"WHAT IS PHOTOTHERAPY?"

AN INFORMATION AID FOR THE EME REPRESENTATIVE

A BASIC FACT SHEET, WRITTEN WITH A MINIMUM OF MEDICAL TERMINOLOGY TO HELP YOU - THE EME REPRESENTATIVE - FAMILIARISE YOURSELF WITH THE PHOTOTHERAPY TREATMENT OF HYBERBILIRUBINEMIA

First, some basic definitions:

BILIRUBIN - a red pigment released from hemoglobin during the normal and also abnormal destruction of the red blood cells. (erythrocytes).

BILIRUBINEMIA - the presence of bilirubin in the circulating blood, where it is normally present in relatively small amounts.

HYPERBILIRUBINEMIA - an abnormally large amount of bilirubin in the circulating blood resulting in clinically apparent jaundice (icterus). A serum bilirubin level concentration of over 15 mg/ 100 ml of blood is now being generally defined as hyperbilirubinemia. At one time a serum bilirubin concentration of 20 mg/100 ml of blood was considered a "safe" level; however, recent tests reported by Dr. Jerold Lucey of the University of Vermont, have shown concentrations as low as 12 mg to 15 mg in sick premature infants can be toxic to human brain cells and cause kernicterus (nuclear jaundice).

JAUNDICE (ICTERUS) - a yellowish staining of the skin and mucous membranes and the excretions due to excessive bile pigments in the circulating blood.

KERNICTERUS (NUCLEAR JAUNDICE) - a grave clinical condition marked by deposit of bilirubin in the nuclei of the brain and spinal cord. Degeneration of the nerve cells (grey matter of the central nervous system) occurs and paralysis and death soon follow.

EXCHANGE TRANSFUSION - previous to phototherapy, this was the treatment of kernicterus. Mortality risk for exchange transfusions with sick infants has been 1%.

PHOTOTHERAPY - a method of reducing the bilirubin level in the premature infant by "bathing" him in broad-spectrum fluorescent light of 100 to 500+ candles which contains energy within the blue spectral energy range of 420 to 460 nanometers. The light produces a photochemical breakdown of the bilirubin as the blood passes through the superficial capillaries of the skin. This broken down bilirubin is then reduced to non-toxic substances which are rapidly excreted in the urine and bile.

Those are the only terms and definitions we need be familiar with now. From them, we know that hyperbilirubinemia occurs when there is too much bilirubin in the blood. We know that bilirubin is obtained from hemoglobin which is obtained from the destruction of the red blood cells. But what causes this chain of events leading to hyperbilirubinemia to occur in approximately 1/5 of all prematurely born infants?

The most frequent causes of hyperbilirubinemia fall into either of two categories: (1) hyperbilirubinemia caused by a nonhemolytic condition or (2) hyperbilirubinemia caused by a hemolytic disease (a disease which destroys the red blood cells and releases hemoglobin).

First, let's examine hyperbilirubinemia caused by a nonhemolytic condition. This would include hyperbilirubinemia in sick premature infants who have anoxia, respiratory distress, acidosis, sepis, or hypoalbuminemia and are receiving drugs. It would also include a condition known as Hyperbilirubinemia of Prematurity. This condition is marked by fragile red blood cells and/or an under-developed liver. It is best explained by comparing a normal,full term baby with a premature infant in this condition.

FULL TERM	PREMATURE
(1) Normal red blood cells	(1) In premature infants, red blood cells are considerably more fragile.
(2) Red blood cells break down and release hemoglobin.	(2) Because of fragility, an excessive amount of red blood cells may break down and release hemoglobin.
(3) The hemoglobin breaks down into bilirubin and globin.	(3) The hemoglobin breaks down into bilirubin and globin.
(4) Liver has developed.	(4) The premature infant's liver is not yet fully developed.
(5) The liver detoxifies the bilirubin.	(5) The liver is incapable of detoxifying <u>all</u> the bilirubin.
(6) The detoxified product is excreted through the infant's kidneys and gastrointestinal tract.	(6) The detoxified product is excreted through the infant's kidneys and gastrointestinal tract.
	(7) Excess bilirubin accumulates in the bloodstream and is deposited in the brain.

In this - HYPERBILIRUBINEMIA OF PREMATURITY - and in other nonhemolytic cases, phototherapy has proven itself effective in both the prophylaxis (prevention) of hyperbilirubinemia in susceptible infants, 1,2 and in the therapeutic reduction of high bilirubin levels in infants with existing hyperbilirubinemia. 3-7

A word of caution must be added. Before phototherapy, the jaundiced skin will appear yellowish. Once phototherapy begins, the bilirubin visible in the skin will be the first bilirubin broken down and discharged in the urine and bile. Therefore, the jaundiced (yellowish) skin colour may disappear before phototherapy has entirely corrected the situation. An infant may still have a serum bilirubin level as high as 8 mg % to 12 mg % and appear to the eye to be non-jaundiced. Hence, it becomes mandatory to follow any infant placed on light therapy with actual tests of serum bilirubin rather than "visual estimates".

The other category - HYPERBILIRUBINEMIA ASSOCIATED WITH HEMOLYTIC DISEASES - is where you as an EME Representative must exercise caution in your statements. HYPERBILIRUBINEMIA IS NOT A HEMOLYTIC DISEASE - it is, however, often a sympton of one. Phototherapy will not cure a hemolytic disease; it will however, in mild and moderate cases of hemolytic disease, curtail the level of hyperbilirubinemia associated with the disease and thereby often reduce the need for an otherwise necessary exchange transfusion. As stated before, after phototherapy the appearance of the skin is not a reliable index of the response to phototherapy nor in this case is it any index of the course of the underlying hemolytic disease. Serum bilirubin concentrations should be determined every 8 to 12 hours and the infant should be monitored for the appearance of anemia in subsequent days since, as previously stated, the hemolytic process itself is not corrected by the phototherapy.

GENERAL COMMENTS

1. Recent experiments have shown the products of the photodecomposition of bilirubin to be non-toxic. 7,8,9

2. Recent controlled experiments have shown a statistically significant difference in the incidence of hyperbilirubinemia in treated infants versus untreated, control group infants. 10,11.

3. Phototherapy has proven as useful and effective in Negro infants as in Caucasians. 12,13,14.

4. Natural daylight equals 10,000 foot candles. Nurseries vary anywhere from 50 to 5,000 foot candels. Phototherapy has traditionally utilised 100 to 500+ foot candels. The EME Phototherapy Lights are capable of emitting up to 600+ foot candles.

5. Studies indicate that bilirubin is most effectively decomposed by light emissions in the blue range, 420 to 460 nanometers and that the greater the energy output of the light source (foot candles) in this wavelength interval, the more rapid the degradation of bilirubin. 15,16.

6. Blue light has proven the most effective source of artificial light for rapidly lowering serum bilirubin levels. 15. The EME Phototherapy Lights emit within the blue energy range by utilising broad spectrum fluorescent lamps (North light).

7. The only noticeable common side-effect of phototherapy is that the infant's stools may be green and loose due to the presence of broken down bilirubin products in the excreted material.

8. The infant's eyes must be covered while undergoing phototherapy treatment.

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