Further extension of the disease beyond the liver is as a rule by direct spread. It is well known that the diaphragm and lung may be involved and that a pleural effusion may occur as with any inflammatory process below the diaphragm, but it is not so well known that from the liver it may spread to the skin, producing a large ulcerating surface. Such a case I was privileged to see in consultation. It was reported and illustrated by Heimbürger (1925) so precisely that the account may profitably be consulted by anyone interested in the histopathology of amoebiasis. There was an area about the size of the palm of one's hand covered with pale oedematous granulations; its appearance was as if a quantity of red brickdust had been ground into the ulcerated surface. It healed rapidly with emetine therapy.

A liver abscess sometimes extends to the abdominal cavity. When this happens the size of the liver may be difficult to determine, and an inflammatory swelling arises which is, as a rule, somewhere continuous with the liver margin. This swelling may extend quite low down in the abdomen, limited either on the right or on the left by the falciform ligament. When it occurs on the left side it is very puzzling, because the occasional abscess arising in the left side of the liver is frequently overlooked. Such an abscess may extend well to the left above the spleen. In ordinary clinical practice it would be regarded as the result of a localized perforation of a peptic ulcer. Such localized intraperitoneal collections of pus from a liver abscess often have a somewhat indirect connexion with the liver, and it may be wise to open them and wash them out. If this course is taken, intentionally or inadvertently, the wound should always be sewn up without drainage and the parent liver abscess aspirated after localization. No harm comes if the patient is immediately put under emetine, and this should, if possible, be invariably done before any intervention is contemplated.

An extremely acute abscess may sometimes perforate into the peritoneal cavity, the case being admitted as an acute abdominal catastrophe. The condition is nearly always such as to preclude any surgical intervention.

Direct Spread from Colonic Lesions

Involvement of the muscular and peritoneal coats of the bowel wall is not uncommon. Sir Leonard Rogers has found amoebae in the lymph adherent to the base of an unperforated ulcer. Actual perforation is rare, but a localized slowly spreading peritonitis is by no means uncommon. It is important to bear in mind its possible existence, because it complicates the diagnosis of other abdominal conditions. The finding of amoebae in the stools makes the diagnosis simple.

Localized abscesses round the appendix are well known but not often seen, and the same may be said of retroperitoneal and perinephric abscesses. If their amoebic origin is not recognized extensive invasion of the abdominal wall may take place.

Ulcers of the colon may be secondarily infected and may produce a localized inflammatory mass which resembles diverticulitis. The lumen of the bowel may be so narrowed by oedema or fibrosis as to produce obstruction. In these cases routine treatment of the dysentery is not always sufficient, and a resection has to be performed.

Finally, the rectum is a common situation for amoebic lesions, and in severe untreated cases these involve the tissues in the ischio-rectal fossa, producing fistulae and ischio-rectal abscess. These may occur in patients who are extremely ill, originating in quite small ulcers without much surrounding infiltration. In more chronic cases the local ulceration may be widespread and, owing to second-

ary infection, the surrounding tissues be infiltrated widely. This may produce a particularly intractable form of stricture requiring colostomy for temporary relief.

Conclusion

It should be remembered that local lesions in the bowel do not as a rule progress thus far, but they do frequently and for a long time act as foci of irritation, exercising a profound influence on the physiology of bowel movement. Such patients have localized spasm and tenderness for years, their bowel is irritable, and they pass frequent stools containing a little mucus. They become bowel-conscious, and haunt the "colon lavatories" in the West End of London and the consulting-rooms of sympathetic physicians. They are diagnosed as cases of anything from spastic constipation to mucous colitis. They require above all things rest, both of the mind and of the colon.

This account by no means exhausts the widespread manifestations of amoebiasis, but enough has been said to show that a knowledge of its manifestations is necessary to every clinician handling cases in or from the East, and to suggest that to the use of the stethoscope must be added that of the microscope and sigmoidoscope as part of the routine examination of patients. By the employment of these methods we arrive at a positive diagnosis of a disease which can be "cured" in a sense that cannot often be used in the case of diseases commonly met with in this country.

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CYSTICERCOSIS EPILEPSY

BY

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The case reported by Dr. W. Blyth (1941), to which he applied the above diagnosis, has many points of similarity to one which came under my notice some years ago, and in which a diffuse cysticercosis of the brain was found post mortem. Out of a large number of post-mortem examinations on mental hospital patients extending over a period of thirty-odd years this was the only case I met with. The condition, though rare, is of interest because—as pointed out by Sir W. P. MacArthur (1941)—of "the importance of considering it as a possible cause of epileptiform attacks commencing in a previously healthy adult after service or residence abroad." The histories of all the cases I found recorded come within this category, though one might extend the term "epileptiform attacks" to those presenting other ill-defined and vague symptoms of cerebral disease. The value of radiographic investigation in the diagnosis is emphasized by Dr. Blyth, but it is not clear from the particulars he gave that these investigations did reveal the presence of cysticerci in the brain tissue of his patient.

References in Literature

The case which came under my observation, and in which unfortunately the possibility of cestode infection as a cause did not occur to me, demonstrated from the necropsy that a condition of cysticercosis of the brain diffuse in distribution produces a clinical picture of epilepsy with dementia difficult to distinguish from the idiopathic form except by the more rapid development of the dementia and of a fatal termination. The cysts examined showed no evidence of calcification. I was so impressed by this massed invasion of the brain by these parasitic cysts that I consulted a number of standard textbooks on psychiatry and cerebral

pathology, but found only scanty reference to the condition, and in some instances none at all, and could discover less than half a dozen cases reported in medical journals in this country over a period of twenty-five years. From these sources I quote the following references:

Von Graefe, it seems, was the first to demonstrate the presence of cysticerci in the vitreous humour. Fagge mentions that as a bladder worm (cysticercus) the parasite is most frequently met with in the eye and in the brain; is often solitary or appears in small numbers, but that probably it is most frequently present in the muscles and subcutaneous tissues, where it is apt to escape notice. Da Costa states that in the brain the chief symptom is violent and increasing epilepsy. Bianchi mentions it in a footnote to his chapter on epilepsy. In *Twentieth Century Practice* (Collins, New York) there is an account of the condition in which it is stated that cysticerci in the brain are more common than echinococci; the symptoms are various—hysteria, hypochondriasis, acute mania, and various motor symptoms resembling Jacksonian epilepsy or cerebral tumour. Osler states that if the ripe ova of *Taenia solium* get into the stomach of man he is liable to become the intermediate host (the part usually played by the pig). This accident, Osler suggests, may occur in an individual the subject of *T. solium*, the most likely manner being by the forcing of mature proglottides into the stomach as by prolonged vomiting. The symptoms produced depend on the number of ova ingested and the localities reached: he groups them under general, cerebrospinal, and ocular.

In his own experience Osler met with only one instance—a patient who was admitted to his wards in a stiff and helpless condition, complaining of numbness and tingling in the extremities and general weakness, and who was first thought to have a peripheral neuritis. On examination subcutaneous nodules were found which proved to be cysticerci. There were none in his eyes, and he had no symptoms pointing to the brain. He saw another, with Friedländer in Berlin, with symptoms of diabetes and anomalous nervous disturbances. The post-mortem examination revealed a cysticercus beneath the valve of Vieussens and pressing on the floor of the fourth ventricle. As to the local distribution of cysticerci, he quotes 155 cases compiled by Stiles: the parasite in 117 was found in the brain, in 32 in the muscles, in 9 in the heart, in 3 in the lungs, subcutaneously in 5, and in the liver in 2. He adds that, except when in the eye, the diagnosis can rarely be made, or, when subcutaneous, one may be excised and examined. Purves-Stewart (1937) quotes a case (reported in the *Centralblatt für Neurologie*, 1902) of a characteristic form of vertigo produced by the presence of a cysticercus in the fourth ventricle. Beattie and Dickson (1925) state that the immature larval or cysticercus stage of *T. solium* may occur in man not only in muscle but in the skin and subcutaneous tissues, where they may be present in large numbers; and in various organs, especially the eye and the brain. In the brain the cysts may attain a considerable size. In the eye they have been known to persist for twenty years. Such infections originate (a) from the patient himself if harbouring the adult cestode, or (b) from a fresh infection from without. Those authors indicate the manner of its dissemination thus: after ingestion by a suitable intermediate host the embryonal shell is dissolved off and the active embryo proceeds to bore its way through the intestinal wall. By way of the lymphatics, or more probably by the mesenteric veins, it reaches the liver (which in the case of *T. echinococcus* forms the special habitat for its bladder-worm stage). In the case of *Cysticercus cellulosae* the embryo travels from the liver, probably by way of the blood stream, to muscles and other tissues of the host's body. In whatever situation the embryo is finally lodged it settles down and becomes passive, loses its hooklets, and undergoes distension with fluid, forming a cyst, and thus entering upon the larval, bladder-worm, or cysticercus stage of its development.

Stitt *et al.* (1938) state that, the segments or ova of *T. solium* having been ingested by the hog, the six-hooked embryo is liberated and becomes encysted chiefly in the tongue, neck, and shoulder muscles as an invaginated scolex ("measly pork"). They point out that the cysticercus of *T. saginata* (*C. bovis*) contains less fluid than *C. cellulosae*, and that, man having ingested the *C. bovis* in raw or imperfectly cooked beef, the adult stage of

T. saginata becomes established in his alimentary canal in about two months. In Abyssinia, they state, this type of cestode infection is said to be universal, and a man without a tapeworm (*T. saginata*) is considered to be a "freak." An important point is the fact that its larval stage scarcely ever appears in man, and it is therefore a much less dangerous parasite than *T. solium*, which readily establishes a larval existence in man if the ova are introduced into the human stomach. If the infection by *C. cellulosae* is not heavy, few symptoms may be observed. It, however, tends to invade the brain, next in frequency the eye, and so cause convulsions, blindness, or death. Instead of being the size of a pea these cysts, when forming in the brain, they add, "may be the size of a walnut or larger. *T. solium* infection is comparatively common in North Germany, but is exceedingly rare in England and the United States." Other references to the condition may be found in works on parasitology and in various Continental reviews and reports. Of actual cases reported in medical journals in this country the following were the only instances I could discover:

- (a) Multiple cysticerci of the brain and epilepsy (J. R. Gilmour, 1902).
- (b) Two cases of cysticercus cellulosae of brain (R. S. Black, 1903).
- (c) A case of cysticercus of brain (W. C. Sullivan, 1903).
- (d) Two cases of cysticercus of brain associated with epilepsy (G. E. Peachell, 1916).
- (e) A case of cysticercus of brain reported at the Bath Clinical Society (Waterhouse, 1911).

Record of a Case

The patient who came under my observation was admitted to the mental hospital certified as a person of unsound mind. He was a well-nourished man aged 31, an ex-soldier who had served in India. He had a history of three months' "behaviour disorder," having become so irritable and strange that he had lost his employment as a machine-man in a factory; he had become unmanageable at home and had threatened to shoot himself. He had no history of psychopathic heredity and none of venereal infection, and had previously enjoyed good health. Physical examination indicated an organic or toxic lesion of the central nervous system; he exhibited a degree of speech impairment with fine lingual tremors, exaggeration of deep reflexes, and Rombergism. Ocular movements were normal, the pupils equal but sluggish (fundi unfortunately not examined), and mental processes retarded, with a degree of confusion bordering on disorientation. He had fleeting visual and aural hallucinations and was unable to converse intelligibly. His habits were neglectful and unclean. The Wassermann reaction of blood and cerebrospinal fluid was negative, and the urine was negative for albumin and sugar. His condition suggested a tentative diagnosis of atypical G.P.I. Within twelve months he developed increasingly frequent epileptiform attacks indistinguishable from true epileptic fits; these were accompanied by a progressive degree of dementia till his death in a series of epileptiform attacks four years after admission to hospital.

P.M. Notes.—The brain unstripped weighed 48 oz.; there was an excess of C.S.F.; the pia arachnoid was congested and thickened, and decortication occurred on stripping it from areas over the cysts. Numerous small cysts were seen scattered over the cortex. They were encapsulated and semi-transparent, ovoid in shape, and varied in size from that of a pea to a little larger. They were mostly single, occasionally in groups of two or three. Their surface distribution appeared to bear a direct relation to the vascular supply, being most numerous in the area of the middle cerebral artery, and this may explain why the brain with its lavish vascular supply appears to be for the parasite a *locus minoris resistentiae*. Most of the cysts made cup-like depressions in the cortex; some were buried in it. They were also present in the sulci and fissures, but were absent on the base. On section of each hemisphere none were observed in the centrum ovale. In the lateral ventricles several of larger size were seen pushing up the ependyma of the floor and sides. In the basal ganglia cysts were present in the region of the lenticular and caudate nuclei and optic thalami. In the midbrain two were located in the right cerebral peduncle in the region of the substantia nigra and in the tegmentum, one below the red nucleus and two pressing into the aqueduct. None were found in the pons or medulla, and only two in the right cerebellar hemisphere.

The spinal cord was not examined. On a moderate computation there were upwards of a hundred cysts scattered throughout the brain. In other organs four were found in the left ventricle of the heart, in the myocardium; none in the liver or other glands. A dissection of the muscles was not undertaken except for those of the right thigh, where one cyst was seen embedded in fibres of the quadratus, which was the only muscle examined. No cestode was found in the alimentary canal. Few of the cysts showed any signs of calcification. On examination under low magnification the cysts had a defined fibrous capsule with some infiltration by small round cells and surrounded by a zone of increased neuroglial fibre growth with an increase of young glial cells. The neurones in close proximity were seen in varying stages of degeneration, grading into normal cortex. The vascular elements were dilated, but there was no evidence of cellular infiltration of the perivascular sheaths. On section of some of the cysts the scolex and hooklets of the embryo were easily identified.

As the degree of calcification of the cysts is determined by time and other factors, it would be of interest to know if the radiographic films of the skull taken by Dr. Blyth revealed the presence of cysts in the cerebrum.

The few instances of this condition of cysticercosis I found reported in this country all occurred in ex-soldiers or in persons who had resided abroad. The cases recorded by Dr. Blyth and Sir W. P. MacArthur are opportune in illustrating the importance of considering the possibility of cestode infection as a cause of epileptiform attacks (and I would add all cases presenting anomalous signs of cerebral disturbance, from neoplasm to early atypical G.P.I.), particularly as at present so many of our countrymen are on active service in Africa and the East.

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MASSIVE ADRENAL CARCINOMA WITH PSEUDO-HERMAPHRODITISM

BY

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Sex characters are primarily determined by the sex chromosome, but hormonal imbalance can alter the picture profoundly. A large group of cases is known in which changes in the adrenal glands can be demonstrated, presenting the "adreno-genital syndrome." In these cases the female sex characters regress, being replaced by those of the male. The changes are mainly dependent on the age of onset and the pathological character of the causative lesion.

A tentative classification may be made, following Broster and others (1932).

1. Clinical:

- Adrenal pseudo-hermaphroditism (before onset of female sex characters).
- Adrenal virilism (onset after puberty).
- Multiglandular syndrome (Cushing, Achard-Thiers, associated with anterior pituitary changes).

2. Pathological:

- Tumours: adenoma, adenocarcinoma, undifferentiated carcinoma.
- Simple hyperplasia (Vines's demonstration of the selective staining of the cortical cells by Ponceau's fuchsin (Broster and Vines, 1933).

The following is the record of an interesting example of Type A.

Case History

A female child aged 2 years 2 months was admitted to the Loughborough General Hospital, under the care of Mr. A. F. Goode, on September 12, 1940. The mother complained that the child had been fretful, and that its abdomen had been swelling during the past few weeks.

On examination the patient was seen to be a thin, puny, wasted infant. Cardiovascular and respiratory systems were normal. The breasts were minute; there was a growth of hair in the axillae and in the pubic region, of male distribution; the clitoris was enlarged, with a small glans, and the rudiments of a prepuce were visible. An enormous ovoid smooth hard mass in the right side of the abdomen could be felt and pushed forward from the loin. The swelling bulged out the right flank; it extended vertically from the costal margin above to the false pelvis below, and medially beyond the umbilicus. It did not move on respiration, and was dull to percussion. The diagnosis was massive adrenal cortical tumour with pseudo-hermaphroditism. On account of its size it was decided that exploratory approach could only be made transperitoneally.

A laparotomy was done by Mr. Goode on September 13. Through a right paramedian incision a very large tumour of the right adrenal gland was seen perched on top of the right kidney, which appeared normal in size but was depressed down into the right iliac fossa. The growth was ovoid in shape, measured 9 in. vertically by 6 in. across, and its surface was covered by a very vascular capsule, fixed superiorly and posteriorly in the region of the portal fissure and inferior vena cava. There were several enlarged para-aortic glands, and the liver was also slightly enlarged. Removal of the main tumour was not attempted. An enlarged para-aortic gland was taken for section, and the abdomen was closed. The child's general condition rapidly deteriorated, and in spite of anti-shock measures she died next day.

Post-mortem Findings.—The vascular capsule over the tumour of the right adrenal gland was incomplete above, where the growth was attached to the diaphragm and inferior vena cava. Para-aortic glands and glands of the portal fissure were infiltrated. The growth itself was of a greasy yellow appearance, was friable, and had large areas of haemorrhagic necrosis. The left adrenal and both kidneys appeared normal. There were no obvious secondaries in liver or lungs. The uterus was very small and the ovaries were represented by two tiny fibrotic nodules.

A portion of the growth was sent for examination, and Prof. G. Haswell Wilson reported as follows: "A cellular malignant tumour with the characters of a carcinoma of the suprarenal cortex. Details are more distinct in the gland specimen, as the tumour itself is the site of extensive necrosis."

Discussion

The formulae of testosterone and corticosterone, the type substances of the male and cortical hormones respectively, as given by Gaddum (1940) are:

and their similarity is apparent. Also to be noted is their close chemical relation to those other sterones oestradiol and progesterone. Clark (1940) states that corticosterone can indeed be synthesized from testosterone.

A further point of interest arises from Broster's (1941) report of a case of "feminism" in the male; this showed