today. This raises the question of more awareness of psychological difficulties by parents because of the easier access to psychiatric reading and terminology through periodicals, the radio and other media. The query then arises—is there in fact more emotional disturbance in the children of our generation or is it only diagnosed more often?

If 'children are no longer pock-marked but Spock-marked' it is because parents, having lost their reliance on family traditions, are turning increasingly to the medical profession for advice about their children’s upbringing and deviations from what is expected.

Although we have experts in the Republic in this field, there are insufficient numbers to meet the need and, particularly because this specialty’s therapy is family-orientated, it requires skilled ancillary personnel to make up such teams. Is the training available sufficient to meet the demand? This kind of experience can only be obtained by actually dealing with children and their parents under the supervision of a skilled child psychiatrist.

The issue is raised because we feel that, firstly, it is a practical problem that has to be met and, secondly, our colleagues in other spheres of medicine would no doubt wish to know much more about this fascinating specialty. If psychiatry has been termed the 'Cinderella of medicine'—what do we call child psychiatry?

Autopsy Findings

The body was that of a thin young White male with a slight degree of jaundice of the conjunctivae. In the right iliac fossa there was a dry blackened area of skin 3 x 1 in., in the centre of which an operation wound was present. There was in addition an 8-inch subcostal incision which showed non-union and necrosis at the ends and under this incision the subcutaneous tissue was extensively necrosed, but no purulent exudate was noted.

The peritoneal sac contained a small amount of turbid fluid containing flakes of fibrin. The right portion of the transverse colon, the caecum and loops of small bowel were adherent to the parietal peritoneum and lower anterior surface of the right lobe of the liver, and as the adhesions were freed thick inspissated pus was found between the adhesions. The left subdiaphragmatic space was normal, but there were a few adhesions present in the right space. A fistula was present between the upper part of the caecum and the wound in the right iliac fossa.

The left pleural sac contained 2 pints of brownish fluid containing small flecks of blackish material. This was the result of the rupture of the lower end of the oesophagus. The right pleural sac and pericardial sac showed no significant pathological change.

Examination of the alimentary tract showed the presence of antemortem digestion of the lower end of the oesophagus with complete lysis of 1 in. of the oesophagus just above the cardio-oesophageal junction, and brownish discoloration of the mucosa was noted above the lysed area. No evidence of amoebiasis or any other pathological change was noted in the rest of the alimentary tract.

The lower anterior edge of the right lobe of the liver, the area in contact with the suppurative pericolic lesion, showed multiple small white necrotic lesions some of which showed evidence of softening, and this lesion had a honey-combed appearance. The rest of the liver showed slight pallor only.

Both suprarenals were the seat of recent massive haemorrhage. The lungs showed congestion and oedema, and the parietal pleura of the left lung showed a few petechial haemorrhages.

The rest of the organs showed no significant change.

Histology. Sections of the wall of the caecum in the region of the fistula showed the presence of focal necrosis and trophozoites of *E. histolytica* (Figs. 1 and 2). Sections of the necrotic lesions in the liver showed the presence of foci of necrosis and trophozoites of *E. histolytica*. Sections of the necrotic tissue under the subcostal incision showed the presence of necrosis and trophozoites of *E. histolytica* (Fig. 3).

Sections of the spleen confirmed the diagnosis of a septic spleen.

DISCUSSION

The outstanding features of this case are the following:

1. Unusual clinico-pathological features of the disease followed by unusual complications.
2. Difficulties in diagnosis.
3. Antemortem digestion of the oesophagus.

1. Unusual Manifestations and Complications

The presence of a solitary acute lesion must indeed be due to an unexplained variation from the normal pathogenesis, which occurs as follows: *Entamoeba histolytica* gains entry to the intestinal wall most frequently in the caecum, with the ascending colon, rectum and rectosigmoid, sigmoid flexure, hepatic flexure, descending colon and transverse colon involved in diminishing frequency. The earliest lesion is a small raised haemorrhagic area, which soon loses its superficial epithelium to become a shallow erosion.2 As the lytic enzymes are produced, so
the organism gains the depths of the mucosal layer, where proliferation occurs. The organisms may then either destroy the muscularis mucosa and enter the submucosa, or spread outwards under the mucosa, coalescing to produce areas of lytic necrosis with relatively healthy areas intervening. In the submucosa, small capillaries and venules may be invaded, and the resultant thrombosis is an important contributing factor in the mucosal necrosis which soon follows. At this stage the picture is that of separate areas of pin-head size ulceration, extending deeply into the larger areas of lytic necrosis, giving rise to the characteristic undermined flask-shaped lesions. Thus far there is purely a chemical digestion, but this is soon followed by secondary bacterial invasion, and now deeper penetration may occur into the muscle layers. Such penetration occasionally occurs with the lytic process itself.

This primary lesion may occur singly or at several closely linked spots, and from this site, the progeny of the primary colonies are squeezed out of the necks of the ulcers, to be carried further down the bowel where the same process is usually repeated at multiple levels. Once this necrotic process is established, the amoebae tend to migrate centrifugally into the adjacent mucosal and submucosal tissue, which thus becomes oedematous. Perforation through the serosa is very unusual at this stage. It appears, therefore, that our case demonstrates such a localized early lesion, without evidence of any distal colonic involvement, but in which the degree of local destruction was advanced to the stage of perforation. One might reasonably expect that with such an acute local process, some degree of upset of bowel function ought to have occurred, for although the full-blown picture of a dysentery is not always seen in acute amoebiasis, diarrhoea without blood or mucus is often a prominent feature even when the lesion is confined to the proximal segment of the large bowel.

The term 'amoebic dysentery' may in fact be misleading, and it should only be applied to cases in which the lesion does involve the distal colon and rectum when blood-stained stools are invariable. The term 'amoebic colitis' is preferable when the lesions are in the proximal large bowel.

The unusual complications in this case were the perforation and cutaneous involvement.

Perforation of intestinal lesions is uncommon, occurring in 1-3% of cases admitted to hospital, but accounts for approximately 20% of deaths from amoebiasis. It may take several forms:

A. Acute intestinal lesions:
   (i) Multiple perforations—usually in fulminating cases, with toxic megacolon, after failure to respond to medical treatment. Duncan and Wilson report a case in which perforation was anticipated, and total colectomy carried out with survival of the patient.
   (ii) Solitary perforation: (a) intraperitoneal, free->peritonitis, or localized->pericolic abscess; (b) retroperitoneal->pericolic abscess or ileus.

B. Amoeboma—central necrosis->rupture
   (i) into adjacent hollow viscera-> internal fistula.
   (ii) free perforation with subsequent adherence to surface by small or large bowel-> internal fistula.

Our case is one of initially localized intraperitoneal perforation, subsequently spreading extra- (retro-) peritoneally, with an insidious course made even more challenging by the solitary nature of the acute lesion. It is in such cases where diagnostic ability must be improved if we are to salvage anything from the critical situation of a spreading retroperitoneal infection in which routine drainage procedures are not always totally effective.

It is of course for the want of such diagnostic acumen that we console ourselves with a therapeutic test of emetine, with or without other anti-amoebic agents. However, when we are faced with a clinical situation of anatomical inaccessibility, superimposed on a basic diagnostic deficiency, we are justified in empiricism especially if there is a very good chance of cure by the correctly chosen drug. Whether a course of emetine, given early, could have benefited this patient is not open to doubt; the final problem was one of uncontrolled bacterial septicaemia.

Cutaneous amoebiasis. Osburn reviewed the literature and found only 61 case reports up to that time. He considers that some of these cases may not have been true E. histolytica infections and quotes Ngai and Fraser who in the first review of the subject in 1933, thought that only 4 of the 30 cases described were authentic. It was suggested by these authors that healthy skin is unable to be invaded by E. histolytica, and they believe that some primary lesion is always present, such as infected wounds of the abdominal wall, peri-anal papillomatia or fissures, or even small ulcerating carcinomas.

The mode of infection of the abdominal wall is most often due to direct invasion secondary to drainage of an amoebic liver or pericolic abscess, or even to appendicectomy, while in several cases there have been no apparent communications of the skin lesion with deeper viscera. Peri-anal skin involvement may be due to implantation of amoebae from infected stools into surface lesions at the anus, or else to direct spread by continuity from an active mucosal lesion in the rectum.

The characteristic features of amoebic ulceration were originally described by Engman and Meleneys in 1931, and this description is still valid. The essential points are as follows:

1. Rapidly spreading ulcerative process with varying degrees of activity along the margins, resulting in an irregular outline.
2. Overhanging edge of epidermis from which some pus can be expressed.
3. Advancing halo of discoloration of the margins of the ulcer.
4. Indolent granulation tissue and debris in the ulcer floor.
5. Extreme tenderness to pressure.

The features of the ulcer in our case fulfilled the above criteria, but unfortunately the diagnosis was not entertained. We had considered the possibility of anaerobic streptococcal infection of the variety described by Meleneys and Meleneys (postoperative synergistic gangrene), a condition which may resemble amoebic ulceration to a remarkable degree, and which may necessitate biopsy for accurate differentiation.

The mode of infection when there is no communication remains speculative; Osburn considered the possibility of blood and lymphatic spread, and stated that direct external contamination is unlikely except in the peri-anal region, where repeated and heavy exposure is more likely to occur.
The response to anti-amoebic drugs is usually satisfactory, but the condition may prove rapidly fatal if allowed to advance without specific drug therapy.

The diagnosis may be established by biopsy and this is suggested as an early routine procedure in all ulcers whose appearance resembles that described above. The unreliability of diagnosis based on laboratory examination of material from the surface of the ulcer parallels that of stool examination in intestinal amoebiasis.

2. Difficulties in Diagnosis

These arise both on the clinical side and in the laboratory. It has already been stated that the bowel symptoms may not be truly dysenteric, which at once tends to dissuade the clinician from making the correct diagnosis. The first essential is an ever-present awareness of the Entamoeba histolytica in all cases. We should recall that the global infestation rate with E. histolytica is about 13%, while the incidence in Africa is 17%, in the USA 10% and in Great Britain between 5 -10%. The number of carriers is potentially great, so that a negative history of known contact with an endemic area is not of clinical importance.

The difficulties in establishing a diagnosis on the basis of identification of the offending organism in the stools are well known and many authors quote low recovery rates of the parasite by routine laboratory methods. Doxiades and Yiotsas\(^\text{7}\) for example give a figure of 10-2% recovery rate of E. histolytica from fresh stool specimens of 2,578 patients who presented with a variety of abdominal complaints including the full blown dysenteric syndrome, while Elsdon-Dew states that 'not only is the amoeba difficult to find, but it is difficult to identify.'\(^\text{11}\) He comments that the task of identification is too often left in the hands of some poorly-trained technician, and points out some of the pitfalls which beset the pathologist. Apart from the problems concerned in making a positive diagnosis, there is equally a tendency to err on the side of false-positive identification, and Elsdon-Dew and Goldman\(^\text{12}\) have discussed this problem. Taking into account the infestation rate as opposed to the actual disease rate, we get some idea of the vast over-all problem facing the diagnostician.

It appears then as if the vast majority of E. histolytica exist as 'commensals' in the bowel, but in order to clarify this point it would be necessary to know whether the colons of this large group do in fact have any superficial pathology which is not severe enough to cause clinical disturbance, and which cannot be detected by routine methods currently at our disposal. Such a series has to my knowledge not been produced. This problem is distinct from that of chronic intestinal amoebiasis described by Candreviotes.\(^\text{13}\)

What are the factors which determine whether an apparently harmless parasite assumes virulence and attacks the bowel wall? Various factors are thought to be operative:

(a) Alterations of the bacterial population in the intestine. Bacteria certainly seem to be responsible for the perpetuation if not the actual production of the pathogenic properties, but the exact mechanism is obscure. The clinical application of this fact is to destroy the bacteria by broad-spectrum antibiotics which in itself is beneficial, but not curative in the acute phase of intestinal involvement.

(b) Nutritional state of the host. Monat\(^\text{14}\) feels that good nutrition in the host undoubtedly minimizes the clinical manifestations, while Elsdon-Dew\(^\text{15}\) feels that the fulminating dysentery seen in the Bantu in Durban is related to their poor diet consisting predominantly of maize products and mineral waters.

(c) Host immunity. This must play a role as witnessed by the fact that natives in certain tropical areas rarely show overt disease, while visitors to these areas rapidly contract an acute illness.\(^\text{16}\)

(d) Spontaneous mutations of avirulent into virulent strains. Unknown factors may promote this change, according to Neal.\(^\text{17}\)

(e) Degree of exposure. As in all infections this may determine the outcome if the dose is excessively large, prolonged or repetitive.\(^\text{18}\)

In view of the low diagnostic yield from routine stool examination, it is obviously desirable to improve this degree of accuracy, and Doxiades and Yiotsas claim to have found positive biopsies in 20.8% of their series of 2,578 cases subjected to rectal biopsy.\(^\text{19}\) Eighty-four percent of these positive biopsies occurred in cases with mild mucosal changes or apparently normal mucosa, and they state that biopsies should therefore be taken even where no ulceration exists. McAllister\(^\text{20}\) concurs in this view, but Chapman\(^\text{21}\) feels that biopsy, best accomplished by a spoon biopsy probe, is only of value at the site of mucosal ulceration.

In considering the migration of amoebae into the oedematous areas away from the actual ulcers, as described above in the discussion of pathogenesis, it becomes apparent that biopsies should always be taken from those oedematous areas even when ulceration does coexist. It has been suggested that the incidence of positive biopsies may be even higher when applied to a series of acute cases only. We must remember that active disease may occur without any active ulceration at all; Faust (quoted by Candreviotes\(^\text{22}\)) failed to note any ulcers in 4 autopsy cases in which he obtained numerous E. histolyticae from the entire length of the colon and rectum.

3. Antemortem Digestion of the Oesophagus\(^\text{23}\) (Fig. 4)

This is a condition to which little or no reference has been made in recent literature. In Muir's Textbook of Pathology,\(^\text{24}\) the following description occurs: 'Antemortem digestion of the oesophagus—peptic oesophagitis—is characterized in its most severe form by haematemesis, and it may be fatal by causing actual perforation of the tube from acute peptic ulceration. It occurs in severe toxic and infective conditions, particularly after operations, and is probably due to the passage of strongly acid gastric contents into the lower oesophagus. The wall of the oesophagus shows brownish discoloration and is in various stages of destruction, and there may be a large comminution with the tissues outside. Although the oesophagus is extensively destroyed, the adjacent part of the stomach wall may be practically unaffected. Recognition of the condition antemortem depends on the histological demonstration of tissue reaction.'

The condition is not uncommon at autopsy,\(^\text{25}\) but the diagnosis is rarely if ever made clinically. Reasons for this are not far to seek; the patient is usually highly toxic and
debilitated, being too weak to vomit the regurgitated acid material. Thus there is no sudden severe retrosternal pain to indicate perforation, such as characterizes the other varieties of spontaneous perforation. Heartburn may be a feature in some patients, as in ours. Mediastinitis may never declare itself because it merely hastens demise from aggravated the lesion and widen the gap in the lower thoracic oesophagus. Hiccough from a different cause may possibly also produce the initial tear in the lower oesophagus diseased by previous acid stasis.

In the event, however, that the condition is diagnosed before death, therapy poses an enormous problem. The only logical procedure consists of cervical oesophagostomy, transthoracic drainage of the site of perforation, and drainage gastrostomy, but this would appear quite unfeasible in the circumstances under which the condition occurs. Local repair of the lesion is totally impractical as the tissues gape and certainly would not take a direct surgical repair. The prognosis thus seems to be universally hopeless.

**SUMMARY**

A case of focal acute amoebiasis is reported with the rare complication of localized intraperitoneal perforation. The difficulties in making the clinical diagnosis are not widely enough appreciated. A negative history, diarrhoeic but not dysenteric stools, and the absence of any abnormal bowel symptoms, contribute to the major clinical diagnostic pitfalls.

Examination of fresh stool specimens is often unreliable, and routine colo-rectal mucosal biopsy should be performed to improve diagnostic accuracy. These biopsies should be taken not only from ulcerated areas, but also from adjacent oedematous mucosa. In the absence of any macroscopic lesion in the distal bowel, biopsy may still be helpful if there is any amoebic activity in the proximal colon or caecum.

Amoebiasis cutis usually occurs as a complication of a visceral amoebic lesion, the possible routes of spread being by continuity along a recent incision into such a lesion, or by blood or lymph spread along vessels connected with such a lesion. Direct external contamination is an unlikely event. However, the skin lesion may occur without any apparent deep visceral involvement. The established lesion may prove rapidly fatal in the absence of specific drug therapy. The major problem in diagnosis is from postoperative synergistic gangrene, and early biopsy of the ulcer margin is indicated for positive differentiation.

Attention is drawn to the rarely diagnosed clinical condition of antemortem digestion of the oesophagus, and the aetiological factors of acid regurgitation and stasis, with severe systemic infection or debility especially postoperatively, are noted. The role of hiccough in the pathogenesis is mentioned. The hopeless prognosis is explained on the basis of the complications of the perforation, superadded to the problem of severe systemic illness, as well as the lack of applicability of the only treatment which seems logical.

I should like to thank Mr. J. A. Myburgh of the Department of Surgery for his very helpful criticism and advice, and Mr. W. Trubshaw to whose unit this case was admitted, as well as Dr. H. van Wyk, Medical Superintendent of Johannesburg Hospital, for permission to publish.

**REFERENCES**