Definition

Jaundice is a condition in which a person's skin and the whites of the eyes are discolored yellow due to an increased level of bile pigments in the blood resulting from liver disease. Jaundice is sometimes called icterus, from a Greek word for the condition.

Description

In order to understand jaundice, it is useful to know about the role of the liver in producing bile. The most important function of the liver is the processing of chemical waste products like cholesterol and excreting them into the intestines as bile. The liver is the premier chemical factory in the body—most incoming and outgoing chemicals pass through it. It is the first stop for all nutrients, toxins, and drugs absorbed by the digestive tract. The liver also collects chemicals from the blood for processing. Many of these outward-bound chemicals are excreted into the bile. One particular substance, bilirubin, is yellow. Bilirubin is a product of the breakdown of hemoglobin, which is the protein inside red blood cells. If bilirubin cannot leave the body, it accumulates and discolors other tissues. The normal total level of bilirubin in blood serum is between 0.2 mg/dL and 1.2 mg/dL. When it rises to 3 mg/dL or higher, the person's skin and the whites of the eyes become noticeably yellow.

Bile is formed in the liver. It then passes into the network of hepatic bile ducts, which join to form a single tube. A branch of this tube carries bile to the gallbladder, where it is stored, concentrated, and released on a signal from the stomach. Food entering the stomach is the signal that stimulates the gallbladder to release the bile. The tube, which is now called the common bile duct, continues to the intestines. Before the common bile duct reaches the intestines, it is joined by another duct from the pancreas. The bile and the pancreatic juice enter the intestine through a valve called the ampulla of Vater. After entering the intestine, the bile and pancreatic secretions together help in the process of digestion.

Causes and symptoms

There are many different causes for jaundice, but they can be divided into three categories based on where they start—before, in, or after the liver (pre-hepatic, hepatic and post-hepatic). When bilirubin begins its life cycle, it cannot be dissolved in water. The liver changes it so that it is soluble in water. These two types of bilirubin are called unconjugated (insoluble) and conjugated (soluble). Blood tests can easily distinguish between these two types of bilirubin.

Hemoglobin and bilirubin formation

Bilirubin begins as hemoglobin in the blood-forming organs, primarily the bone marrow. If the production of red blood cells (RBCs) falls below normal, the extra hemoglobin finds its way into the bilirubin cycle and adds to the pool.

Once hemoglobin is in the red cells of the blood, it circulates for the life span of those cells. The hemoglobin that is released when the cells die is turned into bilirubin. If for any reason the RBCs die at a faster rate than usual, bilirubin can accumulate in the blood and cause jaundice

Hemolytic disorders

Many disorders speed up the death of red blood cells. The process of red blood cell destruction is called hemolysis, and the diseases that cause it are called hemolytic disorders. If red blood cells are destroyed faster than they can be produced, the patient develops anemia. Hemolysis can occur in a number of diseases, disorders, conditions, and medical procedures:
Malaria. The malaria parasite develops inside red blood cells. When it is mature it breaks the cell apart and swims off in the blood. This process happens to most of the parasites simultaneously, causing the intermittent symptoms of the disease. When enough cells burst at once, jaundice may result from the large amount of bilirubin formed from the hemoglobin in the dead cells. The pigment may reach the urine in sufficient quantities to cause "blackwater fever," an often lethal form of malaria.

Side effects of certain drugs. Some common drugs can cause hemolysis as a rare but sudden side effect. These medications include some antibiotic and anti-tuberculosis medicines; drugs that regulate the heartbeat; and levodopa, a drug used to treat Parkinson's disease.
Certain drugs in combination with a hereditary enzyme deficiency known as glucose-6-phosphate dehydrogenase (G6PD). G6PD is a deficiency that affects over 200 million people in the world. Some of the drugs listed above are more likely to cause hemolysis in people with G6PD. Other drugs cause hemolysis only in people with this disorder. Most important among these drugs are anti-malarial medications such as quinine, and vitamins C and K.
Poisons. Snake and spider venom, certain bacterial toxins, copper, and some organic industrial chemicals directly attack the membranes of red blood cells.
Artificial heart valves. The inflexible moving parts of heart valves damage RBCs as they flutter back and forth. This damage is one reason to recommend pig valves and valves made of other organic materials.
Hereditary RBC disorders. There are a number of hereditary defects that affect the blood cells. There are many genetic mutations that affect the hemoglobin itself, the best-known of which is sickle cell disease. Such hereditary disorders as spherocytosis weaken the outer membrane of the red cell. There are also inherited defects that involve the internal chemistry of RBCs.
Enlargement of the spleen. The spleen is an organ that is located near the upper end of the stomach and filters the blood. It is supposed to filter out and destroy only worn-out RBCs. If it has become enlarged, it filters out normal cells as well. Malaria, other infections, cancers and leukemias, some of the hereditary anemias mentioned above, obstruction of blood flow from the spleen—all these and many more diseases can enlarge the spleen to the point where it removes too many red blood cells.
Diseases of the small blood vessels. Hemolysis that occurs in diseased small blood vessels is called microangiopathic hemolysis. It results from damage caused by rough surfaces on the inside of the capillaries. The RBCs squeeze through capillaries one at a time and can easily be damaged by scraping against the vessel walls.
Immune reactions to RBCs. Several types of cancer and immune system diseases produce antibodies that react with RBCs and destroy them. In 75% of cases, this reaction occurs all by itself, with no underlying disease to account for it.
Transfusions. If a patient is given an incompatible blood type, hemolysis results.
Kidney failure and other serious diseases. Several diseases are characterized by defective blood coagulation that can destroy red blood cells.
Erythroblastosis fetalis. Erythroblastosis fetalis is a disease of newborns marked by the presence of too many immature red blood cells (erythroblasts) in the baby's blood. When a baby's mother has a different blood type, antibodies from the mother may leak into the baby's circulation and destroy blood cells. This reaction can produce severe hemolysis and jaundice in the newborn. Rh factor incompatibility is the most common cause.
High bilirubin levels in newborns. Even in the absence of blood type incompatibility, the newborn's bilirubin level may reach threatening levels.

Normal jaundice in newborns

Normal newborn jaundice is the result of two conditions occurring at the same time—a pre-hepatic and a hepatic source of excess bilirubin. First of all, the baby at birth immediately begins converting hemoglobin from a fetal type to an adult type. The fetal type of hemoglobin was able to extract oxygen from the lower levels of oxygen in the mother's blood. At birth the infant can extract oxygen directly from his or her own lungs and does not need the fetal hemoglobin any more. So fetal hemoglobin is removed from the system and replaced with adult hemoglobin. The resulting bilirubin loads the system and places demands on the liver to clear it. But the liver is not quite ready for the task, so there is a period of a week or so when the liver has to catch up. During that time the baby is jaundiced.

Hepatic jaundice

Liver diseases of all kinds threaten the organ's ability to keep up with bilirubin processing. Starvation, circulating infections, certain medications, hepatitis, and cirrhosis can all cause hepatic jaundice, as can certain hereditary defects of liver chemistry, including Gilbert's syndrome and Crigler-Najjar syndrome.

Post-hepatic jaundice

Post-hepatic forms of jaundice include the jaundices caused by failure of soluble bilirubin to reach the intestines after it has left the liver. These disorders are called obstructive jaundices. The most common cause of obstructive jaundice is the presence of gallstones in the ducts of the biliary system. Other causes have to do with birth defects and infections that damage the bile ducts; drugs; infections; cancers; and physical injury. Some drugs—and pregnancy on rare occasions—simply cause the bile in the ducts to stop flowing.

Symptoms and complications associated with jaundice

Certain chemicals in bile may cause itching when too much of them ends up in the skin. In newborns, insoluble bilirubin may get into the brain and do permanent damage. Long-standing jaundice may upset the balance of chemicals in the bile and cause stones to form. Apart from these potential complications and the discoloration of skin and eyes, jaundice by itself is inoffensive. Other symptoms are determined by the disease producing the jaundice.