

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