Occasionally jaundice sets in and persists for many weeks, or even months, without interfering seriously with the nutrition of the child.

3. Acute Yellow Atrophy of the Liver; Malignant Jaundice; Icterus Gravis.

Definition.—Jaundice associated with marked cerebral symptoms and characterized anatomically by extensive necrosis of the liver-cells with reduction in volume of the organ.

Etiology.—This is a rare disease. In a somewhat varied post-mortem and clinical experience no instance has fallen under my observation. On the other hand, a physician may see several cases within a few years, or even within a few months, as happened to Riess, who saw five cases within three months at the Charité, in Berlin. The disease seems to be rare in this country. No case is reported in the Transactions of the Pathological Societies of New York (Vols. I to III) or of Philadelphia (Vols. I to XIII). The disease is more common in women than in men. Of the 100 cases collected by Legg, 69 were in females; and of Thierfelder's 143 cases, 88 were in women. There is a remarkable association between the disease and pregnancy, which was present in 25 of the 69 women in Legg's statistics, and in 33 of the 88 women in Thierfelder's collection. It is most common between the ages of twenty and thirty, but is occasionally seen in young children. It has followed fright or profound mental emotion. Though the symptoms produced by phosphorus poisoning closely simulate those of acute yellow atrophy, the two conditions are not identical.

Morbid Anatomy.—The liver is greatly reduced in size, looks thin and flattened, and sometimes does not reach more than one half or even one third of its normal weight. It is flabby and the capsule is wrinkled. On section the color is of a yellowish brown, yellowish red, or mottled, and the outlines of the lobules are indistinct. The yellow and dark-red portions represent different stages of the same process—the yellow an earlier, the red a more advanced stage. The organ may cut with considerable firmness. Microscopically the liver-cells are seen in all stages of necrosis, and in spots appear to have undergone complete destruction, leaving a fatty, granular *débris* with pigment grains and crystals of leucin and tyrosin. The interlobular tissue may be normal, but in many cases there is a marked proliferation of small cells, which was present in 9 of the 12 cases examined by Riess. Micro-organisms have been noted by several observers. The bile-ducts and gall-bladder are empty.

The other organs show extensive bile staining, and there are numerous hæmorrhages. The kidneys may show marked granular degeneration of the epithelium, and usually there is fatty degeneration of the heart. In a majority of the cases the spleen is enlarged.

Symptoms.—In the initial stage there is a gastro-duodenal catarrh, and at first the jaundice is thought to be of a simple nature. In some instances this lasts only a few days, in others two or three weeks. Then severe symptoms set in—headache, delirium, trembling of the muscles, and, in some instances, convulsions. Vomiting is a constant symptom, and blood may be brought up. Hæmorrhages occur into the skin or from the mucous surfaces; in pregnant women abortion may occur. With the development of the head symptoms the jaundice usually increases. Coma sets in and gradually deepens until death. The body temperature is variable; in a majority of the cases the disease runs an afebrile course, though sometimes just before death there is an elevation. In some instances, however, there has been marked pyrexia. The pulse is usually rapid, the tongue coated and dry, and the patient is in a "typhoid state."

The urine is bile-stained and often contains tube-casts. Leucin and tyrosin are constantly present; the former as rounded disks, the latter in needle-shaped crystals, arranged either in bundles or in groups. The tyrosin may sometimes be seen in the urine sediment, but it is best first to evaporate a few drops of urine on a cover-glass. In the majority of cases no bile enters the intestines, and the stools are clay-colored. The disease is almost invariably fatal. In a few instances recovery has been noted. I saw in Leube's clinic, at Wurzburg, a case which was convalescent.

Diagnosis.—Jaundice with delirium, diminution of the liver volume, delirium, and the presence of leucin and tyrosin in the urine, form a characteristic and unmistakable group of symptoms.

It is not to be forgotten that any severe jaundice may be associated with intense cerebral symptoms. The clinical features in certain cases of hypertrophic cirrhosis are almost identical, but the enlargement of the liver, the more constant occurrence of fever, and the absence of leucin and tyrosin are distinguishing signs. Phosphorus poisoning may closely simulate acute yellow atrophy, particularly in the hæmorrhages, jaundice, and the diminution in the liver volume, but the gastric symptoms are usually more marked, and leucin and tyrosin are stated not to occur in the urine.

No known remedies have any influence on the course of the disease.

II. AFFECTIONS OF THE BLOOD-VESSELS OF THE LIVER.

(1) **Anæmia.**—On the post-mortem table, when the liver looks anæmic, as in the fatty or amyloid organ, the blood-vessels, which during life were probably well filled, can be readily injected. There are no symptoms indicative of this condition.

(2) **Hyperæmia.**—This occurs in two forms. (*a*) *Active hyperæmia.* After each meal the rapid absorption by the portal vessels induces transient congestion of the organ, which, however, is entirely physiological; but it is quite possible that in persons who persistently eat and drink too much

this active hyperæmia may lead to functional disturbance or, in the case of drinking too freely of alcohol, to organic change.

The *symptoms* of active hyperæmia are indefinite. Possibly the sense of distress or fulness in the right hypochondrium, so often mentioned by dyspeptics and by those who eat and drink freely, may be due to this cause. There are probably diurnal variations in the volume of the liver. In cirrhosis with enlargement the rapid reduction in volume after a copious hæmorrhage indicates the important part which hyperæmia plays even in organic troubles. It is stated that suppression of the menses or suppression of a hæmorrhoidal flow is followed by hyperæmia of the liver. Andrew H. Smith has described a case of periodical enlargement of the liver.

(*b*) *Passive Congestion.*—This is much more common and results from an increase of pressure in the efferent vessels or sub-lobular branches of the hepatic veins. Every condition leading to venous stasis in the right heart at once affects these veins.

In chronic valvular disease, in emphysema, cirrhosis of the lung, and in intrathoracic tumors mechanical congestion occurs and finally leads to very definite changes. The liver is enlarged, firm, and of a deep-red color; the hepatic vessels are greatly engorged, particularly the central vein in each lobule and its adjacent capillaries. On section the organ presents a peculiar mottled appearance, owing to the deeply congested hepatic and the anæmic portal territories; hence the term *nutmeg* which has been given to this condition. Gradually the distention of the central capillaries reaches such a grade that atrophy of the intervening liver-cells is induced. Brown pigment is deposited about the centre of the lobules and the connective tissue is greatly increased. In this cyanotic induration or cardiac liver the organ is large in the early stage, but later it may become contracted. Occasionally in this form the connective tissue is increased about the lobules as well, but the process usually extends from the sublobular and central veins.

The symptoms of this form are not always to be separated from those of the associated conditions. Gastro-intestinal catarrh is usually present and hæmatemesis may occur. The portal obstruction in advanced cases leads to ascites, which may precede the development of general dropsy. There is often slight jaundice, the stools may be clay-colored, and the urine contains bile-pigment.

On examination the organ is found to be increased in size. It may be a full hand's-breadth below the costal margin and tender on pressure. It is in this condition particularly that we meet with pulsation of the liver. We must distinguish the communicated throbbing of the heart, which is very common, from the heaving, diffuse impulse due to regurgitation into the hepatic veins, in which, when one hand is upon the ensiform cartilage and the other upon the right side at the margin of the ribs, the whole liver can be felt to dilate with each impulse.

The indications for *treatment* in passive hyperæmia are to restore the balance of the circulation and to unload the engorged portal vessels. In cases of intense hyperæmia eighteen or twenty ounces of blood may be directly aspirated from the liver, as advised by George Harley and practised by many Anglo-Indian physicians. Good results sometimes follow this hepato-phlebotomy. The prompt relief and marked reduction in the volume of the organ which follow an attack of hæmatemesis or bleeding from piles suggests this practice. Salts administered by Matthew Hay's method deplete the portal system freely and thoroughly. As a rule, the treatment must be that of the condition with which it is associated.

(3) **Diseases of the Portal Vein.**—(*a*) *Thrombosis; Adhesive Pylephlebitis.*—Coagulation of blood in the portal vein is rarely seen except in cirrhosis. Exceptional causes are invasion of the branches by cancer, proliferative peritonitis involving the gastro-hepatic omentum, and perforation of the vein by gall-stones. In rare instances a complete collateral circulation is established, the thrombus undergoes the usual changes, and ultimately the vein is represented by a fibrous cord, a condition which has been called *pylephlebitis adhesiva.* In a case of this kind which I dissected the portal vein was represented by a narrow fibrous cord; the collateral circulation, which must have been completely established for years, ultimately failed, ascites and hæmatemesis supervened and rapidly proved fatal.* The diagnosis of obstruction of the portal vein can rarely be made. A suggestive symptom, however, is a *sudden* onset of the most intense engorgement of the branches of the portal system.

Emboli in the branches of the portal vein do not, as a rule, produce infarction, for blood reaches the lobular capillary plexus, as shown by Cohnheim and Litten, through the free anastomosis with the hepatic artery. In rare instances, however, a condition resembling infarction does occur, sometimes in small areas, at others in quite extensive territories. Septic emboli, on the other hand, may induce suppuration.

(*b*) *Suppurative pylephlebitis* will be considered in the section on abscess.

(4) **Affections of the hepatic vein** are extremely rare. Dilatation occurs in cases of chronic enlargement of the right heart, from whatever cause produced. Emboli occasionally pass from the right auricle into the hepatic veins. A rare and unusual event is stenosis of the orifices of the hepatic veins, which I met in a case of fibroid obliteration of the inferior vena cava and was associated with a greatly enlarged and indurated liver.†

(5) **Hepatic Artery.**—Enlargement of this vessel is seen in cases of cirrhosis of the liver. It may be the seat of extensive sclerosis. Aneurism of the hepatic artery is rare, but instances are on record, and will be referred to in the section on arteries.

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* Journal of Anatomy and Physiology, vol. xvii.

† Ibid., vol. xvi.