A STUDY OF THE HAEMATOLOGICAL FINDINGS, SERUM PROTEINS AND LIVER FUNCTION TESTS IN THE NATAL AFRICAN IN HEALTH AND IN AMOEBIASIS

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INTRODUCTION

A notable feature of this century has been the introduction of medical science to large, undeveloped areas throughout the world. Backward, mainly tropical, regions have become fertile fields for study, particularly of nutritional and parasitic disorders, and the application of modern biochemical methods has shown that many inhabitants of these countries lack certain of the biochemical "normal" values seen in Europeans. One of the most striking differences is that of the serum proteins. Although by no means confined to this continent, these differences are commonly found in Africans.

Precise comparison of the results obtained by various authors throughout Africa is not possible as the techniques used for estimating the serum proteins vary greatly. This is true, not only of chemical fractionation, but also of the more recent electrophoretic methods which have the advantage of differentiating the individual globulin fractions. However, all the

techniques show a similar basic pattern, and considerable evidence has now been accumulated that the indigenous African has a lower serum albumin with a higher total globulin, particularly gamma globulin, than healthy Europeans (Mohan,1946; Van Oye and Charles,1951; Holmes et al.,1951,1955; Busson et al.,1953; Arens and Brock, 1954; Edozien,1957; Carr et al.,1958). These differences are reflected in certain liver function tests.

African standards of "normals", particularly with regard to the serum proteins and liver function tests, are a long felt need among clinicians who practise in African hospitals. With the advent of the Non-European Medical School this need has become imperative in Durban. Without such standards investigation of the serum proteins and liver function in patients has limited diagnostic value, and may be misleading.

This thesis is divided into 2 parts.

Part 1 consists of a survey of Africans without apparent disease in order to establish whether the

changes in serum proteins and liver function described elsewhere in Africa are present in Natal. Two African groups are compared:

(a) Natal labourers who exist in slum conditions on a predominantly maize diet, deficient in animal protein.

(b) A higher socio-economic group known to be living in an environment, and receiving a diet more akin to that of Europeans.

The aetiology of the findings is then discussed.

Part II consists of an investigation of African male patients with amoebiasis. This part contains 3 sections:

(a) In the first section the distinction is made between amoebic hepatitis and amoebic liver abscess. The pathological and clinical evidence of amoebic hepatitis is discussed, and compared with the findings in Durban.

(b) The second section consists of a survey of the haematological findings, serum proteins and liver function tests in patients with amoebic dysentery and amoebic liver abscess. The aetiology of the findings are discussed, and the results compared with those reported elsewhere.

(c) In the last section the value of laboratory findings in the differentiation of amoebic liver abscess from cirrhosis of the liver, primary carcinoma of the liver and right basal pneumonia is assessed.

Methods

The detailed techniques of the methods used are shown in Appendix I. In all cases the following investigations were performed:
Haemoglobin (Hb), erythrocyte sedimentation rate (E.S.R.), packed cell volume (P.C.V.), serum bilirubin, alkaline phosphatase, cephalin cholesterol flocculation, and zinc sulphate turbidity. The total serum protein, albumin and globulin were estimated by chemical fractionation. In addition the serum albumin, and the globulin fractions were determined by paper electrophoresis.

The detailed results are shown in Appendix II.

Statistical analysis was that of Students' t-distribution for testing the significance of difference between means. Details are shown in Appendix III.

In all instances the subjects were examined and investigated by the author.

PART 1.

THE HAEMATOLOGICAL FINDINGS, SERUM PROTEIN PATTERN AND LIVER FUNCTION TESTS IN THE APPARENTLY HEALTHY NATAL AFRICAN

Material

Two groups, each of 50 African males of similar ages, were studied. In addition 25 European medical staff of comparable age were used as controls.

Group 1 consisted of the 25 European medical staff.

Group 2 comprised 50 African male members of the hospital nursing staff, their ages ranging from 19 to 41 years. All were healthy as judged by full medical examination, and radiological examination of their chests. All had been receiving an adequate, largely European type of diet for at least 3 years, some for considerably longer. Their main daily meal contained meat and potatoes. They received adequate milk, with fish, butter and eggs 2 to 3 times a week, and cheese occasionally. In addition, they consumed large amounts of maize in the form of mealie meal, samp and mealie rice.

Group 3 was composed of 50 African males, employed as labourers and manual workers, and who attended the casualty department with minor cuts and abrasions. Their ages ranged from 18 to 42 years. All lived in Cato Manor, an African slum suburb which provides the bulk of hospital admissions. Full medical examination of this group was not possible, but they were all apparently healthy with no obvious signs of disease. Their wages varied between £10 and £15 per month and they existed chiefly on a maize, bread, jam, potato, madombe and bean diet, receiving meat rarely more than once a week, and very little milk, with virtually no other forms of dairy produce. The amount spent on food varied between 10/- and £1 per week.

Results

Haematological Findings

The results of the haematological investigations are presented in Table I. In all groups the haemoglobin is within the normal range, although in both African groups it is significantly lower than in the European group($P < 1$ & 0.1%). This is reflected in a lower P.C.V. for the 2 groups, although here the difference is not statistically significant.

However, the lower M.C.H.C. of the 2 African groups is significant ($P < 0.1\%$). Unexplained elevation of the E.S.R. is common in Africans although it is less frequent and less marked in the Male Nursing Staff than in the Cato Manor subjects. The differences between the 3 groups are statistically significant ($P < 0.1\%$).

Table 1. Hb(G./100ml.), E.S.R.(mm./1hr.), P.C.V.(%) and Mean Corpuscular Haemoglobin Concentration(%) (M.C.H.C.)

		Group 1 (Europeans)	Group 2 (Afr.Male Nurses)	Group 3 (Cato Manor Africans)
Hb (G./100ml.)	Mean	17.0	16.2	16.0
	Range	14.2–19.6	14.2–18.2	13.6–19.0
	S.D.	1.31	0.97	1.06
E.S.R. mm./1hr.	Mean	4	11	18
	Range	1–12	1–25	2–40
	S.D.	2.86	6.64	9.58
P.C.V. %	Mean	48.5	47.4	47.3
	Range	42–57	42–55	40.5–55
	S.D.	3.10	2.85	2.95
M.C.H.C. %	Mean	35.1	34.1	33.9
	Range	33–37	31.5–37	31–36
	S.D.	1.00	1.10	0.98

Liver Function Tests

Table 2. Mean values of alkaline phosphatase and zinc sulphate turbidity tests

		Group 1 (Europeans)	Group 2 (Afr.Male Nurses)	Group 3 (Cato Manor Africans)
Alkaline Phosphatase (K.A.units)	Mean	6.1	6.9	9.3
	Range	2.5–9.5	3.5–11	4.5–24
	S.D.	1.7	1.9	3.1
Zinc Sulph. Turbidity	Mean	2.9	5.6	6.3
	Range	1–7	2–15	2–16
	S.D.	1.7	2.6	2.4

Table 3. Percentage with abnormal liver function tests

	Group 1 (Europeans)	Group 2 (Afr.Male Nurses)	Group 3 (Cato Manor Africans)
Serum Bilirubin > 1 mgm.%	0	0	0
Alkaline Phosphatase > 13 units	0	0	6
Cephalin Cholesterol > ++ 48 hrs.	4	22	44
Zinc Sulphate Turbidity > 4 units	16	64	84

The results of 4 liver function tests are reported in Tables 2 and 3. In no instance is the serum bilirubin raised above normal. In only 6% of Group 3 is the alkaline phosphatase beyond the normal range, although there is a significant difference between the mean values of the Cato Manor group and Groups 1 and 2 ($P < 0.1\%$). Despite a clinical impression of the unreliability of the cephalin cholesterol flocculation test, the finding that a result of more than 2+ after 48 hours is present in 44% of the Cato Manor Africans and 22% of the African Male Nursing Staff, indicates a significant difference between each of these groups and that of the European control group. The zinc sulphate turbidity is raised in almost all of the Cato Manor Africans, and in the majority of Group 2. Both African groups show a significant degree of elevation compared with Europeans ($P < 0.1\%$), but there is no significant difference between the 2 African groups.

Serum Proteins

Chemical fractionation of the serum proteins shows significant differences between the 3 groups (see Table 4)

Table 4. Mean values of serum proteins by chemical fractionation in G./100ml.

		Group 1 (Europeans)	Group 2 (Afr.Male Nurses)	Group 3 (Cato Manor Africans)
Total Protein	Mean	7.20	7.49	7.14
	Range	6.30-8.34	6.30-8.82	6.20-8.26
	S.D.	0.54	0.50	0.42
Serum Albumin	Mean	3.94	3.91	3.23
	Range	3.20-5.10	3.05-5.16	2.61-3.91
	S.D.	0.51	0.56	0.29
Serum Globulin	Mean	3.26	3.58	3.91
	Range	2.83-3.88	2.90-4.41	3.05-4.78
	S.D.	0.29	0.31	0.37
A/G Ratio		1.21:1	1.09:1	0.83:1

The above table indicates that the African Male Nurses have a mean total protein which is significantly higher than that of the Cato Manor group ($P < 0.1\%$). The mean serum albumins of the African Male Nurses and the Europeans are similar, whilst that of the Cato Manor Africans is significantly lower than those of the other 2 groups ($P < 0.1\%$). However, the mean total globulin

of the Cato Manor subjects is significantly higher than those of the other 2 groups ($P < 0.1\%$), and that of the African Male Nursing Staff is intermediate, being significantly higher than that of the Europeans and significantly lower than that of the Cato Manor Group ($P < 0.1\%$).

Table 5. Mean values of serum proteins by paper electrophoresis in G./100ml.

		Group 1 (Europeans)	Group 2 (Afr.Male Nurses)	Group 3 (Cato Manor Africans)
Serum Albumin	Mean	4.19	4.26	3.52
	Range	3.58-5.29	3.31-5.27	3.04-4.32
	S.D.	0.47	0.49	0.26
Serum Globulin	Mean	3.01	3.23	3.62
	Range	2.72-3.41	2.75-3.84	2.94-4.47
	S.D.	0.25	0.26	0.31
A/G Ratio		1.39:1	1.32:1	0.97:1

Table 5 shows that the results obtained by paper electrophoresis confirm the findings of chemical

fractionation. In brief, the serum albumin of the Cato Manor Africans is significantly lower than those of the other 2 groups which are similar, whilst the difference between the mean total globulins of the 3 groups are all significant, that of the Cato Manor Africans being highest, and that of the Europeans lowest ($P < 0.1\%$).

Table 6. Mean values of serum globulin fractions by paper electrophoresis in G./100 ml.

		Group 1 (Europeans)	Group 2 (Afr.Male Nurses)	Group 3 (Cato Manor Africans)
$Alpha_1$	Mean	0.30	0.28	0.33
	Range	0.16-0.41	0.21-0.40	0.23-0.41
	S.D.	0.06	0.05	0.04
$Alpha_2$	Mean	0.64	0.59	0.73
	Range	0.54-0.83	0.44-0.87	0.57-0.96
	S.D.	0.07	0.10	0.10
Beta	Mean	0.85	0.79	0.93
	Range	0.65-1.07	0.56-1.06	0.66-1.26
	S.D.	0.09	0.10	0.11
Gamma	Mean	1.22	1.57	1.69
	Range	0.91-1.46	1.13-2.34	1.13-2.36
	S.D.	0.15	0.23	0.22

The mean values of the globulin fractions obtained by paper electrophoresis are shown in Table 6. The striking feature is that the gamma globulins of both African groups are similar, being significantly higher than that of the Europeans($P < 0.1\%$). The $alpha_2$ globulin of the Cato Manor Africans is significantly higher than that of the other 2 groups, and both the $alpha_1$ and beta globulins of the Cato Manor Africans are significantly higher than those of the African Male Nursing Staff($P < 0.1\%$).

Table 7. Comparison of mean gamma globulin in G./100 ml. with zinc sulphate turbidity.

		Gamma Globulin	Zinc Sulph. Turbidity
Group 1 (Europeans)	Mean	1.22	2.9
	Range	0.91-1.46	1-7
	S.D.	0.15	1.7
Group 2 (Afr.Male Nurses)	Mean	1.57	5.6
	Range	1.13-2.34	2-15
	S.D.	0.23	2.6
Group 3 (Cato Manor Africans)	Mean	1.63	6.3
	Range	1.13-2.36	2-16
	S.D.	0.22	2.4

A fairly close correlation between the zinc sulphate turbidity and the gamma globulin level is present. This is shown in Table 7.

Table 8. Percentage of African Male Nursing Staff and Cato Manor Africans with serum proteins within the European range.

	Tot. Prot.	Ser. Alb.	Ser. Glob.	Alpha$_1$	Alpha$_2$	Beta	Gamma
Group 2 (Afr.M. Nurses)	98%	90%	74%	100%	72%	90%	24%
Group 3 (C.M. Afr.)	98%	36%	30%	100%	80%	88%	16%

Table 8 indicates that the serum albumin of the African Male Nursing Staff is within the European range in 90% of subjects, but the gamma globulin falls within European limits in only 24%. Of the Cato Manor Africans, 36% show a European level of serum albumin, and only 16% have a serum gamma globulin within the European range.

Discussion of Findings

The haematological investigations were performed in order to provide standards for comparison with those in amoebiasis, and the findings are not relevant to the present discussion.

Of the 4 liver function tests performed, only the zinc sulphate turbidity and cephalin cholesterol flocculation were commonly abnormal. These 2 tests depend on changes in the serum proteins and are not strictly tests of liver function. Zinc sulphate turbidity can be correlated with the level of the gamma globulin (Kunkel,1947), and elevation of the latter in the African adequately explains the frequency of abnormal zinc sulphate turbidity tests. The mechanisms of cephalin cholesterol flocculation is more complex (Moore et al.,1945), but it is probable that the frequency of positive results in the African is due to quantitative and qualitative alterations in the serum albumin and globulin fractions. In my hands this test has been inconsistent. The reagent is unstable and occasionally conflicting results are obtained on the same serum.

The basic problem to be discussed is that of the serum protein pattern in the African. Both chemical fractionation and paper electrophoresis show that the African labourer from Cato Manor has the characteristic protein disturbance of a low serum albumin and raised globulin that has been reported throughout Africa. The following factors have been suggested to account for this disturbance.

1. Tropical Parasitism

Holmes et al.,(1951) reported the characteristic protein pattern in Uganda Africans. Further studies (Holmes et al.,1955) on 3 groups of Africans, living in different environments and receiving different diets, but all subject to malarial infection (although to a varying extent), indicated that chronic malaria was an important causative factor in Uganda. It is well known that parasitic infections, including malaria, may cause a reduction of the serum albumin and elevation of the gamma globulin (Dole et al.,1945; Taylor et al.,1946, 1949; Macheboeuf et al.,1951; Stauber,1954).

2. Genetic Factors

Rawnsley et al.,(1956), in a comparison of North American Negroid blood donors with Europeans, stated to be sharing the same environment and dietary background, demonstrated a significantly higher gamma globulin level in Negroids than in Europeans, and suggested that this difference is due to genetic factors. Bersohn et al.,(1954) in Johannesburg, found no significant differences between the albumin and total globulin levels of European and African infants at birth, although the gamma globulin level of the African infants was higher than that of the Europeans. Stanier and Thompson (1954) in Uganda, on the other hand, found a significantly lower serum albumin, but no significant difference between the gamma globulin levels of newborn African infants compared with American figures. However, Thompson (1956) showed that by the end of the first year of life the characteristic adult pattern of a low serum albumin and raised gamma globulin had emerged. As by this time 79% of the infants had become infected with malaria

it was felt that the changing pattern may have been largely due to malarial infection.

A genetic cause cannot be entirely excluded at present, but hereditary factors are always difficult to disprove, and because of this they should not be accepted until all other factors are ruled out. In the case of differences in the serum proteins convincing proof of a genetic origin is lacking.

3. Protein malnutrition with or without chronic liver disease.

Arens and Breck (1954) have shown that the serum protein fractions in non-tropical African subjects in Cape Town are intermediate between, and significantly different from, Europeans on the one hand and tropical Africans on the other. It was also shown that compared with Kampala Africans, the Cape Town African has a higher mean total protein due to a higher level of both albumin and globulin of approximately the same order. As tropical parasitism in the sense used by Holmes et al.,(1955) does not occur in Cape Town, these changes in the Cape Town African cannot be attributed

to malaria. It is possible that tropical parasitism may be responsible for the more marked changes found at Kampala, but it is apparent that the similar basic changes found in Cape Town must be due to other factors such as protein malnutrition with or without liver disease.

Table 9. Mean values of serum proteins in G./100 ml. by chemical fractionation in Kampala, Cape Town and Durban.

	Total Protein	Serum Albumin	Serum Globulin	A/G Ratio
Europeans:				
Kampala	7.31	4.25	3.06	1.30:1
Cape Town	7.63	4.27	3.36	1.28:1
Durban	7.20	3.94	3.26	1.21:1
Africans:				
Kampala	7.60	3.27	4.33	0.75:1
Cape Town	8.24	3.55	4.69	0.79:1
Cato Manor	7.14	3.23	3.91	0.83:1
African Male Nurses	7.49	3.91	3.58	1.09:1

Despite the difference in the chemical methods used it can be seen in Table 9 that the figures for Durban Europeans are similar to those in Cape Town and Kampala.

Malaria is unlikely to be a significant factor in Durban as it is now an uncommon disease here. In 1956, of 33,583 African admissions to this hospital, malaria was diagnosed in only 7 cases. As Arens and Broek(1954) state "Chronic liver disease is prevalent throughout the continent. There is much evidence that protein malnutrition may be an important factor in the aetiology of this chronic liver disease. Therefore protein malnutrition and chronic liver disease may individually or together contribute to the low serum albumin." It is extremely difficult in the adult African to separate these 2 factors in order to assess how much disturbance may be due to protein malnutrition, and how much to chronic liver disease. However, if it can be shown that this characteristic pattern is reversible, or is not present in better nourished Africans, it may indicate that if liver disease is a factor it is not necessarily irreversible.

In Durban, the serum protein pattern of the Cato Manor African labourer conforms to that which is seen throughout Africa, but the better nourished Durban African (Group 2) has an albumin level equal to that of Europeans,

although a raised gamma globulin is present in both African groups. Although Edozien(1957) in Nigeria was unable to show alterations in the protein pattern, either according to social status and income or by feeding experiments, there is some evidence that if an adequate diet is received for a sufficient period the albumin level in the African may reach European levels, although hypergammaglobulinaemia persists. Holmes(1954) studied 15 Kampala students at the commencement of their term, and again after they had received an adequate diet for 3 months, and found a marked rise in albumin, whilst the total globulin, which was raised, remained unchanged.

In a further study of 3 patients with protein malnutrition Stanier and Holmes(1954) showed that, following a high protein diet, the albumin invariably rose whilst the high total globulin did not invariably fall. More recently, Schofield (1957) has shown that in West Africans resident in Britain the protein pattern approaches that of the European in the course of time. After 4 - 8 years the albumin reaches the European range, but the gamma globulin, although showing some reduction, remains elevated. It is noteworthy that in this group the albumin and gamma globulin levels closely resemble

those of the better nourished Durban African. In Salisbury, Carr et al.,(1958) report a similar pattern in a small number of African orderlies investigated. It would therefore seem that, whereas the reduced albumin of the African is reversible by environmental factors, the gamma globulin tends to remain elevated for considerably longer, and there is no evidence yet available that it returns completely to European levels.

My results show that 24% of the African Male Nursing Staff, but only 8% of the Cato Manor subjects, have serum protein patterns indistinguishable from those of Europeans. Moreover, of those with a European type of serum protein pattern, all the Cato Manor Africans and all but 2 of the African Male Nursing Staff have levels of Hb, E.S.R.,P.C.V. and liver function tests within the European range. The different incidence of findings between the 2 African groups suggests that environmental factors, of which diet may be the essential one, are responsible. It is doubtful whether the incidence of liver disease in the African is sufficiently high to account for 92% of the Cato Manor subjects possessing a protein pattern abnormal by European standards. Certainly

the incidence of liver disease at routine autopsy at this hospital is far less than this (Wainwright,1958).

Although my results appear to confirm the hypothesis that protein deficiency is the main factor in producing the African type of serum protein pattern, it must be emphasised that direct proof is still lacking. All that has been shown is that in a different environment the albumin reaches European levels, whereas the gamma globulin is much less affected. It has been shown that malaria cannot be the responsible factor in Cape Town and Durban, but it may be that other, less obviously pathogenic, parasites play a part. Throughout much of the continent the African has a high incidence of bilharzial infection, ascaris, trichocephalus and other forms of worm infestation. Although there is little evidence that these affect the serum proteins, it is likely that their incidence will be reduced in those subjects of a higher socio-economic group who are able to afford a better diet. In the same way, repeated exposure to infections, such as tuberculosis, may be of importance, and less exposure will occur in precisely

those Africans able to live in a better environment and receive a better diet. It is impossible to separate diet from other environmental factors, and it may be that the African type of protein pattern is the result of a combination of factors.

It is also possible that hypergammaglobulinaemia in the African is due to different factors from those causing the hypoalbuminaemia. Miller and Bale(1954) have shown experimentally that albumin is formed by the parenchymal cells of the liver whereas there is much evidence that gamma globulin is formed at an extra-hepatic site(Good,1957). Characteristically chronic liver disease produces a raised serum gamma globulin with depression of the albumin, but any condition associated with stimulation and proliferation of the reticulo-endothelial system may give rise to an increased gamma globulin. This occurs not only in liver disease, but also in many chronic infections, particularly those produced by large viruses and protozoa, and also in diseases characterised by granuloma formation. Certainly hypergammaglobulinaemia is not specific and represents a heterogeneous response to multiple types of disease disturbance.

It is now well established that the African has a hypergammaglobulinaemia. Good(1957), in a comprehensive investigation of the site and mechanism of antibody and gamma globulin formation, has shown that the cells of the reticulo-endothelial system, particularly the plasma cells, are responsible both for gamma globulin production and immune responses. The recent demonstration by Gajdusek (1958) of a high incidence of auto-immune antibodies in European subjects with liver disease, collagen disease and paraproteinaemias is of interest in view of Gillman and Gillmans'(1951) opinion that there is indirect evidence of an abnormal reticulo-endothelial and connective tissue response in some of the disease patterns which are common in the African.

Since it is likely that some stimulus to the reticulo-endothelial system occurs in the African in infancy and early childhood it is of great importance to investigate further the development of hypergammaglobulinaemia in the African infant and child. At present the nature of this stimulus remains speculative. There appears to be an environmental factor or factors which begin to act at an early age. These factors may be predominantly dietary, although

parasitic infestation, or repeated exposure to infection may also play a part. This results in a serum protein pattern, and other biochemical changes, which are abnormal by European standards. A somewhat different disease pattern develops, including a greater liability to chronic liver disease. At some stage these changes may become irreversible, but, on our present inadequate evidence, following prolonged exposure to a European type of diet and environment many Africans steadily approximate more closely to European standards.

Summary and Conclusions

It has been shown that whereas the Natal African labourer has a serum protein pattern similar to that described elsewhere in Africa, those of a higher socio-economic group have a European level of serum albumin although the gamma globulin is elevated.

The zinc sulphate turbidity test is elevated, and runs parallel with the gamma globulin level. The cephalin cholesterol flocculation is positive in 44% of the Cato Manor Africans, and in 22% of the African Male Nursing Staff.

The cause of the African type of protein pattern remains obscure. Evidence of a genetic origin is conflicting. An environmental factor is more probable. This may be predominantly dietary, but parasitic infestation and repeated exposure to infection are not excluded. The pattern may be due to a combination of such factors. It has been shown that the low level of serum albumin is commonly reversible under suitable circumstances, whereas the elevated gamma globulin has less tendency to fall to European levels.

PART II.

THE HAEMATOLOGICAL FINDINGS, SERUM PROTEIN PATTERN AND LIVER FUNCTION TESTS IN THE NATAL AFRICAN WITH AMOEBIASIS

Section 1.

An Investigation of the Pathological and Clinical Evidence of Amoebic Hepatitis

It is apparent from the literature that much confusion exists over the pathology and clinical manifestations of hepatic amoebiasis. The terms amoebic liver abscess and amoebic hepatitis are frequently used synonymously, and many authors writing on liver function in amoebiasis fail to make clear what form of amoebic disease of the liver is being described. Others base their findings on the ill-defined condition of amoebic hepatitis which has received widespread, but doubtful, status in the literature. As claims are made that laboratory investigations are of value in the diagnosis of this condition it is important to attempt to define and, if possible, to distinguish it from liver abscess.

Before comparing my findings in proven liver abscess with those reported elsewhere it is therefore necessary to review some of the pathological and clinical evidence of amoebic hepatitis and to compare it with the disease as it is seen in the Durban African.

Pathological Evidence of Amoebic Hepatitis

Although there must be a stage in which Entamoeba histolytica invades the liver before abscess formation, little pathological evidence of this exists. Much of it is based on animal experiments which may not reflect the pathogenesis in human beings.

Councilman and Lafleur(1891) described a case of multiple liver abscesses which also showed diffuse, centrilobular necrosis affecting all parts of the liver, which they attributed to absorption of chemical products of amoebae from the intestinal canal. Rogers(1922) suggested that amoebae reaching the liver become entangled in blood clot in the intra-lobular veins producing congestion of the liver. The vast majority of amoebae then undergo degeneration. This condition he termed

"presuppurative amoebic hepatitis". However, this pathology has never been demonstrated satisfactorily and remains hypothetical.

Craig(1944) states that small fibrous areas are sometimes seen in fatal cases of amoebic dysentery, and regards these as representing foci of infection with E. histolytica which have not resulted in abscess formation. He states that in the earliest stage of abscess formation, "there is an area of capillary congestion surrounding an area composed of cytolysed tissue cells in which may be seen red blood corpuscles, degenerated liver cells, and a few leucocytes, connective tissue cells and granular debris with now and then a trophozoite of Endamoeba histolytica lying in a zone of completely cytolysed material". This may either progress to true abscess formation, or healing occurs with connective tissue replacement. DeBakey and Ochsner(1951) indicate that in the early phase of amoebic hepatitis there is a balance between regression towards healing by scar tissue replacement and progression to suppuration and abscess formation. Blame(1952,1953) has a similar view.

Many writers regard invasion of the liver as common, and Radke(1954) states that the liver is invaded in every patient with amoebiasis. Faust(1944) considers amoebic hepatitis to be due to multiple small colonies of E. histolytica although in most cases the amoebae do not colonize the liver parenchyma but die as the result of an amoebastatic action of the liver. However, pathological proof of the frequency of invasion by amoebae, or of an amoebastatic action by the liver is lacking.

Palmer(1938) in a study of the liver in 19 fatal cases of amoebic dysentery found liver abscess in 13. He concludes that definite hepatitis is associated with active amoebic lesions in the colon, the hepatitis being indicated by a generalised increase in the portal connective tissue in all but 1 of his 19 cases, by lymphocytic and mononuclear infiltration, and by constant degenerative changes of a variable type, notably parenchymateus degeneration, fatty change, haemosiderosis and lysis of hepatic cells. In a review of 9,500 autopsy records Chatgidakis(1954) found 157 cases of amoebiasis, of which 87 had amoebic abscess of the liver and 1 had acute amoebic hepatitis. Her findings

in the case of amoebic hepatitis are unique. Small areas of necrosis were seen usually containing remains of necroded liver cells, and with an infiltration of non-granular cells, lymphocytes and monocytes, the lesions somewhat resembling the lymphomatous nodules of typhoid fever. The portal tracts showed slight lymphoid hyperplasia, and amoebae were seen in the sinusoids both at the periphery of the necrotic tissues, and more distant from these areas. However, this case is exceptional because lobar pneumonia and pneumococcal meningitis were found as well as gross acute amoebic colitis with a generalised peritonitis due to perforation of the colon.

Carrera and Sadun(1952) were unable to find amoebae, hepatic necrosis or other inflammatory reactions in the liver in 7 subjects who died with active amoebic colitis. Kean(1955) reviewed histological sections of the liver in 4,478 consecutive autopsies in the Canal Zone, where amoebiasis is prevalent, and could find no evidence of a diffuse amoebic hepatitis. He quotes several other large autopsy surveys in the tropics with similar negative

findings. Kean et al.,(1956) also reviewed the autopsy findings in 148 cases of amoebiasis and were unable to demonstrate hepatitis.

It has been argued that autopsy material may only reveal the late lesions of amoebic liver abscess, and liver biopsy has been undertaken to show the nature of amoebic hepatitis. Bennin and Moretti(1951), who consider hepatic amoebiasis invariably accompanies intestinal amoebiasis, found an increase in peripertal connective tissue in 4 patients with clinical amoebic hepatitis. Heller et al.,(1953), describing 2 cases of amoebic hepatitis presenting as pyrexia of unknown aetiology, report that liver biopsy showed focal necrosis. Nelson et al.,(1955) performed liver biopsies on 9 patients with intestinal amoebiasis, and report a consistent picture which they consider typical of amoebic hepatitis. This consists of a subacute hepatitis, frequently focal in distribution, and usually most marked in the peripertal areas, manifested principally by lymphocytes and plasma cell infiltration in the peripertal areas. Rarely some increase in the polymorphonuclear cells was noted. In no

instance were amoebae seen. Kasliwal and Bhatia(1956) described the liver biopsy findings in 30 cases of intestinal amoebiasis with enlarged livers. They describe reticulo-endotheliosis, diffuse parenchymal damage, disorganisation of hepatic cell plates and focal necrosis as extremely common. Mononuclear portal infiltration with scanty polymorphonuclears was present in 40%. Fibrosis was conspicuously absent; E. histolytica was not seen. They note the similarity to changes due to other protozoal infections such as malaria and kala azar. Chaudhuri and Saha(1956) performed liver biopsies on 15 cases of amoebic dysentery, and found round cell infiltration of the portal tracts in 12, slight, or patchy, fibrosis of the portal connective tissue in about half, parenchymal degeneration in 8, focal necrosis in 2, fatty changes in 4, and hyperactivity of the Kupffer cells in 7 cases. In no instance were amoebae found.

Kean(1955), on the other hand, reviewed the histological sections of liver biopsy specimens of 50 cases diagnosed clinically as amoebic hepatitis, and found 5 abscesses but in no instance was there evidence of

diffuse amoebic hepatitis.

It is clear from the above review that pathological evidence of amoebic hepatitis is conflicting and the existence of a diffuse form of hepatic amoebiasis remains doubtful.

Clinical Evidence of Amoebic Hepatitis

The clinical state of diffuse amoebic hepatitis has been widely recorded in the literature, but this contains a number of syndromes which are dissimilar. At least 3 types are described.

1. Acute Hepatitis

The syndrome of hepatomegaly, hepatic tenderness, fever, leucocytosis, laboratory evidence of hepatic dysfunction and a response to specific anti-amoebic therapy in patients with or without active intestinal amoebiasis has been well described (Sodeman and Lewis, 1945; Sodeman,1950). From a clinical viewpoint the only differentiation from amoebic liver abscess is that

no pus is seen by aspiration or from rupture of the abscess. It seems possible that such cases frequently have a liver abscess, or, less commonly, multiple small abscesses which respond satisfactorily to conservative anti-amoebic therapy. As this condition is clinically indistinguishable from proven liver abscess little is gained by terming it hepatitis rather than liver abscess.

2. Subacute and Chronic Hepatitis

This controversial condition, of which convincing pathological proof has still to be demonstrated, has resulted in much confusion owing to the ill-defined clinical criteria. Klatskin(1946) found that the presenting symptom may not be that of liver pain, but in virtually all cases subcostal or compression tenderness is present, and hepatomegaly is common. A previous history of dysentery may be obtained. Diarrhoea, systemic disturbance and E. histolytica in the stools may or may not be present. Response to bed rest and emetine may occur, but this does not prove the diagnosis which can never be entirely certain. This state is

rarely associated with concomitant acute dysentery, and it is perhaps significant that its frequency appears to be in inverse proportion to the amount of proven acute forms of amoebiasis occuring in the area. In Durban this form of hepatitis in the African is distinctly rare.

There is a growing body of opinion that E.histolytica is not always invasive and that it may live in the bowel as a commensal. There is little evidence that the vast majority of people who harbour E. histolytica in the intestine develop symptoms, and it is likely that some change in the host-parasite relationship must take place before ulceration and symptoms occur. Phillips (1957) has recently reviewed the evidence in favour of synergism between amoebae and bacteria in producing bowel lesions. Invasion of the liver would seem more likely to occur if there is ulceration of the bowel. Those who believe that the presence of E. histolytica in the stools is necessarily harmful are forced to accept figures such as those of Peake and Eskridge(1950) who estimate that in the United States 10 to 30 million subjects harbour E. histolytica in the bowel, and of these

1 million have amoebic hepatitis! This attitude has become even more alarming as some workers do not include tender hepatomegaly in their essential diagnostic criteria, and regard the presence of cysts of E. histolytica in the stools associated with abnormal liver function tests as sufficient evidence for the diagnosis of amoebic hepatitis (Anderson et al.,1955, 1956;Nelson et al.,1955). This is the logical development of Radke's(1954) views, although Radke, himself, regards as noteworthy the frequency with which liver function tests in amoebic liver abscess remain within the normal range. Until proof of the pathology of subacute and chronic amoebic hepatitis is available it would seem reasonable to limit the diagnosis to those cases of tender hepatomegaly who show evidence of invasion of the bowel wall.

3. Hepatitis associated with Acute Amoebic Dysentery

Payne(1945) reviewed 1,000 cases of amoebiasis in India, the majority having dysenteric symptoms, and found mild amoebic hepatitis (tender hepatomegaly) in at least 50% compared with an incidence of 2.8% for liver abscess.

This form of hepatitis was so common that it was regarded as part of the clinical picture of amoebic dysentery.

It is not necessary to assume that invasion of the liver by amoebae must be the cause of this condition. Laboratory and pathological evidence of hepatic disease in ulcerative colitis is well recognised (Pollard and Block,1948; Kimmelstiel et al.,1952; Bargen,1956), and as Kean(1957) points out, tender hepatomegaly in amoebiasis may be due to the same factors that are responsible for its occurence in ulcerative colitis. However, Loeber and D'Antoni(1947), Sodeman(1950), and Sodeman et al.,(1951) noted the disappearance of hepatomegaly and tenderness both when insoluble compounds such as diiodohydroxyquinoline, effective in colonic amoebiasis, and when chloroquine, effective in hepatic amoebiasis were used. Kasliwal and Bhatia(1956) regard this as evidence that this syndrome is due to E. histolytica and not to bacteria.

Amoebic Hepatitis in the Durban African

In Durban amoebiasis in the African is extremely common, and assumes a virulent form. In 1957, 1247

African patients with proven acute amoebic dysentery were admitted to the medical wards of this hospital, and an even larger number were treated as out-patients. During the past 2 years 77 cases of proven liver abscess have been admitted to 1 medical unit, and of these, 22 had concomitant acute amoebic dysentery. As therefore a fairly high proportion of these patients with liver abscess have associated amoebic dysentery, amoebic hepatitis might also be expected to occur in a proportion of the dysenteric patients. At least 50 autopsies are performed each year on fatal cases of amoebic dysentery yet in no instance has it been possible to demonstrate amoebic hepatitis, despite careful search, including histological examination of the liver sections. Moreover, autopsy records show that 1011 routine autopsies were performed at this hospital on Africans during 1957 without evidence of amoebic hepatitis being found (Roach,1958).

Liver biopsy has been undertaken on 30 patients with tender hepatomegaly and harbouring E. histolytica in their stools, but has either revealed pus from an

abscess or has failed to show any specific hepatic lesions. Both liver biopsy and autopsy findings have commonly shown the picture of lymphocyte and plasma cell infiltration in the periportal areas described by Nelson et al.,(1955), but this is not regarded as specific as it is present in a high proportion of African livers without clinical or pathological evidence of amoebiasis (Wainwright,1958).

Within the past 2 years I have seen and examined in 1 medical unit several hundred patients with proven amoebic dysentery, 77 with proven liver abscess, and several additional cases of liver abscess which either did not require aspiration, or failed to yield pus on aspiration. During this period it has not been possible to recognise a single case of subacute or chronic hepatitis.

Although subacute or chronic hepatitis is rare in the Durban African, tender hepatomegaly associated with acute amoebic dysentery is common. The patient rarely complains of liver tenderness as this is overshadowed by

the acute dysenteric symptoms. There are no specific signs, apart from a slight to moderate diffusely tender hepatomegaly which tends to be obscured by the abdominal signs of acute intestinal amoebiasis. In some cases it is impossible to distinguish liver tenderness from tenderness of the hepatic flexure of the colon.

The following investigation was undertaken to assess the incidence and response of this condition to treatment:

The presence of liver tenderness and hepatomegaly was noted in 100 patients with acute amoebic dysentery on admission and on completion of treatment 27-30 days' later. All had dysenteric symptoms, rectal ulceration and trophozoites of E. histolytica in their stools. Fifty were treated with tetracycline and diiodohydroxyquinoline, and 50, in addition, received emetine or chloroquine. In all cases the stools became normal, with healing of the rectal ulceration, and apparent cure before discharge.

Of the 50 receiving tetracycline and diiodo-hydroxyquinoline hepatomegaly was present on admission in 20, and in 18 of these there was also hepatic

tenderness. On discharge the liver had decreased in size in 12 cases, in 7 there was no change, and in 1 the liver had increased in size. Four patients with no hepatomegaly on admission showed non-tender enlargement on discharge. In 2 minimal hepatic tenderness was still present.

Of the 50 who received emetine or chloroquine in addition, hepatomegaly was present in 21 on admission and of these 18 also had hepatic tenderness. On discharge the liver had decreased in size in 14 cases, in 5 there was no change and in 2 the liver had increased in size. Two patients with no hepatomegaly on admission showed non-tender enlargement on discharge. In 4 slight hepatic tenderness was still present.

The clinical significance of hepatomegaly in the African is obscured by the frequency of non-tender liver enlargement of unexplained aetiology, although this is generally considered to be nutritional or cirrhotic. Neither tetracycline and its derivatives nor diiodohydroxyquinoline are effective agents in amoebic liver abscess(Wilmot et al.,1952). It is not rare, in

this hospital, to see patients develop liver abscess whilst on such a regime(Wilmot *et al.*,1958). Diiodohydroxyquinoline alone produces cure in a comparatively small proportion of dysenteric patients. As both tetracycline with diiodohydroxyquinoline, and emetine or chloroquine had a similar effect in reducing hepatic size and tenderness, it is unlikely that in these cases the liver is undergoing widespread invasion by *E. histolytica*. This syndrome can be adequately explained as non-specific hepatitis due to ulceration of the intestine. Provided that slightly tender hepatomegaly is recognised as a common finding in acute amoebic dysentery, it is best to avoid the term "amoebic hepatitis" for this state, as this immediately brings to mind the much more vague condition of subacute or chronic hepatitis associated with chronic amoebic infection of the bowel. This latter state is one of uncertain clinical criteria, and remains without convincing pathological proof.

Summary and Conclusions

In view of confusion in the literature over the terms amoebic hepatitis and amoebic liver abscess the pathological and clinical evidence of amoebic hepatitis has been reviewed. No convincing pathological proof of the condition exists. The clinical state of amoebic hepatitis appears to consist of 3 syndromes:

(a) Acute Hepatitis. This is indistinguishable from amoebic liver abscess except that pus is not demonstrable. No purpose is served in terming this condition hepatitis rather than abscess, as pus, although undoubtedly present, is not necessarily found by aspiration in all cases of liver abscess, and such cases respond satisfactorily to conservative anti-amoebic therapy.

(b) Subacute and Chronic Hepatitis. The clinical criteria of this condition are ill-defined. If the broad criteria of certain authors are accepted the disease must be regarded as absurdly frequent. It remains a matter of opinion whether this condition really exists. Much depends on whether E. histolytica is regarded as

always being invasive, or whether it commonly lives in the bowel as a commensal. There is little evidence that the majority of people who harbour E. histolytica develop symptoms. This syndrome is commonly reported in areas where proven acute forms of amoebiasis are rare, and there are few reports of it occuring in those regions where amoebiasis is severe.

(c) Hepatitis associated with Acute Amoebic Dysentery. This mild form of hepatitis is so common that it is part of the clinical picture of acute amoebic dysentery. It is not necessarily due to invasion of the liver by E. histolytica.

In the Durban African pathological proof of amoebic hepatitis has not been found. Clinical amoebic liver abscess, which either does not require aspiration, or does not yield pus on aspiration, is seen. This corresponds to the syndrome of Acute Hepatitis. Subacute or Chronic Hepatitis is rare, but Hepatitis associated with Acute Amoebic Dysentery is common. Tetracycline and diiodohydroxyquinoline, which are not effective in proven amoebic infection of the liver, are as effective as emetine and chloroquine in curing this condition.

Section 2.

The Haematological Findings, Serum Protein Pattern and Liver Function Tests in the Natal African with Amoebic Dysentery and Amoebic Liver Abscess.

The confusion over the clinical manifestations and pathology of hepatic amoebiasis is reflected in the literature dealing with liver function in this disorder. For the most part, liver function tests have been undertaken in amoebiasis, either as a diagnostic aid in hepatic amoebiasis, or to estimate hepatic dysfunction in intestinal amoebiasis. In many instances no distinction has been drawn between amoebic hepatitis and liver abscess; in others the tests performed have been fragmentary. Commonly there is a lack of controls in assessing both the diagnosis and therapeutic response of these cases. It is not surprising, therefore, that the literature shows wide differences of opinion on the effect of amoebiasis on liver function and the serum protein pattern.

Review of the Literature

Blane(1953) states that liver function tests are negative in amoebic hepatitis. Sherlock(1955) writes that these tests are usually normal in both amoebic hepatitis and liver abscess, and although the serum alkaline phosphatase level is sometimes moderately raised, none of the tests are of specific practical value. She refers to the work of Zuckerbrod et al.,(1948) who, in a study of 3 proven cases of liver abscess, found that the serum bilirubin, albumin and globulin levels and flocculation tests were within the normal range in 2 cases, whilst the third showed reduced serum albumin with increased globulin and a reversed albumin-globulin ratio. Smitskamp(1952) found liver function tests within the normal range in a small series of proven amoebic liver abscess. Drury(1952) described a proven case of amoebic liver abscess in which the liver function tests were normal. Givner and Chang(1953) did not find liver function tests helpful in establishing the diagnosis in 3 cases of liver abscess and 2 of amoebic hepatitis. Conan(1949) found the alkaline phosphatase normal in amoebic hepatitis.

Popper and Schaffner(1957) state that abnormal results in any of the hepatic tests are the exception rather than the rule in amoebic liver abscess, although serum alkaline phosphatase activity and bromsulphalein retention are sometimes increased, and refer to Brem(1955) who found that in 10 cases cephalin cholesterol flocculation was normal, the serum bilirubin was normal in 9, and the albumin and globulin usually normal. In 7 of 8 cases the alkaline phosphatase was raised, and he noted that those with the most extensive involvement had the highest alkaline phosphatase levels.

Sodeman and Lewis(1945) found that in amoebic hepatitis evidence of impaired liver function may be present, but their data is inadequate. Sodeman(1950) writes that in amoebic hepatitis, and even in very advanced liver abscess, liver function tests may be normal. He states that sometimes the cephalin cholesterol flocculation may be positive, in about 12% there is jaundice, and more commonly there is a disturbance of the serum proteins, but only to a very small extent. He emphasises that in amoebic hepatitis

the physical findings are more marked than laboratory evidence of hepatic dysfunction, in contrast to other forms of liver disease such as infectious hepatitis. Zavala and Hamilton(1952) in a discussion of 7 cases of hepatic amoebiasis, of which 4 were proven to have liver abscess, state that liver function tests are important in diagnosis although even in advanced cases abnormalities of liver function may be slight. They state that occasionally there is reversal of the albumin-globulin ratio or positive cephalin cholesterol flocculation, in contrast to true infectious hepatitis when liver function tests may be seriously impaired in patients with few and slight physical findings.

Patterson and Lawlis(1956) did not find liver function tests helpful in the diagnosis of proven liver abscess. The serum albumin was lowered in 12 of 18 patients, the serum bilirubin raised in 2 of 12, and the cephalin cholesterol flocculation positive in 4 of 16 patients. In no instance was there significant elevation of the alkaline phophatase. They conclude that there is more evidence of liver disease on physical

examination than by laboratory findings. Radke(1954), in a study of 15 cases of amoebic liver abscess, regarded as noteworthy the frequency with which liver function tests remained within the normal range. They were completely normal in 6 out of 12 cases, the icteric index being raised in 4.

Charmot(1956) investigated 26 patients with acute amoebic hepatitis. There was almost invariably an increase in alpha globulin, but the usual laboratory tests for hepatic insufficiency gave irregular results. He concludes that the results are more suggestive of a localized liver lesion than of a diffuse hepatitis, but he states that evidence of hepatic insufficiency might be found in cases with large abscesses. De Vries et al.,(1956), in a review of 24 patients with hepatic amoebiasis, and 29 with liver abscess, found hypo-albuminaemia in 2 and hyperglobulinaemia in 11 of 14 of those with liver abscess. Kean(1955) found abnormal liver function in 17 of 30 cases in which the clinical diagnosis of amoebic hepatitis had been made. He

observes(Kean,1957) that "it appears to be the consensus that approximately 50% of individuals with diffuse amoebic hepatitis will have 1 or more abnormal liver function tests."

However, others find that liver function is disturbed to a much greater extent in hepatic amoebiasis. Coirault et al.,(1952), using a sodium sulphate salting out technique in a study of 6 Indo-Chinese patients with hepatic amoebiasis, found reduction of the serum albumin and elevation of the total globulin, the latter being due to a selective increase in the gamma globulin. In addition, the cephalin cholesterol flocculation was positive, and the zinc sulphate turbidity raised in all cases. Coumel et al.,(1948), studied 37 cases of chronic amoebiasis, amoebic hepatitis and 1 liver abscess, and found evidence of liver dysfunction in all, the cephalin cholesterol flocculation being one of the tests most frequently abnormal. Liver function was more disturbed in those with clinical evidence of hepatic involvement.

A different approach is evident in this work of

Coumel et al.,(1948) as they present evidence of liver dysfunction in intestinal amoebiasis. This aspect had been emphasised by Shute(1947) who found that in 73 cases of amoebiasis (including acute amoebic dysentery, chronic amoebic colitis, and amoebiasis presenting with unusual symptoms, but excluding those proved to have hepatic involvement) the cephalin cholesterol flocculation was 2+ or more in 50.6%. He regarded this as evidence of liver parenchyma damage. Following treatment the cephalin cholesterol flocculation became normal. However, the test was negative in 2 of 5 patients with amoebic hepatitis, and it was felt that the series was too small from which to draw conclusions. Capps and Bennett(1949) performed liver function tests on 15 patients with chronic amoebiasis. In 11 these tests showed mild liver dysfunction, and in 10 liver tenderness was present. Following emetine the abnormal findings returned to normal or dropped sharply, and the liver tenderness responded. From this study they conclude that a specific hepatitis is present in a large percentage of cases of chronic amoebiasis. Mazzitelli(1954) performed liver function tests in 153

patients with chronic amoebic colitis and found that 58.8% showed definite impairment of liver function, whilst only 14.3% had completely normal tests.

This aspect has been further stressed by Andersen et al.,(1955) who place great reliance on cephalin cholesterol flocculation in the diagnosis and therapeutic assessment of amoebic hepatitis. They found this test positive in 21 of 42, and an elevated icteric index in 12 of 49, cases of amoebic hepatitis. Only the abnormal tests were repeated during follow up, and these showed great improvement following treatment. In a later investigation Nelson et al.,(1955) further stress the value of the cephalin cholesterol test. Of 29 patients harbouring E. histolytica in their stools this test was positive in 93%. Liver biopsy was performed in 11 patients, and in 9 changes considered to be typical of amoebic hepatitis were found. These histological findings have already been discussed in the preceding section. Hepatic enlargement and tenderness, regarded by most as cardinal features of amoebic hepatitis(Ochsner and DeBakey,1943; Payne,1945; Sodeman, 1950; De Vries et al.,1956) were not considered essential

for diagnosis, hepatomegaly, with or without tenderness, being present in only 48% of their cases. These writers therefore regard cephalin cholesterol flocculation as the most important single diagnostic criterion of amoebic hepatitis in patients harbouring E. histolytica in the stools. However, it should be borne in mind that these workers have the broadest of views regarding the pathogenicity of E. histolytica, one of them having written(Anderson et al.,1953) that amoebiasis is as protean in its clinical characteristics as syphilis, and stating that it is perhaps even more common in the United States than syphilis or malaria, and equally chronic and capable of relapse. In a later study of 41 patients in whom amoebic hepatitis was diagnosed, Anderson et al.,(1956) found hepatomegaly with or without tenderness in 21 of 40 cases, and positive cephalin cholesterol flocculation in 21 of 38 patients. Following treatment the liver size was reduced in all, and 15 of 19 cases showed reduction of cephalin cholesterol flocculation. They conclude that some correlation exists between physical and laboratory examinations in this condition.

Findings in the Natal African

The following investigation of the findings in Natal Africans was undertaken:

Material

Forty-nine African male patients with acute amoebic dysentery, and 31 with uncomplicated amoebic liver abscess were investigated on the day of admission and on completion of treatment 27-30 days later. Follow up studies were conducted for a further 3 months. All those with amoebic dysentery had dysenteric symptoms, rectal ulceration and trophozoites of *E. histolytica* in their stools. All the cases of liver abscess were proven by aspiration of characteristic pus. In neither group was coexisting disease present. The findings in the 50 African labourers from Cato Manor, presented in Part 1 of this thesis, were used as controls.

Full details of the methods, results and statistical analyses are given in Appendices I to III.

Haematological Findings

Table 10. The haematological findings in acute amoebic dysentery and amoebic liver abscess.

		Amoebic Dysentery		Liver Abscess		African Controls
		0/A	0/D	0/A	0/D	
HB (G./100ml.)	Mean	15.8	16.0	12.5	15.2	16.0
	Range	8.2–19.2	11.7–19.3	9.1–16.1	12.3–19.2	13.6–19.0
Wbc.per cu.mm.	Mean	13,100	9,700	13,900	9,200	—
	Range	6,000–27,000	6,000–20,000	6,300–39,000	4,500–14,700	—
E.S.R. mm./1hr.	Mean	22	19	44	28	18
	Range	3–66	1–53	8–61	2–48	2–40
P.C.V. %	Mean	46.6	47.8	37.9	45.3	47.3
	Range	26–54	35–58	28–48	38–54	40.5–55
M.C.H.C. %	Mean	33.8	33.6	32.9	33.6	33.9

Table 10 indicates that in acute amoebic dysentery the mean Hb and E.S.R. are within the range of the control group, whereas in liver abscess the Hb is frequently reduced, and the E.S.R. elevated. Following treatment there is marked improvement. In both conditions there is commonly a leucocytosis of the same order.

Results

Table 11. Percentage of cases of acute amoebic dysentery and amoebic liver abscess with Hb < 14.5G./100ml., Wbc. > 10,000 per cu.mm., and E.S.R. > 9mm./1hr.

	Amoebic Dysentery		Liver Abscess		African Controls
	O/A	O/B	O/A	O/B	
% with Hb < 14.5G./100ml.	18%	14%	71%	22%	4%
% with Wbc > 10,000 per cu.mm.	73%	31%	74%	28%	16%
% with E.S.R.> 9mm./1hr.	80%	67%	87%	78%	78%

Table 11 indicates that approximately three quarters of those with liver abscess have some degree of anaemia, and a similar percentage of both the dysenteric and liver abscess cases have a leucocytosis.

Liver Function Tests

Table 12. Mean values of alkaline phosphatase and zinc sulphate turbidity tests in acute amoebic dysentery and amoebic liver abscess.

		Amoebic Dysentery		Liver Abscess		African Controls
		0/A	0/D	0/A	0/D	
Alkaline Phosphatase (K.A.units)	Mean	9.1	9.3	13.6	11.5	9.3
	Range	5.5-24.5	3-26	8-25.5	5-21	4.5-24
	S.D.	3.5	3.8	4.2	4.2	3.1
Zinc.Sulph. Turbidity	Mean	5.9	6.8	14.2	12.8	6.3
	Range	2-14	2-18	4-28	4-32	2-16
	S.D.	3.6	4.1	6.6	7.7	2.4

From Table 12 it can be seen that in amoebic dysentery the mean alkaline phosphatase and zinc sulphate turbidity do not differ from those of the African controls, whilst in liver abscess both are clearly raised, although there is a slight reduction on completion of treatment.

Table 13. Percentage of cases of acute amoebic dysentery and liver abscess with abnormal liver function tests.

	Amoebic Dysentery		Liver Abscess		African Controls
	O/A	O/D	O/A	O/D	
Ser.Bilirubin > 1mgm.%	0	0	0	0	0
Alk.Phosphatase > 13units	8	8	37	34	6
Ceph.Cholesterol > ++48hrs.	16	27	12	28	44
Zn.Sulph.Turbidity > 4units	53	67	97	93	84

Table 13 shows that about a third of the patients with amoebic liver abscess have a clearly raised alkaline phosphatase which is still present at the end of treatment. The cephalin cholesterol flocculation is not consistent; in both groups there is a tendency for it to become positive following treatment. Almost all those with liver abscess, and the majority of those with amoebic dysentery have a zinc sulphate turbidity which is above a strictly normal level. It has already been shown that this is extremely common in the symptomless African.

Table 14. Percentage of cases of amoebic dysentery and liver abscess with Hb, E.S.R., serum bilirubin, alkaline phosphatase and zinc sulphate turbidity within the control group range.

	Amoebic Dysentery		Liver Abscess	
	0/A	0/D	0/A	0/D
Hb	87.7	93.9	35.5	87.1
E.S.R.	89.8	98.0	29.1	83.4
Ser.Bilirubin	100	100	100	100
Alk.Phosphatase	98.0	98.0	96.8	100
Zn.Sulph.Turbid.	100	93.7	67.8	73.4

Table 14 indicates that there is little disturbance of the Hb, E.S.R., serum bilirubin, alkaline phosphatase and zinc sulphate turbidity in amoebic dysentery, whereas in liver abscess the Hb and E.S.R. commonly fall outside the control range although most return to the control levels within 30 days. Although the zinc sulphate turbidity is less commonly above the wide control group range there is little tendency for it to fall to the control level within 30 days.

Serum Proteins

Table 15. Mean values of serum proteins by chemical fractionation in G./100ml. in acute amoebic dysentery and amoebic liver abscess.

		Amoebic Dysentery		Liver Abscess		African Controls
		O/A	O/D	O/A	O/D	
Total Protein	Mean	6.52	7.20	7.10	7.56	7.14
	Range	4.48-8.15	5.95-8.40	5.60-8.99	6.16-9.65	6.20-8.26
	S.D.	0.81	0.62	0.90	0.69	0.42
Serum Albumin	Mean	2.57	3.12	1.96	2.97	3.23
	Range	1.11-3.57	2.02-4.14	1.22-3.30	2.19-3.66	2.61-3.91
	S.D.	0.54	0.47	0.50	0.38	0.29
Serum Globulin	Mean	3.95	4.08	5.14	4.59	3.91
	Range	3.00-5.53	3.10-5.06	4.14-7.06	3.47-6.43	3.05-4.78
	S.D.	0.56	0.50	0.76	0.61	0.37
A/G Ratio		0.65:1	0.76:1	0.38:1	0.65:1	0.83:1

Table 15 shows that the total protein in acute amoebic dysentery is significantly lower than in either liver abscess or the control group ($P < 1-0.1\%$). This is due to reduction of serum albumin without alteration of the total globulin. Following treatment the albumin rises to the average range, producing a

rise in total protein to the level of the control group.

Despite a reduction of serum albumin below that of the dysenteric patients, the total globulin in liver abscess is increased so that the total protein remains within the usual range. Following treatment there is an increase in albumin without a corresponding fall in total globulin so that the total protein rises. However, the albumin fraction remains lower than that of the control group. All these changes are statistically significant ($P < 0.1\%$).

In Table 16 the results by paper electrophoresis show similar changes in the serum albumin and total globulin.

Table 16. Mean values of serum proteins by paper electrophoresis in G./100ml. in acute amoebic dysentery and amoebic liver abscess.

		Amoebic Dysentery		Liver Abscess		African Controls
		0/A	0/D	0/A	0/D	
Serum Albumin	Mean	2.83	3.46	2.32	3.40	3.52
	Range	1.56-3.66	2.48-4.34	1.52-3.81	2.93-4.05	3.04-4.32
	S.D.	0.48	0.42	0.54	0.31	0.26
Serum Globulin	Mean	3.69	3.74	4.78	4.16	3.62
	Range	2.87-4.88	2.80-4.58	3.74-6.80	3.07-6.12	2.94-4.47
	S.D.	0.53	0.42	0.79	0.63	0.31
A/G Ratio		0.77:1	0.93:1	0.49:1	0.82:1	0.97:1

The following changes in the globulin fractions are shown in Table 17:

(a) In both amoebic dysentery and liver abscess the alpha fractions are significantly raised ($P < 0.1\%$), being higher in liver abscess. Following treatment they fall to the level of the control group.

(b) The beta globulins in both conditions are somewhat reduced, returning to the average African level following treatment.

(c) The gamma globulin in amoebic dysentery is not significantly altered, whereas in liver abscess it is markedly and significantly raised ($P < 0.1\%$), largely accounting for the rise in total globulin. There is only a slight fall, which is not significant, at the end of 27–30 days' treatment.

Table 17. Mean values of serum globulin fractions by paper electrophoresis in G./100ml. in acute amoebic dysentery and liver abscess.

		Amoebic Dysentery		Liver Abscess		African Controls
		0/A	0/D	0/A	0/D	
$Alpha_1$	Mean	.38	.31	.48	.31	.33
	Range	.20–.56	.20–.48	.31–.73	.19–.48	.23–.41
	S.D.	.09	.07	.10	.07	.04
$Alpha_2$	Mean	.90	.77	1.08	.76	.73
	Range	.57–1.35	.55–1.22	.65–1.57	.48–.99	.57–.96
	S.D.	.18	.12	.20	.14	.10
Beta	Mean	.80	.90	.85	.91	.93
	Range	.58–1.06	.55–1.12	.61–1.14	.58–1.48	.66–1.26
	S.D.	.13	.11	.13	.18	.11
Gamma	Mean	1.61	1.76	2.37	2.18	1.63
	Range	1.02–2.29	1.16–2.42	1.40–3.97	1.39–3.99	1.13–2.36
	S.D.	.33	.33	.73	.53	.22

Table 18. Percentage of cases of amoebic dysentery and liver abscess with serum proteins within the control group range.

	Amoebic Dysentery		Liver Abscess	
	0/A	0/D	0/A	0/D
Albumin	38.8	89.8	9.7	87.1
Globulin*	91.8	95.9	48.4	71.0
Alpha$_1$	79.5	93.9	29.1	90.3
Alpha$_2$	63.3	93.9	29.1	90.3
Beta**	100.0	100.0	93.6	90.6
Gamma***	100.0	95.9	54.9	74.2

Not indicated in this table:

* Two cases of amoebic dysentery on admission, and 1 on discharge had total globulins below the control range.

** Eight cases of amoebic dysentery on admission, and 2 on discharge had beta globulins below the control range. Two cases of liver abscess had beta globulins below the control range.

*** Two cases of amoebic dysentery had gamma globulins below the control range.

Table 18 indicates that in amoebic dysentery the albumin and, less commonly, the alpha globulins fall outside the

control group range, but return to the average levels within 30 days. In amoebic liver abscess all fractions apart from the beta globulin tend to fall outside the control range, particularly the serum albumin and alpha globulins. There is less tendency for the gamma globulin to return to the control level within 30 days.

Follow up Findings

Ten cases with liver abscess and 5 with amoebic dysentery were seen at follow up 1 month after discharge.

The results of the liver function tests in these patients are shown in Table 19. The results follow the same pattern as in the larger groups reported in Tables 12 and 13. The markedly raised zinc sulphate turbidity in liver abscess has fallen 1 month after discharge, although it is still elevated above the control level. This is in contrast to the dysenteric cases in whom the zinc sulphate turbidity continues to fall within the control range. In about half of these cases of liver abscess the alkaline phosphatase is raised on admission, but falls to normal following treatment and remains normal

1 month after discharge. The inconsistency of the cephalin cholesterol flocculation is apparent.

Table 19. Liver function tests in 10 cases of liver abscess and 5 of amoebic dysentery on admission, on discharge and at 1 month follow up.

	Liver Abscess			Amoebic Dysentery		
	0/A	0/D	1 Month	0/A	0/D	1 Month
Ser.Bilirubin % > 1mgm.%	0	0	0	0	0	0
Alk.Phos. Mean	13.7	9.0	9.1	9.3	9.2	9.4
%>13 units	50%	10%	0	0	0	0
Ceph.Cholesterol % > ++48hrs.	30%	30%	60%	20%	0	0
Zn.Sulph.T.Mean	15.6	13.0	9.1	4.6	5.6	6.2
% > 4 units	100%	100%	100%	40%	80%	80%

Tables 20 and 21 show the changes in the serum proteins in these patients. In liver abscess the serum albumin continues to rise to the average African level 1 month after discharge, whilst the globulin, although falling, remains elevated. The total protein is unchanged. In the small number of dysenteric patients seen at 1 month follow up the serum albumin remained at the control level, and the globulin fell although this is not significant statistically.

Table 20. Mean serum proteins by chemical fractionation and paper electrophoresis in G./100ml. in 10 cases of liver abscess and 5 of amoebic dysentery, on admission, on discharge and at 1 month follow up.

	Liver Abscess			Amoebic Dysentery		
Chem.Fractionat.	O/A	O/D	1 Month	O/A	O/D	1 Month
Total Protein	7.39	7.58	7.57	7.13	7.67	7.12
Ser.Albumin	2.13	3.01	3.35	2.92	3.38	3.32
Ser.Globulin	5.26	4.57	4.22	4.21	4.29	3.80
A/G Ratio	0.40:1	0.66:1	0.79:1	0.69:1	.80:1	0.87:1
Paper Electroph.						
Ser.Albumin	2.50	3.45	3.67	3.05	3.77	3.61
Ser.Globulin	4.89	4.13	3.90	4.08	3.90	3.51
A/G Ratio	0.51:1	0.84:1	0.94:1	0.75:1	0.97:1	1.03:1

From Table 21 it can be seen that in liver abscess the gamma globulin has only fallen slightly at 1 month follow up, thereby accounting for the raised total globulin.

Table 21. Mean serum globulin fractions by paper electrophoresis in G./100ml. in 10 cases of liver abscess and 5 of amoebic dysentery, on admission, on discharge and at 1 month follow up.

	Liver Abscess			Amoebic Dysentery		
	O/A	O/D	1 Month	O/A	O/D	1 Month
$Alpha_1$	.47	.29	.33	.45	.34	.34
$Alpha_2$	1.06	.75	.74	1.10	.80	.79
Beta	.91	.93	.83	.90	.95	.82
Gamma	2.45	2.17	2.00	1.64	1.81	1.56

Three cases of liver abscess were followed up for a further 3 months.

Table 22. Liver Function tests in 3 cases of liver abscess, on admission, on discharge, at 1 month and 3 months follow up.

		Case 1	Case 2	Case 3	Mean
Zn.Sulph.T.	O/A	26	12	18	18.7
	O/D	26	9	10	15.0
	1 month.	14	7	6	9.0
	3 months.	7	6	6	6.3
Alk.Phos.	O/A	22	10	10.5	14.2
	O/D	10.5	9	6.5	8.7
	1 month.	9	10	7	8.7
	3 months.	11	11	7.5	9.8
Ceph.Chol.	O/A	0	+	+	—
	O/D	+	0	+	—
	1 month.	0	++	+++	—
	3 months.	0	++	++	—

Table 22 shows the liver function tests in these patients. The zinc sulphate turbidity returns to the control level by the third month following discharge. The alkaline phosphatase which was initially elevated in 1 patient falls to normal following treatment. The cephalin cholesterol fails to reflect the trend of the other tests.

Table 23. The mean gamma globulin in G./100ml. in 3 cases of liver abscess on admission, on discharge, at 1 month and 3 months follow up.

On Admission	On Discharge	1 Month	3 Months
2.60	2.19	1.97	1.64

Table 23 shows that the mean gamma globulin of these cases had returned to the control level by the third month. Comparison of the mean gamma globulin levels with the mean zinc sulphate turbidity in liver abscess (see Tables 19,21, 22 and 23) show that these run parallel, and only return to the average African range 3 months after discharge.

One patient with liver abscess is of particular interest as he returned 6 weeks after discharge with a recurrence of the abscess, and was then followed up for 6 months after his second discharge.

Table 24. The serum proteins in G./100ml. by chemical fractionation and paper electrophoresis in a case of liver abscess.

Chem.Fract.	1st Adm.	1st Dis.	2nd Adm.	2nd Dis.	1 Month	3 Months	6 Months
Tot.Prot.	6.90	7.57	6.11	8.03	8.05	6.87	6.50
Ser.Alb.	1.80	2.90	1.58	3.28	3.91	3.16	3.25
Ser.Glob.	5.10	4.67	4.53	4.75	4.14	3.71	3.25
A/G Ratio	.35:1	.62:1	.35:1	.69:1	.95:1	.85:1	1.00:1
Pap.Elect.							
Ser.Alb.	2.07	3.11	1.81	3.09	3.68	3.88	3.71
Ser.Glob.	4.83	4.46	4.30	4.94	4.37	2.99	2.79
$Alpha_1$	.43	.39	.47	.23	.36	.35	.27
$Alpha_2$	1.14	.89	1.12	.99	.96	.61	.58
Beta	.77	1.12	.72	1.48	1.17	.79	.72
Gamma	2.49	2.06	1.99	2.24	1.88	1.24	1.22
A/G Ratio	.43:1	.70:1	.42:1	.63:1	.84:1	1.29:1	1.34:1

Table 24 illustrates a rise of total protein following treatment with a subsequent fall during the follow up period. On each admission the serum albumin was much reduced, and rose following treatment. The serum globulin was extremely high on admission, and remained raised on discharge, only

falling to the control level between 1 and 3 months after discharge. The alpha fractions show a rise on both admissions with a subsequent fall, and the beta globulin, a rise on each occasion following treatment, with a return to the average range during the follow up period. On the first admission the gamma globulin was markedly raised. Following treatment it fell somewhat, but remained elevated and was at approximately the same level when the patient was readmitted. It was still raised when he was discharged, and only fell to the control range between 1 and 3 months after discharge.

Discussion of Findings in the Natal African

In Durban Africans with acute amoebic dysentery the Hb, E.S.R., serum bilirubin, alkaline phosphatase and zinc sulphate turbidity are usually undisturbed, but leucocytosis is common. In this investigation the cephalin cholesterol test was more frequently positive in the control group than in the patients. Anderson et al.,(1958) report similar results in Africans. The mean gamma globulin is not elevated above the usual African level, and the total globulin is undisturbed. Extensive necrosis in the bowel wall due to

the severe intestinal ulceration characteristic of the disease in the Durban African is sufficient to explain the increase in alpha globulins which return to normal following treatment. Although hepatic dysfunction is a well known cause of reduction of the serum albumin, greatly increased protein loss must be a potent factor in producing the low serum albumin levels found in acute amoebic dysentery so that the finding cannot be regarded as suggestive evidence of liver damage. In all the dysenteric patients symptoms were rapidly controlled. The serum albumin rose to the control mean on completion of treatment and remained at this level during follow up, indicating that cessation of dysentery combined with hospital diet quickly corrected the hypoalbuminaemia. Despite reduction in the serum albumin and elevation of the alpha globulin 98% of these patients have liver function tests within the usual African range.

In Durban Africans with amoebic liver abscess the findings are clearly different. Anaemia, leucocytosis and elevation of the sedimentation rate are common features. Recently Lamont and Peeler(1958) have concluded that in

amoebic liver abscess both the degree of anaemia and leucocytosis bear a striking relationship to the duration of symptoms. However, their findings are questionable as little more than half of their cases were proven by aspiration to have liver abscess, and quite a high proportion of these were complicated by rupture into surrounding structures, or by secondary infection.

The following are the findings in 100 consecutive African male patients (admitted to 1 medical unit) with uncomplicated liver abscess. In all instances the diagnosis was proven by the aspiration of pus.

Table 25. Degree of anaemia related to length of history in 100 cases of amoebic liver abscess.

	Under 7 days	8–28 days	Over 28 days	Total
No anaemia	6 (31.6%)	13 (68.4%)	0	19
Mild(12–14G)	5 (18.5%)	17 (63%)	5 (18.5%)	27
Moderate (9–12G)	3 (6.4%)	33 (70.2%)	11(23.4%)	47
Severe(< 9G)	1 (14.3%)	2 (28.6%)	4(57.1%)	7

Table 26. Degree of anaemia related to amount of pus aspirated from liver abscess.

	Small < 125 ml.	Moderate 125–350 ml.	Large > 350 ml.	Total
No anaemia	15 (78.9%)	4 (21.1%)	0	19
Mild(12–14G)	9 (33.3%)	14(51.9%)	4 (14.8%)	27
Moderate (9–12G)	5 (10.6%)	12(25.5%)	30(63.9%)	47
Severe(< 9G)	0	0	7 (100%)	7

Table 25 indicates that there is some relationship between the duration of symptoms and the degree of anaemia although the correlation is not very close. Table 26 shows clearly that there is a closer correlation between the amount of pus aspirated and the degree of anaemia. Although an abscess may not be entirely emptied by aspiration, in general the larger the abscess the more pus is obtained, so that my findings indicate that the larger the liver abscess the more severe is the anaemia.

Table 27. Length of history related to amount of pus aspirated from liver abscess.

	Small < 125 ml.	Moderate 125–350 ml.	Large > 350 ml.	Total
Under 7 days	8 (53.3%)	4 (26.7%)	3 (20%)	15
8 – 28 days	20(30.8%)	20(30.8%)	25(38.4%)	65
Over 28 days	1 (5%)	6 (30.0%)	15(65.0%)	20

It would seem likely that liver abscesses tend to be larger in those patients with the longest histories, and Table 27 indicates that this relationship tends to occur, although there are exceptions. African patients are often inaccurate in their estimation of the duration of symptoms, and this may explain some of the discrepancies. However, occasional instances undoubtedly occur of very large abscesses developing in a short space of time. Three of the present series with large abscesses had symptoms of less than 7 days' duration.

Table 28. Degree of leucocytosis related to length of history in 100 cases of liver abscess.

	Under 7 days	8–28 days	Over 28 days	Total
< 10,000 per cumm.	3 (10.3%)	19 (65.5%)	7 (24.2%)	29
10,000 – 15,000 per cumm.	7 (16.3%)	27 (62.8%)	9 (20.9%)	43
> 15,000 per cumm.	5 (17.9%)	19 (67.9%)	4 (14.2%)	28

Table 29. Degree of leucocytosis related to amount of pus aspirated in 100 cases of liver abscess.

	Small < 125 ml.	Moderate 125–350 ml.	Large > 350 ml.	Total
< 10,000 per cumm.	11 (37.9%)	5 (17.2%)	13 (44.9%)	29
10,000 – 15,000 per cumm.	12 (27.9%)	18(41.9%)	13 (30.2%)	43
> 15,000 per cumm.	6 (21.4%)	7 (25.0%)	15 (53.6%)	28

Tables 28 and 29 indicate that there is little relationship between the degree of leucocytosis and either the duration of symptoms or the size of the liver abscess.

Elevation of the sedimentation rate is a common feature of amoebic liver abscess. This has been stressed by Blanc(1953), Charmot(1956) and others as a valuable index of hepatic involvement in amoebiasis. My results confirm this. Although the E.S.R. tends to be higher in the average African than in Europeans, the commonly heard statement that the sedimentation rate is of little value in the African because unexplained elevation is so frequent, is not entirely true. My findings indicate that marked elevation is frequent in liver abscess, but is unusual in amoebic dysentery.

Jaundice is an uncommon finding in the African with liver abscess. In my cases the serum bilirubin was not raised. When jaundice is present it is usually associated with a large abscess and is obstructive in type. The incidence is much less than that of 15% found by Sodeman and Lewis(1945) in hepatic amoebiasis.

The alkaline phosphatase is inconsistent, being elevated in about a third of the cases, the mean value lying just above the normal range, although it is within

the control range in all except 1 patient. The shortcomings of the cephalin cholesterol flocculation in the African have already been noted, and it does not appear that the test is of value in the diagnosis of liver abscess in these cases. The zinc sulphate turbidity is markedly elevated, and can be correlated with the increased gamma globulin.

Elevation of the alpha and gamma fractions results in a raised total globulin. Neither of these changes in the globulins necessarily indicates liver disease, as elevation of the alpha fractions commonly occurs in inflammatory lesions with tissue breakdown, whilst gamma globulin increase is found in chronic infections(Flynn, 1954; Jenoks et al.,1956). As none of these cases had dysenteric symptoms it is unlikely that either lack of absorption with increased protein loss or dietary protein deficiency are responsible for the marked reduction of serum albumin in liver abscess. The control level is not reached until 1 month after discharge, and the full serum protein pattern does not return to the average African range until 3 months after treatment, indicating a slower return to the African normal due to a more profound disturbance of

protein metabolism such as might be found in liver disease. Certainly, the findings in most cases are indistinguishable from those due to hepatic dysfunction.

It has been shown in Part 1 of this thesis that reduction of the serum albumin and elevation of gamma globulin is common in the symptomless Durban African. These disturbances must be borne in mind when ascribing changes to specific diseases. Moreover, it is probable that these subjects have less reserve of liver function than well nourished Europeans, and may therefore show earlier or more marked disturbance of liver finction in diseases affecting the liver. In addition, such subjects tend to wait until the disease is gross before seeking medical attention. These factors may be of importance when comparing the findings in Durban with those reported in Europeans, and do not necessarily contradict the many reports of only slight disturbance

of liver function in Europeans with liver abscess. However, the latter reports seem incompatible with those which emphasise the value of liver function tests in the diagnosis of hepatitis associated with chronic amoebiasis. In the Durban African, amoebic hepatitis (as distinct from amoebic liver abscess) and chronic amoebiasis are rarely diagnosed, whereas amoebic dysentery and liver abscess are extremely common. In amoebic dysentery there is little evidence of hepatic dysfunction, in contrast to liver abscess in which the laboratory findings are indistinguishable from those of disordered liver function.

Summary and Conclusions

The literature reveals wide differences of opinion on the value of liver function tests in hepatic amoebiasis. This is partly due to failure to distinguish between the conditions of liver abscess and amoebic hepatitis, and also to the broad criteria frequently adopted for the diagnosis of amoebic hepatitis.

In Durban Africans with acute amoebic dysentery leucocytosis, reduction of the serum albumin and elevation of the alpha globulins are common. These return to the control levels within 27-30 days. These changes can be explained purely by the bowel lesions, and there is little evidence of hepatic dysfunction.

In Durban Africans with amoebic liver abscess anaemia is common. This bears a closer relationship to the size of the abscess than to the duration of symptoms. Leucocytosis is as frequent and of the same

order as in amoebic dysentery, and cannot be related to the size of the abscess or to the duration of symptoms. Marked elevation of the sedimentation rate is common. These haematological findings show considerable improvement within 27-30 days. The serum bilirubin is rarely raised, elevation of the alkaline phosphatase is inconsistent, but the zinc sulphate turbidity is markedly raised, and can be correlated with gamma globulin elevation. The serum albumin is reduced, and the alpha and gamma globulins elevated. The alpha globulins rapidly return to normal, but the serum albumin and gamma globulin do not reach the control levels until 1, and 3 months, respectively, after discharge. The findings are indistinguishable from those due to disordered liver function.

Section 3

The Haematological Findings, Serum Protein Pattern and Liver Function Tests in the Differential Diagnosis of Amoebic Liver Abscess in the Natal African.

In the preceding section it was shown that changes occur in the blood picture, serum proteins and liver function in Durban Africans with liver abscess. However, the diagnostic value of these findings depends on their specificity. In this region the conditions which commonly require to be differentiated from liver abscess are cirrhosis of the liver, primary carcinoma of the liver and right basal pneumonia. The following study was undertaken to compare the laboratory findings in these diseases with those in liver abscess.

Material

Three groups of African male patients were investigated, comprising 10 cases each of cirrhosis of the liver, primary carcinoma of the liver and right basal pneumonia. All could clinically have been confused with amoebic liver abscess. Liver biopsy confirmed the diagnosis in all the

cases of cirrhosis and primary hepatic carcinoma. Those with lobar pneumonia had radiological evidence of right basal consolidation and showed a rapid and complete response to penicillin.

The findings in these 3 groups are compared with those of the 31 patients with liver abscess, and the control group of 50 African male labourers from Cato Manor.

The detailed techniques of the methods used are shown in Appendix I, the detailed results are given in Appendix II, and statistical analyses in Appendix III.

Results

Haematological Findings

Table 30. Comparison of haematological findings in cirrhosis, primary carcinoma of the liver, lobar pneumonia and amoebic liver abscess.

		Cirrhosis	Primary Ca. of Liver	Lobar Pneumonia	Liver Abscess	African Controls
Hb(G./100 ml.)	Mean	13.1	13.5	14.2	12.5	16.0
% < 14.5g./100 ml.		90%	70%	60%	71%	4%
Wbc.per cumm.	Mean	7,800	11,300	13,900	13,900	7,900
% > 10,000 per cumm.		20%	60%	50%	74%	18%
E.S.R.mm./1hr.	Mean	42	40	33	44	18
% > 9mm./1hr.		90%	90%	90%	87%	78%
M.C.H.C.	Mean	34.1	32.2	32.4	32.9	33.9

Table 30 indicates that a slight degree of anaemia is frequent in all the conditions, although in liver abscess this is often more severe. Leucocytosis is not common in cirrhosis, but the white cell count is not helpful in differentiating lobar pneumonia, liver abscess and primary carcinoma of the liver. The mean sedimentation rate in all

the conditions is considerably higher than that of the African controls.

Liver Function Tests

Table 31. Comparison of liver function tests in cirrhosis, primary carcinoma of the liver, lobar pneumonia and amoebic liver abscess.

		Cirrhosis	Primary Ca. of Liver	Lobar Pneumonia	Liver Abscess	African Controls
Alkaline Phosph. (K.A.units)	Mean	17.6	37.5	9.6	13.6	9.3
% > 13units		50%	90%	10%	40%	6%
Zn.Sulph.Turb.	Mean	23.1	16.1	7.4	14.2	6.3
% > 4units		100%	80%	80%	97%	84%
Ser.Bilirubin	Mean	1.3	1.9	0.7	0.4	0.4
% > 1mgm./100 ml.		40%	50%	10%	0	0
Ceph.Cholesterol						
% > ++/48hrs.		80%	70%	20%	12%	44%

In primary carcinoma of the liver the alkaline phosphatase is almost always clearly raised (See Table 31). In both cirrhosis and liver abscess it is elevated in a third to a half of the cases, the mean level being somewhat higher in cirrhosis than in liver abscess. In lobar pneumonia it is not significantly affected. In cirrhosis the zinc sulphate turbidity is markedly elevated, and it is commonly raised, but to a lesser extent, in primary carcinoma and liver abscess. It is only slightly affected in lobar pneumonia. In contrast to liver abscess the serum bilirubin is elevated in about half the cases of cirrhosis and primary carcinoma, but only occasionally in lobar pneumonia. The cephalin cholesterol flocculation is difficult to assess as it is so commonly positive in the controls. However, it is frequently positive in both cirrhosis and primary carcinoma of the liver, and far less often so in liver abscess and lobar pneumonia.

Serum Proteins

Both chemical fractionation and paper electrophoresis of the serum proteins show the same trends.

Table 32. Comparison of serum proteins in G./100 ml. by chemical fractionation and paper electrophoresis in cirrhosis, primary carcinoma of the liver, lobar pneumonia and amoebic liver abscess.

		Cirrhosis	Primary Ca. of Liver	Lobar Pneumonia	Liver Abscess	African Controls
Chem.Fractionation						
Tot.Protein	Mean	7.40	6.94	6.74	7.10	7.14
	Range	5.83-9.42	5.91-7.85	5.90-7.85	5.60-8.99	6.20-8.26
	S.D.	.94	.64	.65	.90	.42
Ser.Albumin	Mean	2.02	1.95	2.47	1.96	3.23
	Range	1.17-3.08	1.15-2.74	1.90-2.85	1.22-3.30	2.61-3.91
	S.D.	.66	.51	.29	.50	.29
Ser.Glob.	Mean	5.38	4.99	4.27	5.14	3.91
	Range	4.36-6.66	3.17-5.89	3.66-5.23	4.14-7.06	3.05-4.78
	S.D.	.71	.82	.50	.76	.37
A/G Ratio		.38:1	.39:1	.58:1	.38:1	.83:1
Paper Electrophoresis						
Ser.Albumin	Mean	2.57	2.39	2.75	2.32	3.52
	Range	1.39-3.50	1.77-3.17	2.19-3.09	1.52-3.81	3.04-4.32
	S.D.	.74	.45	.35	.54	.26
Ser.Glob.	Mean	4.83	4.55	3.99	4.78	3.62
	Range	3.54-6.05	3.06-5.59	3.58-4.93	3.74-6.89	2.94-4.47
	S.D.	.75	.77	.39	.79	.31
A/G Ratio		.53:1	.53:1	.69:1	.49:1	.97:1

Table 33. Comparison of serum globulin fractions in G./100 ml. by paper electrophoresis in cirrhosis, primary carcinoma of the liver, lobar pneumonia and amoebic liver abscess.

		Cirrhosis	Primary Ca. of Liver	Lobar Pneumonia	Liver Abscess	African Controls
$Alpha_1$	Mean	.27	.43	.47	.48	.33
	Range	.17–.44	.31–.51	.32–.62	.31–.73	.23–.41
	S.D.	.08	.07	.10	.10	.04
$Alpha_2$	Mean	.61	.88	1.04	1.08	.73
	Range	.42–1.03	.60–1.16	.73–1.27	.65–1.57	.57–.96
	S.D.	.17	.20	.17	.20	.10
Beta	Mean	.85	.91	.81	.85	.93
	Range	.55–1.38	.73–1.13	.69–.91	.61–1.14	.66–1.26
	S.D.	.28	.13	.06	.13	.11
Gamma	Mean	3.10	2.34	1.67	2.37	1.63
	Range	2.13–3.97	1.32–3.74	1.18–2.49	1.40–3.97	1.13–2.36
	S.D.	.65	.78	.40	.73	.22

Table 32 indicates that:

(a) In all the diseases the serum albumin is reduced significantly below that of the controls ($P < 0.1\%$). The reduction is most marked and of a similar order in

cirrhosis, primary carcinoma and liver abscess.

(b) Conversely, in all the diseases the total globulin is significantly elevated above that of the control group although in lobar pneumonia the elevation tends to be less than in cirrhosis, primary carcinoma and liver abscess.

Table 33 indicates the following changes in the globulin fraction.

(a) The alpha globulins in cirrhosis are significantly lower than in primary carcinoma, lobar pneumonia, liver abscess and the control group($P < 1-0.1\%$). In primary carcinoma, lobar pneumonia and liver abscess they are significantly elevated above that of the controls ($P < 0.1\%$).

(b) In lobar pneumonia the gamma globulin is not significantly disturbed, whereas in each of the other 3 conditions it is significantly elevated above that of the control group($P < 0.1\%$), being highest in cirrhosis.

Discussion

The changes in the blood picture, liver function tests and serum proteins in Europeans with cirrhosis of the liver are fully described(Marrak and Hoch,1949; Martin,1949; Sterling et al.,1949; Popper et al.,1950; Rafsky et al., 1950; Whitman et al.,1950; Franklin et al.,1951; Popper and Schaffner,1952; Welin,1952; Flynn,1954; Mackay et al., 1954; McQueen et al.,1954; Kuhns,1955; Jenoks et al.,1956; Owen and Robertson,1956). Despite the differences between apparently healthy Africans and Europeans, it is evident that in African patients with clinical symptoms and signs of cirrhosis the laboratory findings conform to those described in Europeans.

Reports of the findings in primary carcinoma of the liver differ considerably. Most of the data is based on European subjects. Some note that elevation of the serum bilirubin and alkaline phosphatase is common(Spatt and Graysel,1948; Galluzi et al.,1953; Spellberg,1954; Popper and Schaffner,1957). Others do not find these tests helpful(Holley and Pierson,1948; Lichtman,1953; Edmondson and

Steiner,1954; Sherlock,1955). In a study of the changes in African subjects, Bersohn(1957) found the mean level of the alkaline phosphatase to be higher in primary carcinoma than in cirrhosis. He also noted elevation of the alpha$_2$ and beta globulins, and a dissociation between the alkaline phosphatase and serum bilirubin levels in primary carcinoma.

Spellberg(1954) states that liver function tests in primary carcinoma are variable, and depend to a great extent on the presence of an underlying cirrhosis. If there is marked derangement of the "liver profile" the presence of cirrhosis is likely. My findings indicate that, although the mean values in primary carcinoma of the liver show a pattern of liver function tests and serum proteins different from those in cirrhosis and liver abscess, there is a wide variation in the individual cases. In some instances the findings are similar to those in cirrhosis. In others there is comparatively little disturbance. No correlation could be found from liver biopsy and autopsy findings between either the type of

carcinoma or the degree of accompanying cirrhosis with the liver function tests and protein patterns of the individual cases. However, the alkaline phosphatase was clearly raised in 9 of 10 cases and tended to be considerably higher than in cirrhosis. The alpha globulins were elevated in 7 cases. This was not found in cirrhosis, presumably as they depend on the amount of tissue destruction occurring within the carcinoma. The cephalin cholesterol flocculation, serum bilirubin, zinc sulphate turbidity and albumin and globulin levels were not of assistance in differentiating the 2 conditions, although a normal cephalin cholesterol flocculation associated with normal zinc sulphate turbidity and gamma globulin levels favour primary carcinoma of the liver.

Sherlock(1955) states that a leucocytosis of about 10,000 cells per cu.mm. is usually present in primary carcinoma as opposed to a leucopenia in cirrhosis, and the finding of a leucocytosis in cirrhosis should suggest a complication such as primary liver cell carcinoma. In my cases 7 of the 10 with primary carcinoma had a leucocytosis compared with only 2 in cirrhosis.

Leucocytosis was present in all those cases of primary carcinoma with raised alpha globulins.

In lobar pneumonia the globulins showed a distinct difference from those in liver disease on the one hand, and the African controls on the other. Nine showed elevation of the alpha globulins, such an increase being well described in lobar pneumonia(Longsworth et al., 1939; Flynn,1954; Kuhns,1955; Jencks et al.,1956). The gamma globulin was comparatively undisturbed. Apart from elevation of the serum bilirubin and a raised alkaline phosphatase in 1 case each, the liver function tests remained within the usual African range. This is in contrast to the findings of Zimmerman and Thomas(1950) who found that liver function was widely and almost invariably impaired in American Negroes with pneumococcal pneumonia.

Conclusions

The diagnosis of amoebic liver abscess is primarily clinical, although radiology and aspiration are of notable

assistance. The place of laboratory investigations in the differentiation of this condition from cirrhosis, primary carcinoma of the liver and right basal pneumonia is small. However, comparison of the changes found in these 4 diseases indicate the following points which may be of help occasionally:

1. Leucocytosis is uncommon in cirrhosis.
2. Elevation of the serum bilirubin is uncommon in liver abscess.
3. The alkaline phosphatase tends to be highest and most consistently raised in primary carcinoma. It is rarely elevated in lobar pneumonia.
4. The cephalin cholesterol flocculation test is of little value in the African, but it is unlikely to be negative in cirrhosis.
5. The zinc sulphate turbidity is consistently and most markedly raised in cirrhosis. It is almost always raised, but to a somewhat lesser extent, in primary carcinoma and liverabscess. It is not significantly elevated above the average African range in lobar pneumonia.

6. The serum albumin is commonly reduced in all 4 conditions, but less markedly so in lobar pneumonia.
7. Conversely the total globulin is commonly raised in all 4 conditions, but less markedly so in lobar pneumonia.
8. The alpha globulins are not raised in cirrhosis. They may be raised in primary carcinoma, and are very commonly elevated in liver abscess and lobar pneumonia.
9. Changes in the gamma globulin are paralleled by the zinc sulphate turbidity; a marked elevation occurring in cirrhosis, a somewhat less marked rise in liver abscess and primary carcinoma and usually little change being found in lobar pneumonia.

CONCLUSION OF THESIS

In the first part of this thesis it was shown that the liver function tests and serum proteins of the Natal African labourer differ from those of Europeans, but are similar to those of Africans elsewhere on this continent, and the aetiology of these differences was discussed. It cannot be claimed that this group of Natal Africans is strictly normal in the sense that Europeans are. However, the normality of a group is related to environment. My findings are therefore those of the usual African living in this region, and their practical value is that Africans of this group constitute the vast bulk of admissions to this hospital. The changes described by me in Natal Africans with amoebic dysentery, liver abscess, cirrhosis, primary carcinoma of the liver and lobar pneumonia are of significance when compared with the usual findings in the Natal African labourer, but this does not mean that Europeans with these diseases will necessarily shew the same changes, and it is not valid to relate the findings in Europeans to those in Africans.

For many years E. histolytica was regarded as an essentially invasive parasite(Craig and Faust,1943). This led to the view that wherever there is E. histolytica there is pathology, and doubtless favoured the acceptance of subacute or chronic amoebic hepatitis as an important entity. At the present time our concept of amoebiasis is changing. With the increasing recognition of non-pathogenic E. hartmanni and possibly E. minuta forms(Burrows,1957; Freedman and Elsdon-Dew, 1958; Heare,1957) the modern trend is to regard the finding of trophozoites or cysts in the stools in conditions as vague as subacute or chronic hepatitis as frequently coincidental, and not to attribute a causal relationship to the parasite(Elsdon-Dew,1958).

Review of the literature showed that pathological and clinical evidence of subacute or chronic hepatitis is slight, and opinion is divided over the diagnostic value of liver function tests in this doubtful condition. In Durban we have found little pathological or clinical evidence of the state, although a mild form of acute hepatitis associated with acute amoebic dysentery is common.

This state may be a non-specific reaction of the liver to the bowel lesions.

In the Natal African the changes in the haematological findings, serum albumin and alpha globulins which occur in amoebic dysentery are not necessarily due to liver dysfunction. In amoebic liver abscess the changes in liver function and the serum proteins are much more marked, and are indistinguishable from those produced by disordered hepatic function. Although on the whole these changes form different patterns from those found in cirrhosis, primary carcinoma of the liver and lobar pneumonia they cannot be regarded as diagnostic. The diagnosis of amoebic liver abscess remains primarily clinical, and in this disease, as in so many others, the place of laboratory investigations is secondary. In the African in particular liver function tests should be interpreted with caution, and related to the patient's condition at the bedside.

Review of the literature showed that in many instances different findings from mine are described,

particularly in amoebiasis and primary carcinoma of the liver. However, not only do European "normals" differ from the Natal African, but in certain instances the diseases assume a different form and severity. For example, amoebiasis in the Natal African is acute and virulent, whereas in Europeans it is commonly much milder and of vague symptomatology.

It has been shown that the findings in Natal Africans of a higher socio-economic group, living in a better environment, and receiving a diet more akin to that of Europeans differ from those of the African labourer. It is probable that the African labourer, as his social and economic conditions improve, will tend to show similar values to this group. However, at the same time the diseases which are at present common in the labouring Natal African are likely to diminish in frequency, and as a result the striking changes in the laboratory findings produced by some of these diseases will be found less frequently.

It has been suggested that following prolonged exposure to a European type of diet and environment the findings in the African steadily approximate that of Europeans. In the same way it is likely that a European type of disease pattern will replace that which the African has at present. Little amoebiasis is seen in Durban Europeans, and it is certainly not common in Africans who have been able to adopt the European mode of life. On the other hand, it is in precisely this group of Africans that we find common European diseases appearing such as peptic ulceration and appendicitis.

At the commencement of this thesis it was stated that the introduction of medical science to undeveloped, backward regions has shown that many inhabitants of these countries have different "normal" values from those of Europeans. However, this is a changing, evolving process, and, to some extent at least, it is the impact of medical science which determines the "normal". As medical science progresses and indicates beneficial dietary and other measures for the prevention of disease it is conceivable that our own "normals" will change. It appears that in

Durban Africans we are dealing with a changing pattern of both "normal" biochemical values and diseases. Within the foreseeable future it is possible that they will become indistinguishable from those of Europeans. By the time this occurs, although both racial groups may have similar findings, the pattern may be different from that which is accepted by us as "normal" today.

APPENDIX I

Description of Methods

In order to increase the practical value of this investigation identical methods to those in use at the hospital routine clinical laboratory were selected.

Venous blood was collected between 9 and 10 a.m., and examined on the same day.

Haematological Investigations

The white cell count was estimated by the standard procedure in use in all clinical laboratories. The erythrocyte sedimentation rate was estimated by the Wintrobe method, and the packed cell volume subsequently obtained. Haemoglobin was read as oxyhaemoglobin in a colorimeter. The mean corpuscular haemoglobin concentration was calculated by dividing the haemoglobin in G./100 ml. by the packed cell volume.

Liver Function Tests

The routine liver function tests in use at the hospital clinical laboratory were performed.

Serum bilirubin

This was estimated by the method of King and Wootton (1956), normal range 0.1 - 0.8 mgm./100 ml. serum.

0.5 ml. of diazo reagent is layered above 1 ml. of serum in a stoppered centrifuge tube. After standing for 10 minutes 0.5 ml. of saturated ammonium sulphate and 8 ml. of 85% ethyl alcohol are added. The mixture is stoppered and thoroughly mixed, and allowed to lie on its side for 30 minutes, and then filtered. The filtrate is compared with a methyl red standard in a colorimeter using a green filter.

Calculation:

Serum bilirubin in mgm./100 ml. serum=

$$\frac{\text{Reading of test}}{\text{Reading of standard}} \times 4$$

Serum Alkaline Phosphatase

This was estimated by the method of King and Wootton (1956), normal range 3 - 13 units.

2 ml. of alkaline buffer solution and 2 ml. of disodium phenyl phosphate substrate are placed in a conical centrifuge tube which is placed in a water-bath at 37°C. for 3 minutes. Without removal of the tube from the bath, 0.2 ml. of serum is added and mixed. Exactly 15 minutes later 1.8 ml. of dilute Folin-Ciocalteu phenol reagent is added and the mixture centrifuged.

A control is made by placing 2 ml. of buffer and 2 ml. of substrate in another conical centrifuge tube, and then adding 1.8 ml. of dilute Folin-Ciocalteu reagent followed by 0.2 ml. of serum, and finally centrifuging the mixture.

4 ml. of the supernatant from the test and control solutions are pipetted into test tubes, and 2 ml. of 15%

sodium carbonate added. The mixtures are shaken and placed in the water-bath for 10 minutes to bring up the colour.

A standard is made up of 4 ml. of standard phenol solution and reagent, to which 2 ml. of 15% sodium carbonate is added. A blank of 3.2 ml. of water and 0.8 ml. of dilute Folin-Ciocalteu, treated with 2 ml. of sodium carbonate is also made up.

The solutions are read in a colorimeter using a red filter.

Calculation:

Serum alkaline phosphatase in King-Armstrong units per 100 ml. =

$$\frac{\text{Reading of (test - control)}}{\text{Reading of (standard - blank)}} \times 30$$

Cephalin Cholesterol Flocculation Test

The method of Hanger(1939) was used, normal range 0 - ++ after 48 hours.

0.2 ml. of serum is added to 4 ml. of normal saline in a test tube, followed by 1 ml. of cephalin cholesterol antigen. The mixture is left at room temperature in the dark, and the amount of flocculation noted after 24 and 48 hours. A control of 4 ml. of normal saline and 1 ml. of cephalin cholesterol antigen is also made up.

Zinc Sulphate Turbidity Test

The method of Kunkel(1947) was used, normal range, using Kingsbury sulphosalicylic acid standards, 0 - 4 units(Maclagan,1951).

0.05 ml. of serum is added to 3 ml. of a buffered zinc sulphate reagent, mixed, and allowed to stand for 30 minutes. The turbidity is read visually against sulphosalicylic acid standards used for C.S.F. protein estimations. When the turbidity exceeds that of the highest standard the mixture is diluted with 3 ml. (or multiples of 3 ml.) of buffered zinc sulphate reagent until the turbidity is such that it can be read on the scale.

Calculation:

Zinc sulphate turbidity in units =

$$\frac{\text{Sulphosalicylic acid standard}}{10} \times \text{Dilution(if used)}$$

Note Many laboratories use barium sulphate standards as described by Kunkel(1947). The normal range given with these standards is 0 - 8 units. As a check from time to time the sera were also read colorimetrically against barium sulphate standards, and it was found that the latter gave results in units almost exactly double that of the visual technique using sulphosalicylic acid standards.

The Serum Proteins

For the chemical estimation of the total protein, serum albumin and globulin 27.2% sodium sulphate(Pregl, 1937) and Weichselbaum's(1946) reagent were used.

0.4 ml. of serum is placed in a test tube and 9.6 ml. of 27.2% sodium sulphate added. The mixture is well shaken and 3 ml. is transferred to a fresh tube for total protein estimation. Both tubes are then incubated for at least 1 hour.

Total Protein:

3 ml. of Weichselbaum's reagent is added to the tube containing 3 ml. of serum-sulphate mixture. A blank is made up of 3 ml. of 27.2% sodium sulphate and 3 ml. of Weichselbaum's reagent. A standard is made up of 3 ml. of standard protein mixture (0.2 ml. of standard serum with 4.8 ml. of standard protein) and 3 ml. of Weichselbaum's reagent. The test, standard and blank are then allowed to stand for at least 30 minutes at room temperature.

Serum Albumin:

The serum-sulphate mixture is filtered in an incubator through Whatman's No. 44 filter paper. 3 ml. of Weichselbaum's reagent is then added to 3 ml. of the clear filtrate, and the mixture allowed to stand at room temperature for at least 30 minutes. The tests, standard and blank are then read in a colorimeter using a blue filter.

Calculation:

For both total protein and serum albumin in mgm./100ml.

$$= \frac{\text{Test} - \text{Blank}}{\text{Standard} - \text{Blank}} \times 6.9$$

Serum globulin in mgm./100ml. = Total protein - serum albumin.

Paper Electrophoresis

Paper electrophoresis was performed in a horizontal bath, using barbitone buffer, pH 8.6, and platinum electrodes. 0.025 ml. of serum is applied to 3 cm. wide strips of Whatman No. 1 filter paper. After running the strips in a current of 2 milli-amps. per strip for 18 hours, and drying and staining for 5 minutes with 1% bromophenol blue in a 95% ethanol solution saturated with mercuric chloride, the strips are washed successively with distilled water, dioxan and ether. They are finally dried in a hot air oven at 105°C. for 15 minutes. The stained strips are then immersed in liquid paraffin at 100°C for 10 minutes, and scanned with a commercial photo-electric densitometer. The individual curves in the resulting graphs are then cut out and carefully weighed, and the percentage concentrations of the various protein fractions computed. The resulting percentages are not multiplied by any coefficient. The total protein obtained by the chemical method is used to convert the percentage values of the protein fractions to absolute values in G./100 ml. All sera are examined in duplicate,

and a difference of more than 2% between the same protein fractions is not accepted. Standardisation is made as rigorous as possible. All estimations were made personally.

Note on Paper Electrophoresis

Within the last decade a formidable literature on paper electrophoresis has arisen, and there has been much discussion over the relative merits and accuracy of numerous techniques. Much of the disagreement in the literature has been due to the uncritical comparison of results obtained by different techniques. Paper electrophoresis is comparatively crude, but, although there is a relatively large degree of technical error, it is a satisfactory means of following the gross changes in the serum proteins found in the African, and valid comparisons can be made provided a single, consistent method is used.

It should be borne in mind that quantitative comparison of results obtained by paper electrophoresis

with those obtained by chemical methods cannot be made as entirely different physical properties of the protein fractions are measured. However, although it is not to be expected that identical results will be obtained, my findings indicate that there is a fair degree of correlation between the 2 methods, paper electrophoresis showing consistently higher albumin levels.

Provided the same technique is used under rigorously standardised conditions, and the investigations are performed by the same observer it is possible to obtain satisfactory results by paper electrophoresis, although the method is not suitable for routine use.

In addition to performing all the tests in duplicate, and repeating all those which showed a difference of more than 2% between the same protein fractions, every 25th specimen was estimated 6 times to check reproducibility.

The following are the results in per cent of 4 such checks:

1.							Mean	S.D.
Alb.	49.5	51.2	50.3	51.1	50.5	51.1	50.6	.66
Glob.	50.5	48.8	49.7	48.9	49.5	48.9	49.4	.66
$Alpha_1$	4.4	4.3	4.4	4.7	4.4	4.5	4.5	.15
$Alpha_2$	11.0	11.0	10.4	10.6	10.6	10.0	10.6	.38
Beta	12.6	12.8	12.6	12.4	13.3	12.5	12.7	.32
Gamma	22.5	20.7	22.3	21.2	21.2	21.9	21.6	.71
A/G Ratio	.98:1	1.05:1	1.01:1	1.05:1	1.02:1	1.05:1	1.02:1	

2.							Mean	S.D.
Alb.	46.8	47.1	47.6	47.5	47.2	46.6	47.1	.39
Glob.	53.2	52.9	52.4	52.5	52.8	53.4	52.9	.39
$Alpha_1$	5.3	4.7	4.2	4.3	4.6	4.5	4.6	.39
$Alpha_2$	9.6	9.3	8.5	9.8	9.2	8.0	9.1	.69
Beta	15.9	15.7	15.2	15.2	14.5	15.9	15.4	.54
Gamma	22.4	23.2	24.5	23.2	24.5	25.0	23.8	1.00
A/G Ratio	.88:1	.89:1	.91:1	.91:1	.89:1	.87:1	.89:1	

3.							Mean	S.D.
Alb.	55.6	56.5	55.6	56.4	58.3	58.8	56.9	1.49
Glob.	44.4	43.5	44.6	43.6	41.7	41.2	43.1	1.49
$Alpha_1$	4.6	4.5	4.7	4.2	3.9	3.8	4.3	.38
$Alpha_2$	9.2	8.5	9.5	9.1	9.0	8.7	9.0	.36
Beta	11.7	11.3	13.0	12.1	12.4	11.8	12.0	.72
Gamma	18.9	19.2	17.2	18.2	16.4	16.9	17.8	1.12
A/G Ratio	1.25:1	1.30:1	1.25:1	1.29:1	1.41:1	1.43:1	1.32:1	

4.							Mean	S.D.
Alb.	56.0	56.4	53.7	55.6	57.3	56.8	56.0	1.26
Glob.	44.0	43.6	46.3	44.4	42.7	43.2	44.0	1.26
$Alpha_1$	4.8	4.6	4.3	4.0	4.1	4.0	4.3	.33
$Alpha_2$	9.7	9.9	10.1	8.6	8.9	8.5	9.3	.70
Beta	12.1	13.1	14.4	13.9	13.0	12.5	13.1	.86
Gamma	17.4	16.0	17.5	17.9	16.7	18.2	17.3	.81
A/G Ratio	1.28:1	1.30:1	1.16:1	1.25:1	1.35:1	1.32:1	1.27:1	

APPENDIX II

DETAILED RESULTS

Haematological Investigations

Group 1 (Europeans)

	Hb (G./100ml.)	Wbc (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
E1	16.2	8,000	7	51.5	35.5
E2	14.2	10,000	10	42.0	34.0
E3	17.6	7,700	3	50.5	35.0
E4	17.0	8,300	4	48.0	35.5
E5	16.2	7,600	4	47.0	34.5
E6	17.8	13,300	2	51.5	34.5
E7	19.0	5,700	3	52.0	36.5
E8	17.0	9,800	1	49.0	34.5
E9	19.6	11,400	2	57.0	34.5
E10	15.9	6,100	4	48.0	33.0
E11	17.2	6,200	1	47.0	36.5
E12	15.9	5,800	5	46.0	34.5
E13	14.9	9,700	3	45.5	34.0
E14	17.3	7,600	4	47.0	37.0
E15	17.3	8,600	2	50.0	34.5
E16	18.4	10,000	3	52.5	35.0
E17	18.7	13,000	12	50.5	37.0
E18	16.4	6,700	1	46.5	35.5
E19	16.0	8,000	2	45.5	35.0
E20	15.7	11,100	7	45.0	35.0
E21	16.4	11,800	8	47.0	35.0
E22	16.0	8,000	5	47.0	34.0
E23	16.6	6,000	2	48.0	34.5
E24	16.6	10,100	6	47.5	35.0
E25	18.6	10,200	3	51.0	36.5
Mean	17.0	8,700	4	48.5	35.1
Range	14.2	5,700-	1-	42.0-	33.0-
	19.6	13,300	12	57.0	37.0
S.D.	1.21	----	2.86	3.10	1.00

Group 2 (African Male Nurses)

	Hb (G./100ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
N1	17.3	3,200	3	48.0	36.0
N2	16.6	8,200	2	50.0	33.0
N3	17.8	7,600	10	51.5	34.5
N4	16.3	8,300	3	51.0	32.0
N5	14.6	7,700	13	42.0	35.0
N6	16.3	4,700	3	48.0	34.0
N7	16.9	5,600	10	49.5	34.0
N8	16.3	6,100	24	49.0	33.5
N9	15.9	10,200	8	47.5	33.5
N10	16.0	5,800	14	44.5	36.0
N11	14.8	5,400	14	45.5	32.5
N12	17.2	5,400	3	50.0	33.0
N13	16.1	7,000	2	48.0	33.5
N14	14.8	5,600	7	45.0	33.0
N15	17.3	5,700	11	49.0	35.5
N16	15.4	4,800	25	45.0	34.0
N17	16.1	6,200	10	43.5	37.0
N18	16.8	8,000	9	48.0	35.0
N19	14.8	4,400	8	42.5	35.0
N20	16.6	6,000	4	50.0	33.0
N21	16.2	8,100	16	45.5	35.5
N22	16.6	7,200	19	46.5	35.5
N23	18.2	5,800	8	51.0	35.5
N24	15.4	6,000	24	45.0	34.0
N25	15.1	4,500	6	45.0	33.5
N26	17.0	5,700	10	51.0	33.5
N27	15.6	4,500	19	47.0	33.0
N28	16.6	8,100	11	48.0	34.5
N29	16.9	7,800	9	49.0	34.5
N30	17.0	5,700	10	51.0	33.5
N31	16.2	6,700	5	46.0	35.0
N32	15.0	6,800	10	42.0	35.5
N33	15.3	4,700	6	46.5	33.0
N34	16.9	6,000	17	49.0	34.5
N35	15.8	8,500	15	47.0	33.0
N36	14.5	7,800	16	42.0	34.5
N37	17.3	4,800	5	49.5	35.0
N38	15.2	4,400	24	46.5	32.5

	Hb (G./100ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
N38	15.7	6,000	14	46.0	34.0
N40	15.9	5,700	6	48.0	33.0
N41	16.1	8,000	4	47.0	34.0
N42	15.1	3,900	1	55.0	33.0
N43	16.6	5,900	14	48.5	34.0
N44	15.8	6,200	18	45.0	34.0
N45	15.2	4,700	1	53.5	34.0
N46	14.2	5,000	3	45.0	31.5
N47	16.1	6,500	15	47.0	34.0
N48	16.0	4,400	4	47.5	33.5
N49	15.9	9,900	12	46.0	34.5
N50	15.6	4,000	23	45.0	34.5
Mean	16.2	6,300	11	47.4	34.1
Range	14.2- 18.2	3,200- 10,200	1- 23	42.0- 55.0	31.5- 37.0
S.D.	.97	----	6.64	2.85	1.19

Group 3 (Cato Manor Africans)

A1	14.7	6,900	10	42.0	35.0
A2	15.2	7,300	22	44.0	34.5
A3	17.0	14,600	6	49.0	34.5
A4	16.0	13,000	26	47.0	34.0
A5	16.0	7,500	8	48.5	33.0
A6	15.6	7,000	25	43.5	36.0
A7	13.6	8,600	27	40.5	33.5
A8	15.8	10,400	10	47.0	33.5
A9	18.2	6,200	8	52.5	34.5
A10	16.2	7,600	15	48.0	34.0
A11	16.9	8,600	8	49.0	34.5
A12	15.6	15,400	34	45.5	34.5
A13	16.8	6,100	10	49.5	34.0
A14	16.2	14,000	17	48.5	33.5
A15	15.9	8,900	15	46.0	34.5

	Hb (G./100ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
A16	15.6	6,900	7	46.0	34.0
A17	15.2	4,800	23	45.0	34.0
A18	15.6	8,400	7	46.0	34.0
A19	16.4	5,300	18	48.0	34.0
A20	14.9	7,600	29	43.0	34.5
A21	16.9	7,600	13	51.5	33.0
A22	17.6	5,000	16	51.0	34.5
A23	17.3	11,900	17	49.0	35.5
A24	14.7	6,500	38	45.0	33.0
A25	15.8	9,400	14	45.5	35.0
A26	15.9	7,500	34	46.0	34.5
A27	15.2	5,400	30	46.0	33.0
A28	16.2	8,100	15	49.0	33.0
A29	15.6	6,000	10	47.0	33.0
A30	14.9	12,000	40	44.5	33.5
A31	15.7	5,700	14	46.0	34.0
A32	15.9	5,300	2	50.5	31.5
A33	16.4	7,700	19	48.5	34.0
A34	16.6	7,800	12	50.0	33.0
A35	15.9	10,000	24	45.5	35.0
A36	15.2	7,600	15	49.0	31.0
A37	15.2	6,300	31	45.5	33.5
A38	16.2	7,800	17	50.0	32.5
A39	14.0	6,100	35	43.0	32.5
A40	16.9	6,400	9	50.0	34.0
A41	16.0	7,900	8	48.0	33.5
A42	13.9	6,700	13	43.0	32.5
A43	16.3	4,400	10	48.0	34.0
A44	16.6	4,200	22	47.0	35.5
A45	19.0	8,200	5	55.0	34.5
A46	17.1	8,300	24	50.5	34.0
A47	16.9	6,200	7	49.0	34.5
A48	16.0	11,000	27	46.0	35.0
A49	15.2	6,400	23	44.0	34.5
A50	18.2	6,900	11	52.5	35.0
Mean	16.0	7,900	18	47.3	33.9
Range	13.6-19.0	4,200-15,400	2-40	40.5-55.0	31.0-36.0
S.D.	1.06	---	9.58	2.95	.98

Liver Function Tests

Group 1 (Europeans)

	Ceph. Chol. 24 hrs.	Ceph. Chol. 48 hrs.	Zn.Sulph. Turb.	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100ml.)
E1	+	+	3	5	.5
E2	+	+	2	5	.5
E3	++	+++	5	6	.5
E4	0	0	4	7	.5
E5	0	0	3	4	.5
E6	0	+	2	7	.6
E7	0	+	7	7.5	.4
E8	+	++	2	6	.6
E9	0	++	1	7	.7
E10	0	+	3	4	.5
E11	++	++	1	6.5	.6
E12	+	+	2	5	.6
E13	0	+	1	8	.4
E14	0	0	1	5	.5
E15	+	+	3	7.5	.5
E16	+	+	3	9.5	.5
E17	+	+	2	8	.4
E18	+	+	3	5.5	.6
E19	+	+	3	4	.4
E20	+	+	1	6	.5
E21	0	0	4	9	.4
E22	++	++	6	7	.6
E23	+	+	6	5.5	.4
E24	++	++	2	5	.4
E25	+	++	3	2.5	.5
Mean	—	—	2.9	6.1	.50
Range	—	—	1-7	2.5-9.5	.4-.7
S.D.	—	—	1.66	1.68	.08

Group 2 (African Male Nurses)

	Ceph. 24 hrs.	Chol. 48 hrs.	Zn.Sulph. Turb.	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100 ml.)
N1	0	0	4	10	.8
N2	+	+	7	5	.4
N3	++	++	5	11	.5
N4	0	0	3	7	.4
N5	0	0	6	6	.4
N6	0	0	6	7	.6
N7	0	0	9	6	.4
N8	+++	++++	12	6.5	.5
N9	0	+	5	9	.5
N10	0	+	8	7	.5
N11	++	+++	8	7	.4
N12	+++	++++	2	5.5	.4
N13	0	+	6	11	.4
N14	0	+	5	5.5	.5
N15	++++	++++	3	5	.5
N16	+++	++++	8	8	.4
N17	0	+	5	9	.4
N18	+++	++++	5	6.5	.4
N19	0	+	5	7.3	.4
N20	+	+	4	5.5	.5
N21	0	+	5	6.7	.5
N22	0	+	4	4	.4
N23	+	+	4	4.3	.5
N24	+	+	5	7	.5
N25	++	++	7	5	.4
N26	++	++	5	8	.6
N27	+++	+++	5	5.5	.5
N28	++	++	4	8	.5
N29	+	+	3	5	.5
N30	0	+	15	8	.6

	Ceph. 24 hrs.	Chol. 48 hrs.	Zn.Sulph. Turb.	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100 ml.)
N31	0	0	5	8	.5
N32	0	0	9	9	.5
N33	0	+	9	5	.4
N34	++	+++	9	8	.4
N35	++	+++	4	7	.4
N36	0	+	3	9	.5
N37	0	+	7	9	.4
N38	0	+	4	8	.5
N39	+	+	4	5.5	.5
N40	+++	+++	9	5	.4
N41	++	++	6	6.7	.4
N42	0	0	3	8	.4
N43	0	++	3	5	.4
N44	0	0	5	9	.5
N45	+	+	3	3.5	.6
N46	0	0	8	6.5	.4
N47	+	+	6	4	.5
N48	+	+	2	9.5	.4
N49	0	++	4	9	.3
N50	+++	+++	6	5	.4
Mean	---	---	5.6	6.9	.46
Range	---	---	2-15	3.5-11	.3-.8
S.D.	---	---	2.55	1.85	.09

Group 3 (Cato Manor Africans)

A1	+	+	3	9	.4
A2	+	++	7	7	.4
A3	+++	++++	12	7.5	.4
A4	+++	++++	7	7	.4
A5	+	++	6	11	.4
A6	+	++	10	6.5	.4
A7	+++	++++	6	9	.4
A8	+++	++++	9	4.5	.4
A9	+++	++++	6	10	.5
A10	+++	++++	9	6.5	.4

	Ceph. 24 hrs.	Chol. 48 hrs.	Zn.Sulph. Turb.	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100 ml.)
A11	0	0	5	11	.4
A12	++	+++	7	8	.4
A13	++++	++++	6	9	.4
A14	0	0	5	10	.4
A15	+++	++++	6	16	.4
A16	0	0	3	9	.4
A17	+++	++++	5	9	.4
A18	+++	++++	6	9.5	.5
A19	+++	+++	7	8.5	.4
A20	+++	++++	4	9.5	.4
A21	+++	++++	6	9	.5
A22	0	0	5	12	.4
A23	+++	++++	5	12	.4
A24	+++	++++	7	9	.4
A25	0	0	5	6	.4
A26	+++	++++	7	16	.4
A27	++	+++	7	9	.5
A28	++	++	6	7	.6
A29	+	++	7	7	.7
A30	+	+	3	6.5	.4
A31	++	++	4	6	.4
A32	+	+	6	9	.4
A33	++	++	4	10.5	.4
A34	++	++	7	9.5	.4
A35	+	++	5	10.5	.5
A36	+	+	5	12	.4
A37	+++	++++	7	7	.6
A38	+++	++++	8	8	.5
A39	+	+	7	8.5	.4
A40	+	+	6	9.5	.4
A41	+	+	2	7.5	.4
A42	++	++	6	9.5	.5
A43	++	++	7	10	.4
A44	+++	++++	16	9.5	.5
A45	+	++	6	24	.5
A46	++	++	5	11.5	.4
A47	+	+	3	9.5	.5
A48	+	++	5	9	.4

	Ceph. 24 hrs.	Chol. 48 hrs.	Zn.Sulph. Turb.	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100 ml.)
A49	++	+++	10	7	.4
A50	++	++	6	8.5	.4
Mean	—	—	6.3	9.3	.43
Range	—	—	2-16	4.5-24	.4-.7
S.D.	—	—	2.36	3.05	.07

Serum Proteins

Chemical Fractionation

Group 1 (Europeans)

	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
E1	8.10	5.10	3.00	1.70:1
E2	7.65	4.80	2.85	1.68:1
E3	8.34	5.04	3.30	1.53:1
E4	7.66	3.78	3.88	.97:1
E5	7.56	4.10	3.46	1.18:1
E6	7.11	3.45	3.66	.94:1
E7	7.36	4.21	3.15	1.34:1
E8	7.70	4.52	3.18	1.42:1
E9	7.33	3.72	3.61	1.03:1
E10	7.10	3.54	3.56	.99:1
E11	6.83	3.67	3.16	1.16:1
E12	6.40	3.20	3.20	1.00:1
E13	6.30	3.20	3.10	1.03:1
E14	6.50	3.40	3.10	1.10:1
E15	7.10	3.77	3.33	1.13:1
E16	6.42	3.59	2.83	1.27:1
E17	7.40	4.02	3.38	1.19:1
E18	7.06	4.02	3.04	1.32:1
E19	6.96	3.81	3.15	1.21:1
E20	6.96	4.13	2.83	1.46:1

	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
E21	6.74	3.48	3.26	1.07:1
E22	8.05	4.24	3.81	1.11:1
E23	6.74	3.81	2.93	1.30:1
E24	7.06	3.81	3.25	1.17:1
E25	7.50	4.02	3.48	1.16:1
Mean	7.20	3.94	3.26	1.21:1
Range	6.30- 8.34	3.20- 5.10	2.83- 3.88	.95:1- 1.70:1
S.D.	.54	.51	.29	—

Group 2 (African Male Nurses)

N1	7.50	4.45	3.05	1.46:1
N2	7.80	4.08	3.72	1.10:1
N3	8.23	4.42	3.81	1.16:1
N4	8.06	5.16	2.90	1.78:1
N5	7.96	4.50	3.46	1.30:1
N6	7.56	3.92	3.64	1.08:1
N7	7.86	3.45	4.41	.78:1
N8	8.82	4.62	4.20	1.10:1
N9	6.68	3.07	3.61	.85:1
N10	8.24	4.80	3.44	1.40:1
N11	7.70	4.00	3.70	1.08:1
N12	6.60	3.10	3.50	.89:1
N13	7.31	3.83	3.48	1.10:1
N14	7.65	4.41	3.24	1.36:1
N15	6.70	3.10	3.60	.86:1
N16	8.05	4.20	3.85	1.09:1
N17	7.85	4.69	3.16	1.48:1
N18	6.79	3.10	3.69	.84:1
N19	7.19	3.94	3.25	1.21:1
N20	7.01	3.36	3.65	.92:1
N21	7.97	4.38	3.59	1.22:1
N22	7.83	4.30	3.53	1.22:1
N23	7.62	4.12	3.50	1.18:1
N24	6.93	3.05	3.88	.79:1
N25	7.45	4.38	3.07	1.43:1

	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
N26	7.69	4.13	3.56	1.16:1
N27	6.92	3.10	3.82	.81:1
N28	7.56	3.88	3.68	1.05:1
N29	7.56	4.50	3.06	1.47:1
N30	7.35	3.60	3.75	.96:1
N31	7.25	3.84	3.41	1.13:1
N32	6.74	3.31	3.43	.97:1
N33	7.01	3.45	3.56	.97:1
N34	7.22	3.24	3.98	.81:1
N35	8.05	4.62	3.43	1.35:1
N36	7.82	4.59	3.23	1.42:1
N37	7.15	3.39	3.76	.90:1
N38	7.92	4.27	3.65	1.17:1
N39	8.05	4.45	3.60	1.24:1
N40	7.90	4.00	3.90	1.03:1
N41	7.20	3.88	3.32	1.17:1
N42	7.40	3.80	3.60	1.06:1
N43	7.35	3.42	3.93	.87:1
N44	7.85	3.99	3.86	1.03:1
N45	6.30	3.15	3.15	1.00:1
N46	7.20	3.20	4.00	.80:1
N47	7.83	4.62	3.21	1.44:1
N48	7.01	3.60	3.41	1.06:1
N49	7.25	3.20	4.05	.79:1
N50	7.70	4.07	3.63	1.12:1
Mean	7.49	3.91	3.58	1.09:1
Range	6.30- 8.82	3.05- 5.16	2.90- 4.41	.78:1- 1.78:1
S.D.	.50	.56	.31	—

Group 3 (Cato Manor African)

A1	6.71	3.05	3.66	.83:1
A2	6.71	2.73	3.98	.69:1
A3	6.95	2.78	4.17	.67:1
A4	6.85	2.67	4.18	.64:1
A5	6.62	3.00	3.62	.83:1

	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
A6	7.06	2.78	4.28	.65:1
A7	7.49	3.53	3.96	.89:1
A8	7.37	3.21	4.16	.77:1
A9	7.06	3.00	4.06	.74:1
A10	7.06	3.21	3.85	.83:1
A11	7.40	3.38	4.02	.84:1
A12	7.70	2.93	4.77	.61:1
A13	7.84	3.26	4.58	.71:1
A14	7.60	3.59	4.01	.90:1
A15	7.05	3.20	3.85	.83:1
A16	6.40	3.20	3.20	1.00:1
A17	7.05	3.42	3.63	.94:1
A18	6.73	3.42	3.31	1.03:1
A19	7.49	3.42	4.07	.84:1
A20	6.95	3.20	3.75	.85:1
A21	7.16	3.53	3.63	.97:1
A22	6.95	3.20	3.75	.85:1
A23	6.95	3.32	3.63	.91:1
A24	7.05	2.99	4.06	.74:1
A25	6.84	3.32	3.52	.94:1
A26	7.69	3.42	4.27	.80:1
A27	7.07	3.15	3.92	.80:1
A28	6.85	3.05	3.80	.80:1
A29	7.07	3.37	3.70	.91:1
A30	6.74	3.15	3.59	.88:1
A31	6.20	3.15	3.05	1.03:1
A32	6.96	3.76	3.20	1.17:1
A33	7.40	3.37	4.03	.84:1
A34	6.85	3.37	3.48	.97:1
A35	7.95	3.91	4.04	.97:1
A36	6.74	2.94	3.80	.77:1
A37	7.72	3.37	4.35	.77:1
A38	7.20	3.10	4.10	.76:1
A39	6.40	2.83	3.57	.79:1
A40	6.95	3.05	3.90	.78:1
A41	7.40	3.81	3.59	1.06:1
A42	7.07	3.26	3.81	.86:1
A43	7.56	3.59	3.97	.90:1
A44	8.26	3.48	4.78	.73:1
A45	7.56	3.37	4.19	.80:1

	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
A46	7.29	3.48	3.81	.91:1
A47	7.29	3.16	4.13	.77:1
A48	7.61	3.48	4.13	.84:1
A49	6.95	2.61	4.34	.60:1
A50	7.19	2.83	4.36	.65:1
Mean	7.14	3.23	3.91	.83:1
Range	6.20-8.26	2.61-3.91	3.95-4.78	.60:1-1.17:1
S.D.	.42	.29	.37	—

Paper Electrophoresis

Group 1 (Europeans)

	Ser. Alb.	Ser. Glob.	A/G Ratio	$Alpha_1$	$Alpha_2$	Beta	Gamma
E1	5.24	2.86	1.83:1	.24	.83	.87	.92
E2	4.86	2.79	1.74:1	.16	.68	.98	.97
E3	5.29	3.05	1.73:1	.40	.55	.65	1.45
E4	4.29	3.37	1.27:1	.33	.67	.91	1.46
E5	4.50	3.06	1.47:1	.36	.60	.83	1.27
E6	4.01	3.10	1.29:1	.33	.61	.85	1.31
E7	4.23	3.13	1.35:1	.32	.54	.85	1.42
E8	4.58	3.12	1.47:1	.41	.69	.90	1.12
E9	4.18	3.15	1.33:1	.35	.76	.84	1.20
E10	3.98	3.12	1.28:1	.37	.64	.92	1.19
E11	3.89	2.94	1.32:1	.25	.68	.84	1.17
E12	3.59	2.81	1.28:1	.20	.56	.81	1.24
E13	3.58	2.72	1.32:1	.21	.66	.79	1.06
E14	3.70	2.80	1.32:1	.26	.56	.70	1.28
E15	3.85	3.25	1.18:1	.29	.67	.86	1.43
E16	3.65	2.77	1.32:1	.28	.58	.77	1.14
E17	4.14	3.26	1.27:1	.32	.69	.97	1.28
E18	3.97	3.09	1.28:1	.28	.57	.88	1.36
E19	3.99	2.97	1.34:1	.28	.69	.83	1.17
E20	4.19	2.77	1.51:1	.31	.74	.81	.91

	Ser. Alb.	Ser. Glob.	A/G Ratio	$Alpha_1$	$Alpha_2$	Beta	Gamma
E21	3.64	3.10	1.17:1	.28	.61	.84	1.37
E22	4.64	3.41	1.36:1	.31	.68	1.07	1.35
E23	3.96	2.78	1.42:1	.27	.54	.73	1.24
E24	4.25	2.81	1.51:1	.26	.56	.75	1.24
E25	4.45	3.05	1.46:1	.31	.65	.90	1.19
Mean	4.19	3.01	1.39:1	.30	.64	.85	1.22
Range	3.58- 5.29	2.72- 3.41	1.17:1- 1.83:1	.16- .41	.54- .83	.65- 1.07	.91- 1.46
S.D.	.47	.25	---	.06	.07	.09	.15

Group 2 (African Male Nurses)

N1	4.73	2.77	1.71:1	.22	.56	.78	1.21
N2	4.44	3.36	1.32:1	.28	.59	.80	1.69
N3	4.57	3.66	1.25:1	.40	.81	.90	1.55
N4	5.27	2.79	1.89:1	.21	.52	.90	1.16
N5	4.71	3.25	1.45:1	.26	.56	.77	1.66
N6	4.57	2.99	1.53:1	.29	.56	.80	1.34
N7	4.02	3.84	1.05:1	.25	.76	.79	2.04
N8	4.99	3.83	1.30:1	.26	.47	.76	2.34
N9	3.70	2.98	1.24:1	.31	.49	.81	1.37
N10	5.00	3.24	1.54:1	.27	.47	.70	1.80
N11	4.40	3.30	1.33:1	.26	.51	.79	1.74
N12	3.63	2.97	1.22:1	.23	.58	.69	1.47
N13	4.20	3.11	1.35:1	.31	.48	.75	1.57
N14	4.64	3.01	1.54:1	.26	.64	.59	1.52
N15	3.65	3.05	1.20:1	.21	.44	.78	1.62
N16	4.51	3.54	1.27:1	.27	.69	.89	1.69
N17	4.86	2.99	1.63:1	.28	.47	.74	1.50
N18	3.54	3.25	1.09:1	.21	.64	.88	1.52
N19	4.26	2.93	1.45:1	.24	.45	.69	1.55
N20	3.56	3.45	1.03:1	.30	.63	.91	1.61
N21	4.66	3.31	1.41:1	.27	.54	.96	1.54
N22	4.89	2.94	1.66:1	.25	.49	.56	1.64
N23	4.66	2.96	1.57:1	.30	.66	.73	1.27
N24	3.64	3.29	1.11:1	.34	.69	.85	1.41
N25	4.45	3.00	1.48:1	.34	.57	.78	1.31

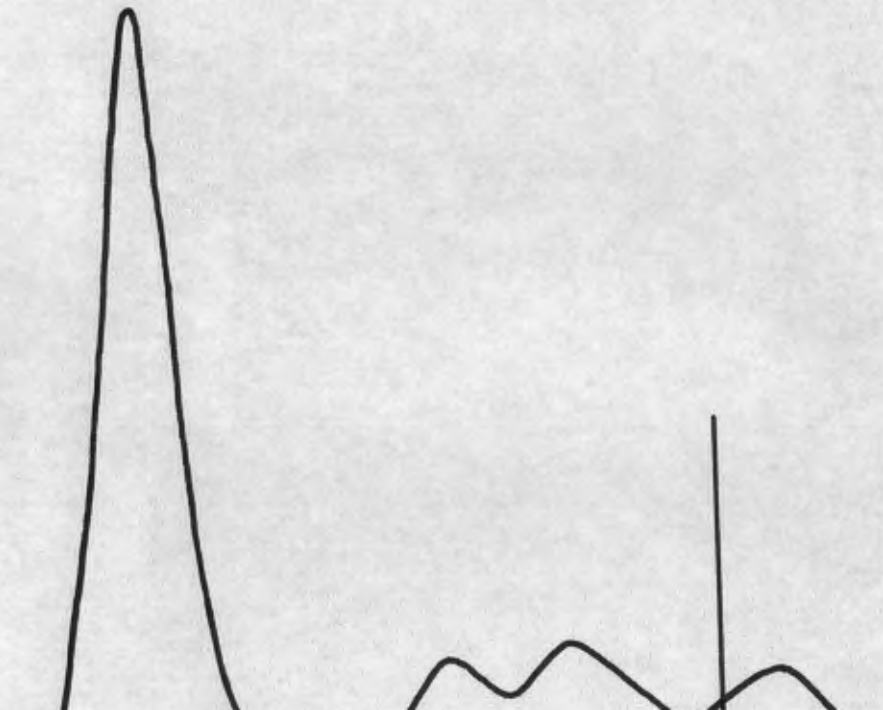
	Ser. Alb.	Ser. Glob.	A/G Ratio	$Alpha_1$	$Alpha_2$	Beta	Gamma
N26	4.39	3.30	1.33:1	.32	.57	.95	1.46
N27	3.31	3.61	.92:1	.24	.80	.90	1.67
N28	4.34	3.22	1.35:1	.37	.66	.91	1.28
N29	4.61	2.95	1.56:1	.26	.73	.83	1.13
N30	3.79	3.56	1.06:1	.21	.56	.84	1.95
N31	4.15	3.10	1.34:1	.25	.46	.91	1.48
N32	3.69	3.05	1.21:1	.22	.47	.66	1.70
N33	3.88	3.13	1.24:1	.24	.62	.72	1.55
N34	3.69	3.53	1.05:1	.32	.87	.83	1.52
N35	4.81	3.24	1.48:1	.25	.52	.69	1.78
N36	4.76	3.06	1.56:1	.29	.61	.75	1.41
N37	3.57	3.58	1.00:1	.24	.68	1.06	1.60
N38	4.68	3.24	1.44:1	.28	.70	.77	1.49
N39	4.68	3.37	1.39:1	.34	.60	.80	1.63
N40	4.60	3.30	1.39:1	.33	.58	.74	1.65
N41	4.38	2.82	1.55:1	.28	.45	.57	1.52
N42	4.15	3.25	1.28:1	.29	.62	.80	1.54
N43	3.92	3.34	1.17:1	.29	.66	.83	1.65
N44	4.36	3.49	1.25:1	.34	.71	.93	1.51
N45	3.55	2.75	1.29:1	.26	.55	.74	1.20
N46	3.75	3.45	1.09:1	.22	.53	.71	1.99
N47	4.73	3.10	1.53:1	.22	.48	.57	1.83
N48	3.86	3.15	1.23:1	.32	.60	.85	1.38
N49	3.76	3.49	1.08:1	.34	.67	.83	1.65
N50	4.29	3.41	1.26:1	.24	.55	.85	1.77
Mean	4.26	3.23	1.32:1	.28	.59	.79	1.57
Range	3.31-5.27	2.75-3.84	.92:1-1.89:1	.21-.40	.44-.87	.56-1.06	1.13-2.34
S.D.	.49	.26	---	.05	.10	.10	.23
Group 3 (Cato Manor Africans)							
A1	3.75	2.96	1.27:1	.34	.60	.89	1.13
A2	3.31	3.40	.97:1	.26	.65	.81	1.68
A3	3.29	3.66	.90:1	.34	.65	.85	1.82

	Ser. Alb.	Ser. Glob.	A/G Ratio	Alpha$_1$	Alpha$_2$	Beta	Gamma
A4	3.23	3.62	.89:1	.33	.70	.84	1.75
A5	3.25	3.37	.96:1	.33	.57	.93	1.54
A6	3.37	3.69	.91:1	.35	.64	.90	1.80
A7	3.92	3.57	1.10:1	.35	.81	.82	1.59
A8	3.52	3.85	.91:1	.38	.67	1.01	1.79
A9	3.32	3.74	.89:1	.38	.61	.98	1.77
A10	3.34	3.72	.90:1	.40	.68	.85	1.79
A11	3.54	3.86	.92:1	.41	.91	.97	1.57
A12	3.37	4.33	.78:1	.37	.96	1.13	1.87
A13	3.68	4.16	.88:1	.31	.92	.98	1.95
A14	3.82	3.78	1.01:1	.33	.85	.92	1.68
A15	3.58	3.47	1.03:1	.32	.65	.89	1.61
A16	3.28	3.12	1.05:1	.30	.64	.79	1.39
A17	3.33	3.72	.90:1	.37	.71	.88	1.76
A18	3.39	3.34	1.01:1	.35	.65	.83	1.51
A19	3.53	3.96	.89:1	.36	.73	.98	1.89
A20	3.35	3.00	.93:1	.38	.68	.88	1.66
A21	3.74	3.42	1.09:1	.32	.59	.98	1.53
A22	3.46	3.49	.99:1	.31	.85	.83	1.50
A23	3.41	3.54	.96:1	.36	.78	.91	1.49
A24	3.17	3.88	.82:1	.33	.78	.99	1.78
A25	3.43	3.41	1.01:1	.30	.70	.86	1.55
A26	3.84	3.85	1.00:1	.39	.79	.79	1.88
A27	3.56	3.51	1.01:1	.30	.76	.90	1.55
A28	3.22	3.63	.89:1	.34	.65	1.08	1.56
A29	3.78	3.29	1.15:1	.29	.58	.87	1.55
A30	3.40	3.34	1.02:1	.30	.79	.96	1.29
A31	3.26	2.94	1.11:1	.29	.63	.79	1.23
A32	3.70	3.26	1.13:1	.35	.59	.88	1.44
A33	3.57	3.83	.93:1	.35	.88	1.19	1.41
A34	3.44	3.41	1.01:1	.29	.72	.86	1.54
A35	4.32	3.63	1.19:1	.31	.82	.95	1.55
A36	3.35	3.39	.99:1	.32	.73	.86	1.48
A37	3.69	4.03	.92:1	.38	.86	1.08	1.71
A38	3.18	4.02	.79:1	.33	.82	.94	1.93
A39	3.10	3.30	.94:1	.33	.72	.66	1.59
A40	3.55	3.40	1.04:1	.29	.70	.81	1.60
A41	4.06	3.34	1.22:1	.34	.69	1.05	1.26
A42	3.58	3.49	1.03:1	.27	.70	.89	1.55
A43	3.86	3.70	1.04:1	.29	.70	1.10	1.61
A44	3.79	4.47	.85:1	.23	.83	1.05	2.36
A45	3.49	4.07	.86:1	.29	.88	1.26	1.64

	Ser. Alb.	Ser. Glob.	A/G Ratio	$Alpha_1$	$Alpha_2$	Beta	Gamma
A46	3.77	3.52	1.07:1	.28	.64	1.00	1.60
A47	3.62	3.67	.99:1	.33	.86	1.04	1.44
A48	3.94	3.37	1.07:1	.38	.73	.93	1.63
A49	3.04	3.91	.78:1	.32	.62	.98	1.99
A50	3.52	3.67	.96:1	.32	.70	.97	1.68
Mean	3.52	3.62	.97:1	.33	.73	.93	1.63
Range	3.04-4.32	2.94-4.47	.78:1-1.27:1	.23-.41	.57-.96	.66-1.26	1.13-2.36
S.D.	.26	.31	—	.04	.10	.11	.22

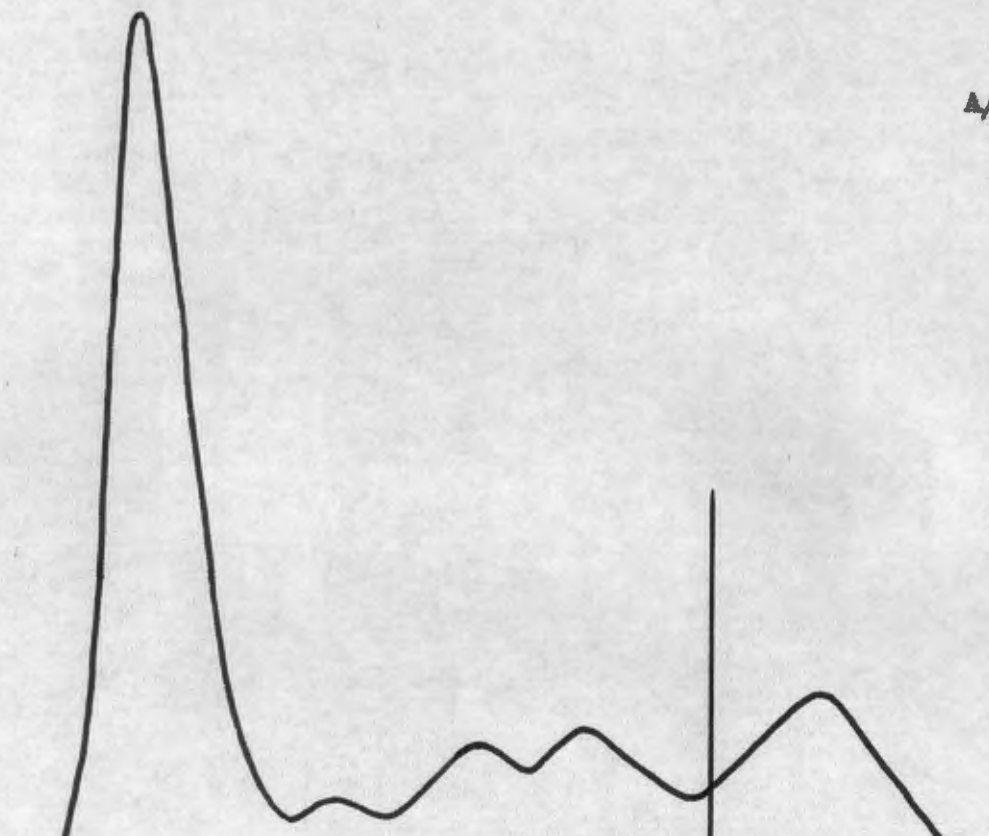
European Electrophoretic Pattern E9

Total Protein	7.33
Serum Albumin	4.18
Serum Globulin	3.15
$Alpha_1$	.35
$Alpha_2$	.76
Beta	.84
Gamma	1.20
A/G Ratio	1.33:1

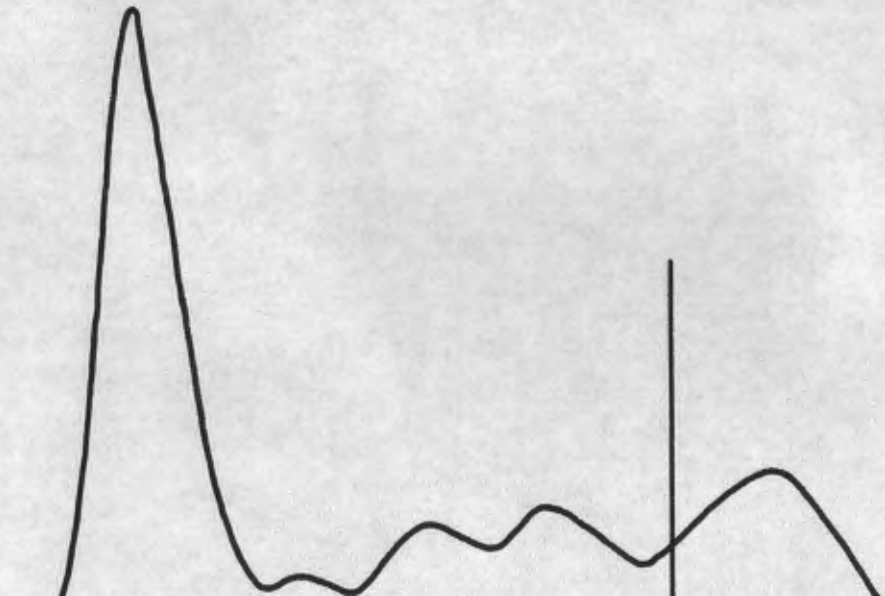


African Male Nursing Staff Electrophoretic Pattern N44

Total Protein	7.85
Serum Albumin	4.36
Serum Globulin	3.49
$Alpha_1$	.34
$Alpha_2$	.71
Beta	.93
Gamma	1.51
A/G Ratio	1.25:1



Total Protein	7.07
Serum Albumin	3.56
Serum Globulin	3.51
$Alpha_1$	.30
$Alpha_2$	.76
Beta	.90
Gamma	1.55
A/G Ratio	1.01:1



Haematological Investigations

Amoebic Dysentery On Admission

Case No.	Hb (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
1.	8.2	14,100	66	26.0	31.5
2.	18.1	10,300	11	54.0	33.5
3.	18.2	8,300	12	52.0	35.0
4.	14.8	18,800	6	46.0	32.0
5.	15.9	16,900	23	46.0	34.5
6.	15.9	9,400	22	46.0	34.5
7.	18.1	7,500	3	53.0	34.0
8.	15.7	10,000	11	50.0	31.5
9.	15.6	21,000	36	46.5	33.5
10.	15.8	10,700	12	43.0	36.5
11.	13.7	17,000	45	43.0	32.0
12.	15.9	11,000	32	45.0	35.5
13.	12.8	16,000	52	34.0	37.5
14.	13.2	11,000	40	39.0	34.0
15.	18.0	6,000	15	51.0	35.5
16.	19.2	12,000	3	54.0	35.5
17.	15.2	12,000	43	43.0	35.5
18.	15.0	8,000	19	42.0	36.0
19.	17.7	21,000	9	50.0	35.5
20..	12.1	10,700	34	37.0	33.0
21.	13.5	17,000	22	40.0	34.0
22.	17.7	9,000	22	53.0	33.5
23.	15.2	7,000	9	45.0	34.0
24.	14.6	16,400	24	45.5	32.0
25.	18.8	9,000	22	54.0	35.0
26.	15.3	18,000	35	45.0	34.0
27.	16.0	16,000	29	48.0	33.5
28.	12.8	19,000	25	42.0	30.5
29.	17.9	11,000	11	50.0	36.0
30.	14.6	12,000	12	45.0	32.5
31.	17.0	16,000	34	47.0	36.0
32.	15.4	10,000	15	45.0	34.0
33.	15.5	22,000	14	46.0	34.0
34.	15.6	17,000	13	47.0	33.0
35.	15.2	27,000	24	47.0	32.5

Case No.	Hb. (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
36.	19.0	11,000	6	54.0	35.0
37.	16.2	16,000	22	49.0	33.0
38.	17.0	8,000	30	48.0	35.5
39.	14.4	13,000	40	43.0	33.5
40.	15.6	12,000	29	46.0	34.0
41.	15.5	11,000	27	48.0	32.5
42.	17.0	12,000	8	51.0	33.5
43.	17.0	13,000	14	51.0	33.5
44.	14.4	10,000	29	49.0	29.5
45.	16.4	17,000	24	50.0	33.0
46.	14.6	11,000	45	44.0	33.0
47.	17.2	11,000	11	51.0	34.0
48.	16.3	6,000	3	48.0	34.0
49.	17.2	14,000	3	52.0	33.0
Mean	15.8	13,100	22	46.6	33.8
Range	8.2-19.2	6,000-27,000	3-66	26-54.0	29.5-37.5
S.D.	1.97	—	14.1	5.43	1.57
Amoebic Dysentery On Discharge					
1.	11.7	6,800	53	35.0	33.5
2.	15.9	7,500	32	46.0	34.5
3.	17.0	9,600	16	48.0	35.5
4.	16.0	17,400	10	46.0	35.0
5.	15.8	9,000	16	45.0	35.0
6.	16.3	9,300	8	50.0	32.5
7.	19.1	8,200	2	56.0	34.0
8.	19.3	7,200	7	58.0	33.0
9.	16.2	10,900	27	46.0	35.0
10.	14.8	13,700	13	45.0	33.0
11.	17.4	9,000	21	48.0	36.0
12.	14.0	8,000	37	43.0	32.5
13.	14.9	6,300	22	47.0	32.0
14.	12.4	7,500	36	39.5	31.5
15.	17.2	7,500	19	51.0	34.0

Case No.	Hb. (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
16.	16.8	11,000	5	51.0	33.0
17.	13.2	7,000	23	44.0	30.0
18.	17.2	7,000	8	49.0	35.0
19.	17.5	11,000	2	49.0	35.5
20.	15.4	13,000	36	43.0	36.0
21.	13.8	11,000	39	41.0	34.0
22.	17.8	10,000	10	51.0	35.0
23.	18.8	6,000	2	58.0	32.5
24.	16.6	7,000	6	47.0	35.5
25.	18.0	9,000	1	55.0	33.0
26.	14.0	20,000	9	42.0	33.5
27.	19.0	9,000	4	54.0	35.0
28.	14.2	9,000	20	46.0	31.0
29.	17.4	8,000	19	50.0	35.0
30.	14.8	12,000	12	45.0	33.0
31.	16.4	10,000	25	49.0	33.5
32.	14.8	10,000	20	46.0	32.0
33.	16.0	16,000	12	48.0	33.5
34.	15.6	11,000	22	48.0	32.5
35.	17.4	14,000	24	49.0	35.5
36.	16.8	12,000	6	53.0	32.0
37.	15.2	6,000	39	46.0	33.0
38.	17.0	7,000	25	49.0	35.0
39.	14.8	10,000	33	45.0	33.0
40.	14.8	8,000	33	42.0	35.5
41.	16.2	10,000	26	50.0	32.5
42.	18.4	9,000	8	52.0	35.5
43.	16.3	7,000	35	48.0	34.0
44.	15.2	9,000	34	46.0	33.0
45.	16.0	11,000	23	49.0	33.0
46.	14.4	8,000	40	45.0	32.0
47.	15.6	9,000	16	50.0	31.5
48.	15.6	6,000	2	50.0	31.0
49.	16.3	13,000	3	51.0	32.0
Mean	16.0	9,730	19	47.8	33.6
Range	11.7- 19.3	6,000- 20,000	1- 53	35.0- 58.0	30.0- 36.0
S.D.	1.67	----	12.71	4.46	1.50

Amoebic Liver Abscess On Admission

Case No.	Hb (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
1.	11.1	6,500	56	32.5	34.0
2.	16.1	14,100	9	48.0	33.5
3.	13.0	13,100	46	41.0	32.0
4.	9.2	9,800	61	30.0	30.5
5.	11.2	12,500	58	34.0	33.0
6.	14.6	6,300	33	45.0	32.5
7.	10.6	8,000	54	32.0	33.0
8.	9.3	39,000	61	28.0	33.0
9.	12.3	15,000	55	36.0	34.0
10.	10.8	17,000	59	32.0	34.0
11.	11.6	8,100	54	36.0	32.0
12.	14.4	10,600	32	42.0	34.5
13.	10.9	19,000	54	37.0	29.5
14.	13.9	11,800	32	42.0	33.0
15.	16.1	8,600	11	48.0	33.5
16.	12.6	20,100	50	38.5	33.0
17.	12.9	11,700	53	37.0	35.0
18.	12.6	14,400	42	38.0	33.0
19.	15.8	19,200	24	45.0	35.0
20.	11.1	11,200	59	31.5	35.0
21.	14.6	19,900	16	45.0	32.5
22.	9.8	13,600	30	31.5	31.0
23.	13.2	12,000	10	41.0	32.0
24.	10.1	21,700	59	31.0	32.5
25.	13.1	11,600	8	41.5	31.5
26.	15.0	11,000	49	42.0	36.0
27.	15.2	9,000	47	43.0	35.5
28.	12.0	17,000	54	38.0	31.5
29.	9.1	10,600	61	30.5	30.0
30.	15.0	13,000	44	44.0	34.0
31.	10.4	15,000	57	34.0	30.5
Mean	12.5	13,900	44	37.9	32.9
Range	9.1–16.1	6,300–39,000	8–61	28.0–48.0	29.5–36.0
S.D.	2.15	——	17.50	5.73	1.63

Amoebic Liver Abscess On Discharge

Case No.	Hb (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
1.	13.1	8,400	26	39.0	33.5
2.	15.4	10,700	38	45.5	33.5
3.	15.3	6,200	33	46.0	33.0
4.	13.6	6,700	45	38.0	36.0
5.	13.9	8,200	37	42.0	33.0
6.	15.9	6,800	40	45.5	35.0
7.	13.2	9,500	48	40.0	33.0
8.	12.7	8,400	31	39.0	32.5
9.	13.9	10,400	45	42.5	33.0
10.	14.6	14,700	25	44.5	33.0
11.	17.4	4,500	4	50.0	35.0
12.	17.2	9,100	15	48.0	36.5
13.	15.8	13,200	18	46.0	34.5
14.	17.6	9,200	7	48.0	36.5
15.	16.4	6,800	7	51.0	32.0
16.	15.4	9,200	29	44.0	35.0
17.	15.9	7,500	32	48.0	34.5
18.	14.6	8,500	44	43.5	33.5
19.	19.2	8,100	16	54.0	35.0
20.	14.4	12,000	47	42.0	34.0
21.	15.2	7,800	31	44.5	34.0
22.	14.1	13,800	40	43.5	32.5
23.	16.0	14,000	2	51.0	31.5
24.	15.4	11,000	28	45.0	34.0
25.	15.2	9,000	18	48.0	33.0
26.	16.6	9,000	26	46.0	36.0
27.	16.0	7,000	18	48.0	33.0
28.	14.6	7,000	34	46.0	32.0
29.	12.3	6,000	39	41.0	30.0
30.	14.8	9,000	31	45.0	33.0
31.	16.2	13,000	8	51.0	32.0
Mean	15.2	9,200	28	45.3	33.6
Range	12.3- 19.2	4,500- 14,700	2- 48	38.0- 54.0	30.0- 36.5
S.D.	1.53	---	13.28	3.85	1.51

Liver Function Tests

Amoebic Dysentery On Admission and On Discharge

Case No.	Cephalin Flocculation 24hrs.		48hrs.		Cholesterol Esterification 24hrs.		48hrs.		Zn.Sulph. Turb.		Alk.Phos. (K.A.units)		Ser.Bilirubin (mgm./100 ml.)	
	O/A	O/D	O/A	O/D	O/A	O/D	O/A	O/D	O/A	O/D	O/A	O/D	O/A	O/D
1.	0	0	0	0	3	8	6	9	.4	.6				
2.	+++	++++	++	+++	14	18	10	7	.6	.6				
3.	++	+++	+++	++++	7	12	6	7.5	.6	.5				
4.	0	+	+	++	3	4	7.5	9	.4	.4				
5.	0	+	0	++	4	7	11	8.5	.3	.4				
6.	+	++	+	++	3	5	8	7	.4	.4				
7.	+	+	+	+	4	3	8	6.5	.6	.5				
8.	0	0	0	+	4	5	6.5	6.5	.5	.5				
9.	0	++	+	+	4	5	8.5	8.5	.6	.5				
10.	0	0	+	++	3	4	6.5	8	.4	.4				
11.	+	++	++	++	14	18	9	8	.5	.4				
12.	+	+	+	++	3	2	9	7	.3	.3				
13.	+++	++++	+	+	8	8	10	11	.5	.5				
14.	+	+	0	0	7	7	20	22	.5	.5				
15.	0	+	+	+	10	12	6	8	.8	.5				
16.	+	++	0	0	4	3	15	14.5	.3	.4				
17.	0	+	0	++	6	7	8.5	9	.4	.4				
18.	+	+	0	+	5	6	7	7.5	.3	.5				
19.	0	+	0	+	5	5	10	10.5	.4	.5				
20.	0	0	+	+	11	17	9.5	8	.3	.3				
21.	0	+	+	++	14	9	8	7.5	.4	.3				
22.	++	+++	0	+	14	9	14	6.5	.6	.3				
23.	0	+	+	++	3	3	8.5	10	.5	.4				
24.	+	+	+	+	7	7	6	8	.5	.5				
25.	0	+	+	+	2	3	10.5	11.5	.7	.6				
26.	0	+	++	+++	10	14	10	11	.4	.4				
27.	0	+	++	+++	5	7	6	7	.5	.5				
28.	0	+	+	++	3	5	8	16	.4	.5				
29.	++	++++	+	++	6	7	6.5	8.5	.5	.4				
30.	++	++	+	++	8	9	12	10.5	.5	.5				
31.	++	+++	0	+	9	7	10	12	.3	.6				
32.	+	++	+	++	5	4	6.5	8	.4	.3				
33.	+	+	+	++	5	4	24.5	26	.7	.6				
34.	+	+	+	+++	7	14	8.5	7	.4	.3				

Case No.	Cephalin Cholesterol 24hrs.		48hrs.		Zn.Sulph. Turb.		Alk.Phos. (K.A.units)		Ser.Bilirubin. (mgm./100 ml.)	
	0/A		0/D		0/A	0/D	0/A	0/D	0/A	0/D
35.	+	++	++	+++	3	5	9	10	.5	.4
36.	+	+	++++	++++	4	3	9.5	7	.5	.6
37.	+	++	+	+	4	10	7.5	8	.5	.5
38.	+	++	+	++	8	4	8.5	9	.6	.6
39.	+	++	++	+++	14	9	9	7.5	.5	.6
40.	+	+	+	++	5	9	8	9.5	.4	.4
41.	+	+	++	+++	4	7	7	7	.5	.5
42.	++	++++	++	+++	2	3	13	10	.6	.6
43.	0	+	+	++	3	5	9	12.5	.4	.5
44.	+	++	++	+++	7	7	7	9	.3	.4
45.	+++	++++	+++	++++	3	4	7	6.5	.4	.5
46.	0	+	+	+++	2	3	5.5	10	.4	.5
47.	0	+	0	+	3	3	6	3	.4	.4
48.	0	+	0	+	3	5	7	8.5	.5	.5
49.	0	+	0	0	2	2	9.5	7.5	.4	.5
Mean	—	—	—	—	5.9	6.8	9.1	9.3	.47	.47
Range	—	—	—	—	2–14	2–18	5.5–24.5	3–26	.3–.8	.3–.6
S.D.	—	—	—	—	3.56	4.05	3.47	3.79	.11	.09

Five Cases of Amoebic Dysentery On Admission, On Discharge and at 1 Month Follow Up.

Case No.	Cephalin Cholesterol 48 hrs.			Zn.Sulph. Turb.			Alk.Phos. (K.A.units)		
	0/A	0/D	1 mth.	0/A	0/D	1mth.	0/A	0/D	1mth.
1.	+	++	++	4	7	7	11	8.5	10
2.	0	+	+	5	5	6	6.5	6.5	5
3.	++	+	+	4	5	8	8.5	8.5	8
4.	++++	+	+	8	8	8	10	11	12
5.	+	+	+	2	3	2	10.5	11.5	12
Mean	—	—	—	4.6	5.6	6.2	9.3	9.2	9.4

Amoebic Liver Abscess On Admission and On Discharge

Case No.	Cephalin Flocculation 24hrs.	Cephalin Flocculation 48hrs.	Cholesterol Flocculation 24hrs.	Cholesterol Flocculation 48hrs.	ZnSulph. Turb.		Alk.Phos. (K.A.units)		Ser.Bilirubin. (mgm./100 ml.)	
	0/A		0/D		0/A	0/D	0/A	0/D	0/A	0/D
1.	0	0	0	+	20	8	14	8	.4	.5
2.	++	+++	0	+	9	6	10	5	.4	.5
3.	0	0	0	0	4	6	12	7	.4	.4
4.	+	+	+	+	28	18	18	21	.4	.4
5.	0	+	0	+	26	26	22	10.5	.5	.4
6.	0	+	0	+	7	28	13	14	.4	.4
7.	0	0	+	+	26	32	15	19	.4	.4
8.	+	++	0	0	7	9	23	16	.5	.5
9.	0	+	0	0	12	10	12	12	.4	.4
10.	0	0	0	0	16	10	14	14	.4	.5
11.	0	+	+	+	14	4	13	8.5	.5	.4
12.	0	+	++	+++	10	7	9.5	7	.3	.4
13.	0	+	0	0	12	9	10	9	.4	.3
14.	+++	++++	+++	++++	14	10	14	9.5	.5	.5
15.	0	+	0	+	18	10	10.5	6.5	.4	.4
16.	0	+	+	++	5	8	12.5	12.5	.4	.5
17.	+	++	++	+++	14	20	12	10.5	.4	.5
18.	+	++	+++	++++	18	24	14	17	.4	.4
19.	+	++	+++	++++	12	9	12	8	.5	.4
20.	++	+++	+	++	18	18	12.5	10	.4	.4
21.	0	+	+	+	7	8	10.5	12.5	.5	.5
22.	0	+	+	++	9	10	17.5	6	.4	.4
23.	+	++	0	+	18	8	15.5	10.5	.3	.3
24.	+	+++	+	+++	14	24	11.5	15.5	.3	.4
25.	+	++	+	++	28	16	10	16	.5	.5
26.	0	+	+++	+++	10	16	8	5.5	.8	.5
27.	0	+	+	++	16	7	14	14.5	.8	.4
28.	0	0	+++	++++	10	4	11	12.5	.5	.4
29.	0	+	+	+++	8	6	25.5	17	.5	.5
30.	+	++	0	+	11	16	8	8	.4	.5
31.	0	++	+	++	20	9	18	14	.3	.6
Mean	—	—	—	—	14.2	12.8	13.6	11.5	.44	.44
Range	—	—	—	—	4–28	4–32	8–25.5	5–21	.3–.8	.3–.6
S.D.	—	—	—	—	6.6	7.7	4.2	4.2	.11	.07

Ten cases of Liver Abscess On Admission, On Discharge and at 1 Month Follow Up.

Case No.	Cephalin Cholesterol			Zn Sulph. Turb.			Alk.Phos. (K.A.units)		
	0/A	0/D	1 mth.	0/A	0/D	1 mth.	0/A	0/D	1mth.
1.	0	+	++	20	8	8	14	8	7
2.	+++	+	0	9	6	5	10	5	8.3
3.	0	+	0	26	26	14	22	10.5	9
4.	+	0	++	12	9	7	10	9	10
5.	++++	++++	++++	14	10	8	14	9.5	11.5
6.	+	+	+++	18	10	6	10.5	6.5	7
7.	++	++++	++++	18	24	9	14	17	13
8.	++	++++	++++	12	9	8	12	8	9
9.	+++	++	++++	18	18	16	12.5	10	9
10.	+	++	++++	9	10	10	17.5	6	7
Mean	—	—	—	15.6	13.0	9.1	13.7	9.0	9.1

Serum Proteins

Chemical Fractionation

Amoebic Dysentery On Admission

Case No.	Total Protein	Serum Albumin	Serum Globulin	A/G Ratio
1.	4.71	1.26	3.45	.37:1
2.	7.57	2.38	5.19	.46:1
3.	7.25	2.65	4.60	.58:1
4.	6.43	2.57	3.86	.67:1
5.	7.24	2.83	4.41	.64:1
6.	7.35	3.34	4.01	.83:1
7.	7.70	3.57	4.13	.87:1
8.	6.44	2.99	3.45	.87:1
9.	7.27	2.89	4.38	.66:1
10.	5.52	2.13	3.39	.63:1
11.	7.82	2.94	4.88	.60:1
12.	6.30	2.69	3.61	.75:1
13.	7.86	2.88	4.98	.58:1
14.	6.61	2.69	3.92	.69:1
15.	7.88	3.26	4.62	.71:1
16.	7.25	3.47	3.78	.92:1
17.	6.41	2.42	3.99	.61:1
18.	6.87	3.32	3.55	.94:1
19.	6.42	2.66	3.76	.71:1
20.	5.20	1.11	4.09	.27:1
21.	6.84	2.56	4.28	.60:1
22.	7.05	2.56	4.49	.57:1
23.	6.40	2.95	3.45	.86:1
24.	5.67	2.56	3.11	.82:1
25.	6.84	2.99	3.85	.78:1
26.	5.89	2.03	3.86	.53:1
27.	6.14	2.36	3.78	.62:1
28.	4.48	1.42	3.06	.47:1
29.	5.70	2.13	3.57	.60:1
30.	6.50	2.74	3.76	.73:1
31.	6.58	2.13	4.45	.48:1
32.	5.48	2.13	3.35	.64:1

Case No.	Total Protein	Serum Albumin	Serum Globulin	A/G Ratio
33.	6.50	2.34	4.16	.56:1
34.	6.74	1.93	4.81	.40:1
35.	6.15	2.50	3.65	.68:1
36.	6.50	2.96	3.54	.84:1
37.	5.89	2.21	3.68	.60:1
38.	6.99	3.29	3.70	.89:1
39.	8.15	2.62	5.53	.47:1
40.	5.20	1.54	3.66	.42:1
41.	5.96	2.50	3.46	.72:1
42.	6.55	2.89	3.66	.79:1
43.	6.93	2.41	4.52	.53:1
44.	6.83	2.42	4.41	.55:1
45.	6.30	2.46	3.84	.64:1
46.	5.70	2.16	3.54	.61:1
47.	6.80	2.60	4.20	.62:1
48.	6.51	3.20	3.31	.97:1
49.	6.20	3.20	3.00	1.07:1
Mean	6.52	2.57	3.95	.65:1
Range	4.48- 8.15	1.11- 3.57	3.00- 5.53	.27:1- 1.07:1
S.D.	.81	.54	.56	---
Amoebic Dysentery On Discharge				
1.	5.95	2.02	3.93	.51:1
2.	8.02	3.45	4.57	.75:1
3.	8.40	4.03	4.37	.92:1
4.	7.25	3.45	3.80	.91:1
5.	8.28	3.76	4.52	.83:1
6.	8.03	4.14	3.89	1.06:1
7.	7.89	3.58	4.31	.83:1
8.	7.15	3.45	3.70	.93:1
9.	7.51	3.08	4.43	.70:1
10.	6.00	2.69	3.31	.81:1
11.	8.00	3.15	4.85	.65:1
12.	6.63	3.05	3.58	.85:1
13.	8.30	3.66	4.64	.79:1
14.	7.86	3.21	4.65	.69:1

Case No.	Total Protein	Serum Albumin	Serum Globulin	A/G Ratio
15.	8.08	3.32	4.76	.70:1
16.	7.05	3.63	3.42	1.06:1
17.	6.50	2.85	3.65	.78:1
18.	7.10	3.54	3.56	.99:1
19.	6.10	2.86	3.24	.88:1
20.	6.50	2.26	4.24	.53:1
21.	7.10	2.96	4.14	.71:1
22.	7.30	2.85	4.45	.64:1
23.	7.01	3.25	3.76	.86:1
24.	7.17	3.54	3.63	.98:1
25.	7.10	2.95	4.15	.71:1
26.	6.90	2.36	4.54	.52:1
27.	7.27	3.15	4.12	.76:1
28.	7.36	3.29	4.07	.81:1
29.	6.79	3.10	3.69	.84:1
30.	6.69	3.10	3.59	.86:1
31.	7.14	2.40	4.74	.51:1
32.	6.83	3.26	3.57	.91:1
33.	8.00	3.15	4.85	.65:1
34.	8.00	2.94	5.06	.58:1
35.	6.93	2.73	4.20	.65:1
36.	6.89	3.34	3.55	.94:1
37.	6.89	2.16	4.73	.46:1
38.	6.80	3.25	3.55	.92:1
39.	8.03	3.25	4.78	.68:1
40.	7.11	3.05	4.06	.75:1
41.	7.83	3.45	4.38	.79:1
42.	7.01	3.45	3.56	.97:1
43.	7.31	3.05	4.26	.72:1
44.	6.60	2.34	4.26	.55:1
45.	6.90	2.95	3.95	.75:1
46.	6.50	2.84	3.66	.78:1
47.	7.14	2.89	4.25	.68:1
48.	7.22	3.62	3.60	1.01:1
49.	6.41	3.31	3.10	1.07:1
Mean	7.20	3.12	4.08	.76:1
Range	5.95-8.40	2.02-4.14	3.10-5.06	.51:1-1.07:1
S.D.	.62	.47	.50	---

Amoebic Liver Abscess On Admission

Case No.	Total Protein	Serum Albumin	Serum Globulin	A/G Ratio
1.	6.78	2.41	4.37	.55:1
2.	7.80	3.30	4.50	.73:1
3.	6.95	2.34	4.61	.51:1
4.	8.19	1.83	6.36	.29:1
5.	7.85	1.67	6.18	.27:1
6.	6.34	2.20	4.14	.53:1
7.	8.26	2.20	6.06	.36:1
8.	6.05	1.83	4.22	.43:1
9.	7.21	1.57	5.64	.28:1
10.	6.90	1.80	5.10	.35:1
11.	7.00	1.91	5.09	.38:1
12.	7.90	2.26	5.64	.40:1
13.	6.11	1.58	4.53	.35:1
14.	8.25	1.92	6.33	.30:1
15.	7.60	2.37	5.23	.45:1
16.	6.10	1.59	4.51	.35:1
17.	7.03	1.43	5.60	.26:1
18.	8.16	1.95	6.21	.31:1
19.	8.15	2.89	5.26	.55:1
20.	7.02	1.64	5.38	.30:1
21.	8.30	3.30	5.00	.66:1
22.	6.20	1.58	4.62	.34:1
23.	7.15	2.10	5.05	.42:1
24.	6.10	1.22	4.88	.25:1
25.	8.99	1.93	7.06	.27:1
26.	6.10	1.80	4.30	.42:1
27.	6.50	1.60	4.90	.33:1
28.	6.20	1.80	4.40	.41:1
29	5.00	1.38	4.22	.33:1
30.	6.40	1.73	4.67	.37:1
31.	6.79	1.55	5.24	.30:1
Mean	7.10	1.96	5.14	.38:1
Range	5.00-8.99	1.22-3.30	4.14-7.06	.25:1-.73:1
S.D.	.90	.50	.76	—

Amoebic Liver Abscess On Discharge

Case No.	Total Protein	Serum Albumin	Serum Globulin	A/G Ratio
1.	7.10	3.10	4.00	.78:1
2.	7.13	3.66	3.47	1.05:1
3.	7.60	3.46	4.14	.84:1
4.	8.78	3.58	5.20	.69:1
5.	6.69	2.19	4.50	.49:1
6.	7.75	2.44	5.31	.46:1
7.	7.94	2.62	5.32	.49:1
8.	6.16	2.55	3.61	.71:1
9.	6.37	2.23	4.14	.54:1
10.	7.57	2.90	4.67	.62:1
11.	7.70	3.62	4.08	.89:1
12.	7.02	2.65	4.37	.61:1
13.	8.03	3.28	4.75	.69:1
14.	7.46	2.94	4.52	.65:1
15.	7.49	2.92	4.57	.64:1
16.	7.80	3.34	4.46	.75:1
17.	8.45	3.34	5.11	.65:1
18.	9.65	3.22	6.43	.50:1
19.	7.80	3.01	4.79	.63:1
20.	7.42	2.77	4.65	.60:1
21.	7.63	3.10	4.53	.68:1
22.	7.05	2.99	4.06	.74:1
23.	7.30	2.75	4.55	.60:1
24.	8.32	3.10	5.22	.59:1
25.	7.55	2.52	5.03	.50:1
26.	7.46	3.15	4.31	.73:1
27.	6.74	3.15	3.59	.88:1
28.	7.76	3.15	4.61	.68:1
29.	7.49	2.95	4.54	.65:1
30.	8.13	2.64	5.49	.48:1
31.	7.11	2.74	4.37	.63:1
Mean	7.56	2.97	4.59	.65:1
Range	6.16- 9.65	2.19- 3.66	3.47- 6.43	.46:1- 1.05:1
S.D.	.69	.38	.61	---

Five cases of Amoebic Dysentery On Admission, On Discharge and at 1 Month Follow Up.

Case No.	Total Protein			Serum Albumin			Serum Globulin			A/G Ratio		
	O/A	O/D	1mth.	O/A	O/D	1mth.	O/A	O/D	1mth.	O/A	O/D	1mth.
1.	7.24	8.28	7.75	2.83	3.76	3.88	4.41	4.52	3.87	.64:1	.83:1	1.00:1
2.	6.44	7.15	6.50	2.99	3.45	3.15	3.45	3.70	3.35	.87:1	.92:1	.94:1
3.	7.27	7.51	7.25	2.89	3.08	3.15	4.38	4.43	4.10	.66:1	.70:1	.77:1
4.	7.86	8.30	7.17	2.88	3.66	3.15	4.98	4.64	4.02	.58:1	.79:1	.78:1
5.	6.84	7.10	6.90	2.99	2.95	3.25	3.85	4.15	3.65	.78:1	.71:1	.89:1
Mean	7.13	7.67	7.12	2.92	3.38	3.32	4.21	4.29	3.80	.69:1	.80:1	.87:1

Ten cases of Amoebic Liver Abscess On Admission, On Discharge and at 1 Month Follow Up.

1.	6.78	7.10	7.34	2.41	3.10	3.20	4.37	4.00	4.14	.55:1	.78:1	.77:1
2.	7.80	7.13	7.46	3.30	3.66	3.97	4.50	3.47	3.49	.73:1	1.05:1	1.14:1
3.	7.85	6.69	7.45	1.67	2.19	3.40	6.18	4.50	4.05	.27:1	.49:1	.84:1
4.	6.11	8.03	8.05	1.58	3.28	3.91	4.53	4.75	4.14	.35:1	.69:1	.94:1
5.	8.25	7.46	8.79	1.92	2.94	3.89	6.33	4.52	4.90	.30:1	.65:1	.79:1
6.	7.60	7.49	7.79	2.37	2.92	3.76	5.23	4.57	4.03	.45:1	.64:1	.93:1
7.	8.16	9.65	8.40	1.95	3.22	3.20	6.21	6.43	5.20	.31:1	.50:1	.62:1
8.	8.15	7.80	6.63	2.89	3.01	2.88	5.26	4.79	3.75	.55:1	.63:1	.77:1
9.	7.02	7.42	7.56	1.64	2.77	2.52	5.38	4.65	5.04	.30:1	.60:1	.50:1
10.	6.20	7.05	6.20	1.58	2.99	2.72	4.62	4.06	3.48	.34:1	.74:1	.78:1
Mean	7.39	7.58	7.57	2.13	3.01	3.35	5.26	4.57	4.22	.40:1	.66:1	.79:1

Paper Electrophoresis

Amoebic Dysentery On Admission

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	Alpha$_1$	Alpha$_2$	Beta	Gamma
1.	1.57	3.14	.50:1	.40	.81	.58	1.35
2.	2.69	4.88	.55:1	.45	1.23	.92	2.28
3.	3.05	4.20	.73:1	.49	.84	.76	2.11
4.	2.73	3.70	.74:1	.30	.76	.98	1.66
5.	2.95	4.29	.69:1	.44	1.20	.91	1.74
6.	3.41	3.94	.87:1	.46	.93	.82	1.73
7.	3.51	4.19	.84:1	.55	.84	1.05	1.75
8.	3.12	3.32	.94:1	.36	.74	.87	1.35
9.	2.84	4.43	.64:1	.49	1.35	.91	1.68
10.	2.35	3.17	.74:1	.40	.83	.77	1.17
11.	3.07	4.75	.65:1	.54	1.01	.91	2.29
12.	2.96	3.34	.89:1	.45	.99	.59	1.31
13.	3.24	4.62	.70:1	.56	1.12	.89	2.05
14.	3.07	3.54	.87:1	.35	1.03	.89	1.27
15.	3.66	4.22	.87:1	.31	1.02	.84	2.05
16.	3.58	3.67	.98:1	.39	1.03	.94	1.31
17.	2.53	3.88	.65:1	.51	1.18	.88	1.31
18.	3.42	3.45	.99:1	.34	.71	.87	1.53
19.	3.12	3.30	.95:1	.36	.81	.80	1.33
20.	1.60	3.60	.44:1	.33	.83	.63	1.81
21.	2.83	4.01	.71:1	.39	.80	.84	1.98
22.	2.90	4.15	.70:1	.32	.93	.77	2.13
23.	3.31	3.09	1.07:1	.32	.73	.77	1.27
24.	2.80	2.87	.98:1	.33	.78	.64	1.12
25.	3.09	3.75	.82:1	.38	1.07	.92	1.38
26.	2.33	3.56	.65:1	.37	.77	.63	1.79
27.	2.94	3.20	.92:1	.39	.71	.73	1.37
28.	1.56	2.92	.53:1	.39	.84	.67	1.02
29.	2.69	3.01	.89:1	.34	.68	.71	1.28
30.	3.13	3.37	.93:1	.33	.69	.79	1.56
31.	2.41	4.17	.58:1	.45	1.03	1.03	1.66
32.	2.41	3.07	.79:1	.36	.57	.70	1.44
33.	2.17	4.33	.50:1	.48	1.16	.93	1.76
34.	2.51	4.23	.59:1	.28	.99	.76	2.20
35.	2.84	3.31	.86:1	.40	.85	.75	1.31

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	$Alpha_1$	$Alpha_2$	Beta	Gamma
36.	2.93	3.57	.82:1	.26	.72	.82	1.77
37.	2.27	3.62	.63:1	.47	.93	.81	1.41
38.	3.34	3.65	.92:1	.33	.69	.85	1.78
39.	3.29	4.86	.68:1	.40	1.26	1.00	2.20
40.	2.03	3.17	.64:1	.29	.73	.63	1.52
41.	2.72	3.24	.84:1	.20	.64	.76	1.64
42.	3.16	3.39	.93:1	.31	.83	.70	1.55
43.	2.88	4.05	.71:1	.53	1.10	.80	1.62
44.	2.83	4.00	.71:1	.26	.84	1.06	1.84
45.	2.82	3.48	.81:1	.35	1.00	.63	1.50
46.	2.50	3.20	.78:1	.34	1.04	.67	1.15
47.	2.89	3.91	.74:1	.23	.96	.90	1.82
48.	3.27	3.24	1.01:1	.24	.66	.65	1.69
49.	3.20	3.00	1.07:1	.34	.71	.68	1.27
Mean	2.83	3.69	.77:1	.38	.90	.80	1.61
Range	1.56-3.66	2.87-4.88	.44:1-1.07:1	.20-.56	.57-1.35	.58-1.06	1.92-2.29
S.D.	.48	.53	——	.09	.18	.13	.33

Amoebic Dysentery On Discharge

1.	2.48	3.47	.71:1	.40	.73	.88	1.46
2.	3.83	4.19	.91:1	.34	.85	.95	2.05
3.	3.94	4.46	.88:1	.33	.78	.93	2.42
4.	3.31	3.94	.84:1	.38	.81	.96	1.79
5.	4.28	4.00	1.07:1	.37	.78	1.09	1.76
6.	4.34	3.69	1.18:1	.48	.91	1.02	1.28
7.	4.07	3.82	1.07:1	.43	.80	1.12	1.47
8.	3.43	3.72	.92:1	.37	.71	.93	1.71
9.	3.52	3.99	.88:1	.29	.84	.95	1.91
10.	2.74	3.26	.84:1	.32	.82	.96	1.16
11.	3.61	4.39	.82:1	.45	.96	.88	2.10
12.	3.17	3.46	.92:1	.27	1.00	.91	1.28
13.	4.13	4.17	.99:1	.37	.88	.99	1.93
14.	3.77	4.09	.92:1	.33	.83	1.07	1.86
15.	3.81	4.27	.89:1	.30	.78	.95	2.24

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	Alpha$_1$	Alpha$_2$	Beta	Gamma
16.	3.80	3.25	1.17:1	.32	.75	.80	1.38
17.	3.09	3.41	.91:1	.26	.74	.88	1.53
18.	3.80	3.30	1.15:1	.22	.65	.86	1.57
19.	3.07	3.03	1.01:1	.24	.70	.63	1.46
20.	2.60	3.90	.67:1	.31	.63	.75	2.21
21.	3.03	4.07	.74:1	.35	.71	.84	2.17
22.	3.25	4.05	.80:1	.33	.78	.91	2.03
23.	3.60	3.41	1.06:1	.34	.76	.90	1.41
24.	3.76	3.41	1.10:1	.30	.64	.90	1.57
25.	3.49	3.61	.97:1	.31	.78	.80	1.72
26.	2.52	4.38	.58:1	.39	.79	.92	2.28
27.	3.44	3.83	.90:1	.25	.71	.99	1.88
28.	3.68	3.68	1.00:1	.24	.71	1.09	1.64
29.	3.38	3.41	.99:1	.24	.71	.94	1.52
30.	3.16	3.53	.90:1	.31	.66	.97	1.59
31.	3.14	4.00	.79:1	.31	.97	.90	1.82
32.	3.58	3.25	1.10:1	.27	.55	.87	1.56
33.	3.42	4.58	.75:1	.36	1.22	1.00	2.00
34.	3.42	4.58	.75:1	.21	.86	1.09	2.42
35.	3.36	3.57	.94:1	.27	.79	1.03	1.48
36.	3.55	3.34	1.06:1	.23	.69	.89	1.53
37.	2.75	4.14	.66:1	.38	.96	.92	1.88
38.	3.58	3.22	1.11:1	.21	.60	.91	1.50
39.	3.81	4.22	.90:1	.20	.75	.92	2.35
40.	3.49	3.62	.96:1	.21	.68	.74	1.99
41.	3.81	4.02	.95:1	.23	.71	.90	2.18
42.	3.58	3.43	1.04:1	.24	.70	.79	1.70
43.	3.36	3.95	.85:1	.34	.96	1.03	1.62
44.	3.08	3.52	.88:1	.24	.78	.77	1.73
45.	3.28	3.62	.91:1	.23	.65	.55	2.19
46.	3.15	3.34	.94:1	.37	.84	.70	1.44
47.	3.56	3.58	.99:1	.27	.79	.84	1.68
48.	4.11	3.11	1.32:1	.25	.58	.69	1.61
49.	3.41	3.00	1.14:1	.29	.65	.84	1.22
Mean	3.46	3.74	.93:1	.31	.77	.90	1.76
Range	2.48-4.34	2.80-4.58	.57:1-1.33:1	.20-.48	.55-1.22	.55-1.12	1.16-2.42
S.D.	.42	.42	——	.07	.12	.11	.33

Amoebic Liver Abscess On Admission

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	$Alpha_1$	$Alpha_2$	Beta	Gamma
1.	2.34	4.44	.53:1	.37	.65	.85	2.57
2.	3.81	3.99	.95:1	.31	1.15	.79	1.74
3.	2.57	4.38	.59:1	.55	1.32	.63	1.88
4.	2.10	6.09	.34:1	.46	.71	.98	3.94
5.	1.92	5.93	.32:1	.47	1.01	.61	3.84
6.	2.60	3.74	.70:1	.44	.83	.73	1.74
7.	2.14	6.12	.35:1	.40	.97	.94	3.81
8.	2.03	4.02	.50:1	.45	.94	.83	1.80
9.	2.24	4.97	.45:1	.48	1.13	.90	2.46
10.	2.07	4.83	.43:1	.43	1.14	.77	2.49
11.	2.63	4.37	.60:1	.36	.98	.74	2.29
12.	3.03	4.87	.62:1	.46	1.48	.86	2.07
13.	1.81	4.30	.42:1	.47	1.12	.72	1.99
14.	2.35	5.90	.40:1	.51	1.36	1.14	2.89
15.	3.45	4.15	.83:1	.38	.91	.89	1.97
16.	1.92	4.18	.46:1	.57	1.32	.73	1.56
17.	1.96	5.07	.39:1	.55	1.05	.84	2.63
18.	2.48	5.68	.44:1	.55	1.22	1.00	2.91
19.	2.89	5.26	.55:1	.56	1.17	1.13	2.40
20.	2.20	4.82	.46:1	.37	.93	.98	2.54
21.	3.35	4.95	.68:1	.73	1.57	.95	1.70
22.	1.80	4.40	.41:1	.68	1.10	1.00	1.62
23.	2.23	4.92	.45:1	.61	1.04	.92	2.35
24.	1.56	4.54	.34:1	.48	.80	.70	2.56
25.	2.19	6.80	.32:1	.62	1.31	.90	3.97
26.	2.35	3.75	.63:1	.39	.93	.87	1.56
27.	2.35	4.15	.57:1	.38	1.06	.75	1.96
28.	2.34	3.86	.61:1	.44	1.29	.73	1.40
29.	1.60	4.00	.40:1	.50	.83	.84	1.83
30.	1.95	4.45	.44:1	.44	1.11	.66	2.24
31.	1.52	5.27	.29:1	.46	1.03	.88	2.90
Mean	2.32	4.78	.49:1	.48	1.08	.85	2.37
Range	1.52-3.81	3.74-6.80	.29:1-.95:1	.31-.73	.65-1.57	.61-1.14	1.40-3.97
S.D.	.54	.79	---	.10	.20	.13	.73

Ten cases of Amoebic Liver Abscess on Admission, On Discharge and at 1 Month Follow Up.

Case No.	Serum Albumin			Serum Globulin			A/G Ratio		
	O/A	O/D	1mth.	O/A	O/D	1mth.	O/A	O/D	1mth.
1.	2.34	3.77	4.05	4.44	3.33	3.29	.53:1	1.13:1	1.23:1
2.	3.81	4.05	4.47	3.99	3.08	2.99	.95:1	1.31:1	1.49:1
3.	1.92	2.94	3.51	5.93	3.75	3.94	.32:1	.78:1	.89:1
4.	1.81	3.09	3.68	4.30	4.94	4.37	.42:1	.63:1	.84:1
5.	2.35	3.24	4.28	5.90	4.22	4.51	.40:1	.77:1	.95:1
6.	3.45	3.66	3.97	4.15	3.83	3.82	.83:1	.96:1	1.04:1
7.	2.48	3.53	3.58	5.68	6.12	4.82	.44:1	.58:1	.74:1
8.	2.89	3.48	3.18	5.26	4.32	3.45	.55:1	.81:1	.92:1
9.	2.20	3.40	3.01	4.82	4.02	4.55	.46:1	.85:1	.66:1
10.	1.80	3.33	2.91	4.40	3.72	3.29	.41:1	.90:1	.88:1
Mean	2.50	3.45	3.67	4.89	4.13	3.90	.51:1	.84:1	.94:1

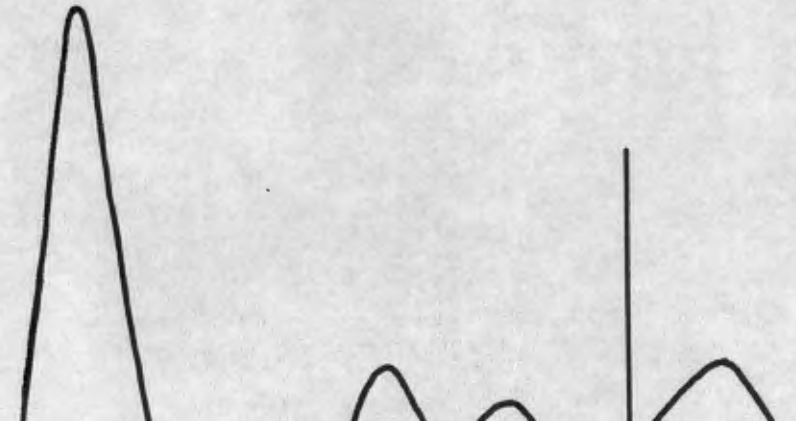
Case No.	Alpha$_1$			Alpha$_2$			Beta			Gamma		
	O/A	O/D	1mth.	O/A	O/D	1mth.	O/A	O/D	1mth.	O/A	O/D	1mth.
1.	.37	.26	.29	.65	.49	.63	.85	.75	.66	2.57	1.83	1.71
2.	.31	.23	.25	1.15	.70	.61	.79	.74	.65	1.74	1.41	1.48
3.	.47	.19	.36	1.01	.60	.69	.61	.58	.71	3.84	2.38	2.18
4.	.47	.23	.36	1.12	.99	.96	.72	1.48	1.17	1.99	2.24	1.88
5.	.51	.30	.36	1.36	.81	.84	1.14	1.18	1.05	2.89	1.95	2.26
6.	.38	.35	.39	.91	.65	.78	.89	.89	.81	1.97	1.94	1.84
7.	.55	.33	.30	1.22	.81	.81	1.00	.99	.84	2.91	3.99	2.87
8.	.56	.41	.32	1.17	.87	.66	1.13	.87	.69	2.40	2.17	1.79
9.	.37	.27	.36	.93	.82	.92	.98	.91	.94	2.54	2.02	2.33
10.	.68	.28	.28	1.10	.69	.55	1.00	.97	.76	1.62	1.78	1.70
Mean	.47	.29	.33	1.06	.74	.75	.91	.93	.83	2.45	2.17	2.00

Amoebic Dysentery On Admission

Electrophoretic Pattern

Case No. 35

Total Protein	6.15
Serum Albumin	2.84
Serum Globulin	3.31
$Alpha_1$	.40
$Alpha_2$	.85
Beta	.75
Gamma	1.31
A/G Ratio	.86:1



Amoebic Dysentery On Discharge

Case No. 35

Electrepheretic Pattern

Total Protein	6.93
Serum Albumin	3.36
Serum Globulin	3.57
$Alpha_1$	.27
$Alpha_2$	.79
Beta	1.03
Gamma	1.48
A/G Ratio	.94:1

Amoebic Liver Abscess On Admission Case No.23

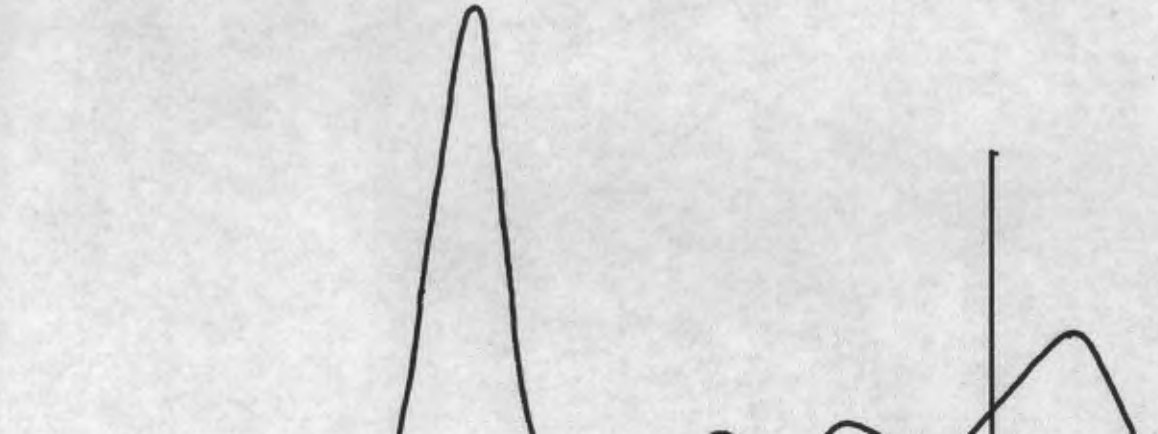
Electrophoretic Pattern

Total Protein	7.15
Serum Albumin	2.23
Serum Globulin	4.92
$Alpha_1$	.61
$Alpha_2$	1.04
Beta	.92
Gamma	2.35
A/G Ratio	.45:1

Amoebic Liver Abscess On Discharge Case No. 23

Electropheretic Pattern

Total Protein	7.30
Serum Albumin	3.23
Serum Globulin	4.07
$Alpha_1$	.31
$Alpha_2$	.72
Beta	.91
Gamma	2.13
A/G Ratio	.79:1



Haematological Investigations

Cirrhosis of the Liver

Case No.	Hb (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
1.	12.9	3,900	54	36.5	35.0
2.	14.4	4,600	48	41.0	35.0
3.	13.0	4,900	53	42.0	31.0
4.	16.9	15,200	5	50.0	34.0
5.	10.1	8,500	63	30.0	33.5
6.	12.8	6,200	17	38.0	33.5
7.	11.0	13,500	48	32.0	34.5
8.	13.1	7,500	48	39.0	33.5
9.	14.1	5,400	31	39.0	36.0
10.	13.0	8,600	51	38.0	34.0
Mean	13.1	7,800	42	38.6	34.0
Range	10.1- 16.9	3,900- 15,200	5- 63	30.0- 50.0	31.0- 36.0
S.D.	1.86	—	18.3	5.48	2.0

Primary Carcinoma of the Liver

1.	12.8	9,900	49	39.0	33.0
2.	16.6	23,300	18	51.9	32.5
3.	16.0	11,000	35	45.0	35.5
4.	9.5	4,000	55	31.0	30.5
5.	11.4	17,000	49	38.0	30.0
6.	9.5	5,000	60	31.0	30.5
7.	13.9	14,400	35	43.0	32.5
8.	19.4	5,000	3	57.5	33.5
9.	14.2	10,400	42	44.0	32.5
10.	11.6	12,700	53	36.5	32.0
Mean	13.5	11,300	40	41.6	32.5
Range	9.5- 19.4	4,000- 23,300	3- 60	31.0- 57.5	30.0- 35.5
S.D.	3.19	—	17.8	8.95	1.66

Lobar Pneumonia

Case No.	Hb. (G./100 ml.)	Wbc. (cu.mm.)	E.S.R. (mm./1hr.)	P.C.V. (%)	M.C.H.C. (%)
1.	12.9	9,000	53	38.0	34.0
2.	14.9	13,400	34	44.0	34.0
3.	14.6	11,600	42	42.5	34.5
4.	15.0	13,000	21	45.0	33.5
5.	14.2	23,000	34	43.0	33.0
6.	14.2	5,000	7	48.0	29.5
7.	13.2	8,000	43	44.0	30.0
8.	13.6	5,500	47	43.5	31.5
9.	14.4	41,000	14	46.0	31.5
10.	15.1	9,000	31	45.0	33.5
Mean	14.2	13,900	32.6	43.9	32.5
Range	12.9- 15.1	5,000- 41,000	7- 53	38.0- 48.0	29.5- 34.5
S.D.	.77	---	14.78	2.61	1.76

Liver Function Tests

Cirrhosis of the Liver

Case No.	Ceph. Chol. 24hrs.	48hrs.	Zn.Sulph. Turb	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100 ml.)
1.	+++	++++	40	9	.4
2.	++++	++++	24	15	.9
3.	++++	++++	16	17	1.6
4.	++++	++++	24	42	3.5
5.	+++	++++	24	9	2.2
6.	+	+	16	11.5	.5
7.	+++	++++	16	12	.9
8.	0	+	22	26.5	.5
9.	++++	++++	9	16.5	1.6
10.	+++	++++	40	13	.6
Mean	---	---	23.1	17.6	1.3
Range	---	---	9- 40	9- 42	.4- 3.5
S.D.			10.14	10.13	.98

Primary Carcinoma of the Liver

Case No.	Ceph. Chol. 24hrs.	48hrs.	Zn.Sulph. Turb.	Alk.Phos. (K.A.units)	Ser.Bilirubin (mgm./100 ml.)
1.	+++	++++	19	46	.5
2.	+	+	7	17	.6
3.	+	++++	4	21	2.3
4.	+++	++++	8	72.5	7.9
5.	+	+	6	85	2.7
6.	++++	++++	24	17	.4
7.	++++	++++	30	30.5	.5
8.	++++	++++	36	13	2.1
9.	0	0	3	53	1.6
10.	++++	++++	24	20	.8
Mean	—	—	16.1	37.5	1.9
Range	—	—	3-36	13-85	.4-7.9
S.D.	—	—	11.98	25.7	2.26

Lobar Pneumonia

1.	0	0	12	10	.4
2.	+	++	5	9	.5
3.	+	++	14	24	.5
4.	+	+	6	9	.5
5.	+	+	7	10	.7
6.	0	+	7	7	.6
7.	+	+++	3	8	.4
8.	0	0	3	5.5	2.2
9.	0	+	8	6.5	.9
10.	+++	++++	9	7	.5
Mean	—	—	7.4	9.6	.72
Range	—	—	3-14	5.5-24	.4-2.2
S.D.	—	—	3.57	5.27	.55

Serum Proteins

Chemical Fractionation

Cirrhosis of the Liver

Case No.	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
1.	7.43	1.69	5.74	.29:1
2.	6.90	1.50	5.40	.28:1
3.	7.90	2.60	5.30	.49:1
4.	9.42	2.76	6.66	.41:1
5.	6.70	1.36	5.34	.25:1
6.	6.90	2.54	4.36	.58:1
7.	5.83	1.17	4.66	.25:1
8.	7.69	1.69	6.00	.28:1
9.	7.64	3.08	4.56	.68:1
10.	7.64	1.82	5.82	.31:1
Mean	7.40	2.02	5.38	.38:1
Range	5.83- 9.42	1.17- 3.08	4.36- 6.66	.25:1- .68:1
S.D.	.94	.66	.71	—

Primary Carcinoma of the Liver

1.	6.48	1.15	5.33	.22:1
2.	6.98	2.66	4.32	.62:1
3.	6.90	1.60	5.30	.30:1
4.	7.14	2.31	4.83	.48:1
5.	6.10	1.47	4.63	.32:1
6.	7.85	1.96	5.89	.33:1
7.	6.93	1.94	4.99	.39:1
8.	7.50	1.70	5.80	.29:1
9.	5.91	2.74	3.17	.86:1
10.	7.64	1.97	5.67	.35:1
Mean	6.94	1.95	4.99	.39:1
Range	5.91- 7.85	1.15- 2.74	3.17- 5.89	.22:1- .86:1
S.D.	.64	.51	.82	—

Lobar Pneumonia

Case No.	Total Protein	Ser.Albumin	Ser.Globulin	A/G Ratio
1.	5.95	1.90	4.05	.47:1
2.	7.10	2.61	4.49	.58:1
3.	7.85	2.62	5.23	.50:1
4.	6.61	2.79	3.82	.73:1
5.	6.84	2.63	4.21	.62:1
6.	7.11	2.44	4.67	.52:1
7.	5.90	2.24	3.66	.61:1
8.	5.91	2.19	3.72	.59:1
9.	7.12	2.42	4.70	.51:1
10.	7.02	2.85	4.17	.68:1
Mean	6.74	2.47	4.27	.58:1
Range	5.90-7.85	1.90-2.85	3.66-5.23	.47:1-.73:1
S.D.	.65	.29	.50	---

Paper Electrophoresis

Cirrhosis of the Liver

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	Alpha$_1$	Alpha$_2$	Beta	Gamma
1.	2.38	5.05	.47:1	.25	.49	.77	3.54
2.	2.32	4.58	.51:1	.22	.46	.61	3.29
3.	3.15	4.75	.66:1	.17	.42	.80	3.36
4.	3.37	6.05	.56:1	.44	.62	1.21	3.78
5.	1.39	5.31	.26:1	.21	.63	.99	3.48
6.	3.36	3.54	.95:1	.29	.55	.57	2.13
7.	1.70	4.13	.41:1	.22	.62	.73	2.56
8.	2.41	5.28	.46:1	.28	1.03	1.38	2.59
9.	3.50	4.14	.85:1	.34	.56	.92	2.32
10.	2.15	5.49	.39:1	.28	.69	.55	3.97
Mean	2.57	4.83	.53:1	.27	.61	.85	3.10
Range	1.39-3.50	3.54-6.05	.26:1-.95:1	.17-.44	.42-1.03	.55-1.38	2.13-3.97
S.D.	.74	.75	---	.08	.17	.28	.65

Primary Carcinoma of the Liver

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	Alpha$_1$	Alpha$_2$	Beta	Gamma
1.	1.97	4.51	.44:1	.50	.98	.84	2.19
2.	3.17	3.81	.83:1	.32	1.13	.81	1.55
3.	2.67	4.23	.63:1	.48	1.03	1.01	1.71
4.	2.63	4.51	.58:1	.41	.75	1.06	2.29
5.	1.77	4.33	.41:1	.51	1.16	.79	1.87
6.	2.55	5.30	.48:1	.45	.67	1.13	3.05
7.	2.16	4.77	.45:1	.48	.86	.85	2.58
8.	1.91	5.59	.34:1	.35	.60	.90	3.74
9.	2.85	3.06	.93:1	.31	.70	.73	1.32
10.	2.25	5.39	.42:1	.44	.89	.94	3.12
Mean	2.39	4.55	.53:1	.43	.88	.91	2.34
Range	1.77-3.17	3.06-5.59	.34:1-.93:1	.31-.51	.60-1.16	.73-1.13	1.32-3.74
S.D.	.45	.77	---	.07	.20	.13	.78

Lobar Pneumonia

1.	2.27	3.68	.62:1	.32	.73	.81	1.82
2.	3.09	4.01	.77:1	.45	1.09	.83	1.64
3.	2.92	4.93	.59:1	.46	1.12	.86	2.49
4.	2.93	3.68	.80:1	.48	.91	.78	1.51
5.	2.86	3.98	.72:1	.62	1.24	.79	1.33
6.	2.96	4.15	.71:1	.35	1.15	.91	1.74
7.	2.32	3.58	.65:1	.55	.97	.86	1.20
8.	2.19	3.72	.59:1	.58	1.27	.69	1.18
9.	2.91	4.21	.69:1	.43	.96	.79	2.03
10.	3.04	3.98	.76:1	.42	.95	.81	1.80
Mean	2.75	3.99	.69:1	.47	1.04	.81	1.67
Range	2.19-3.09	3.58-4.93	.59:1-.80:1	.32-.62	.73-1.27	.69-.91	1.18-2.49
S.D.	.35	.39	---	.10	.17	.06	.40

Cirrhosis of the Liver

Electrophoretic Pattern

Case No. 5

Total Protein	6.70
Serum Albumin	1.39
Serum Globulin	5.31
$Alpha_1$	.21
$Alpha_2$	.63
Beta	.99
Gamma	3.48
A/G Ratio	.26:1

Primary Carcinoma of the Liver

Case No. 4

Electrophoretic Pattern

Total Protein	7.14
Serum Albumin	2.63
Serum Globulin	4.51
$Alpha_1$	.41
$Alpha_2$	.75
Beta	1.06
Gamma	2.29
A/G Ratio	.58:1

Lobar Pneumonia

Case No. 5

Electrophoretic Pattern

Total Protein	6.84
Serum Albumin	2.86
Serum Globulin	3.98
$Alpha_1$	.62
$Alpha_2$	1.24
Beta	.79
Gamma	1.33
A/G Ratio	.72:1

APPENDIX III

STATISTICAL RESULTS

Student's 't' method for estimating the significance of differences between the means of various groups has been used for statistical analysis.

To obtain the standard deviation the following formula has been used:

$$S.D. = \sqrt{\frac{\Sigma(x - \bar{x})^2}{n - 1}}$$

To estimate 't' the following formula has been used:

$$s^2 = \frac{\Sigma(x - \bar{x})^2 + \Sigma(y - \bar{y})^2}{(m + n) - 2}$$

$$s = \sqrt{\frac{\Sigma(x - \bar{x})^2 + \Sigma(y - \bar{y})^2}{(m + n) - 2}}$$

$$t = \frac{\bar{x} - \bar{y}}{s\sqrt{\frac{1}{m-1} + \frac{1}{n-1}}}$$

where x and y are individual results, $\bar{x}$ and $\bar{y}$ the means of the series and m and n the number of individual results in each series.

P has been found by reference to Tables of 't'.
P values of $< 0.1\%$ are regarded as highly significant, $< 1\%$ as significant and $< 5\%$ as possibly significant.

Groups:

Group 1 = 25 Europeans
Group 2 = 50 African Male Nursing Staff
Group 3 = 50 Cato Manor Africans
Group 4 = 49 Patients with Amoebic Dysentery On Admission
Group 5 = 49 Patients with Amoebic Dysentery On Discharge
Group 6 = 31 Patients with Amoebic Liver Abscess On Admission
Group 7 = 31 Patients with Amoebic Liver Abscess On Discharge
Group 8 = 10 Patients with Cirrhosis of the Liver
Group 9 = 10 Patients with Primary Carcinoma of the Liver
Group 10 = 10 Patients with Right Basal Lobar Pneumonia

N S = Not Significant

	Groups Compared	't'	P	
Hb				
	1 & 2	2.999	< 1%	
	2 & 3	.986	> 25%	N S
	1 & 3	3.561	< .1%	
E.S.R.				
	1 & 2	5.029	< .1%	
	2 & 3	3.724	< .1%	
	1 & 3	7.132	< .1%	
P.C.V.				
	1 & 2	1.530	> 10%	N S
	2 & 3	.173	> 25%	N S
	1 & 3	1.634	> 10%	N S
M.C.H.C.				
	1 & 2	3.817	< .1%	
	2 & 3	.962	> 25%	N S
	1 & 3	4.973	< .1%	
Zn. Sulph. Turb.				
	1 & 2	4.810	< .1%	
	2 & 3	1.425	> 20%	N S
	1 & 3	6.432	< .1%	
Alk.Phosphatase				
	1 & 2	1.824	> 5%	N S
	2 & 3	4.761	< .1%	
	1 & 3	4.882	< .1%	
Ser.Bilirubin				
	1 & 2	1.823	> 5%	N S
	2 & 3	1.850	> 5%	N S
	1 & 3	3.946	< .1%	

	Groups Compared	't'	P	
Total Protein				
	1 & 2	2.287	< 5%	
	2 & 3	3.788	< .1%	
	1 & 3	.531	> 25%	N S
Chem. Fractionat.				
Ser. Albumin				
	1 & 2	.224	> 25%	N S
	2 & 3	7.567	< .1%	
	1 & 3	7.694	< .1%	
Ser. Globulin				
	1 & 2	4.307	< .1%	
	2 & 3	4.853	< .1%	
	1 & 3	7.707	< .1%	
Paper Electroph.				
Ser. Albumin				
	1 & 2	.592	> 25%	N S
	2 & 3	9.456	< .1%	
	1 & 3	7.997	< .1%	
Ser. Globulin				
	1 & 2	3.470	< .1%	
	2 & 3	6.761	< .1%	
	1 & 3	8.548	< .1%	
$Alpha_1$				
	1 & 2	1.613	> 10%	N S
	2 & 3	5.935	< .1%	
	1 & 3	2.632	< 2%	
$Alpha_2$				
	1 & 2	2.266	< 5%	
	2 & 3	7.005	< .1%	
	1 & 3	3.882	< .1%	
Beta				
	1 & 2	.773	> 25%	N S
	2 & 3	6.387	< .1%	
	1 & 3	.965	> 25%	N S
Gamma				
	1 & 2	6.893	< .1%	
	2 & 3	1.335	> 10%	N S
	1 & 3	8.342	< .1%	

	Groups Compared	't'	P	
Total Protein				
	4 & 5	4.667	< .1%	
	6 & 7	2.266	< 5%	
	3 & 4	4.355	< .1%	
	3 & 5	.565	> 25%	N S
	3 & 6	.273	> 25%	N S
	3 & 7	3.429	< .1%	
	4 & 6	2.999	< 1%	
	5 & 7	2.418	< 2%	
Chem. Fractionat.				
Ser. Albumin	4 & 5	5.425	< .1%	
	6 & 7	8.899	< .1%	
	3 & 4	7.560	< .1%	
	3 & 5	1.431	> 10%	N S
	3 & 6	18.143	< .1%	
	3 & 7	3.462	< .1%	
	4 & 6	4.951	< ,1%	
	5 & 7	1.520	> 10%	N S
Ser. Globulin				
	4 & 5	1.212	> 20%	N S
	6 & 7	3.146	< 1%	
	3 & 4	.420	> 25%	N S
	3 & 5	1.942	> 5%	N S
	3 & 6	9.809	< .1%	
	3 & 7	6.250	< .1%	
	4 & 6	8.082	< .1%	
	5 & 7	4.090	< .1%	
Paper Electroph.				
Ser. Albumin				
	4 & 5	6.844	< .1%	
	6 & 7	9.634	< .1%	
	3 & 4	8.891	< .1%	
	3 & 5	.849	> 25%	N S
	3 & 6	13.450	< .1%	
	3 & 7	1.884	> 5%	N S
	4 & 6	4.389	< .1%	
	5 & 7	.677	> 25%	N S

Amoebic Liver Abscess On Discharge

Case No.	Ser. Alb.	Ser. Glob.	A/G Ratio	Alpha$_1$	Alpha$_2$	Beta	Gamma
1.	3.77	3.33	1.13:1	.26	.49	.75	1.83
2.	4.05	3.08	1.31:1	.23	.70	.74	1.41
3.	3.75	3.85	.97:1	.19	.77	.62	2.27
4.	3.91	4.87	.80:1	.28	.48	1.04	3.07
5.	2.94	3.75	.78:1	.19	.60	.58	2.38
6.	3.25	4.50	.72:1	.23	.61	1.02	2.64
7.	3.15	4.79	.66:1	.37	.57	.84	3.01
8.	3.09	3.07	1.00:1	.26	.68	.74	1.39
9.	2.93	3.44	.85:1	.27	.68	.71	1.78
10.	3.11	4.46	.70:1	.39	.89	1.12	2.06
11.	3.77	3.93	.96:1	.32	.84	1.11	1.66
12.	3.04	3.98	.76:1	.36	.91	.76	1.95
13.	3.09	4.94	.63:1	.23	.99	1.48	2.24
14.	3.24	4.22	.77:1	.30	.81	1.16	1.95
15.	3.66	3.83	.96:1	.35	.65	.89	1.94
16.	3.39	4.41	.77:1	.48	.94	1.05	1.94
17.	3.87	4.58	.84:1	.46	.97	1.11	2.04
18.	3.53	6.12	.58:1	.33	.81	.99	3.99
19.	3.48	4.32	.81:1	.41	.87	.87	2.17
20.	3.40	4.02	.85:1	.27	.82	.91	2.02
21.	3.62	4.01	.90:1	.34	.89	.79	1.99
22.	3.33	3.72	.90:1	.28	.69	.97	1.78
23.	3.23	4.07	.79:1	.31	.72	.91	2.13
24.	3.45	4.87	.71:1	.27	.76	1.02	2.82
25.	2.98	4.57	.65:1	.34	.99	.84	2.40
26.	3.42	4.04	.85:1	.26	.70	.90	2.18
27.	3.55	3.19	1.11:1	.21	.63	.81	1.54
28.	3.66	4.10	.89:1	.31	.76	.90	2.13
29.	3.33	4.16	.80:1	.28	.71	.97	2.20
30.	3.53	4.60	.77:1	.35	.89	.74	2.62
31.	2.96	4.15	.71:1	.42	.77	.95	2.01
Mean	3.40	4.16	.82:1	.31	.76	.91	2.18
Range	2.93-4.05	3.07-6.12	.58:1-1.31:1	.19-.48	.48-.99	.58-1.48	1.39-3.99
S.D.	.31	.63	——	.07	.14	.18	.53

	Groups Compared	't'	P	
Ser. Globulin	4 & 5	.517	> 25%	N S
	6 & 7	3.425	< 1%	
	3 & 4	.914	> 25%	N S
	3 & 5	1.625	> 10%	N S
	3 & 6	9.347	< .1%	
	3 & 7	5.143	< .1%	
	4 & 6	7.385	< .1%	
	5 & 7	3.590	< .1%	
$Alpha_1$				
	4 & 5	4.456	< .1%	
	6 & 7	7.812	< .1%	
	3 & 4	3.729	< .1%	
	3 & 5	1.810	> 5%	N S
	3 & 6	9.830	< .1%	
	3 & 7	1.614	> 10%	N S
	4 & 6	4.812	< .1%	
	5 & 7	0	0	N S
$Alpha_2$				
	4 & 5	4.105	< .1%	
	6 & 7	7.269	< .1%	
	3 & 4	4.102	< .1%	
	3 & 5	.337	> 25%	N S
	3 & 6	5.684	< .1%	
	3 & 7	1.752	> 5%	N S
	4 & 6	10.574	< .1%	
	5 & 7	1.114	> 25%	N S
Beta				
	4 & 5	4.112	< .1%	
	6 & 7	1.482	> 10%	N S
	3 & 4	5.394	< .1%	
	3 & 5	1.303	> 10%	N S
	3 & 6	2.868	< 1%	
	3 & 7	.609	> 25%	N S
	4 & 6	1.698	> 5%	N S
	5 & 7	.302	> 25%	N S

	Groups Compared	't'	P	
Gamma	4 & 5	2.256	< 5%	
	6 & 7	1.179	> 20%	N S
	3 & 4	.356	> 25%	N S
	3 & 5	2.307	< 5%	
	3 & 6	6.746	< .1%	
	3 & 7	6.532	< .1%	
	4 & 6	6.381	< .1%	
	5 & 7	4.390	< .1%	
Total Protein	8 & 9	1.277	> 20%	N S
	9 & 10	.697	> 25%	N S
	8 & 10	1.824	> 5%	N S
	3 & 8	1.409	> 10%	N S
	6 & 8	.909	> 25%	N S
	3 & 9	1.264	> 20%	N S
	6 & 9	.521	> 25%	N S
	3 & 10	2.516	< 2%	
	6 & 10	1.171	> 25%	N S
Chem.Fractionat. Ser. Albumin	8 & 9	.265	> 25%	N S
	9 & 10	2.808	< 2%	
	8 & 10	1.964	> 5%	N S
	3 & 8	9.351	< .1%	
	6 & 8	.303	> 25%	N S
	3 & 9	11.073	< .1%	
	6 & 9	.055	> 25%	N S
	3 & 10	7.540	< .1%	
	6 & 10	3.029	< 1%	
Ser. Globulin	8 & 9	.113	> 25%	N S
	9 & 10	2.369	< 5%	
	8 & 10	4.026	< .1%	
	3 & 8	9.646	< .1%	
	6 & 8	.884	> 25%	N S
	3 & 9	6.667	< .1%	
	6 & 9	.535	> 25%	N S
	3 & 10	2.653	< 2%	
	6 & 10	2.613	< 2%	

Groups Compared	't'	P		
Paper Electroph. Ser. Albumin				
8 & 9	.656	>	25%	N S
9 & 10	1.997	>	5%	N S
8 & 10	.698	>	25%	N S
3 & 8	7.308	<	.1%	
6 & 8	1.160	>	25%	N S
3 & 9	10.992	<	.1%	
6 & 9	.369	>	25%	N S
3 & 10	8.140	<	.1%	
6 & 10	2.352	<	5%	
Ser. Globulin				
8 & 9	.823	>	25%	N S
9 & 10	2.051	>	5%	N S
8 & 10	3.127	<	1%	
3 & 8	8.485	<	.1%	
6 & 8	.176	>	25%	N S
3 & 9	6.449	<	.1%	
6 & 9	.808	>	25%	N S
3 & 10	3.289	<	1%	
6 & 10	3.041	<	1%	
$Alpha_1$				
8 & 9	4.718	<	.1%	
9 & 10	1.042	>	25%	N S
8 & 10	5.145	<	.1%	
3 & 8	3.100	<	1%	
6 & 8	6.248	<	.1%	
3 & 9	6.285	<	.1%	
6 & 9	1.495	>	20%	N S
3 & 10	7.821	<	.1%	
6 & 10	.285	>	25%	N S

Groups Compared	't'	P	
$Alpha_2$			
8 & 9	3.285	< 1%	
9 & 10	1.973	> 5%	N S
8 & 10	5.720	< .1%	
3 & 8	2.981	< 1%	
6 & 8	6.617	< .1%	
3 & 9	3.544	< .1%	
6 & 9	2.740	< 1%	
3 & 10	7.789	< .1%	
6 & 10	.566	> 25%	N S
Beta			
8 & 9	.624	> 25%	N S
9 & 10	2.246	< 5%	
8 & 10	.449	> 25%	N S
3 & 8	1.527	> 10%	N S
6 & 8	0	0	N S
3 & 9	.348	> 25%	N S
6 & 9	1.250	> 20%	N S
3 & 10	3.203	< 1%	
6 & 10	.911	> 25%	N S
Gamma			
8 & 9	2.377	< 5%	
9 & 10	2.422	< 5%	
8 & 10	5.944	< .1%	
3 & 8	13.055	< .1%	
6 & 8	2.834	< 1%	
3 & 9	5.582	< .1%	
6 & 9	.112	> 25%	N S
3 & 10	.450	> 25%	N S
6 & 10	2.890	< 1%	

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BRIEF SUMMARY OF THESIS

SUBMITTED FOR THE DEGREE OF DOCTOR OF MEDICINE

BY

S.J. POWELL, M.B.,Ch.B.(Cape Town), M.R.C.P.(Ed.)

The haematological findings, serum proteins and liver function tests in a group of Natal African labourers from Cato Manor are compared with those in African Male Nurses and in Europeans. The findings are also compared with those reported elsewhere in Africa, and the possible causes of the differences discussed.

The pathological and clinical evidence of amoebic hepatitis is reviewed and compared with the disease as it is seen in Durban Africans.

The haematological findings, serum proteins and liver function tests in Natal Africans with amoebic dysentery and with amoebic liver abscess are compared with each other and with those of Cato Manor Africans. The literature is

reviewed and the significance of the findings discussed.

The changes in amoebic liver abscess are compared with those found in cirrhosis of the liver, primary carcinoma of the liver and right basal lobar pneumonia.

30th January, 1958.

Details of Published Work

By
S.J. Powell

1. Diphetarsone in the Treatment of Acute Amoebic Dysentery
(In Association with A.J.Wilmot & R.Elsden-Dew)
J.Trop.Med. & Hyg. 1957:60, 16.

2. A Comparison of Erythromycin, Spiramycin & Novobiocin in the Treatment of Acute Amoebic Dysentery.
(In Association with A.J.Wilmot & R.Elsden-Dew)
J.Trop.Med. & Hyg. 1958:61,58.

3. The Comparative Value of Emetine and Chloroquine in Amoebic Liver Abscess.
(In Association with A.J.Wilmot & E.B.Adams)
Am.J.Trop.Med. & Hyg. 1958:7,197.

4. Diverticulum of the Left Ventricle
Amer.Heart Journal 1958:55,518.

5. Erythromycin in Amoebic Liver Abscess
(In Association with A.J.Wilmot & R.Elsden-Dew)
Am.J.Trop.Med. & Hyg. 1959:8,pp. not available.

The following papers have been accepted for publication and are at present in the press.

1. Unexplained Peculiarities in the African Electrocardiogram
Brit.Heart Journal.

2. Hepatic Amoebiasis
(In Association with A.J.Wilmot & R.Elsden-Dew)
Trans.Roy.Soc.Trop.Med. & Hyg.

3. The Serum Protein Pattern & Liver Function Tests in the Natal African.
S.Afr.J.Lab. & Clin.Med.

2/...

4. The Serum Protein and Liver Function Tests in Acute Amebic Dysentery and Amebic Liver Abscess.
Am.J.Trop.Med. & Hyg.

5. The Serum Protein Pattern and Liver Function Tests in the Differential Diagnosis of Amebic Liver Abscess.
Am.J.Trop.Med. & Hyg.

DIVERTICULUM OF THE LEFT VENTRICLE: CASE REPORT WITH SPECIAL REFERENCE TO ELECTROCARDIOGRAPHIC FINDINGS

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Durban, South Africa

From the Department of Medicine, University of Natal

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Diverticulum of the Left Ventricle: Case Report With Special Reference to Electrocardiographic Findings

S. J. Powell, M.B., M.R.C.P. (Edin.), Durban, South Africa

The subject of diverticulum of the left ventricle of the heart was reviewed, in 1951, by Skapinker,[1] who also reported the successful removal of a diverticulum in an infant. Of the 13 cases he found in the literature, only one survived infancy without surgical resection, and this one died as a result of rupture of the diverticulum at the age of 4 years. Since then, Parsons,[2] in addition to describing a case of ventricular extension into the abdominal wall in an infant, has drawn attention to a similar case previously mentioned by him, and to cases described by Formijne[3] and Snellen and associates.[4] In all these latter cases evidence of associated congenital cardiac abnormalities was present. The following is the report of a ventricular diverticulum in a young adult without disability or other discernible congenital defects in the cardiovascular system.

CASE REPORT

A male Bantu, aged 17 years, came to Durban to seek work. On registering for employment he was seen by the medical officer and referred to hospital for investigation. He was symptom-free but had had a swelling in the upper abdomen for as long as he could remember. This had slowly enlarged as he grew older. Confirming this history, his mother said that the swelling was present at birth and had increased steadily in size.

On examination there was a swelling in the epigastric region 2 inches above the umbilicus (Fig. 1). It was 1 inch wide by 2.5 inches long, somewhat pear-shaped and hanging vertically downward. A large tortuous, pulsating vessel passed down from beneath the xiphisternum to become continuous with the base of the swelling, while the base of the tumor was attached to the umbilicus by a nonpulsating cordlike structure. The swelling appeared to be just under, but not attached to, the skin. There was marked systolic expansile pulsation. However, when the superior feeding vessel was obliterated by manual compression, the tumor became smaller, and slight, independent systolic contraction could be seen. On compressing the tumor itself, premature beats occurred both in the mass and the heart. Systolic and diastolic thrills could be felt over the feeding vessel and tumor. A soft diastolic murmur was present at the cardiac apex, and, as this was traced down to the feeding vessel, it became progressively louder and rougher, and to it became added a loud, rough systolic murmur.

The rest of the cardiovascular system was normal. The apex beat was in the fifth left intercostal space 3 inches from the midline. The heart sounds at the base were normal and closed.

From the Department of Medicine, University of Natal, Durban, South Africa.
Received for publication Oct. 25, 1957.

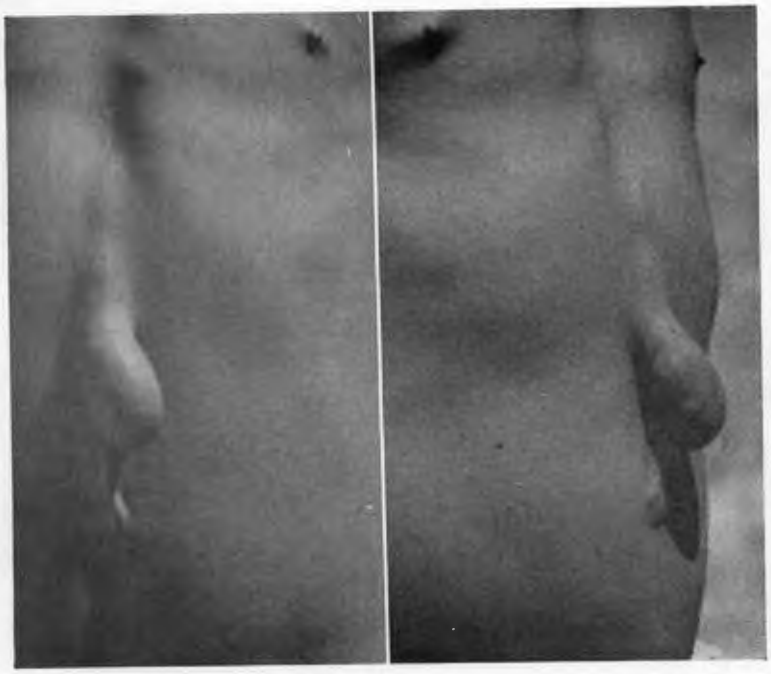

Fig. 1.—The patient with diverticulum protruding in epigastric region.

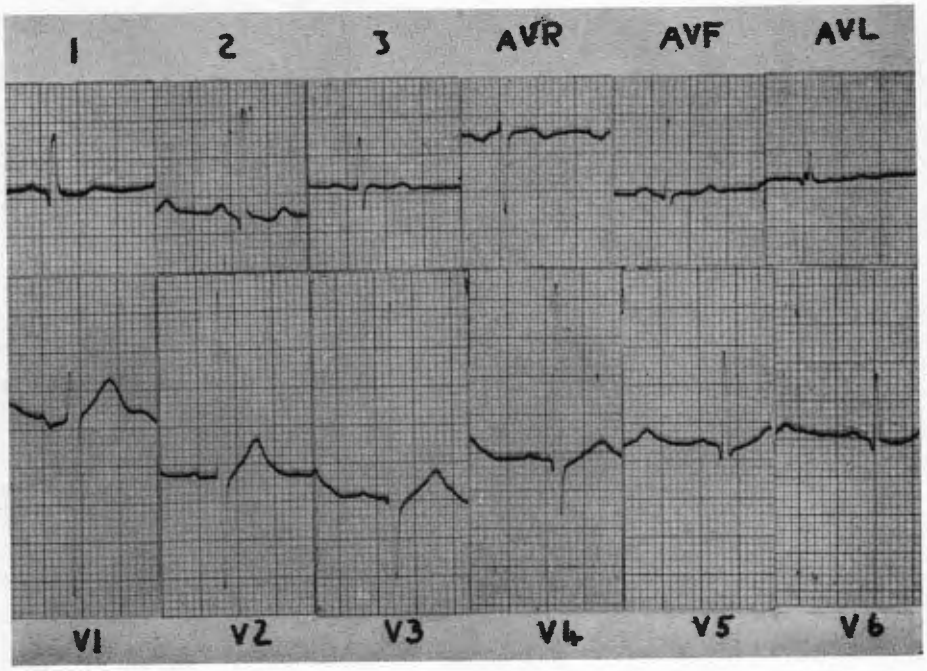

Fig. 2.—Standard electrocardiogram showing no abnormality.

The pulse rate was 86 per minute. It was regular and not collapsing. The blood pressure was: left arm 115/70 mm. Hg, right arm 95/70 mm. Hg, left leg 120/75 mm. Hg, and right leg 130/85 mm. Hg. Physical development was average, and no abnormality was found in any other system.

Radiologic Investigations.—Straight x-ray and screening of the heart showed that the heart was not enlarged, but there was a prominent, vigorously pulsatile pulmonary outflow tract. A soft tissue swelling could be seen overlying the abdomen.

Angiography: At first a polythene catheter was passed down into the channel connected to the pouch, and 50 c.c. of 70 per cent diodone was injected. This demonstrated the sac, and

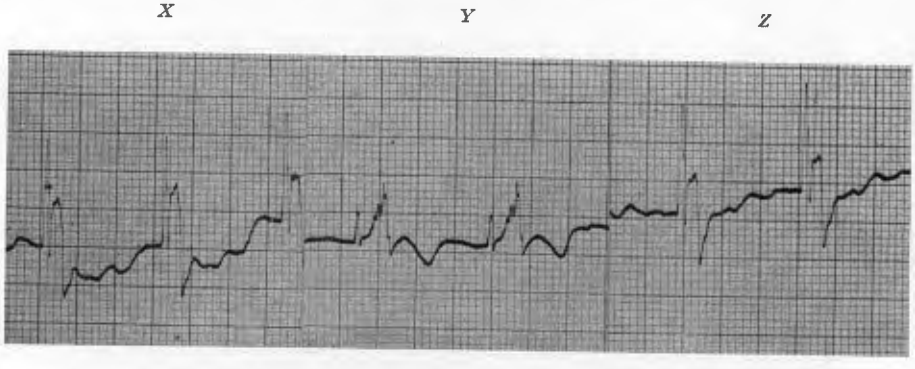

Fig. 3.—*X* and *Z* electrode on either side, adjacent to mass. *Y* electrode on mass. Double QRS complexes present.

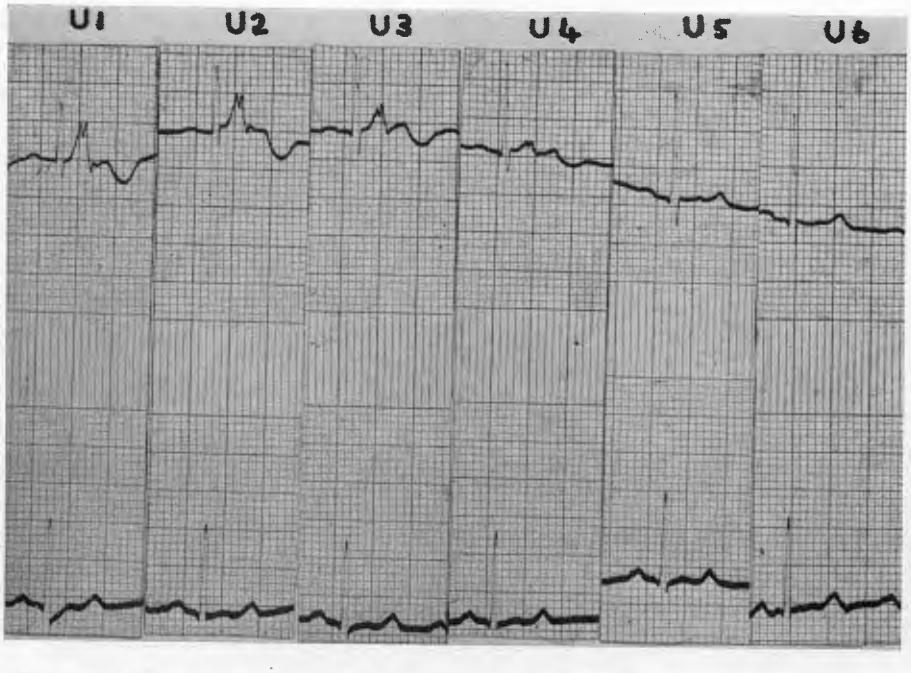

Fig. 4.—Two-channel electrocardiogram consisting of Lead II and unipolar Leads U1-U6 taken successively in a line from a point adjacent to the mass to the cardiac apex. These show a double QRS complex in U1 which gradually diminishes in amplitude until it is absent in U6.

a vertical channel above it, apparently with a double lumen, some 12 cm. long. A further angiogram was performed with the catheter passed upward in the channel connected to the pouch, and 60 c.c. of 70 per cent diodone was injected. The catheter passed into the left ventricle, and the left ventricle, aorta and its branches were outlined. The channel appeared to enter the left ventricle at, or near, its apex.

Although surgery was advised, the patient refused to remain in hospital, and returned to his home in the country. He has not attended for follow-up.

DISCUSSION AND ELECTROCARDIOGRAPHIC FINDINGS

An electrocardiogram using the standard and routine chest leads showed no abnormality, but with leads taken adjacent to and directly over the mass a double QRS complex was seen (Figs. 2 and 3).

A two-channel electrocardiogram using Lead II in one channel, and a unipolar lead placed at 6 sites (U1—U6) taken successively in a line leading from the mass to the cardiac apex showed the following: (1) The initial complex corresponded in timing, in expected contour, and in elevation with the normal

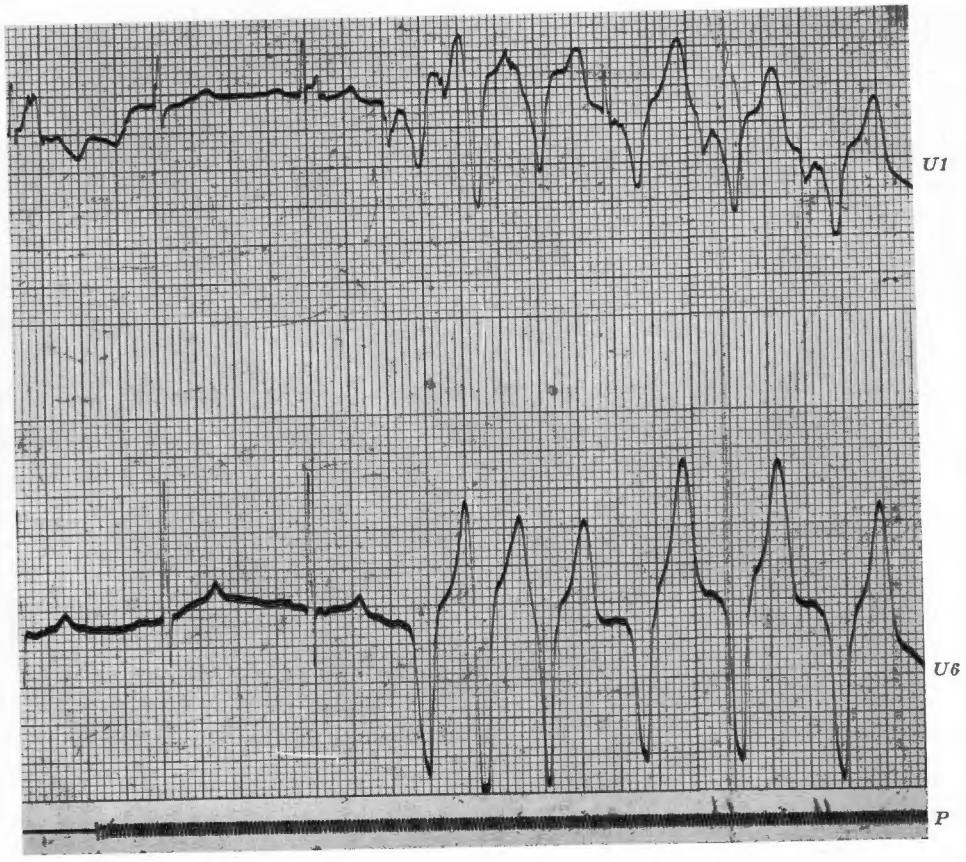

Fig. 5.—Two-channel electrocardiogram with pressure applied to mass. The premature beats show double, retrograde QRS complexes in U1, and single, retrograde complexes occurring at least 0.08 second later in U6. (P = pressure.)

cardiac QRS. (2) It was followed immediately by a broadened and notched QRS complex ending at least 0.12 second after the normal QRS. An inverted T wave followed. (3) As the leads progressed further from the mass toward the cardiac apex there was a gradual diminution in amplitude of the additional QRS until it was completely absent in U6 (Fig. 4).

The electrocardiographic findings indicated the presence of actively depolarizing and repolarizing muscle in the tumor wall.

A continuous electrocardiographic strip taken with premature systoles being produced by compressing the tumor revealed the following (Fig. 5):

In Lead U1 double retrograde QRS complexes were seen, while the corresponding complexes in U6 were single and retrograde. The onset in U1 was at least 0.08 second earlier than the apparent onset in U6 (which was sufficiently remote from the tumor not to pick up its potential). This apparent delay could be explained only by assuming that an ectopic focus in the tumor stimulated the heart muscle by retrograde spread of the depolarization wave from the tumor via a connecting cardiac muscular channel to the heart muscle, the apparent delay representing the time taken for the wave to spread from the tumor up to the heart. This clearly indicated that the muscle in the tumor was part of the normal cardiac syncytial mass.

SUMMARY

1. A case of diverticulum of the left ventricle of the heart in a male Bantu, aged 17 years, is described.

2. The electrocardiographic findings are discussed. These clearly show the presence of an ectopic focus in the tumor, capable of initiating impulses and conducting them to the heart via a connecting cardiac muscular channel. This finding was of considerable value in diagnosis.

Thanks are due to Dr. A. J. Wilmot for allowing me to study this patient who was under his care, to Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, for permission to publish this case, and to Dr. L. Norris for his help with the two-channel electrocardiograms.

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THE COMPARATIVE VALUE OF EMETINE AND CHLOROQUINE IN AMEBIC LIVER ABSCESS

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Since Conan (1948) showed that chloroquine was effective in the treatment of hepatic amebiasis, the activity of the drug has been confirmed by numerous reports, and many now believe that chloroquine is the drug of choice in this condition, chiefly because it is held to be as efficacious as emetine hydrochloride but less toxic (Hoekenga and Maximo, 1950; Patterson and Lawlis, 1956). Few comparative studies have been made. Harinasuta (1951), however, has compared emetine and chloroquine in two groups of 45 patients suffering from amebic liver abscess and concluded that "chloroquine appeared to possess nearly as much curative value as emetine in the treatment of amebic liver abscess. It might be somewhat inferior on the point of relapse rate."

The purpose of the present study is to assess the relative efficacy of emetine and chloroquine in the treatment of amebic liver abscess.

MATERIAL AND METHODS

Alternate male African patients diagnosed as suffering from amebic liver abscess were given emetine or chloroquine. The diagnosis was established in all cases by aspiration within three days of admission, and in all the pus removed was characteristic and bacteriologically sterile. The finding of an associated disease excluded a patient from the series. All patients were kept under observation in hospital for 30 days and thereafter they were asked to report for follow-up at 1 month, 3 months and 6 months after discharge.

Emetine hydrochloride was given by intramuscular injection in a daily dose of 1 grain. Each patient received two courses, the first lasting 10 days and the second 6 days, with an interval of 14 days between courses. Chloroquine was given as the diphosphate (250 mgm. tablets equivalent to 150 mgm. of the base) or the sulphate (200 mgm. tablets equivalent to 150 mgm. of the base) in a dose of 4 tablets immediately, followed by 2 tablets 6 hours later and then 1 tablet twice daily for 29 days. In addition, all patients received 600 mgm. diiodohydroxyquinoline thrice daily for 20 days.

When there was active amebic dysentery in addition to liver abscess in cases receiving chloroquine, we added tetracycline in a dosage of 1 gram daily for 10 days. We did this because we felt that treatment with chloroquine and diiodohydroxyquinoline is inadequate for active bowel disease, and because we believe that tetracycline is ineffective in amebic liver abscess (Wilmot *et al.*, 1952; Wilmot, 1956). At the outset it was decided that both emetine and chloroquine would be given if a patient did not respond or deteriorated on treatment with one of these drugs alone, and that if a patient relapsed after leaving hospital he would be given the drug he had not previously received.

The series consisted of 19 patients treated with emetine, 5 of whom had amebic dysentery as well as liver abscess, and 16 treated with chloroquine 3 of whom had associated dysentery.

RESULTS

One patient treated with chloroquine was given emetine after 10 days as it was felt that he was deteriorating clinically and that it was unjustifiable to continue with the single drug. A further case in the chloroquine series had not responded fully after 32 days and was then given emetine. Both recovered satisfactorily.

Apart from these 2 cases there was no apparent difference in the initial response to these two drugs. The relapse rate however was significantly different. In the emetine series there were no relapses, but among the patients treated with chloroquine, 4 returned within 6 weeks with symptoms and signs of liver abscess and pus was obtained at aspiration. These 4 patients were treated with emetine with a satisfactory result.

As is usual with African patients the follow up was incomplete. In the chloroquine series, apart from the relapses, 4 patients reported at 1 month

[1] The Amoebiasis Research Unit is sponsored by the following bodies: The South African Council for Scientific and Industrial Research, The Natal Provincial Administration, The University of Natal, The United States Public Health Service (Grant E/1592).

and one of these again at 3 months. They were assessed as "cures". In the emetine series 11 patients returned at 1 month, 4 again at 3 months and 2 again at 6 months. All were well.

Apart from a moderate rise in pulse rate in some of the patients treated with emetine no toxic reactions were encountered with either drug.

DISCUSSION

Amebic liver abscess is a dangerous condition which may terminate fatally, usually by rupture of the abscess into adjoining viscera or from secondary bacterial infection. For this reason treatment should not only be effective in immediate control but reduce the incidence of relapse to a minimum.

In the doses used in this trial, chloroquine appears to be as effective as emetine in the alleviation of the initial symptoms, but it carries the risk of a significant relapse rate which we did not find with emetine. Although the follow up obtained was unsatisfactory, particularly in the chloroquine series, this does not invalidate the results as the African is reluctant to return to hospital when he is well. However, if symptoms recur, he is likely to return to this hospital as there is no alternative free medical service in the area.

The growing preference for chloroquine in the treatment of hepatic amebiasis is largely based on the fear of toxic reactions to emetine, but provided this drug is not used in patients with heart lesions and the dose is reduced in the young and the aged, we have not experienced serious toxic reactions in these or other patients who number many hundreds. Two courses of emetine were used because it has been shown that a single 10-day course carries a relapse rate of 7.7 per cent which a second course reduces to less than 1 per cent (Wilmot, 1949). The dose of chloroquine was that usually recommended in the literature, and it is felt that the period of administration was adequate.

We think that there are grounds for caution in recommending the substitution of chloroquine for emetine in the treatment of amebic liver abscess, and that the disease represents a greater risk to the patient than the possible occurrence of emetine toxicity. Chloroquine might prove more effective in a higher dose, but Wilkinson (1953) has reported a number of side effects when 450 mgm. of the base were given daily. It might also be possible to achieve satisfactory results by combining emetine and chloroquine and so reduce the total emetine dosage.

SUMMARY

Patients in whom the presence of an amebic liver abscess was confirmed by the aspiration of characteristic pus were treated in two groups with emetine or chloroquine. Of 19 cases given emetine all responded well initially and none relapsed. Sixteen patients were treated with chloroquine. Initial response was poor in 2 and they were given emetine with a satisfactory result. Four of the remaining 14 patients returned within six weeks with a recurrence of the abscess.

ACKNOWLEDGMENT

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A Comparison of Erythromycin, Spiramycin and Novobiocin in the Treatment of Acute Amoebic Dysentery

By S. J. POWELL, A. J. WILMOT and R. ELSDON-DEW

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In recent years antibiotics have found an established place in the treatment of acute amoebic dysentery. Of those tested by this Unit the tetracyclines have been the most effective (Armstrong *et al.* 1950, Elsdon-Dew *et al.* 1952, Elsdon – Dew 1954 – 1955, and Wilmot 1955–1956). This paper reviews the results of clinical trials with three of the newer antibiotics.

Material and Methods

All patients placed on trial were African males complaining of diarrhoea with blood and mucus, showing ulceration on sigmoidoscopy, and with actively motile *Entamoeba histolytica* in the stools and ulcer scrapings. Severely ill patients and those with complications or co-existing disease were excluded.

Sigmoidoscopy was performed on admission, and subsequently on the 5th, 10th, 20th and 27th days after commencing treatment. When patients appeared to be deteriorating clinically, or when ulcers failed to heal at the end of treatment, daily stool examinations and more frequent sigmoidoscopy were done.

Results were classified into the following categories:—

i. *Success:* Symptom-free, ulcers healed and no parasites demonstrated.
ii. *Probable Failure:* Persistent open ulceration, but no parasites demonstrable in in stools or scrapings.
iii. *Absolute Failure:* Trophozoites still present, with or without open ulceration.
iv. *Failure with cysts:* Patient symptom-free and ulcers healed, but with cysts of *E. histolytica*.

Cases remaining or becoming absolute failures following completion of their course of the test drug (i.e. after 10 days) were removed from trial at this point, and given other treatment.

Patients still classified as probable failure at 27 days were usually given other treatment.

In the assessment at follow-up, the term recurrence is used in preference to relapse in view of the possibility of reinfection.

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Results

Erythromycin

The dosage given was 500 mg. 6-hourly for 10 days.

One patient vomited every time he was given erythromycin. After three days the drug was discontinued and the vomiting ceased. He responded satisfactorily to other treatment. No other cases showed toxic manifestations.

Table I shows that by the 27th day no patients had trophozoites in their stools, although two (10%), who were symptom-free, still harboured parasites, as cysts were present in their stools. A further three patients (14%) had open ulceration without demonstrable parasites. The remaining 15 cases (71%) appeared to be cured.

Follow-up was incomplete, but one month after discharge eight of the 15 patients classified as "success" returned, and of these six re-

TABLE I. RESULTS OF TREATMENT WITH ERYTHROMYCIN 500 MG. 6 HOURLY FOR 10 DAYS IN 21 CASES OF ACUTE AMOEBIC DYSENTERY.

Days after start of Therapy	*10*	*15*	*20*	*27*	*Cumulative 27 days*
Assessment	*No. of Cases*	*No. of Cases*	*No. of Cases*	*No. of Cases*	*No. of Cases*
Success	11=52%	16=76%	18=86%	15=71%	15=71%
Probable Failure	8=38%	3=14%	1= 5%	3=14%	3=14%
Absolute Failure	1= 5%	1= 5%	1= 5%	—	—
Failure with cysts	—	—	—	2=10%	2=10%

TABLE II. RESULTS OF TREATMENT WITH SPIRAMYCIN 250 MG. 6 HOURLY FOR 10 DAYS IN 30 CASES OF ACUTE AMOEBIC DYSENTERY.

Days after start of Therapy	*10*	*15*	*20*	*27*	*Cumulative 27 days*
Assessment	*No. of Cases*	*No. of Cases*	*No. of Cases*	*No. of Cases*	*No. of Cases*
Success	10=33%	18=60%	23=80%	25=83%	25=83%
Probable Failure	20=67%	12=40%	3=10%	2= 7%	2= 7%
Absolute Failure	—	—	1= 3%	—	1= 3%
Failure with cysts	—	—	2= 7%	1= 3%	1= 3%

mained " successes," but two had open rectal ulceration with trophozoites of *E. histolytica*. In addition two patients classified as " failure with cysts " returned, and both stool examinations and sigmoidoscopy were normal. One month later four of the eight remaining " successes " returned for follow-up, and continued as " successes." A further month later, i.e., three months after discharge, three of these four " successess " returned, two continuing to be " successes," but one now had open rectal ulceration and trophozoites. This represents a known recurrence rate of 18% within three months of discharge.

Spiramycin

The dosage given was 250 mg. 6-hourly for 10 days. Two patients developed itching, papular rashes on the trunk whilst on treatment. These were not severe, and cleared up shortly after treatment was completed. No other clinical side-effects were seen.

One case developed signs of liver abscess on the 18th day and, although at this stage he was a " success " from the intestinal standpoint, he was removed from the trial as no longer conforming to our criteria.

It can be seen from Table II that at the 27th day, one case (3%) showed open ulceration with trophozoites; another patient, although symptom-free, had cysts. A further two cases (7%) still had rectal ulceration, although parasites could not be found and the patients were symptom-free. The remaining 25 cases (83%) appeared to be cured.

Follow-up was incomplete, but one month after discharge 14 of the 25 patients classified as " successes " returned; of these 12 remained " successes," one had open ulceration with trophozoites and another had dysenteric symptoms with open ulceration, although *E. histolytica* was not found. In addition, one patient who was classified as " failure with cysts " returned after one month, when cysts were still found, and a further patient, regarded as a " probable failure " at 27 days, continued to have open rectal ulceration, without *E. histolytica* being found. One month later eight of the 12 remaining " successes " re-attended, seven continuing as " successes," but one was found to have open ulceration with trophozoites. Five of these seven " successes " returned for a final check one month later, and of these four remained " successes," but in one trophozoites were now present. This represents a known recurrence rate of 18% within three months of discharge.

Novobiocin

Two dosage schedules for novobiocin were used, one consisting of 250 mg. 6-hourly and the other of 500 mg. 6-hourly, both for 10 days. Cases were allocated alternately so that 10 patients were on the 250 mg. schedule and nine received the 500 mg. schedule. As the results for the two groups were almost identical, they have been combined for this study.

During treatment one patient receiving the smaller dosage developed a mild, generalised, itching papular rash which cleared up shortly after the course was completed. No other clinical side-effects were noted.

Table III shows that at 27 days nine cases (48%) had or had had open ulceration and trophozoites in the stools despite the novobiocin course, three more cases (16%) had persistent rectal ulceration without demonstrable parasites, and one further case (5%) was removed from the series at 20 days and given other treatment owing to persistent open ulceration and the presence of cysts. The remaining six cases (32%) appeared to be cured.

At one month follow-up, five out of the six cases classified as " successes " returned, and of these, two remained as " successes," one had open rectal ulceration with trophozoites, one had open rectal ulceration but no demonstrable parasites; and in one case cysts of *E. histolytica* were present. In addition one patient, classified as a " probable failure " at 27 days, returned one month after discharge with open ulceration and trophozoites. One month later the two remaining successes re-attended, and were still classified as " successes." One of these two returned for a final check one month later, by which time open ulceration and trophozoites

TABLE III. RESULTS OF TREATMENT WITH NOVOBIOCIN 250–500 MG. 6 HOURLY FOR 10 DAYS IN 19 CASES OF ACUTE AMOEBIC DYSENTERY.

Days after start of Therapy	*10*	*15*	*20*	*27*	*Cumulative 27 days*
Assessment	*No. of Cases*	*No. of Cases*	*No. of Cases*	*No. of Cases*	*No. of Cases*
Success	2=10%	5=26%	4=21%	6=32%	6=32%
Probable Failure	12=63%	6=32%	5=26%	3=16%	3=16%
Absolute Failure	4=21%	1= 5%	4=21%	—	9=48%
Failure with cysts	1= 5%	3=16%	1= 5%	—	1= 5%

were once more present. This represents a known recurrence rate of 50% within three months of discharge.

Discussion

Several reports on the use of erythromycin in acute amoebic dysentery have appeared in the literature, and it has been shown to produce satisfactory results, although a significantly high relapse rate is reported (Steigmann *et al.* 1953, Halawani *et al.* 1955, Jung *et al.* 1955, Shafei 1955, Villarejos 1955, and Nor El-Din 1956).

Reports on spiramycin are fewer, although as this drug possesses a similar anti-bacterial spectrum to erythromycin it is to be expected that results in acute amoebic dysentery would be comparable. Satisfactory immediate results have been reported, but with a high relapse rate (Charmot and Delahousse 1956). Bonan *et al.* (1956) using a higher dosage of 3 grammes daily obtained satisfactory immediate cure and a much reduced relapse rate.

Novobiocin has a different anti-bacterial spectrum, and might be expected to produce different results. McHardy *et al.* (1956) obtained a relatively poor cure rate, with a high incidence of side-effects, chiefly skin rashes.

A correct assessment of these drugs can only be made by comparison with those antibiotics already tested.

TABLE IV. RESULTS OF TREATMENT OF ACUTE AMOEBIC DYSENTERY WITH ANTIBIOTICS. ASSESSMENT AT 27 DAYS.

Antibiotic	*No. of Cases*	*Successes*	*Combined Failure Rate*
Tetracycline	36	35 (97%)	1 (3%)
Chlortetracycline	52	48 (92%)	2 (4%)
Oxytetracycline	49	45 (92%)	4 (8%)
Erythromycin	21	15 (71%)	2 (10%)
Spiramycin	30	25 (83%)	2 (7%)
Novobiocin	19	6 (32%)	10 (53%)

Table IV illustrates our results with erythromycin, spiramycin and novobiocin compared with previous trials of tetracycline, chlortetracycline, and oxytetracycline (Elsdon-Dew *et al.* 1952, Wilmot 1955-1956). These results are strictly comparable as our methods of selection and assessment have not varied.

Under the heading "Combined Failure Rate" we have included all those in whom either trophozoites or cysts have been found at 27 days. Table IV shows that erythromycin and spiramycin are both effective in the treatment of acute amoebic dysentery, although probably less so than the tetracyclines. Novobiocin, on the other hand, is relatively ineffective, as less than a third of the cases could be regarded as "successes" 27 days after commencing treatment.

TABLE V. RESULTS OF TREATMENT OF ACUTE AMOEBIC DYSENTERY WITH ANTIBIOTICS. FOLLOW-UP AT 1 AND 3 MONTHS.

Antibiotic	*No. Attending*	*Recurrence at 1 month**	*Total recurrence at 3 months**
Chlortetracycline	26 (54%)	6 (13%)	6 (13%)
Oxytetracycline	20 (48%)	2 (5%)	3 (7%)
Erythromycin	10 (59%)	2 (12%)	3 (18%)
Spiramycin	16 (59%)	3 (11%)	5 (18%)
Novobiocin	6 (75%)	3 (38%)	4 (50%)

* The percentage given is based on the number of classified as "success" at 27 days.

The follow-up studies as shown in Table V are incomplete, and no figures for tetracycline are available, but it is evident that there is a similar and significantly high relapse rate for both erythromycin and spiramycin. As 50% of the small number of immediate successes obtained with novobiocin had recurrences within three months of discharge, the overall value of this antibiotic in acute amoebic dysentery appears to be slight.

It should be emphasised that no claim is made that any single antibiotic is the treatment of choice in acute amoebic dysentery. In order to reduce the high recurrence rate when even the tetracyclines are used alone, a direct acting amoebicide, such as di-iodohydroxyquinoline should be given concurrently (Elsdon-Dew *et al.* 1952).

It is noteworthy that one patient developed signs of an amoebic liver abscess eight days after completing a course of spiramycin. This

conforms with our experience that not only are antibiotics valueless in the treatment of simple amoebic liver abscess, but patients may actually develop this condition whilst responding to antibiotic treatment of intestinal amoebiasis (Wilmot 1955-1956).

Summary

The results of treatment of acute amoebic dysentery with erythromycin, spiramycin and novobiocin have been compared.

Erythromycin and spiramycin produce similar results, and are effective, although there is a significantly high relapse rate. Novobiocin has little value in the treatment of the condition. None of these drugs appear to be as satisfactory as the tetracyclines.

A direct-acting amoebicide should always be used in addition to an antibiotic.

Antibiotics do not prevent the occurrence of liver abscess.

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The White Blood Cell Count in the Indigenous People of East Africa

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Clinicians and pathologists in East Africa are familiar with the fact that the distribution of the white blood cells in the indigenous people differs from that usually accepted as normal in Europeans. In fact, it is found that most Africans have a high lymphocyte count.

It was the purpose of this investigation to ascertain the differences in the white blood cell counts at various locations at differing altitudes.

A previous article (Moore 1956) demonstrated the differences to be found in the white blood cell counts of men, women, and pregnant women at Mwanza, Tanganyika Territory.

For this study three locations were visited, viz: Kikale, in the Eastern Province of Tanganyika, less than 50 feet (15 metres) above sea level; Mwanza, in the Lake Province, on the south-eastern shore of Lake Victoria, 3,721 feet (1,145 metres) above sea level; and Eldoret in the Rift Valley Province of Kenya, 6,778 feet (2,085 metres) above sea level.

A total of 378 people was examined. The numbers were made up as follows: 60 (36 male, 24 female) at Kikale; 221 (136 male, 85 female) at Mwanza; and 97 (88 male, 9 female) at Eldoret. All the people examined were over 14 years of age. For the purposes of this investigation, results for males and females have been combined. None of the females admitted to being pregnant.

Method

For the total white cell count 20 cmm. of blood were withdrawn from the finger into a pipette. The blood was put directly into the diluting fluid and mixed. A thin film was

Diphetarsone in the Treatment of Acute Amoebic Dysentery

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Bis-(p-arsonophenylamino) 1-2 ethane or diphetarsone (trade name Bemarsal) has been the subject of a number of clinical trials in intestinal amoebiasis by French workers Schneider and Dupoux (1953), Charmot *et al.* (1953), Canet and Ghéstin (1953), Crosnier *et al.* (1953), Collomb and Sankale (1953), Lebon *et al.* (1954), and Sohier *et al.* (1954). Immediate results particularly in acute cases have been good, but there has been a tendency to recurrence—in one series 40% over a ten-month period (Lebon *et al.* 1954). The drug has been well tolerated and no serious side-effects have been reported.

In this paper we record the results of treatment with Diphetarsone in acute amoebic dysentery.

Material and Method

Forty-four African patients were treated with 2½ grammes of diphetarsone daily for 10 days. On admission all complained of diarrhoea with blood and mucus, showed ulceration at sigmoidoscopy and trophozoites of *Entamoeba histolytica* were present in either stools or scrapings from ulcers. Severely ill patients were excluded as were those with coexisting disease.

Sigmoidoscopy was done on the 10th, 15th, 20th, and 27th days after beginning treatment and stools were examined on the same days. When patients appeared to be deteriorating clinically or when ulcers had failed to heal after the end of treatment, daily stool examinations and more frequent sigmoidoscopy were performed.

The following categories are used to express results:—

Success—Sympton free, ulcers healed and no parasites demonstrable.

Probable Failure — Ulceration still present, but no amoebae found in stools or scrapings.

Absolute Failure — Open ulcerations and trophozoites of *E. histolytica* present.

Failure with Cysts — Symptom free, ulcers healed, but cysts of *E. histolytica* found in stools.

Cases remaining or becoming absolute failures after completion of the course of diphetarsone (10 days) were removed from the series and given other drugs. Patients still classified as probable failures at 27 days were given other treatment.

Results

The results are shown in the table. It can be seen that by the 27th day after the commencement of treatment 6 patients (14%) had or had had open ulceration and trophozoites of *E. histolytica* in the stools despite the 10-day course of diphetarsone. One of these had deteriorated symptomatically while on treatment. A further three patients (7%), although symptom-free, harboured parasites, as cysts were found in the stools. Another three cases (7%) still had open ulceration at sigmoidoscopy although parasites could not be found and these patients were without symptoms. The remaining 32 (73%) appeared to be cured.

There was no clinical evidence of toxicity to diphetarsone. Serial urinalysis and blood counts performed in 10 cases remained normal.

Follow-up was incomplete, but one month after discharge 25 of the 32 patients classified as " success " returned for check. Of these 18 remained successes, one was a probable failure and six had open rectal ulceration with trophozoites of *E. histolytica.* This represents a recurrence rate of 19%.

* The Amoebiasis Research Unit is under the joint sponsorship of the South African Council for Scientific and Industrial Research, the Natal Provincial Administration, and the University of Natal.

RESULTS OF TREATMENT WITH DIPHETARSONE IN 44 CASES OF ACUTE AMOEBIC DYSENTERY

Assessment at:	*10 days* *		*15 days* *		*20 days* *		*27 days* *		*Cumulative 27 days* *	
	No. Cases	*%*	*No. Cases*	*%*	*No. Cases*	*%*	*No. Cases*	*%*	*No. Cases*	*%*
Success	26	59	36	82	36	82	32	73	32	73
Probable Failure	15	34	5	11	4	9	3	7	3	7
Absolute Failure	3	7	–	–	1	2	2	4	6	14
Failure with Cysts	–	–	–	–	–	–	3	7	3	7

* After commencement of 10-day course of treatment

Discussion

When compared with other amoebicides used singly in similar cases diphetarsone appears to be an effective agent both for the immediate suppression of parasites and for the control of symptoms. In our hands, there is a parasitic failure rate of 28% with emetine given in a dose of 1 grain daily for 10 days (Armstrong *et al.* 1949) as compared with 20% for diphetarsone. Emetine bismuth iodide (3 grains daily for 10 days) gave a 28% failure rate, diiodohydroxyquinoline 24%, and carbarsone 42% (Armstrong *et al.* 1950).

As with other direct amoebicides diphetarsone proved to be inferior to the wide-spectrum antibiotics in the immediate control of acute amoebic dysentery. With chlortetracycline the parasitic failure rate was only 2% at 27 days and similar results were obtained with oxytetracycline and tetracycline (Armstrong *et al.* 1950, 1952; Elsdon-Dew 1955; Wilmot 1956).

Follow-up was incomplete, but one month after discharge 19% of our patients suffered parasitic recurrence. It seems unlikely that many of these were reinfections and it appears that there is a significant relapse rate with this drug.

This trial indicates that diphetarsone is a useful drug in acute dysenteric amoebiasis, comparable with emetine, diiodohydroxyquinoline and emetine bismuth iodide when used singly. Our clinical impression is that symptomatic relief may not be as rapid as with emetine. Diphetarsone was well tolerated. Like all available remedies it seems likely that it should be used in combination with other agents if satisfactory immediate results and permanent parasitic cure are to be obtained.

Summary

Forty-four patients suffering from acute ulcerative amoebic dysentery were treated with diphetarsone, 2·5 grammes daily for 10 days.

73% of patients were classified as successful, 20% as parasitic failure, and one month later 19% of the successes had suffered parasitic recurrence.

The immediate results compare favourably with those obtained with other direct amoebicides.

No toxic symptoms were encountered.

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