is considerable concern. In Taiwan, women who had been exposed to high doses of these compounds through eating contaminated cooking oil had infants with dental and gum anomalies, hyperpigmentation, deformed nails, and acne. Affected infants showed decreased fetal growth, height, weight, and head circumference, and developmental delay. Attempts have been made to study the effect of another environmental agent, dioxin, which was a contaminant in the herbicide agent orange. These studies, involving Viet Nam veterans as well as in inhabitants of Viet Nam, have been exceedingly difficult to carry out because of epidemiologic problems with determining individuals at risk. At the present time, it does not appear that there is an acceptable amount of evidence that such exposure caused either an increase in birth defects or an increase in behavioral teratogenesis in the offspring.

Thalidomide

The thalidomide story is extremely well known and is unlikely to be repeated because this agent has not been on the market for nearly 30 years, except as an experimental agent in the treatment of leprosy. It remains, however, as a regrettable lesson in the inability to predict human fetal toxicity from animals studies as well as the first painful reminder that the placenta does not form a protective barrier to the fetus.

Warfarin Derivatives

The woman who requires anti-coagulation medication during pregnancy poses a very difficult problem. Warfarin compounds (such as Coumadin) cause the fetal warfarin syndrome: midface hypoplasia, abnormal ears and skull formation, central nervous system malformation, chondrodysplasia punctate, and digit malformation. Mental retardation occurs in affected offspring. Should a mother insist on continuing a pregnancy to term, fetal affects may be minimized by using heparin for the first trimester or later and switching to Coumadin before delivery. Perinatal hemorrhage is a threat with both Coumadin and heparin derivatives.

**FETAL DRUG THERAPY**

Not all drug effects are adverse to the fetus. An example of taking advantage of therapeutic drug passage across the placenta is the use of digoxin and furosemide administration to the mother when her fetus has congestive heart failure. The administration of corticosteroids to mothers threatening premature delivery may prevent hyaline membrane disease if the fetus is not carried to term. It is hoped that more compounds which can be used therapeutically for the benefit of the fetus will be developed in the future.

**REFERENCES**

5. Brent RL, Holmes LB. Clinical and basic science lessons from the thalidomide tragedy: what have we learned about the cause of limb defects. Teratology. 1988;38:241–251

**SUGGESTED READING**


**Self-Evaluation Quiz**

15. Although the effects of drugs on the fetus can occur at any time during gestation, the period of highest vulnerability is:
   A. First trimester.
   B. Second trimester.
   C. Third trimester.
   D. One month prior to conception.
   E. The perinatal period.
16. Each of the following drugs cause withdrawal syndrome in the newborn except:
   A. Amtriptyline.
   B. Cocaine.
   C. Methamphetamine.
   D. Meperidine.
   E. Marijuana.
17. The effects on infants born to cocaine-dependent mothers include each of the following, with the possible exception of:
   A. Decreased sleep.
   B. Increased risk of sudden infant death syndrome.
   C. Central nervous system bleeding.
   D. Urinary tract abnormalities.
   E. Seizures.
18. Of the following drugs given to pregnant women, which has not been associated with significant risk to the fetus? Captopril.
   B. Propranolol.
   C. Enalapril.
   D. Phenytin.
   E. Tetracycline.
19. A newborn is growth-retarded with midface hypoplasia, thin upper lip, micrognathia and upturned nose. The description is characteristic of exposure to what drug? Lithium.
   B. Hydantoins.
   C. Ethanol.
   D. Trimethadione exposure.
   E. Valproic acid.
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<thead>
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<th>Date</th>
<th>Event</th>
<th>Location</th>
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<tr>
<td>April 9–14</td>
<td>Pediatrics 1991</td>
<td>Kauai/Maui, Hawaii</td>
</tr>
<tr>
<td>May 24–26</td>
<td>Pediatric Advances</td>
<td>Kiawah Island, South Carolina</td>
</tr>
<tr>
<td>June 21–23</td>
<td>Clinical Pediatrics</td>
<td>Washington, DC</td>
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<tr>
<td>March 16–21</td>
<td>Spring Session</td>
<td>San Diego, California</td>
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<tr>
<td>October 26–31</td>
<td>Annual Meeting</td>
<td>New Orleans, Louisiana</td>
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These programs feature subject matter which is coordinated with the PREP curriculum and are eligible for PREP credits.

For further information, contact: CME, Department of Education, American Academy of Pediatrics, PO Box 927, Elk Grove Village, IL 60009-0927. (800) 433-9016. In Illinois (800) 421-0589.

American Academy of Pediatrics
PRCP Program for Renewal of Certification in Pediatrics

Guides for Record Review

Neonatal Hyperbilirubinemia

Supplement to Pediatrics in Review
This guide has been prepared by the American Board of Pediatrics as an integral part of the record review required for renewal of certification in general comprehensive pediatrics. Its purpose is to provide the pediatrician with criteria for assessing patient records dealing with specific problems. Important elements to be included in the record appear in the margins; those printed in italics are important under certain circumstances. Please note that these guides do not purport to articulate standards of care. They are designed solely to address record keeping issues.

The guides focus on the elements of the history and physical examination relevant to specific problems and are not meant to discourage a more thorough history and physical examination as appropriate for the patient and the particular circumstances.

The guides will be updated periodically. Because of rapid changes in knowledge about drugs and their availability, drugs and dosages included in these guides should be verified in current sources.

A table of international units is included in each guide.

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DIFFERENTIAL DIAGNOSIS

The most important differentiation is between the infant with physiologic jaundice and the infant with an abnormal degree of jaundice, or with an important underlying disease. Criteria which rule out the diagnosis of physiologic jaundice are listed in Table 2. Clinical jaundice appearing within the first 24 hours of age is abnormal. Similarly, clinical jaundice persisting beyond 2 weeks of age in the full-term infant requires an explanation. A total serum bilirubin concentration exceeding 11.5 mg/dL in a white, full-term, formula-fed infant or 14.5 mg/dL in a white full-term, breast-fed infant requires investigation (Table 2). The rate of increase of physiologic jaundice is generally relatively slow, with a peak at about 3 to 4 days of age. Except in infants receiving human milk, a rapid increase of serum bilirubin concentration, exceeding 5 mg/dL per day, also requires investigation.1,2

HISTORY

It is always important to record biographic data about every patient, but it is especially important to record such information in a jaundiced infant because of the association of prematurity and other events of pregnancy with hyperbilirubinemia. In evaluating a neonate with jaundice, it is important to determine whether there has been any unexplained illness during pregnancy which might indicate the presence of a congenital infection, although most such infants will have some degree of direct hyperbilirubinemia. Infants whose mothers have pre-eclampsia or diabetes mellitus are also predisposed to jaundice. It is well recognized that a prior pregnancy may result in sensitization to blood group antigens, with resultant isoimmunization of the fetus, but it is often forgotten that spontaneous or induced abortions have similar potential; women who have had abortions may not have been treated with immunoglobulin. The presence of maternal fever, prolonged rupture of membranes, or evidence of amnionitis such as a foul odor provides a clue that sepsis should be included in the differential diagnosis, although most such infants have some degree of direct hyperbilirubinemia. The labor and delivery should also be investigated because traumatic delivery, utilizing vacuum extraction or forceps, may result in bruising of the infant or formation of a cephalhematoma, providing a source of additional bilirubin for transport and excretion.3 The Apgar score will provide a clue to fetal asphyxia, which causes hypoxia and acidosis, and an increased risk of bilirubin encephalopathy (kernicterus).2

Because infants with congenital hemolytic anemia may be jaundiced in the first few days after birth, a family history of jaundice, anemia, gallbladder disease, or splenectomy would be helpful in diagnosis. The occurrence of jaundice in a previous sibling of the infant may also indicate the presence of a hemolytic anemia or other familial problem such as congenital hepatic abnormalities1 or cystic fibrosis.

The time of onset of jaundice would be helpful because jaundice before 24 hours of age indicates an increased rate of hemolysis, most commonly due to isoimmunization. To investigate the possibility that the enterohepatic circulation is a major cause of the problem, the caloric and fluid intake of the infant should be determined, as well as the presence of vomiting, delayed passage of meconium, or failure to begin to gain weight after the normal initial weight loss. If jaundice has its onset somewhat later during the neonatal period, it will be important to know whether the infant is breast- or bottle-fed, because breast feeding is the major cause of jaundice that persists beyond the first few days after birth.
PHYSICAL EXAMINATION

The infant’s temperature should be recorded as a baseline measurement because fever or hypothermia raises the possibility of sepsis in a jaundiced infant. The length and weight should be recorded because small size may indicate that the infant is preterm or small for gestational age; both circumstances are associated with an increased incidence of jaundice. On the other hand, the infant who is large for gestational age may have a diabetic or prediabetic mother, another condition predisposing to jaundice. The head circumference should be measured because microcephaly is commonly seen with intrauterine infections, which may be associated with jaundice. The presence of a cephalhematoma or extensive bruising may indicate that hemorrhage is the source of the hyperbilirubinemia. Enlargement of either the liver or the spleen may indicate the presence of hemolytic anemia or congenital infection. An abdominal mass or distention may indicate the presence of partial bowel obstruction such as pyloric stenosis, or adrenal hematoma, which are rare causes of persistent jaundice. Finally, the skin should be carefully examined. Plethora may indicate that polycythemia is present; pallor is a clue to the presence of anemia; and petechiae may be present in the infant with congenital infection, sepsis, or severe hemolytic disease.

LABORATORY STUDIES

In the presence of jaundice, the mother’s blood group should be determined to indicate the possibility of ABO or Rh incompatibility. The infant’s routine evaluation should include a total serum bilirubin concentration, determination of the blood groups, and a Coombs test. The first of these will determine the level of hyperbilirubinemia, and whether the degree of jaundice may be considered to be physiologic (see Table 2). The latter two tests will determine whether isoimmunization is present, and thus whether further studies are needed.1,2

When a significant level of hyperbilirubinemia exists and isoimmunization has been ruled out, the fractions of serum bilirubin should be determined. As previously indicated, direct hyperbilirubinemia will not be discussed (see Table 1 for differential diagnosis).1,1 The infant with indirect hyperbilirubinemia should have a hematocrit performed to rule out polycythemia or anemia (hemolysis). A reticulocyte count to ascertain whether hemolytic disease is likely to be present should be done, and a blood smear examined to determine erythrocyte morphology; the presence of spherocytes suggests hereditary spherocytosis, although spherocytes are also seen with ABO incompatibility. Erythrocyte fragmentation is present in disseminated intravascular coagulation, and other nonspecific abnormalities may be present in infants with other congenital hemolytic anemias. Most other studies done in the infant with hyperbilirubinemia (platelet count, leukocyte count and differential cell count, serum IgM concentration, urinalysis for reducing substances, and so forth) are more helpful in the diagnosis of direct hyperbilirubinemia than indirect hyperbilirubinemia.1,2
MANAGEMENT

Jaundice in the neonate is a unique problem because of the risk of bilirubin encephalopathy (kernicterus) at that age. The goal of therapy is to keep the serum bilirubin concentration at a level which minimizes the risk of encephalopathy, while treating any underlying cause and avoiding treatment that is more risky than the jaundice. In general, the two forms of therapy most commonly used are phototherapy and exchange blood transfusion. Each reduces the serum bilirubin concentration, the first by photoisomerization of bilirubin to a product that is more readily excreted, and the latter by direct removal. In certain circumstances, the administration of a drug such as phenobarbital, which induces activity of the hepatic enzymes, may be indicated, but the onset of action is slow and unacceptable sedation may result.

One of the most frequent clinical problems affecting the neonate is the hyperbilirubinemia associated with breast feeding. Multiple mechanisms are involved, including the possibility that some mothers have substances in their milk that aggravate the physiologic tendency to hyperbilirubinemia. Also important is the possibility that the infant is not feeding frequently enough, or not getting a sufficient volume at the time of feeding. These factors increase the enterohepatic circulation, and reduce the amount of bilirubin eliminated in the stools. Once the other causes of jaundice have been eliminated, it seems reasonable to assume that the otherwise healthy, breast-fed term infant has hyperbilirubinemia as a result of breast feeding. No treatment is indicated except to monitor the serum bilirubin concentration to insure that it does not reach potentially toxic levels (probably > 20 mg/dL). The risk of encephalopathy in such infants is exceedingly small. If the serum bilirubin concentration approaches 20 mg/dL, it may be worthwhile to supplement or interrupt breast feeding for 24 hours, substituting a proprietary formula, and to measure the serum bilirubin concentration to document the prompt decline. Such a procedure has both diagnostic and therapeutic benefits. If there is concern that the amount of human milk is inadequate, supplemental feeding with a proprietary formula for 24 to 48 hours may have a salutary effect. Frequent feeding to insure an adequate fluid and caloric intake is important. Monitoring the infant's weight may be helpful in determining the adequacy of intake. Phototherapy is unnecessary unless the above maneuvers fail and the serum bilirubin concentration exceeds 18 to 20 mg/dL. In the rare infant who has even higher concentrations of bilirubin, or who has any suggestion of lethargy, poor suck and/or poor feeding, more aggressive treatment is warranted.

Whatever the cause of the indirect hyperbilirubinemia, the goal of management is to keep the serum bilirubin concentration below a level that is associated with encephalopathy. Numerous guidelines have been published; Table 3 contains consensus criteria for the need for either phototherapy or exchange transfusion. Preterm infants are at greater risk of encephalopathy. Similarly, the presence of acidosis, hypoxia, hypoglycemia, or sepsis increases the risk of kernicterus; thus, aggressive management of hyperbilirubinemia in preterm infants is warranted at a lower serum bilirubin concentration. In making decisions regarding institution of therapy, the limits of accuracy of the laboratory measurement of serum bilirubin concentration must be remembered. Transcutaneous bilirubinometers are used in some nurseries, but should not be relied upon in determining whether a given infant needs therapy. In the infant who has had a measured serum bilirubin concentration, the bilirubinometer may provide comparative data of clinical value, provided that the institution has standardized the relationship between directly measured and bilirubinometer values.

In general, phototherapy should be instituted when the serum bilirubin concentration, taken in conjunction with the known rate of increase and the age of the infant, indicates the
possibility that the serum bilirubin concentration will increase to a point at which encephalopathy would be possible. Clinical observations of jaundice are not reliable once phototherapy has been instituted; serum bilirubin concentrations should be measured repeatedly until it is clear that the bilirubin concentration is decreasing. Following discontinuation of phototherapy, the serum bilirubin concentration should be measured again in six to twelve hours, because a rebound upwards may occur. In general, the otherwise well infant can be discharged from the hospital if the increase following the discontinuation of treatment does not exceed 2 or 3 mg/dL. If home phototherapy is used, the same recommendations for measuring serum bilirubin concentrations should be followed.

Exchange transfusion is indicated when the serum bilirubin value has already reached a concentration at which the infant is at risk for encephalopathy, when the infant has symptoms of early encephalopathy, or when the rate of increase is such that a dangerous serum concentration of bilirubin can be anticipated. All infants with clinically significant hemolytic disease of the newborn should be treated with phototherapy in an effort to avoid should be treated with phototherapy to prevent, if possible, the need for a second exchange transfusion. The indications for a repeat exchange transfusion are the same as those for the first.

FOLLOW-UP EVALUATION

In the otherwise normal infant with jaundice, no specific follow-up is needed once it is determined that the serum bilirubin concentration is declining. In the infant with hemolytic disease, however, significant hemolysis may continue and produce severe anemia within several weeks after birth, particularly in infants with isoimmunization who have not required exchange transfusion. In such infants, regular determinations of hemoglobin and/or hematocrit are needed until it can be assured that the value is stable or increasing.

FAMILY EDUCATION

In the mother who is breast feeding her infant, the occurrence of jaundice that is attributed to breast feeding may cause considerable anxiety, and requires counseling. Such mothers should be assured that their milk is not bad for the infant, and they should be encouraged to plan to breast feed subsequent infants, if that is their desire. Instructions regarding frequent breast feeding and insuring that the infant suck well are important. If temporary interruption of breast feeding is needed, assistance in maintaining the milk supply by pumping the breasts will be needed. Mothers who have Rh-negative blood types should be aware of the need for gamma globulin (RhoGAM) administration in the event of spontaneous or induced abortion so that the information will be communicated to any physicians involved in her care.
TABLE 1
Etiology of Direct (Conjugated) Hyperbilirubinemia

I. Infections
   A. Bacterial sepsis
   B. Intrauterine infections (TORCH, syphilis)
   C. Perinatally-acquired viral infections (herpes, enterovirus, hepatitis B)

II. Obstructions
   A. Biliary atresia
   B. Bile plugs
   C. Choledochal cyst

III. Cholestatic syndromes (e.g., arteriohepatic dysplasia)

IV. Giant cell hepatitis

V. Inborn metabolic errors and other genetic diseases
   A. α1-Antitrypsin deficiency
   B. Cystic fibrosis
   C. Galactosemia
   D. Tyrosinosis

VI. Severe hemolytic disease

TABLE 2
Criteria Which Rule Out the Diagnosis of Physiologic Jaundice in Full-term Infants

1. Clinical jaundice in the first 24 hours after birth

2. Total serum bilirubin concentration exceeding 14.5 mg/dL in a breast-fed infant or 11.5 mg/dL in a formula-fed infant*

3. Direct serum bilirubin concentration exceeding 2 mg/dL.

4. Clinical jaundice persisting for more than two weeks

* These values apply to healthy, white, full-term infants only. No similar data are available for breast-fed versus formula-fed black, Hispanic or Oriental infants in the United States. In the Collaborative Perinatal Project, 95% of black infants had maximal serum bilirubin concentrations of < 13 mg/dL. This population included infants with hemolytic disease and the majority were formula fed.

TABLE 3
Serum Bilirubin Concentrations Commonly Used as Indications for Phototherapy or Exchange Transfusion*

<table>
<thead>
<tr>
<th>Birth Weight</th>
<th>Phototherapy**</th>
<th>Exchange**</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1500 g</td>
<td>8 to 10 mg/dL</td>
<td>10 to 15 mg/dL</td>
</tr>
<tr>
<td>1501 to 2500 g</td>
<td>10 to 12 mg/dL</td>
<td>15 to 18 mg/dL</td>
</tr>
<tr>
<td>&gt; 2500 g</td>
<td>15 to 18 mg/dL</td>
<td>20 mg/dL</td>
</tr>
</tbody>
</table>

* These guidelines represent the current practice of many experts. They have not been validated by appropriate clinical studies.

** In the presence of hemolysis, or risk factors such as hypoxia, acidosis, hypoalbuminemia, or sepsis, these values should be reduced by 2 mg/dL. The smaller and younger the infant is within the weight group, the more liberal use of treatment is indicated. These values do not pertain to healthy infants whose jaundice is believed to be due to breast feeding.
**Conversion Table to Standard International (SI) Units**

I. Hematology
   - Hemoglobin g/dL x 0.155 = mmol/L
   - Platelets/mm³ = count/μL = 10⁶ cells/L
   - Leukocytes/mm³ = count/μL = 10⁶ cells/L
   - Erythrocytes/mm³ = count/μL = 10⁶ cells/L
   - Hematocrit % x 0.01 = vol RBC/vol whole blood
   - Reticulocytes % x 0.01 = (1)

II. Blood Pressure mm Hg (torr) x 1.333 = mbar

III. Blood Gases
   - 1 mm Hg = 133.322 Pa
   - Base excess mEq/L = mmol/L
   - pH value = same

IV. Blood Chemistries
   - Acetone mg/dL x 0.1722 = mmol/L
   - Acetaminophen μg/mL x 6.62 = μmol/L
   - Albumin g/dL x 144.9 or g/L x 14.49 = μmol/L
   - Aldosterone ng/dL x 0.0277 = nmol/L
   - Ammonia mgN/dL x 0.714 = mmol/L
   - Bicarbonate mEq/L = mmol/L
   - Bilirubin mg/dL x 17.10 = μmol/L
   - Blood urea nitrogen mg/dL x 0.357 = mmol urea/L
   - Calcium mg/dL x 0.25 = mmol/L
   - Carotene IU x 0.6 or μg/dL x 0.01863 = μg
   - Ceruloplasmin mg/dL x 0.0662 = μmol/L
   - Chloride mEq/L = mmol/L
   - Cholesterol mg/dL x 0.0259 = mmol/L
   - Complement component (C3) mg/dL x 0.01 = g/L
   - Copper μg/dL x 0.157 = μmol/L
   - Cortisol μg/dL x 27.59 = nmol/L
   - Creatine mg/dL x 76.26 = μmol/L
   - Creatinine mg/dL x 88.40 = μmol/L
   - Digoxin ng/mL x 1.28 = nmol/L
   - Enzymes
     - Alanine aminotransferase (ALT, SGPT) U/L = U/L
     - Aldolase = U/L
     - Sibley-Lehninger units/mL = U/L
     - Amylase = U/L
     - Somogyi units/dL = U/L
     - Aspartate aminotransferase (AST, SGOT) U/L = U/L
     - Creatine kinase (CK) U/L = U/L
     - Phosphatase
       - Bodansky units/dL = U/L
       - King-Armstrong units/dL = U/L
     - Fatty acids mg/dL x 0.0354 = mmol/L
     - Ferritin ng/mL x 1 = μg/L

PCO₂ mm Hg x 0.1333 = kPa
P0₂ mm Hg x 0.1333 = kPa
<table>
<thead>
<tr>
<th>Test</th>
<th>Unit Conversion</th>
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<tr>
<td>a-Fetoprotein ng/mL x 1</td>
<td>μg/L</td>
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<tr>
<td>Fibrinogen mg/dL x 0.01</td>
<td>g/L</td>
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<tr>
<td>Folic acid μg/dL x 22.65</td>
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<tr>
<td>Glucose mg/dL x 0.0555</td>
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<tr>
<td>Glycerol mg/dL x 0.1086</td>
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<tr>
<td>Haptoglobin mg/dL x 0.01176</td>
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<tr>
<td>17-Hydroxyprogesterone mg/d x 2.759</td>
<td>μmol/d</td>
</tr>
<tr>
<td>Insulin IU x 0.04167</td>
<td>mg</td>
</tr>
<tr>
<td>or μU/mL x 1.0</td>
<td>mU/L</td>
</tr>
<tr>
<td>Iodine μg/dL x 78.8</td>
<td>nmol/L</td>
</tr>
<tr>
<td>Iron μg/dL x 0.1791</td>
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</tr>
<tr>
<td>Iron binding capacity μg/dL x 0.1791</td>
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</tr>
<tr>
<td>17-Ketosteroids mg/d x 3.467</td>
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</tr>
<tr>
<td>Lead μg/dL x 0.0483</td>
<td>μmol/L</td>
</tr>
<tr>
<td>Lipoprotein mg/dL x 0.01</td>
<td>g/L</td>
</tr>
<tr>
<td>Magnesium mg/dL x 0.4114</td>
<td>mmol/L</td>
</tr>
<tr>
<td>or mEq/L x 0.5</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Phosphorus mg/dL x 0.3229</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Potassium mEq/L</td>
<td>mmol/L</td>
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<tr>
<td>Prednisone mg x 2.79</td>
<td>μmol/L</td>
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<tr>
<td>Protein g/dL x 10</td>
<td>g/L</td>
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<tr>
<td>Salicylate mg/dL x 0.0724</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Sodium mEq/L</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Theophylline μg/mL x 5.55</td>
<td>μmol/L</td>
</tr>
<tr>
<td>Thyroid-stimulating hormone μU/mL x 1</td>
<td>mU/L</td>
</tr>
<tr>
<td>Thyroxine μg/dL x 12.87</td>
<td>nmol/L</td>
</tr>
<tr>
<td>Transferrin mg/dL x 0.01</td>
<td>g/L</td>
</tr>
<tr>
<td>Triglycerides mg/dL x 0.01</td>
<td>g/L</td>
</tr>
<tr>
<td>Triiodothyronine ng/dL x 0.0154</td>
<td>nmol/L</td>
</tr>
<tr>
<td>Urea nitrogen mg/dL x 0.357</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Uric acid mg/dL x 59.48</td>
<td>μmol/L</td>
</tr>
<tr>
<td>Vitamin A μg/dL x 0.0349</td>
<td>μmol/L</td>
</tr>
<tr>
<td>Vitamin B₂ pg/dL x 0.738</td>
<td>pmol/L</td>
</tr>
<tr>
<td>Vitamin C mg/dL x 56.78</td>
<td>μmol/L</td>
</tr>
<tr>
<td>Vitamin E μg/dL x 2.322</td>
<td>μmol/L</td>
</tr>
<tr>
<td>Xylose mg/dL x 0.0667</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Zinc μg/dL x 0.153</td>
<td>μmol/L</td>
</tr>
</tbody>
</table>

V. Urine or Stool
- Coproporphyrin μg x 1.53 - nmol
- Epinephrine μg/d x 5.458 - nmol/d
- Vanilmandelic acid mg/d x 5.046 - μmol/d
- Homovanillic acid mg/d x 5.489 - μmol/d

VI. Energy
- Kcal x 4.1868 - KJ (Kilojoule)
- Rad x 0.01 - Gy (Gray) (joule/kg)

VII. Radionuclide Activity
- Curie (Ci) x 37 - GGq (Gigabecquerel)
REFERENCES


