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# SUBCLINICAL ALCOHOLIC HEPATITIS ASSOCIATED WITH POST-ALCOHOLIC HYPOGLYCAEMIA

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As early as 1911 Mallory[1] described a lesion in the chronic progressive alcoholic cirrhotic that he considered characteristic, which consisted of a peculiar hyaline change ('alcoholic hyalin'). This change was often associated with polymorphonuclear and mononuclear cells and with hepatic necrosis. The lesion of hepatic necrosis, not always associated with alcoholic hyalin, has since been noted in chronic alcoholics, both cirrhotic and non-cirrhotic,[1-25] and has been related to preceding ethanol excess.[12, 15, 22, 23, 25] Beckett *et al.*,[22, 23] among others,[6, 7, 12, 25] described an accompanying symptom complex, which consisted of anorexia, nausea and vomiting, upper abdominal pain, and fever with or without icterus. We have now observed a number of cases following alcoholic debauch, where liver biopsy showed foci of necrosis and where the performance of daily biochemical tests showed a transient hepatitis. In these cases the symptom complex described by Beckett *et al.*[22, 23] was not observed, the lesion having been found during the investigation of patients admitted to King Edward VIII hospital in post-alcoholic hypoglycaemia.

Previously[26, 27] we had thought that the alcoholic hepatitis might be a factor in the production of the post-alcoholic hypoglycaemia, since both conditions were present in all our cases. We now feel that the hypoglycaemia and the hepatitis are related only by their common aetiology, ethyl alcohol. The hypoglycaemia appears to be related to alcohol metabolism and to an action of ethyl alcohol on glycogen synthesis and gluconeogenesis in starving patients,[28, 29] while the hepatitis is probably due to the action of ethyl alcohol, either direct or indirect, on the liver cells. The mechanism of post-alcoholic hypoglycaemia, however, has not yet been fully elucidated, and pituitary or adrenal dysfunction might also be involved in some cases.

As a number of our patients, in whom the lesion was noted after excessive intake of alcohol, were heavy weekend drinkers rather than chronic alcoholics, the possibility was considered that ethyl alcohol was a hepatic toxin. We also wondered whether frequent production of hepatic necrosis by alcohol excess in chronic alcoholics might not play a part in the production of cirrhosis in the alcoholic.

In this paper I describe our liver-biopsy findings, and also discuss the possible role of ethyl alcohol in the production of the acute lesion. The findings related to post-alcoholic hypoglycaemia will be described in a subsequent paper.

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## MATERIAL AND METHODS

Thirty Africans (21 males) and 4 Indians (1 male) were studied, admitted to King Edward VIII Hospital in hypoglycaemic coma following excessive intake of ethyl alcohol. The patients were aged from 23 to 65 years and the alcohol intake included Zulu beer, shimeyane, gavine,[26] gin, brandy, wine spirit, and cane spirit. One patient (case 2/62) was admitted on 2 separate occasions. Of these patients 12 were regarded as chronic alcoholics, being uncontrollable drinkers and obviously suffering ill-effects, both in health and in personal relationships, from excessive drinking. Most of the remainder were heavy weekend drinkers (many of them married) who had jobs and never showed any desire for alcohol during their hospitalization (more than 2 weeks). These we did not classify as chronic alcoholics (though this is a difficult problem[30, 31]).

Investigation included clinical examination, biochemical and haematological investigation, and liver biopsy. Biochemical investigations, by the methods described in a previous paper,[26] included daily total serum bilirubin, alkaline phosphatase, serum glutamic oxaloacetic acid transaminase (SGOT), and urinary urobilin and bilirubin.

In the previous article[26] we described the gross changes such as general architectural pattern, large fatty vacuoles, and presence or absence of glycogen, in 17 liver biopsies. We have now re-examined these 17 biopsies and also examined biopsies taken from a further 17 patients. Each of the following has been assessed separately: intralobular foci of degeneration and necrosis with or without inflammatory cell infiltration, alcoholic hyalin, the amount of fatty change (including both fatty vacuolation and fine intercellular change), the degree of fibrosis, the inflammatory cell infiltration in the portal tracts, the glycogen and haemosiderin content, and the presence of bile thrombi and bile-duct hyperplasia. The liver biopsies were performed on 5 patients on admission, on 4 on the first day after admission, on 6 on the second day, on 7 on the third day, on 5 on the fourth day, on 3 on the fifth day, on 2 on the seventh day, and on 1 each on the ninth, eleventh, twelfth and thirteenth days. In one case (patient 14/60) 2 biopsies were performed during hospitalization, while another patient (case 2/62) was biopsied on both admissions. The specimens were fixed immediately in formalin and absolute alcohol, and all were stained with haematoxylin and eosin. The majority of specimens were also stained by silver impregnation for reticulin, Weigert's iron haematoxylin Van Gieson stain for collagen, prussian-blue reaction for haemosiderin, and Best carmine for glycogen. On 5 specimens, taken on admission, periodic-acid/Schiff

TABLE I. EXAMPLES OF RESULTS

| Days | Case 1/62 | | | | Case 2/63 | | | | Case 3/63 | | | | Case 4/63 | | | | Case 5/63 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *T* | *B* | *AP* | *U* | *T* | *B* | *AP* | *U* | *T* | *B* | *AP* | *U* | *T* | *B* | *AP* | *U* | *T* | *B* | *AP* | *U* |
| 1 | 86 | 0·6 | 9 | Nil | 160 | 1·3 | 20 | Nil | 70 | 0·4 | 6 | Trace | | | | | 110 | 0·9 | 5 | |
| 2 | 60 | 0·5 | 10 | Nil | 110 | 1·8 | 18 | Trace | 40 | 0·8 | 6 | | 150 | 1·8 | 7 | | | | | |
| 3 | | | | Nil | 90 | 1·6 | 16 | ++ | 40 | 0·5 | 9 | Trace | 128 | 0·9 | 9 | | 50 | 0·3 | 7 | |
| 4 | 112 | 1·0 | 8 | + | 200 | 1·3 | 17 | ++ | 40 | 0·6 | 5 | + | 86 | 0·7 | 7 | Trace | 70 | 0·4 | 6 | Nil |
| 5 | 38 | 0·9 | 7 | +++ | 200 | 1·0 | 17 | +++ | 20 | 0·6 | 5 | + | | | | Trace | | | | |
| 6 | 50 | 0·9 | 8 | Trace | 110 | 1·2 | 14 | +++ | | | | | 120 | 0·4 | 9 | Trace | 48 | 0·4 | 5 | |
| 7 | 34 | 1·2 | 8 | Trace | | | | | | | | Trace | 144 | | | Nil | 74 | 0·3 | 5 | Nil |
| 8 | | | | | 138 | 0·8 | 16 | | | | | Nil | | | | | | | | |
| 9 | 36 | 0·6 | 11 | Nil | | | | ++ | | | | | | | | | | | | |
| 10 | 30 | | | Nil | 110 | | 13 | + | | | | | 12 | 0·5 | 9 | | 24 | 0·5 | 9 | |
| 16 | | | | | 50 | 0·5 | 11 | Nil | | | | | | | | | | | | |

T = SGOT B = Serum bilirubin AP = Serum alkaline phosphatase U = Urinary urobilin Nil = Not increased.

stain was also used for glycogen, while on 18 specimens fat staining was performed with Sudan III. Eight specimens were stained with phosphotungstic-acid/haematoxylin for alcoholic hyalin.[32]

Haematological investigation was performed by standard methods.

## RESULTS

### *Clinical Findings*

All our 34 patients presented in hypoglycaemic coma following excessive intake of alcohol. After treatment with intravenous dextrose solution, 14 recovered immediately and completely and required no further treatment. Two patients (cases 8/60 and 2/62) developed delirium tremens and 3 (cases 5/60, 13/62, and 2/63) alcoholic psychosis. Four patients (cases 7/60, 11/60, 19/60, and 25/60) remained mentally dull or confused for a few days after dextrose therapy, and this was considered to be the result of prolonged hypoglycaemia. One patient (case 1/60) developed acute pancreatitis, one (case 6/60) porphyria, one (case 1/62) an aspiration pneumonia, and one (case 12/62) developed lobar pneumonia and died 5 days after admission. Six patients (cases 5/60, 14/60, 20/60, 22/60, 5/62 and 10/62) had pulmonary tuberculosis, of whom one (5/62) had an associated pneumothorax and one (10/62) developed miliary tuberculosis with meningitis. One patient (case 9/60) developed pyrexia with pulmonary symptoms 2 weeks after admission, and another (case 6/62) had a fever (100°F) for a few days after hospitalization. The liver was palpable in 17 patients and tender in 1, but in none was it grossly enlarged. In no patients was splenomegaly, ascites, or any sign of hepatic failure noted, and in none was jaundice or gastro-intestinal haemorrhage observed. Pellagrinous dermatitis was found in 7 patients, poor nutrition in 11, and fair nutrition in the remainder (16). The dietary history of the patients was poor (particularly in the cases of chronic alcoholism), showing an intake high in carbohydrate and low in protein.

### *Biochemical Findings*

Table I shows examples of the results found in our patients. Results from some of the patients have already been published,[26] and it can be seen that all suffered from a transient subicteric hepatogenous jaundice following alcoholic excess.

### *Liver-Biopsy Findings*

*Foci of necrosis* were found in 18 specimens and were almost always accompanied by an inflammatory cell reaction consisting of neutrophils, monocytes and round

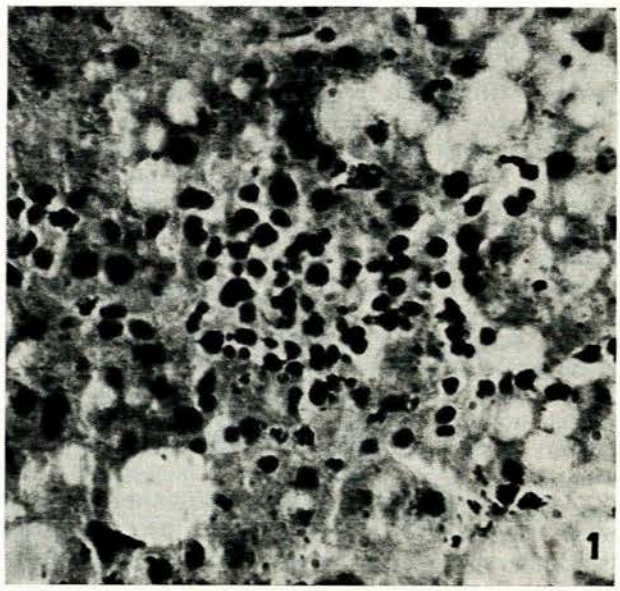


*Fig. 1.* Liver biopsy, case 14/60, early chronic alcoholic, shimeyane drinker. Focus of necrosis with inflammatory cell reaction; severe fatty change, grade 4. (Fibrosis, group I; siderosis, grade 4.) (H. & E.)

cells in varying numbers (Figs. 1—4). There was no correlation between the foci of necrosis and fatty change, and in one case (22/60) there was virtual absence of fat.

*Alcoholic hyalin* was not noted in any sections (2 doubtful), but few were stained with phosphotungstic acid/haematoxylin or other special stains.

*Fatty change* (*lipohepatosis*). On haematoxylin-and-eosin section, fatty change was absent in 1 case, mild in 4 cases, moderate in 5, severe in 18 and extreme in 7. In

18 cases, where tissue was stained with Sudan III, it was graded from 0 to 4 according to the proportion of cells showing fatty change, and was considered to be grade 1

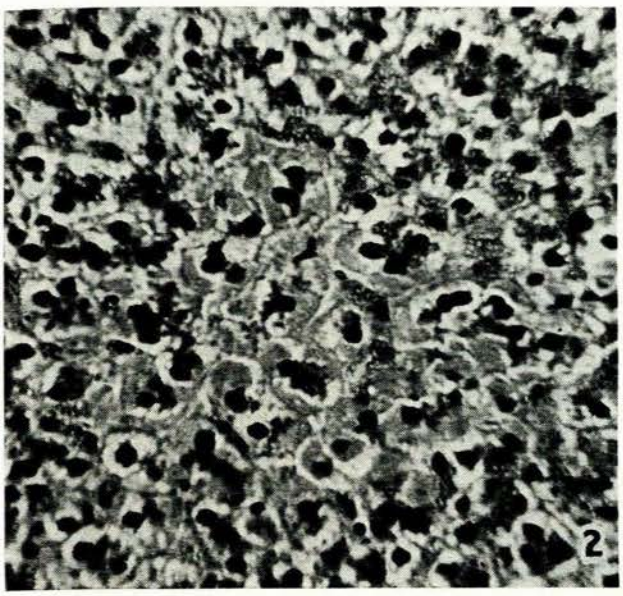

*Fig. 2.* Liver biopsy, case 12/62, ? chronic alcoholic, heavy drinking 1 year. Focus of eosinophilic necrosis (? ischaemic) without inflammatory cell reaction; fine intercellular fatty change severe; siderosis grade 5. (Fibrosis, group IV.) (H. & E.)

(1 — <25%) in 3 cases, grade 2 (25 — <50%) in 1 case, grade 3 (50 — <75%) in 2 cases, and grade 4 (75 — 100%) in 12.

*Inflammatory cells in the portal tracts.* Acute or chronic inflammatory cells were found in the portal tracts of al-

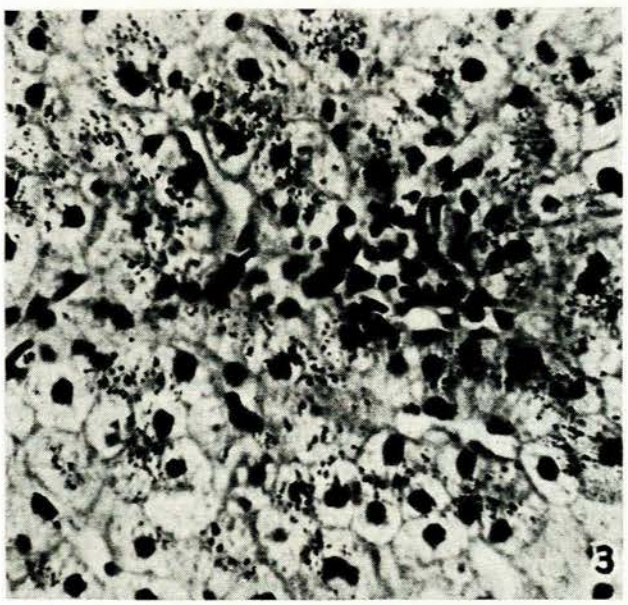

*Fig. 3.* Liver biopsy, case 4/62, heavy weekend drinker. Siderosis grade 4; focus with inflammatory cell infiltration; fatty change mild. (Fibrosis, group I.) (H. & E.)

most all sections. This is a common finding in liver sections from Africans at this hospital.[33, 34]

*Fibrosis* was classified according to Higginson *et al.*[34] as follows: group I = non-fibrotic liver; group II = slight portal fibrosis; group III = moderate fibrosis ('periportal fibrosis'); group IV = severe diffuse septal fibrosis (mild cirrhosis); and group V = severe cirrhosis. Group I was found in 11 cases, group II in 12, group III in 5, group IV in 3, and group V in 4 (Fig. 4).

*Haemosiderin* was graded according to Wainwright,[35] from 0 to 5. In grade 0 haemosiderin was absent, in grade 1 it was found in the Kupffer cells only, in grade 2 in the

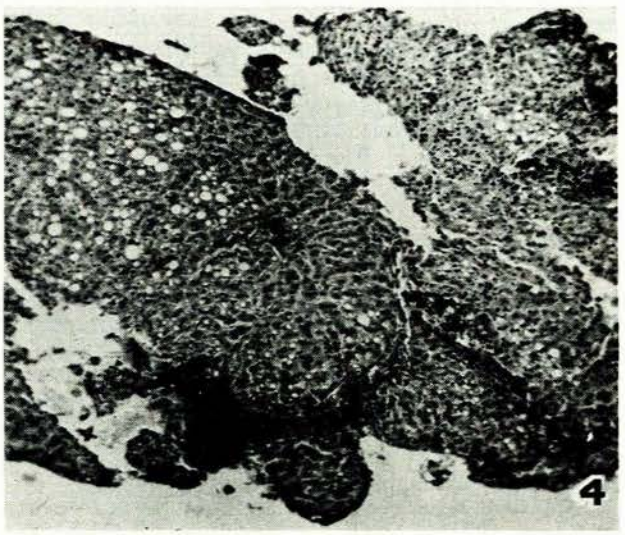

*Fig. 4.* Liver biopsy, case 8/60, chronic alcoholic, heavy drinker 25 years. Cirrhosis with intralobular focus of necrosis and inflammatory cell reaction; severe fatty change. (Siderosis, grade 2.) (H. & E.)

parenchyma at the periphery of the lobules, in grade 3 throughout the lobule, in grade 4 in clumps also in the portal tracts and parenchyma, and in grade 5 more severely than in grade 4. It was found to be grade 0 in 5 cases, grade 1 in 2 cases, grade 2 in 4 cases, grade 3 in 7 cases, grade 4 in 8 cases, and grade 5 in the remainder (Figs. 2 and 3).

*Glycogen.* On admission this was found to be absent in 5 cases but varied from grade 1 to 4 in the remainder.

*Bile thrombi* were not noted but were probably more difficult to assess because of the large quantities of haemosiderin on our sections. Bile-duct hyperplasia, accompanied by cirrhotic change, was noted in 3 cases.

*Haematological Findings*

The white blood count, performed on the first or second day of admission, was under 11,000 per cu.mm. in 21 patients. In 9 patients, it varied from 11,000 to 28,000/cu.mm. The haemoglobin was below normal in 2 patients. One of these had miliary tuberculosis; the cause of the anaemia in the other was not elucidated.

*Comment*

None of our patients, except one, had accompanying clinical symptoms such as abdominal pain, nausea or vomiting, while anorexia was rare and only found in the

chronic alcoholic. One patient developed severe abdominal pain and was proved to be suffering from acute pancreatitis. Thus our cases differed from those of Beckett *et al.*,[23] the hepatitis being subclinical and only being found during investigation of the post-alcoholic hypoglycaemia. Siefe *et al.*[10] noted the absence of clinical features in patients who had been admitted in a state of acute alcoholism, and Volwiler *et al.*[6] noted this in 2 of their patients. Volwiler and Jones[5] emphasized that the severity of active intrahepatic necrosis and inflammation often did not correlate with the clinical impression.[4, 14] We have noted clinical symptoms and signs following alcoholic excess, in particular, jaundice, fever and leucocytosis; and these have been mentioned by other writers,[6, 7, 12, 15-17, 21-25] but are not a necessary accompaniment of the hepatic lesion.

In all our cases we found, by biochemical investigation, a transient hepatogenous jaundice. It seems probable that the small scattered foci of hepatic necrosis account, at least partly, for this. The foci were unassociated with cirrhosis in the majority of our cases and there was no correlation between them and fatty change. This absence of correlation has been noted by others previously.[5, 6, 10, 12, 13, 18, 24] Alcoholic hyalin was not found in association with necrosis in our cases and its absence has also been noted by several others.[13, 14, 18, 22, 23] The presence of alcoholic hyalin might indicate a poor prognosis,[36] which was not found in our cases. Siderosis was common and severe in most. Gillman and Gillman[37] suggested that siderosis, which is extremely common in Southern Africa, is the result of very chronic malnutrition but many now feel it is due to a high iron intake.

There have been numerous reports of foci of hepatic necrosis in chronic alcoholics with cirrhosis.[1-23, 25] Siefe *et al.*,[10] Popper *et al.*,[14] Shorter and Baggenstoss,[18] and Green *et al.*,[25] among others observed the lesion in chronic alcoholics without cirrhosis. Beckett *et al.*,[22, 23] also observed it in patients with and without cirrhosis and they emphasized the preceding alcoholic debauch in its aetiology.[12, 15, 25] We have now found it, accompanied by transient biochemical hepatitis,[25] in heavy weekend drinkers, without accompanying clinical symptoms, after intake of excess alcohol. On this evidence, there appears to be a relationship between the foci of necrosis and preceding acute alcoholic excess, and the question arises whether ethyl alcohol is not acting as a toxin in the production of the hepatic necrosis. It is probable that certain circumstances are required before ethyl alcohol can cause hepatic injury. It seems that an excessive intake of alcohol (acute excess or frequent chronic excess) is an important factor, and it is possible that malnutrition is a predisposing condition. Infection, glycogen depletion, and individual idiosyncrasy, might also play a part in its production in some cases. Supportive experimental evidence for the toxic action of ethyl alcohol has been given by Ogata[38] and Cameron *et al.*,[39] who showed that hepatic necrosis resulted from the introduction of ethyl alcohol into the portal circulation. In addition, increased SGOT activity has been noted by Bang *et al.*,[40] Hed,[41] and also by ourselves,[26] following the intake of ethyl alcohol.

The possibility that toxic factors were present in the alcohol was considered because the majority of the patients had been drinking home brews or distillates (Zulu beer, shimeyane and gavine) but, for the reasons given in our previous article,[26] it was not thought likely, though it is possible that impurities are produced during the distillation of one of them (gavine). However, there was no clinical, biochemical or histological difference between the patients taking African liquor and those drinking commercially produced alcohol. We also wondered whether the foci of necrosis were the result of infection, but associated infection was not present in all our patients, and foci of necrosis were less frequently found on examination of 82 post-mortem liver specimens from patients in whom acute infection was prevalent. It is, however, possible that the liver of the chronic alcoholic is more susceptible to infection. The possibility that hypoglycaemia was the cause was also considered, but we were unable to find a comparable series of cases of prolonged hypoglycaemia from which liver-biopsy material could be obtained. It is possible that a liver with glycogen depletion is more prone to injury by ethyl alcohol.

The role of malnutrition in the production of the hepatitis is difficult to assess, for it is known that hepatic necrosis can be produced in animals on inadequate diets.[42-45] In addition, on experimental and clinical grounds it seems likely that patients on imbalanced diets would be more vulnerable to hepatic toxins.[42, 46, 47] Nevertheless, this does not necessarily indicate that patients on adequate diets or of good nutrition may not suffer similar damage after excess alcohol. Beckett *et al.*[22, 23] considered that most of their patients were not malnourished, while Chalmers *et al.*[8] found hepatic necrosis in steady drinkers who were regularly employed and eating at least one good meal a day. It is noteworthy, however, that ethyl alcohol, itself, can increase the demands for certain nutrients.[48, 49] The majority of our patients either showed evidence of poor nutrition, gave a history of an inadequate diet, or received an income on which an adequate intake would not be possible without frequent subsidy from other sources. It appears, therefore, that poor nutrition could have played a part in the production of the hepatitis in our patients.

## SUMMARY

Focal hepatic necrosis was observed on liver biopsy in chronic alcoholics and heavy weekend drinkers, after hypoglycaemic coma following alcoholic debauch. An accompanying transient (biochemical) hepatitis was also noted.

It was felt that the hypoglycaemia and the hepatitis were only related by their common aetiology, ethyl alcohol. In the literature, foci of hepatic necrosis have been noted on numerous occasions in chronic alcoholics (with and without cirrhosis), and, preceding acute alcoholic excess, has appeared to be an important factor in its aetiology. In a number of cases reported here, foci of hepatic necrosis were found, but the patients were heavy weekend drinkers rather than chronic alcoholics. It seemed probable that ethyl alcohol was the toxin concerned in the production of the lesion, though other factors, such as malnutrition, might have played a part.

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# BLOOD SUPPLY TO FORE-, MID- AND HINDGUT IN CASE OF NON-ROTATION OF 2nd STAGE ASSOCIATED WITH ABNORMAL DUODENUM AND ABUNDANT SPLENIC TISSUE

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Although many cases of arrest of intestinal rotation in the 2nd stage have been recorded,[1] Bleloch[2] rightly points out that the associated vascular arrangements have been described in comparatively few of these. The present report describes the arrangement of the bowel and its blood supply in an instance of complete second-stage non-rotation of the midgut loop found in a White female aged 79 years, dissected during 1963 in our laboratories. The associated abnormal arrangement of the duodenum and pancreas and the presence of an unusual volume of splenic tissue in this individual form the subject of other papers.

## DISPOSITION OF BOWEL AND PERITONEAL ATTACHMENTS (*Fig.* 1)

In the thorax the oesophagus, after passing behind the arch of the aorta, lay in the midline to the right of and anterior to the descending aorta. The cardia, embraced by the loops of the diaphragmatic crura, lay anterior to the beginning of the abdominal aorta in the midline at the level of the 12th thoracic vertebra, the inferior vena cava being closely related to its right border (Fig. 2). The stomach was normal in all respects.

The small intestine joined the pylorus 2·5 cm. to the LEFT of the midline at the level of the 2nd lumbar vertebra. Its proximal 13·3 cm. formed a freely mobile peritoneal-covered U loop with its concavity upwards, which was related to the head, neck and part of the body of the pancreas. Following this U loop was a 10-cm. vertical limb of bowel anchored to the posterior body wall in relation to the inferior vena cava; from this a further 10-cm. loop of small bowel proceeded to the right, its concavity facing downwards. Peritoneum covered the inferior, anterior, superior and part of the posterior aspects of this last loop, so that a deep peritoneal fossa was formed between it and the lower pole of the right kidney. The exact site of the duodeno-jejunal junction defies anatomical definition.

The ensuing coils of small bowel, measuring 2·43 m. in total length, and also the caecum and proximal 20 cm. of colon, occupied the free border of the mesentery. The root of the mesentery, 20 cm. in length, began at the lower pole of the right kidney, 10 cm. from the midline, and extended from the right upper to the left lower quadrant of the abdomen, skirting the brim of the pelvis to end to the left of the midline at the level of the promontory of the sacrum just short of the left sacro-iliac joint. The right leaf of the mesentery became continuous with the attenuated pelvic mesocolon, whereas the left leaf continued to anchor an ascending limb of colon to the posterior body wall in the left half of the abdominal cavity.

The mobile caecum (Fig. 3), 8·75 cm. in width by 5·23 cm. in depth above the ileocolic valve, lay slightly to the left of the midline at the pelvic brim; the terminal ileum entered it from the right side and the 2·5-cm. appendix projected upwards and to the right from its superior aspect. The proximal 20 cm. of colon, leading from the caecum, was also supported by the mesentery; it formed a mobile knuckle of bowel dipping into the pelvis and closely related to the relatively immobile pelvic colon. From this pelvic loop, a tortuous ascending limb of colon, 20 cm. long, ran up the posterior body wall behind the