JAUNDICE.*

Jaundice, a visible staining of the tissues with bile pigments, is a symptom more frequently encountered in diseases of tropical and sub-tropical areas than in Western medical practice because of the greater frequency of diseases which cause a high degree of blood destruction or exert their toxic effects upon the cells of the liver or of the reticuloendothelial system.

Normally bilirubin is present in the blood in a concentration of 1 in 250,000. If for any reason the bilirubin increases to a concentration of about 1 in 50,000, it begins to be excreted into the urine, and at about the same point jaundice becomes clinically obvious. This condition of hyperbilirubinæmia may arise in three ways :—

(a) Obstructive Jaundice.

Obstruction within the bile ducts causes a rise of pressure, and the bile excreted by the hepatic cells is reabsorbed by the hepatic capillaries. This obstruction may occur in the finest bile capillaries, in the medium-sized bile ducts, or in the extra-hepatic bile duct system (Hepatic duct, common bile duct). The obstruction may be due to causes within the ducts (*e.g.*, gall stones) or in the wall of the duct (cholangitis, congenital obliteration), or be due to pressure on the duct from outside (tumour, gumma, hydatid, cirrhosis, etc).

In obstructive jaundice all the constituents of the bile are retained in the body, viz., bile pigment (causing jaundice) ; bile salts (causing itching and bradycardia) ; cholesterol (causing xanthoma). In addition, absence of the bile from the intestine interferes with the absorption of vitamin K, causing hyporothrombinæmia and a consequent tendency to hæmorrhage.

The bile pigment manufactured in the reticuloendothelial system (largely outside the liver) cannot, by reason of the obstruction, be excreted by the normal channels. Consequently, the stools are pale and they are also bulky with excess of fats from malabsorption due to the absence of bile salts. The urine contains abundant bile pigment and bile acids. Bile pigment may be found in the urine before jaundice is evident clinically in the skin or conjunctivæ. The van Bergh reaction is direct positive.

(b) Hæmolytic Jaundice.

This is due to excessive destruction of the red blood cells, leading to the formation by the cells of the reticuloendothelial system of bilirubin in excess of the quantity that the liver cells, themselves healthy, can excrete. In consequence, jaundice develops, due solely to hyperbilirubinæmia. Bile pigment is never present in the urine in true hæmolytic jaundice, but only urobilin. The van Bergh reaction is indirect.

This type of jaundice is seen in pernicious anæmia, acholuric jaundice, severe M.T. malaria, blackwater fever, transfusion with incompatible blood, and in the action of the venom of certain snakes.

(c) Toxic Jaundice.

The essential lesion is damage to the glandular cells of the liver, usually accompanied by damage to the hepatic ducts. The bilirubin carried to the liver cannot be there further elaborated, owing to the damage to the glandular cells, and, owing to the blockage of the ducts, any bile that is formed is obstructed in its escape. Bile pigment is generally present in the urine of cases of this type, but in the later stages urobilin alone may be found.

The van Bergh reaction is biphasic.

This group is a large one and includes the jaundice that may occur in all forms of hepatitis, acute, subacute or chronic, and the causative agencies are very varied.

These may be classified as :—

(A) Chemical.

1. Organic : Trinitrotoluene, tetrachlorethane, carbon tetrachloride, chloroform, alcohol and cinchophen.
2. Inorganic: Arsenic, phosphorus, gold.

(B) Organismal.

1. Bacterial : Typhoid, especially paratyphoid B, typhus, pneumonia, septicæmia and pyæmia.
2. Spirochætal : Syphilis, relapsing fever, Weil's disease.

(C) Virus Diseases.

These include yellow fever, and probably epidemic infective hepatitis.

(D) Unknown Agencies.

As in eclampsia, some cases of acute yellow atrophy, and possibly some classified as catarrhal jaundice.

With this etiology the degree of damage to the liver cells and the degree of associated primary or secondary cholangitis is very inconstant, and the intensity of the jaundice, the amount of bile in the urine and the colour of the stools may vary within wide limits.

The remainder of this section is devoted to two conditions in which jaundice is the predominant symption, and in which no specific etiological agency has been identified.

Catarrhal Jaundice.

Cases occur in all climates of sporadic "catarrhal jaundice," said to be associated with a catarrhal gastro-duodenitis which causes œdema at the mouth of the common bile duct and in the ampulla of Vater, and with exudation of thick mucus, consequent obstruction of the duct. The onset is usually associated with gastric symptoms, anorexia, nausea and sometimes vomiting, which precede the jaundice by a few days.

The van den Bergh reaction is a prompt direct positive, and such cases are generally considered truly obstructive and catarrhal in origin. This condition affects chiefly adolescents and young adults, and lasts for a variable period, perhaps a week or two, after which the jaundice gradually fades. A light diet, rich in carbohydrate and poor in fat, is usually prescribed, along with a morning saline, and recovery is the rule.

There are also encountered cases which, although apparently sporadic, show evidence of a toxic agency. The liver is often found to be tender and enlarged, and the van den Bergh reaction is of the biphasic type commonly found in cases of toxic jaundice. Occasionally in Great Britain, and more commonly in other countries, temperate as well as tropical, small localised outbreaks of jaundice of this type are met with, individual cases being clinically indistinguishable from the sporadic catarrhal jaundice mentioned above. There is a growing trend of opinion towards considering these to be of a toxic type due to a communicable infection probably with a virus.

Recent investigations in Denmark by means of

* Reprinted from *The Journal of the Western Australian Nurses.*

previous page next page