CYSTICERCOSIS AS SEEN IN THE BRITISH ARMY, WITH SPECIAL REFERENCE TO THE PRODUCTION OF EPILEPSY.¹

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The above title was chosen because my experience of cysticercosis has been limited, almost entirely, to soldiers serving or discharged. It is not suggested that the disease is in any way peculiar to members of His Majesty’s Army.

Many persons who hear of the investigation which is the subject of this discourse, are curious to know how it came about, so in anticipation of such questions I give an introductory explanation on this point. The inquiry was undertaken to determine, as far as possible, the degree of responsibility of cysticercosis for epilepsy in the Army. On an average, about a hundred soldiers are discharged the Service every year because of epilepsy. Some of these men slipped past the recruiting officers by making a false answer on attestation, and denying that they had ever suffered from fits; in others,

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² In the writer’s opinion, there is nothing new to be said regarding the important literature of cysticercosis. He believes, too, that it would best serve his purpose to give an independent account of the disease as actually studied in the Army. For these reasons the traditional review of the literature of the subject has been expressly omitted. Those interested will find an extensive bibliography in the monographs, “Le Cysticercus cellulosae chez l’Homme et chez les Animaux,” Y. Vosgien, 1911; and “Les Cysticercoses du Neuromé,” P. Schmitte, 1928.
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an inherent epileptic taint became evident only after enlistment. When these two groups, and various oddments, are deducted from the total, there remains a considerable number of men previously healthy and coming of sound nervous stock who developed epilepsy in adult life during or after service abroad. Long before the problem of epilepsy in the Service came within my particular province, I had been much concerned regarding the possible etiology of this form of the disorder for it could not be attributed to neoplasm, specific disease, or traumatic lesions of the brain. For years past there has been diagnosed in the Army, and sometimes published, an occasional case of cysticercosis complicated by epilepsy. These were regarded by most, including myself, as isolated curiosities of medicine, and their significance as outcrops of an extensive hidden infestation was not appreciated. At one time or another I saw some half-dozen of these earlier cases, and viewing them as a whole it seemed peculiar that they should all have some outstanding sign which could not well escape notice, and which, naturally would lead to investigation. For example, one man had sixty subcutaneous nodules; another almost as many; a third showed a cysticercus in the eye, and so forth. It appeared to me that if these men had presented less obvious signs of cysticercosis, if instead of dozens of palpable nodules, only one or two, or perhaps none, had developed, then the true nature of their malady might not have been suspected. As I expressed it to various colleagues at the time, it was as though we were detecting enteric fever only in those patients who pass a quart of blood per rectum. About six years ago, this belief received striking support. The pathologist to a civil hospital asked for my opinion regarding the nature of some puzzling bodies in the brain of a confirmed epileptic who had died in a fit. According to the history given me then, the man, during the long course of his disease, had been seen by various practitioners of experience, including some with special neurological knowledge, but nothing had been noticed to arouse suspicion that his infirmity was other than some form of textbook epilepsy. On examination of the brain, the bodies in question proved to be cysticerci. This was the most important step in my education for the case linked up the known group of frank cysticercosis, and the presumed obscure or cryptic variety.

Further evidence was collected, here I must mention Lieutenant-Colonel R. Priest's independent work, and by 1929, when it first became my duty to write a part of the Annual Report on the Health of the Army, I felt justified in stressing the close association of epilepsy in the Service and cysticercosis. And finally, eighteen months ago, the War Office issued orders that all men who develop epilepsy in the suggestive circumstances I have mentioned, are to be sent to Millbank for special investigation.

In one way or another, over sixty military cases of proved cysticercosis have been collected, but I do not stress the figure for we know that it is far from complete. There are several figures, however, which I consider most significant; twenty cases of cysticercosis have been diagnosed in Millbank in
the present year, and besides others still under suspicion, eight men who
returned from India during the past trooping season have already been
proved to suffer from cysticercosis. Of these eight, six were invalided
home as ordinary epilepsy, and the other two, though not so diagnosed, had
been under medical observation in India because of a history of some kind
of seizure.

As everyone knows, there are two tapeworms of which man is the sole
host in the adult stage—Taenia saginata and T. solium; the former passes
its cysticercus stage normally in the ox, and the latter in the pig. T. saginata
is not known to cause human cysticercosis, and if it ever does so, the
infestation must be very rare. Whereas the intermediate stage of T. solium,
given the opportunity, develops rapidly in man, and the embryos have a
predilection for the brain, where they select especially the grey matter. I
believe that the preference for the eye, especially in textbooks, is only
apparent, and is due to the fact that parasitization of the eye, unlike that of
other parts of the body, cannot fail to attract attention. In the pig the
development of the cysticercus is said to be complete in from three to four
months; I presume this refers to its structural maturity, for in man the
the parasite may be observed to increase in size for years. Unlike what
happens in the case of cesturus and an echinococcus, each cysticercus
includes only one larval scolex; so that a single egg of a T. solium of one
generation cannot eventually give rise to more than one adult worm of the
next generation.

**Incubation Period.**

The period that may elapse between the invasion of the body and the
onset of manifest symptoms is difficult to fix. The main reason is that in
the great majority of our cases there is no evidence of infestation with an
adult T. solium at any time—that is to say, the patient shows no sign of
intestinal tæniaisis while under prolonged observation, he has not been
treated for tapeworm in the past, and denies having ever passed segments.
And owing to the greater liability to bowel diseases in the tropics and the
consequent watch likely to be kept on the condition of the dejecta, and also
owing to the general absence of a water-carriage sewage system, I think
that it would be more difficult to overlook a tapeworm infestation abroad
than in this country.

In a minority of cases there is a history of T. solium to suggest the
probable time of infestation. In some of these the symptoms commenced
while the patient was actually in hospital undergoing treatment for tape­
worm; in others there was a latent period of several years between com­
plete expulsion of a worm and the eventual development of clinical
cysticercosis.

**Prodromal Symptoms.**

In many instances the process of invasion gives rise to little or no general
reaction, and so as a rule prodromal symptoms are absent or so mild that
their significance is appreciated only long afterwards. Sometimes at about
the probable period of invasion there is a record of admission to hospital for headache and unidentified fever, or for myalgia or rheumatic pains, but these latter are usually of a degree so indefinite as not to impress the patient’s memory. Again, there may be a history of temporary localized or diffuse oedematosus swelling of those muscles that later are found to harbour cysticerci.

None the less, many men who show advanced infestation of the muscles at the time fits commence declare that they have never had a day’s illness, nor missed a game of football or hockey for reasons of health. One man who was a keen pugilist first became aware that something was wrong when a cyst, which had formed on one wrist, interfered with the strings of his boxing glove during a fight. This drew attention to the presence of several other cysts, but he continued to appear in the ring as usual. A year later he had his first epileptic fit.

**Established Disease.**

If palpable cysts appear, diagnosis is a simple matter, but it must be understood that parasites may invade the body even in large numbers and give no outward or visible sign of their presence. Years later, if they calcify, they may be discovered, perhaps accidentally, by radiological examination.

The number of palpable cysts varies widely in different cases, and also alters in the same patient from time to time. Large cysts that have been under observation for years may vanish completely within a few days, while others appear in sites where previously there was none. Because of this coming and going, patients sometimes imagine that the nodules migrate from place to place. There may be dozens of these palpable cysts or only one; but there is always a larger number in the tissues than show themselves, for when they eventually calcify and become visible in radiographs, more may be seen, say, in one arm than could be palpated at any time in whole body. As a rule, the palpable cysts eventually disappear, with or without accompanying calcification; sometimes, however, calcareous cysts, if sufficiently superficial, remain perceptible as small hard nodules.

The cysts may be detected in the muscles or subcutaneous tissues of any part of the body—the head and face, including the eyelids and lips, trunk and limbs, but rarely in the hands or feet. They are found more commonly in the upper half of the body, not because the parasites are more numerous here, but because of the better cover afforded by the larger masses of muscle in the lower half.

Their size depends mainly on their age and their situation. If in firm tissues which are equally resistant on all sides they tend to be small and rounded, as in the substance of the brain. In the muscles they are oval and lie between the fibres, separating them, and when fully grown may attain a length of twenty millimetres or more. Their size, when palpated
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in muscle, usually suggests that of a pea or a hazel nut, but I have encountered examples which in the tissues felt as large as a pigeon's egg.

There is one developmental peculiarity of the cysts which I believe to be of transcendent importance in the pathogenesis of the disease. Its most evident outward expression is seen in the manner in which cysts may continue to appear, singly or in crops, over a long period of time measured in years, while others already in evidence may increase in size in the same way. If we can explain this phenomenon, I think that we solve at the same time the mystery of the extraordinary alterations in the character and extent of the symptoms exhibited by an individual patient at different times, and thus replace a bewildering medley of clinical groups by one intelligible and composite whole. I have found no satisfactory explanation in the literature; if such exists, it is so little known that I make no apology for introducing the matter here. On many occasions men known to have suffered from cysticercosis for several years have shown me one or more cysts which they declared had just recently appeared. But when one of these "new" cysts was examined it always proved to be, not a recent formation, but one of long standing, the contained larva dead and in some stage of degenerative change. All such cysts had one peculiar character in common—the tenseness of the cyst capsule owing to the large amount of contained fluid. Therefore, I thought it reasonable to suppose that the death of the larva was associated in some way with an increase in the quantity of fluid, so that a cyst originally flaccid and not differing appreciably in consistence from the surrounding muscle would be detectable for the first time when it became tense and firm; just as a vein in the arm, previously impalpable, can be felt when it is distended with blood through the application of a tourniquet. There was always the possibility, however, that these men were unwittingly in error, and that some accidental circumstance had merely brought into evidence cysts which they had not noticed before. Recently a case occurred where we are not dependent on the accuracy of the patient's story regarding the late appearance of such cysts. An ex-soldier who has suffered from cysticercosis for seven years was in Millbank in March, 1933. The palpable cysts were counted with great care and their position accurately noted. He was discharged from hospital and kept under observation. In July he was readmitted showing three "new" cysts in situations where none had been apparent before. They felt firm on palpation and had a bulk about that of a hazel nut, but somewhat elongated as is usual in muscle. All three were excised and each was found to be so tense with fluid as to resemble a tightly-inflated Rugby football in miniature. As I had ventured to prophesy before excision, the larvæ were dead and undergoing post-mortem changes, the head and neck being so firmly adherent to the surrounding invagination of the bladder that no degree of digital pressure nor any manipulation could evaginate them, and in the end they had to be dissected out. I think that this confirms my theory regarding the late appearance of cysts in the
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tissues. I think, too, that allied changes affecting the intracerebral parasites explain the long delay in the onset of brain symptoms in certain cases of cysticercosis, a latent period that may extend to at least six years from the time that cysts are first detected in the body.

The following composite account is based on an examination of the brains of four men who died of cerebral cysticercosis, and is an attempt to correlate the morbid processes which follow the development of the cysticercus and the accompanying clinical symptoms of brain involvement. In the brain the cysticercus becomes enclosed by a wall of sclerosed neuroglia, corresponding to the fibrous capsule found in extracranial tissues. I do not know how long this neuroglial wall takes to form, but it was well defined in the brain of a man who died eighteen months after his first prodromal symptom of severe headache. It is not the time factor alone that determines the extent of this sclerosis, for the wall around individual parasites in the same brain may differ greatly in thickness. Small round cells and a few plasma cells are present between the delimiting neuroglia and the surrounding normal brain tissues.

Unless the parasites have invaded the brain in overwhelming numbers, or have lodged in some particularly responsive centre, they cause little nervous disturbance while in their relatively quiescent stage, otherwise it seems impossible that anyone could survive for years—as we know to be a fact—with 200 cysticerci present in the brain.

In short, my belief is that cysticerci while alive usually enjoy a relative tolerance on the part of the host, but that after their death they act as foreign irritants and bring about the changes next to be described, partly by their toxic effects and partly by increased pressure, like that which leads to the appearance of "new" cysts in the muscles. Surrounding the dead and disintegrating cysticercus the tissues are seen to be undergoing active degenerative changes, with a marked cellular response both within and without the neuroglial wall which itself is involved in the destructive process. To the naked eye the degenerating tissues may be visible around the cysticercus as a discoloured ring, perhaps three millimetres or thereabouts in depth, shading off into the normal brain tissue. If the patient survives, the damaged tissues may undergo necrosis. This dead area which may extend for at least five millimetres beyond the cysticercus, is ringed off from the normal brain substance by a distinct encircling wall of sclerosed neuroglia.

So that several pathological processes may share, with differing degrees of responsibility, in the production of the clinical picture—the active destructive changes which I suggest are associated with the death and degeneration of the parasite; the tissue necrosis following this severe reaction; and sometimes, possibly, an excessive neuroglia sclerosis around the cysticercus.

1 Since the above was written, this patient has been re-examined by Major Dixon (December 19); thirteen more "new" cysts have become evident since July.
In the brain of one man who died in status epilepticus six years after his initial fit, parasites could be seen in the three main stages I have mentioned. Obviously he had survived the reaction to those which were surrounded by a necrosed area, disturbances possibly expressed in the original onset of fits; I believe that the parasites which were causing the active degeneration I have described, were responsible for his death; and that the small and relatively quiescent cysticerci held potentialities for later mischief had the patient not succumbed.

I do not know of any other explanation which will cover the following observed clinical facts: that commonly parasites are present in the body for at least one or two years, and sometimes much longer (e.g. six and eleven years) before brain symptoms become evident; that when these develop they are subject to periods of exacerbation, followed by intervals of relative or absolute quietude; and that the character of the symptoms may vary so markedly that an individual patient seen at intervals by different observers has been diagnosed as delusional insanity, disseminated sclerosis, and cerebral tumour.

After a variable period determined in part by the resistance of the host the parasites in the body tissues die and often undergo calcareous change. What I take to be the classical teaching regarding the sequence of events is, that the fluid in the cyst becomes gelatinous and the whole contents reduced to a caseous mass which eventually calcifies. I do not doubt that this may happen, but up to the present I have not seen any stage of this process in an excised cyst. Our experience has been that calcification commonly commences in the scolex, and in a number of instances we have found an undegenerated bladder wall with its fluid contents unchanged appreciably, and in this a calcified scolex which in at least four cases was lying quite free. So that calcification of the scolex may be complete while the cyst capsule and its other contents are unaffected. From a comparison of excised cysts and radiographic appearances, I believe that calcification may stop at this point and go no further, the cyst wall collapsing through the escape or absorption of the fluid, and disappearing. Or the collapsed cyst may be flattened by the pressure of the surrounding muscle and calcify in an extended form. Again, after calcification the cyst may retain much of its original shape, supported, presumably, by solidified fluid, thus withstanding the pressure of the tissues. Here, too, the degenerative process often commences in the scolex which may be seen heavily calcified at a time when the surrounding mass shows only as a ghost-like shadow. Occasionally the appearances suggest a calcium invasion spreading from without inwards, and from analogy, there seems no reason why this should not take place.

Thus it is evident that the appearance of a calcified cyst is determined mainly by its original size and the presence or otherwise of a host capsule; by the degree of collapse undergone by the cyst wall and the position it assumed; and the final disposition of the contents.
The time that may elapse before calcareous changes commence depends on many variable factors, the most variable being the duration of life of the parasite, for the longevity of individual parasites even in the same host may differ by years. But there is always the possibility that some of the invaders may not have survived long, and so recognizable calcium deposits might reasonably be expected somewhere in the body in a case of a four or five years' duration, or even less. I believe that generally about three years are required after the death of the cysticercus for the scolex to calcify. Radiological examinations made at any relatively early stage should, if negative, be repeated after appropriate intervals.

Calcification in the brain is a much more dilatory process as a rule, and this degenerative change may be complete in the muscles at a time when the cerebral cysts remain unaffected. One ex-soldier in this series was operated on eleven years after the onset of fits. Several cysts removed from the cerebral cortex showed no signs of calcareous change although the cysts in the muscles had then been heavily calcified for three years, and some of them for five years. In one of our cases, radiographs showed calcified cysts in the brain only, and none elsewhere, presumably because the brain alone had been invaded.

Of the nervous manifestations of cysticercosis by far the commonest and most striking is epilepsy. Some people may find this term objectionable, but I use it deliberately for there is no symptomatic character of the type of epilepsy that may be produced to differentiate it from one or other of the classical forms of that disorder. The attacks may be like those of petit mal, or may be Jacksonian in type, with or without loss of consciousness. They may show all the stages of the textbook fit of major epilepsy, with aura, biting of the tongue, relaxation of sphincters, postepileptic stupor, and so forth. In some instances they are irregular in character and show no clear-cut sequence of stages. In this connexion it may be mentioned that a number of ex-soldiers (eventually proved to suffer from cysticercosis) have been diagnosed and demonstrated as cases of cerebral tumour in several teaching hospitals in London and elsewhere.

There may be a long history of incomplete fits—often regarded as hysterical—prior to the commencement of fully-developed major attacks. For example, one man used to stand for a few seconds with his teeth clenched and his left wrist flexed. After about a year of such attacks, one day he was carried into hospital unconscious and within the next twenty-four hours had three major fits, followed by five more in rapid succession a day or two later. Another man, during a period of two years, had momentary attacks during which he flexed his head to one side, being quite conscious of the movements but unable to control them. Three years ago major attacks developed, commencing with three in one day. They still continue at intervals, and recently I learned that when falling in one such fit he had dislocated both shoulders at the same time.

Sometimes fits commence at about the time that cysts are first detected.
In other cases there may be a long latent period between the appearance of cysts and the first seizure. One man developed subcutaneous cysts in 1922. He showed no outward sign of brain involvement for six years, and then, in 1928, had a major epileptic fit. He went down hill rapidly, and eighteen months after his first seizure he is recorded as being very weak and tremulous, scarcely able to walk or stand, and having great difficulty in understanding and answering questions. He died six months later. Again, cysts may become palpable only subsequent to the onset of epileptic attacks. To illustrate this sequence I cite the case of a soldier who developed fits and was discharged the Service as suffering from certified “true epilepsy.” Not for four years did cysts commence to show themselves in the muscles, and they have continued to appear at intervals during the succeeding three years, including 1933.

The other signs of involvement of the central nervous system are less dramatic than epilepsy, but they cover an extraordinarily wide range. When we reflect that the embryos may lodge in any part of the brain, it is easy to realize that any symptoms, motor, sensory or mental, which accompany focal lesions in the brain may be produced in cysticercosis. The picture may be that of cerebral tumour with all or any of its classical symptoms, or may resemble disseminated sclerosis, or if there is a hyperinfestation, acute encephalitis. One soldier in this last category survived for only seven days after admission to hospital on his first complaining of intense headache.

With or without fits, psychical disturbances may predominate at times, and the considered diagnoses in cases later proved to be cysticercosis included (besides that par nobile fratum, hysteria and neurasthenia)—melancholia, acute mania, delusional insanity, and dementia praecox. As well as gross mental disturbance which suggests diagnoses such as these, there may be mental dullness, impairment of memory, temporary periods of disorientation, or a change in disposition, so that a previously efficient soldier may become careless and untrustworthy. Indeed, Colonel Benson, commanding the Q. A. Military Hospital, Millbank, has told me with some feeling that if any breach of ward discipline is reported, usually a cysticercosis patient proves to be the delinquent.

I want to make it quite clear that the foregoing are mere phases or stages of the infestation, determined, as I believe, by the waxing and waning of parasites in the brain, and that they are not clinical entities. Some authors divide the disease into so-called “types”—one characterized by headache, another by vertigo, a third by sensory changes, and so on to about twenty in all—and state the relative frequency of each of these in figures. This is a misleading representation of the disease. It results from basing the symptomatic account on published cases collected from the literature. Naturally each was described in whatever stage the observer happened to see it. If these same patients had been examined a year or two earlier, or later, many of them would have been classified as belonging
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to quite a different "type." Major H. B. F. Dixon has shown indefatigable energy in ascertaining the early and subsequent histories of the patients in our series. To-night he has brought summaries of the clinical histories of over sixty military cases of proved cysticercosis, and anyone interested can see here how an individual patient when observed from first to last may be seen to pass through every one of the so-called "types" of this terrible disease. An early emphatic entry, "No Fits," does not preclude a final one, "Died in status epilepticus."

Diagnosis.

As is the case in many other maladies, the great impediment to the diagnosis of cysticercosis is the failure to think of the disease actually present. Everybody knows that man may serve as the intermediate host of T. solium, but often there is a hiatus between this theoretical knowledge and its practical application. Even as a house physician I was aware of the great danger of handling tapeworm segments carelessly; yet I turn pale when I think of the number of cases of cysticercosis that I must have missed since then. Several times when taking part in examinations for post-graduate qualifications in medicine, I have provided an exceptionally easy case of cysticercosis for the clinical test, mainly as an interesting experiment. The first occasion, over seven years ago, may be taken as typical of the rest. The diagnoses offered included Von Recklinghausen's disease, secondary malignant deposits, and even nodular leprosy. None of these qualified candidates suggested cysticercosis or mentioned it in his discussion as a possibility, however remote.

I do not know of any English textbook that deals with this disease in an adequate manner. In some the account is too brief or too incomplete to be of much practical help. The account in others is a compilation of excerpts from various sources, which only serves to give the reader a misleading picture of what he may encounter. One large and generally admirable work dismisses cysticercosis in two and a half lines, and even in this space room is found to emphasize its rarity. While in the long and detailed account of the causes of epileptiform convulsions it is not even mentioned.

The history in itself may be suggestive—the onset of fits in adults without any evidence of a familial or personal epileptic taint, who are not suffering from neoplasm, syphilis, or the effects of head injury. A history of residence abroad increases the probability of cysticercosis, but persons who have never left England are not exempt from attack. And, of course, infestation may take place at any age. Two of this series were boys aged 13.

The most generally helpful sign in diagnosis is the presence of palpable cysts in the body tissues, and the patient under suspicion should be examined thoroughly from head to foot. Cysts, if not numerous, are easily overlooked. On one occasion I examined an epileptic patient with great care and pro-
nounced him free from palpable cysts; just as I turned away the sun came out behind him and threw into relief a tiny elevation above one clavicle which I had failed to detect before. This proved to be due to a cysticercus, the only one discovered in his body. Often the patient himself is aware of the presence of nodules, and if these are seated deep in the muscles and difficult to feel, he may demonstrate some digital manipulation, such as manoeuvring a cyst against a bone, which experience has taught him to be effective.

In order to demonstrate the parasite, a suitable cyst is excised under local anaesthesia, and freed from any adhering tissue. The host capsule is incised carefully so as not to injure the cysticercus which is gently extracted. The appearance of the translucid membrane with its central "milk spot," representing the invaginated scolex, is characteristic. If alive, the parasite may evaginate the head and neck, or may be induced to do so by immersion in warm saline. Pressure applied to the bladder may succeed if these methods fail, but when the larva is dead and the scolex adherent to the surrounding membrane, dissection will be necessary to display its characters, sometimes a laborious but not otherwise difficult task. When calcareous degeneration is so extensive as to mask the structure of the parasite, the calcium can be dissolved by weak HCl which does not affect the hooklets.

Diagnosis in cysticercosis is more easily established if palpable cysts are present; the disease is no less likely if they have been absent throughout, for in many persons the parasites lodge deep in the muscles, and so escape detection. If palpable cysts are recorded as universally present, at some period, in any considerable series of patients, this is good evidence that many cases of infestation are being overlooked.

When the embryos are active in the body, no doubt an eosinophilia results. But in the established disease when these small parasites have been walled off, as would be expected there is usually no help to be gained from blood-counts.

Presumably for the same reason, the complement-fixation and skin tests—which are group reactions—have not the high degree of success of the corresponding tests in schistosomiasis, filariasis and hydatid disease; and when carried out in known cases of cysticercosis they are more often negative than positive. The complement-fixation test, however, has been of great value on occasion, and in two instances it was positive at a time when all other methods of diagnosis gave no help. Its accuracy was confirmed later in both of these. The greatest need at present is for some reliable means of detecting infestation when the parasites either are actually limited to the brain, or if present elsewhere in the body as well, cannot be discovered. For this purpose the complement-fixation test with its existing limitations does not offer any prospect of success; for although a positive result points to parasitism, a negative result is valueless and may accompany even very heavy infestation.

There are no constant changes in the spinal fluid, and such deviations
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from the normal as may occur have no positive diagnostic significance. Even when profound cerebral disturbance is present, the spinal fluid may remain unaffected. None of our patients showed an eosinophil reaction.

Major W. K. Morrison, lately radiologist at the Q. A. Military Hospital, Millbank, has made a study of the radiological appearances of calcifying and calcified cysts. He compiled the accompanying diagram from the radiopacities shown in twelve cases of cysticercosis, and although many more positive radiographs have been obtained since then, it has not been found necessary to add to the figures in the diagram. The somewhat fanciful names have been found useful in reporting the presence of parasites.

In practice the diagnosis may become apparent only in course of time—the onset of fits may bring to notice pre-existing cysts in the tissues; or the appearance of belated cysts may suggest the nature of an epilepsy of long standing. Again, the calcification of parasites not previously detectable may determine a diagnosis by radiological examination.

No single series of examinations and tests, however thorough and complete, can be relied on to exclude cysticercosis. Therefore, I emphasize the necessity of keeping cases of suspected or possible cysticercosis under observation, examining them at intervals, and making further X-ray examinations after the lapse of six months or a year.

Diagnosis in some unknown proportion of cases of infestation is impossible during life, and even post-mortem the presence of a few parasites—perhaps only one or two in all—probably would not be detected unless the pathologist has cysticercosis in mind, knows what to look for and leaves no scrap of brain substance more than four millimetres square, unsearched.

I am indebted to Major Morrison for the following notes on X-ray examination:

"For routine investigation the following regions are radiographed—skull, lateral view only; root of neck; upper arms; fore-arms; thighs; legs. The films and the intensifying screens should be free from 'grain.' The Potter-Bucky diaphragm is used only for the skull radiograph. The standard of radiograph aimed at in each region should be that of the ordinary exposure conditions for bone detail, with a very slight under-exposure. It is useless to take the radiograph under special conditions to show soft tissues only.

"The parasite may be represented by a final radiopacity in any form from the calcified scolex (about one millimetre in diameter) up to a fully-grown elliptical cyst (about twenty-three millimetres long). Radiographs are viewed in the ordinary way, but owing to the faint shadow produced by the early calcifying parasite, the search of the soft tissues must be a very thorough one. It is quite easy to miss a small section of a calcified parasite at the upper or lower margins of the films, or to miss one overlying normal bone shadows. Oblique illumination is sometimes of value. If single shadows are observed, the patient's skin should be examined for the presence of warts, scars, or red ink tattoo marks. The shadows often imitate simple film stains, and in case of doubt the radiograph should be repeated. Films should be filed for comparison with those taken at a later date."

It is scarcely necessary to add that the radiologist should familiarize himself with the structure of cysticerci and their developmental history in the body.
Though it may seem incredible to members of a scientific society, I have been asked what is the good of “going to all this trouble” to establish a diagnosis of cysticercosis when little or nothing can be done for the sufferer. There is one obvious advantage to a soldier, for the disease when acquired abroad is rightly held to be attributable to military service. Apart from this material gain there are other advantages. Men labelled as epileptics, and labelled wrongly as was proved later, have protested, literally with tears in their eyes, that epilepsy was unheard of in their families, and have asked with dismal forebodings whether the disease would show itself in their children. Further, when mental deterioration has advanced so far that the subject becomes certifiable as insane, it is an indescribable relief to all his family and connexion—even though the unfortunate victim himself may gain no benefit—to know that the slur of familial lunacy has been removed.
FIG. 1.—One calcified cyst and several calcified scoleces. (Eight years after removal of unidentified tapeworm, 6 years after first nodule detected, and 4 years after initial fit). 
A +; B +.

FIG. 2.—Two calcifying cysts. (Seven years after initial fit, and three years after first nodule; no tapeworm). 
A +; B -.
A + = Cysticercus excised and identified. B + = Complement-fixation and skin tests positive. 
B - = Complement-fixation and skin tests negative.
Fig. 3.—Two calcifying cysts. (Six years after initial fit; no nodules detected; no tapeworm.)

B—.

Fig. 4.—A calcareous scolex, presumably free, at lower extremity of calcifying cyst. (Same year as initial fit, but the appearance of the parasite shows that infestation has lasted for several years; subcutaneous lipomata rendered search for palpable cysts ineffective; no tapeworm.)

B— = Complement-fixation and skin tests negative. B+ = Complement-fixation and skin tests positive.
Cysticercosis as seen in the British Army

Prognosis.

Prognosis in this disease is a matter of extreme difficulty for there is no method of ascertaining during life the number of cysticerci and their distribution in the brain, or of forecasting the vagaries of their future behaviour on which so much depends. The most dangerous time is from the sixth to the eighth year for then the grave intensification of existing cerebral disturbances most frequently takes place, and subjects who have remained free from outward signs of extensive brain involvement are most likely to show them about this time.

The general tendency is one of retrogression, as evidenced by signs of mental deterioration which may be so marked as to necessitate institutional segregation. On the other hand, some patients in spite of persisting epilepsy remain mentally alert; while in four of the series, fits have ceased after a duration which ranged from a few months to twenty years.

Treatment.

The luminal and bromide series are sometimes helpful in controlling fits, but no medicinal treatment so far employed has had any curative effect. Indeed the observations on tissue changes which follow the death of intracerebral cysticerci suggest that the destruction of large numbers of these parasites at the same time—supposing that some chemical of lethal power were forthcoming—might only make matters worse for the sufferer. Of two men who received intravenous injections of antimony tartrate, one developed a crop of "new" cysts afterwards, while in the other, the cysts already present increased in size and the cerebral symptoms became aggravated. These results cannot be attributed to treatment, for similar phenomena are commonly seen in untreated patients, but they are such as would be expected to follow the administration of a parasiticidal drug, unless, possibly, this were employed very early in the course of an infestation before the embryo had attained larval maturity.

The large numbers of parasites found in the brain and their wide distribution there do not encourage a general resort to surgery. The successful removal of cerebral cysts is reported occasionally in the medical press, but before claiming a cure or appraising the degree of permanent improvement, time must be given for any other parasites in the brain to die off, and this may mean years of observation. In actual practice a temporary amelioration of symptoms after removal of one or more cysts has been followed by the death of the patient in status epilepticus.

I should not favour operation except when some restricted and constant localizing sign is present (e.g. aphasia). It is important to remember that muscular spasm if confined to some particular part during a single fit does not necessarily indicate a limited cortical involvement, for when an individual patient is watched in a series of several fits, each of them may be observed to affect a different group of muscles.

The possible extent of indigenous cysticercosis in England is unknown,
for no one has yet searched for the disease. That it can be contracted here is shown by one of our cases, a man who has never left this country. Intestinal infestation by *T. solium* contracted in England is believed to be rare to-day, but there is always a possibility of infested persons returning from abroad, and recently I heard of a man resident for over four years in

![Image: One calcified cyst and many calcified scolexes in brain. Of twenty-six patients who showed calcified parasites somewhere in the body, positive radiographs of the brain were obtained in five. Calcification in the brain, when it occurs, appears to affect mainly the scolex, and sixty-six cerebral parasites out of seventy-one detected in films showed the scolex type of calcification.](image)

a country district in England, who has been passing segments of *T. solium* throughout this period. To intensify this risk of chance infestation there is a common practice in rural England, as I recently learned to my amazement, of using human dejecta as a fertilizer for vegetable gardens and fields. But apart from the possibility of home-acquired cysticercosis, there are many people in Great Britain who have been exposed to a risk of
Although this type of calcification is well known, the radiograph is included because of the patient's history: Aged 40, served in India 1913-20, Egypt 1920-23; has not left England since 1923. No tapeworm; "never had a day's illness." In October, 1932, had a major epileptic fit, followed by a second in 1933, which was witnessed, and certified as "true epilepsy." The condition shown above is general throughout body. No nodules, and none noticed at any time. Duration of infestation unknown, but clearly it has existed for many years.

[This and the preceding plates are from radiographs provided by the Queen Alexandra Military Hospital, Millbank, London.]
contamination abroad—discharged soldiers and sailors of the regular forces, the tens of thousands who served abroad during the late war, civilians who follow their avocations beyond the seas, and, of late, the crowds of tourists who explore tropical countries with a zeal undamped by any knowledge of preventive medicine. I should like to enlarge somewhat on the group mentioned first, in case my words may reach the general practitioner. Our experience suggests that there are many undiscovered sufferers from cysticercosis among army reservists and discharged soldiers. Men who develop fits after leaving the Army hide their disability and deny it if questioned, for they live in dread of losing their few weekly shillings of reservists' pay. Fits to them mean epilepsy, and they know that (true) epilepsy as a "constitutional" disease is regarded as not attributable to military service. Several ex-soldiers who had lost job after job because of "fits"—one of them an inmate of a workhouse—made despairing appeals ad misericordiam, and have been found to be victims of cysticercosis. There must be many others throughout the country.

Our investigation gives at least some indication of the serious extent of cysticercosis in the Army. It is not a military disease in any sense, and there is nothing in the ordinary life of a soldier to render him more likely to contract infestation than other persons resident in the countries where British troops serve. Of these, we know that the disease has originated in India, Egypt and Malaya, and probably in other countries as well, but because of changes of station during the long presymptomatic period so commonly present, it is often impossible to say where the disease was contracted. I see no reason to suppose that the European civilians in these countries are entirely exempt from attack.

If a commission of persons who have studied this disease, especially in its more elusive forms, were to search diligently in lunatic asylums, institutions for epilepsy, and general hospitals, I think that the results would be surprising. If such an inquisition did not bring to light hundreds of unsuspected cases of cysticercosis, I should regard this as the greatest miracle since Moses smote the rock.

[We regret that owing to limitations of space we are unable to reprint the discussion which followed Colonel MacArthur's paper.—Ed.]