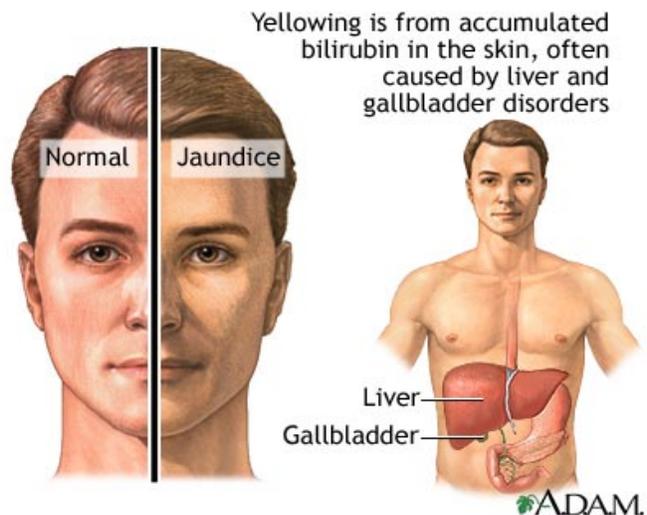


## Jaundice

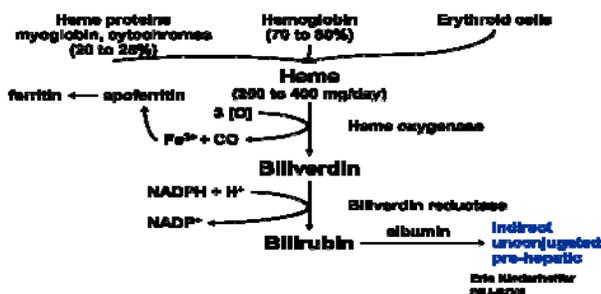
The human body, a collection of complex mechanisms, running smoothly without much input from its operator, finds a way to showcase its shortcomings whenever they arise. Pain or discomfort or the inability to do something in our everyday life has us running to the ‘mechanic’ while the more subtle signs are allowed to linger. One of the most striking indications of a problem within is a change in pigmentation, and jaundice is a common and rather explicit, though not very subtle clue that something is wrong.

Jaundice, a pigmentation (“*jaune*”, French: “yellow”) of the skin and mucosal membranes, specifically, conjunctiva overlying the sclera of the eyes, is due to the increased level of bilirubin in the blood, which dissolves in the subcutaneous adipose tissues giving the skin the characteristic yellow color. In order to understand the potential cause of hyperbilirubinemia, one must understand the physiology of the production and excretion of bilirubin to see the probable challenges along the pathway.

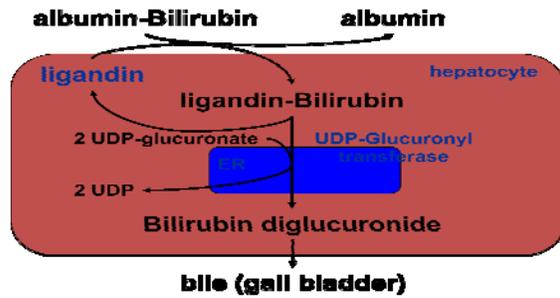


During regular hemolysis, most of the parts of the erythrocytes are recycled, and some, in particular, the heme group of the hemoglobin, is not. It goes through several oxidation/reduction reactions to become bilirubin, a molecule that is very poorly soluble in water, and thus, must be transported while in the bloodstream. This ‘free’ bilirubin is transported to the liver, where it is conjugated - joined with a glucuronic acid to be more water soluble. This, now ‘conjugated’ bilirubin is now free to leave the liver with the bile via the common bile duct to eventually make its way to the duodenum, where it can take one of two pathways: either be changed into stercobilin and be excreted with feces or be reabsorbed by the intestinal cells and be transported to the kidneys and be excreted as urobilin in the urine.

### Bilirubin Production

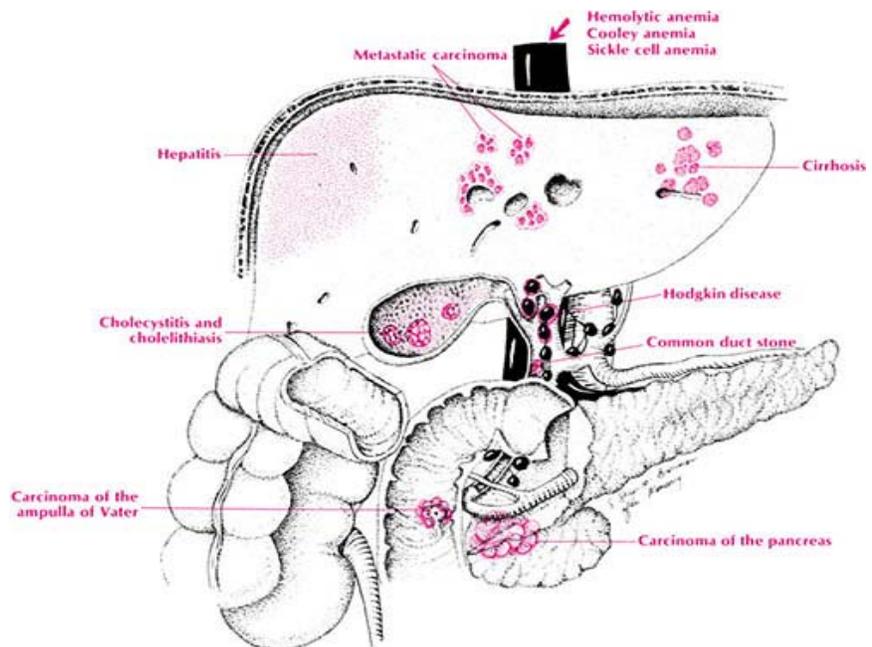


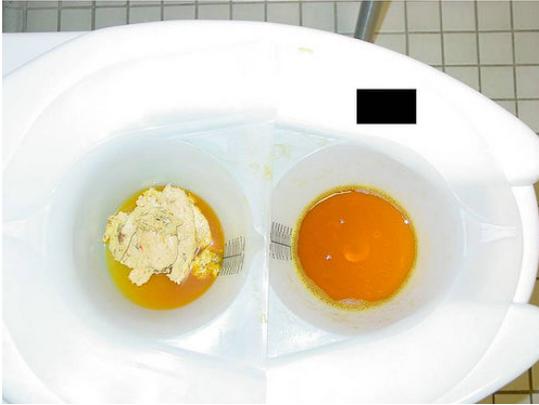
# Bilirubin Processing



A problem anywhere along this tract – increased production, decreased conjugation, decreased secretion by the liver, or blockage of the ducts – can lead to the buildup of bilirubin in the serum that would eventually lead to jaundice. In an attempt to categorize the causes, they are broken down from the perspective of where the problem occurred in relationship to the liver. If, for example the problem is due to the increased production of bilirubin, (by the increased erythrocyte hemolysis due to malaria or hemolytic anemia), it is considered ‘pre hepatic’; if the problem is with the conjugation or the secretion of the bilirubin, and thus, has occurred in the liver, it is ‘hepatic’, and, if after – ‘post hepatic’, such as the very common problem of gallstones or pancreatic cancer, both of which contribute to obstruction of the common duct (before the duodenum).

Physical representation of where the pathology may occur:





Since bilirubin (its forms) is what gives the feces and urine its color, a decrease in excretions would be another sign of jaundice – feces become a striking pasty ‘clay’ white color while the urine is very dark yellow-brown. (On picture, the sample on the left has the white stool while the sample on the right is the darker urine only)

There are many diseases whose symptoms include jaundice – see summary table.

Jaundice described above is pathological: a symptom of a disease. Another type of jaundice, physiological, is common in newborn infants and is considered completely normal (around 70% of infants exhibit signs). Upon birth, fetal hemoglobin is being destroyed and replaced by adult hemoglobin, taxing a new liver with an immature pathway: the increase of hemoglobin destruction leads to build up of bilirubin, causing temporary jaundice.



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