COLONIC PERFORATIONS IN AMOEBIASIS*

E. M. BARKER, F.R.C.S., *Department of Surgery, University of Natal*

Perforation of the colon is an uncommon and serious occurrence in the course of acute or chronic amoebic dysentery. This complication has been variously estimated as occurring between 1 % and 3 % of cases of amoebic dysentery. Ochsner and de Bakey[9] in a large series found a 1.5% occurrence while Wilmot[15] noted a 1.1 % occurrence at Durban. Although forming only a small percentage of the total number of cases, perforations account for a significant proportion of the deaths from amoebiasis, estimated at between 10 % and 20 % in different series.[2, 4, 10, 13] The figures for King Edward VIII Hospital, Durban, are 13%:[15] with a yearly admission of between 2,000 and 3,000 patients with amoebiasis the cases of perforation number about 20 or 30.

Perforation associated with fulminating dysentery is widely regarded as almost inevitably fatal and the condition is usually dismissed as a terminal complication for which treatment is of little avail. Those authors who discuss the possibility of treatment recommend operative intervention as an urgent measure. Recent experience however has shown that the condition is not necessarily fatal, and that with few exceptions operation is not the treatment of choice.

PATHOLOGY

The initial lesion produced by invasion of the bowel wall by *Entamoeba histolytica* is a minute hemispherical elevation in the mucosa, which marks a deeper-lying patch of necrosis. This elevation breaks down to form a pin-head ulcer, with a deeper and more extensive prolongation in the submucosa. As the condition progresses, these ulcers become larger and more profuse and range in size up to several inches in diameter. In typical cases the base of the ulcer consists of the muscular coat of the gut, covered by grey, black or yellow slough, and the mucosa between the ulcers appears relatively normal, although adjacent ulcers may communicate tunnel-fashion on a deeper plane. In the vast majority of cases the original lesion occurs in the caecum, followed in order of frequency by the ascending colon, recto-sigmoid, hepatic flexure, splenic flexure, descending colon, and transverse colon. It would appear that the sites most frequently involved are those where stasis of large-bowel contents is normally most marked. Mild or severe secondary infection of these ulcers is inevitable.[2,3,8] Bleeding occurs as a result of direct destruction of vessel walls by the histolytic ferment produced by the amoeba.

The untreated case may progress to chronicity, and will take on either a generalized or a localized development. In the generalized type there is gross thickening of the wall of the whole colon, with multiple ulcers present in various stages of formation, and of varying size and depth. Some healing of ulcers may take place, but further ulceration, chronic inflammation and thickening of the bowel wall will always occur. This type of lesion may, though rarely, result in unexpected perforation of the bowel, which is not necessarily associated with an acute episode of dysentery. When such a perforation occurs it is commonly extraperitoneal, or walled off by previous adhesions, and leads therefore to pericolic abscess formation,[5] but it may rupture directly into the general peritoneal cavity.

The localized form of chronic amoebic enteritis, on the other hand, results in the formation of an amoebic granuloma or amoeboma. This condition usually presents as a painful tender abdominal mass in a patient who may or may not have a past history of dysentery. Macroscopically, a firm nodular granulomatous mass is found, with multiple small abscesses in the centre. There is mucosal destruction with the formation of a deep irregular ulcer having a typical sloughing base. This form of chronic amoebiasis may also proceed to perforation.[6] Warning of impending perforation is sometimes given by an acute exacerbation of pain and local tenderness at the site of the amoeboma.

In the commoner acute form of amoebic dysentery the ulcerative process may be limited by the muscle layer, as already described, and may either heal or become chronic. On the other hand it may progress to an acute fulminating necrotizing enteritis. Necropsy of these cases shows a darkly coloured large bowel covered with fibrinous exudate, with some free fluid in the peritoneal cavity. The wall of the large gut is thickened, soggy and, to use Sir Leonard Rogers' description, 'of the consistency of wet blotting paper'. It is often possible, even with the gentlest handling, to detach a piece of the wall of the colon. The mucosa is studded from caecum to rectum with ulcers, and in some cases total denudation of the mucosa is found to have occurred. The bowel contains multiple black sloughs. This process often involves the appendix and may extend for some inches up the ileum. Wilmot[15] quotes one fatal case in which a relatively normal-looking mucosa was studded with multiple ulcers set on a base of serous coat only. In its most advanced form this type of dysentery sometimes leads to massive gangrene of extensive sections of the large gut, with profuse haemorrhage.

A particularly noticeable feature of this fulminating type of dysentery is the frequent finding of fibrinous exudate and

* Presented at the South African Medical Congress, Durban, September 1957.

free fluid, sometimes associated with pericolic abscesses, about macroscopically unperforated bowel.[2, 5] This would seem to indicate that migration of amoebae and secondary invaders occurs across a bowel wall which no longer offers a normal barrier to infection. Thus generalized or localized peritonitis sometimes occurs in fulminating amoebic dysentery without any frank perforation.

CLINICAL PICTURE

The clinical picture of acute fulminating amoebic dysentery is reminiscent of that of bacillary dysentery. Twenty or thirty stools are passed each day, consisting almost entirely of blood and mucus, with occasional black mucosal sloughs and, rarely, whole casts of large bowel mucosa. However, even in advanced cases, the stool may consist of watery exudate only with no blood or mucus.[15] Abdominal pain, seldom observed in milder forms of amoebiasis, is constantly present and tenderness is of a severity found only with generalized peritonitis. In fact these patients are suffering from a true generalized peritonitis even before macroscopic perforation occurs. Rigidity, even localized, is seldom found, for the patient is too ill to manifest this sign. In the early stages the abdomen is soft and doughy, there is progressive abdominal distension, and free intraperitoneal fluid may occasionally be clinically detectable. Rectal examination reveals marked tenderness in the pouch of Douglas and often ulcers are palpable as granular areas low in the rectum. Intractable hiccup may occur, and is regarded as a grave prognostic sign.[15]

The toxaemia which is so marked a feature in these cases of fulminating dysentery is due in part to the dehydration and electrolyte imbalance consequent on the severe diarrhoea and in part to the ileus which usually occurs, even in the absence of frank perforation. It must be recognized that, although the patient continues to pass blood and mucus, a state of paralytic ileus exists and must be treated.

When macroscopic perforation occurs during the course of fulminating amoebic dysentery the clinical picture differs little from that already described. The patient becomes, if possible, a little more ill, the abdomen more distended and tender, and ileus, if not already established, now becomes evident. Rigidity is as infrequent as it is in cases of fulminating dysentery. The only positive signs of perforation are the presence of much free fluid in the peritoneum and X-ray evidence of free intraperitoneal gas. Clinical assessment of the presence of free gas in such cases is notoriously unreliable, and a lateral radiograph of the patient's abdomen taken in bed in the supine position is necessary. This should not unduly disturb the patient. Despite the presence of ileus, stools continue to be passed with undiminished frequency.

TREATMENT

In considering the treatment of perforation of the amoebic colon a clear distinction must be drawn between the 3 types described above, viz. those occurring extra-peritoneally or into a peritoneal space already walled off by adhesions, giving rise to pericolic abscesses; secondly, true intraperitoneal perforations of amoebic granulomata or of acute amoebic ulcers, these perforations not being associated with severe dysentery; and lastly the intraperitoneal perforations of bowel which occur during the course of fulminating dysentery.

Treatment of a pericolic abscess should be initially conservative if this is at all possible. This will allow time for the causative amoebic process to be adequately controlled by emetine and for the secondary invading organisms to be dealt with by antibiotics. If operation is undertaken too early there is grave danger that the patient's relatively quiescent amoebiasis will flare into a dysentery that may be difficult to control. There is also a risk of the formation of faecal fistula, which tends to be intractable, and of the development of amoebiasis cutis, which even with emetine and broad-spectrum antibiotics is sometimes a terrifying complication. The surgeon's hand, however, will be forced if the abscess enlarges rapidly while under treatment or threatens to rupture into the general peritoneal cavity. Where conservative measures are possible the approach to treatment is similar to that employed in the management of an appendix abscess—absolute bowel rest achieved by sedation and repeated gastric aspiration, controlled intravenous therapy, and the use of emetine and intravenous oxytetracycline. Surgical drainage is almost invariably indicated eventually, and should be undertaken as soon as the amoebiasis is well controlled.

Case 1.

V.R., a 27-year-old African female, was admitted to the gynaecological ward on 10 January 1957, with a diagnosis of pelvic abscess. Her complaint was mainly of 6 weeks' pain in the right iliac fossa. E.U.A. revealed a possible early pregnancy and she was discharged for further observation. A few days later (on 30 January) she was re-admitted with the same pain still present. Pregnancy was now confirmed by a frog test and the diagnosis of ectopic pregnancy, torsion of ovarian tumour, or pelvic infection associated with pregnancy, were considered. Soon after admission a vague mass became palpable in the right supra-inguinal region. Her complaints failed to respond to a long course of penicillin and streptomycin. By 22 March her pain was much worse, and the supra-inguinal mass was much more evident. An adjacent inguinal swelling had also developed, and a diagnosis of inguinal and iliac adenitis with suppuration was made. The iliac abscess was drained, with immediate improvement in the patient's general condition. Four days after operation her condition began again to deteriorate and on 1 April her operation wound was discharging thin pus. Two minute areas of skin necrosis were observed at the edge of the wound and this observation raised the first suspicion of amoebiasis. On close questioning the patient gave a history of amoebic dysentery many years ago, but denied any disturbance of bowel function in the last few years. The diagnosis was proved by the finding, in material aspirated from the depths of the wound, of free forms of *E. histolytica*. At no time were amoebae or cysts observed in pus swabs taken from the spontaneous discharge from the wound. A full course of emetine and tetracycline resulted in improvement. This was, however, extremely slow and weeks elapsed before recovery was sufficiently advanced to permit discharge from the ward. This case illustrates some of the dangers resulting from failure to recognize the true nature of amoebic pericolic abscesses.

Perforation of an amoebic granuloma, or of an amoebic ulcer which is not associated with acute dysentery, presents as a sudden abdominal catastrophe. The diagnosis is seldom made pre-operatively, because the history and clinical examination do not provide sufficient evidence upon which to differentiate this condition from other intra-abdominal catastrophes; early operation must be undertaken as for any 'acute abdomen'. Provided the causative pathology is recognized at operation, and energetic anti-amoebic treatment instituted immediately, these cases respond well and have a prognosis no worse than perforations of the colon from other causes. Direct suture or exteriorization are the procedures of choice, depending on the site of perforation. Excessive mobilization of unhealthy gut is of course to be avoided, and unless exteriorization is technically simple, direct suture

with drainage is to be preferred. The condition of the bowel wall in this type of case is generally good and no great technical difficulty in suturing need be anticipated.

The most difficult type of perforation to treat and the one with the poorest prognosis is *that occurring during the course of fulminating dysentery*. Most authors regard the condition as inevitably fatal and make no recommendations regarding treatment.[1,4,7,8,11-13] Some who do consider the possibility of treating the condition advise immediate operation.[2, 9, 10, 14] However, Wilmot[15] and Oschner and DeBakey[9] are of the opinion that operation accomplishes nothing and merely makes worse the condition of a gravely ill patient. Several different operative procedures have been described,[9, 11, 14] each of which merits examination in the light of what is known of the pathology of this condition:

1. *Exteriorization of Affected Segments of Bowel.* Any attempt at this may leave the operator with a segment of bowel very radically exteriorized and completely detached from the rest of the colon. Further, in the majority of cases, the widespread involvement of the large bowel would necessitate exteriorization to an extent approaching total colectomy, a suggestion which experience of this serious condition rules out.

2. *Suture of Perforations.* Because of the nature of the bowel wall, the more attempts that are made at suturing a perforation, the more numerous will the perforations become. The gentlest attempt to pass an eyeless needle through the bowel wall may detach as much as a square inch of tissue.

3. *Defunctioning Ileostomy.* This procedure, first recommended by Theron,[14] was devised to defunction the colon, limit peritoneal contamination, and permit healing of the perforation. However, in view of the fact that ileus is invariably established, if not before perforation, then at a time that perforation occurs, this in itself 'defunctions' the colon as well as any ileostomy; an ileostomy cannot prevent dissemination through the peritoneum of material already in the large bowel. At the stage when perforation occurs, no further small-bowel content is entering the colon and ileostomy can only result in added fluid and electrolyte loss.

4. *Simple Drainage of the Peritoneal Cavity.* It is becoming more commonly recognized that the insertion of a tube drain into the peritoneal cavity can seldom hope to keep empty an area of more than a few square inches around itself. Furthermore, necropsies performed on fatal cases of perforation rarely show much faecal contamination of the peritoneum. By the time the perforation occurs the colon is empty of faeces and immobile. The exudate and blood which it contains run away as stools with practically no peristaltic assistance. The additional contamination resulting from gross perforation into an already infected peritoneum consists of blood and exudate only. Drainage of these cases should then, logically, be applied to all cases of fulminating amoebic dysentery with marked tenderness and any detectable quantity of free fluid, since these patients indubitably suffer from peritonitis. Further, to be effective, drainage should include complete toilet of the peritoneum, and no case of fulminating dysentery can be rendered fit enough to stand up to this procedure.

It would seem then, that operation in this type of case is both useless and dangerous. The majority of patients die of 'toxaemia,' which usually means dehydration and electrolyte imbalance, leading to peripheral vascular collapse. Toxic absorption and so called 'septic shock' may be involved as contributory causes, and must be treated concomitantly with the fluid and electrolyte disturbance. The primary aetiological agent, the amoeba, also requires treatment. The alternative method of treatment to be suggested is directed to the control of dehydration, shock and infection, and is based on the underlying pathology where the perforation of the immobile and empty colon is only an extension of a process, the lethal factors of which are already in operation. These concepts simplify the problems involved in treatment, but they by no means reduce the care and vigilance required to save the patient's life. 'Toxaemia' and 'shock' can be overcome by adequate fluid and electrolyte replacement, controlled clinically and by laboratory aids. In the first 24 hours 4–8 litres are required, at least half of which should be isotonic saline to replace the salt lost. At least 500 ml. of blood are required daily, and more if the haemoglobin and haematocrit readings indicate a marked anaemia. The blood pressure, which is usually below 100 mm.Hg systolic, is a valuable indication of the response to the intravenous fluids and l.-noradrenaline should be used if the pressure remains at this low level for some hours. Oxytetracyline must be given by means of intravenous infusion, and emetine should always be used except in the presence of myocardial disease. This is important, for emetine is still the most reliable amoebicide available and failure to use it may result, in those cases who survive the acute episode, in a true chronic amoebic peritonitis.[15] Ileus is treated by sedation and by effective gastric or upper intestinal aspiration.

Where these measures have been fully applied there have been gratifying recoveries of patients who otherwise, it is believed, would have died.

Case 2.

J.M., an African male of 36 years, was admitted on 22 November 1955, with a 6-day history of diarrhoea and blood and mucus in the stools. He had about 10 stools per 24 hours at the time of admission. His abdomen was noted at this time to be vaguely tender. By 24 November he was extremely ill, and his abdomen was distended and markedly tender. A provisional diagnosis of colonic 'leak' was made, but fluids were still administered freely by mouth throughout this day. By the following day he was severely shocked, and at 4.00 p.m. an infusion of intravenous glucose in distilled water was started. He died 6 hours later, and post-mortem revealed multiple ulcers of the colon with areas of necrosis of the gut wall. In the light of the post-mortem findings, it would appear that little could have been accomplished by surgery. It is specifically in this type of case that intensive therapy to counteract dehydration and toxaemia, together with anti-amoebic treatment are indicated.

Case 3.

G.T., an African male of 45 years, was admitted on 4 January 1955. He had had two previous attacks of dysentery, one in January 1954, and a second in July 1954, both treated with full courses of emetine, and both having responded well to this treatment. His present attack had lasted 7 weeks at the time of admission. The day after admission his condition deteriorated and signs of generalized peritonitis were obvious. There was clinical evidence of free gas in the peritoneum. Gastric aspiration and intravenous therapy with electrolyte solution and tetracycline were instituted. By 10 January he was sufficiently well for fluids to be given by mouth. These were not well tolerated, and on the following day gastric aspiration was again used, with good effect. By 20 January he was relatively well and all therapy was discontinued. However, on 25 January his condition again deteriorated and a small abscess became evident in the right flank. This was aspirated and a small quantity of malodorous pus accompanied by gas was evacuated. By 7 February he became very debilitated, and his abscess was re-aspirated, with findings similar to those obtained at the first aspiration. Despite blood trans-

fusions and further intravenous tetracycline, he died on 14 February. Permission for necropsy was not obtained.

The cause of this patient's death, occurring some weeks after he had been successfully tided over an acute intraperitoneal perforation of fulminating amoebic dysentery appears to have been the presence of one or more pericolic abscesses resulting from his earlier bowel perforation. Although surgical drainage of his abscesses may have been indicated once the acute phase of peritonitis was safely passed, and may have altered the outcome, this case illustrates that a fatal termination may occur many days or weeks after successful treatment of the acute phase of perforation. It is imperative that amoebic perforations of the bowel should be treated by physicians and surgeons in full collaboration, for the condition cannot be regarded as either entirely 'medical' or entirely 'surgical'.

Case 4.

P.M., an African male aged 14 years, was admitted on 18 July 1955. He had suffered for the past 4 weeks from diarrhoea with frequent stools containing blood and mucus. On admission, his general condition was good and his abdomen was recorded as being diffusely tender, though soft. Rectal examination revealed some tenderness in the recto-vesical pouch. A course of emetine was started forthwith. On 23 July he complained of increasingly severe abdominal pain, and on examination was found to be severely shocked, with a fast feeble pulse and a temperature of 101·5° F. His abdomen was extremely tender and guarded, with release tenderness, and absent liver-dullness to percussion. There were no bowel sounds present. X-ray of the abdomen revealed the presence of free gas in the peritoneal cavity, and of fluid levels in the small bowel. Gastric aspiration and intensive intravenous fluid and antibiotic (oxytetracyline) therapy were started immediately. The patient's response, though slow, was satisfactory, and by 30 July he was considered to be out of danger. However, on 2 August his temperature again rose, and it continued to 'spike' until, on 9 August, a pelvic abscess became evident. In view of the amoebic basis of his condition, operation was delayed, and the patient's condition was carefully watched. His temperature gradually settled on conservative measures, and his abscess became progressively smaller. He was discharged from hospital as cured on 31 August 1955.

STATISTICAL

During the period 1 January, 1955, to 31 July, 1956, approximately 3,000 cases of amoebic dysentery were admitted to King Edward VIII Hospital. These were exclusive of those classified under 'amoebiasis' (i.e. hepatitis, asymptomatic cyst-passing, and other non-dysenteric complications). Of these cases of amoebic dysentery, a random selection has been made of 1,000 cases which have been reviewed in some detail, with particular reference to the complication of perforation of the bowel. More than half the patients (534 cases) were noted to have abdominal tenderness when first examined, or to develop this sign during the course of treatment. Those with only moderate tenderness were usually not very ill, while those whose tenderness was recorded as 'acute' or 'severe' or 'with guarding and rigidity' had in general, dysentery of a more fulminating type. Exceptions were found in cases of elderly patients or babies dying from amoebic dysentery without or with only slight abdominal tenderness. In addition to the cases showing tenderness there were cases classified as perforations, the diagnosis being most often made clinically on the basis of severe shock, ileus, marked abdominal tenderness and the presence of free fluid and gas in the peritoneal cavity. In only 3 cases was the diagnosis of perforation proved beyond doubt by X-ray or necropsy. The numbers of these cases are shown in Table I.

TABLE I. CASES WITH ABDOMINAL TENDERNESS

		%
Moderate Tenderness	433	43·3
Severe Tenderness	82	8·2
Perforation of Bowel	19	1·9
Proved	3	
Clinically diagnosed	16	
Total	534	

There were in all 74 deaths, a mortality of 7.4%, the immediate causes of which are set out in Table II. Permission for necropsy was not often obtained, which accounts for the large number classified as 'exact cause unknown'. Not all of the other cases dying of 'known causes' were proved at necropsy, but they are included in their respective categories if the clinical findings were definite enough for a diagnosis to be made. Of the 74 deaths 14 cases (19.2%) were found to be due to perforation of the colon, the true figure probably being higher, as the 'unknown causes' would be expected to have included some perforations. Of the 6 cases in which death took place from massive necrosis of the large bowel, only one presented clinically as a case of perforation, all the others being recorded as presenting only moderate to severe abdominal tenderness. The one case presenting clinically as a perforation has not been included in any table among cases of perforation.

TABLE II. IMMEDIATE CAUSES OF DEATH

		%
Acute stage of Perforation	12	16·3
Chronic Peritonitis following Perforation	2	2·9
Intestinal Haemorrhage	3	4
Massive Necrosis of Bowel (all proved at necropsy)	6	8
Other causes (Pneumonia, Cardiac failure, etc.)	14	18·8

Those 14 cases which died as a result of perforation are classified in Table III. Those who received no intravenous therapy died almost on admission, and no time was available for resuscitation. Of those classified as receiving 'some intravenous therapy' some died too soon after admission to permit of adequate treatment, but in many cases the full range and possibilities of available therapy (e.g. blood transfusion and intravenous oxytetracyline) were not for some reason used. Of those 4 cases who received what is considered to be the full routine of therapy for this type of case and who nevertheless died, 2 died without recovering from the acute phase of shock associated with perforation, and 2 died of chronic peritonitis after having recovered from the acute episode — one dying 4 weeks and one 5 weeks after perforation.

TABLE III. DEATHS ACCORDING TO INTRAVENOUS THERAPY

Received no intravenous therapy	2
Received some intravenous therapy	8
Received full routine of therapy as described earlier	4

Of the 19 cases of perforation encountered in this survey, 14 died as already indicated, and 5 recovered after intensive conservative treatment. Of these 5, 2 were proved by X-ray to have perforated. The remainder were clinical diagnoses only. This represents a recovery rate of 26%, a figure that can certainly in our experience be further improved. A patient who is not moribund on admission and who survives up to

24 hours should have considerably more than a 26% chance of recovery, this latter figure being the survival rate in this series for all cases of perforation.

This serves to indicate that the prognosis of this complication, in the absence of massive and frank gangrene of the bowel, is not as gloomy as has hitherto been believed, and that intensive and energetic fluid replacement with broad-spectrum antibiotics and emetine is the treatment of choice in preference to surgical interference.

SUMMARY

The pathology of intestinal amoebiasis is briefly surveyed with particular reference to the changes preceeding and accompanying peritonitis and perforation.

The signs and symptoms of these complications are discussed and emphasis is laid on the pathology, clinical presentation and treatment of perforation occuring with fulminating dysentery, as against that occurring with quiescent or chronic dysentery, or amoeboma.

The significance of pericolic abscess is also discussed, together with the approach to treatment of this complication; its underlying pathology should be recognized and anti-amoebic treatment instituted in good time.

The views of different authors on the treatment of perforation and our conclusions based on some 2,000 cases annually of amoebic dysentery are presented.

A random selection of 1,000 cases of amoebic dysentery, encountered in 18 months, has been reviewed and typical case reports are also presented.

It is argued that operative treatment has rarely, if ever, any place in the management of colonic perforations occurring during the course of fulminating amoebic dysentery. Emphasis is laid on the possibility of saving a greater number of these cases by means of treatment with intravenous fluid and electrolyte therapy, blood replacement and antibiotic and anti-amoebic therapy. This form of treatment, to achieve success, demands great care and attention to detail.

Bowel perforations due to amoebiasis but not associated with fulminating dysentery should, like bowel perforations due to any other causes, be treated by operation. The presence of amoebiasis introduces special risks and complications, the possibility of which must be recognized. They can be forestalled by adequate anti-amoebic treatment.

I wish to thank Prof. A. E. Kark, F.R.C.S., for his help and encouragement in the preparation of this paper. My thanks are also due to Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, for permission to publish case reports and analyses of hospital records.

REFERENCES

1. Andre, M. and Cornet, L. (1955): Med. Trop. (Marseille), **15**, 143.
2. Craig, C. F. (1944): *Etiology, diagnosis and treatment of Amoebiasis*. Baltimore: Williams and Wilkins.
3. Elsdon-Dew, R. (1946): S. Afr. Med. J., **20**, 580.
4. Howe, P. (1945): Surg. Gynec. Obstet., **81**, 387.
5. Hayman, L. D. and Weaver, T. H. (1953): Gastroenterology, **23**, 97.
6. Manson-Bahr, P. (1951): *Manson's Tropical Diseases*, 13th ed. London: Cassell.
7. Mackie, T. T., Hunter, G. W. and Worth, C. B. (1945): *Manual of Tropical Medicine*. Philadelphia: Saunders.
8. Naunton-Morgan, C. (1947): *British Surgical Practice*, vol. 1. London: Butterworth.
9. Oschner, A. and DeBakey, M. (1952): Int. Clin., **1**, 68.
10. Rives, J. D., Heibner, W. C. and Powell, J. L. (1955): Surg. Clin. N. Amer., **35**, 1421.
11. Rogers, L. and Megaw, J. W. D. (1946): *Tropical Medicine*, 5th ed. London: Churchill.
12. Stewart, I. (1953): Proc. Roy. Soc. Med., **46**, 613.
13. Strong, R. (1944): *Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases*, 7th ed. Philadelphia: Blakiston.
14. Theron, P. (1947): Brit. Med. J., **2**, 123.
15. Wilmot, A. J. (1949): *Clinical Manifestations of Amoebiasis in the Bantu*, M.D. Thesis, Univ. Oxford.