cardiorespiratory disease in an infant or child with hyperventilation who is dehydrated or is a known diabetic ; and the possibility that the overventilation is due to salicylates or other drugs must always be borne in mind.

Gilbert's Syndrome

Bilirubin is formed from broken-down haemoglobin in the reticuloendothelial cells and passes in the blood to the liver. There it becomes conjugated with glucuronic acid by liver cells and in that form enters the bile. Various diseases can cause a chronic excess of unconjugated bilirubin in the bloodstream, with consequent jaundice. Among them are infections, heart disease, and hepatic and biliary disease.[1] This so-called unconjugated hyperbilirubinaemia can also be of congenital origin, and the commonest variety is Gilbert's syndrome.[2]

It was first described by A. Gilbert and P. Lereboullet in 1901[3] as a benign familial disorder occurring in the absence of overt haemolysis or other evidence of hepatocellular dysfunction. The mildness of the jaundice (serum bilirubin levels of 1–6 mg./100 ml.) makes the disease difficult to trace in a family from the history alone, and in only a few studies have the relatives been systematically examined by serum bilirubin estimations.[4] Such a study has recently been reported by L. W. Powell and his colleagues,[5] who examined 122 first-degree relatives of 42 patients with Gilbert's syndrome. They found raised serum bilirubin values in 16% of the propositi and 27% of the siblings and concluded that the disorder was inherited as an autosomal dominant. In another recent study P. M. Smith and his colleagues[6] found evidence of dominant transmission in a group of patients with Gilbert's syndrome whose serum bilirubin values ranged from 2.4 to 4.2 mg./100 ml. However, in a group with lower values (1.2–2.2 mg./100 ml.) they could find no evidence of the disease in the families, and they suggested that the disorder in those patients might be acquired.

The problem of whether Gilbert's syndrome is a single entity or a syndrome of varied aetiology is unlikely to be resolved until the nature of the defect in bilirubin metabolism is established. All attempts to show that bilirubin conjugation is impaired by a decrease in glucuronyl transferase activity have failed, though this may be the cause of the more severe cases of unconjugated hyperbilirubinaemia with serum bilirubin levels of 6.4–19.9 mg./100 ml.[7] B. H. Billing and her colleagues[8] have produced indirect evidence that the defect lies in an uptake of bilirubin in the cell. But further work is needed. A slight reduction in red-cell survival has been found in about half the patients.[9] I. M. Arias[7] separates such patients into a separate group called "compensated haemolytic disease," but this degree of haemolysis is slight and is insufficient to cause jaundice in the absence of simultaneous impairment of the capacity of the liver to remove bilirubin. Furthermore, some reduction in red-cell survival is found in many other varieties of jaundice, suggesting that it may be a secondary effect.

There are many other features of Gilbert's syndrome that need to be explained. Why do patients with such mild jaundice so frequently have symptoms ? Fatigue and nausea, with abdominal pain or discomfort in the right upper quadrant of the abdomen, affected 69% of the patients in the series of L. W. Powell and his colleagues.[5] The symptoms and depth of jaundice tend to become more marked during infections, after taking alcohol, or after strenuous exercise,[10] and it is perhaps not surprising that these patients are diagnosed as having either chronic or relapsing hepatitis. The symptoms may be related to the chronic anxiety state that results, since the relatives who are shown to have raised levels of serum bilirubin have rarely had symptoms. The full investigation of suspected cases of Gilbert's syndrome, including liver biopsy to show normal hepatic histology, is therefore essential, for in this way symptoms may be allayed and unwarranted invalidism prevented.

Anterior Resection for Rectal Carcinoma

About three-quarters of the cancers of the large bowel are situated in the rectum and rectosigmoid. The introduction of the abdominoperineal excision of the rectum by W. E. Miles in 1908, based as it was on a careful consideration of the lymphatic spread of the tumour in outward, downward, and upward directions, was an immense step forward in the management of these growths.[1] Yet it was a victory gained only at the expense of a permanent colostomy. In the nineteen thirties and forties surgeons began to explore the possibility of a more conservative approach with conservation of the anal sphincters. Such procedures included first the abdominosacral resection,[2] then various types of abdominoanal pull-through procedures,[3 4] and finally the anterior resection of the rectum, developed particularly at the Mayo Clinic.[5] The problems of sacral fistulae with the first and anal incontinence with the second have largely relegated these operations to surgical history, but the anterior resection with colorectal anastomosis has passed firmly into the surgeon's armamentarium.

An important stimulus in these developments, quite apart from the desirability of avoiding a colostomy, was the demonstration that the tumour rarely spreads more than 2 cm. beyond the lower border of the growth. Hence an excision of 5 cm. of distal bowel could be considered to be adequate.[6–8] Lateral spread of the tumour is more prone to occur when the lesion is situated in the infraperitoneal portion of the rectum. Naunton Morgan[9] found that this type of spread occurred in 14.5% of cases after excision of tumours of the lower third of rectum, but in only 5.2% of cases when the

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