have a hypotensive effect by virtue of its adrenolytic properties. Knox and Slessor (1955) found that 25 mg. of chlorpromazine intramuscularly did not affect the height of a histamine-induced rise in blood pressure in their case, but abolished the symptoms, such as headache, pallor, and sweating, which were associated with the rise. In our case 25 mg. of chlorpromazine was given intramuscularly to the patient in the supine position. It produced a fall in blood pressure to normotensive levels which lasted for 4½ hours (Fig. 4). There was no significant fall in the blood pressure when chlorpromazine was given in the same dosage under the same conditions after the operation (Fig. 5). This suggests that chlorpromazine has a powerful adrenolytic and noradrenolytic action in the human subject, and that it may be used as an alternative to phentolamine in the premedication of patients before operative removal of a phaeochromocytoma. Dundee (1954) used chlorpromazine intramuscularly for this purpose, and found that, although it did not completely prevent a rise in blood pressure during positioning the patient on the operating table, it was very effective in preventing a hypertensive attack during handling of the tumour.

Summary

A case of phaeochromocytoma is described which showed sustained hypertension, increased metabolism, impaired dextrose tolerance, polyuria, and albuminuria. These findings became normal after removal of the tumour.

The use of phentolamine in the diagnosis and operative management and the effect of reserpine and chlorpromazine on the blood pressure are described.

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The proceedings of the National Smoke Abatement Society's conference at Bournemouth last year have recently been published (price 12s. 6d. net from the Society, Palace Chambers, Bridge Street, London, S.W.1). They include Sir Ernest Smith's presidential address, papers on recent progress in domestic heating by centralized methods, on local authorities and the Beaver Report and the Clean Air Bill, and by members of the Beaver Committee. The Des Vœux Memorial Lecture was given by Dr. A. Parker on "The Destructive Effects of Air Pollution on Materials."

SERUM IRON IN THE DIAGNOSIS OF HEPATOBIILIARY DISEASE

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The differentiation between obstructive and non-obstructive jaundice may sometimes be difficult in spite of the laboratory aids at present available. It is therefore interesting to note reports of an association between viral hepatitis and an abnormally high serum iron level. Earliest publications were those by Warburg and Krebs (1927) and Locke *et al.* (1932), who each described a single case of "icterus" in which a raised serum iron level occurred. Hemmeler (1939) first reported that the serum iron was elevated in "catarrhal icterus" and normal in obstructive jaundice. Since then only a few reports have appeared in the literature on this subject.

The present study was undertaken to investigate the value of the serum iron determination as a routine procedure in the diagnosis of hepatobiliary disease.

Methods and Material

Over a period of 15 weeks 104 serum iron studies were conducted on 63 patients with hepatobiliary disease. This group consisted of 21 cases of acute infective hepatitis, 8 cases of homologous serum hepatitis, 6 cases of "subacute viral hepatitis" (defined below), 14 cases of cirrhosis of the liver, 12 cases of extrahepatic obstruction, and 2 cases of chlorpromazine "hepatitis." There was also one case of haemochromatosis.

The diagnosis of viral hepatitis was based on the history of a constitutional disturbance (anorexia, nausea, lassitude, generalized body pains), pyrexia, tenderness in the right hypochondrium, jaundice, and regression of the disease on conservative treatment. If injections had not been given during the preceding six months the cases were termed "infective hepatitis" (endemic viral hepatitis), whilst those that had had injections two to six months before the onset of the illness were designated "homologous serum hepatitis." None of the patients had been given a blood transfusion or had received iron therapy prior to the onset of the disease.

Specimens were collected at 9 a.m.—that is two hours after the morning meal. Before any glassware was used in the laboratory (including the tubes into which the blood was collected) it was cleaned with chromic acid, then with concentrated nitric acid, and rinsed well with glass-distilled water. The serum iron was determined by the method of Kitzes *et al.* (1944), which takes approximately two hours to perform; several estimations may be carried out simultaneously. Our normal range for this method is 70–185 µg. per 100 ml.

Results

The results are recorded in Figure 1 and Table I. At the initial estimation, which was carried out during the first week of admission, 16 out of 21 cases of infective hepatitis had serum iron values above 220 µg. per 100 ml. The mean value of the 21 initial estimations was 292 µg. per 100 ml.

Nineteen of these 21 cases had values above 220 $\mu\text{g.}$ during some stage of the disease. The mean of all the highest serum iron levels obtained in the cases of infective hepatitis was 309 $\mu\text{g. per 100 ml.}$ This figure is in agreement with the findings of Peterson (1952) (295 $\mu\text{g. per 100 ml.}$) and Christian (1954) (297 $\mu\text{g. per 100 ml.}$).

In this series three out of eight cases of homologous serum hepatitis had values above 220 $\mu\text{g. per 100 ml.}$ during some

FIG. 1.—Highest recorded serum iron values in patients with hepatobiliary disease.

TABLE I.—Results of Serum Iron Estimations in Hepatobiliary Disease

	Total No. of Cases	No. of Cases with Serum Iron Values Above 220 $\mu\text{g. per 100 ml.}$	
		On Initial Estimation	On Initial or Subsequent Estimation
Infective Hepatitis	21	16	19
Homologous Serum Hepatitis	8	1	3
"Subacute Viral Hepatitis"	6	6	6
Cirrhosis of the Liver	14	1	1
Gall-stone Obstruction	5	0	0
Carcinoma Head of Pancreas	7	0	0
Chlorpromazine "Hepatitis"	2	0	0

stage of the disease. Normal serum iron levels were more frequent in this group than in infective (endemic) hepatitis. It is interesting to note that Peterson (1952), in his series of 60 cases of viral hepatitis, found five with serum iron levels below 200 $\mu\text{g. per 100 ml.}$, four of which were cases of homologous serum hepatitis.

Of the group with non-viral hepatobiliary disease (cirrhosis, gall-stone obstruction, carcinoma of the head of the pancreas, and chlorpromazine "hepatitis") only 1 out of 28 cases had a serum iron level above 220 $\mu\text{g. per 100 ml.}$ The mean of the highest values obtained in this group was 141 $\mu\text{g. per 100 ml.}$ A significant difference is thus noted between this group and that of infective hepatitis.

Illustrative Cases

Case 1. Infective Hepatitis.—On admission to hospital a 40-year-old man stated that for the previous eight days he had suffered from acute nausea, had vomited frequently, had had no appetite, and had experienced a feeling of extreme fatigue with generalized aches and pains. Two days before admission he had noticed that his skin was yellow in colour. The results of the biochemical investigations are shown in Fig. 2. Against medical advice he started to walk about, when his serum bilirubin was 4.2 mg. per 100 ml. This occasioned a return of symptoms, with a marked rise in the serum iron level despite the fact that the serum bilirubin level continued to fall.

Case 28. Homologous Serum Hepatitis.—A woman aged 20 was admitted to hospital with a two-days history of headaches, rigors, and anorexia. Examination on the third day of her illness revealed a spleen which was just palpable. She was febrile—99.8° F. (37.7° C.)—and a white-cell count showed 4,800 cells per c.mm. At this stage her serum iron was 227 $\mu\text{g. per 100 ml.}$ and the diagnosis was "pyrexia of unknown origin." During the next 10 days an occasional specimen of urine showed the presence of excess urobilin. However, she felt perfectly well and expressed a desire to leave hospital. On the thirteenth day of her illness her urine showed the presence of bilirubin and a complete battery of liver-function tests were carried out. These revealed grossly abnormal function, and her serum iron was 400 $\mu\text{g. per 100 ml.}$ On the sixteenth day she became clinically jaundiced and a diagnosis of viral hepatitis was made. (As the patient had received penicillin injections 60 days previously for an attack of tonsillitis this case was classified as homologous serum hepatitis.) The results of investigations and of treatment are shown in Fig. 3.

Case 32. Subacute Viral Hepatitis.—A 17-year-old scholar was admitted to hospital in October, 1954. He stated that for two days before admission he had had nausea, weakness, dizziness, faintness, and stiffness of the neck. Examination revealed a macular rash, enlarged lymph nodes in the posterior triangle of the neck, a spleen which was just palpable, and a liver enlarged two fingerbreadths below the costal margin. The white-cell count was 10,600 per c.mm.; lymphocytes 67%, many of which were atypical. The Paul-Bunnell test was negative on two occasions. During the ensuing months he felt listless, often went to sleep in the afternoons, and "could not look at food." Examination seven months after the onset of the illness revealed an enlarged tender liver two fingerbreadths below the costal margin. Flocculation tests were abnormal and the total serum bilirubin was 0.4 mg. per 100 ml. There was

FIG. 2.—Case 1. Serum iron and serum bilirubin values.

excess urobilin in the urine. Serum iron levels at weekly intervals were 334, 264, and 232 $\mu\text{g. per 100 ml.}$ At no stage of the disease was there any clinical jaundice.

Case 29. Subacute Viral Hepatitis.—A 32-year-old medical practitioner stated that during January, 1955, he had an attack of gastro-enteritis with generalized body pains and rigors. He was also very anorexic during this period, which lasted approximately one week. Since then he had suffered from tiredness, weakness, and loss of appetite. He had found it physically difficult to carry on with his medical practice, whereas previously he had been extremely active. Examination during the initial attack and at various times

since then revealed the presence of excess urobilin in the urine. In April physical examination revealed tenderness in the right hypochondrium but no other abnormality. On April 20 the serum iron was 268 $\mu\text{g. per 100 ml.}$ and the serum bilirubin 1.2 mg. per 100 ml. On May 18 the serum iron was 243 $\mu\text{g. per 100 ml.}$ and the serum bilirubin 0.2 mg. per 100 ml. Daily intramuscular injections of 1,000 $\mu\text{g.}$ of vitamin B₁₂ were started on May 18. On the third day of treatment the serum iron was 132 $\mu\text{g. per 100 ml.}$ and the serum bilirubin 0.4 mg. per 100 ml. At no stage of the disease was there any obvious clinical jaundice.

Case 60. Haemochromatosis Complicated by Post-infective Hepatitis.—A 39-year-old medical practitioner

FIG. 3.—Case 28. Results of investigations and treatment.

stated that he had had an attack of infective hepatitis 10 years previously and three recurrences within a period of six months. Since then he had felt tired and "physically exhausted, whereas other people seemed to carry on." He also experienced attacks of "dragging discomfort" in the right hypochondrium, especially towards the end of the day. In addition he had an extreme loss of libido. Friends observed that his complexion had darkened a little. The liver was palpable three fingerbreadths below the costal margin, but no obvious clinical jaundice was noted. Liver-function tests showed gross derangement. Serum iron levels on two occasions were 416 and 326 $\mu\text{g. per 100 ml.}$ An exploratory laparotomy revealed an enlarged liver, spleen, and lymph nodes. Pathological report (Dr. B. J. P. Becker): "Sections showed the presence of intense iron pigmentation in the liver cells, in the Kupffer cells, and in the portal tracts. There was increased fibrous tissue in the portal tracts and early portal cirrhosis was present. There was a heavy lymphocytic and mononuclear infiltrate in the portal tracts together with slight bile-duct proliferation. No evidence of hepato-cellular necrosis has been observed. Sections of a lymph node removed from the neighbourhood of the porta hepatis showed the presence of heavy deposits of haemosiderin. These observations indicated the presence of haemochromatosis with early portal cirrhosis. In addition, in my experience the heavy cellular infiltrate of the portal tracts was very unusual in uncomplicated cases of haemochromatosis, and in my opinion there was evidence in the liver of a persistent post-infective hepatitis. Summary: Haemochromatosis complicated by post-infective hepatitis."

Case 43. Cirrhosis of Liver.—This was the only case of portal cirrhosis with a serum iron level above 220 $\mu\text{g. per 100 ml.}$ A 76-year-old man stated that three months before admission to hospital his abdomen became swollen and had steadily increased in size. Alcoholic intake had been exces-

sive, but latterly he had shown some moderation in this respect. Physical examination revealed a massive ascites. After this had been aspirated no liver or spleen could be palpated. The serum iron levels were 232 and 259 $\mu\text{g. per 100 ml.}$ He was treated with vitamin-B complex, repeated abdominal paracentesis, and mercurial diuretics. While in hospital he became jaundiced and his condition deteriorated progressively. He died three weeks after admission from a terminal bronchopneumonia. Pathological report (Dr. C. B. Chatgidakis): "Sections of liver showed the characteristic features of an advanced Laennec's cirrhosis. A heavy non-granular cell infiltrate was present in the interlobular stroma and numerous foci of necrosis and regeneration were noted in the parenchyma. Bile duct proliferation was slight. No alcoholic hyaline or fat was present. No animal parasites were noted. Summary: Laennec's cirrhosis, ascites, and splenomegaly. The histological features are suggestive of post-infective cirrhosis."

Discussion

Normal serum iron values in Europeans are 70–185 $\mu\text{g. per 100 ml.}$ In this study only values above 220 $\mu\text{g. per 100 ml.}$ were regarded as significantly raised. Other investigators have considered values above 200 $\mu\text{g. per 100 ml.}$ as significant, and, as may be seen in Table II, all have noted significantly increased values in most cases of viral hepatitis. The cause of this hypersideraemia is not yet known.

There is no constant linear relationship between the serum iron level, the serum bilirubin, and the severity of the disease. Frequently the maximal sideraemia occurred after the maximal bilirubinaemia, and quite often convalescent cases were found to have raised serum iron levels. This conforms with the findings of other investigators (Vahlquist, 1941; Brochner-Mortensen, 1942; Hemmeler, 1951; Peterson, 1952). Furthermore, Hemmeler has observed a rapid decrease in serum iron values when the complication of acute yellow atrophy occurs.

Diagnostic difficulties occur most in those patients with subacute viral hepatitis. This comprised: (1) the post-hepatitis syndrome (Caravati, 1944; Sherlock and Walshe, 1946); (2) the subacute pre-cirrhotic stage of viral hepatitis as described by Sherlock (1948); and (3) the cases often designated as hepatitis sine icterus. All these patients told of periods of chronic ill-health (lassitude, anorexia, etc.) varying from a few months to many years. In some this illness had followed an overt clinical attack of infective hepatitis, but in others no such history was obtained. Periodic examination revealed the occasional presence of excess urobilin in the urine, a fluctuating bilirubinaemia not exceeding 2 mg. per 100 ml., and transient derangement of liver-function tests. All these cases had abnormally elevated serum iron readings—mean value 309 mg. per 100 ml. Most of these cases have previously been termed the post-hepatitis syndrome, which was thought to be psychogenic in origin. It is suggested, however, that the clinical findings in conjunction with the raised serum iron level indicate that the disease is still in an active phase and that prolonged bed rest is necessary. The hepatic lesion in these cases may be as severe as that encountered in the overt icteric cases (Mallory, 1947; Axenfeld and Brass, 1942), and it is in this condition that the serum iron level as an indicator of activity may find its greatest value.

Four of the cases of viral hepatitis were given 1,000 $\mu\text{g.}$ of vitamin B₁₂ by intramuscular injection daily. This was followed by an appreciable fall in the serum iron level on the second day of treatment and coincided with an increased feeling of well-being.

All the cases with extrahepatic obstruction had serum iron levels below 220 $\mu\text{g. per 100 ml.}$ This is in conformity with the findings of previous investigators (Table II). In two cases the jaundice followed the administration of chlorpromazine. This type of jaundice has been ascribed to obstruction in the biliary canaliculi (Van Ommen and Brown, 1955; Bersohn and Wallace, 1955).

TABLE II.—*Previous Investigations of Serum Iron in Hepatobiliary Disease (in $\mu\text{g. per } 100 \text{ ml.}$)*

	Viral Hepatitis		Cirrhosis		Post-hepatic Obstruction	
	Total No.	No. >200 $\mu\text{g.}$	Total No.	No. >220 $\mu\text{g.}$	Total No.	No. >220 $\mu\text{g.}$
Hemmeler (1939) ..	10	5			11	0
Skouge (1939) ..	5	2			4	0
Waldenström (1940) ..	9	4				
Vahlquist (1941) ..	25	10				
Brochner-Mortensen (1942) ..	26	17	5	0	19	1
Bjerre and Christoffersen (1942, 1944) ..	54	44	13	0	18	0
Rambert <i>et al.</i> (1946) ..	14	7	5	0	7	0
Skjetne (1948) ..	24	20	23	0	26	0
Ducci <i>et al.</i> (1952) ..	62	36	68	0	27	2 3*
Peterson (1952) ..	60	55	15	0	10	0
Christian (1954) ..	20	17	23	1	13	1
	309	217	152	1	135	2

* The diagnosis in these 3 cases was doubtful.

There were 14 cases of cirrhosis of the liver in this series—seven were cases of “alcoholic” cirrhosis, one was a case of “cardiac” cirrhosis, one a case of biliary cirrhosis, and in five the aetiology was obscure. Of the 14 cases, one had a serum iron level above 220 $\mu\text{g. per } 100 \text{ ml.}$ The clinical diagnosis in this case was alcoholic cirrhosis. The pathological report stated that the histological features were suggestive of post-infective cirrhosis (see above). In the medical literature we have found one case of uncomplicated cirrhosis out of a total of 155 cases in which the serum iron level was above 220 $\mu\text{g. per } 100 \text{ ml.}$

The only diseases in which the serum iron level may be greatly raised are infective hepatitis and haemochromatosis. (A minimal rise may occur in cases of pernicious anaemia and haemolytic anaemia.) In other infectious or inflammatory diseases the serum iron is in the low or low normal range (Cartwright *et al.*, 1946).

The serum iron level might be of particular value in differentiating the great majority of cases of viral hepatitis from those of extrahepatic obstruction and cirrhosis of the liver. Thus, should the serum iron level in a clinically doubtful case of jaundice be greater than 220 $\mu\text{g. per } 100 \text{ ml.}$, the diagnosis of cirrhosis of the liver or extrahepatic obstruction is unlikely and the most probable diagnosis is viral hepatitis. The obvious exception to this is the rare occasion when a patient with haemochromatosis develops an extrahepatic obstruction or cirrhosis of the liver. No conclusions whatsoever can be drawn if the serum iron level is low or normal.

We endorse the view of Christian (1954), who considers it a “real oversight” that so few studies of this relatively simple investigation have appeared in the English literature. There can be no doubt that this test constitutes a valuable adjunct to clinical diagnosis. It should be emphasized, however, that the test is only an adjunct to diagnosis, and that in the words of Himsworth (1950) “there is yet no test which approaches in value a careful clinical assessment of the patient and none which can be interpreted without it.”

Summary

The serum iron was estimated 104 times in 63 patients with hepatobiliary disease.

The serum iron level was found to be significantly raised, above 220 $\mu\text{g. per } 100 \text{ ml.}$, in the majority of cases of viral hepatitis, and to be below 220 $\mu\text{g. per } 100 \text{ ml.}$ in nearly all cases of cirrhosis of the liver and extrahepatic obstruction.

The value of this test in the diagnosis of hepatobiliary disease is discussed.

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ANIMAL RESERVOIR OF RINGWORM INFECTION IN BRITAIN*

In many of the major mycoses which affect both man and animals the spread of infection from one type of host to the other is infrequent, and both are subject to the hazard of infection from an external reservoir such as the soil. Two well-known examples of such mycoses are the systemic coccidioidomycosis (caused by *Coccidioides immitis*), which is endemic in arid regions of North and South America, and histoplasmosis (caused by *Histoplasma capsulatum*), which occurs in certain more humid regions of the United States and elsewhere. The first of these has a high incidence among wild soil-burrowing desert rodents, and attacks man as well as farm and domestic animals, the disease often being mild or the infection even subclinical. Infected desert rodents were at first suspected of being the effective source of the pathogen in man, but it now seems more probable that this is to be found in dust from certain soils. Histoplasmosis, which attacks both man and animals and is often a mild disease or present in subclinical form, is most probably also a soil dust-borne infection; in favour of this view is some recent evidence that certain regions in which histoplasmosis is endemic have soils of a similar kind.

There is, however, another type of mycosis affecting both man and animals in which the infecting agent is endogenous, and in which the usual mode of spread of infection is probably from man to man and from animal to animal. An example of this type is moniliasis, caused by the yeast *Candida albicans*, which is a common inhabitant of the mucous surfaces of apparently normal

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