be said to compare quite favourably in this respect with other branches of medicine, such as cardiology. I hope, therefore, that this subject may be of interest, particularly in view of the therapeutic possibilities, of modern neurosurgery.

I wish to thank the Staff of the West End Hospital for Nervous Diseases for permission to refer to case notes, and in particular Dr. Worster-Drought, and Mr. G. C. Knight. I would also emphasise that the diagnosis and treatment of such cases is essentially a matter of team work on the part of the Hospital Staff.

CYSTICERCOSIS

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Although Leuckart (1856) first elucidated the life cycle of *tænia solium*, it was not until 1933 that the pioneer work of W. P. MacArthur drew attention to the importance and prevalence of cysticercosis as a clinical entity in this country: with the collaboration of H. B. F. Dixon, D. W. Smithers and W. K. Morrison working at the Queen Alexandra Military Hospital, Millbank, he worked out the clinical features, pathological changes, and radiographic appearances of cysticercosis. He drew attention to the prevalence of the condition amongst soldiers who had been stationed abroad—particularly in India. As tænia solium infection of pigs and man is common on the continent of Europe, the Middle East, India, and the Far East, it is certain that a large number of our fighting personnel will return after the war with the unpleasant manifestations of this infection. The condition of cysticercosis must therefore always be kept in mind when dealing with a patient who has resided abroad, and who has developed such manifestations as fits, symptoms suggesting some degenerative process of the nervous system or a psychic disorder.

A picture of the pathological processes and the protean manifestations of the condition of cysticercosis may be visualised by the consideration of the following typical case.

W.C., æt 44, commenced his adult life in the Regular Army, serving on the North-West Frontier of India from 1918 to 1924. During that period he had several attacks of what was considered to be malaria—bouts of fever, with intense headaches and severe pains in his muscles. After returning to this country he suffered from repeated epileptiform fits from 1927 to 1933. Early in 1942 he was referred to hospital with a diagnosis of left renal colic, as he had suffered for a period of four months from sudden attacks of violent and commanding pain which commenced in the left lumbar region and radiated round the abdomen to the area of the umbilicus. The duration of each attack varied from a few minutes to several hours. With the increasing frequency and severity of the attacks he had noticed that pain of a similar character and distribution was being radiated to the right side to encircle the trunk, suggestive rather of the girdle pains of tabes. After each attack he would feel exhausted and pass into a deep sleep for several hours. He denied any possibility of a syphilitic infection in early adult life, the reflexes in his lower limbs were found to be intact, his pupillary reflexes were normal, and there was no disorder of sensation. The urine was free of red cells, but in order to exclude the possibility of a renal calculus an X-ray examination of the renal tract was carried out. The film showed, dotted about the abdominal area, numerous small well-defined elongated opacities having the characteristic features of calcified cysticerci. Further films of the entire body demonstrated the presence of numerous calcified cysticerci scattered through the musculature of the extremities; one was demonstrated in the skin, but there were none visible in the brain or spinal cord.

Incidence in Great Britain.

As the result of a wise provision of Parliament in the year 1582, which made the sale of "mesell pork" a crime in this country, the tænia solium virtually does not exist in England and Wales, or in Scotland. In parts of Ireland where the social circumstances of the people are more primitive it is not uncommon. In Dixon and Smithers' review of seventy-one cases of cysticercosis (which included those reported in the literature) only one appeared to have been acquired in this country. Tænia saginata, which is seen so commonly, produces eggs which are morphologically identical with those of the pig worm, but does not utilise man as the intermediate host for the cysticercus stage.

Mode of infection.

This patient became infected in one of two ways. He may have been the host of a taenia solium as the result of eating measly pork which had been insufficiently cooked. He could then

develop an auto-infection from eggs produced in his own intestine, either by direct invasion of the mucosal lining of his bowel, or by infection of his upper alimentary tract by his hands which had been infected from his own excreta. Alternatively, he may never have been the host of a tapeworm, but become infected with eggs from eating fruit or green vegetables which had been manured or contaminated by infected human excreta. In the East, it is a common sight to see "coolie" gardeners sprinkling the family excreta on their market garden. A point to be remembered when salad is on the menu in an Eastern restaurant!

Development of adult tænia solium.

In the life cycle of the tænia solium the larvæ or *cysticercus cellulosæ* stage occurs commonly in the pig, less commonly in man, and very occasionally in the dog: the worm exists only in the human intestine. Following the ingestion of infected and insufficiently cooked pork the cysticercus is hatched in the small intestine following the tryptic digestion of the cyst wall. The head or scolex rapidly buries itself in the mucous membrane of the jejunum after fixing itself on to the surface with its double row of hooklets, of which there are about twenty-eight in the rostellum. The larva further relies on its four cupped suctional pockets for attachment to the *mucosal* surface. As the worm grows the terminal proglottides, which are composed of a hermaphrodite sexual unit, become gravid and ultimately break off, each discharging about 10,000 eggs. These eggs are spherical in shape, and measure about 35 μ in diameter. Each egg is composed of an embryo with three pairs of hooklets on its surface and a surrounding thin protective capsule.

Migration of larvæ.

The capsule of the embryo is absorbed in the small intestine of the host of the worm or an intermediate host, and the embryo clings to the mucous membrane by means of its hooklets. It then penetrates the mucosa and enters a vein or lymph channel. The method of dissemination is probably through the general systemic circulation, although it is difficult to explain the fact that cysticerci are seldom found in the lungs, kidney, spleen, thyroid and bone marrow, through which such a large percentage of the blood flows. The embryos appear to prefer certain tissues —the majority make their way into the brain, where they prefer grey matter to white. Others settle in the muscular system, and may even be found in the heart muscle. The subcutaneous tissue and retina appear to be other favourite haunts. It would seem that the brain is their favourite resting-place: in one autopsy between 2–300 cysticerci were found in the brain, but none could be detected in any other tissue.

Formation of the cysticercus.

When the larva has come to its final point of rest, it forms a capsule about itself which is known as the cysticercus. This process of encapsulation which separates the embryo from the host takes about three months to complete.

The brain reacts to the embryo rather differently than other tissues of the body. The capsule becomes surrounded by a zone of round cells—mainly plasma and endothoid cells—and outside this is a layer of sclerosis or gliosis. In this state, the host, if not overwhelmed by excessive numbers of larvæ, or if a vital area has not been infiltrated, lives with remarkably little discomfort in a state of symbiosis.

Mortification of the cysticercus.

Eventually after the passage of time varying from 2–10 years the embryo dies. As the result of the mortification process in the embryo a toxic substance is produced and the capsule becomes distended with necrotic fluid. In the brain the toxic substance permeates the capsule and invades the surrounding tissue causing necrosis, intense irritation and tissue reaction.

It is this process which gives rise to the late cerebral manifestations of cysticercosis. In the subcutaneous tissues and muscles the cysticerci which have been soft and not palpable during the life of the embryo, become distended, and may be either seen or felt. After a variable period, usually extending over a few months, the inflammatory reaction subsides, the cyst shrinks and becomes calcified. The process of calcification seldom occurs in the brain. In the rare instances in which it does occur only the scolex becomes calcified; this may be recognised as a minute pinhead shadow in the radiograph.

CLINICAL MANIFESTATIONS OF CYSTICERCOSIS

Premonitory symptoms.

There may be premonitory symptoms during the period of invasion by the parasites—these are characterised by the occurrence of headache, unidentified fever, and myalgia. Sometimes the invading parasites produce localised swellings and oedema in the muscles. This patient complained of having had "touches of malaria"—a condition often misdiagnosed during the invasion period.

If the invasion process takes place very slowly there may be no premonitory symptoms whatever.

Should there be a rapid and massive invasion of the nervous system, the victim develops a picture which is very similar to that seen in acute encephalitis, and this may be fatal within a week of onset.

If massive invasion takes place more slowly, the patient may live for several months with irregular fever, headache, vomiting, and all the manifestations of increasing intracranial pressure, including papilloedema, motor or sensory changes, mental stupor, and ultimately coma preceding death.

When invasion has taken place slowly, as has occurred in this patient, there is an interval of a few years before the late manifestations develop, arising from the mortification process. These are:—

(1) *Palpable cysts.*

These are usually noted in the subcutaneous tissue of the scalp, face, eyelids, trunk and limbs, but are seldom seen in the hands or feet. The tongue and retinæ are also favourite sites. The cyst is usually oval in shape, measuring about 1 × 2 cms. and feels like a small bean under the skin. In this patient there was only one which could be recognised, in the right cheek.

(2) *Symptoms of cerebral involvement.*

The commonest symptom of cerebral involvement is the production of epileptic attacks. These may commence as mild attacks of *petit mal*, which may be hardly noticed by the patient.

Another possible early result of local irritation in the motor area is the occurrence of a periodic spasm of a group of muscles, such as a sudden contraction of the fingers of the hand or some other unusual act, which may be considered a manifestation of hysteria.

Ultimately a major epileptic attack will occur, either of the Jacksonian type or as a generalised convulsion. Fits may then recur at varying intervals, and the patient might succumb in status epilepticus.

In this patient fits commenced three years after leaving India and continued for a period of six years. No doubt this latter period represented the phase of mortification of successive cysticerci in his brain. His recent symptom of girdle pains was considered to be an epileptic aura as it was always followed by the typical post-epileptic period of somnolence. This theory was substantiated during his stay in hospital, when a particularly severe attack of pain was followed by a generalised convulsive seizure—the first for a period of nine years. As further attacks of girdle pains were prevented by the administration of phenobarbitone, it seemed that the theory as to their causation was substantially correct.

Other manifestations of cerebral involvement less dramatic then epilepsy may simulate the clinical pictures of various degenerative diseases of the nervous system—disseminated sclerosis, chronic encephalitis, cerebral tumour, etc.

(3) *Psychological changes.*

There may be insidious psychological changes noted in victims of cysticercosis—mental dullness, impairment of memory, periods of disorientation, or a change in disposition, so that a person originally alert and efficient becomes careless and untrustworthy. Clinical states simulating the various psychoses may develop, e.g. hysteria, neurasthenia, melancholia, mania, delusional insanity, or schizophrenia.

The manifestations of this disease are as protean as those of malaria, syphilis, and hysteria.

Diagnostic Features.

(1) *Presence of tænia solium* infection of the intestine, as shown by the presence of gravid segments in the fæces. The segments of T. solium are distinguished from those of T. saginata by the fact that the gravid uterus of the former has approximately nine lateral evaginations compared with fifteen of the latter.

(2) *Inspection and palpation* of the whole body surface in search of subcutaneous or intramuscular cysticerci. The retinæ and tongue should be carefully examined. Any suspicious nodule should be excised for microscopic examination.

(3) **Radiographic examination** gives evidence of cysticercosis only some years after the process of invasion, when the parasite is dead and the cyst calcified. Repeated X-ray examinations of the whole body should be carried out at yearly intervals after the onset of late cerebral symptoms. As calcification does not occur until at least three years have elapsed after the beginning of the process of mortification, diagnosis by this means is necessarily much more delayed than by the former method. As calcification in the brain occurs only very rarely, and then only in the scolex, it follows that radiographic investigation of the skull area alone is of no practical value. The demonstration of a calcified cysticercus in the muscles is strong presumptive evidence of cerebral involvement. Morrison and more recently Brailsford have described in detail the various radiographic manifestations of the disease.

(4) *The blood picture* is of little diagnostic value as most people who have lived for any length of time in the tropics show a degree of eosinophilia. An eosinophilic reaction occurs during the process of invasion, and also at the time of mortification. The demonstration of an increase of the eosinophilic count in the presence of fits is, however, suggestive.

(5) *The cerebrospinal fluid* may show signs of cellular reaction by an increase in the lymphocytic count during the process of invasion. The fluid in this patient was normal in all respects, and the large number of cases investigated in this way by Dixon and Smithers showed no feature of diagnostic value.

(6) *Complement fixation and skin tests* have unfortunately not the high degree of success of the corresponding tests in schistosomiasis, filariasis, and hydatid disease. A negative reaction does not exclude the presence of the disease, but a positive reaction is of the greatest clinical significance.

Prognosis.

The prospect of survival depends on the intensity of the cerebral involvement. If the host survives the period of invasion he may succumb in status epipepticus six to ten years later. If he survives the irritative phase during the process of mortification the prognosis improves as each year advances after the twelfth year since his original infection.

TREATMENT

Prophylaxis.

As there are no methods of treatment of the established disease, prophylaxis is of paramount importance. The wise legislative measures instituted in this country in 1582 are indicative. In addition, the following regulations should be enforced: confinement of pigs to pens in infected areas, careful inspection of all pork at the slaughter house, and efficient sterilisation of all pork products by heating to 100° C. or by freezing. The direct use of human excreta as a manure for gardens should be prohibited.

Therapy.

Should segments or eggs be discovered in the fæces, energetic treatment to destroy the worm must be instituted without delay because of the danger of auto-infestation.

Extract of *aspidium felix mas* or malefern is the anthelminthic of choice. This remedy has been utilised for more than 2,000 years, and has yet to be improved upon. The oleoresin of felix mas which contains 24 per cent of the active principle felicin is used in capsules containing 1 c.c. each.

For success in treatment attention to detail is of the utmost importance. For 48 hours beforehand the patient should have no fatty food, and for the last 24 hours fluids only. On the final evening ½ oz. of sodium sulphate in a full glass of water is given to further cleanse and

expose the surface of the cestode. On the following morning at half-hourly intervals three capsules, each containing 1 c.c. of the ext. felix mas, are administered. For children 1 minim is given for each year of life in each dose. Two hours later a second sodium sulphate purge should wash away the tapeworm which has been paralysed by the action of the felicin. Care must be taken to confirm the expulsion of the head. Should the treatment be unsuccessful in removing the head, the same ritual of treatment should be repeated after an interval of ten days.

An even more efficient, but rather more dangerous, method is the use of carbon-tetrachloride. The same ritual as above is followed, except that the fats need not be excluded, and a 3 c.c. capsule is administered in one dose. For children 3 minims are given for each year of life. The toxic effects of this drug may produce in susceptible subjects symptoms of acute liver atrophy —intense jaundice, subcutaneous haemorrhages, and eventually convulsions and death. For this reason it should never be administered to patients who have been addicted to alcohol, or who suffer from cirrhosis of the liver or chronic renal disease. It has been found that by giving large doses of calcium lactate and gluconate, both by mouth and intramuscularly for three days before the treatment, the toxic symptoms are greatly reduced or altogether avoided.

Phenobarbitone and bromide are the limited available drugs during the late irritative phase of the disease.

Surgical intervention is of no avail, as the sites of irritation are always multiple, but decompression may be necessary for the relief of raised intra-cranial pressure.

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SURGERY IN RELATION TO MENINGITIS

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Exploring the role of surgery in meningitis unfolds an ever-expanding field. How best to peruse the subject is a moot decision, but two main aspects loom to the fore: firstly, the problem of the acute stage of the disease, and secondly the sequelae arising out of incomplete resolution of the inflammatory processes.

Acute Meningitis

Admittedly in the acute stage of meningococcal meningitis, and other forms of primary meningitis, apart from lumbar puncture or the comparable operations of cisternal or ventricular tapping, surgical intervention assumes but limited application. The position in relationship with possible sequelae, however, is somewhat different, for the formation of scar tissue is apt to induce obliterative changes within the cerebro-spinal fluid pathways, thereby causing obstruction within the subarachnoid spaces of the brain, or the spinal cord.

With secondary varieties of meningitis, such as the septic type, the surgical aspect is paramount, as immediate relief is to be sought in the eradication of the primary focus of infection. The source of infection, nevertheless, is not always so obtrusive, because either the pathway of transmission is not revealed, or lack of co-incident symptoms veils the picture—in other words, a fissured fracture involving the accessory nasal sinuses may not be visualised on the radiograph, or the manifestations of meningitis in a patient with middle ear disease may not be predetermined by the loss of cochlear or vestibular function. The perforating qualities of a particular wound can escape recognition pending the development of meningeal infection; such a lack of realisation may be accounted for out of the limited dimensions of the surface wound,