, as, apart from its cost, it is liable to leak and is easily broken.

After considerable experiment the following pattern has been devised.

An empty ghi tin is placed upright and cut across the top medially. A similar cut is made parallel across the front of the tin about three inches from the bottom and the cut extended up each side to meet the top cut, the part removed being shaped like a banker's shovel (fig. 1). A modified pattern is made by cutting perpendicularly through the top to within three inches of the base, and then horizontally through half the tin (fig. 2). The reservoir for the poison solution is the lower three inches of the tin and about one inch depth of solution is ample. A stout wire is passed across the top of the tin about two inches from the back and looped above to enable the tin to be hung on a wall or other support.

The portion of wire across the inside of the tin serves as a support for gunny or other material. The gunny should extend to the bottom of and also cover the floor of the tin. The upper part of the gunny can be wetted with the poison solution by laying the tin flat. The lower part is of necessity always wet.

These ghi tin fly traps can be turned out very quickly at a cost which