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REMARKS ON (i.) CHRONIC HÆMOLYTIC (ii.) PANCREATIC JAUNDICE

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THE following brief sketch gives an outline of our present knowledge about two forms of jaundice which have become prominent in recent years.

Chronic Hæmolytic Jaundice.

With the progress of time it sometimes happens that the current opinion of one period, after becoming discredited, if not forgotten, subsequently regains general acceptation. This swing of the pendulum is seen in the views held, during the lifetime of middle-aged members of our profession, on the mechanism of jaundice. Thirty years ago jaundice was divided into obstructive (due to gross changes in the bile-ducts) and non-obstructive (with no obvious cause). Various explanations were current to explain non-obstructive jaundice, such as suppression of the bile secretion by the liver, and the formation of bile in the blood or elsewhere from effete blood-pigment; this was therefore called hæmatogenous, in

contradistinction to obstructive or hepatogenous jaundice. The experimental investigations of Minkowski and Naunyn on geese (1886) and others showed that bile was manufactured only in the liver, and that jaundice could not occur when that organ was removed from the circulation. Stadelmann (1881-3) and, later, W. Hunter, proved that the jaundice caused by toluylenediamine was really due to inflammation and obstruction by viscid bile of the small intrahepatic ducts, and it was concluded, partly from microscopic examination and partly from analogy, that the jaundice seen in various infective and toxic conditions—such as septicæmia, pneumonia, acute yellow atrophy, hæmoglobinuria, snake-bite, and poisoning by drugs—in which no obstruction was visible, was also caused by intrahepatic cholangitis. Or, as Hunter summed up in "Allbutt's System of Medicine" (1897, IV, 81): "Instead, then, of the two varieties of jaundice formerly described—one hepatogenous or obstructive, the other hæmatogenous or non-obstructive—it is necessary now to recognise one class only. All jaundice is hepatogenous, the result of absorption of bile formed and excreted by the liver." The cases in which no gross obstruction was forthcoming were spoken of as toxæmic or hæmohepatogenous. The view expressed by Hunter is that generally accepted at the present time, though, as will be shown later, the pendulum is beginning to swing back to the hæmatogenous origin as regards certain cases. Before going on to this question it may be mentioned that at different dates Frerichs, Liebermeister, Szubinski, Minkowski, Pick, and others suggested that in certain conditions, such as toxæmia, disturbance of the metabolism of the liver cells may lead to secretion of bile directly into the blood-vessels or lymphatics of the liver instead of into the bile capillaries (diffusion or acathetic jaundice, jaundice from parapedesis, paracholia). This hypothesis, which is obviously difficult or impossible to prove, was framed to explain cases of non-obstructive or toxæmic jaundice. The channel by which bile when dammed up in the liver reaches the circulation was shown by Saunders (1803), Fleischl (1874), and Vaughan

Harley (1892), to be the lymphatics, ligature of the thoracic duct preventing the jaundice which would naturally follow ligature of the common bile duct. More recent experimental work has thrown doubt on this generally accepted view, and it has been shown that a fistula of the thoracic duct does not prevent the occurrence of jaundice (Wertheimer and Lapage, Mendel and Underhill, Whipple and King).

To return to the possible hæmatogenous origin of jaundice, which has been brought up again by the recognition of a special form of chronic jaundice variously labelled as acholuric, hæmolytic with splenomegaly, familial splenomegalic cholæmia. The condition is often hereditary, familial, or may be congenital and lifelong and remarkably free from symptoms. The main exception to the last statement is that some cases have attacks of colic, which appear to be connected with small pigment calculi in the gall-bladder, as these were found in five out of six necropsies on congenital cases (Guizzetti). These calculi, however, could not have anything to do with the jaundice, as they were confined to the gall-bladders, and should be regarded as a complication. This chronic hæmolytic jaundice may also be acquired, and is then accompanied by very considerable anæmia and less jaundice than in the congenital, hereditary, and familial forms. The spleen is nearly always enlarged, and as the jaundice, never very deep, often varies and may even almost disappear for a time, the mild cases show clinical transitions between chronic splenic anæmia and hypertrophic biliary (Hanot's) cirrhosis, especially that variety in which the spleen is enlarged before the liver (metasplenomegalic biliary cirrhosis); and the acquired cases may resemble pernicious anæmia with splenic enlargement. The urine contains urobilin, but is free from bile pigment (acholuria); hence, in former times, when bile in the urine was considered proof that discoloration of the skin was due to jaundice, these cases were spoken of as "urobilin" jaundice. In 1885 Murchison gave an account of a family, a later generation of which was shown in 1909 to have this condition, as shown by recent blood tests (Hutchison and Panton).

Wilson (1890, 1893) reported a family in this country with one necropsy, and Minkowski (1900) entered with considerable detail into the subject, and recorded a necropsy in which there was no evidence of biliary obstruction. The condition did not, however, attract much attention, and the tendency was probably, if I may judge from my own attitude, to regard the jaundice as due to obstruction of the minute bile ducts in the liver. The work that really separated this from other forms of jaundice was Chauffard's discovery (1907-8) that the red blood corpuscles of such patients are fragile when exposed to hypotonic solutions of common salt, are smaller than normal, and show basophil granulations, whereas in obstructive jaundice the red blood corpuscles are more resistant to hypotonic saline solutions, and are larger than normal. These characters have been so generally confirmed that they may be regarded as diagnostic, but observations as to the presence or absence of fragility of the red blood corpuscles in the rare disease hypertrophic biliary (Hanot's) cirrhosis are necessary before the relation between it and chronic hæmolytic jaundice can be settled. An attempt to explain these cases on the lines of inflammatory obstruction in the small intrahepatic bile ducts fails from the absence of any microscopic changes in the ducts; and the suggestion that the bile is so viscid that it blocks the ducts cannot stand against the observation that in a patient on whom cholecystotomy had been performed for suspected gall-stone large quantities of normal and strikingly fluid bile were discharged. The cause of the abnormal fragility of the red blood corpuscles is unknown, but it has been thought to be due either to inadequacy of the red bone-marrow or to a poison produced by some inborn error of metabolism. It would be interesting to have some information about the resistance to hypotonic saline solutions of the red blood corpuscles in the common or so-called physiological jaundice of newly-born infants; for at birth there is an increased number of red blood corpuscles followed by a correspondingly increased hæmolysis. However brought about, this fragility of the red blood corpuscles provides an excess of hæmoglobin,

and from this bilirubin is formed, it is now supposed, in the circulation. In a recent review of the subject, Thayer and Morris have collected evidence to show that bile pigment may be found in old hæmorrhages, and on these grounds it is argued that hæmolytic jaundice is really hæmatogenous in origin. It has been suggested that hæmoglobin is transformed into bilirubin by a tryptic ferment in the presence of a carbohydrate, such as glycogen or dextrose; and that though these conditions are usually provided by the liver, the change may be carried out in the tissues. The hæmolysis, which is the essential factor in the jaundice, is not caused by a hæmolysin, but is due either simply to the fragility of the corpuscles or to the activity of the spleen (Minkowski, Banti). There does not appear to be convincing proof that the hæmolysis is mainly due to the spleen; if there was, it is obvious that splenectomy would be the proper treatment. At present there is not sufficient evidence as to the effect of splenectomy on hæmolytic jaundice, and in the hereditary, familial, and congenital cases, which hardly suffer at all from the jaundice, it would not be justifiable; but in the acquired form, in which the patient is often really ill, cures have been reported after splenectomy (Micheli, Banti). Simple drainage of the gall-bladder, which would be reasonable on the supposition that there was infection of the biliary system, has been seldom tried; in one case in which it was carried out jaundice returned after the fistula was closed. With regard to medical treatment, in the congenital, hereditary, and familial cases, there is usually little or nothing calling for relief. In acquired cases in which anæmia may be advanced iron does good, whilst arsenic has been shown to be useless. Possibly some means may be discovered by which the fragility of the red blood corpuscles can be obviated.

Pancreatic Jaundice.

The pancreas now plays a much more important part in the production of obstructive jaundice than was recognised ten years ago, mainly as the result of Mayo Robson's

advocacy. Chronic pancreatitis, except in connection with grave diabetes, attracted little or no attention until it was shown that it might by cicatricial contraction compress the common bile duct and so imitate malignant disease of the head of the pancreas; in fact, it is highly probable that some of the cases formerly described as "scirrhus" of the pancreas were inflammatory rather than carcinomatous. It is true that chronic pancreatitis does not always act in this way; the reason why it causes jaundice in some cases and not in others is that the anatomical relations of the head of the pancreas and the common bile duct are not always the same; in 62 per cent. of bodies the common bile duct is completely embedded in the head of the pancreas and so would be compressed, whereas in the remaining 38 per cent. the duct lies behind in a deep groove (Helly) and would not suffer. In jaundice due to chronic pancreatitis the clinical picture may resemble that of malignant disease obstructing the bile ducts, and in such cases Cammidge's tests are of great value in the differential diagnosis. These tests, namely, examination of the urine for the "pancreatic reaction" and analysis of the fæces especially with regard to the total quantity of fat and the relative percentages of the saponified and unsaponified fat, are complicated, and with their inventor's growing experience have undergone considerable change and extension. They have been adversely criticised, and there can be little doubt that they are more successful in their inventor's hands than in those of his critics. They require time and practice, and, generally speaking, should be carried out by an expert, and are hardly adapted for ordinary clinical work. But in my experience they may establish a diagnosis when ordinary clinical methods are inconclusive.

Chronic pancreatitis may be initiated in various ways, but is usually due to a calculus in the lower end of the common bile duct. The indurated head of the pancreas may be easily mistaken for carcinoma in the course of an operation planned for the removal of a gall stone, but abandoned under this misconception. Fortunately in some such incomplete opera-

tions manipulation of the parts appears to have extruded the calculus into the duodenum, and so to have, unintentionally so to speak, brought about a cure. It appears that chronic pancreatitis initiated by a stone in the common duct may persist and advance after the calculus has been passed. The surgical treatment of chronic pancreatitis consists in thorough drainage of the bile ducts by means of cholecystotomy or cholecystenterostomy. This procedure has also been advocated by Mayo Robson (1910) in order to prevent the late sequel of the disease, namely, diabetes. Removal is of course the proper means of dealing with calculi in the common duct; but in patients who are not fit for operation or decline it, two methods of medical treatment are worth a trial: (*a*) Urotropin, which has been shown by Crowe to be excreted into the biliary system, may be combined with sodium salicylate, which increases the flow of bile; in this way the gall-bladder and ducts can be flushed and disinfected. (*b*) A vaccine of a micro-organism obtained from the patient's stools and proved to be agglutinated by his blood serum may be given. I have employed these means with relief, but they cannot be regarded as curative or in the same light as operation.

Under the heading of *catarrhal jaundice* more than one condition is probably included. This title is most suitably applied to cases with vomiting and diarrhœa in which jaundice follows painlessly and lasts for a few weeks. But in practice, even though the manifestations of gastro-intestinal catarrh are absent, it is often employed to describe cases of mild jaundice for which no cause is obvious; and it is also given to mild cases of epidemic jaundice which are more conveniently classified as infective. The usual explanation of catarrhal jaundice is that inflammation of the mucous membrane of the duodenum spreads into the cavity of the biliary papilla or ampulla of Vater, and that the swollen mucous membrane, aided by a plug of mucus, causes obstruction. This has often been doubted, especially as opportunities for observing the actual morbid changes are rare. In a case fatal from accident

on the eighth day of the disease Eppinger (1909) found hyperplasia of the lymphoid tissues in the mucous membrane of the common bile duct where it passes through the walls of the duodenum. He compared this lymphoid tissue to the tonsil and suggested that its function is to protect the duct against infection from the intestine, and drew an analogy between catarrhal jaundice and tonsillitis. According to Mayo Robson (1904), prolonged catarrhal jaundice is pancreatic in origin, and is due to pressure exerted on the terminal part of the common bile duct by the head of the pancreas, which is enlarged from catarrhal inflammation. This was shown to exist in fifteen cases of prolonged catarrhal jaundice in which the abdomen was opened; further, a positive "pancreatic reaction" in the urine was found in forty-two out of fifty-three cases (or 79 per cent.) of catarrhal jaundice (Cammidge, 1910). Whilst it is not clear why jaundice is so extremely rare in cases of severe acute pancreatitis such as give rise to the symptoms now sometimes described by the phrase "the acute abdomen," there seems to be good evidence that some cases of so-called catarrhal jaundice are pancreatic in origin.

Jaundice may, of course, be due to other diseases of the pancreas, such as carcinoma, pancreatic and peripancreatic cysts, large calculi in Wirsung's duct compressing the termination of the common bile duct, hydatid cysts arising in the head of the gland, or gummatous disease. These, except the first two, are extremely rare. Two clinical features, though far from constantly present, have been specially correlated by Mayo Robson with jaundice of pancreatic origin: they are pruritus and hæmorrhages.

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