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 recognize 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