infection were more likely to have mothers who had gonorrhea, trichomoniasis, or bacterial vaginosis during pregnancy compared with controls. A new registry, created to monitor CMV cases in the United States and Canada, will improve our ability to identify infants who have congenital CMV and to develop prevention and treatment strategies.

Peter Sherman, MD
Director of Adolescent Clinics
Assistant Professor of Pediatrics
Bronx Municipal Hospital Center
Albert Einstein College of Medicine
Bronx, NY

**Comment:** Sexual history-taking in our teenage patients is a more complicated undertaking in this new era of proliferating STDs. With the significant implications for health and disease in the newborn, pediatricians who care for teenagers must be vigorous in their extensive and careful screening procedures for all those who are sexually active. With multiple partners over time, and even more complex sexual histories in those unidentified or current partners, the risk of STD with serious potential outcomes for newborn babies cannot be overstated. Complete counseling must include careful description of the potential risks of such infections, including HIV, even in what appear to be low-risk populations.

Steven P. Shelov, MD
Abstracts Editor
American Academy of Pediatrics

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PRCP

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Guides for Record Review

Anemia

Supplement to Pediatrics in Review
This guide has been prepared by the American Board of Pediatrics (ABP) 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 bold-face type in the margins; other elements to be considered are printed in italics.

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.

The guides are planned, written, and reviewed by an ABP committee composed primarily of practicing pediatricians. Appropriate subject experts are consulted during the preparation of the guides.

Please note that these guides do not purport to articulate standards of care. They are designed solely to address record keeping issues.

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INTRODUCTION

Anemia is defined as a reduced number of erythrocytes or a reduced concentration of hemoglobin, with a resultant decrease in oxygen-carrying capacity. Normal hematologic values vary according to age (Table). During childhood, black patients tend to have somewhat lower values than those of white patients. During adolescence and young adulthood, males usually have higher erythrocyte counts and hemoglobin concentrations than females. Thus, these demographic factors must be taken into account in determining whether a given patient is anemic. The causes of anemia also vary at differing ages; for example, nutritional iron deficiency is seen almost exclusively during infancy and in adolescent girls.

In evaluating a child with anemia, it is helpful to consider the likely origin: (1) blood loss; (2) a reduced production of erythrocytes, as occurs in patients with chronic infection or renal disease; or (3) increased destruction of erythrocytes, on either an intrinsic basis (eg, sickle cell disease, spherocytosis) or an extrinsic basis (eg, drug-induced). As indicated below, the size and characteristics of erythrocytes may provide important clues to the likely cause of anemia.

Because the etiology of anemia is complex, the emphasis in this guide will be upon those mild to moderate anemias that are encountered most commonly in ambulatory pediatric practice, in patients from 6 months of age through adolescence.
HISTORY

Unless anemia is severe, symptoms are commonly absent. As discussed below, the diagnosis is frequently made after routine screening studies during the course of a health maintenance examination. However, some children may be pale, easily fatigued, irritable, or have behavioral problems such as breath-holding and tantrums. Older children may complain of reduced exercise tolerance, palpitations, or syncope; inattentiveness and poor school performance may be noted.2

For the infant found to be anemic, it is important to know whether he or she was born prematurely because reduced iron stores may not have been corrected.1 Similarly, evidence for blood group incompatibility and iso-immunization during the newborn period should be sought.3 Because of the frequency of iron deficiency in infants, it is particularly important to inquire about the diet, to ascertain whether the infant has been breast fed or formula fed, and whether the formula was iron fortified.4 Early introduction of cow milk may result in reduced iron absorption and may cause gastrointestinal blood loss in some infants.5 It should be determined whether there has been any evidence of bleeding such as recurrent epistaxis, melena, or menorrhagia. Easy bruisability may be a clue to a hemorrhagic disorder or to a blood dyscrasia such as leukemia. Long-term therapy with certain drugs may cause depression of the bone marrow; patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency may have acute hemolysis following exposure to certain drugs such as sulfonamides.1 Thus, exposure to drug therapy and to other chemicals or toxins should be identified and noted in the record. Although chronic illness will not be considered in this guide, it is a common cause of persistent anemia.1 A history of pica should be sought, especially in infants living in older housing or in other environments that may have an increased potential exposure to lead.2 In adolescents, sustained exercise such as long-distance running may cause subtle gastrointestinal blood loss.3 If the child has been previously treated for anemia, the results should be documented; the recurrence of iron deficiency after appropriate treatment, for example, would provide important evidence for blood loss. Children with congenital heart disease, especially those with artificial valves, may have anemia as the result of increased destruction of erythrocytes.3

FAMILY HISTORY2

Many anemias, particularly hemolytic anemias, are hereditary in origin. Thus, it should be remembered that black children may have sickle cell disease or G6PD deficiency, and that those of southeast Asian or Mediterranean origin are more likely to have thalassemia. A family history of anemia, episodes of jaundice, gallbladder disease beginning in early life, or of splenectomy may indicate the presence of one of the familial anemias. If the family has lived or traveled recently in areas where diseases such as malaria or worm infestations are common, those causes of anemia may need to be investigated.
PHYSICAL EXAMINATION

A complete physical examination should be done in an effort to identify clues to underlying illness and to determine whether the anemia is causing any physiologic ill effects. When the cause is nutritional, other evidence for poor nutrition may be apparent, although infants with iron deficiency anemia can actually be overweight because of a large intake of milk. The skin and mucous membranes should be inspected for the presence of pallor, petechiae, or purpura. The presence of tachycardia or a systolic ejection murmur indicates that the anemia is clinically significant. The presence of lymphadenopathy, hepatomegaly, or splenomegaly would raise concerns about an underlying disease such as lymphoma or leukemia. Skeletal abnormalities are commonly associated with rare causes of anemia such as Diamond-Blackfan syndrome. Finally, evidence for a chronic disease, such as rheumatoid arthritis or renal insufficiency, should be sought.

LABORATORY EVALUATION

As noted above, anemia is frequently identified as a result of routine screening studies, particularly of hemoglobin or hematocrit. Although the yield of routine determinations of hematocrit in well-nourished, middle- and upper-class populations is low, it is commonly recommended that hemoglobin concentration or hematocrit be measured toward the end of the first year after birth, at the time of school entry, during late childhood, and during adolescence. Such screening studies are ideally done when the child is well; screening at the time of acute illness may result in an incorrect diagnosis because of the temporary depression of bone marrow that commonly occurs at such times. Borderline values should be re-measured before an extensive evaluation is undertaken. In interpreting the screening values, it should also be remembered that, in some children, the normal value may be outside the normal range. For example, a child with cyanotic congenital heart disease can be expected to have a higher than normal hemoglobin concentration or hematocrit; the usual normal value might indicate the presence of a relative anemia in such patients.

The origins of most anemias can be determined by a relatively simple and inexpensive series of basic studies, including erythrocyte indices, especially mean corpuscular volume, a reticulocyte count, and a peripheral blood smear; in black patients, sickle cell disease should be ruled out. Anemias may conveniently be classified on the basis of erythrocyte size; the figure provides a simple flow diagram indicating the most likely diagnoses for anemias of various morphologic types. Microcytic anemias are commonly due to iron deficiency, and the reticulocyte count will tend to be decreased, but lead poisoning may cause a similar pattern. The free erythrocyte protoporphyrin (FEP) concentration is increased in both conditions, but particularly high in lead poisoning. Thalassemia trait disorders also present as microcytic anemia; the reticulocyte count is usually normal, as is the FEP. Most electronic counting equipment used to determine hemoglobin concentration and erythrocyte size also provides an erythrocyte distribution width (RDW). The RDW, a measure of the variation of size and shape of erythrocytes, is increased in anemia due to iron deficiency and lead poisoning but is normal in anemia due to thalassemia trait.
The reticulocyte count (see Figure) is particularly helpful in the evaluation of normocytic anemia, providing a major clue whether bone marrow depression on the one hand, or bleeding or hemolysis on the other, is responsible for anemia. Transient erythroblastopenia of childhood is an example of the former condition; autoimmune hemolytic anemia and intrinsic defects of erythrocytes (eg, spherocytosis, hemoglobin disorders, enzyme deficiencies) account for most of the latter conditions. Of course, chronic blood loss may also eventually result in iron deficiency.

Depending upon the initial findings and the most likely diagnoses, further tests are sometimes indicated. If blood loss is suspected, several stool determinations for the presence of occult blood should be done. It is seldom necessary to document iron deficiency if the history is consistent with this diagnosis and if the peripheral blood smear shows typical microcytosis and hypochromia, but in older children, in whom the diagnosis may be more questionable, serum iron and ferritin determinations may be useful. Hemoglobin electrophoresis, including measurement of hemoglobin A2, is required to confirm the diagnosis of a hemoglobinopathy such as sickle cell disease or thalassemia. The presence of spherocytes on the peripheral blood smear or other evidence of rapid hemolysis may indicate the need for a Coombs test to rule out an antibody-mediated anemia. Spherocytes may also indicate the presence of hereditary spherocytosis, which can be confirmed by an incubated osmotic fragility test; such tests should be done while the patient's condition is stable, rather than during or immediately following a hemolytic episode, or a falsely negative result may be obtained. Similarly, studies for G6PD deficiency should be done between rather than during hemolytic episodes because erythrocytes just produced by the bone marrow may have enzyme values within the range of normal. In the rare patient with macrocytic anemia, serum vitamin B12 and folic acid concentrations should be determined. Bone marrow examination may be needed in some patients, such as those suspected of having transient erythroblastopenia, and are required if a malignancy such as leukemia is suspected. Many pediatricians would probably prefer to obtain consultation with a pediatric hematologist-oncologist before embarking on such studies. Finally, if an underlying nonhematologic disease is suspected, studies such as urinalysis or erythrocyte sedimentation rate may be indicated.

FOLLOW-UP EVALUATION AND THERAPY

If the initial history, physical findings, and results of screening laboratory studies indicate the likelihood of iron deficiency, it is reasonable to begin a therapeutic trial of oral iron therapy before a more extensive laboratory investigation is carried out. Further studies are then indicated if the anemia does not respond promptly to treatment or if it recurs after an initial response. Iron is usually given in the form of ferrous sulfate, in a dose of 6.0 mg of elemental iron/kg/day. Repeat hemoglobin or hematocrit determination should be obtained after one month, at which time iron deficiency anemia should be corrected. (Hemoglobin concentration is expected to increase 0.1 to 0.3 g/dL/day after two to three days of appropriate iron therapy.) This therapy is continued for two to three months after the hemoglobin concentration has returned to normal so that iron stores will be repleted. A subsequent
decrease in hemoglobin concentration suggests the possibility of blood loss. In most infants and young children, it will also be necessary to modify the diet by reducing the intake of cow milk and increasing the intake of foods containing iron.2

The management of other acute or chronic anemias is beyond the scope of this discussion, but a few points deserve attention. In a child with sickle cell disease or one who has required splenectomy, the risk for bacteremia and overwhelming sepsis must be kept in mind.10 Such patients should be immunized with pneumococcal and meningococcal vaccines in addition to the usual childhood immunizations, and should receive daily penicillin prophylaxis,11 at least until 6 years of age, although compliance has been a problem in many studies. In any event, such children should be evaluated promptly whenever a febrile illness develops, and presumed to have bacteremia until proven otherwise. The performance of a blood culture and the prompt institution of antibiotic therapy are essential. If a child is known to have G6PD deficiency, a notation to that effect should be written prominently on the chart so that exposure to oxidant drugs (eg, a sulfonamide, an antimalarial drug, some antipyretic medications) and certain foods (eg, fava beans) is avoided.

PATIENT AND FAMILY EDUCATION

As noted above, parents of infants or children with iron deficiency anemia need to be informed of the importance of continuing iron therapy beyond the time that the hemoglobin concentration and/or hematocrit have returned to normal, and of modifying the diet appropriately. Parents of children with G6PD deficiency need to be aware of the drugs and foods that may precipitate hemolysis. Families must be alert to the need for prompt medical attention when fever develops in a child who has sickle cell disease. Some centers have succeeded in teaching families how to palpate the abdomen so that an early diagnosis of acute splenic sequestration can be made.12 Maintenance of hydration and the use of analgesic drugs during intercurrent illnesses and in the early phases of painful crises may be helpful.1

For a child with lead poisoning, separation from the hazardous environment until all sources of lead have been removed is essential. Exposure to lead-containing dust during renovations, for example, can rapidly increase the blood lead concentration.13

Finally, families of patients affected by any of the hereditary anemias should be given the benefit of genetic counseling, if available or appropriate, so that they can take advantage of such services as prenatal diagnosis.
TABLE
NORMAL BLOOD VALUES FROM
6 MONTHS OF AGE TO ADULTHOOD*

<table>
<thead>
<tr>
<th>Age, years</th>
<th>Medial</th>
<th>Lower Limit</th>
<th>Median</th>
<th>Lower Limit</th>
</tr>
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<tbody>
<tr>
<td>0.5 to 2</td>
<td>12.5</td>
<td>11.0</td>
<td>77</td>
<td>70</td>
</tr>
<tr>
<td>2 to 5</td>
<td>12.5</td>
<td>11.0</td>
<td>79</td>
<td>73</td>
</tr>
<tr>
<td>5 to 9</td>
<td>13.0</td>
<td>11.5</td>
<td>81</td>
<td>75</td>
</tr>
<tr>
<td>9 to 12</td>
<td>13.5</td>
<td>12.0</td>
<td>85</td>
<td>76</td>
</tr>
<tr>
<td>12 to 14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>13.5</td>
<td>12.0</td>
<td>85</td>
<td>77</td>
</tr>
<tr>
<td>Male</td>
<td>14.0</td>
<td>12.5</td>
<td>84</td>
<td>76</td>
</tr>
<tr>
<td>14 to 18</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>14.0</td>
<td>12.0</td>
<td>87</td>
<td>78</td>
</tr>
<tr>
<td>Male</td>
<td>15.0</td>
<td>13.0</td>
<td>86</td>
<td>77</td>
</tr>
<tr>
<td>18 to 49</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>14.0</td>
<td>12.0</td>
<td>90</td>
<td>80</td>
</tr>
<tr>
<td>Male</td>
<td>16.0</td>
<td>14.0</td>
<td>90</td>
<td>80</td>
</tr>
</tbody>
</table>

* On average, black children have hemoglobin concentrations approximately 0.5 g/dL less than those of white children. Because of the differing values at different ages, adult norms should not be used for determining the presence of anemia in infants and young children.

From Stockman JA III: Anemia: shortcuts to diagnosis. The Child’s Doctor 1:9, 1984
FIGURE
CLASSIFICATION OF ANEMIA
on the basis of morphologic characteristics
(adapted from various sources)

MICROCYTIC
Iron deficiency
Thalassemias
Lead poisoning
Chronic disease*
FEP or RDW
Increased
Lead poisoning
Iron deficiency

NORMOCYTIC
Reticulocyte count
Decreased
Chronic disease
Aplastic anemia
Transient erythroblastopenia
Leukemia
Erythrocyte aplasia
Increased
Bleeding
Hemolysis (most congenital erythrocyte disorders)

MACROCYTIC**
Reticulocytosis
Folic acid deficiency
Vitamin B₁₂ deficiency
Hypothyroidism
Liver disease
Drugs (eg, methotrexate)
Preleukemia
Leukemia
Aplastic anemia
Congenital pure erythrocyte aplasia

* Most children with anemia due to chronic disease have normocytic anemia. Some, however, may have microcytosis.

**Normal neonates and infants and children with Down syndrome also have macrocytic erythrocytes.
REFERENCES


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 = 133.22 Pa
    Base excess mEq/L = mmol/L
    PCO₂ mm Hg x 0.1333 = kPa
    PO₂ mm Hg x 0.1333 = kPa
    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
    Sibley-Lehninger units/mL = U/L
    Amylase
    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
<table>
<thead>
<tr>
<th>Substance</th>
<th>Conversion Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferritin ng/mL x 1</td>
<td>= μg/L</td>
</tr>
<tr>
<td>α-Fetoprotein ng/mL x 1</td>
<td>= μg/L</td>
</tr>
<tr>
<td>Fibrinogen mg/dL x 0.01</td>
<td>= g/L</td>
</tr>
<tr>
<td>Folic acid μg/dL x 22.65</td>
<td>= nmol/L</td>
</tr>
<tr>
<td>Glucose mg/dL x 0.0555</td>
<td>= mmol/L</td>
</tr>
<tr>
<td>Glycerol mg/dL x 0.1086</td>
<td>= mmol/L</td>
</tr>
<tr>
<td>Haptoglobin mg/dL x 0.01176</td>
<td>= μmol/L</td>
</tr>
<tr>
<td>17-Hydroxycorticosteroids 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>
<td>= μmol/L</td>
</tr>
<tr>
<td>Iron binding capacity μg/dL x 0.1791</td>
<td>= μmol/L</td>
</tr>
<tr>
<td>17-Ketosteroids mg/d x 3.467</td>
<td>= μmol/d</td>
</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>
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</tr>
<tr>
<td>or mEq/L x 0.5</td>
<td>= mmol/L</td>
</tr>
<tr>
<td>Phosphorus mg/dL x 0.3229</td>
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</tr>
<tr>
<td>Potassium mEq/L</td>
<td>= mmol/L</td>
</tr>
<tr>
<td>Prednisone mg x 2.79</td>
<td>= μmol</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>
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</tr>
<tr>
<td>Transferrin mg/dL x 0.01</td>
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<tr>
<td>Triglycerides mg/dL x 0.01</td>
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<tr>
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<td>Vitamin A μg/dL x 0.0349</td>
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<td>Vitamin B12 Pg/dL x 0.738</td>
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<td>Vitamin C mg/dL x 56.78</td>
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<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>
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</table>

V. Urine or Stool

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Coproporphyrin μg x 1.53</td>
<td>= nmol</td>
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<tr>
<td>Epinephrine μg/d x 5.458</td>
<td>= nmol/d</td>
</tr>
<tr>
<td>Vanilmandelic acid mg/d x 5.046</td>
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</tr>
<tr>
<td>Homovanillic acid mg/d x 5.489</td>
<td>= μmol/d</td>
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</tbody>
</table>

VI. Energy

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<th>Conversion Factor</th>
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</thead>
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<tr>
<td>Kcal x 4.1868</td>
<td>= KJ (Kilojoule)</td>
</tr>
<tr>
<td>Rad x 0.01</td>
<td>= Gy (Gray) (joule/kg)</td>
</tr>
</tbody>
</table>

VII. Radionuclide Activity

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<thead>
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<th>Substance</th>
<th>Conversion Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Curie (Ci) x 37</td>
<td>= GGq (Gigabequerel)</td>
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</table>