AMOEBIC LIVER ABSCESS

IN

NATAL AFRICAN CHILDREN

Being a thesis submitted for
the Degree of Doctor of Medicine
in the University of Cape Town

By

JOAN N. SCRAGG

July, 1961

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Plate 1. Reproduction from Virchows Archives showing the title of a paper published by Lösch from Russia in 1875. Also shown are Lösch's drawings of the amoebae described in this paper

XVII.

Massenhafte Entwickelung von Amöben im Dickdarm.

Aus der Klinik von Prof. E. Eichwald.

Von F. Lösch,
klinischem Assistenten und Privatdocenten der inneren Medicin in St. Petersburg.

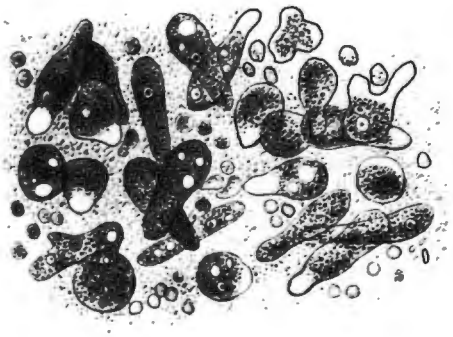
(Hierzu Taf. X. Fig. 1—3.)

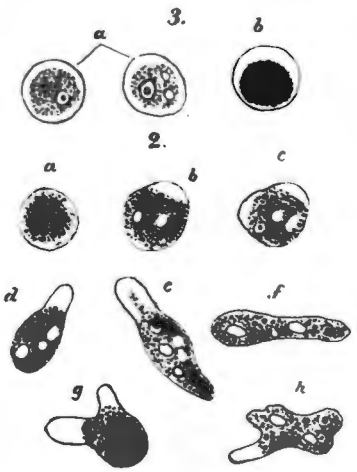
Im Dickdarm des Menschen sind Amöben bisher so selten beobachtet worden, dass selbst R. Leuckart [1]), dieser gründliche und vielerfahrene Forscher auf dem Gebiete der menschlichen Parasiten, an ihrem Vorkommen daselbst zweifelt und den einzigen bisher von Lambl [2]) veröffentlichten Fall als Verwechselung pathologischer zelliger Gebilde mit Amöben ansieht. Der vorliegende Fall, den ich im Verlaufe mehrerer Monate in der Klinik von Prof. Eichwald zu beobachten Gelegenheit hatte, bietet daher grosses Interesse dar.

J. Markow, Bauer aus dem Archangelschen Gouvernement, 24 Jahre alt, wurde den 3. November 1873 in die Klinik aufgenommen.

Seiner Aussage nach kam er zum ersten Male im Sommer 1871 nach der Hauptstadt, um Arbeit zu suchen und erkrankte hierselbst an einem hartnäckigen, heftigen Durchfall, der mehrere Monate anhielt und ihn schliesslich dermassen entkräftete, dass er nicht im Stande war sich selbst im Bette aufzurichten. In

[1]) Die menschlichen Parasiten von R. Leuckart. 1863. S. 140.
[2]) Aus dem Prager Josef-Kinder-Spitale in Prag. I. Theil. S. 262.





INTRODUCTION

A great difference of opinion still exists with regard to the incidence of amoebic dysentery in infants and young children. While many authors have stated that amoebic dysentery does not occur, or is a medical curiosity in childhood, others have stressed that this is not so and that it should be kept in mind in investigating diarrhoea in infants and children even in temperate zones.

By the term amoebiasis one assumes that infection with Entamoeba histolytica has occurred. This infection may not have given rise to symptoms or signs. Hoare (1950 and 1952) stated that "Although E. histolytica is a pathogenic parasite, capable of invading the tissues of the host with the production of lesions and clinical symptoms of disease, in most cases it does not manifest any evidence of virulence, for in up to 90% of persons harbouring this parasite the infection is symptomless and the host is a carrier. In most carriers the amoebae live in the lumen of the gut as harmless commensals ..."

From many of the reports of amoebiasis it is impossible to determine whether an author is discussing asymptomatic amoebiasis or amoebic dysentery. This failure to correlate the finding of E. histolytica in the stools of children with the actual number with dysenteric signs has led to much confusion.

Many authors still believe that the complications of amoebic dysentery are much less frequent in children than in adults.

Faced with the problem of a high incidence of amoebic infection and its complications in the African child in Durban, I was prompted to search the literature on this subject in order to compare the incidence, complications and mortality as found in Natal African children, with the disease in childhood elsewhere and to compare and contrast the findings in children with those in adults.

HISTORICAL REVIEW

It was Lösch in Russia who in 1875 identified amoebae in the stools of a young peasant from Archangel. He was able to infect a dog with these amoebae (which he named "Amoeba coli") and at post-mortem examination of the dog there was ulceration of the large intestine with amoebae in the ulcers. The patient when he died showed a similar condition of ulceration with amoebae present in the large intestine.

Some modern workers still cite Lambl as the discoverer of the intestinal amoeba of man in 1859. According to Dobell (1919) the paper published by Lambl in 1859 made no mention of amoebae. Dobell stated that the work in which amoebae were mentioned appeared in 1860 and probably few people have really seen this work, which is very difficult to obtain. Dobell believed that the small "amoebae" described by Lambl in the intestine of a child who died of enteritis were in fact small degenerating individuals of the flagellate Trichomonas hominis.

Dobell (1919) quoted Koch as furnishing the next discovery of importance which was published in 1887. Koch had occasion in 1883 to carry out autopsy examinations on 5 cases of dysentery in Egypt, 2 of them complicated by liver abscess. Sections of the intestinal ulcers of 4 and of the liver abscess in 1 revealed the presence of amoebae.

In the same year of 1887 Pfeiffer saw amoebae similar to those

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described by Lösch in the stools of children suffering from dysentery and Kartulis also found amoebae in liver pus. This latter author had earlier (1886) reported on the finding of amoebae in 150 patients with dysentery in Egypt.

Osler in America, in 1890, discovered amoebae in liver abscesses and this led Councilman and Lafleur in 1891 to publish a masterly study on "amoebic dysentery" and "amoebic abscess of the liver" which terms they introduced. They gave the organism the name of "Amoeba dysenteriae" and it was not until 1903 that it received the name "histolytica" from Schaudinn.

According to Councilman and Lafleur (1891), Leukart (1880) reported that Sonsino in Cairo had found large numbers of amoebae in the intestinal mucus of a child who died of dysentery.

It is interesting to note that in some of the earlier references to the disease in children, the diagnosis was made because of the presence of the complication of liver abscess. Thus probably the earliest reference in the literature is that of Brown who in 1824 reported "a case of great enlargement, suppuration and ulceration of the liver in a child aged 12 months". This occurred in a little Negress in Cuba, following dysentery.

Reviewing the literature makes it obvious that amoebiasis is a highly controversial subject. The disease in childhood appears to be the subject of even greater controversy.

INTESTINAL AMOEBIASIS IN CHILDHOOD

Incidence of Entamoeba histolytica infection

Many surveys among children in different parts of the world had revealed that infection with E. histolytica (not necessarily producing dysentery) was common in certain areas. Willets (1914) had reported an incidence of 25% positive stools in Philippine children.

Dobell (1921) in a study on the incidence of intestinal protozoa in the inhabitants of Britain, found that there was an appreciable incidence of E. histolytica in the children examined, with for example the following variations: 1.6% in Leeds, 3.1% in Bristol and 5.3% in Sheffield.

In 1930 Meleney, and later in 1931 Milam and Meleney, conducted an epidemiological study of amoebiasis in Tennessee showing the incidence of infection to be very high (38%), appearing first in the 2 year old group. However, no correlation could be found between the occurrence of dysentery and the harbouring of E. histolytica.

In southwestern Virginia, Faust (1930) found that 16.5% of the age group up to 5 years harboured E. histolytica. Later in 1931 he pointed out the value of diagnosis of cases in which only cysts are passed as contrasted with those in which active trophozoites are seen. His study suggested a racial tolerance to the parasite, as in the most heavily infected communities cysts were more common than trophozoites

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and vice versa. Examination of stools from children in orphanages in New Orleans showed that 55.5% were infected, but cysts only were recovered from these stools.

Later in 1933 this same author reported on the parasitic findings in a cross section of New Orleans patients with especial reference to children. Of those infected with E. histolytica only about 10% manifested symptoms of dysentery or chronic diarrhoea. It was found that there was an appreciable incidence of infection in the 1 to 5 year old group, but maximum incidence was not reached till early adult life. However, by comparison with a series of 2,000 patients in Panama river villages, the incidence of E. histolytica rose to a peak (30%) in the first decade, then declined only to reach a second peak at about 40 to 45 years. In the most heavily infected group of villages a peak of 90% was reached before the age of 16 years. This author concluded that E. histolytica although causing manifest clinical symptoms in only a small proportion of those infected, is a grave menace particularly in children who constitute a distinctly non-immune element of the population.

From all over the world reports have appeared of the incidence of E. histolytica in the stools of children.(Macchiavello 1938, Bustos 1938, Ivanhoe 1943, Beltran 1944, Miller 1947, Miller et al. 1947, Sigalas et al. 1950, Rojas 1950 and 1953, Anand 1954, Neghme 1954, Neghme et al. 1954a and 1954b, Young 1955, Roman 1955, Pessoa 1957, and others).

These stool surveys did not necessarily correlate the finding of E. histolytica in the stools with the actual number of children suffering from dysentery.

Amoebic dysentery

Among the earliest references to amoebic dysentery in children were those of Cahen (1891), Strong (1898) and Harris (1898). These authors regarded the disease as rare at an early age.

In 1901, Amberg, in an important contribution, gave a review of the early literature on amoebic dysentery in children. He had studied 5 cases under the services of Osler in the Johns Hopkins Dispensary between 1900 - 1901. This author stressed that amoebae might be found only after repeated stool examinations.

An editorial in the Journal of the American Medical Association in 1902 commented on the infrequency of this disease in childhood.

In 1903, Futcher, also reporting from Johns Hopkins Hospital on a study of 119 cases of amoebic dysentery, pointed out as of special interest the rather frequent occurrence of amoebic dysentery in young children. Eleven (9.4%) of the cases occurred in the first decade, 6 of these being between 1 and 5 years. This author commented, "It should teach us to keep in mind the possibility that a chronic dysentery or diarrhoea in young children may be of amoebic origin."

From this time onwards conflicting views on the incidence of amoebic dysentery in childhood were published.

De Oliveira (1905) pointed out that although previous observers regarded amoebic dysentery as unknown in infants, it occurred not infrequently. On the other hand Bobillier (1913) stressed the rarity of the condition in children under 2 years of age in Brazil.

Further attention was drawn to amoebic dysentery in childhood by Debuys (1914a, 1914b), Cannata (1916 and 1923), Spolverini (1922), Maggiore (1923) and Petzetakis (1925).

Up until the mid 1920's most of the literature had come from tropical and sub-tropical countries. However, it was now becoming apparent that infection with this parasite was to be found even in temperate zones, as shown by the reports of Young (1921) from England and Perrin et al. (1923) from France.

Despite the fact that the literature contained numerous references to this condition in children, it was still considered by many to be unusual in childhood. Musser (1927) believed that it was a disease of adult life and was in fact a clinical curiosity in childhood. Another author holding this view was Zaturjan (1935) who regarded the condition as rare, of a more benign course and free from complications in children.

From areas of endemic amoebiasis reports of dysentery in

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children continued to appear (Pardo 1926; Gerbasi 1927 and 1931; Perry et al. 1929 and 1932; Archer 1932; Owen et al. 1934 and Dendale 1934).

That amoebic dysentery might even be found in breast-fed infants was stressed by Trabaud (1928 and 1935), Chueca (1928) and Izar (1932). This latter author quoted Tarro (without reference or year) as recording the ages of 30 infants dying of amoebiasis in whom necropsies were done; 21 of these were under 6 months, 4 of them being only 16, 18, 21 and 23 days old respectively.

Manson-Bahr (1932) believed that ulcers did not develop for a year, possibly longer, after oral infection with E. histolytica. Basing his conclusions on this belief and the fact that in his long experience he had never come across the disease under the age of 5 years, he suggested that Izar's cases in very young infants were in fact bacillary dysentery. He pointed out that large macrophages, abundant in the stools of bacillary dysentery, closely simulate and are often mistaken for E. histolytica. However, there appears to be no foundation for this belief of Manson-Bahr's regarding the interval between oral infection and the manifestation of signs from ulceration of the bowel.

As late as 1934, Craig also stated that amoebic dysentery occurred rarely in children, not because of any inherent insuscept-

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ibility, but because of the lesser chance of exposure to the infection.

Numerous reports of symptomatic disease caused by E. histolytica now began to appear in the literature (Gillot et al. 1936, Sabri et al. 1937, Howell and Knoll 1941, Montanes 1943 and 1949, Beltran et al. 1944, Costa Mascaro 1945, Raghaven 1945, Delthill 1946, Reddy et al. 1948, Cardelle et al. 1948, D'Antoni 1948 and 1949, Adams et al. 1949, De Silva 1949, Sarrouy et al. 1950, Goldenberg 1952, Treuer 1952, Suarez et al. 1952, Crawford 1953, Anderson et al. 1953, Castillo 1953 and 1955, Riopedre 1953, Debbas 1953 and Vidal Freyre 1953 and 1954. Most of these authors stressed that amoebic dysentery in children was not as rare as had been formerly thought.

It is of interest to note the report of Repetto (1952) of an infant who developed dysentery with blood and mucus in the stools at the age of 46 hours. From the stools of this neonate E. histolytica were isolated on the twenty-third day of life. Other reports of proved amoebic dysentery in very young neonates are those of Fonzo (1935) and Weiser (1953).

Conclusions

There is no doubt that infection with E. histolytica is world wide and that many children as well as adults harbour this parasite without signs or symptoms of disease being manifested. However, study

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of the literature reveals that much controversy exists about the frequency with which amoebic dysentery occurs in children. The picture is further confused through failure of some authors to distinguish between symptomatic and asymptomatic amoebiasis.

In many parts of the world where amoebic dysentery is common in adults (Egypt, other parts of North Africa, the Middle and Far East, Spain, Italy, Mexico and Cuba) it is also commonly found in children. While dysentery in children appears to occur in other endemic areas (Brazil, Argentina, Chile and the Southern States of America) the incidence does not appear to be great. It is a distinctly rare condition in children in non-endemic countries as may be judged by the few single case reports from such places as England, Metropolitan France and other European countries.

AMOEBIC LIVER ABSCESS IN CHILDREN

After Brown (1824) reported his case of liver abscess associated with dysentery in a child, there appeared a number of single reports of liver abscess in childhood (Miller 1851, Chapple 1861, Dulles 1879, Moore 1881, Menger 1881, Swift 1882, Easmon 1887, Huybertsz 1888, Pereira 1890, Neal 1892, Le Blond 1892, Legrand 1894, Finizio 1896 and Johnston 1897). All authors stressed the rarity of the condition in children.

It was not until 1895 that Slaughter reported the first

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proved instance of amoebic liver abscess. This occurred in a boy of 7 years in the United States of America. Amoebae were demonstrated in the multiple abscesses found at necropsy.

The second proved case to be published was that of Gneftos (1900). This occurred in Alexandria in a child of 6 years who died after surgical drainage of the abscess. The pus was reported to contain dead amoebae.

Other authors reporting on liver abscess in adults commented on the absence of this condition in children. Gutierrez (1904) among 647 amoebic liver abscesses found none under 10 years of age and only 2.2% between 10 and 20 years of age.

Some referred to amoebic liver abscess in childhood as an extreme rarity (Oddo 1904), while others stated that the condition did not exist in children.

A number of later reports have accredited Duckworth with having reported a case of amoebic liver abscess in a child in 1887. However, in his paper of 1887 he stated that tropical abscess of the liver was unknown in children, and he repeated this statement again in 1914 and 1920.

Great confusion existed in the early literature with regard to the aetiology of abscesses of the liver in childhood. In some,

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reported as following dysentery, amoebic origin was probable, but seldom was proof established by finding amoebae in the pus or wall of the abscess. Thus Amberg (1901) endeavoured to clarify these earlier reports. He stated that unfortunately in the great majority of liver abscesses in children which are reported following dysentery there is no mention made of amoebae, although in some of them one may suspect amoebic origin. Amberg, discarding various cases because dysentery was not mentioned or because of the presence of roundworm, arrived at a total of 12 of almost certain amoebic origin. The report of Easmon (1887) which Amberg discarded because dysentery was not mentioned occurred in Accra in a child of $3\frac{1}{2}$ years and may well have been amoebic in aetiology, although amoebae were not reported in the pus. It is a well established fact that not all cases of amoebic liver abscess have dysentery or a history of antecedent dysentery.

It was Legrand in 1906 who made the first really detailed study of the subject of liver abscess in children. He consulted verbally, and by circular, 250 practitioners in Egypt, and hospital personnel with long experience. In his "Mémoires Originaux" he reported on 112 collected cases of liver abscess of varying aetiology. Thirty-one of these he regarded as of amoebic origin. He himself in 1894 had reported 2 liver abscesses as amoebic in aetiology occurring in children in Suez, but in neither had E. histolytica been demonstrated in the pus. Explaining the rarity of amoebic liver abscess

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in children Legrand said that it was probably because amoebic dysentery itself was in reality also rare in children.

The case accredited to Menger (1881) was reported by Legrand as occurring in Germany, but, in fact, it occurred in the U.S.A. Legrand mentioned 4 cases reported by Rouis, Chapple, Nealy and Hall, the references to which he could not trace. It appears that Chapple's case was reported in 1861 (as mentioned above). 'Nealy' must read Neal (1892). The case of Hall (1892) was reported as traumatic in origin and that of Rouis (1896) occurred in Algeria.

This complication of liver abscess must be unusual in many parts of the world, judging by the number of authors who have been prompted to report one or a few cases (Boucher 1909, Niblock 1911, Alfaro 1912, Acuna 1912, Razetti 1913, Guzman 1919, Carvallo 1920, Carrau 1922, Cade et al. 1922, Navarro et al. 1925, Ludlow 1926, Panayotatoe 1926a and 1926b, Pozzo et al. 1928, Leroy des Bares 1930, Biggam 1932, Aldunate 1933, Sweet 1934, Fonzo 1934, Streicher 1935, Carbonnell y Salazar et al. 1936, Mohammed 1937, Smith 1943, Osburn 1944, Aguirre et al. 1944, Stransky et al. 1948, Shanmugaratnam 1949, Soragni 1949, Tupas et al. 1951, Salas Martinez et al. 1953, De la Maza et al. 1953, Smith et al. 1955, and Moreno 1956).

A report by Streicher in 1935 was entitled "Liver Abscess (amebic) in a boy aged seven years". At operation, however, the

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author described a "multiple suppurative cholangitis" which he drained through the gallbladder. Amoebae were not present in the pus or the stools. As a suppurative cholangitis is not the usual hepatic lesion of amoebic aetiology and is characteristically that due to ascariasis, this report has been excluded from the statistics of collected cases (appendix 1).

In some reports (Baeza 1933, Beretervide et al. 1940, Ortiz Machado 1941, Jelliffe 1951) amoebic aetiology could only be assumed, especially in the absence of aspiration of the contents of the abscess. Others have been excluded from the collected series because the aetiology was in doubt (Judd 1910, Muggia 1924, Bonaba 1935, Carbonnel y Salazar et al. 1936, Gonzalez 1935, Huard et al. 1936a).

A great deal of inaccuracy exists in the manner that various authors reports are quoted. For example, Rojas (1950) quoted Osburn (1944) as having reported 3 amoebic liver abscesses in children, whereas in fact only 1 had been so diagnosed by Osburn, the other 2 being intestinal amoebiasis. This same author (Rojas) quoted Aguirre et al. (1944) as reporting 5 amoebic liver abscess in children, whereas they had reported only 1. Even as recently as 1960 Jayaratne et al. quoted Osburn as having recorded 2 cases from Cape Town. This refers to the same paper as above and was of a liver abscess in a child in Salisbury, Southern Rhodesia. No case of amoebic liver abscess in a child has yet been reported from Cape Town.

Further confusion is caused by authors stating that amoebic liver abscess is unknown in early childhood. For example, Debbas (1953) stated that the complication of liver abscess was unknown under 7 years of age. However, as shown in appendix 1, this statement was entirely unfounded.

Of interest was the report of Jiménez et al. (1947) who described multiple amoebic liver abscesses in an infant of 2 months of age. At this stage this was the youngest recorded case. Since then there have been the reports of Fernandez de la Arena y Santé (1956) who reported a proved amoebic liver abscess in an infant 28 days old, and in the same year Jiménez et al. reported a further one in an infant also 28 days old.

Torroella et al. (1956), in an excellent paper, reported amoebic liver abscess in 14 patients under the age of 6 years which occurred in Mexico over a period of 11 years.

The finding of nucleated cysts in the pus from a case of liver abscess in an African infant, casts serious doubt on the correctness of the parasitic aetiology in the 4 cases reported by Senecal et al. (1957).

Salas et al. (1958) recorded 17 amoebic liver abscesses in children, compiled ffom necropsy statistics over a peried of 9 years. From that date the following cases have been reported: 1 by Wagle et al. (1958) from Bombay, 4 by Biagi (1958) from Mexico City, 1 from the

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United States of America by Burnside et al. (1959), 16 by Walt (1959) from Durban, 1 by Ravina et al. (1960) from Israel, 1 by Jayaratne et al. (1960) from Ceylon, and 10 by Macdougall (1960) from Nairobi.

The largest published series to date is that of Scragg (1960) concerning 53 cases among African children in Durban.

As stated by D'Antoni (1947) "the number of recorded amebic liver abscesses (in children) is small, but it is probable that many are not reported as, for example, the case of an infant of 4 weeks of age who died in Spanish Honduras". Jayaratne et al. (1960) also refer to 5 cases seen at necropsy at the Children's Hospital, Colombo, since 1952 which have not been published.

I am aware of other cases of amoebic liver abscess in children which have not been published. Wilmot (1949) observed an African infant in Durban, aged 18 months, who at necropsy had 2 large abscesses occupying most of the liver substance.

Prior to the start of this study in 1951, amoebic liver abscess in African children was not unknown at King Edward VIII Hospital, Durban (Klenerman 1951). I have also examined 6 infants with amoebic liver abscess who had come under the care of surgeons in this hospital.

There is obviously great confusion as to the incidence of the complication of liver abscess in childhood. Some authors categorically state that it does not occur, while others stress that it is very rare. In order to clarify this, all the reported cases that have been collected by careful search of the literature have been tabled in appendix 1.

The term amoebic hepatitis has purposely not been referred to in the above review. This has received much attention in adults and there are also reports of amoebic hepatitis in children (Gillot and Dendale 1936, Cardelle and Saenz 1948, Soragni 1949, Tupas et al. 1951, Lestrade et al. 1956, Biagi 1958 and others). However, no instance of so-called diffuse, non-suppurative amoebic hepatitis has been observed among a large number of necropsies on African children at King Edward VIII Hospital. Pathological proof of such a lesion appears to be lacking.

Roach (1958) reported from Durban that on routine histological examination of the liver of all necropsy cases not a single case of diffuse hepatitis attributable to the amoeba had been seen. In some cases pre-suppurative lesions were present in association with liver abscess. From neither necropsy nor biopsy material had it been possible to find diffuse non-suppurative amoebic hepatitis. Powell et al. (1959), also reporting from Durban, are of the same opinion.

The experience in Durban appears similar to that of some workers elsewhere. Da Silva (1954) found no evidence of diffuse hepatic lesion in Rio de Janeiro; Spellberg (1954) believed that the difference between hepatitis and hepatic abscess was quantitative rather than qualitative, the only clear difference being in response to therapy. Harrison (1958) also stated that "no clear cut distinction between hepatitis and abscess is warranted as they differ only in degree. Kean (1955) reviewed histological sections of the liver in 4,478 consecutive necropsies in the Canal Zone and failed to demonstrate diffuse lesions produced by E. histolytica. Kean et al. (1956) also reviewed the histological findings in 148 fatal cases of amoebiasis and were unable to find proof of a diffuse hepatitis. Kean is of the opinion that "...the only proved amoebic lesion of the liver is the abscess. This may be single and large or multiple and small. There is no proof of the existence of a diffuse lesion or a widespread focal lesion".

Conclusions

Amoebic liver abscess in children appears to be unusual in most parts of the world, judging by the isolated case reports.

Review of the literature since 1824 shows that Durban is unique as far as the incidence of liver abscess in children is concerned, for, apart from this area, the only series of any size

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have been reported from Mexico, consisting of two studies numbering 17 and 14 cases, respectively, and another from Kenya of 10 cases.

It is of interest that while amoebic liver abscess is very common among adults in such places as Egypt, India and the Far East, no large series in children has been reported from these areas.

AMOEBIASIS IN CHILDREN IN NATAL

Incidence of Entamoeba histolytica infection

Asymptomatic amoebiasis appears to be much less common among African children in Natal than elsewhere, as judged by the chance finding of E. histolytica in the stools. While cysts of E. histolytica are not infrequently reported in the stools of children in other parts of the world this finding is less common in Durban. The more usual finding is of trophozoites of E. histolytica in children who have the signs of dysentery.

No stool survey for E. histolytica has been conducted in African children in Durban. In 1946, Elsdon-Dew gave an incidence of E. histolytica in 27.5% of 20,243 stool specimens from African and Indian patients in King Edward VIII Hospital. Of these 42 were children under 10 years. Later, Elsdon-Dew et al. (1952) in a study of intestinal parasites in the Natal African, found an overall incidence of E. histolytica in just over 5%. As a single examination by their technique revealed only 70% of infections, the authors considered the incidence in this race to be about 10%.

Amoebic dysentery

Elsdon-Dew (1945) and Armstrong et al. (1949) pointed out that amoebic dysentery in Natal and particularly in Durban affects predominantly the African population and occurs rather uncommonly in

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Indians and to only a small extent in Europeans.

No accurate statistics exist for the incidence of amoebic dysentery in the African child population of Natal. In Durban it is a common disease accounting for the out-patient attendance of hundreds of children annually.

As the vast majority of cases of amoebic dysentery in African children have of necessity to be treated as out-patients, the figures of hospital admissions can in no way be taken as indicative of the probable incidence of the disease.

In our busy Paediatric out-patient department, with an annual attendance of about 60,000, there are laboratory facilities for immediate direct examination of stools. Records have been kept for some years of all cases of proved amoebic dysentery in children 10 years and under. The average number of such cases treated as out-patients is 30 per month. However, in only some stools from children whose complaint is diarrhoea with blood and mucus, is E. histolytica found on a single examination. The instability of shack residence, distance from hospital, transport difficulties and other factors, do not permit of controlled follow-up of children with symptoms of dysentery, thus repeated stool examinations of children attending as out-patients is not often possible.

A valuable aid to diagnosis is sigmoidoscopic examination. The large number of cases, however, leaves little time for such an

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examination as a routine measure, so that many cases of probable amoebic dysentery are therefore treated symptomatically.

An average of 150 cases of amoebic dysentery are treated annually as in-patients. The upper age limit for admission to this Unit is about 7 years. The cases for admission tend to be selected on the basis of severity. In 1959, 162 cases were treated in hospital, the age distribution of which was as follows:-

0 - 6 months	9
7 - 11 "	21
1 - 2 years	63
- 3 "	22
- 4 "	19
- 5 "	13
- 6 "	13
over 6 "	2
Total	162

It will be noted that the greatest incidence of amoebic dysentery is found between the ages of 1 - 2 years.

The occurrence of amoebiasis in all parts of the world is better correlated with poor sanitation and low socio-economic standards than it is with climate and geographical consideration (Platou and Beaver, 1958).

The urban and immediate peri-urban African population of Durban is still largely housed under slum conditions with little or no sanitation. It is from this environment, which exposes the infant to amoebiasis from the moment of birth, that most of our hospital cases are drawn.

In Durban amoebic dysentery in the white child is not common. Enquiry of private practitioners and paediatricians reveals that they see few cases.

At Addington Hospital which is the white children's hospital in Durban, there have only been 45 cases of amoebic dysentery in 6 years (March 1954 - March 1960) and during this time only 2 cases of amoebic liver abscess occurred (Wallace 1960).

No attempt has been made to investigate the incidence of amoebic dysentery among Indian children attending the out-patient department. It occurs much less frequently than among African children, and accounts for very few attendances or admissions to the Paediatric department.

Amoebic liver abscess

It has been the opinion of some workers that the native races in the tropics are less often affected by amoebic liver abscess than the white race.

Craig (1944) stated "... that race has something to do with the incidence of amoebic abscess of the liver, is proven by the fact that it is less commonly encountered in the native races in the tropics than in the white races". He believed that this was due to a partial immunity of the natives from constant reinfection since childhood. Manson-Bahr (1951) was also of this opinion.

Among white children in Natal amoebic liver abscess is a rare condition (Wallace 1960). During the 9 years period under discussion I have seen 8 cases of liver abscess in Indian children, only 4 of which were of amoebic aetiology, the others being due to ascariasis. Amoebic dysentery is less common in these two racial groups, so that it is to be expected that this complication would be rarely seen.

In Durban a high incidence of fulminating dysentery and its complications occurs in the indigenous African (Elsdon-Dew 1949). African children share in this high incidence of the disease and its complications.

It is interesting to note that Legrand as early as 1906 commented that race was of no importance with regard to the complication of amoebic liver abscess in children, and he listed the wide variety of races in 31 collected cases.

Conclusions

The incidence of infection with *E. histolytica* has not been assessed for the 3 main racial groups of children in Natal.

Amoebic dysentery is very common in the African child population, occurs uncommonly in the Indian, and to only a small extent in white children.

The highest incidence of amoebic dysentery (and its complications) in African children is found between the ages of 1 and 2 years.

The complication of liver abscess is rarely seen in white or Indian children, but is not uncommon in African children.

CLINICAL MATERIAL

The study of the particular complication of liver abscess occurring in African children over a period of 9 years (1951 - 1960) forms the basis for this thesis.

The clinical material comprises 115 cases of amoebic liver abscess (appendix 2).

It was necessary to exclude one from the series. This was a female of 17 months in whom amoebic liver abscess was proved by aspiration and who clinically had recovered from the hepatic complication. This child developed convulsions and cerebro-spinal fluid changes and the development of an amoebic brain abscess was postulated. At this stage, however, the parents removed the child from hospital and the outcome is unknown. Three other infants with proved amoebic liver abscess were dead on arrival at hospital.

With these exceptions, all cases of amoebic liver abscess admitted to the Paediatric Department in this 9 year period are documented.

All the subjects, with the exception of 12, were clinically examined and investigated by myself.

UNPROVED CASES

Fourteen were not proved by the demonstration of pus either by aspiration or at necropsy. It is proposed to deal briefly with these and then to discuss in detail the remaining 100 patients.

The criteria for diagnosis in these 14 patients were:

(i) Proved amoebic dysentery with trophozoites of E. histolytica in the stools of all.

(ii) Tender hepatomegaly in all. The presence of a palpable mass in the liver in 10 of the 14.

(iii) Complete resolution on specific anti-amoebic therapy in all but 2 patients, who died.

The age range was from 9 months to 6 years, 3 of them being 1 year or less.

Eight were males and 6 were females.

All had fever. A leucocytosis (above 15,000/c.mm.) was present in all cases with a range from 16,000 to 32,000 per c.mm. (average 24,000/c.mm.).

There was a normocytic normochromic anaemia (< 10 g./100 ml.) in 10. The range being 6.5 to 12.8 g./100 ml. with a mean of 8.7 g./100 ml.

In 13, radiological examination was carried out. Three showed

/elevation..

elevation of the right dome of the diaphragm and 1 slight elevation of the left dome of the diaphragm (associated with a mass in the left hepatic lobe). One case showed collapse of the middle lobe of the right lung with a normal diaphragm and in 8 there was no radiological abnormality.

Ten cases had a palpable mass in the large tender liver. In 8 the mass was located in the right lobe of the liver, in 1 it was in the left lobe, and 1 had a mass centrally placed in the liver. In 1 who exhibited a mass in the right lobe, a similar mass appeared in the left lobe of the liver 21 days later.

In 3, transcostal aspiration was attempted because as marked elevation of the diaphragm was present it was feared that rupture through the diaphragm might occur. However, no pus was obtained, but the mass, large liver and elevated diaphragm resolved with anti-amoebic treatment.

The reasons for not aspirating the remaining 7 were, firstly, because the masses were hard and non-fluctuant and, secondly, as they were under observation it was felt safe to review the situation day by day.

There is no doubt that amoebic liver abscesses can and do resolve on specific anti-amoebic therapy without aspiration (Rogers 1933). Therefore in the presence of a hard mass and if imminent rupture is not anticipated, it appears reasonable to delay aspiration.

Every case received emetine and chloroquine. For the concomitant amoebic dysentery diiodohydroxyquinoline was used in 7, tetracycline in 3, chlortetracycline in 3 and succinylsulphathiazole in 1.

There was complete resolution of all but two cases on the above treatment.

Three of these 14 cases are worthy of mention:

Case No. 20. A 3 year old male was found to have blood, mucus and trophozoites of E. histolytica in the stools. He was gravely ill with a large, tender liver 10 cm. below the costal margin, containing a hard, diffuse mass centrally placed. Anaemia (8 g./100 ml.), leucocytosis (27,000/c.mm.) and a high temperature were present. Treatment with emetine, chloroquine and diiodohydroxyquinoline started immediately after admission. Aspiration was delayed in view of the hardness and lack of definition of the mass. The liver and mass remained unchanged and death occurred after 6 days. Necropsy was refused.

Case No. 27. A 2 year old female with a history of dysentery 3 months previously was admitted seriously ill. Trophozoites of E. histolytica were present in the one stool examined. High temperature, anaemia (7.4 g./100 ml.) and leucocytosis (27,000/c.mm.) were present. The liver was tender and much enlarged with a hard, diffuse mass in the right hepatic lobe. Emetine, chloroquine and diiodohydroxyquinoline were started immediately after admission. Aspiration was delayed as the mass was hard. The patient died 51 hours later. Necropsy was refused.

In retrospect, it might have been wise to have attempted aspiration in these 2 cases in view of the fatal outcome. In the first, lack of response after a few days of appropriate treatment should have prompted exploratory needling, which may well have saved this child.

/The..

The second did not survive long enough to receive more than 2 doses of emetine and 2 days treatment with chloroquine, but here too, in view of the serious condition, aspiration might have had a beneficial effect.

Case No. 33. A 5 year old male on admission had a large, tender liver $8\frac{1}{2}$ cm. below the costal margin with a hard mass in the right lobe. This mass disappeared on treatment, but the liver did not return to normal size and remained tender for 11 days after completing a course of emetine. While still on chloroquine another mass became apparent in the left hepatic lobe. A further course of emetine was given and chloroquine was continued, with complete recovery.

The clinical picture of tender hepatomegaly associated with amoebic dysentery, which resolves on specific anti-amoebic therapy, without hepatic aspiration becoming necessary is a familiar one in adults but is a much less common finding in children. That only 4 such cases have been encountered in this long period under discussion is evidence that the more usual clinical picture of hepatic amoebiasis in children is one of manifest abscess in the form of an actual mass in the liver, or evidence of diaphragmatic elevation when transcostal aspiration usually yields pus.

PROVED CASES - 100

The criteria for diagnosis were:

(i) Demonstration of pus either by aspiration or at necropsy.

(ii) Demonstration of pus from rupture of an abscess (this occurred only once).

All statistical data shown hereafter has been based on the findings in these 100 cases.

Age distribution

The age distribution is shown in figure 1. The youngest was 8 weeks and the oldest 5 years. Thirty-two were 1 year or less in age, 12 of them being 6 months or under.

The maximum incidence falls in the age group 1 to 2 years (the age group most affected with intestinal amoebiasis). Forty-one of the cases were in this age group.

In the series of 6 cases reported by de la Maza et al. (1953) only 1 was older than 2½ years, while none of the 5 cases reported by Fernandez de la Arena y Santé (1956) was over 2 years. The series of Torroella et al. (1956) showed the youngest to be 3 months and the oldest 5 years with 9 of the 14 falling in the group 6 months to 2 years. Salas et al. (1958) reported 17 cases of amoebic liver abscess in children of which 5 were 1 year or less and 6 between 1 and 2 years. In Walt's cases (1959) the youngest was 7 months and the oldest 4 years, with 12 of the 16 being 2 years or under. Figure 2 shows the age distribution of 154 collected cases.

Sex distribution

Fifty-eight of the 100 patients were males (figure 3).

The striking predominance of males in the reported series of

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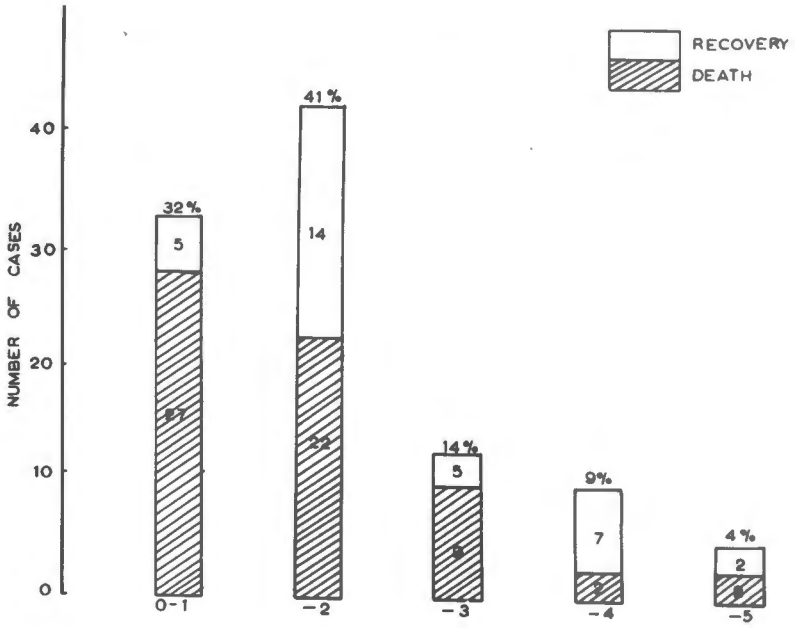


FIG.1. AGE INCIDENCE AND OUTCOME IN AUTHOR SERIES OF 100 CASES OF AMOEBIC LIVER ABSCESS IN CHILDREN

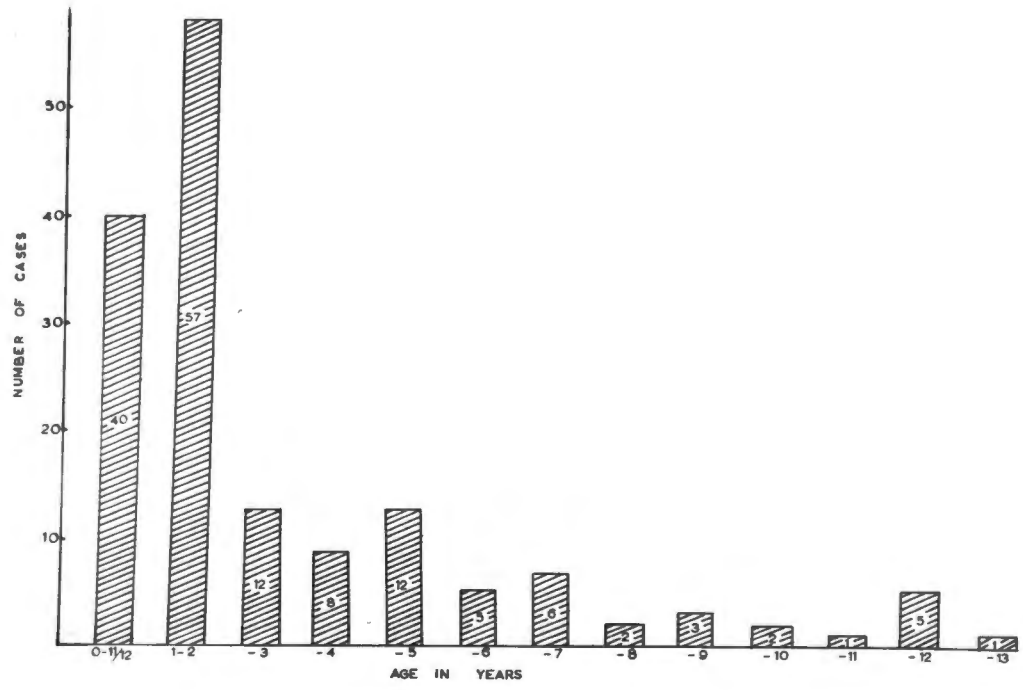


FIG 2. AGE INCIDENCE IN 154 COLLECTED CASES OF AMOEBIC LIVER ABSCESS IN CHILDREN

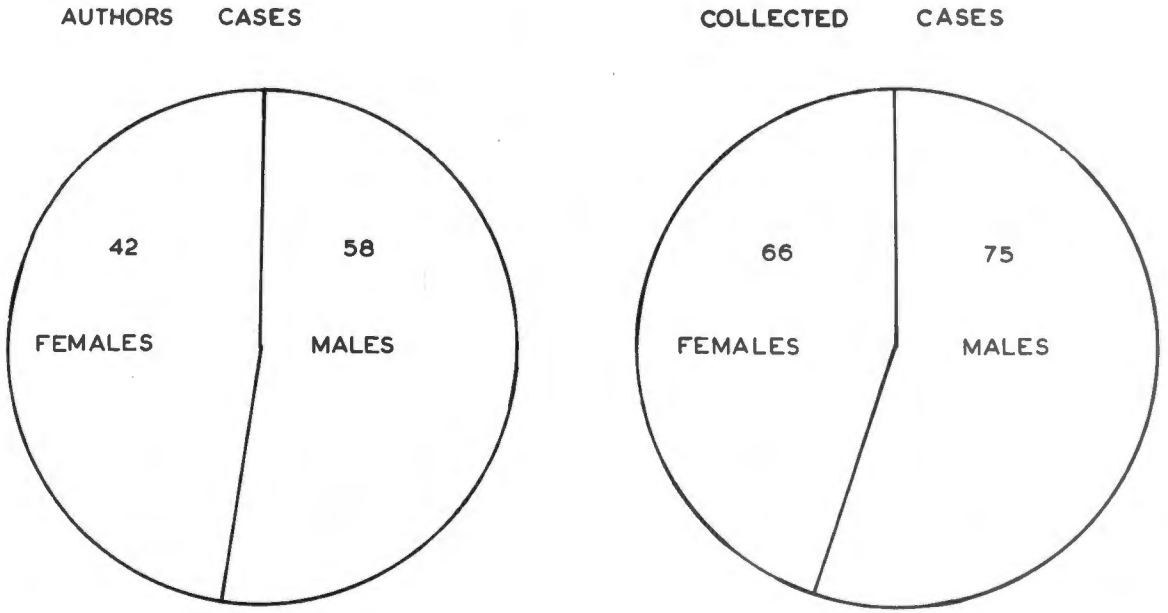


FIG. 3. SEX INCIDENCE IN AUTHORS AND COLLECTED CASES OF AMOEBIC LIVER ABSCESS

amoebic liver abscess in adults, though unexplained, is an undoubted fact. This has been shown by a number of authors (Ludlow 1926, Biggam and Ghalioungui 1933, Craig 1934, Huard and Meyer-May 1936b, Palazuelos and Lerner 1941, Ochsner and DeBakey 1943, Wilmot 1949 and others) who report a predominance of males of about 80-90%.

Legrand (1906) was of the opinion that "the child is more or less sexless with regard to the liver before puberty". He reported that where sex had been stated in 31 collected cases, there was no significant predominance in the male sex.

Often sex is not stated when one or a few cases have been reported in children. Biagi (1958 and 1959) and Salas et al. (1958) state that the hepatic lesions in children are encountered with equal frequency in the sexes.

In the series of 14 children reported by Torroella et al. (1956) there were almost twice as many females as males. Salas et al. (1958) in a series of 17 found 70% to be females and Scragg (1960) reported 62% males in a series of 53 cases.

Where sex has been stated in the collected cases (figure 3) it will be seen that 75 (53.2%) were males and 66 (46.8%) were females out of a total of 141.

Thus the striking predominance of between 80-90% in the male sex in adult cases is not a feature of the condition in children.

Clinical features

Pain. All authors stress that pain is a constant feature of hepatic abscess. In small infants it is difficult to determine whether pain is present. However, the facial expression and the frequency of a respiratory "grunt" suggests that it is. Even in the absence of lung involvement or diaphragmatic pleurisy the movements of respiration appear to cause pain and if the child is observed lying still in bed a grunt with each breath will often be heard.

Fever. This was present in all cases (figure 4). The temperature usually fluctuated between 102-103°F. until appropriate therapy was begun.

While some authors stress that fever is an important sign in adults (Craig 1934, Palazuelos et al. 1941, Manson-Bahr 1944), others state that fever may be absent or low grade (Ludlow 1926, Biggam and Ghalioungui 1933, Wilmot 1949, Lamont and Pooler 1958).

In most of the isolated case reports of amoebic liver abscess in children fever has been a prominent feature and has been stressed by Aguirre and Rivera (1944), Lestrade et al. (1956), Fernandez de la Arena y Santé (1956) and Torroella et al. (1956).

Thus in children as in adults fever is an important finding. However, in children the fever is striking, usually being high and swinging and only very rarely low grade.

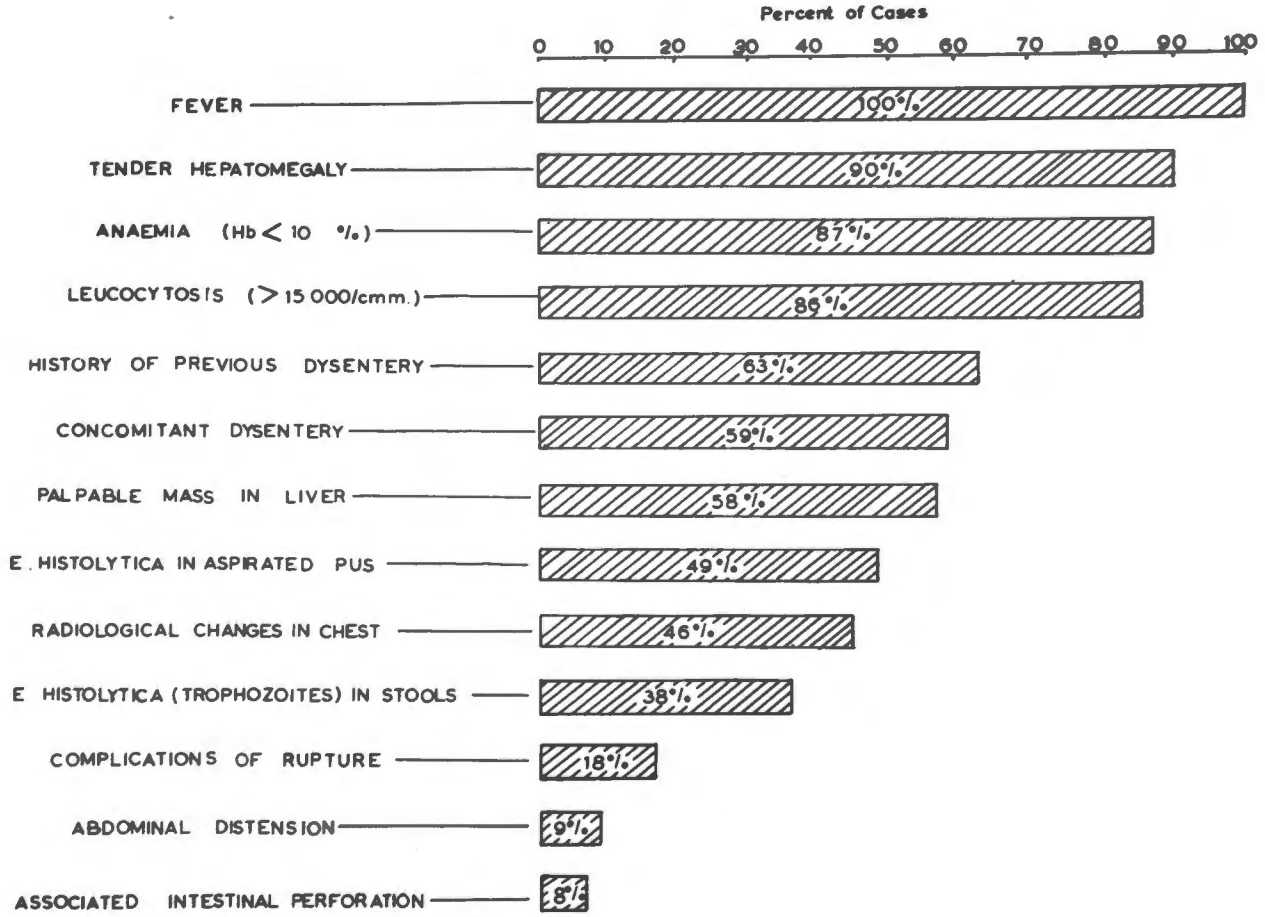


FIG. 4 CLINICAL FINDINGS IN 100 CASES OF AMOEBIC LIVER ABSCESS.

Tender hepatomegaly. Tender hepatomegaly was present in all but 10 cases (90%).

Nine had apparent tender hepatomegaly, but in addition generalised abdominal distension and tenderness was present. This feature of abdominal distension will be referred to later.

In the 10 cases where tenderness was not a striking feature the correct diagnosis was not made until necropsy. Five of these showed neither tenderness nor hepatomegaly. The remaining 5 had hepatic enlargement (varying from 6 - 10 cm. below the costal margin) but tenderness was not elicited.

Intercostal tenderness is a constant sign in adults (Wilmot 1949) and of value in differentiating other causes of hepatomegaly and in focussing attention on the liver when other signs indicate involvement of another organ such as lung, pericardium or peritoneal cavity. Intercostal tenderness may be difficult to elicit in infants, but direct palpation of the liver is usually exquisitely painful.

The following case summaries briefly illustrate the difficulty that may arise in diagnosis:

Case No. 14. A male infant of 9 months was admitted with a history of diarrhoea with blood and mucus of 3 weeks duration. He was ill-looking with a high temperature. The liver was $4\frac{1}{2}$ cm. below the costal margin. It was difficult to decide whether the liver was tender in this small crying infant, but after a few days it was thought that tenderness was not present. Hepatic amoebiasis was considered but the diagnosis was discarded. He was treated as a case of amoebic dysentery with diiodohydroxyquinoline and oxytetracycline, despite the stool being negative for E. histolytica. Anaemia and leucocytosis were present. There was no response to treatment and death occurred on the 10th day in hospital. Necropsy showed multiple minute amoebic liver abscesses, diffuse amoebic ulceration of the colon, peritonitis and an abscess in relation to the descending colon.

Case No. 29. A 3 year old male was seriously ill on admission with signs of bronchopneumonia. There was nothing of note in the past history and diarrhoea was not present. He had a high temperature, anaemia and leucocytosis. The liver was palpated 4 cm. below the costal margin and was non-tender. X-ray of the chest confirmed the presence of bronchopneumonia. He failed to respond to penicillin and later to streptomycin and died on the 11th day in hospital. At necropsy a diffuse bronchopneumonia and thrombosis of cerebral vessels was found. A small amoebic abscess 4 cm. in diameter was found in the right hepatic lobe. Although this did not account for death it illustrates that an amoebic abscess may be present without antecedent or concomitant dysentery.

Case No. 47. A female infant of 14 months with severe kwashiorkor was admitted moribund, with a history of diarrhoea for 3 weeks. The liver was noted to be 4 cm. below the costal margin and was not tender. She was considered too ill for X-ray. Soon after admission convulsions occurred. The cerebrospinal fluid was normal. Death occurred 21 hours later. Necropsy showed multiple small liver abscesses in which E. histolytica were found. Ulcers were found in the caecum but no amoebae were found in these lesions.

Case No. 54. A male of 3 years, with pulmonary tuberculosis, was seriously ill on admission. There was a history of diarrhoea with blood in the stools 3 weeks previously. X-ray of the chest revealed a tuberculous cavity. The liver was $4\frac{1}{2}$ cm. below the costal margin and was not tender. There was an anaemia and leucocytosis. He died 35 hours after admission. At necropsy there was an ulcerative colitis with amoebae present and early multiple amoebic liver abscesses. The presence of cavitating pulmonary tuberculosis was confirmed.

In 3 of the 4 cases described above the hepatic lesions were multiple and small, and in the fourth case single and small, which probably explains why the liver enlargement was not pronounced.

The following cases had hepatomegaly, but tenderness was not a feature:

Case No. 2. A two year old male was admitted with a history of diarrhoea with blood and mucus of 2 months duration. He was seriously ill with a high temperature and signs of bronchopneumonia. The liver was $6\frac{1}{2}$ cm. below the costal margin, firm and not tender. The stool contained blood and mucus but no E. histolytica. Subsequently, Shigella flexneri was cultured from the stool. There was anaemia and leucocytosis. Initially the bronchopneumonia appeared to respond to penicillin and sulphadiazine but crepitations persisted at the base of the right lung. Chloramphenicol was now given without further improvement and he died on the 8th day in hospital. At necropsy there was one large abscess in the right hepatic lobe and multiple small abscesses throughout the liver. The abscess in the right lobe had ruptured into the right lung, in which an abscess cavity was found in the lower lobe. There was extensive amoebic colitis. E. histolytica were present in both the bowel and hepatic lesions.

Case No. 80. A female infant of 8 months had attended the out-patient department with a history of 4 weeks diarrhoea. She had received chlortetracycline for one week without improvement. On admission she was gravely ill and dehydrated. The liver was palpable 6 cm. below the costal margin and did not appear tender. Intravenous therapy and tetracycline was started. The liquid stool contained no blood, mucus or E. histolytica. She died 41 hours after admission. At necropsy there was a single amoebic abscess of the liver, without evidence of active intestinal amoebiasis.

Case No. 95. A male infant of 9 months was admitted moribund with the signs of right pleural effusion, confirmed by X-ray. There was a history of diarrhoea with blood and mucus for 3 weeks. The liver was 4 cm. below the costal margin, smooth and was not tender. The stool was normal in appearance. As there did not appear to be undue respiratory distress it was decided to resuscitate the infant before aspirating the chest. However, he died 12 hours after admission and before aspiration. Necropsy showed a large amoebic abscess in which E. histolytica were found, situated posteriorly in the right lobe of the liver. The abscess had ruptured through the diaphragm into the right pleural cavity with resultant empyeme. There were also multiple small abscesses in the liver. Amoebic colitis was present, but no amoebae were demonstrated in the bowel lesions. This case illustrates how attention may be focussed on another organ when the complication of rupture has occurred. Here the liver was no longer greatly enlarged or tender because the abscess had already emptied itself into the pleural space.

Case No. 113. A female child of 2 years was admitted with signs of bronchopneumonia. There was a history of diarrhoea with blood in the stools 2 months earlier. The liver was 8 cm. below the costal margin, but was not tender. There was anaemia and leucocytosis.

/X-ray..

X-ray of the chest suggested miliary tuberculosis. Stool examinations were all negative for E. histolytica. No anti-amoebic treatment was given. Death occurred after 5 days. Necropsy confirmed the presence of tuberculosis with miliary tubercles throughout the liver. However, in addition there were 3 large liver abscesses in which amoebae were present. There was no bowel lesion.

Case No. 114. A male infant of 9 months with a history of diarrhoea for 2 days was seriously ill on admission. The liver was 6 cm. below the costal margin and was not tender. Anaemia and leucocytosis were present. The infant remained ill and febrile and the diarrhoea continued. Repeated stool examinations were negative for E. histolytica. He died suddenly after 9 days. Necropsy showed a liver abscess (with amoebae present) in the right lobe, which had ruptured into the right pleural cavity. There was amoebic colitis with amoebae present.

Abdominal distension. Abdominal distension may mask the diagnosis of amoebic liver abscess. Fulminating intestinal amoebiasis is commonly seen in African children. They may present with abdominal distension and generalised tenderness due to peritonitis either from frank perforation of the colon or a "seepage" through a grossly diseased bowel. In 1959, 23.7% of 59 cases of amoebiasis which came to necropsy had such peritonitis.

Abdominal distension from other causes may present difficulty. Howell and Knoll (1941) drew attention to the fact that amoebic dysentery in children may simulate typhoid fever. Experience among African children confirms this observation. In 3 of the 9 following cases a tentative diagnosis of typhoid fever was made:

Case No. 43. A male child of 4 years had had diarrhoea for 2 weeks. He was ill, pale and had a high temperature. The abdomen was distended and diffusely tender. The liver appeared to be about 6 cm. below the costal margin, the edge being difficult to determine. There was anaemia, but no leucocytosis (9,000/c.mm.). The green, liquid stool contained no blood, mucus or E. histolytica and culture was negative. He was treated with chloramphenicol as a possible case of typhoid fever. However, this diagnosis was not confirmed on further investigation and death occurred 8 days after admission. Necropsy showed multiple amoebic liver abscesses, ulcerative amoebic colitis with perforation of the bowel and generalised peritonitis.

Case No. 71. A female infant of 1 year was found to have amoebic dysentery. On admission the liver was noted to be about 6 cm. below the costal margin and there was slight generalised tenderness of the abdomen. Crepitations were present in both lungs. The X-ray was suggestive of pulmonary tuberculosis. The white cell count was 8,000/c.mm. and the haemoglobin 11.2 g./100 ml. The treatment consisted of emetine, diiodohydroxyquinoline and tetracycline. Four days later the abdomen became distended and more tender. Amoebic peritonitis was thought to have occurred. The infant died the following day. Necropsy confirmed the presence of amoebic colitis and pulmonary tuberculosis, but peritonitis was not present. There were multiple amoebic liver abscesses.

Case No. 75. A malnourished female of 9 months was seriously ill with diarrhoea and vomiting. Apart from a high temperature the only finding of note was a large liver 8 cm. below the costal margin and abdominal distension. The stools were green, watery and contained mucus, but no blood or E. histolytica. A provisional diagnosis of bacillary dysentary or typhoid fever was made and chloramphenicol was given. There was no response to treatment, the abdomen became more distended and she died three days later. Necropsy showed multiple large amoebic abscesses in the right hepatic lobe and ulcerative amoebic colitis.

Case No. 82. A seriously ill male of 4 years was admitted with proved amoebic dysentery. Dullness and crepitations were present in the left lung. The liver was palpated about 2 cm. below the costal margin and there was generalised abdominal distension and tenderness. Anaemia and leucocytosis was present. He was treated with gastric suction, intravenous fluids, emetine and intravenous tetracycline. There was no response to treatment and death occurred 3 days later. Necropsy showed a single amoebic abscess 3 inches in diameter, situated in the left hepatic lobe, which

/had..

had ruptured into the left lung. By contiguity with the abscess there was a fibrinous pericarditis. Amoebic ulcerative colitis with perforation of the bowel and peritonitis was also present.

Case No. 83. A female child of 5 years with amoebic dysentery was admitted seriously ill, with abdominal distension and tenderness. The liver appeared enlarged about 6 cm. below the costal margin, but the edge could not be accurately palpated. There was anaemia and leucocytosis. A diagnosis of amoebic peritonitis was made. Emetine, intravenous tetracycline and gastric suction was started. Death occurred 19 hours later. Necropsy confirmed the diagnosis of amoebic colitis with perforation and peritonitis. Two amoebic abscesses about 4 cm. in diameter were found, one in the left and one in the right lobe of the liver.

Case No. 90. A female child of 2 years with kwashiorkor had a one week history of diarrhoea, with a little blood in the stools. She had a very distended, tender abdomen. Although the liver was enlarged it was difficult to determine the exact size. E. histolytica were not found in the stool. There was anaemia but no leucocytosis (11,000/c.mm.). Possible bacillary dysentery or typhoid fever was considered and chloramphenicol prescribed. Death occurred 32 hours after admission. Necropsy showed multiple amoebic abscesses throughout the liver and amoebic ulceration of the colon.

Case No. 96. A female infant of 10 months had a history of diarrhoea with blood and mucus of 3 weeks duration. She was febrile and very ill with abdominal distension. Generalised tenderness prevented accurate palpation of the liver, but the latter appeared enlarged. The haemoglobin was 11.2 g./100 ml. and the white cell count 13,000/c.mm. E. histolytica were not found in the stool examined on admission. Oxytetracycline was started. Forty-eight hours later a stool containing blood and mucus was found to contain E. histolytica. The liver edge was still not felt but the liver was very tender. As hepatic amoebiasis appeared likely, emetine and chloroquine were now commenced and diiodohydroxyquinoline was given for the bowel lesion. However, signs of bronchopneumonia developed and death occurred the following day. At necropsy there was a single abscess in the left lobe of the liver, ulcerative amoebic colitis and bronchopneumonia.

Case No. 98. A male child of 2 years with kwashiorkor and bronchopneumonia had a history of diarrhoea with blood in the stools for 3 months. The liver was noted to be about 6 cm. below the costal margin. There was abdominal distension and generalised tenderness. High fever, severe anaemia (Hb. 3.2 g./100 ml.) and leucocytosis (24,000/c.mm.) were present. Penicillin was started and a blood

/transfusion..

transfusion was given. The loose stool contained no blood, mucus or E. histolytica. The following day the abdomen was more distended and he died shortly afterwards. At necropsy there was an abscess of the right hepatic lobe (containing E. histolytica) which was leaking into the peritoneal cavity with resultant peritonitis. There was ulcerative amoebic colitis, bilateral bronchopneumonia and left sided pleurisy.

Case No. 104. A male infant of 1 year, moribund on admission, had a history of diarrhoea with blood and mucus in the stools for 2 weeks. Crepitations were present in the left lung. The liver was palpable about 6 cm. below the costal margin and the abdomen was distended. Dysentery was present. He died 8 hours after admission. Necropsy showed an amoebic liver abscess 2-3 cm. in diameter situated in the right hepatic lobe. There was an ulcerative amoebic colitis with an early fibrinous peritonitis without demonstration of frank perforation of the bowel.

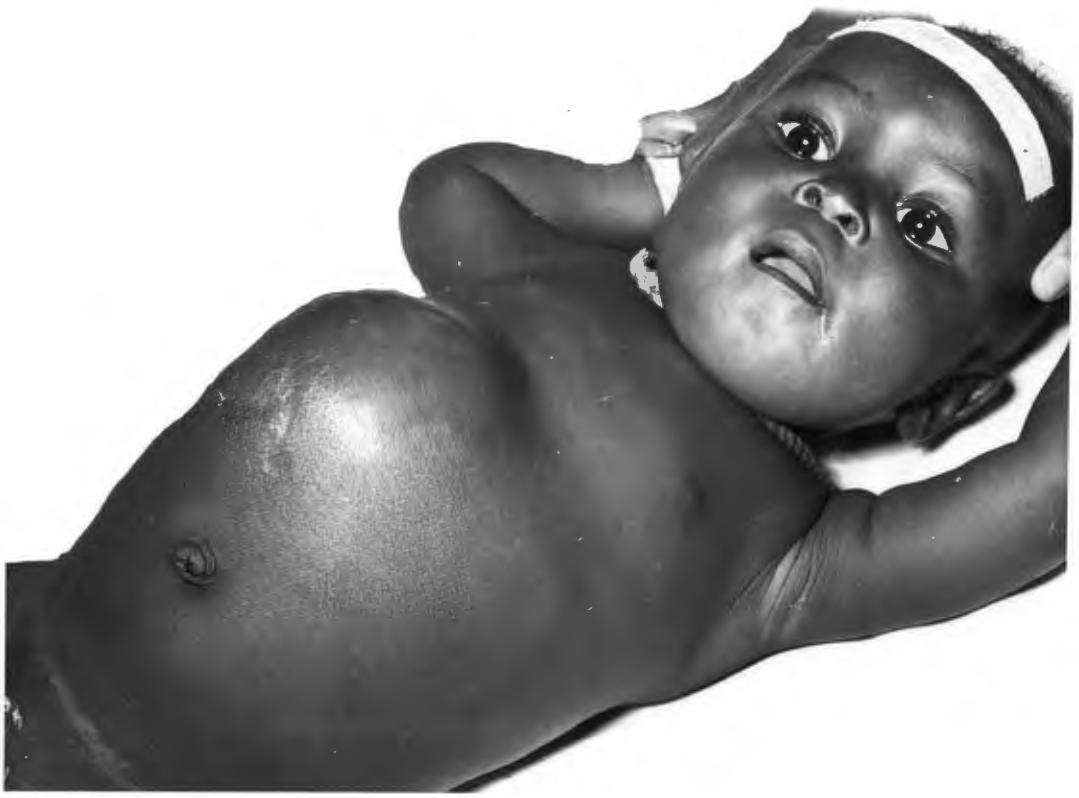
Palpable mass in the liver. A palpable mass was present in 58 cases. (Plate 2 is an example of a visible mass arising from the liver)

Six cases each had 2 masses in the liver, 4 of them a mass in both right and left lobes, 1 a mass in the right lobe and another centrally placed in the liver, and 1 case had 2 masses in the right lobe. In 4, pus was aspirated from the separate abscesses, while in 1, pus was aspirated from the mass in the right, but not from that in the left lobe and in the remaining case death occurred very rapidly before aspiration was possible.

In 16 cases no mass was palpated in the large, tender liver, but pus was obtained by transcostal needling. In 6 of these 16 cases the liver enlarged under observation.

Location and number of abscesses. The location of abscesses clinically determined in 74 cases (figure 5) was as follows:

Plate 2. Case No. 105. Male infant aged 10 months with a large abscess in the right lobe of the liver. A total of 410 ml. of pus was aspirated from this abscess



1.	Right lobe	51 (68.9%)
2.	Left lobe	4 (5.4%)
3.	Central - at junction of lobes of liver	13 (17.6%)
4.	Multiple	6 (8.1%)

Abscesses are said to be characteristically single and to occur most often in the right lobe. In this series it will be seen that by clinical determination 68 (91.9%) were single and only 6 (8.1%) multiple. In 7 additional cases the location was uncertain and aspiration was not carried out. In the remaining 19 the correct diagnosis was not made before death.

If, however, these figures are determined from necropsy findings (figure 6) it will be seen that in 52 cases the findings were as follows:

1.	Right lobe	15 (28.8%)
2.	Left lobe	4 (7.7%)
3.	Central - at junction of lobes of liver	1 (1.9%)
4.	Multiple	32 (61.5%)

In adults figures vary widely from 37% (Craig 1934) to 89% (DeBakey and Oschner 1951) for single lesions. Similarly location of right lobe abscesses varies from about 65% to over 90% (Gutierrez 1904,

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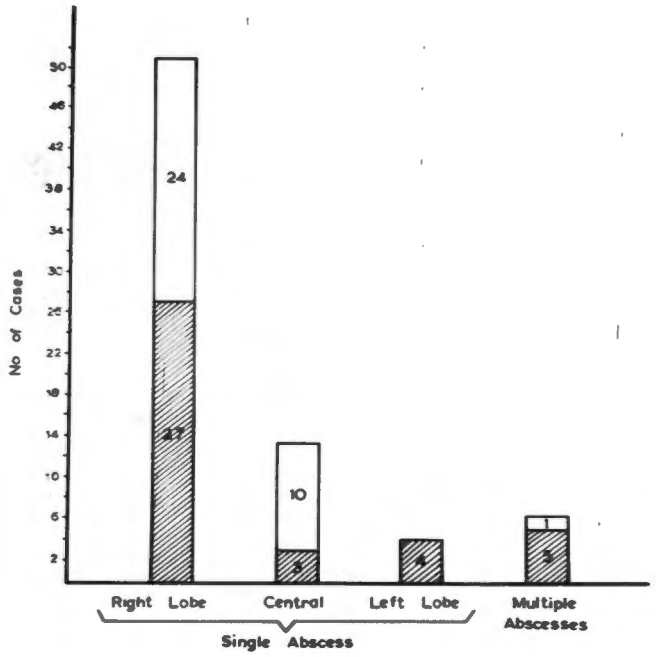

FIG. 5

NUMBER AND LOCATION OF ABSCESSES
CLINICALLY DETERMINED IN 74 CASES

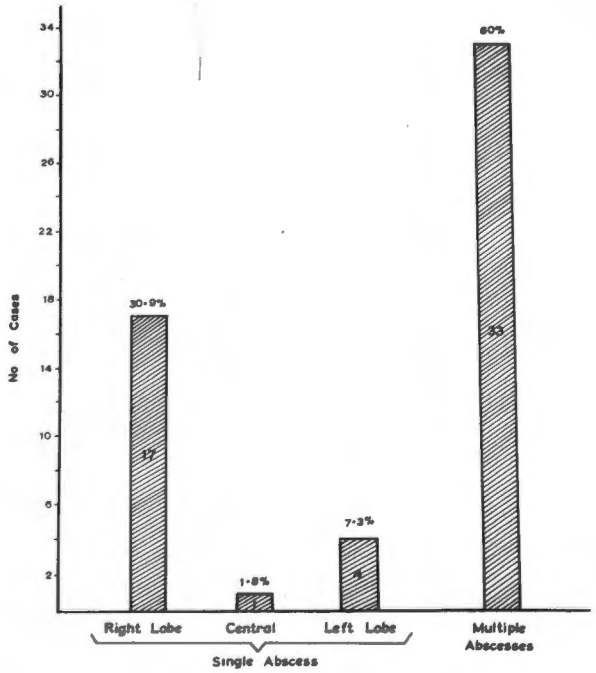

FIG. 6

NUMBER AND LOCATION OF ABSCESSES
AT NECROPSY IN 55 CASES

Chen et al. 1931, Huard et al. 1936b, Tribedi et al. 1951 and DeBakey and Ochsner 1951).

Craig (1944) pointed out that the old idea that amoebic abscesses were usually solitary is most erroneous. It is obvious that the most extensive liver involvement will be shown by fatal cases, so that multiple abscesses are much more frequently observed in necropsy studies than in patients in whom the condition has been recognised and treated.

This is well illustrated by Clark (1925) who reported a solitary abscess in only 42% and multiple lesions in 58% of 95 necropsies. In his series the right lobe was involved in 55.7%. Very similar figures are given by Flores-Barroeta et al. (1959) in 92 necropsy cases.

In children the location and number of lesions appears similar to that reported for adults only where the statistics are compiled from necropsy records.

Rogers (1922) stressed that where multiple amoebic liver abscesses were present the amoebic ulceration of the bowel was clinically active and acute, but usually completely latent clinically in the more chronic single large variety of liver abscess. My findings support this as far as multiple abscesses are concerned. In 32 cases of multiple liver abscesses coming to necropsy, there was acute and extensive amoebic

/ulceration..

ulceration of the colon in all but 10. Two of these 10 showed ulcerative lesions confined to the caecum, in 2 the bowel lesions were healing when death occurred after 14 days and 23 days respectively and in 6 there was no demonstrable lesion. However, the bowel was also extensively involved in 13 of 20 cases of single hepatic abscess at necropsy.

Haematological findings

Leucocytosis. The white blood cell count was done in 90.

Taking a level of 15,000 w.b.c's per c.mm. as a significant rise, there was a leucocytosis in 77 (85.5%). A leucocytosis between 15,000 and 30,000 w.b.c's per c.mm. was observed in 63 and between 31,000 and 50,000 w.b.c's per c.mm. in 14 others. In the remaining 12 it ranged between 6,000 and 14,000 w.b.c's per c.mm. (figure 7). The average for all cases estimated was 23,000 w.b.c's per c.mm.

Torroella et al. (1956) in their series of 14 children reported a leucocytosis of 11,000 to 40,000 w.b.c's per c.mm. in 58%, while Walt (1959) in 16 cases estimated the white cell count in 11 and found a leucocytosis of 15,000 w.b.c's per c.mm. or more in 6 (54.5%).

Reports of leucocytosis in adults suggests that this is present in about 70 to over 80% (Ludlow 1926, Biggam and Ghalioungui 1933, Craig 1934, Manson-Bahr 1944, Rogers and Megaw 1946, Wilmot 1949, DeBakey and Oschner 1951, Lamont and Pooler 1958, Powell 1958 and

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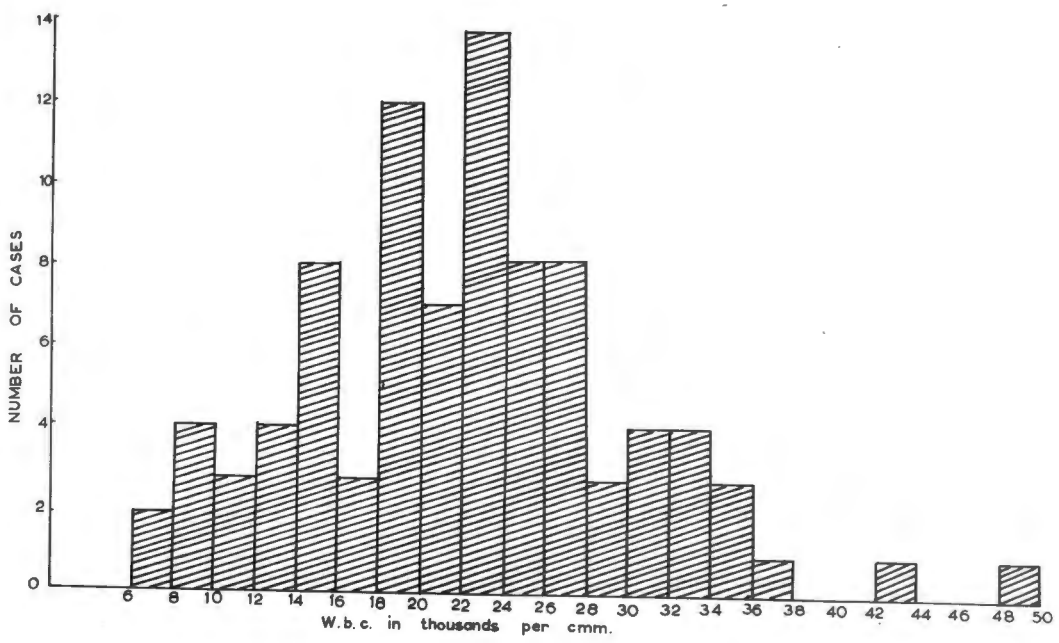


FIG. 7. LEUCOCYTE COUNT — 90 CASES

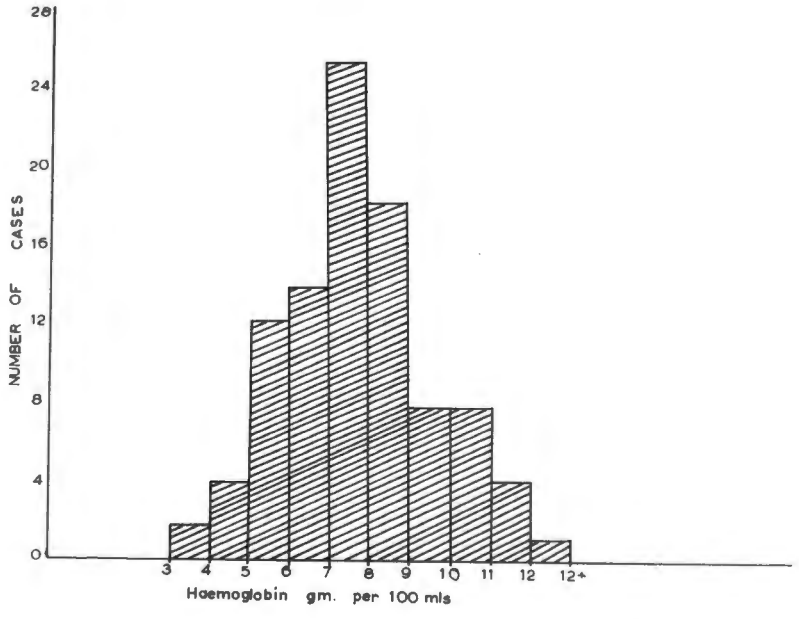


FIG. 8. HAEMOGLOBIN ESTIMATION — 94 CASES

Islam et al. 1960).

My findings in children show that although leucocytosis is found in about the same proportion, the degree of elevation appears greater than in adults.

In adults Lamont and Pooler (1958) found that a short history was related to an appreciable leucocytosis, while those with a long history had a less marked leucocytosis. Powell (1958) found little correlation between the degree of leucocytosis and the duration of symptoms. In my series no correlation was found between length of history and the level of the white cell count.

The size of the abscess (at necropsy or as judged by the amount of pus aspirated) also bore no relationship to the degree of leucocytosis.

Two fatal cases with very high counts were found to have single hepatic lesions at necropsy. Another with a very high count recovered and the two lowest counts of 8,000 and 9,000 w.b.c's per c.mm. respectively occurred in children who at necropsy had multiple hepatic lesions. These findings do not confirm the opinion that a pronounced leucocytosis indicates multiple abscesses with a bad prognosis (Rogers 1922).

Anaemia. Haematological examination was done in 94 (figure 8).

A normocytic, normochromic anaemia (< 10 g./100 ml.) was present in 82 (87.2%). The mean haemoglobin of all cases was 7.7 g./

/100 ml. ..

100 ml. (range 3.2 to 13.2 g./100 ml.).

Anaemia is a common feature of the disease in adults. In Durban, Lamont and Pooler (1958) in a study of amoebic liver abscess in adults, found the degree of anaemia to be related to the duration of symptoms. In my series, the children with the longest histories tended to have the lowest levels of haemoglobin (figure 9). However, this finding might also be expected in many other diseases.

In his adult series Powell (1958) found a closer correlation between the amount of pus aspirated and the degree of anaemia than the length of history and anaemia. In my series of children, however, there did not appear to be a close correlation between these two factors.

Anaemia is an important feature of the disease. Severe anaemia may be present when the patient is first seen or may develop rapidly, so that blood transfusion is quite frequently required.

Liver function tests

Liver function tests were not performed as a routine. Where they were done they appeared to be of no aid in diagnosis.

Fernandez de la Arena y Santé (1956) and Torroella et al. (1956) carried out liver function tests in small series of cases of amoebic liver abscess in children with inconclusive results.

From the cases studied in adults (Sodeman 1950, Givner and

/Chang..

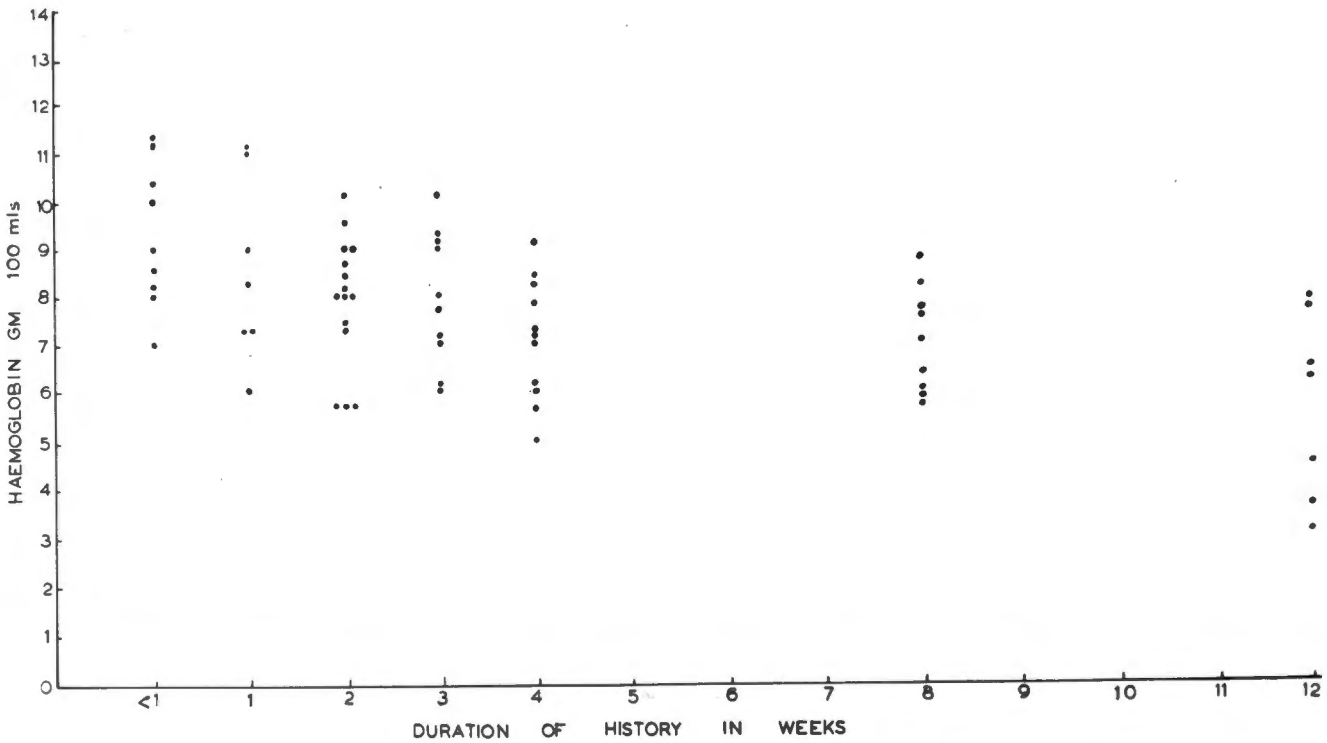


FIG. 9. HAEMOGLOBIN LEVEL RELATED TO DURATION OF HISTORY —— 65 CASES

Chang 1953, Sherlock 1955, Brem 1955, Patterson and Lawlis 1956, Popper and Schaffner 1957, and Powell 1958) liver function tests in amoebic liver abscess have no diagnostic or prognostic significance.

Radiological Investigation. Frequently the patients were considered too ill for this investigation. Many died soon after admission and before X-ray examination. However, radiological investigation was done in 70 (figure 10).

Screening, considered by many to be a valuable aid to diagnosis, could seldom be done because it entailed time and handling of a seriously ill child. Lamont and Pooler (1958) reported that postero-anterior and lateral radiographs of the chest provided the necessary information in 84% of their adult cases without need for screening.

In 21 (30%) of the 70 cases, elevation of the diaphragm was present and was the only radiological finding.

In 3 there was elevation of the dome of the diaphragm but in addition there was evidence of rupture of the liver abscess into the lung (in two instances on the right, and in one of the left side).

Two showed large right-sided pleural effusions due to empyema having resulted from rupture of the liver abscess into the pleural cavity.

In a further 6, although the diaphragm was normal, lung changes -

/usually..

NO ABNORMALITY	33 (47·2%)
ELEVATION DOME OF DIAPHRAGM — NO LUNG CHANGES	21 (30%)
ELEVATION DOME OF DIAPHRAGM — ASSOCIATED RUPTURE OF LUNG	3 (4·3%)
MASSIVE PLEURAL EFFUSION — RUPTURE WITH EMPYEMA	2 (2·8%)
LUNG CHANGES — DIAPHRAGM NORMAL	6 (8·6%)
LUNG CHANGES DUE TO NON-AMOEBIC PATHOLOGY	5 (7·1%)

FIG 10. RADIOLOGICAL FINDINGS — 70 CASES

usually basal, were present. These appeared either as collapse and/ or consolidation.

Five exhibited lung changes due to non-amoebic pathology. Three were due to tuberculosis and 2 to bronchopneumonia.

Thus in 32 (45.7%) there were radiological changes due to amoebiasis. Plates 3 - 7 show the radiological changes present in 5 cases.

It seems that radiology is less often helpful in children than in adults, among whom the majority (80 - 90%) exhibit diaphragmatic elevation (Sodeman and Lewis 1945, Wilmot 1949, DeBakey and Oschner 1951, Lamont and Pooler 1958 and Islam et al. 1960).

History of dysentery. A history of antecedent dysentery with blood and mucus in the stools was obtained in 63. Diarrhoea without blood and mucus was the complaint in 8, and in 29 there was no previous history of diarrhoea or dysentery.

Concomitant unproved amoebic dysentery. Stools were examined in 92. Dysentery with blood and mucus present in the stools was found in 59.

In adults the figures for antecedent or concomitant dysentery vary from less than 30% to over 60% in some series (Mayer-May et al. 1936, Manson-Bahr 1944, Sodeman and Lewis 1945, Banker 1947, Wilmot 1949 and DeBakey and Oschner 1951) while others report this finding as varying from about 50% to over 80% (Rogers 1930, Chen et al. 1931, Biggam et al.

/1933..

Plate 3. Case No. 16. Male child aged 2 years. Radiograph of chest showing marked elevation of right dome of diaphragm. Nineteen ml. of pus removed by transcostal aspiration. Subsequent rupture into lung with recovery

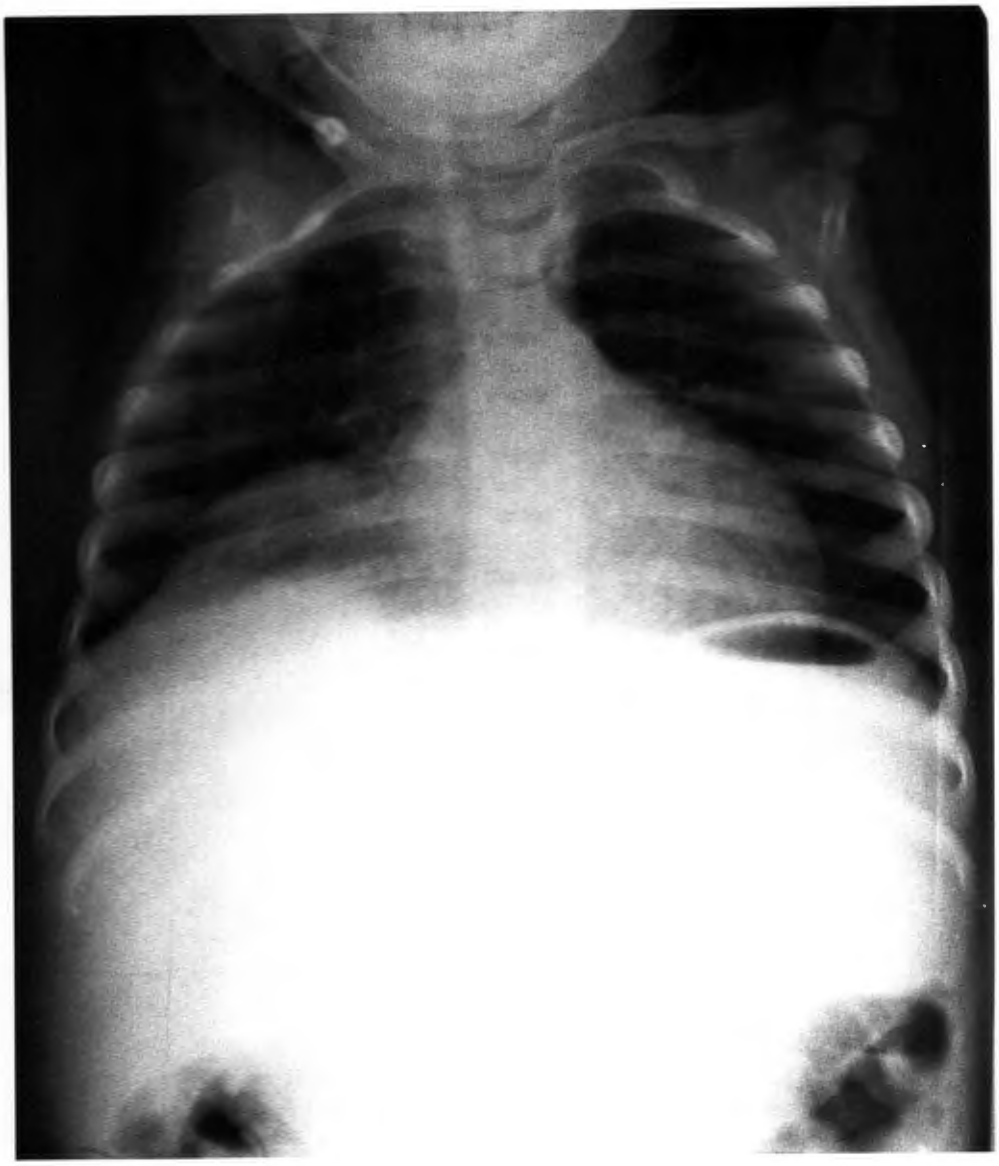
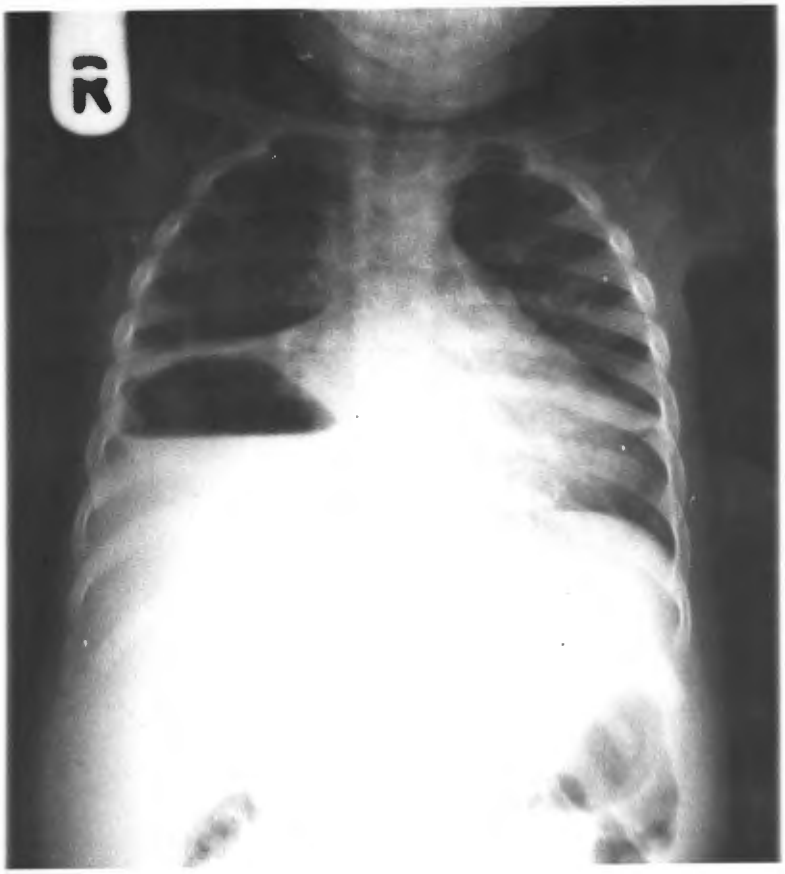


Plate 4. Case No. 41. Male infant aged 11 months. Postero-anterior and lateral radiographs of chest after aspiration (total of 535 ml. of pus removed). Marked elevation of right dome of diaphragm with air in the abscess cavity in the liver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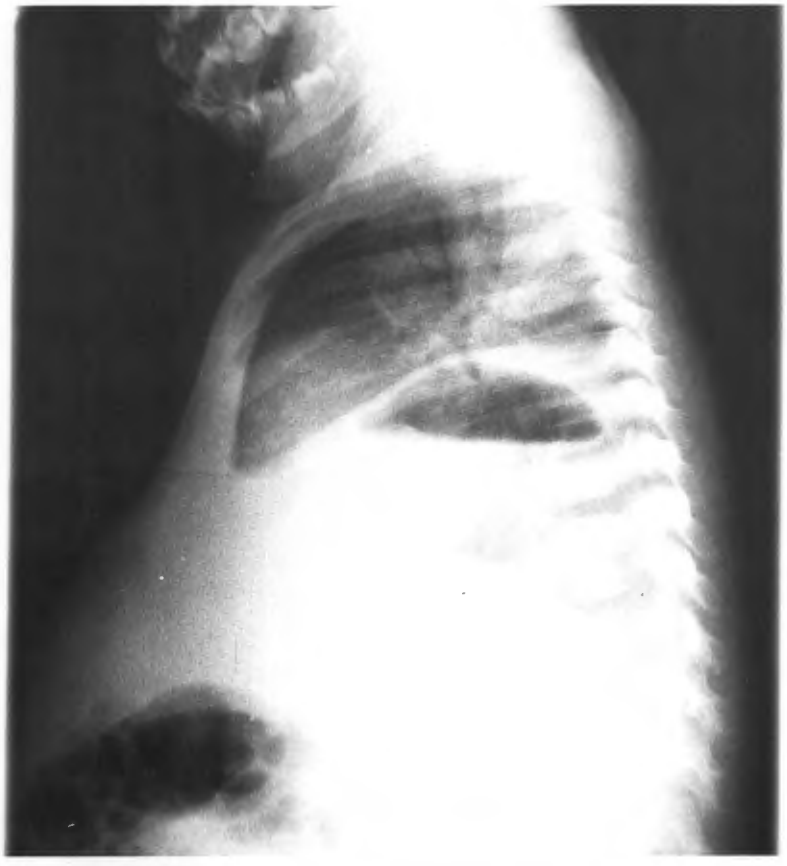
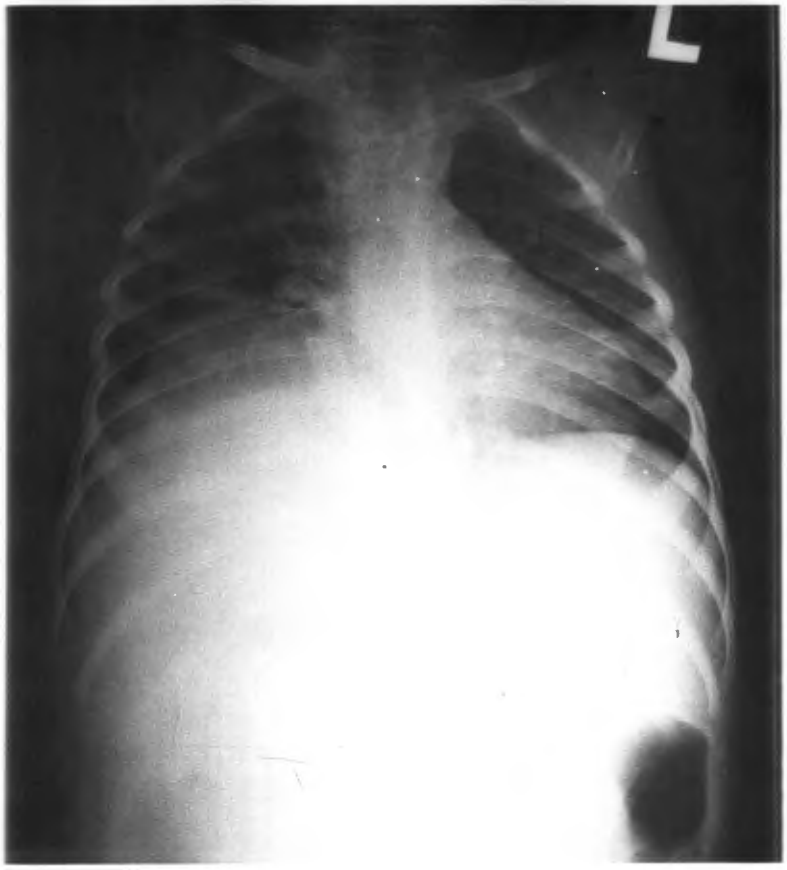


Plate 5. Case No. 45. Male child aged 3 years

(a) Postero-anterior radiograph of chest showing marked elevation of right dome of diaphragm before aspiration

(b) Postero-anterior radiograph of chest after aspiration (total of 390 ml. of pus removed) with air in the abscess cavity in the liver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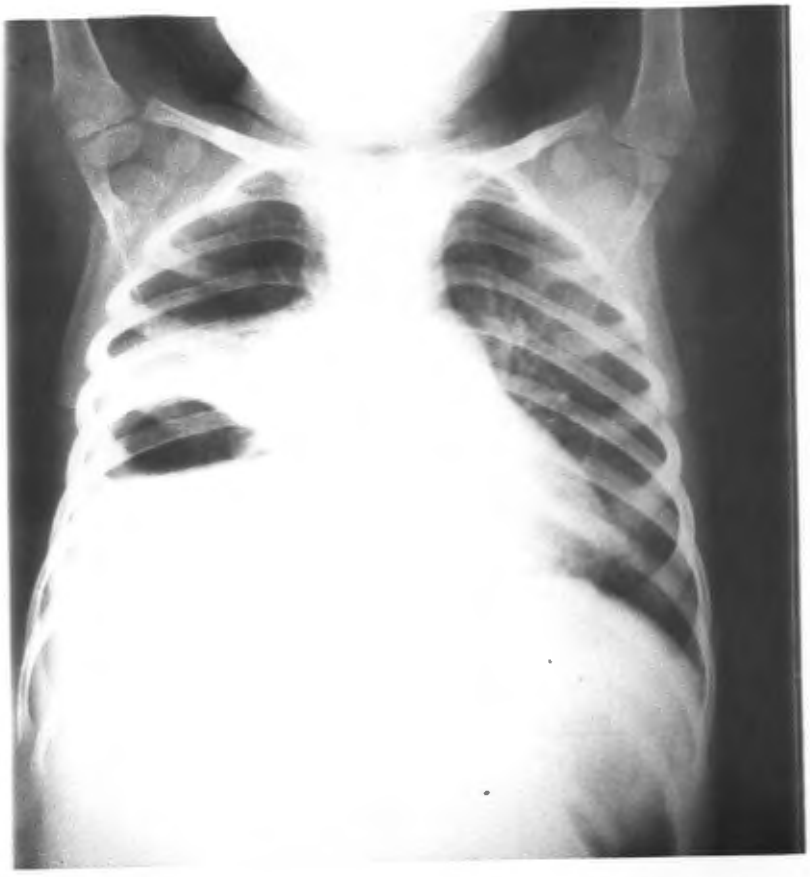
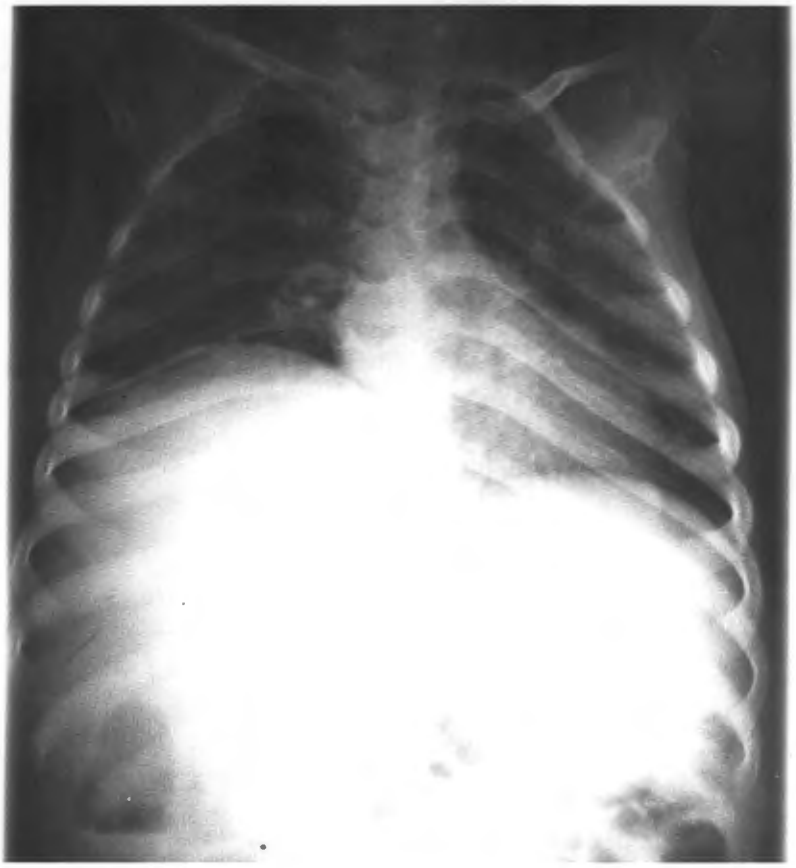


Plate 6. Case No. 62. Female child aged 2 years. Postero-anterior and lateral radiographs of chest showing elevation of right dome of diaphragm before aspiration (total of 926 ml. of pus removed



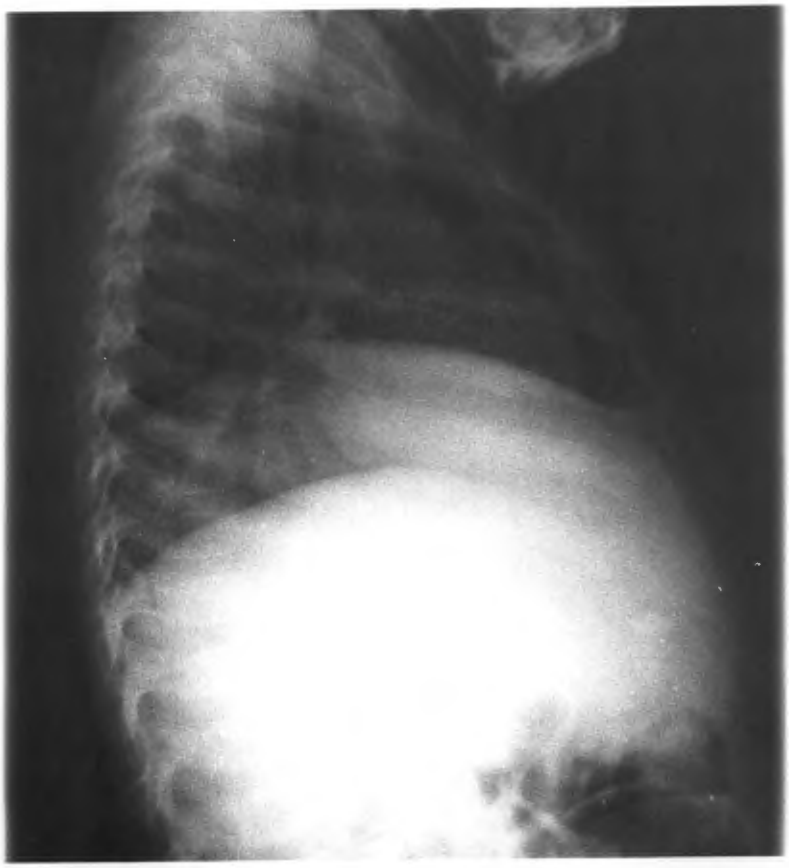
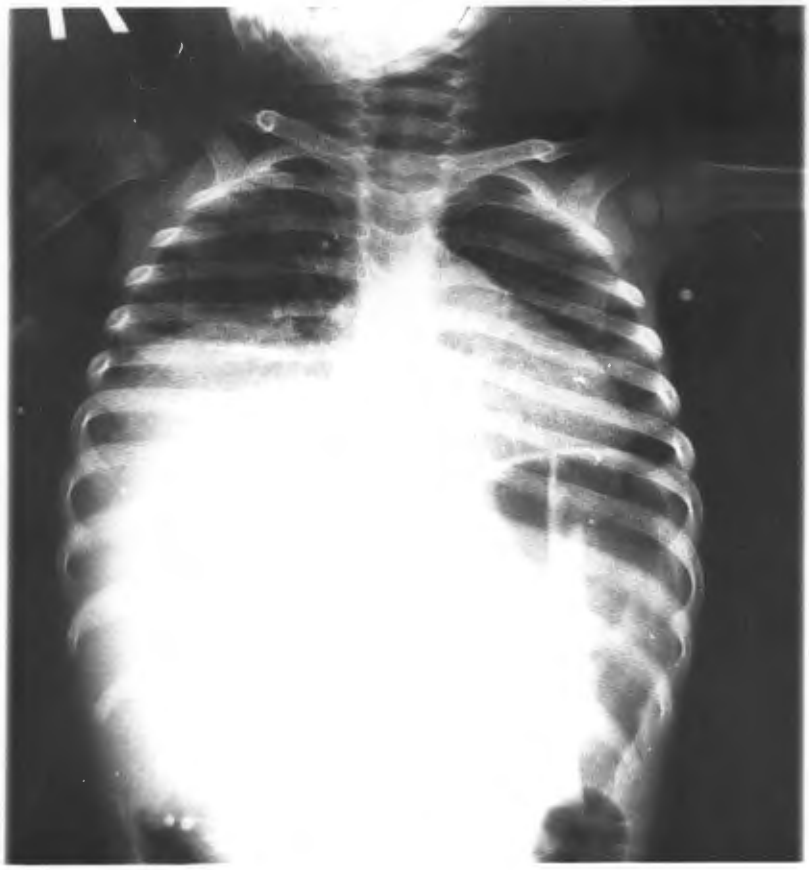
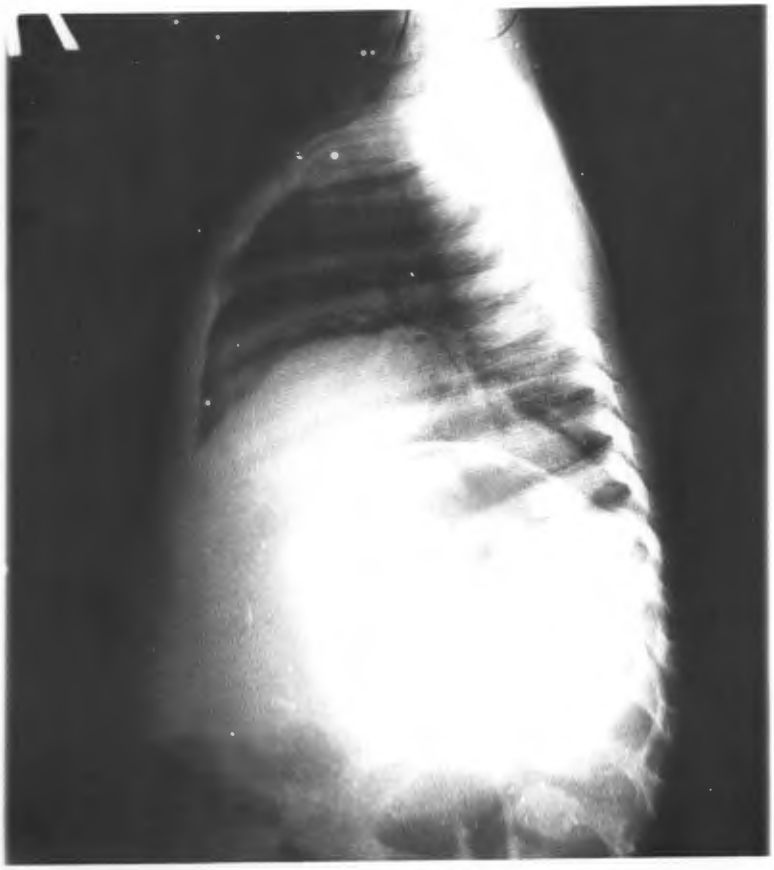


Plate 7. Case No. 107. Male infant aged 20 months. Postero-anterior and lateral radiographs of chest showing marked elevation of right dome of diaphragm. A single transcostal aspiration drained 150 ml. of pus





1933, Palazuelos et al. 1941 and Craig 1944).

Concomitant proved amoebic dysentery. In 59 in whom clinical diagnosis of amoebic dysentery was made E.histolytica was found in the stools of 35 (59.3%). If this figure of 35 is considered in relation to the number in whom stools were examined, then only 38% had "positive" stools. However, in many instances (because of rapid death) only one stool examination was possible.

In the series of 14 children with amoebic liver abscess reported by Torroella et al. (1956), 70% had dysentery with blood and mucus in the stools, but in only 50% was amoebic dysentery proved by finding trophozoites of E.histolytica in the stools.

It has been pointed out (Goldenberg et al. 1952, Young 1955, and many others) that a single stool examination will reveal only about one third to one half of the cases of E. histolytica infection. However, sigmoidoscopy is a valuable aid to diagnosis. A specimen obtained by scraping the bowel wall will often yield a positive result when stool examination is negative.

DeBakey and Oschner (1951) reported finding E. histolytica in 47% of their adult cases, while Castorina (1951) in 56 adults found E. histolytica in 54.5% of stools examined. Sodeman and Lewis (1945) found positive stools in only 29.4%. It seems that in only about half the cases can it be expected to find E. histolytica in the stools.

It is noteworthy that wherever E. histolytica was found in the stools in my series they were in every instance trophozoites. This is in contrast to the disease in adults where amoebic liver abscess frequently occurs when dysentery is quiescent, and an appreciable number of adult cases have cysts of E. histolytica in the stools. Adams and Maegraith (1960) state that it is most unusual for evidence of amoebiasis of the liver to appear during a frank dysenteric attack; it most commonly makes its appearance during a remission.

In 52 cases coming to necropsy, the bowel was free of active lesions in only 13. Three of these 13 had been treated with specific anti-amoebic therapy when death occurred 16, 20 and 36 days respectively, after admission. In 2 others, who died after 14 and 23 days respectively, a full 10 day course of emetine had been given and the bowel lesions showed evidence of healing. In children with amoebic liver abscess it appears, in contrast to the finding in adults, that frank amoebic dysentery is much more likely to be present. In view of these necropsy findings it seems likely that if repeated stool examinations are possible the number "positive" may be expected to be greater than 50%.

Nature of pus

Appearance. Many authors report finding typical "anchovy-coloured" pus. Others refer to the pus as "chocolate-coloured". Craig (1944) stated that of the various descriptive names for the contents of amoebic liver abscesses the best is "anchovy-sauce" or "chocolate-sauce". This author was of the opinion that variations in colour occur when a complicating pyogenic infection is present and the intensity of the latter often results in the contents appearing green or yellow.

Banker (1947) reported that in 78% of 130 cases studied in adults the pus was typically "chocolate-coloured" and in the remainder was "brownish-yellow". DeBakey and Oschner (1951) reported that "in amoebic hepatic abscess the demonstration of "chocolate sauce" pus following aspiration usually establishes the diagnosis, for this type of pus is pathognomonic. Berne (1942) found that in one third of 74 adult cases of amoebic liver abscess the pus was creamy-white in colour.

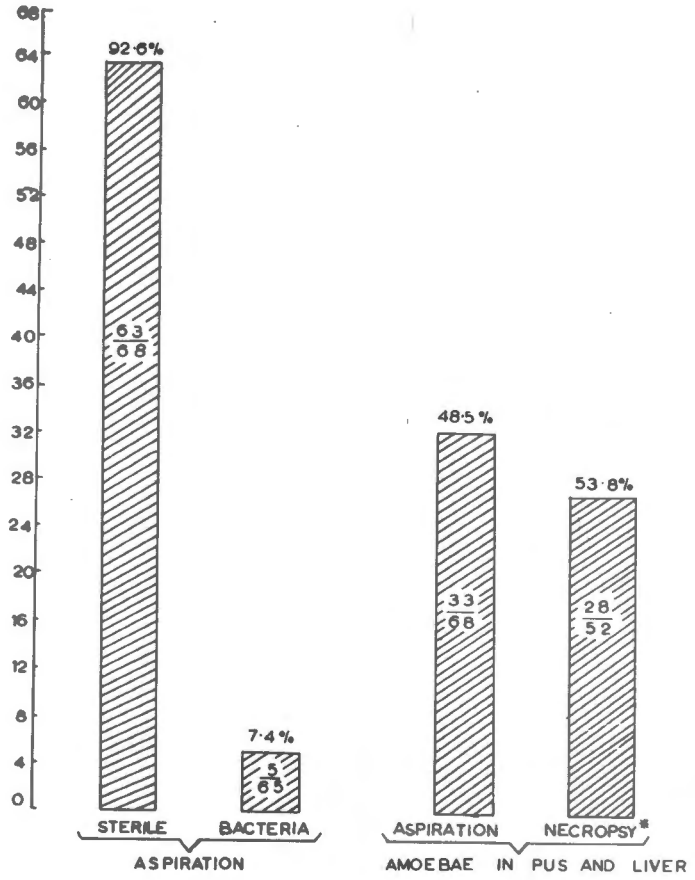
While the aspiration of anchovy coloured pus is usually against an abscess being pyogenic in origin, yellow or even greenish coloured pus may be obtained from amoebic abscesses. In my experience, classical "anchovy" pus should not be anticipated as it is usually grey-green or yellow at first aspiration and only at subsequent aspirations when blood has entered the abscess does it take on the pink or red-brown colour. The colour was no indication that secondary infection was present.

On four occasions at the first aspiration an almost colourless or slightly amber, clear fluid was obtained at the beginning of the procedure, which, as aspiration continued, changed in colour and consistency to resemble classical yellow pus. In each of these E. histolytica was found in the aspirated material. Where an abscess communicates with a large bile duct the aspirate may be coloured by dark green bile.

Roach (1958) found that in 77 cases of amoebic liver abscess the contents were yellow in over 90% of unaspirated abscesses. He concluded that "only rarely at necropsy does the liver abscess contain the "anchovy-type" material considered by many to be characteristic".

Parasitological findings. E. histolytica was found in 33 (48.5%) of 68 aspirated (figure 11). Of the 35 in which E. histolytica was not found 17 had not received any emetine before aspiration. In the remaining 18 emetine had been given for 2 days to 6, and for a period of 3 to 7 days in 11. In 1 a full 10 day course of emetine had been given before aspiration.

Many authors in referring to the incidence of E. histolytica in liver pus, do not state whether their patients were already receiving emetine before aspiration took place. After a grain or two of emetine the likelihood of finding E. histolytica in pus becomes remote.



* 19 NOT ASPIRATED
5 NO AMOEBAE IN ASPIRATED PUS
4 AMOEBAE ALSO PRESENT IN ASPIRATED PUS

FIG. 11. INCIDENCE OF STERILE ABSCESSES AND AMOEBAE IN ASPIRATED PUS AND IN LIVER AT NECROPSY

In 19 cases not aspirated E. histolytica were found in the liver at necropsy. In 5 the aspirated pus did not contain E. histolytica, but these were found in the liver at necropsy. In none of these 5 had emetine been given before death. In 4 E. histolytica was found both at aspiration and necropsy. In all 4 death was rapid, 3 received only 1 dose of emetine and 1 received none.

In a total of 52 necropsies E. histolytica was found in the liver in 28 (53.8%). Combining the figures for "positive" pus and those cases "positive" at necropsy, E. histolytica was thus recovered from the liver in 57%. Plate 8 shows amoebae in sections from 2 necropsy studies.

Opinions differ regarding the incidence of amoebae in the abscess contents in reported series in adults. Meyer-May et al. (1936) found amoebae in the pus in only 7 of 51 cases, Chen et al. (1931) in 65% of 40, DeBakey and Oschner (1951) in 20% of 263, Castorina (1951) in 14% of 56 and Biagi et al. (1958) 68% of 79 cases. According to Manson-Bahr(1944) very rarely can amoebae be demonstrated in aspirated pus.

Torroella et al. (1956) in their series of 14 children found amoebae in the pus of 7 (50%) Biagi et al. (1958) stated that "it seems a fact that in amoebic hepatic lesions in childhood parasites are abundant and practically always encountered in histological study

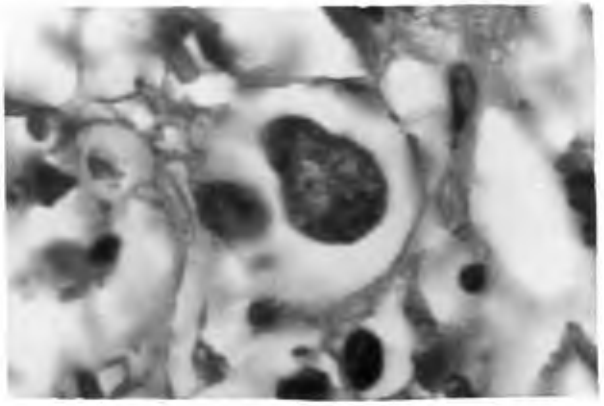
/(Salas..

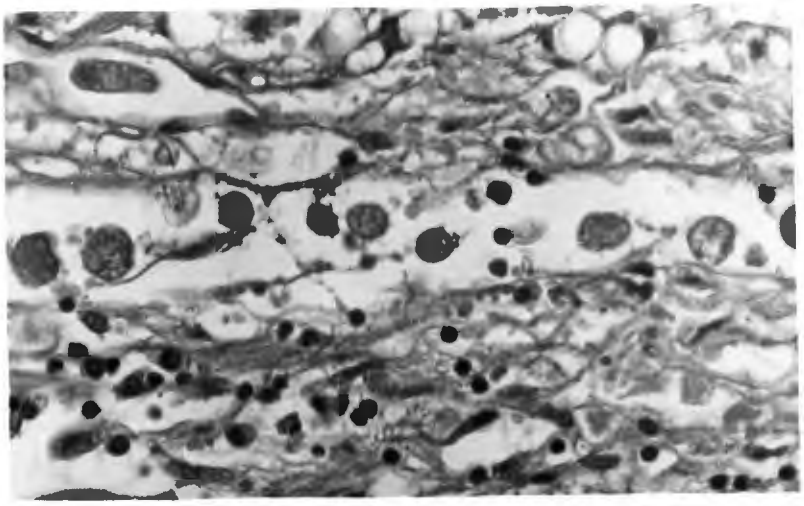
Plate 8.

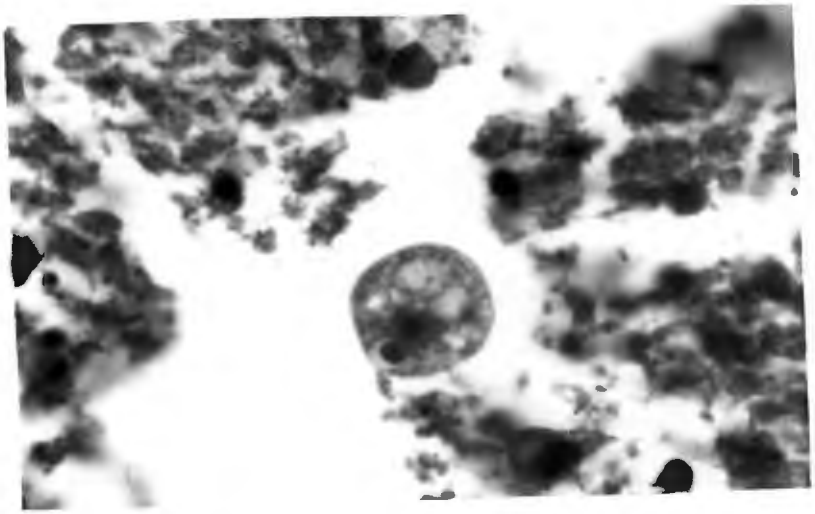
(a) Case No. 33. Amoeba in histological section of liver (approx X 1125)

(b) Case No. 33. Numerous amoebae in histological section of liver (approx X 300)

(c) Case No. 43. Amoeba lying free in pus at edge of liver abscess (approx X 1350)







(Salas et al. 1958) notwithstanding that this is not successful in adults".

Walt (1959) reporting on amoebic liver abscess in children stated that "to find amoebae in aspirated pus, is most difficult and usually unsuccessful". However, the Amoebiasis Research Unit, Durban, in a series of 75 consecutive liver abscesses in adults, have observed and/or isolated amoebae from 84% (Maddison et al. 1959). It is now my custom to submit fresh pus from the first aspiration to this Unit and amoebae have been found in 22 of the last 28 specimens of pus examined.

The specimen should be obtained towards the end of aspiration when the amoebae in the wall of the abscess are more likely to find their way into the pus.

Bacteriological findings. The pus is characteristically bacteriologically sterile and was so in 63 (92.6%) of 68 cases aspirated (figure 11). In 4 bacteria were isolated from the first aspirate. E. histolytica was also present in 2 of these. In another, bacteria were found in the second aspirate. In one further case, the pus was sterile and contained E. histolytica and secondary infection only occurred when a small sinus from a residual subcutaneous abscess formed 32 days later, aspiration having long since ceased. In none of these cases had rupture occurred.

This finding is in accordance with reports by many authors. Rogers (1922) found 86% of 87 cases of amoebic liver abscesses to be bacteriologically sterile. Chen et al. (1931) reported sterile

/abscesses..

in 82% of their patients.

Maddison et al. (1959) carried out a study of the liver pus from 75 consecutive adult cases of amoebic liver abscess. In no instance was the abscess infected by bacteria on the initial aspiration. Six cases subsequently became infected. These authors rightly point out that unless reports make a distinction between ruptured and unruptured abscesses, the figures given for the incidence of bacterial infection in amoebic liver abscesses are misleading.

Complications

Rupture of abscess. Rupture occurred in 18 cases (15 of them fatal).

The following brief table summarises the site of rupture in order of frequency and the outcome:

Rupture into pleural cavity	5	(5 deaths)
" " peritoneal cavity	5	(5 ")
" " lung	4	(2 ")
" " lung and pleural cavity	2	(2 ")
" through abdominal wall	2	(1 ")
	18	

Six of these cases were moribund on admission and died within 12 hours, 3 with rupture into the pleural cavity, 2 with rupture into the peritoneal cavity and 1 with rupture into both lung and pleural cavity.

No case of frank rupture into the pericardium occurred in this series. However, in 1 case (a 6 month old infant with multiple amoebic abscesses of the liver) a fibrinous pericarditis was present at necropsy with an abscess in the left hepatic lobe adherent to stomach, diaphragm and pericardium, without frank rupture being present. In this case E. histolytica was demonstrated in the liver. In another case, recorded above as rupture into the lung, a second abscess of the left hepatic lobe (with E. histolytica demonstrated) was adherent to the pericardium with a fibrinous pericarditis, but again no frank rupture was shown.

An infant of 7 months who had had 2 abscesses aspirated on 7 occasions in all, was found at necropsy to have a very large abscess in the right lobe which had extended to form a perinephric abscess on the right side, but this had not ruptured into the kidney. There was, in addition, another healing abscess of the right lobe.

Manson-Bahr (1944) gave a figure of 11% of adult cases complicated by rupture, stating that rupture was most apt to occur into the pleura with lung and pericardium the next commonest sites.

Craig (1944) compiled the observations of 8 authors. Out of 624 cases of amoebic liver abscess in adults, 192 (30.8%) were accompanied by rupture into some cavity or viscus. The commonest site was pleura (70) then lung (47), pericardium (37), stomach and lumbar region (each 8) and colon and inferior vena cava (each 6). Bile ducts, duodenum and kidneys accounted for the remainder. None were reported to have ruptured into the peritoneal cavity. In contrast, Wilmot (1949) found rupture into the peritoneal cavity (11 instances) to be the commonest site among 20 cases of rupture. Kean (1956) and Flores-Barroeta et al. (1959) also found the abdominal cavity to be among the commonest sites of rupture.

In children rupture of an abscess appears to occur with the same frequency as reported for adults. In a few series reported in children Torroella et al. (1956) found rupture in 28% of 14, Salas et al. (1958) in 35% of 17 and Macdougall (1960) in 20% of 10 cases.

Rupture into the pericardial cavity, a rare event in adults (Kern 1945), has been reported in children. Torroella et al. (1956) and Salas et al. (1958) each reported this complication in 2 patients.

The following summary illustrates the difficulty encountered when the case presents with rupture having already occurred:

> A female child of 2 years, with kwashiorkor, was admitted gravely ill. No history was available. The liver was noted to be 8 cm. below the costal margin and was tender. There were

/signs..

signs of right pleural effusion. The stool examined on admission, contained blood, mucus and E. histolytica. She died 6 hours later. Necropsy disclosed a single abscess in the right hepatic lobe which had ruptured through the diaphragm, with a resultant empyema on the right side. There was, in addition, pneumonic consolidation of the lung, and the whole of the large gut showed extensive amoebic ulceration.

Brain abscess. This occurred in 2 cases (28 and 94). In neither were there clinical signs to arouse suspicion of brain abscess. In both, amoebae were found in the brain tissue. The first was an infant of 5 months with a very large abscess in the right hepatic lobe and multiple small abscesses in the left hepatic lobe. The second was a 3 year old child who also had a very large abscess in the right hepatic lobe which had ruptured into the right pleural space and there were multiple abscesses in the right lung. This child showed no evidence of bowel ulceration.

This rare complication of amoebiasis almost invariably follows on abscess of the liver and/or lung. Cytolysis of tissue is a feature and amoebae are found in that part of the tissue in immediate contact with the inner surface of the abscess cavity (Craig 1934).

Kartulis (1904) stated that brain abscess occurred in 3% of his cases of amoebic dysentery. Legrand (1912) collected particulars of 45 cases, 26 of which occurred in Egypt. He was of the opinion that the proportion of 3% reported by Kartulis was much too high. Two reported by Legrand were in children, one of 5 years (his own personal

/case..

case) and one of 14 years. However, the former was a clinical diagnosis of brain abscess in a proved case of hepatic amoebiasis, but necropsy was refused.

Clark (1925) reported the brain involved in 4 of 186 necropsies on cases of amoebiasis. In 1942 Stein and Kazan claimed to report the 60th case in the literature stating that in only 15 of the 60 were amoebae actually seen in the brain. Orbison et al. (1951) collected 83 reports of this condition in 22 of which amoebae were demonstrated in the brain lesion. Kean et al. (1956) reported 2 in which the amoebic aetiology was definitely established. Recently reporting from Durban, Powell and Neame (1960) reported another proved case in an adult African male.

With regard to reports in children, Jiménez et al. (1947) quoted the unpublished case of Aballi and Labourdette of an amoebic brain abscess which occurred in an infant of 8 months in Cuba in 1941. Cardelle et al. (1948) also recorded the occurrence in an infant of 14 months in Cuba. Other single instances of this rare complication in children have been reported by Herrera (1952), De la Maza et al. (1953), Fernandez de la Arena y Santé (1956) and Vizcarrondo et al. (1959).

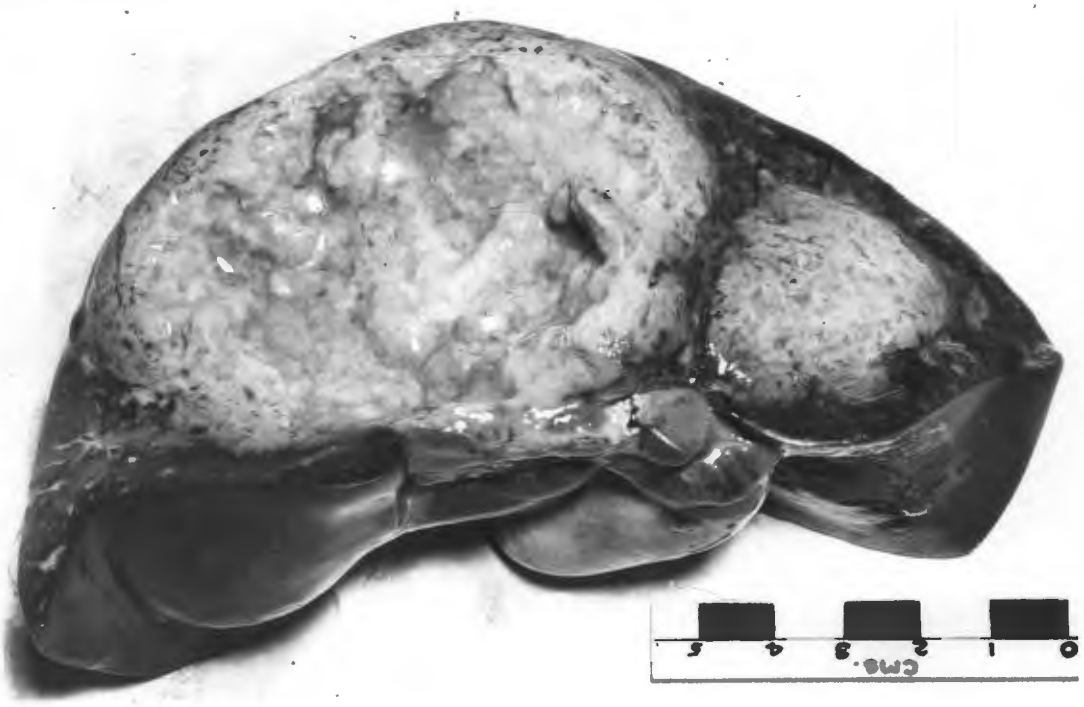
Jaundice. Clinical jaundice occurred in only 2 cases (38 and 84). One was a moribund infant of 4 months who was admitted deeply jaundiced.

/Death..

Plate 9. Examples of amoebic liver abscesses producing jaundice by mechanical obstruction to bile ducts

(a) Case No. 38. Male infant aged 4 months. Deep clinical jaundice and a large irregular liver. Practically the entire liver substance is occupied by 2 large abscesses, producing mechanical obstruction to bile ducts and a mucocoele of the gallbladder

(b) Case No. 84. Female child aged 2 years. Large liver with increasing clinical jaundice. Seven large abscesses occupy almost the entire liver. Two abscesses in the porta hepatis accounted for the biliary obstruction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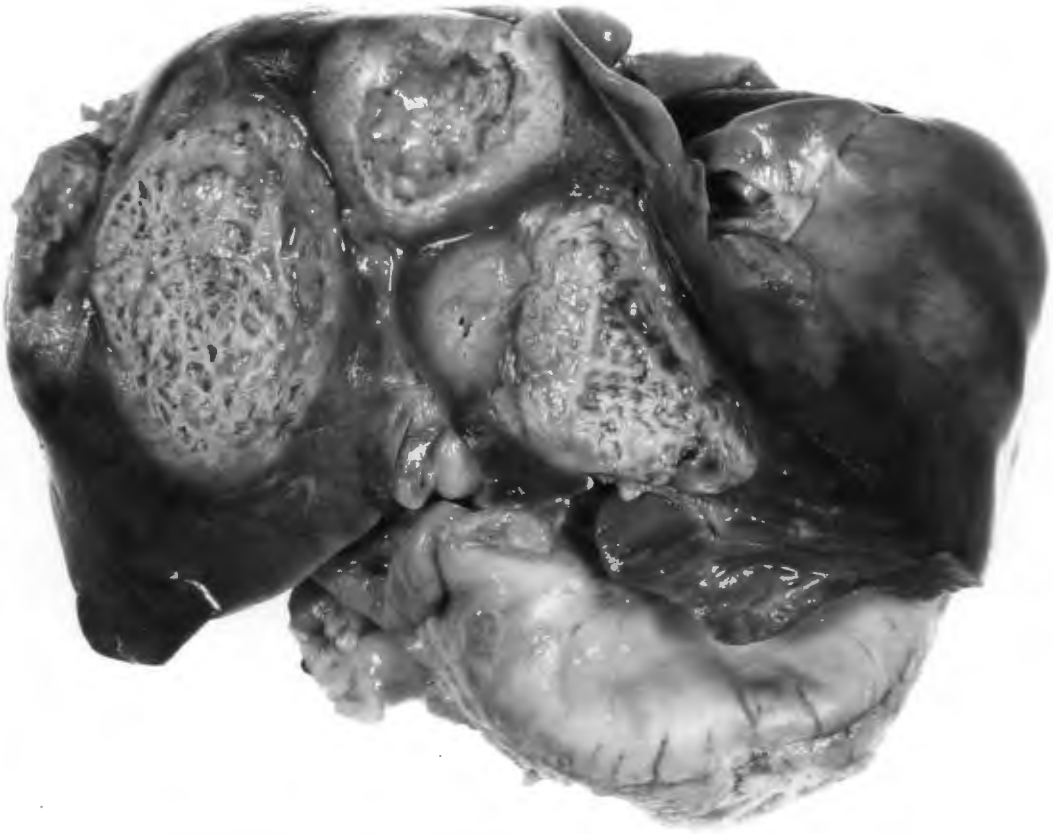

Death occurred one and a half hours after admission. At necropsy there were 2 large abscesses (with amoebae present) producing mechanical obstruction to the biliary system with a mucocoele of the gallbladder (plate 9a). The second was a 2 year old child, who developed progressive jaundice and at necropsy was found to have 7 large amoebic abscesses occupying almost the entire liver (plate 9b). Two abscesses in the region of the porta hepatis evidently accounted for the obstructive jaundice.

Jaundice as an unusual complication of amoebic liver abscess has been described (Rogers and Megaw 1946, Manson-Bahr 1944). Reddy et al. (1945) reported 2 instances in which mechanical obstruction (to the right hepatic and common bile duct respectively) by amoebic liver abscesses caused jaundice.

DeBakey and Ochsner (1951) reported an incidence of jaundice in 12.9% of their 263 cases of hepatic amoebiasis, while Kean (1956) in a series of 148 fatal cases of amoebiasis reported an incidence of jaundice of 14% in both those patients with and without liver abscess. Lamont and Pooler (1958) found jaundice in 6 of their 16 fatal cases.

Jaundice is thus an infrequent complication, but when present suggests a serious prognosis.

Mortality

There were 62 deaths. No cases have been excluded, however

/short..

short the survival. This figure includes 1 case who, in addition to 3 amoebic liver abscesses, had pneumococcal meningitis secondary to otitis media.

In this series of 100 proved cases 29 died within 48 hours of admission. The duration of survival of these cases is shown below:

11	-	died within 6 hours of admission
6	-	" " 7 - 12 " "
5	-	" " 13 - 24 " "
7	-	" " 25 - 48 " "

Hepatic amoebiasis untreated is usually fatal. If one excludes those in whom specific anti-amoebic therapy with emetine and chloroquine was not given (15 cases) and those in whom rapid death precluded the giving of more than one or two doses of emetine and chloroquine (24 cases) the mortality rate was 23%. If this figure is further corrected by including 14 cases of almost certain amoebic aetiology, then the mortality rate was 21.1%.

The prognosis in infants 1 year or less in age appears very grave. It will be noted (figure 1) that only 5 infants recovered out of the 32 cases in this age group.

The expected mortality rate in children has not been assessed as only one large series (Scragg 1960) in children has been reported.

Most authors reporting on the complication of amoebic liver abscess in childhood stress the high mortality. Published series in children report the following mortality rates: Torroella et al. (1956) 85.7% of 14 cases; Walt (1959) 25% of 16 cases; Macdougall (1960) 90% of 10 cases. The 17 cases reported by Salas et al. (1958) were necropsy studies, only 7 of which had been diagnosed during life. These authors did not state the number, if any, which recovered during the 9 year period under review.

From the cases of amoebic liver abscess in children collected from the literature (appendix 1) where the outcome is stated, it can be assessed that in 211 cases there were 76 deaths, a mortality rate of 36%.

In adults the mortality rate appears to be very much lower. Rogers (1922) reported a rate of 14.4% of 111 collected cases conservatively treated. Thurston (1924) recorded a rate of 14% of 64 cases. Manson-Bahr (1944) stated that in the early years of the last century the mortality rate for amoebic liver abscess was very high, about 50 - 80%, but with advances in diagnosis and treatment this had fallen considerably. In his series it was 6%, which he stated was probably the average death rate.

Wilmot (1949) found a mortality rate of 11.7% in 77 consecutive adult Africans with proved amoebic liver abscess. DeBakey and Ochsner (1951) reported an overall mortality rate of 22.2%. In their own series the mortality rate was 100% with multiple abscesses, but only 11% with

/single..

single lesions.

Multiple lesions undoubtedly worsen prognosis. Of the 62 cases dying in my series, in 52 a necropsy was performed. Thirty-two (61.5%) of these exhibited multiple hepatic lesions (figure 7). Plates 10 and 11 are examples of extensive hepatic necrosis.

The prognosis is dependent in some measure, too, on the extent of the primary amoebic lesions. Thus, of 52 coming to necropsy, the bowel was free of active lesions in only 13. Thirty-nine had extensive amoebic ulceration, 8 of these, in addition, having peritonitis, which undoubtedly contributed to the high death rate.

It appears that in children the hepatic complication supervenes more acutely during the phase of active amoebic colitis than is the case in adults, in whom a latent period of many months or years may elapse between the primary bowel lesion and the hepatic complication.

If the hepatic complication is recognised and promptly and adequately treated, it would appear that the mortality rate to be expected in children is probably about 25%, which is appreciably greater than that for adults.

Plate 10. Examples of extensive liver necrosis

Case No. 92. Liver from a female child aged 2 years. A single aspiration of a mass in the right hepatic lobe drained 94 ml. of pus. Necropsy showed that rupture into the right pleural space had occurred. There was in addition an abscess centrally placed in the liver

Case No. 124. Liver from a female child of 14 months. (Not in the present series). Aspiration of an abscess in the right hepatic lobe drained 45 ml. of pus. Necropsy showed 8 large and many small abscesses in the liver

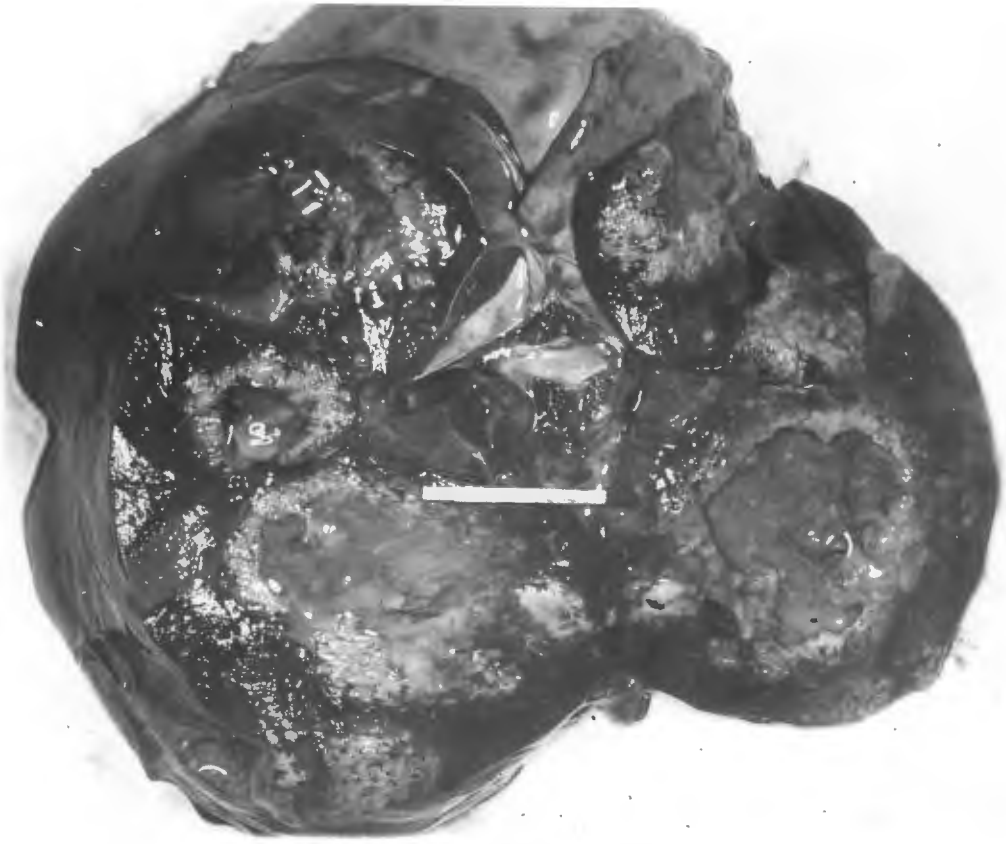

Plate 11. Examples of extensive liver necrosis

Case No. 105. Liver from an infant aged 10 months. From the right hepatic lobe a total of 410 ml. of pus had been drained (5 aspirations). Necropsy showed a chronic healing abscess in the right hepatic lobe and another abscess in the left hepatic lobe

Case No. 107. Liver from a child aged 20 months. A single transcostal aspiration drained 150 ml. of pus. Necropsy showed the diaphragm adherent to the right hepatic lobe. The liver contained 2 very large and many smaller abscesses


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2
3
4
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Treatment

Specific Anti-amoebic Therapy

Emetine. All cases in which the correct diagnosis was made received emetine hydrochloride. Many died so rapidly that there was time for only one or a few doses. In those that survived long enough emetine was given for 10 days, the daily dose varying from gr. $\frac{1}{8}$ to gr. $\frac{1}{4}$ intramuscularly, depending on the weight of the patient.

It was occasionally necessary to repeat the course of emetine before complete resolution occurred. Thus 10 cases received a further 7 day course of emetine.

Chloroquine. Chloroquine diphosphate or chloroquine sulphate was used in an initial dose of 300 mg. of base, followed in 6 hours by 150 mg. of base, followed thereafter by 75 mg. of base twice daily for 21 days.

Diiodohydroxyquinoline. This was frequently used where dysentery was present. The usual dose was 200 mg. thrice daily for 15 days.

These drugs were well tolerated by the children. In no instance were there obvious signs of toxicity.

Antibiotics.

(i) Penicillin was used during the period when aspirations were carried out as a precaution against secondary infection.

(ii) Oxytetracycline or chlortetracycline was used when dysentery was present in those patients in the series up till the end of 1956, the usual dose being 125 mg. 6-hourly for 6 to 7 days.

(iii) From 1957 onwards, tetracycline (125 mg. 6-hourly for 6 to 7 days) was the antibiotic used in the presence of dysentery.

Aspiration of the abscess. This was done in 68 cases.

Twenty-two were aspirated once only, 33 on 2 to 4 occasions, 10 on 5 to 8 occasions. Three required 12, 13 and 17 aspirations respectively. From this latter patient 1,688 cc. of sterile pus was removed.

The often-stressed danger of haemorrhage caused by aspiration was not encountered, despite the fact that pre-aspiration emetine had frequently not been given, or perhaps only one dose had been administered. It was felt that the danger from rupture was much greater than that from haemorrhage, so that aspiration was usually carried out as soon as the diagnosis was made.

Surgical drainage. One 5 month old infant, after a single aspiration, was treated by surgical drainage with good result. Another child of 2 years, after 7 aspirations, developed a midline sinus with secondary infection and a residual small abscess was evacuated surgically. Rapid and complete cure followed. A third child, aged 3 years, was found to have secondary infection of the abscess and, after the second aspiration, was submitted for surgical drainage; cure followed. A fourth patient

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was submitted for surgical drainage after 8 aspirations because the quantity of pus was not decreasing and general improvement had not occurred. After drainage of a very large abscess in the right hepatic lobe complete recovery resulted.

Supportive measures

Intravenous fluids and blood transfusions were used when indicated. The maintenance of hydration and electrolyte balance is of paramount importance in the presence of serious dysentery.

Discussion

The use of emetine in children has been the subject of much controversy for many years. There have been widely conflicting beliefs that emetine is well tolerated by children (Archibald 1914), while others have stressed its danger and contra-indication (Buchman 1926, Merle 1949). Some have stated that extreme caution is required (Petzetakis 1924). Leroy des Barres (1930) was of the opinion that infants and children tolerate emetine poorly. There are authors who have claimed unsatisfactory results with emetine in children (Maggiore 1923). Deutsch (1927) believed that the usual very small doses recommended for children were ineffective. Teitel (1929) stated that there was little danger if a good quality preparation was used in appropriate doses in children.

There are reports of peripheral neuritis following emetine (Kilgore 1916) and death in children as a result of emetine (Snell 1915, Buchman 1927, Stern 1928).

The report of Snell (1915) was of amoebic dysentery in a 5 year old child who died from emetine poisoning. It is hardly surprising that death occurred considering that the child initially received gr. $\frac{1}{3}$ intravenously, followed in a haphazard way by very large doses (for this age) varying from gr. $\frac{1}{2}$ to gr. 1, to a total dose of gr. $10\frac{2}{3}$.

The toxic signs reported by Kilgore (1916) occurred in 4 children between the ages of 4 and 8 years, the total doses varying from 4 to 6 grains, which must be considered excessive for the ages and probable weights of these children. It is not surprising that trouble ensued.

Few fatalities from emetine have been reported. These have usually resulted from excessively large doses. Providing the recommended dose and duration of treatment is not exceeded emetine still appears to be the drug of choice in amoebiasis, especially in the presence of hepatic complications. The dangers of emetine as a "myocardial toxicant" have been grossly exaggerated in the past (Adams 1956).

Rail (1947), too, in evaluating the treatment in 150 adult cases of amoebiasis stated that "the toxic effects of emetine had been conspicuous by their absence".

Emetine remains the most effective drug available for the

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clinical control of amoebiasis and its complications and there is no other drug which can equal it in this respect (Adams 1952). Although emetine cannot be relied upon completely to eradicate the primary bowel infection, when given in the presence of an extra-intestinal lesion, such as hepatic abscess, it will sterilize the latter.

Wilmot (1949) showed a relapse rate of 7.7% in 39 adult cases receiving a single course of emetine compared with a relapse rate of less than 1% in 40 cases receiving a second course. In the children under discussion it was necessary to repeat the course of emetine in 10 cases, in none of whom were any obvious toxic signs encountered.

Care should be exercised in its use and the advocated dose should not be exceeded. The usual adult dose of 1 grain (0.065 gm.) daily for 10 days has been variously adapted for children. Some authors appear to be over-cautious, advocating very small doses such as one-twelfth to one-twentieth of the adult dose. A dose of 1 mg. per kilogram of body weight per day for children is recommended by most authors (Strong 1945, Torroella *et al.* 1956, Moreno *et al.* 1956, Platou and Beaver 1958).

Chloroquine is a drug of great value in hepatic amoebiasis, possessing, as it does, amoebicidal qualities, almost complete absorption from the intestine and considerable localization in the liver

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(Conan 1948, Berliner et al. 1948, Conan et al. 1950).

Since the efficacy of chloroquine in amoebic liver abscess was first described (Conan 1948 and 1949, Murgatroyd et al. 1948, Manson-Bahr 1949, Emmett 1949, Sharma 1951) it has been widely and successfully used in this condition. Some advocate chloroquine as the drug of choice in this condition (Lane 1951), others feel that there is little to choose between chloroquine and emetine (Woodruff 1959). However, chloroquine therapy alone is followed by a significant relapse rate (Harinasuta 1951, Wilmot et al. 1958) so that a combination of chloroquine and emetine is probably the best form of drug treatment for amoebic liver abscess, despite the fact that some authors do not recommend their simultaneous use (Platou and Beaver 1958).

Chloroquine seems to be singularly non-toxic (Alving et al. 1948) and even in infants and small children the drug has been found to be harmless (Berberian et al. 1948).

Antibiotics have a place in the treatment of amoebic dysentery (Hargreaves 1945, Armstrong et al. 1949, 1950 and 1952, Most et al. 1950 and 1951, and others). Of these the tetracyclines have proved to be the most useful (Elsdon-Dew et al. 1952, Elsdon-Dew 1955 and Wilmot 1955). Antibiotics are of no value in the treatment of amoebic liver abscess (Killough et al. 1951, Wilmot et al. 1952 and 1958, Martin et al. 1953, Sadun et al. 1956).

The probability of sterilizing the infection is maximal when several drugs, probably acting in different ways, are given concurrently (Adams 1952). Powell et al. (1960) have shown that chloroquine apparently has a potentiating effect on tetracycline and when the three drugs tetracycline, diiodohydroxyquinoline and chloroquine were given together the success of treatment was 94%. These authors conclude, therefore, that it seems reasonable that chloroquine should be given in addition, not only to safeguard the liver, but also because of the enhanced efficacy of combined therapy.

As amoebic dysentery occurs commonly among African children in Durban and as the hepatic complication occurs not infrequently, it is our practice to treat all cases of amoebic dysentery with the combination of the above three drugs.

Faust and Jung (1956) advocated that "in all cases of intestinal amebiasis chloroquine should be given as prophylaxis against amebic hepatitis or liver abscess. While amebic liver abscess is uncommon in children, in consideration of its gravity and the low cost and low toxicity of the drug, prophylaxis is indicated".

With regard to aspiration of the abscess, most authorities agree that conservative treatment, consisting of specific anti-amoebic therapy combined with aspiration repeated as necessary, is the treatment of choice, except where secondary infection is present. DeBakey and

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Ochsner (1951) showed a striking difference in mortality with conservative therapy, consisting of emetine with or without aspiration and open surgical drainage.

Wilmot (1960) considers that the main indications for open drainage are:

(i) Failure to aspirate pus from patients with suspected liver abscess whose condition is not responding to emetine and/or chloroquine.

(ii) Secondarily infected abscesses which do not respond to aspiration and local and systemic antibiotics.

(iii) In some cases after rupture has occurred, in order to drain the pus from other loci.

(iv) Cases not improving despite repeated aspiration and specific therapy.

In retrospect, in view of the high mortality rate, it might have been advisable to undertake surgical drainage in a few selected patients who required numerous aspirations. For instance 3 (Nos. 5, 41 and 79) had repeated aspirations and failed to show the expected rapid response to this and specific anti-amoebic treatment. At necropsy on 2 of them it was felt that adequate surgical drainage might have saved these children. In the remaining case necropsy was refused. A further patient (No. 60) died after a total of 12 aspirations (7 from an abscess in the right and 5 from an abscess in the left hepatic lobe). This case

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at necropsy showed that while 2 large abscesses had been satisfactorily drained the liver was studded with multiple abscesses varying in size from 1 cm. to 5 cm. in diameter. It is doubtful if surgery would have saved the child.

The necessity to aspirate repeatedly is not an absolute indication that surgical drainage is indicated. In 3 cases (Nos. 51, 62 and 70) although many aspirations (13 in the former and 7 in each of the latter 2) were necessary before complete recovery, there was at no time anxiety about the outcome, as these children showed continued improvement and the amount of pus though large at first, steadily decreased at each aspiration.

CONCLUSIONS

The literature reveals that the hepatic complication of amoebiasis in childhood appears to be much less frequent in most parts of the world than it is in Durban. Even in many other areas where amoebiasis is common, this complication is regarded as distinctly rare in childhood, as judged by the isolated case reports.

In this study of the particular complication of amoebic hepatic abscess in Natal African children, stress has been laid on the usual clinical features to be expected in this disease. These features have been compared and contrasted with the condition in adults.

The noteworthy features of difference in children from those in adults are:

1. The hepatic complication supervenes more acutely and often at the height of the primary intestinal lesion.
2. The distinct predominance of the complication in the male sex in adults is not apparent in children.
3. Fever is a more striking feature.
4. Abdominal distension, which may cause difficulty in diagnosis, appears to be a more common finding in children than in adults.

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5. Severe anaemia is a commoner feature.

6. Leucocytosis while occurring in about the same proportion of cases in adults, appears to be of a higher order in children.

7. Radiology is less often helpful in children than in adults.

8. Multiple hepatic lesions appear to occur more frequently than in adults.

9. The complications of amoebic liver abscess are similar to those in adults and while rupture of the abscess occurs as frequently and the sites of rupture are similar, few cases of rupture of the abscess in children have survived with the exception, perhaps, of rupture into the lung. This is in contrast to this complication in adults, in whom there are numerous instances of rupture followed by a favourable outcome.

10. The overall mortality is very much higher than in adults. Even allowing for correction of the rate by exclusion of cases of rapid death and those not receiving any or only a few doses of the recommended specific therapy, the mortality rate is still much greater than that in adults.

The great difference between the mortality rate in adults and children requires further consideration. In adults, death is uncommon if there are no complications and cure usually results with frequent aspiration and specific anti-amoebic therapy.

A number of children die in which there seems to be an insufficient cause for death. There are instances of death where the abscess was well contained within the liver without having produced gross necrosis. Perhaps the child is less able to withstand this pathological condition in his liver and may die more easily from the toxic effects of the disease.

Multiplicity of hepatic lesions undoubtedly worsens the prognosis. This holds for adults too. Children, however, appear to have a higher incidence of multiple lesions. This may be explained by the fact that as so many come to necropsy multiple lesions are revealed more often than in adults. This explanation does not appear entirely valid, as in some adult necropsy studies the proportion of multiple abscesses has been high. We do not know if the vast majority of adults who recover had, in fact, only one abscess unassociated with multiple small abscesses. Perhaps adults are better able to withstand multiple hepatic abscesses.

The very high mortality rate of this condition in our African children may in part be due to their poor general health and nutritional state. Other complications such as perforation of the bowel in some measure contribute to the high mortality. More important, however, is the fact that the African child is so often submitted very late for treatment of most diseases. This is illustrated by the large number who died within minutes or hours of admission. The late stage at which

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treatment is instituted obviously contributes to the high death rate in this disease.

It has been questioned whether some of these deaths could have been due to emetine. In all in whom emetine was used great care was taken to ensure that the recommended dose and duration of treatment was not exceeded. Routine electrocardiographic studies were not done so that the possibility of death in some instances from myocarditis cannot be excluded.

The usual total dose of emetine recommended for adults is 10 grains. The dose employed in these children varied from about one-eighth to one-quarter of the adult dose, while that of chloroquine was one-half the usual adult dose. It might be that this disproportion of dosage is a contributory factor to the higher mortality.

An answer to these problems may be found by treating one random group of cases on emetine alone and another on chloroquine alone and comparing the mortality rate of the two groups.

Apart from the above mentioned differences, in all other respects the condition does not materially differ from that in adults. The management and treatment is the same.

The criteria for surgical drainage have been stated. Very few cases were subjected to surgical drainage. In retrospect, however,

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it is felt that a few other selected cases should have been submitted for this form of treatment, especially where many aspirations were necessary and the expected improvement was not apparent.

Illustrative cases are recorded which draw attention to the difficulties that may arise in diagnosis. Failure of response to therapy shown by a continuing high fever and increasing anaemia in children with dysentery or a history thereof should suggest the possible presence of an hepatic complication. However, concomitant or antecedent dysentery can be expected in only about two-thirds of cases.

In an area where amoebiasis of a severe type is unfortunately so prevalent in the African race and where there exists an acute awareness of its clinical manifestations and complications, the diagnosis of amoebic liver abscess is nevertheless often difficult and not infrequently missed. Cases continue to be revealed at necropsy which one feels should have been diagnosed clinically. This condition should always be kept in mind in the differential diagnosis of hepatomegaly, especially tender hepatomegaly, even in the absence of dysentery or a history of dysentery.

Owing to the overwhelming number of ill African children it is not feasible to admit all cases of dysentery for investigation and treatment. In view of this state of affairs it has become necessary to attempt to prevent this (and other serious complications of amoebiasis) with the best means at our disposal in the form of out-patient therapy.

From the study of others in which the usefulness of chloroquine has been proved, it appears rational to prescribe this drug (in addition to other recommended amoebicides) in all cases of intestinal amoebiasis, especially where circumstances prevent hospitalisation and thus preclude the use of emetine. Not only is chloroquine advocated in order to prevent the hepatic complication, but recent work suggests that chloroquine is indicated in combination with other amoebicides, including tetracycline, for its apparent potentiating effect on the latter, thus greatly increasing the efficacy of treatment of the primary intestinal infection. If all cases of amoebiasis were treated with chloroquine it would seem reasonable to expect that the incidence of the secondary manifestations of amoebiasis would be reduced. No child at this hospital who has received chloroquine in combination with other amoebicides has ever been admitted later with an amoebic liver abscess. This combination of therapy has been employed for out-patients for a number of years. This fact, though gratifying, does not appear to be the whole answer to the prevention of this complication. Probably only a small proportion of cases of acute amoebic dysentery are presented for treatment early in the disease, so that one can expect this serious complication to continue to occur.

With improvement in sanitation and hygiene, and this constitutes an urgent necessity among the African population, it is to be expected that severe acute amoebiasis will become less prevalent. If along with

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this the African can be educated to present himself and his child for treatment early in disease, then it can be hoped that the incidence of this serious condition and its complications will be much reduced.

SUMMARY

One hundred cases of proved amoebic liver abscess in African children have been studied over a period of nine years.

The literature relating to amoebiasis in children, and especially to the complication of liver abscess is reviewed. Study of the literature indicates that the reported incidence of amoebic liver abscess in children in other parts of the world, is small by comparison with that in Durban, even in those places where the incidence in adults is comparable.

The diagnostic criteria are stated. The clinical presentation and course of the disease is described. Comparison of amoebic liver abscess in children with the disease in adults indicates that although the laboratory and clinical findings are similar, there are certain differences. It is noteworthy that in children the hepatic complication supervenes more acutely than it does in adults and often occurs at the height of the primary intestinal lesion. The incidence of multiple hepatic lesions is high in children. The high mortality in children is notable; this is probably in part due to late presentation for treatment.

Illustrative case summaries are recorded drawing attention to the difficulties that may arise in diagnosis. It is stressed that this condition should be kept in mind in the differential diagnosis of hepatomegaly, especially tender hepatomegaly in children, even in the absence of antecedent or concomitant dysentery.

The necessity for thorough treatment of amoebic dysentery in children is emphasized, and the use of a drug such as chloroquine to prevent hepatic involvement is advocated. The criteria for surgical drainage have been stated. It is suggested that selected cases should be submitted for this form of treatment.

The need for improvement in hygiene and sanitation among the African population is stressed.

APPENDIX 1

REPORTED CASES OF AMOEBIC LIVER ABSCESS IN CHILDREN

*	Unpublished (Quoted by Legrand, 1906)
**	Cysts of E. histolytica in aspirated pus (an improbable finding)
***	Unpublished (Quoted by Jiménez and Saenz, 1947)
X	Unpublished (Quoted by Cardelle and Saenz, 1948)
ø	Criteria for diagnosis not stated
D	Died
R	Recovered
F	Female
M	Male
NS	Not stated

APPENDIX 1
REPORTED CASES OF AMOEBIC LIVER ABSCESS IN CHILDREN

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Brown	1824	Cuba	Negro	1	1	F	D
Miller	1851	India	White	1	10	M	R
Chapple	1861	India	White	1	17/12	M	D
Dulles	1879	U.S.A.	White	1	12	M	R
Moore	1881	England	White	1	3 6/12	F	D
Menger	1881	U.S.A.	Mexican	1	9	M	R
Swift	1882	U.S.A.	White	1	3	F	D
Caravias*	1885	France	White	1	12	M	R
Easmon	1887	W. Africa	African	1	3 6/12	F	R
Huybertsz	1888	Ceylon	NS	1	6	M	R
Pereira	1890	India	White	1	20/12	F	R
Henoch	1890	Germany	?	2	?	?	Both D
Tshernoff	1891	Russia	White	1	5	M	R
Neal	1892	Br. Guiana	NS	1	5	M	D

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Le Blond	1892	France	?	3	?	?	?
Legrand	1894	Egypt	Italian	1	5	F	D
"	"	"	Arab	1	3	M	R
Slaughter	1895	U.S.A.	Negro	1	7	M	D
Finizio	1896	Italy	NS	1	4	M	D
Rouis	1896	Algeria	?	1	?	?	?
Johnston	1897	U.S.A.	White	1	13	F	D
Niklaoui*	1897	Egypt	Arab	1	12	F	R
Brossard*	1898	Egypt	Arab	1	3	F	D
"	"	"	Greek	1	5	M	R
Rigazzi*	1900	Egypt	Italian	1	18/12	M	D
Neuishi*	1900	Egypt	Arab	1	9	M	R
Gneftos	1900	Egypt	White	1	6	M	D
Arnott	1903	St.Helena	NS	1	2½	NS	D

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
De Oliveira	1903	Brazil	NS	3	5/12	NS	R
"	"	"	NS		17/12	F	R
"	"	"	NS		21/12	M	R
Boucher	1909	Viet-nam	Sino-Annamite	1	8/12	F	R
Niblock	1911	India	Indian	1	11/12	M	R
Alfaro	1912	Argentina	Russian	1	2	M	D
Acuna	1912	Argentina	White	1	2 5/12	M	R
Razetti	1913	Venezuela	White	2	21/12	F	R
"	"	"	White		13/12	F	D
Guzman	1919	Chile	?	1	4 8/12	F	R
Carvallo	1920	Peru	Indian	1	18/12	F	R
Carrau	1922	Argentina	White	1	20/12	M	D
Cade et al.	1922	France	White	1	9	NS	R
Navarro et al.	1925	Argentina	White	1	12	M	R

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Ludlow	1926	Korea	Korean	3	7	M	NS
"	"	"	Korean		10	M	NS
"	"	"	Korean		2	M	R
Panayotatou	1926(a)	Egypt	?	4	8/12	?	D
"	"	"	?		10/12	?	D
"	"	"	?		16/12	?	D
" **	1926(b)	"	NS		5	F	R
Pozzo et al.	1928	Argentina	White	1	12	M	R
Leroy des Barres	1930	Cochin China	Franco-Annamite	1	8/12	F	R
Gerbasi	1931	Sicily	White	1	3/12	NS	D
Biggam	1932	Egypt	NS	1	3/12	M	D
Biggam et al.	1933	Egypt	NS	1	8	NS	NS
Aldunate	1933	Chile	NS	1	3 6/12	M	R
Fonzo	1934	Italy	White	1	11/12	M	D

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Sweet	1934	China	Chinese	1	5	F	R
Mohammed	1937	Egypt	NS	1	2	F	D
Aballi et al.***	1941	Cuba	?	1	8/12	M	D
Smith	1943	Nigeria	NS	1	< 3	NS	D
Ochsner et al.	1943	U.S.A.	NS	2	< 9	NS	NS
Osburn	1944	S. Rhodesia	African	1	17/12	NS	D
Aguirre et al.	1944	Chile	NS	1	6	NS	D
Rogers	1946	India	NS	2	Both < 10	NS	NS
Buccelato	1947	Italy	White	1	22/12	M	R
Jiménez et al.	1947	Cuba	White	1	2/12	M	D
Aballi et al.[X]	1948	Cuba	?	1	14/12	F	D
D'Antoni	1948	Sp.Honduras	NS	1	1/12	NS	D
Stransky et al.	1948	Philippines	Filipino	1	7	M	D
Reddy et al.	1948	India	Indian	2	<10	NS	NS

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Soragni	1949	Italy	White	1	11	M	R
Shanmugaratnam	1949	Malaya	Chinese	1	6/12	F	D
Lambillon et al.	1949	Belgian Congo	African	2	5	M	R
"	"	"	African		3	M	D
Bol.Méd.Hosp. Inf.Méx.	1951	Mexico	White	1	3/12	M	D
Tupas et al.	1951	Philippines	Filipino	2	7	NS	D
"	"		Filipino		7	NS	D
Debbas Ø	1953	Egypt	NS	6	All between 6-7	NS	NS
De la Maza et al.	1953	Chile	White	6	6/12	M	D
"	"	"	White		15/12	F	D
"	"	"	White		16/12	M	D
"	"	"	White		2	M	R
"	"	"	White		2	M	D
"	"	"	White		8	F	R

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Chatgidakis	1953	South Africa	African	2	Both <18/12	NS	Both D
Salas Martinez et al.	1953	Mexico	White	3	1	F	D
"	"	"	White		18/12	F	D
"	"	"	White		5	F	D
Smith	1955	U.S.A.	White	1	17/12	M	D
Torroella et al.	1956	Mexico	White	14	5 1)	5M 9F	2 R
"	"	"	White		5 1-2)		12 D
"	"	"	White		4 3-5)		
Jiménez et al.	1956	Cuba	White	1	28/365	F	D
Moreno et al.	1956	Cuba	Negro	1	16/12	M	R
Lestrade et al.	1956	Viet-Nam	Vietnamese	3	4	M	D
"	"	"	Vietnamese		5	F	D
"	"	"	Vietnamese		6	F	R
Fernandez de la Arena y Santé	1956 "	Cuba	White	5	28/365	F	D

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Fernandez de la Arena y Santé	1956	Cuba	White		2/12	NS	D
"	"	"	White		8/12	M	D
"	"	"	White		16/12	F	D
"	"	"	White		2	NS	D
Senecal et al.**	1957	West Africa	African	4	6/12	F	D
"	"	"	African		2	F	D
"	"	"	African		3	M	D
"	"	"	African		2	M	R
Salas et al.	1958	Mexico	White	17	5<1	5M 12F	All D
"	"	"	White		6 1-2		
"	"	"	White		6 2-4		
Biagi	1958	Mexico	White	4	NS	NS	NS
Wagle et al.	1958	India	NS	1	6/12	NS	D
Vizcarondo et al.	1959	Venezuela	White	2	15/12	NS	D
"	"	"	White		2 6/12	NS	D

AUTHOR	YEAR	COUNTRY	RACE	No.	AGE (years)	SEX	OUTCOME
Burnside et al.	1959	U.S.A.	Negro	1	6/12	M	D
Walt	1959	South Africa	African	16	3 < 1	9M 7F	12 R
"	"	"	African		7 1-2		4 D
"	"	"	African		4 2-3		
"	"	"	African		2 3-4		
Ravina et al.	1960	Israel	Iraqi	1	1	F	D
Jayaratne et al.	1960	Ceylon	Sinhalese	1	2	F	D
Scragg	1960	South Africa	African	53	10 0-6/12	33M 20F	
"	"	"	African		7 6/12-1		23 R
"	"	"	African		21 1-2		30 D
"	"	"	African		10 2-3		
					5 > 3		
MacDougall	1960	Kenya	African	10	4 < 1	8M 2F	1 R
"	"	"	African		6 1-2		9 D

APPENDIX 2

SUMMARY OF FINDINGS IN 100 CASES AMOEBIC LIVER ABSCESS

D	Diarrhoea only
Rec	Recovered
RL	Right hepatic lobe
LL	Left hepatic lobe
C	Centrally placed in liver
(S)	Subsequent surgical drainage
RIL	Rupture into lung
RIP	Rupture into pleural space
(D)	Abdominal distension
*	Pleural aspiration
(C)	Pus coughed up
X	Non amoebic pathology

APPENDIX 2

SUMMARY OF FINDINGS IN 100 CASES AMOEBIC LIVER ABSCESS

Case No.	1	2	3	4	5	6
Age - years	2	2	11/12	2	8/12	2/12
Sex	M	M	M	M	F	F
Duration history of dysentery - wks	-	8	2/7 D	-	4	2
Dysentery on admission	-	+	-	+	-	-
Trophozoites E.histolytica in stool	-	-	-	-	-	-
Liver - cm. below costal margin	10	$6\frac{1}{2}$	9	4 → 8	9	8
Mass palpable in liver	RL	-	RL	-	RL	LL
Number of aspirations	2		1	3	17	
Total pus aspirated - mls.	200		40	299	1688	
E.histolytica in aspirated pus	-		+	+	-	
X-ray changes	-			+	+	+
Hb -g. per 100 ml.	6.0	7.4	9.0	7.1	8.3	8.0
W.b.c. - thousands per c.mm.	33.0	26.0		16.0	30.0	20.0
Duration survival - days	Rec	8	<1	36	36	1

Case No.	7	8	9	10	13	14
Age - years	3	2	5	18/12	5/12	9/12
Sex	M	F	F	M	M	M
Duration history of dysentery-wks	-	-	-	-	4	3
Dysentery on admission	+	-	+	-	+	+
Trophozoites E.histolytica in stool	+	-	+	-	-	-
Liver - cm. below costal margin	6	7	±5	±7	7	4½
Mass palpable in liver	RL	-	RL	LL	RL	-
Number of aspirations	4		1	1	1(S)	
Total pus aspirated - mls.	200		50	160	580	
E.histolytica in aspirated pus	-		-	-	-	
X-ray changes	+		-	+	-	
Hb -g. per 100ml.	8.0	9.1	13.2	7.7	7.7	6.0
W.b.c. - thousands per c.mm.	22.0	43.0	9.6	35.0	20.0	26.0
Duration survival -days	Rec	2	Rec	<1	Rec	10

Case No.	15	16	19	21	22	24
Age - years	4	2	20/12	2	2	3
Sex	M	M	F	M	F	M
Duration history of dysentery-wks	12	4/7	-	4	-	3
Dysentery on admission	+	+	-	-	+	+
Trophozoites E.histolytica in stool	+	+	-	-	+	+
Liver - cm. below costal margin	5	4 → 7	9	6	8	7
Mass palpable in liver	RL	-	RL	RL	RL	RL
Number of aspirations		1	1	3	3	3
Total pus aspirated - mls.	±300(C)	19 ±100(C)	200	150	92	90
E.histolytica in aspirated pus		-	+	+	-	-
X-ray changes	+(RIL)	+(RIL)		-	-	-
Hb -g. per 100ml.	7.4	8.3		7.1	6.7	7.2
W.b.c. - thousands per c.mm.	23.0	16.0		19.0	16.0	26.0
Duration survival - days	Rec	Rec	<1	Rec	Rec	Rec

Case No.	28	29	31	32	33	34
Age - years	5/12	3	20/12	3	14/12	6/12
Sex	M	M	M	M	F	F
Duration history of dysentery-wks	4/7	-	2	-	3/7	-
Dysentery on admission	+	-	+	+	+	-
Trophozoites E.histolytica in stool	-	-	-		-	-
Liver - cm. below costal margin	7½	4	5	6	8	6
Mass palpable in liver	RL	-	C	-	RL	C
Number of aspirations	1		4			
Total pus aspirated - mls.	4		230			
E.histolytica in aspirated pus	-		-			
X-ray changes	-	-	+			
Hb -g. per 100ml.	7.0	8.2	7.5		8.6	5.6
W.b.c. - thousands per c.mm.	23.0	16.0	27.0		26.0	21.0
Duration survival - days	13	11	Rec	<1	<1	4

Case No.	35	38	39	40	41	42
Age - years	3/12	4/12	4	3	11/12	2
Sex	M	M	F	F	M	M
Duration history of dysentery-wks	8	3/7 D	-	-	-	8
Dysentery on admission	+	-	+	+	+	+
Trophozoites E.histolytica in stool	+		-	+	+	-
Liver - cm. below costal margin	7	7½	9	8	8	±8
Mass palpable in liver	C	RL C	-	RL	-	RL LL
Number of aspirations	1		2	2	7	6
Total pus aspirated - mls.	205		12	55	535	194
E.histolytica in aspirated pus	+		-	-	-	-
X-ray changes			+	-	+	+
Hb -g. per 100ml.	5.8		9.5	10.2	4.8	5.6
W.b.c. - thousands per c.mm.	19.0		14.0	26.0	21.0	19.0
Duration survival - days	7	<1	Rec	Rec	44	Rec

Case No.	43	45	46	47	48	50
Age - years	4	3	21/12	14/12	3	5/12
Sex	M	M	M	F	M	M
Duration history of dysentery-wks	2	-	-	3/7 D	-	12
Dysentery on admission	-	-	-	-	+	+
Trophozoites E.histolytica in stool	-	-	-		-	-
Liver - cm. below costal margin	±6 (D)	7	$6\frac{1}{2}$	4	4 → 9	7
Mass palpable in liver	-	RL	-	-	LL	RL
Number of aspirations		5	4		2	2
Total pus aspirated - mls.		390	80		180	134
E.histolytica in aspirated pus		-	-		-	-
X-ray changes		+	+		-	-
Hb -g. per 100ml.	7.4	5.0	7.6	9.4	8.6	7.7
W.b.c. - thousands per c.mm.	9.0	23.0	27.0		19.0	23.0
Duration survival -days	8	20	Rec	< 1	18	8

Case No.	51	52	53	54	55	56
Age - years	3	2	5	3	3	3
Sex	F	M	F	M	F	M
Duration history of dysentery-wks	-	1	1	3	2	4
Dysentery on admission	-	+	+	-	+	-
Trophozoites E.histolytica in stool	-	+	+		+	-
Liver - cm. below costal margin	3 → 7	4	5 (D)	$4\frac{1}{2}$	6	5
Mass palpable in liver	-	-	-	-	-	-
Number of aspirations	13					1
Total pus aspirated - mls.	1135					160
E.histolytica in aspirated pus	+					+
X-ray changes	+	-		+ X	-	+
Hb -g. per 100ml.	10.6	9.0	6.0	9.2	8.0	6.2
W.b.c. - thousands per c.mm.	18.0	24.0	26.0	27.0	24.0	22.0
Duration survival - days	Rec	23	5	1	7	13

Case No.	57	58	60	61	62	63
Age - years	4/12	3	2	4	2	2
Sex	M	M	M	M	F	M
Duration history of dysentery-wks	-	2	2	1	3	-
Dysentery on admission	-	-	-	-	+	+
Trophozoites E.histolytica in stool	-		-	-	+	-
Liver - cm. below costal margin	$5\frac{1}{2}$	9	$8\frac{1}{2}$	6	8	7
Mass palpable in liver	C	RL	RL LL	RL	C	RL
Number of aspirations	1	1	12	2	7 (S)	1
Total pus aspirated - mls.	27	200	320	27	926	18
E.histolytica in aspirated pus	-	-	+	-	+	+
X-ray changes	-		-	-	-	+
Hb -g. per 100ml.			9.0	11.4	6.1	8.3
W.b.c. - thousands per c.mm.			17.0	26.0	34.0	20.0
Duration survival - days	<1	<1	16	Rec	Rec	Rec

Case No.	64	65	66	67	68	69
Age - years	5/12	13/12	4	17/12	6/12	2
Sex	M	M	F	F	F	M
Duration history of dysentery-wks	4	5/7 D	2	12	4	-
Dysentery on admission	+	-	+	+	-	+
Trophozoites E.histolytica in stool	+	-	-	+	-	-
Liver - cm. below costal margin	3 → 7½	3 → 8	4 → 9	8	8	5 → 9
Mass palpable in liver	RL	RL	-	C	RL LL	C
Number of aspirations	1	2	1	5	3	3
Total pus aspirated - mls.	30	12	14	675	100	95
E.histolytica in aspirated pus	-	+	-	-	-	-
X-ray changes	+ X	-	+	-		+
Hb -g. per 100 ml.	5.6	8.0	5.7	6.1	8.6	7.0
W.b.c. -thousands per c.mm.	33.0	35.0	30.0	24.0	26.0	16.0
Duration survival - days	< 1	8	Rec	Rec	2	Rec

Case No.	70	71	72	73	75	76
Age - years	7/12	1	1¼/12	10/12	9/12	3
Sex	M	F	F	M	F	F
Duration history of dysentery-wks	-	1	2	-	5/7 D	3
Dysentery on admission	+	+	+	-	-	+
Trophozoites E.histolytica in stool	-	+	+	-	-	+
Liver - cm. below costal margin	3½ → 9	±6 (D)	8	6	8 (D)	10
Mass palpable in liver	RL	-	-	RL	-	C
Number of aspirations	7		5	3		2 (S)
Total pus aspirated - mls.	252		270	74		7
E.histolytica in aspirated pus	-		-	+		-
X-ray changes	-	+ X	+	-		-
Hb -g. per 100 ml.	7.0	11.2	9.5	9.1	10.6	9.0
W.b.c. - thousands per c.mm.	27.0	8.0	12.0	28.0	6.0	15.0
Duration survival - days	Rec	5	7	Rec	13	Rec

Case No.	77	78	79	80	82	83
Age - years	2	2	7/12	8/12	4	5
Sex	M	F	M	F	M	F
Duration history of dysentery-wks	2/7	-	4	4	1	3
Dysentery on admission	-	+	+	-	+	+
Trophozoites E.histolytica in stool	-	-	-	-	+	+
Liver - cm. below costal margin	±7	7	7	6	±2(D)	±6(D)
Mass palpable in liver	C	-	RL RL	-	-	-
Number of aspirations	3	4	7			
Total pus aspirated - mls.	87	155	203			
E.histolytica in aspirated pus	+	-	+			
X-ray changes	+	+	+		+(RIL)	
Hb -g. per 100 ml.	11.6	7.6	6.0	7.2	7.4	7.6
W.b.c. - thousands per c.mm.	11.4	19.0	36.0	30.0	24.0	32.0
Duration survival - days	Rec	Rec	14	2	3	< 1

Case No.	84	85	86	87	88	89
Age - years	2	2	2	2	3/12	2
Sex	F	M	M	F	M	F
Duration history of dysentery-wks	4	4	1	12	8	3
Dysentery on admission	+	+	+	-	+	+
Trophozoites E.histolytica in stool	+	+	-	-	+	+
Liver - cm. below costal margin	7	±7	8	4→8	±9	7½
Mass palpable in liver	RL LL	-	RL	-	RL	-
Number of aspirations	2		1	3	1	1
Total pus aspirated - mls.	179		164	428	25	32
E.histolytica in aspirated pus	+		+	+	+	+
X-ray changes	X +			+		+
Hb -g. per 100 ml.	4.9	7.0	7.4	4.4	6.3	8.0
W.b.c. - thousands per c.mm.	36.5		27.0	23.0	23.0	32.0
Duration survival - days	10	<1	<1	Rec	<1	3

Case No.	90	91	92	93	94	95
Age - years	2	9/12	2	13/12	3	9/12
Sex	F	M	F	M	F	M
Duration history of dysentery-wks	1	12	-	5/7 D	-	3
Dysentery on admission	-	+	+	-	-	-
Trophozoites E.histolytica in stool	-	+	+	-	-	
Liver - cm. below costal margin	?(D)	8	±9	6	8	4
Mass palpable in liver	-	LL	RL	C	-	-
Number of aspirations		1	1	2	3 *	
Total pus aspirated - mls.		163	94	60	90	
E.histolytica in aspirated pus		+	+	+	-	
X-ray changes				-	+(RIP)	+(RIP)
Hb -g. per 100 ml.	8.4	3.6	7.2	10.1	7.0	7.1
W.b.c. - thousands per c.mm.	11.8		19.5	10.0	31.0	34.0
Duration survival - days	1	<1	<1	Rec	10	<1

Case No.	96	97	98	99	100	101
Age - years	10/12	16/12	2	4	2	4
Sex	F	M	M	M	F	F
Duration history of dysentery-wks	3	2	12	8	-	4/7
Dysentery on admission	+	+	-	+	+	+
Trophozoites E.histolytica in stool	+	-	-	+	+	+
Liver - cm. below costal margin	? (D)	4 → 8	±6 (D)	6	8	7
Mass palpable in liver	-	-	-	RL	-	C
Number of aspirations		1		4		2
Total pus aspirated - mls.		39		174		160
E.histolytica in aspirated pus		+		-		+
X-ray changes		+	-	+		+
Hb -g. per 100ml.	10.2	10.1	3.2	8.7	10.5	11.2
W.b.c. - thousands per c.mm.	13.0	15.6	24.0	20.0	50.0	31.0
Duration survival - days	3	Rec	2	Rec	<1	Rec

Case No.	103	104	105	106	107	108
Age - years	9/12	1	10/12	2	20/12	2
Sex	F	M	M	M	M	F
Duration history of dysentery-wks	1	2	4	12	8	2
Dysentery on admission	+	+	+	+	-	+
Trophozoites E.histolytica in stool			-	+	-	+
Liver - cm. below costal margin	0	±6 (D)	7	7½	±8	7
Mass palpable in liver	0	-	RL	C	-	RL
Number of aspirations			5	2	1	2
Total pus aspirated - mls.			410	87	150	96
E.histolytica in aspirated pus			+	+	+	-
X-ray changes			-	-	+	-
Hb - g. per 100ml.		8.0	9.2	6.4	5.9	8.2
W.b.c. - thousands per c.mm.		24.0	14.0	28.0	21.0	18.0
Duration survival - days	<1	<1	14	Rec	<1	5

Case No.	109	110	111	112	113	114
Age - years	1	4/12	1	19/12	2	9/12
Sex	M	M	F	F	F	M
Duration history of dysentery-wks	8	8	-	-	8	2/7 D
Dysentery on admission	-	+	-	+	-	-
Trophozoites E.histolytica in stool	-	+	-	-	-	-
Liver - cm. below costal margin	9	3 → 8	7	5	8	6
Mass palpable in liver	RL	C	RL	RL	-	0
Number of aspirations	4	3	3	2		
Total pus aspirated - mls.	404	52	73	95		
E.histolytica in aspirated pus	+	+	+	-		
X-ray changes	-	-	-	-	+ X	
Hb - g. per 100ml.	7.0	8.2	6.2	7.0	7.5	8.7
W.b.c. - thousands per c.mm.	19.0	28.0	9.0	21.0	20.0	23.0
Duration survival - days	5	Rec	Rec	Rec	5	9

Case No.	115	116	117	118		
Age - years	11/12	4	19/12	5		
Sex	M	F	F	F		
Duration history of dysentery-wks	2/7 D	2	2	2		
Dysentery on admission	-	+	-	+		
Trophozoites E.histolytica in stool	-	+	-	+		
Liver - cm. below costal margin	6	6	7	7		
Mass palpable in liver	RL	RL	RL	-		
Number of aspirations		3	8 (S)	1		
Total pus aspirated - mls.		197	423	55		
E.histolytica in aspirated pus		+	+	+		
X-ray changes		-	-	+		
Hb - g. per 100ml	5.7	5.7	8.4	9.0		
W.b.c. - thousands per c.mm.	22.0	16.0	14.0	23.0		
Duration survival - days	<1	Rec	Rec	Rec		

ACKNOWLEDGMENTS

I wish to record my thanks to Professor H.L. Wallace, Head of the Department of Paediatrics, University of Natal, Professor E.B. Adams, Head of the Department of Medicine, and Dr. N.M. Mann for their interest in this study.

I am especially indebted to Dr. A.J. Wilmot and Dr. S.J. Powell of the Department of Medicine, and Dr. R. Elsdon-Dew, Director of the Amoebiasis Research Unit, Durban, for their valuable criticism and advice.

Thanks are also due to Dr. Pauline Klenerman and all my other colleagues in the Paediatric Department for permission to examine cases under their care and for referring patients to me for study, and to Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, Durban, for facilities.

I am grateful, too, to Professor J. Wainwright, Dr. S. Kallichurum and the staff of the Department of Pathology for the necropsy and histology studies; Dr. W. Hift for his help in translating the German literature; Mrs. B. Robinow, Medical Librarian, University of Natal, for a great deal of help in tracing and obtaining the literature; Mrs. J.R. Rudder of the Department of Medicine for the typescript; Miss M. Goddard for preparing the histograms; and finally Mrs. P. Kruger for the photography.

REFERENCES

ABALLI, A.A., CARBONELL, and PRADO (1947). Absceso hepatico amebiano en un niño de 8 meses de edad. Personal communication quoted by Jiménez and Saenz, 1947.

ABALLI, A.A., and LABOURDETTE, J.M. (1948). Absceso hepatico amebiano y absceso cerebral en una niña de 14 meses. Staff-meeting Hosp.Mun.Inf. (Not published). Quoted by Cardelle and Saenz (1948).

ACUNA, M. (1912). A proposito de un caso de absceso hepatico en la primera infancia. Arch.Lat.-amer.Pediat., 5, 213.

ADAMS, A.R.D., and SEATON, D.R. (1949). Amoebiasis in England as a Household Disease. Brit.med.J., 2, 136.

ADAMS, A.R.D. (1952). Current Therapeutics - Amoebicides. Practitioner, 168, 419.

ADAMS, A.R.D. (1956). Treatment of Intestinal Amoebiasis. J.trop.Med.Hyg., 59, 45.

ADAMS, A.R.D., and MAEGRAITH, B.G. (1960). Clinical Tropical Diseases, 2nd Edit., Oxford, Blackwell.

AGUIRRE, S.P., and RIVERA, J.M. (1944). Los Abscesos Hepaticos en la infancia (con relación de dos casos). Rev.chil. Pediat., 15, 736.

ALDUNATE, R. (1933). Absceso hepatico de origen amebiano en un niño de 3½ anos. Rev.chil.Pediat., 4, 155.

ALFARO, A. (1912). Sobre un caso de absceso del higado en un niño de dos anos. Arch.lat.-amer.Pediat., 6, 88.

ALVING, A.S., EICHELBERGER, L., CRAIGE, B. Jr., JONES, R., MERRILL WHORTON, C., and PULLMAN, T.N. (1948). Studies on the chronic toxicity of chloroquine. J.clin.Invest., 27, 60.

AMBERG, S. (1901). A contribution to the study of Amoebic Dysentery in children. Bull.Johns Hopk.Hosp., 12, 355.

ANAND, B.R. (1954). A Survey of Carriers of Intestinal Parasites in Rajasthan with special reference to E.histolytica. J.Indian. med.Ass., 23, 398.

ANDERSON, H.H., BOSTICK, W.L., and JOHNSTONE, H.G. (1953). Amebiasis. Pathology, Diagnosis and Chemotherapy. Springfield, Illinois, Charles C. Thomas.

ANDERSON, H.H., NELSON, T.L., HRENOFF, A.K., and FISH, C.H.(1954). Antibiotic Synergism in Amebiasis. Amer.J.trop.Med.Hyg., 3, 254.

ARCHER, J.G. (1932). The increasing incidence of Amoebic Dysentery as a Warning for More Study of Diarrheas, New Orleans med.surg. J., 85, 418.

ARCHIBALD, R.G. (1914). Emetine Treatment of Dysentery in Young Children. J.trop.Med.Hyg., 17, 161.

ARMSTRONG, T.G., WILMOT, A.J., and ELSDON-DEW, R. (1949). The Treatment of Amoebic Dysentery in the Bantu African. Trans. roy.Soc.trop.Med.Hyg., 42, 597.

ARMSTRONG, T.G., WILMOT, A.J., and ELSDON-DEW, R. (1950). Aureomycin and Amoebic Dysentery. Lancet, 2, 10.

ARMSTRONG, T.G., WILMOT, A.J., and ELSDON-DEW, R. (1952). Terramycin in the Treatment of Amoebic Dysentery. S.Afr.med.J., 26, 766.

ARNOTT, W.J.J. (1903). Case of Liver Abscess in a child aged 2½ years. Brit.med.J., 1, 189.

BAEZA, G. (1933). Un probable absceso hepático en un niño de 9 anos. Rev.chil.Pediat., 4, 600.

BANKER, D.D. (1947). Amoebic Abscess of the Liver. Indian Physician, 6, 254; abstr., Trop.Dis.Bull., 45, 338, 1948.

BELTRAN, E. (1944). El problema de la amibiasis en la Républica Mexicana. Rev. de med.trop. y parasitol; bacteriol. clin. y lab., 10, 40.

BELTRAN, E., and LARENAS, R.M. (1944). Investigación de protozoarios en materias fecales de niños con "diarrea". Rev.Inst. Salubr.Enferm.trop.(Méx)., 5, 23.

BERBERIAN, D.A., and DENNIS, E.W. (1948). Field experiments with chloroquine diphosphate. Amer.J.trop.Med., 28, 755.

BERETERVIDE, E., ROSSI, R., and MIDLIN, S. (1940). Absceso de probable origen amebiano de higado en un niño 8 anos, curación. Arch.argent.Pediat., 13, 420.

BERLINER, R.W., EARLE, D.P. Jr., TAGGART, J.V., ZUBROD, C.G., WELCH, W.J., CONAN, N.J., BAUMAN, E., SCUDDER, S.T., and SHANNON, J.A. (1948). Studies on the chemotherapy of the human malarias. The physiological disposition, antimalarial activity and toxicity of several derivatives of 4-aminoquinoline. J.clin.Invest.Suppl., 27, 98.

BERNE, C.J. (1942). Diagnosis and Treatment of Amebic Liver Abscess. Surg.Gynec.Obstet., 75, 235.

BIAGI, F.F. (1958). Algunas observaciones clinicas sobre 46 casos de amibiasis en niños. Bol.méd.Hosp. infant.(Méx)., 15, 633.

BIAGI, F.F. (1959). Amibiasis Generalidades. Rev.Gastroent. Méx., 24, 1.

BIAGI, F.F., and NAVARRETE, F. (1958). Busqueda de amibas en abscesos hepaticos. Rev. lat.-amer. de Microbiologia, 1, 243.

BIGGAM, A.G. (1932). Amoebic Dysentery in Infancy. Correspondence, Lancet, 2, 1457.

BIGGAM, A.G., and GHALIOUNGUI, P. (1933). Amoebic Liver Affection: Symptoms and Treatment, with Review of 25 cases. J.trop.Med. Hyg., 36, 270.

BOBILLIER, C. (1913). La Dysenterie Amibienne du tout petit enfant, Thèse, Paris.

BOLETIN MEDICO DE HOSPITAL INFANTIL MÉXICO (1951). Un caso de Amibiasis con Absceso del Higado (Case Report), 8, 809.

BONABA, J. (1935). Abscesos hepáticos multiples y pleuresia diaphragmática putrida consecutiva a apendicitis abscesada evolucionando durante tres meses. Arch.urug.Med., 6, 590.

BOUCHER, H. (1909). Un cas d'abcès du foie observé chez une métis sino-annamite de huit mois. Le Caducée, Paris, 7, 93.

BREM, T.H. (1955). The use of Hepatic Function Tests in the Diagnosis of Amebic Abscess of the Liver. Amer.J.Sci., 229, 135.

BROSSARD (1897). Not published. Quoted by Legrand, 1906.

BROWN, J. (1824). Case of great enlargement, suppuration and ulceration of the liver of a child aged 12 months. Trans. med.-chir.Soc.Edinb. (1821-1823), 665.

BUCCELATO, G. (1947). Considerazioni su un caso di epatite amebica. Riv.pediat.sicil., 2, 273.

BUCHMAN, M. (1926). Behandlung von Amöben dysenterie bei Erwachsenen und Kindern mit "Yatren 105". Arch.f.Schiffs-u. Trop.-Hyg. 30, 148; abstr. Trop.Dis.Bull., 23, 648, 1926.

BUCHMAN, M. (1927). Zur Therapie der Amöbenruhr bei Kindern, Dtsch.med.Wschr., 53, 928; abstr. Trop.Dis.Bull., 25, 224.

BURNSIDE, W.W. Jr., and CUMMINS, S.D. (1959). Amebic Abscess of the Liver in a six-month-old infant. J.Pediat., 55, 516.

BUSTOS, A. (1938). Parasitosis intestinal. Rev. Gastroent. Méx., 3, 75.

CADE, A., and RIGAL (1922). Dysenterie amibienne infantile autochtone compliquée d'hepatite suppurée. Lyon méd., 131, 301.

CAHEN, (1891). Ueber Protozoen im Kindlichen Stuhle. Dtsch.med. Wschr, No. 27, 853. Quoted by Councilman and Lafleur, 1891.

CANNATA, S. (1916). Sulla dissenteria amebica nell' infanzia. Pediatria (Napoli), 24, 359.

CANNATA, S. (1923). Contributo alla conoscenza dell' amebiasi nell' età infantile. Pediatria (Napoli), 31, 1193.

CARAVIAS, (1885). Not published. Quoted by Legrand, 1906.

CARBONELL Y SALAZAR, A., and SANCHEZ SANTIAGO, B. (1936). Absceso Hepático De Una Niña De Catorce Meses. Arch.Med.infant., 5, 143.

CARDELLE, G., and SAENZ, J.C. (1948). La Amibiasis en la Infancia, Pediat. Amér., 6, 14.

CARRAU, A. (1922). Absceso hepático amebiana en un niño de 20 meses de edad. Arch.lat.-amer. Pediat., 16, 172.

CARVALLO, C.J. (1920). Gran absceso hepatico disentérico en una niña de dieciocho meses. Crón.méd.(Lima), 37, 208.

CASTILLO, C. (1953). Amibiasis en el niño. Rev.méd.Cordoba, 41, 166.

CASTILLO, C. (1955). Amibiasis en la infancia. Gac.méd.Caracas, 63, 374.

CASTORINA, S. (1951). Sulla frequenza dell' eziologia amebica nelle formazioni ascessuali del fegato in Sicilia. Acta. med.ital.Mal.infett., 6, 73.

CHAPPLE, R. (1861). Case of Abscess of the Liver in a child 17 months old. Trans.med.phys.Soc.Bombay, 6, 47.

CHATGIDAKIS, C.B. (1953). The pathology of hepatic amoebiasis as seen on the Witwatersrand. S.Afr.J.clin.Sci., 4, 230.

CHEN, S.M., VAN GORDER, G.W., and YUAN, Y.K. (1931). Amoebic Abscess of liver. Study of 48 cases with particular attention to treatment. Nat.med.J.China, 17, 393.

CHUECA, F. (1928). Disenteria amebiana en la infancia. Crón.méd. (Lima), 45, 25; abstr. Trop.Dis.Bull., 25, 615, 1928.

CLARK, H.C. (1925). Distribution and complications of amebic lesions found in 186 post-mortem examinations. Amer.J. trop.Med., 5, 157.

CONAN, N.J. (1948). Chloroquine in Amebiasis. Amer.J.trop.Med., 28, 107.

CONAN, N.J. (1949). The treatment of hepatic amebiasis with chloroquine. Amer.J.Med., 6, 309.

CONAN, N.J., HEAD, J.A., and BREWER, A.E. (1950). Pleural and hepatic amoebiasis treated with chloroquine. Trans.roy.Soc. trop.Med.Hyg., 43, 659.

COSTA MASCARO, A. (1945). Le disenteria amebiana en la infancia. Rev.chil.Pediat., 16, 662.

COUNCILMAN, W.T., and LAFLEUR, H.A. (1891). Amoebic Dysentery. Johns Hopk.Hosp.Rep., 2, 395.

CRAIG, C.F. (1934). Amebiasis and Amebic Dysentery. London, Bailliére, Tindall and Cox.

CRAIG, C.F. (1944). The Etiology, Diagnosis and Treatment of Amebiasis. Baltimore, Williams and Wilkins.

CRAIG, C.F., and FAUST, E.C. (1943). Clinical Parasitology, Ed., 2, Philadelphia, Lea and Febiger.

CRAWFORD, K.P. (1953). Diarrhea in infants and children. J. Kentucky med.Ass., 51, 425.

D'ANTONI, J.S. (1947). Amebiasis; in Mitchell-Nelson Textbook of Pediatrics, Ed., 4, Philadelphia, W.B. Saunders Company.

D'ANTONI, J.S. (1948). Chronic Diarrheas. Amer.J.Surg., 75,332.

D'ANTONI, J.S. (1949). The Pattern of the Literature of Amebiasis: 1932-1947. A Commentary on Trends. Presidential Address, American Society of Tropical Medicine, 1948. Amer.J.trop. Med., 29, 269.

DA SILVA, J.R. (1954). Contribução ao estudo do comprometimento hepático na amibiase. Bol.Centro Estudos Hosp. Servidores Estado (Rio de Janiero) May Suppl., p.2 and 144; abstr. Trop. Dis.Bull., 53, 187, 1956.

DE BAKEY, M.E., and OCHSNER, A. (1951). Hepatic Amebiasis. A 20 Year Experience and Analysis of 263 Cases. Int.Abstr.Surg., 92, 209.

DEBBAS, E. (1927). Contribution a l'étude de la dysenterie amibienne et de son traitement chez les enfants. Arch.Méd. Enf., 30, 257.

DEBBAS, E. (1953). La dysenterie amibienne chez les enfants et les nourrissons. Sem.Hop.Paris, 21, 1082.

DEBUYS, L.R. (1914a). Amebic Dysentery in Children. J.Amer. med. Ass., 63, 1806; abstr. Trop.Dis.Bull., 5, 202, 1915.

DEBUYS, L.R. (1914b). Amebic Dysentery in Children. Amer.J.Obstet., 70, 509; abstr. Trop.Dis.Bull., 6, 50, 1915.

DE LA MAZA, V., and GUZMAN, A. (1953). Absceso Hepatico Amebiano, Absceso Pulmonar Amebiano, Absceso Cerebral Amebiano, Peritonitis y Meningitis Amebianas en el Niño. Rev.chil. Pediat., 24, 421.

DELTHILL, M.P. (1946). Dysenterie amibienne mortelle contractee en France chez un enfant de 18 mois. Arch.franç.Pediat. 3, 173.

DENDALE, R. (1934). Contribution a l'étude l'amibiase infantile. Thése, Universite D'Alger.

DE OLIVEIRA, O. (1903). Dysenterische Leberabszesse bei Säuglingen. Mschr. Kinderheilk., 2, 290.

DE OLIVEIRA, O. (1905). La dysenterie amibienne chez l'enfant. Mémoires Originaux, Arch. Méd. Enf., 8, 193.

DE SILVA, P.C.C. (1949). Amoebiasis in Infancy. Brit.med.J., 2, 1208.

DEUTSCH, D. (1927). Zur Therapie der Amöbenruhr bei Kindern. Deut.Med.Woch., 53, 360; abstr. Trop.Dis.Bull. 25, 615, 1928.

DOBELL, C. (1919). The Amoebae Living in Man; A Zoölogical Monograph, London, John Bale, Sons and Danielsson.

DOBELL, C. (1921). A Report on the Occurrence of Intestinal Protozoa in the Inhabitants of Britain with special reference to Entamoeba histolytica. Spec.Rept.Ser.Med.Res.Counc. (London). 59, 1-71, 1921.

DUCKWORTH, D. (1887). Tropical Abscess of the Liver. Lancet, 1, 811.

DUCKWORTH, D. (1914). The Diagnosis and Treatment of Tropical Hepatic Abscess. Practitioner, 93, 1.

DUCKWORTH, D. (1920). The Diagnosis and Treatment of Tropical Hepatic Abscess. J.trop.Med.Hyg., 23, 149.

DULLES, C.W. (1879). Abscess of the liver; operation; recovery. Philad.med.Times, 9, 300.

EASMON, J.F. (1887). A case of abscess of the liver in a child three years and a half old. Lancet, 2, 310.

ELSDON-DEW, R. (1945). Amoebiasis in Durban. Nature, 156, 118.

ELSDON-DEW, R. (1946). Some Aspects of Amoebiasis in Africans. S.Afr.Med.J., 20, 580, 620.

ELSDON-DEW, R. (1949). Endemic Fulminating Amebic Dysentery. Amer.J.trop.Med., 29, 337.

ELSDON-DEW, R. (1955). Antibiotics in Acute Ulcerative Amebic Colitis. Antibiotics Annual 1954-55, Medical Encyclopedia, Inc., New York, N.Y. p. 869.

ELSDON-DEW, R., and FREEDMAN, L. (1952). Intestinal Parasites in the Natal Bantu. S.Afr.J.clin.Sci., 3, 59.

ELSDON-DEW, R., ARMSTRONG, T.G., and WILMOT, A.J. (1952). Antibiotics and Amoebic Dysentery. Lancet, 2, 104.

EMMETT, J. (1949). Refractory amebic abscess of the liver treated with chloroquine. J.Amer.med.Ass., 141, 22.

FAUST, E.C. (1930). Study of intestinal protozoa of representative sampling of population of Wise County, Southwestern Virginia. Amer.J.Hyg., 11, 371.

FAUST, E.C. (1931). The incidence and Significance of Infection with Endamoeba histolytica in New Orleans and the American Tropics. Amer.J.trop.Med., 11, 231.

FAUST, E.C. (1933). Parasitic infections in New Orleans, based on a cross section of charity and Welfare clinic patients with especial reference to children's clinics. J.Pediat., 2, 53.

FAUST, E.C., and JUNG, R.C. (1956). Protozoan and Metazoan Parasitoses of the Intestinal Tract. Pediat.Clin.N.Amer., 3, 169.

FERNANDEZ DE LA ARENA Y SANTÉ, M. del. C. (1956). El absceso hepatico amebiano en el lactante. Arch.Med.infant., 25, 1.

FINIZIO, G. (1896). Su di un ascesso epatico in un bambino. Pediatria (Napoli) 4, 340.

FLORES-BARROETA, F., NUÑEZ, V., and BIAGI, F.F. (1959). Observaciones sobre amibiasis en material de Autopsia. Pren.méd.méx., 24, 141.

FONZO, F. (1934). Contributo clinico allo studio degli ascessi epatici nell' età dell lattante. Lattante, 5, 72.

FONZO, F. (1935). Amebiasi in un neonato di tre giorni. Lattante, 6, 238.

FUTCHER, T.B. (1903). A study of the cases of amebic dysentery occurring at the Johns Hopkins Hospital. J.Amer.med.Ass., 42, 480.

GERBASI, M. (1927). Contributo alla conoscenza della dissenteria amebica nei bambini. Pediatria (Napoli), 35, 689.

GERBASI, M. (1931). Osservazioni e ricerche sulla amebiasi nei lattanti. Pediatria (Napoli), 40, 65.

GILLOT, V., and DENDALE, R. (1936). Étude clinique de l'Amibiase infantile. Rapport au Congres de l'amibiase. Maroc. méd., 16, 198.

GIVNER, D., and CHANG, D.J. (1953). Problems in diagnosis and complications of amebic infection of the liver. Amer.J. dig.Dis., 20, 32.

GNEFTOS, P. (1900). Ein dysenterischer Leberabscess bei einem sechsjahrigen Kinder. Dtsch.med.Wschr., 25, 515.

GOLDENBURG, C., SHLAES, W.H., and MINTZER, S. (1952). A Clinical report of amebiasis in Infants under one year of age. J.Pediat., 40, 290.

GONZALEZ, U. (1935). Hepatitis amebiana. Rev.chil.Pediat., 6, 214.

GUTIERREZ, U. (1904). Los abscesos del higado en Chile. Impr. Barcelona. Santiago. Quoted by Aguirre and Rivera, 1944.

GUZMAN, L. (1919). Absceso del higado en una niñita de 4 anos 8 meses. Rev.méd.Chile, 47, 741.

HALL, F.W.G. (1892). Traumatic hepatic abscess in a young subject. Indian med. Rec., 3, 7. Quoted by Amberg, 1901.

HARGREAVES, W.A. (1945). Chronic Amoebic Dysentery : A New Approach to Treatment. Lancet, 2, 68.

HARINASUTA, C. (1951). A comparison of chloroquine and emetine in the treatment of amoebic liver abscess. Indian med. Gaz., 86, 137.

HARRIS, H.F. (1898). Amoebic Dysentery. Amer.J.med.Sci., 115, 384.

HARRISON, T.R. ed. (1958). Principles of Internal Medicine, Ed., 3, New York, McGraw-Hill.

HENOCH, E. (1890). Quoted by Legrand 1906.

HERRERA, J.M. (1952). Meningo-Encefalitis por Ameba Histolitica. Arch.méd.panameñ., 1, 149.

HOARE, C.A. (1950). Amoebiasis in Great Britain with special reference to carriers. Brit.med.J., 2, 238.

HOARE, C.A. (1952). The food habits of Entamoeba histolytica in its commensal phase. Parasit. 42, 43.

HOWELL, K.M. and KNOLL, E.W. (1941). Amebiasis in Infants and in Children. Amer.J.Dis.Child., 61, 54.

HUARD, P., and MEYER-MAY, J. (1936a). L'abcès du foie chez l' enfant. Rev.méd.-chir.Mal.Foie, 2, 417.

HUARD, P., and MEYER-MAY, J. (1936b). Les abcès du foie. Paris, Masson et Cie.

HUYBERTSZ, H. (1888). Hepatic Abscess in a child: Incision: Recovery. Ceylon med.J. 1887-1888, 1, 29.

ISLAM, N., ALAM, K.S., and QUADERI, M.A. (1960). Hepatic Amoebiasis. J.trop.Med.Hyg., 63, 131.

IVANHOE, G.L. (1943). Studies on the transmission of amebiasis in a children's home in New Orleans. Amer.J.trop.Med., 23, 401.

IZAR, G. (1932). Amebiasi infantile. Rif.med. 48, 1435; abstr., Trop.Dis.Bull., 30, 22, 1933.

JAYARATNE, S., and DE SILVA, C.C. (1960). A Perforated amebic liver abscess with empyema in a child of 2 years. J.trop.Pediat., 5, 115.

JELLIFFE, D.B. (1951). Amoebic hepatitis treated with chloroquine. Report of a successful case in an African boy. J.trop.Med. Hyg., 54, 114.

JIMÉNEZ, M.R., and SAENZ, J.C. (1947). Absceso Hepático Amebiano en un Lactante de dos meses de edad. Médica (Matanzas), 6, 85.

JIMÉNEZ, M.R., BENS, S.S., BORGES, F., and DE VARONA, E. (1956). Abscesso hepático amebiana en un lactante. Arch.Med.infant., 25, 122.

JOHNSTON, G.B. (1897). Symptoms and Treatment of Hepatic Abscess. With report of Eighteen cases. Trans.Amer.surg.Ass., 15,225.

JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION (1902). Editorial, Amoebic Dysentery in Children, 38, 253.

JUDD, J.R. (1910). A Case of abscess of liver in an Eight-Year-Old Boy - Operation, Recovery. Arch.Pediat., 27, 451.

KARTULIS, S. (1886). Zur Aetiologie der Dysenterie in Aegypten. Virchows Arch.path.Anat., 105, 521. Quoted by Dobell, 1919.

KARTULIS, S. (1887). Zur Aetiologie der Leberabscesse; Lebende Dysenterie - Amöben im Eiter der dysenterischen Leberabscesse. Zbl.Bakt., 1. Abt. Orig., 2, 745; Quoted by Dobell, 1919.

KARTULIS, S. (1904). Gehirnabszesse nach dysenterischen Leberabszessen. Zbl. Bakt., 1 Abt., 37, 527; Quoted by Legrand 1912.

KEAN, B.H. (1955). Amebic Hepatitis. Absence of diffuse lesions at autopsy and in biopsies. Arch.intern.Med., 96, 667.

KEAN, B.H., GILMORE, H.R. Jr., and VAN STONE, W.W. (1956). Fatal Amebiasis: Report of 148 fatal cases from the Armed Forces Institutes of Pathology. Ann.intern.Med., 44, 831.

KERN, F. Jr. (1945). Amebic pericarditis. Arch.intern.Med., 76, 88.

KILGORE, A.R. (1916). Peripheral neuritis following emetine treatment of amebic dysentery. Boston med.surg.J.; abstr. Trop.Dis.Bull., 8, 442, 1916.

KILLOUGH, J.H., and MAGILL, G.B. (1951). Terramycin in Epidemic Typhus, Amebic Dysentery and Typhoid. J.Amer.med.Ass., 147, 1737.

KLENERMAN, P. (1951). Personal communication.

KOCH, R., and GAFFKY, G. (1887). Bericht über die Thätigkeit der zur Erforschung der Cholera im Jahre 1883, nach Egypten und Indien entsandten kommission. Arb.Gesundh Amte (Berl) 3, 1; Quoted by Dobell, 1919.

LAMBILLON, J., and BEHEYT, P. (1949). L'amibiase hépatique au Congo belge. Ann.Soc.belge Méd.trop., 29, 307.

LAMBL, W.D. (1860). Beobachtungen und Studien aus dem Gebiete der pathologischen Anatomie und Histologie Aus dem Franz Josef-Kinder-Spitale in Prag. (Loschner and Lambl) 1 Theil, Sect. 13(6) "Zur Pathologie des Darms" p. 365. Quoted by Dobell, 1919.

LAMONT, N.McE., and POOLER, N.R. (1958). Hepatic Amoebiasis. Quart.J.Med., 27, 389.

LANE, R. (1951). The treatment of hepatic amoebiasis with chloroquine. J.trop.Med.Hyg., 54, 198.

LE BLOND, V. (1892). Diagnostic et traitement des abcès du foie, Thèse, Paris, Nov. 1892.

LEGRAND, H. (1894). Deux cas d'abcès du foie (abcès des pays chauds, Egypte) chez l'enfant. Bull.Acad.Méd., (Paris) 3. S., 32, 155.

LEGRAND, H. (1906). Abcès du foie chez l'enfant (mémoire). Arch.Méd.Enf., 9, 129, 216.

LEGRAND, H. (1912). Les abcès dysentériques du cerveau (amibiase encéphalique). Presse méd.Egypte, 4, 309, 341.

LEROY DES BARRES (1930). Un cas d'abcès amibien du foie chez une enfant de huit mois. Rev.méd.-chir.Mal.Foie, 5, 42.

LESTRADE, P., and GUÉRINEAU, P. (1956). L'Hépatite Amibienne du jeune enfant. Arch.franç Pédiat., 13, 728.

LEUKART, K.G.F.R. (1880). Die Parasiten des Menschen. Quoted by Councilman and Lafleur, 1891.

LÖSCH, F. (1875). Massenhafte Entwickelung von Amöben im Dickdarm. Virchows Arch.path.Anat., 65, 196.

LUDLOW, A.I. (1926). Amebic Liver Abscess. China med.J., 40,1165.

MACDOUGALL, L.M. (1960). Amoebiasis and its complications in African infants. E.Afr.med.J., 37, 279.

MACCHIAVELLO, A. (1938). Investigaciones sobre la bacteriologia e immunologia del tifo exantematico, Santiago, Soc.Imp. y Lito. Universo, Ahumada, 32, 222.

MADDISON, S.E., and POWELL, S.J. (1959). Bacterial Infection of Amoebic Liver Abscess. Med.Proc., 5, 514.

MAGGIORE, S. (1923). Nota sulla dissenteria amebica nell' infanzia. Pediatria (Napoli), 31, 913.

MANSON-BAHR, P. (1932). Amoebic Dysentery in Infancy (correspondence), Lancet, 2, 1304.

MANSON-BAHR, P. (1944). The Dysenteric Disorders, Ed. 2, London, Cassell.

MANSON-BAHR, P. (1949). The treatment of Amoebic liver abscess with chloroquine. J.trop.Med.Hyg., 52, 91.

MANSON-BAHR, P. (1951). Mansons Tropical Diseases, Ed. 13, London, Cassell.

MARTIN, G.A., GARFINKEL, B.T., BROOKE, M.M., WEINSTEIN, P.P., and FRYE, W.W. (1953). Comparative efficacy of amebacides and antibiotics in acute amebic dysentery. J.Amer.med.Ass., 151, 1055.

MELENEY, H.E. (1930). Community surveys for endameba histolytica and other intestinal protozoa in Tennessee, I. J.Parasit., 16, 146.

MENGER, R. (1881). Case of hepatic abscess, successfully treated under Lister's antiseptic method. Texas med.surg.Rec. (Galveston) 1, 136.

MERLE, F. (1949). La conessine dans le traitement de la dysenterie amibienne de l'enfant. Rev.Palud.Méd.trop., 7, 220.

MEYER-MAY, J., and HUARD, P. (1936). Note sur l'étiologie des abcès du foie. Bull.Soc.Path.exot., 29, 61.

MILAM, D.F., and MELENEY, H.E. (1931). Investigations of Endameba histolytica and other intestinal protozoa in Tennessee, II. An epidemiological study of amebiasis in a rural community. Amer.J.Hyg., 14, 325.

MILLER, H. (1851). Case of hepatic abscess in a boy ten years of age, treated by external incision. Trans.Med.Phys.Soc. Bombay (1849-50) 10, 313.

MILLER, M.J. (1947). Studies on amoebiasis; clinical study of children passing cysts of entamoeba histolytica. Canad. med.Ass.J., 57, 373.

MILLER, M.J., and CHOQUETTE, L.P.E. (1947). Studies on Amoebiasis in Canada. Part I. The incidence of Intestinal Protozoal Infections in Two Institutionalized Groups in Canada. Canad.J.Res. (E)., 25, 1.

MOHAMMED, A.S. (1937). Amoebic abscess of liver rupturing into stomach of a child 2 years of age. Egypt.med.Ass., 20, 147.

MONTANES, R.M. (1943). Disenteria amebiana en el niño lactante. Med.colon., 2, 280.

MONTANES, R.M. (1949). Disenteria amebiana en el lactante y niño pequeño. Med.colon., 14, 223.

MOORE, N. (1881). Abscess of liver in a child. Trans.path.Soc. Lond., 32, 132.

MORENO, O., CORRAL, P.J., GUTIERREZ, V.O., and ARZOLA, C.R. (1956). Absceso Hepático Amebiana multiple en el lactante. Reporte de un caso curado. Rev.cubana Pediat., 28, 597.

MOST, H., and VAN ASSENDELFT, F. (1950). Laboratory and Clinical Observations on the effect of Terramycin in the Treatment of Amebiasis. Ann.N.Y.Acad.Sci., 53, 427.

MOST, H., and VAN ASSENDELFT, F. (1951). Laboratory and Clinical Observations on the effect of Terramycin in the Treatment of Amebiasis. Amer.J.trop.Med., 31, 284.

MUGGIA, A. (1924). Su di un caso di ascesso multiplo del fegato. Clin.pediat.Modena, 6, 502.

MURGATROYD, F., and KENT, R.P. (1948). Refractory amoebic liver abscess treated by chloroquine. Trans.roy.Soc.trop.Med.Hyg., 42, 15.

MUSSER, J.H. (1927). Amebiasis. Amer.J.med.Sci., 174, 1.

NAVARRO, J.C., and BERETERVIDE, E. (1925). Absceso hepatico en un niño de doce anos; (curación). Arch.lat.-amer. Pediat., 3, S., 19, 1127.

NEAL, F. (1892). Abscess of the left lobe of the liver in a boy of five. Brit.Guiana med.Ann., p. 173.

NEGHME, A. (1954). Estado actual de nuestros conocimientos sobre amebiasis en Chile. Semana méd. 60th Anniversary 1894-1954, 63.

NEGHME, A., and SILVA, R. (1954a). Data on the epidemiology and prophylaxis of Amebiasis in Chile. Riv. di Parassit., 15, 557.

NEGHME, A., SILVA, R., and SOTOMAYOR, R. (1954b). Enteroparasitosis entre escolares de la provincia de Santiago. Bol.chil. Parasit., 9, 70.

NIBLOCK, W.J. (1911). Notes on some interesting surgical cases. Infantile hepatic abscess; operation recovery. Indian med. Gaz., 46, 134.

NIKLAOUI (1897). Unpublished. Quoted be Legrand, 1906.

NOUISHI (1900). Unpublished. Quoted by Legrand, 1906.

OCHSNER, A., and DE BAKEY, M. (1943). Amebic hepatitis and hepatic abscess, analysis of 181 cases with review of literature. Surgery, 13, 460, 612.

ODDO, C. (1904). In Traité des maladie de L'Enfance. Grancher, J., Comby, J., and Marchant, A.B., Paris, Masson, Abcès du Foie, p. 616.

ORBISON, J.A., REEVES, N., LEEDHAM, C.L., and BLUMBERG, J.M. (1951). Amoebic brain abscess: Review of the literature and report of five additional cases, Medicine (Baltimore), 30, 247.

ORTIZ MACHADO, M. (1941). Absceso hepâtico en un niño de 12 meses de edad. Bol.Soc.cubana Pediat., 12, 137.

OSBURN, H.S. (1944). Amoebiasis in infancy. Clin.Proc., 3, 405.

OSLER, W. (1890). On amoeba coli dysentery and dysenteric liver abscess. Johns Hopk.Hosp.Bull., 1, 53.

OWEN, W.B., HONESS, R.F., and SIMON, J.R. (1934). Protozoal infestation of American Indian children. J.Amer.med.Ass., 102, 913.

PALAZUELOS, P.G., and LERNER, J.M. (1941). Diagnostico y tratamiento del abscesso hepatico amebiano. Rev.méd. Chile, 69, 67.

PANAYOTATOU, A. (1926a). L'Amibiase Intestinale et ses localisation extra-intestinales. Thésis. Quoted by Jiménez and Saenz, 1947.

PANAYOTATOU, A. (1926b). Abces amibien du foie chez une filette de cinç ans guéri par la traitment medical. Bull.Soc.méd. Hôp.Paris, 3, ser., 50, 1548.

PARDO, J.M. (1926). Amebic Dysentery in Children. Medicina ibera (Madrid) 20, 245, summarised in J.Amer.med.Ass., 87, 368.

PATTERSON, M., and LAWLIS, V. (1956). The diagnosis and management of amebic liver abscess. Amer.Practit.(Philad), 7,1995

PEREIRA, M. (1890). A case of hepatic abscess in an European female child. Indian med.Gaz., 25, 170.

PERRIN, M., REMY, A., and ZUBER, R. (1923). Un cas de dysenterie amibienne autochtone chez un enfant de 10 ans. Arch.Méd.Enf., 26, 168.

PERRY, H.M., and BENSTED, H.J. (1929). Investigations in Egypt of some Acute Bacillary Intestinal Infections. Trans.roy. Soc.trop.Med.Hyg., 22, 511.

PERRY, H.M., and BENSTED, H.J. (1932). Amoebic Dysentery in Infancy (correspondence). Lancet, 2, 1358, 1457.

PESSOA, S.B. (1957). Parasitoses Intestinais nos dois primeiros anos de Vida. Brasil-méd., 71, 13.

PETZETAKIS, M. (1924). Traitement systematique de l'amibiase par les injections intraveineuses d'émétine. Presse méd., 32, 705; abstr. Trop.Dis.Bull., 22, 7, 1925.

PETZETAKIS, M. (1925). Contribution a l'étude clinique de therapeutique de la dysenterie amibienne chez le nourrisson et dans l'enfance. Arch.Méd.Enf., 28, 601.

PFEIFFER, (1887). Weitere Untersuchungen über Parasiten In Blut und in der Lymph bei den Packenprocessen, Corr. Blall-Algem. Artztlich. Verem in Thuringen, 1888. Quoted by Councilman and Lafleur, 1891.

PLATOU, R.V., and BEAVER, P.C. (1958). Intestinal Protozoa, Amoebiasis, in Diseases of Children in the Subtropics and Tropics, Trowell, H.C., and Jeliffe, D.B., London, Edward Arnold.

POPPER, H., and SCHAFFNER, F. (1957). Liver Structure and Function, New York, The Blakiston Division, McGraw-Hill Book Company.

POWELL, S.J. (1958). A study of the haematological findings, serum proteins and liver function tests in the Natal African in health and in amoebiasis, M.D. Thesis, University of Cape Town.

POWELL, S.J. (1959). The Haematological Findings, Serum Protein Pattern and Liver Function Tests in Acute Amoebic Dysentery and Liver Abscess. Amer.J.trop.Med.Hyg., 8, 331.

POWELL, S.J., WILMOT, A.J., and ELSDON-DEW, R. (1959). Hepatic Amoebiasis. Trans.roy.Soc.trop.Med.Hyg., 53, 190.

POWELL, S.J., WILMOT, A.J., and ELSDON-DEW, R. (1960). Potentiating Effect of Quinolines on the Action of Tetracycline in Amoebic Dysentery. Lancet, 1, 76.

POWELL, S.J., and NEAME, P.B. (1960). A Case of Amoebic Brain Abscess. Brit.med.J., 2, 1136.

POZZO, F., and OLIVAN, N. (1928). Absceso hepático amebiano en un niño de 12 anos. Sem.méd., 35, pt. 2, 1483.

RAGHAVAN, P. (1945). Amoebiasis in Children. A brief note on its incidence, with a report of a case of amoebic dysentery in a child four and a half months old. Indian Physician, 4, 169.

RAIL, G.A. (1947). A comparative study of the therapeutic effects of some of the drugs used in the treatment of amoebic dysentery. J.trop.Med.Hyg., 50, 3.

RAVINA, A., JOSEPH, A., and COHEN, W. (1960). Fatal Amoebiasis in Infancy. Harefuah, 58, 157.

RAZETTI, L. (1913). Sobre los abscesos del higado en la infancia. Arch.lat.-amer.Pediat., 7, 12.

REDDY, D.G., and RANGAM, C.M. (1945). Jaundice in Amoebic Liver Abscess. A report of 2 cases. Indian med.Gaz., 80, 501.

REDDY, D.G., and THANGAVELU, M. (1948). Some aspects of amoebiasis in Madras. Indian med.Gaz., 83,557.

REPETTO, G. (1952). Amebiasis en un recien nacido. Rev.chil. Pediat., 23, 357.

RIGAZZI, (1900). Not published. Quoted by Legrand, 1906.

RIOPEDRE, R. (1953). Contribucion al estudio de la amebiasis infantil. Pren.méd.argent., 40, 2164.

ROACH, G.G. (1958). The Pathology of Amoebiasis, Proc. 6th Int. Congresses on Tropical Medicine and Malaria, Lisbon Sept. 5-15, In press.

ROGERS, L. (1922). Amoebic liver abscess; its pathology, prevention and cure, Lettsomian Lectures I, II, III, Lancet, 1, 463, 569, 677.

ROGERS, L. (1933). Tropical liver, hepatitis and abscess. Practitioner, 131, 117.

ROGERS, L., and MEGAW, J.W.D. (1946). Tropical Medicine, Ed., 5, London, J. and A. Churchill.

ROJAS, R.D. (1950). Amibiasis infantil y amibiasis del lactante. Rev.colomb.Pediat., 10, 5.

ROJAS, R.D. (1953). Amibiasis infantil y amibiasis del lactante. Pediat.Amer., 10, 529.

ROMAN, E. (1955). Symptomatologie des protozooses intestinales chez l'enfant; étude d'une soixantaine de cas observé a la clinique médicale infantile. J.Méd.Lyon, 36, 809.

ROUIS, J.L. (1896). Recherches sur les suppurations endemiques du foie d'apres des observations recueillies, Paris, J.B. Bailliere et fils. Quoted by Ludlow, 1926.

SABRI, I.A., ABBOUD, M.A., and ALI, M. (1937). Dysentery in infancy. Arch.Dis.Child., 12, 225.

SADUN, E.H., VIRANUVATTI, V., and HARINASUTA, T. (1956). Treatment of Amebic Liver Abscess with Oral and Intravenous Administration of Aureomycin and Terramycin. Gastroent., 30, 257.

SALAS MARTINEZ, M., RODRIGUEZ, E.C., RASCON, J.C., GONZALEZ, J.V., POUMAIN, G.L. and BARRERA, A. (1953):

(1) Amibiasis con Perforación Intestinal y Peritonitis.

(2) Amibiasis con Abscesso Hépatico, Diafragmitis y Pleuroneumonitis de Mismo Origen Parasitario.

(3) Amibiasis por Peritonitis con Perforación.

Bol.méd.Hosp.infant.(Méx), 10, 177, 185, 194.

SALAS, M.M., ANGULO, O.H., and ESPARZA, H.S. (1958). Patologia de la Amibiasis en los Niños. Bol.méd.Hosp.Inf.Méx., 15, 185.

SARROUY, C., and GILLOT, F. (1950). L'Amibiase Infantile. Gaz.méd.France, 57, 753.

SCHAUDINN, F. (1903). Untersuchungen über die Fortpflanzung einiger Phizopoden, Arb. Gesundh Amte. (Berl.) 19, 547. Quoted by Dobell, 1919.

SCRAGG, J.N. (1960). Amoebic liver abscess in African children. Arch.Dis.Child. 35, 171.

SENECAL, J., LARIVIÈRE, M., DUPIN, H., and TRENOU, R. (1957). Quelques aspects des abcès amibiens du foie chez le nourrisson Africain. Bull. méd. Afr.occid. franç., 2, 349.

SHANMUGARATNAM, K. (1949). Amoebic Liver Abscess in an Infant. Proc.Alumni. Ass. King Edw. VII Coll. Med., 2, 322.

SHARMA, L.R., SUNDER RAO, A.R., and ANDERSON, R.K. (1951). Chloroquine treatment of Amoebic Liver Abscess. Indian med.Gaz., 86, 6.

SHERLOCK, S. (1955). Diseases of the Liver and Biliary System, Oxford, Blackwell.

SIGALAS, R., PAUTRIZEL, R., and NOGUÉS, C. (1950). Étude du parasitisme intestinal chez les enfants de la région bordelaise. J.Méd.de Bordeaux, 126, 626; abstr., Trop.Dis.Bull., 47, 357, 1950.

SLAUGHTER, R.M. (1895). Abscess of the liver in children, with report of a case of amoebic abscess. Va.med.Mon., 22, 722.

SMITH, A., KIDD, F.H., and HARSHBARGER, M. (1955). Amebic abscess of the liver in an infant. J.Pediat., 47, 234.

SMITH, E.C. (1943). Child mortality in Lagos, Nigeria. Trans. roy.Soc.trop.Med.Hyg., 36, 287.

SNELL, J.A. (1915). Death of a child probably due to emetine. China med.J., 19, 164; abstr. Trop.Dis.Bull., 6, 447, 1915.

SODEMAN, W.A. (1950). Amebiasis panel; clinical picture of hepatic amebiasis. Amer.J.trop.Med.Hyg., 30, 141.

SODEMAN, W.A., and LEWIS, B.O. (1945). Amebic Hepatitis. Amer.J.trop.Med., 25, 35.

SORAGNI, E. (1949). L'epatite amebica nell infanzia. Clin. Pediat., Modena, 31, 414.

SPELLBERG, M.A. (1954). In Diseases of the Liver, Amebic Abscess of the Liver (Amebic Hepatitis), New York, Grune and Stratton.

SPOLVERINI, L. (1922). Sulla dissenteria amebica infantile. La Pediatria, 30, 1.

STEIN, A., and KAZAN, A. (1942). Brain abscess due to entamoeba histolytica. J.Neuropath., 1, 32.

STERN, E. (1928). Anaphylaktischer Chok und Tod nach 0.02g intraglutäaler Emetinein spritzung bei einem kinde. Arch. Schiffs-u. Tropenhyg., 32, 610.

STRANSKY, E., and DAUIS LAWAS, D.F. (1948). On some tropical aspects of liver pathology in pediatrics. Ann.Pediat. (Basel), 170, 131.

STREICHER, M.H. (1935). Amebic liver abscess in a boy aged 7 years. Illinois med.J., 67, 468.

STRONG, L.W. (1898). Two Cases of Amoebic Enteritis. M.S. Rep. Boston City Hosp., p. 249. Quoted by Howell and Knoll, 1941.

STRONG, R.P. (1945). Amoebiasis; in Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases, Ed. 7, Philadelphia, The Blakiston Company.

SUAREZ, H., ORTEGA, R., and BRIEVA, L. (1952). Tratamiento de la disenteria amebiana en el lactante. Rev.chil.Pediat., 23, 60.

SWEET, L.K. (1934). Amebic abscess of the liver. Report of a case in a child five years old. J.Pediat., 5, 750.

SWIFT, (1882). Quoted by Legrand, 1906.

TEITEL, D. (1929). Zur Frage de Behandlung der Amöbendysenterie bei Säuglingen Weiner. Klin. Wschr., 42, 235.

THURSTON, E.O. (1924). Liver abscess. A series of sixty-four cases. Lancet, 2, 1008.

TORROELLA, J.M., LOPEZ, T.G., and VILLARREAL, R. (1956). Consideraciones sobre el absceso hepatico en los niños. Bol.méd.Hosp.infant. (Méx.), 13, 1023.

TRABAUD, J. (1928). La dysenterie amibienne chez l'enfant au sein. La Médicine, 2, 1174.

TRABAUD, J. (1935). Les aspects médicaux de l'amibiase en Syrie. J. Egypt. med. Ass., 18, 579; abstr. Trop. Dis. Bull., 33, 541, 1936.

TREUER, C. (1952). La rectoscopia en la amebiasis del nino. Rev.chil.Pediat., 23, 58.

TRIBEDI, B.P., and ROY, A.R. (1951). An uncommon complication of amoebic liver abscess. Ind.J.med.Sci., 5, 242.

TSHERNOFF (1891). Quoted by Legrand, 1906.

TUPAS, A.V., and DAUIS-LAWAS, D.F. (1951). La complication hepatique de l'amibiase chez l'enfant. J. Philipp.med. Ass., 27, 397.

VIDAL FREYRE, A. (1953). Amibiasis infantil; sintomatologia y tratamiento. Pren.Pediát. (B.A.), 4, 15.

VIDAL FREYRE, A. (1954). Amibiasis infantil; sintomatologia y tratamiento. Dia. méd., 26, 219.

VIZCARONDO, E., DOMINGUEZ, A., and SÁNCHEZ ROMERO, G. (1959). La amibiasis hepatica en el niño. Arch. Venez. Puericult., 22, 131.

WAGLE, M.M., JADHAV, M.A., TEJANI, A., and APTE, N.V. (1958). Amoebic Liver Abscess in an infant six months old. J.Child Health, 7, 107.

WALLACE, H.L. (1960). Personal communication.

WALT, F. (1959). Amoebic liver abscess in infancy. J.trop.Ped., 5, 77.

WEISER, J. (1953). Amebic patient 13 days old. J.Amer.med.Ass., (correspondence), 52, 1554.

WILLETS, D.G. (1914). Intestinal Parasitism, particularly Entamoebiasis, in Patients of the Philippine General Hospital, Manila, Phillip. J.Sci.Sect.B.Trop.Med. 9, 81; abstr. Trop.Dis.Bull., 4, 297, 1914.

WILMOT, A.J. (1949). Clinical Manifestations of Amoebiasis in the Bantu, D.M. Thesis, Oxford University.

WILMOT, A.J. (1955). A comparison of Tetracycline and Its Derivatives in Amebiasis. Antibiotics Annual 1955-56, Medical Encyclopedia, Inc., New York, N.Y., p. 319.

WILMOT, A.J. (1960). Personal communication.

WILMOT, A.J., ARMSTRONG, T.G., and ELSDON-DEW, R. (1952). Aureomycin in Amoebic Liver Abscess. Amer.J.trop.Med. Hyg., 1, 429.

WILMOT, A.J., POWELL, S.J., and ADAMS, E.B. (1958). The comparative value of emetine and chloroquine in amebic liver abscess. Amer.J.trop.Med.Hyg., 7, 197.

WILMOT, A.J., POWELL, S.J., and ELSDON-DEW, R. (1958). Erythromycin in amebic liver abscess. Amer.J.trop.Med.Hyg., 7, 656.

WOODRUFF, A.W. (1959). Current therapeutics - amoebiasis. Practitioner, 183, 92.

YOUNG, M.L.(1921). A case of amoebic dysentery in a boy aged three. Lancet, 1, 122.

YOUNG, M.M. (1955). Report of a survey for intestinal parasites in school children in a rural mountain county in Tennessee. Sth.med.J. (Bgham, Ala.), 48, 46.

ZATURJAN, A.T. (1935). Protozoen-Infektionen des Darmtraktus in Kindersalter in Armenien Arb. d. Tropeninstituts d. Volksgandht. Kommisariat. d. S.S.R. Armenien. 2, 347. (German Summary p. 375) abstr., Trop. Dis. Bull., 33, 530, 1936.

AMOEBIC LIVER ABSCESS IN NATAL AFRICAN CHILDREN

Summary of thesis presented by Jean M. Serrag

One hundred cases of proved amoebic liver abscess in African children have been studied over a period of nine years.

The literature relating to amoebiasis in children, and especially to the complication of liver abscess is reviewed. Study of the literature indicates that the reported incidence of amoebic liver abscess in children, in other parts of the world, is small by comparison with that in Durban, even in those places where the incidence in adults is comparable.

The diagnostic criteria are stated. The clinical presentation and course of the disease is described. Comparison of amoebic liver abscess in children with the disease in adults indicates that although the laboratory and clinical findings are similar, there are certain differences. It is noteworthy that in children the hepatic complication supervenes more acutely than it does in adults and often occurs at the height of the primary intestinal lesion. The incidence of multiple hepatic lesions is high in children. The high mortality in children is notable; this is probably in part due to late presentation for treatment.

Illustrative case summaries are recorded drawing attention to the difficulties that may arise in diagnosis. It is stressed that this condition should be kept in mind in the differential diagnosis of hepatomegaly, especially tender hepatomegaly in children, even in the absence of antecedent or concomitant dysentery.

The necessity for thorough treatment of amoebic dysentery in children is emphasised, and the use of a drug such as chloroquine to prevent hepatic involvement is advocated. The criteria for surgical drainage have been stated. It is suggested that selected cases should be submitted for this form of treatment.

The need for improvement in hygiene and sanitation among the African population is stressed.