Pre-Meeting Preparation:
Please read the following enclosures, corresponding to the screening procedures:

1) **Hematocrit or Hemoglobin**: “Screening for Iron Deficiency” *(PIR 2002)*
   - excerpt from “Diagnosis and Prevention of Iron-Deficiency and Iron-Deficiency Anemia in Infants and Young Children” *(Pediatrics 2010)*

2) **Lead**: Screening for Lead Exposure *(Excerpts - AAP Policy Statement)*
   - update on Maryland lead testing requirements (2015)
   - update on DC lead screening guidelines (2016)

3) **Tuberculosis**: TB Risk-Assessment Questionnaire; “TST- CDC Fact Sheet”

4) **Dyslipidemia**: Screening for Dyslipidemia *(Excerpts- AAP Statement)*;

**Homework:**
* Bring in an article or resource addressing a “clinical controversy” or “current event” related to this week’s screening procedures (e.g. unclear benefits of universal iron deficiency or lipid screening)

**Conference Agenda:**
- Review Health Maintenance II
- Complete Health Maintenance II Cases
- **Round-table discussion** of “clinical controversy”/ “current events” articles

**Post-Conference:** Board Review Q&A

**Extra-Credit:**
Review these AAP clinical practice guidelines, corresponding to the listed screening procedures:

1) Hct or Hgb: “Diagnosis & Prevention of Iron Deficiency” *(Clinical Report; 2010)*
2) Lead: “Prevention of Childhood Lead Toxicity” *(Policy Statement; July 2016)*
3) Tuberculosis: “Pediatric Tuberculosis” Francis J. Curry National TB Center, 2010
Screening for Iron Deficiency

Ann Chen Wu, MD,* Leann Lesperance, MD, PhD,* Henry Bernstein, DO†

Objectives After completing this article, readers should be able to:

1. Determine the most common cause of iron deficiency in the United States.
2. Describe the pathogenesis of iron deficiency.
3. List populations at high risk for iron deficiency.
4. Outline the common signs and symptoms of iron deficiency.
5. Specify the American Academy of Pediatrics recommendations for screening for iron deficiency.

In the March and April issues of Pediatrics in Review, we published a two-part article on managing anemia in a pediatric office practice. This article expands on the various tests for iron deficiency, including some relatively new ones. These articles should be read as complementary.—RJH

Introduction
Iron deficiency is the most common nutritional deficiency in the world, responsible for a staggering amount of ill health, lost productivity, and premature death. Although its prevalence in the United States has declined since the late 1960s, iron deficiency with or without anemia still is seen frequently in infants, toddlers, adolescent females, and women of childbearing age. In fact, iron deficiency anemia remains the most common hematologic disease of infants and children.

Definitions
Anemia is defined as a low hemoglobin (Hgb) concentration or red blood cell (RBC) mass compared with age-specific norms. Anemia may be caused by decreased RBC production, increased RBC destruction, or blood loss. Based on the size of the RBC, hematologists categorize anemia as macrocytic, normocytic, or microcytic.

Iron is found in different compartments within the body. Total body iron (measured by ferritin), transport iron (measured by transferrin saturation), serum iron, and other hematologic and biochemical markers are used to describe the degrees of iron deficiency. Iron depletion refers to the earliest stage of diminishing iron stores in the setting of insufficient iron supply. Iron deficiency (without anemia) develops as these iron stores are depleted further and begin to impair Hgb synthesis. Finally, iron deficiency anemia results when the iron supply is insufficient to maintain normal levels of Hgb.

Epidemiology
According to current World Health Organization estimates, most of the world’s population may be iron-deficient, and at least one third (approximately 2 billion people) have anemia due to iron deficiency. As recently as the late 1960s, iron

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHr</td>
<td>reticulocyte hemoglobin content</td>
</tr>
<tr>
<td>FEP</td>
<td>free erythrocyte protoporphyrin</td>
</tr>
<tr>
<td>Hct</td>
<td>hematocrit</td>
</tr>
<tr>
<td>Hgb</td>
<td>hemoglobin</td>
</tr>
<tr>
<td>MCV</td>
<td>mean corpuscular volume</td>
</tr>
<tr>
<td>RBC</td>
<td>red blood cell</td>
</tr>
<tr>
<td>RDW</td>
<td>red blood cell distribution width</td>
</tr>
<tr>
<td>TIBC</td>
<td>total iron-binding capacity</td>
</tr>
<tr>
<td>TfR</td>
<td>serum transferrin receptor</td>
</tr>
<tr>
<td>TfSats</td>
<td>transferrin saturation</td>
</tr>
<tr>
<td>WIC</td>
<td>Women, Infants and Children</td>
</tr>
<tr>
<td></td>
<td>(Special Supplemental Food Program)</td>
</tr>
<tr>
<td>ZPP</td>
<td>zinc protoporphyrin</td>
</tr>
</tbody>
</table>

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†Associate Professor of Pediatrics, Harvard Medical School, Boston, MA.

Drs Wu and Bernstein have a grant from the Bayer Corporation to study the reticulocyte hemoglobin content test.
deficiency with or without anemia was highly prevalent in the United States. In 1971, the American Academy of Pediatrics Committee on Nutrition promoted the early use of iron-fortified formula instead of cow milk. One year later, the federal government introduced the Special Supplemental Food Program for Women, Infants and Children (WIC) Act to address iron and other nutritional deficiencies.

These initiatives have had a tremendous impact on the health and well-being of children. In one review of children ages 6 to 60 months who were participating in public health programs (such as WIC), the prevalence of anemia declined from 7.8% in 1975 to 2.9% in 1985.

Unfortunately, iron deficiency with or without anemia remains relatively common in the United States. In the third National Health and Nutrition Examination Survey (1988 to 1994), 13% of 1-year-olds, 5% of 2-year-olds, 9% of adolescent females (12 to 15 y), and 11% of women of childbearing age (16 to 49 y) were iron-deficient. Iron deficiency anemia was present in 3% of the toddlers studied, 2% of the adolescent females, and 3% to 5% of the women of childbearing age. Despite efforts at prevention and early detection, severe cases still occur.

The high prevalence of iron deficiency anemia in developing countries most often is attributed to nutritional deficiencies worsened by chronic blood loss due to parasitic infections and malaria. In the United States and other industrialized nations, the most common cause of iron deficiency with or without anemia is insufficient dietary iron. Infants, toddlers, adolescents, and pregnant women are particularly susceptible because of their relatively rapid growth and increased demand for iron.

The use of iron-fortified formula helps ensure adequate iron supplies for infants. However, toddlers often have diets that contain minimal amounts of iron-rich foods and large amounts of cow milk. The early introduction of whole cow milk (before 1 year of age) and consumption of greater than 24 oz/d after the first year of life increase the risk of iron deficiency.

Iron is absorbed from the gastrointestinal tract and transported in the blood bound to transferrin. Excess iron is stored primarily in the liver, bone marrow, and spleen as ferritin.

The developing fetus builds iron stores from maternal supplies. Unless maternal iron deficiency is severe, a normal term infant is born having sufficient iron stores for at least 4 to 6 months of postnatal growth. During the first months of life, the newborn uses iron at a high rate for accelerated growth and expansion of blood volume. By 4 months of age, an infant’s iron stores have decreased by 50% (and birthweight usually has doubled). The preterm infant has less time to accumulate iron in utero and, therefore, is born with lower iron stores. In addition, the preterm infant has a demonstrably faster rate of postnatal growth than the term infant and may deplete iron stores within 2 to 3 months.

Adequate iron must be available to meet these demands. Although the majority of iron in the body is conserved and reused, some is lost through the gastrointestinal tract, skin, and urine. During the first year of life, normal infants need to absorb approximately 0.8 mg/d of dietary iron (0.6 mg for growth, 0.2 mg to replace ongoing losses).

Toward the end of the second year of life, this swift
rate of growth begins to slow, so routine diets tend to include sufficient iron-rich foods to meet demands. Iron requirements increase again during adolescence due to rapid growth; adolescent females need additional iron to replace losses from menstruation.

There are two types of dietary iron: heme and non-heme. Heme iron already has been incorporated into the heme molecules of Hgb and myoglobin and is well absorbed by the body. Approximately 10% of the iron in a typical Western diet is heme iron, derived from meat, poultry, and fish. The majority of dietary iron is non-heme, in the form of iron salts. The bioavailability (amount absorbed by the body) of nonheme iron is highly variable and influenced by several factors, including current diet and the amount of iron already present in the body. Bran, dietary fiber, calcium, tannins (in tea and coffee), and oxalates, phytates, and polyphenols (in certain plant-based foods) inhibit iron absorption. Absorption is enhanced by reducing substances such as hydrochloric acid and ascorbic acid. The consumption of heme iron, even in small amounts, enhances the absorption of nonheme iron. Absorption of iron also is increased when total body stores are decreased or when the demand for iron increases, such as during adolescent growth spurts.

Mature human milk and cow milk contain the same amount of iron, approximately 0.5 mg/L; fortified formulas contain 10 to 13 mg/L. However, about 50% of iron from human milk is absorbed compared with only 10% from cow milk and less than 5% from iron-fortified formula. The reasons for the enhanced bioavailability of iron from human milk are not well understood, but they include a lower concentration of calcium and a higher concentration of ascorbic acid in human milk.

Clinical Aspects

The signs and symptoms of iron deficiency with and without anemia depend on the degree of deficiency and the rate at which the anemia develops. Children who have iron deficiency or mild-to-moderate anemia may show few, if any, signs or symptoms. Pallor is the most frequent sign of iron deficiency anemia. As the degree of anemia worsens, fatigue, exercise intolerance, tachycardia, cardiac dilatation, and systolic murmurs may develop. Splenomegaly can be found in 10% to 15% of affected patients. Infants and toddlers may demonstrate irritability and anorexia. However, even severe anemia may be asymptomatic; in one study of severe cases, 45% were diagnosed incidentally.

Iron deficiency anemia in infancy and early childhood is associated with developmental delays and behavior disturbances that may be irreversible. Numerous studies have documented lower test scores of mental and motor development among infants who had iron deficiency and iron deficiency anemia. In some follow-up studies, test results were normal after reversal of the anemia, but in others, developmental delays persisted, despite adequate treatment. The extent and persistence of brain involvement seem to depend on the age at which anemia first develops as well as its degree and duration. Although additional study is needed, the evidence linking iron deficiency and cognitive impairment is compelling. Iron supplements even have been shown to improve learning and memory in nonanemic iron-deficient adolescent females.

Iron deficiency anemia also is associated with poor growth and may produce other systemic abnormalities, such as blue sclerae, koilonychia, angular stomatitis, increased susceptibility to infection, and functional alterations in the gastrointestinal tract. Iron deficiency increases lead absorption and has been associated with pica, which may result in plumbism.

Making the Diagnosis

The differential diagnosis for anemia in children is broad, but it narrows once the anemia is classified further as microcytic. Iron deficiency and thalassemia minor are the most common causes of microcytic anemia in children. Microcytosis also results from lead poisoning, chronic disease (eg, inflammation, infection, cancer), sideroblastic anemia, and other rare conditions.

An array of tests can be used for evaluating anemia, but there is no single “best” test to diagnose iron deficiency with or without anemia. The “gold standard” for identifying iron deficiency is a direct test—bone marrow biopsy with Prussian blue staining. However, bone marrow aspiration is too invasive for routine use, so indirect assays generally are used. Hematologic tests are based on RBC features (eg, Hgb, mean corpuscular volume [MCV]), and biochemical tests are based on iron metabolism (eg, zinc protoporphyrin [ZPP], serum ferritin concentration). Hematologic tests generally are more readily available and less expensive than biochemical tests. However, biochemical tests detect iron deficiency before the onset of anemia and, therefore, may be worth the additional expense because the deleterious effects of iron deficiency appear to begin before anemia develops. A new hematologic test, reticulocyte hemoglobin content (CHr), may help diagnose iron deficiency before anemia is present.

Laboratory Parameters

The various hematologic and biochemical parameters used for screening and diagnosis are discussed below.

Laboratory Parameters

The various hematologic and biochemical parameters used for screening and diagnosis are discussed below.
Tables 1 and 2 summarize the values for these parameters along the spectrum from normal to iron deficiency anemia. In most cases, the results from several tests are necessary to make a definitive diagnosis.

**Hematologic Markers (Table 1)**

Measurement of Hgb, the concentration of oxygen-carrying protein, is a more sensitive and direct test for anemia than is measurement of hematocrit (Hct), the percentage of whole blood that is occupied by RBCs. Anemia generally is defined as Hgb values below the 5th percentile in a healthy reference population: less than 11.0 g/dL (110 g/L) for infants 6 months to 2 years of age. Both measurements are inexpensive, readily available tests for anemia and are used most commonly to screen for iron deficiency. However, Hgb and Hct are late markers of iron deficiency, are not specific for iron deficiency anemia, and are less predictive as the prevalence of iron deficiency anemia decreases.

The MCV, the average volume of RBCs, is reported in automated analyses, but it also can be calculated as the ratio of Hct to RBC count. MCV is useful for categorizing anemia as microcytic, normocytic, and macrocytic.

The red blood cell distribution width (RDW) measures variations in the size of RBCs and increases with iron deficiency. In one study of adults, high RDW (%/Hb) was 71% to 100% sensitive and 50% specific in diagnosing iron deficiency. Another study of 12-month-

**Table 1. Hematologic Markers for Identifying Iron Deficiency**

<table>
<thead>
<tr>
<th>Hematologic Marker</th>
<th>Normal</th>
<th>Iron Depletion</th>
<th>Iron Deficiency Without Anemia</th>
<th>Iron Deficiency Anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL) (g/L)</td>
<td>N</td>
<td>≥11 (110)</td>
<td>N</td>
<td>D</td>
</tr>
<tr>
<td>Mean corpuscular volume (fl)</td>
<td>N</td>
<td>70 to 100</td>
<td>N</td>
<td>D</td>
</tr>
<tr>
<td>Red blood cell distribution width (%)</td>
<td>&lt;15</td>
<td>&lt;15</td>
<td>N</td>
<td>I</td>
</tr>
<tr>
<td>Reticulocyte hemoglobin content (pg)</td>
<td>≥29</td>
<td>≥29</td>
<td>&lt;29</td>
<td>&lt;29</td>
</tr>
<tr>
<td>Reticulocytes</td>
<td>1 to 5</td>
<td>1 to 5</td>
<td>1 to 5</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

N = normal, I = increased, D = decreased
*Values for ages 6 mo to 2 y.

**Table 2. Biochemical Markers for Identifying Iron Deficiency**

<table>
<thead>
<tr>
<th>Biochemical Marker</th>
<th>Normal</th>
<th>Iron Depletion</th>
<th>Iron Deficiency Without Anemia</th>
<th>Iron Deficiency Anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum ferritin (mcg/dL) (mcg/L)</td>
<td>N</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>Serum iron (mcg/dL) (mcmol/L)</td>
<td>N</td>
<td>&lt;115 (20.6)</td>
<td>D</td>
<td>&lt;60 (10.7)</td>
</tr>
<tr>
<td>Total iron-binding capacity</td>
<td>N</td>
<td>360 to 390 (64.4 to 69.8)</td>
<td>N/l</td>
<td>≥350 (73.4)</td>
</tr>
<tr>
<td>Transferrin saturation (%)</td>
<td>N</td>
<td>&lt;30</td>
<td>D</td>
<td>&lt;20</td>
</tr>
<tr>
<td>Serum transferring receptor (nmol/L)</td>
<td>&lt;35</td>
<td>≥35</td>
<td>≥35</td>
<td>≥35</td>
</tr>
<tr>
<td>Zinc protoporphyrin/Heme (mcmol/mol)</td>
<td>&lt;40</td>
<td>&lt;40</td>
<td>≥40</td>
<td>≥70</td>
</tr>
</tbody>
</table>

N = normal, I = increased, D = decreased
old infants found that high RDW (>14%) was 100% sensitive and 82% specific. Because of its relatively low specificity, RDW is not as useful alone as a screening test, but it is used frequently in conjunction with MCV to differentiate among various causes of anemia. For example, RDW is high in iron deficiency anemia, but low in thalassemia minor.

The reticulocyte count measures circulating immature RBCs and decreases with iron deficiency. However, the reticulocyte count increases with blood loss. In severe cases of iron deficiency anemia coupled with blood loss, the reticulocyte count may be slightly elevated. This parameter often is used for assessing the response to iron supplements.

CHr, the concentration of iron-containing protein in reticulocytes, can be measured in some hematology laboratories by using the same automated flow cytometer that provides RBC and reticulocyte indices. CHr has been shown to be an early indicator of iron deficiency in healthy subjects receiving recombinant human erythropoietin. A retrospective laboratory analysis performed on 210 children showed that low CHr was the best predictor of iron deficiency compared with Hgb, MCV, serum iron, RDW, and transferrin saturation.

**Biochemical Markers (Table 2)**

Ferritin is a storage compound for iron, and serum ferritin levels normally correlate with total iron stores. As iron stores are depleted, serum ferritin levels decline and are the earliest marker of iron deficiency. Serum ferritin has high specificity for iron deficiency, especially when combined with other markers such as Hgb. However, the test is expensive and has limited availability in a clinic setting; therefore, it is not used commonly for screening. In addition, serum ferritin is an acute-phase reactant that can become elevated in the setting of inflammation, chronic infection, or other diseases.

Serum iron concentration can be measured directly and generally decreases as iron stores are depleted. However, serum iron may not reflect iron stores accurately because it is influenced by several additional factors, including iron absorption from meals, infection, inflammation, and diurnal variation.

Total iron-binding capacity (TIBC) measures the availability of iron-binding sites. Extracellular iron is transported in the body bound to transferrin, a specific carrier protein. Hence, TIBC indirectly measures transferrin levels, which increase as serum iron concentration (and stored iron) decreases. Unfortunately, this test also is affected by factors other than iron status. For example, TIBC is decreased with malnutrition, inflammation, chronic infection, and cancer.

Transferrin saturation (Tfsat) indicates the proportion of occupied iron-binding sites and reflects iron transport rather than storage. Tfsat is calculated from two measured values: serum iron concentration divided by TIBC, expressed as a percent. Low Tfsat implies low serum iron levels relative to the number of available iron-binding sites, suggesting low iron stores. Tfsat decreases before anemia develops, but not early enough to identify iron depletion. Tfsat is influenced by the same factors that affect TIBC and serum iron concentration and is less sensitive to changes in iron stores than is serum ferritin.

Serum transferrin receptor (TfR) also can be detected in some laboratories via immunoassay. This receptor is present on reticulocytes and is shed from the membrane as the reticulocyte matures. With tissue iron deficiency, there is a proportional increase in the number of transferrin receptors. Although not a readily available test, TfR is useful as an early marker of iron deficiency, but it also may differentiate between iron deficiency anemia and anemia of chronic disease. In one study, TfR was increased in patients who had iron deficiency anemia, but not in patients who had anemia in the setting of acute infection.

ZPP is formed when zinc is incorporated into protoporphyrin in place of iron during the final step of heme biosynthesis. Under normal conditions, the reaction with iron predominates, but when iron is in short supply, the production of ZPP increases and the ZPP/heme ratio becomes elevated. ZPP/heme reflects iron status during hemoglobin synthesis and detects iron deficiency before the onset of anemia. This test is reported most accurately as the ZPP/heme ratio, but it also is reported simply as ZPP. ZPP is not the same as free erythrocyte protoporphyrin (FEP) or erythrocyte protoporphyrin, which is created in the laboratory when zinc is stripped from ZPP and also is used as a marker of iron deficiency without
anemia. Although ZPP and FEP can be measured by using affordable, clinic-based methods, both are elevated with lead poisoning and chronic disease, making them less useful for the diagnosis of anemia.

**Diet**
Dietary history may be suggestive of iron deficiency and has been studied as a possible marker for microcytic anemia. In one study of healthy inner-city children between the ages of 15 and 60 months, dietary iron deficiency was defined as: 1) fewer than 5 servings per week of each of meat, cereals or bread, vegetables, and fruit; 2) more than 16 oz per day of milk; or 3) daily intake of fatty snacks or sweets or greater than 16 oz of soda. As a screening test for microcytic anemia, the study found that diet history had 71% sensitivity, 79% specificity, and 97% negative predictive value. Similarly, low specificity was demonstrated in another prospective study that used a questionnaire to assess diet, WIC participation, and medical and family history. Response to a clinical trial of iron therapy is used by most clinicians as a practical method of diagnosing iron deficiency anemia.

Because of the low specificity of dietary history for iron deficiency anemia, it cannot eliminate the need for further laboratory testing. However, dietary history may be useful in identifying children at low risk for iron deficiency (negative predictive value) and is essential for the prevention and management of iron deficiency anemia.

**Prevention**
Clinicians play an essential role in preventing iron deficiency and its related medical and developmental problems. Primary prevention involves counseling at routine health supervision visits from infancy through adolescence to help ensure that ingestion of dietary iron is adequate (Table 3). Secondary prevention involves regular screening, prompt diagnosis, and appropriate treatment of iron deficiency.

Hgb and Hct are the most commonly used screening tests for iron deficiency. They are readily available and cost-effective markers of anemia. However, the use of anemia as a marker for iron deficiency depends on the prevalence of iron deficiency anemia in a population. Increased prevalence improves the positive predictive value of anemia as a screening test for iron deficiency.

The American Academy of Pediatrics recommends universal screening with Hgb or Hct once between ages 9 and 12 months of age and again 6 months later in communities and populations that have a high prevalence of iron deficiency anemia, including children eligible for WIC, children of migrant workers, or recently arrived refugee children. For communities that have low rates of anemia, selective screening at the same intervals is recommended for children at risk for iron deficiency, including preterm or low-birthweight infants, infants fed a diet of noniron-fortified infant formula, infants introduced to cow milk before age 12 months, breastfed infants who are receiving inadequate dietary iron after age 6 months, and children who consume more than 24 oz of cow milk per day.

After 2 years of age, routine screening usually is not necessary. Risk can be assessed regularly and children screened who have a previous history of iron deficiency, evidence of low iron intake, or special health needs that increase the risk for iron deficiency (e.g., chronic infection, inflammatory disorders, chronic or acute blood loss, restricted diets, use of medications that interfere with iron absorption).

The American Academy of Pediatrics recommends screening all adolescents once between ages 11 and 21.

<table>
<thead>
<tr>
<th>Age of Child</th>
<th>Preventive Counseling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>• Encourage breastfeeding exclusively for 4 to 6 months after birth and continue breastfeeding until 12 months of age.</td>
</tr>
<tr>
<td></td>
<td>• For infants who are not breastfed, recommend only iron fortified formula (10 to 12 mg/L).</td>
</tr>
<tr>
<td></td>
<td>• For breastfed infants who were preterm or of low birthweight, recommend 2 to 4 mg/kg per day of iron drops to a maximum of 15 mg/d and screen for anemia before 6 months of age.</td>
</tr>
<tr>
<td>4 to 6 months</td>
<td>• Recommend starting iron-fortified infant cereal.</td>
</tr>
<tr>
<td>6 months</td>
<td>• Recommend two or more servings per day of iron-fortified infant cereal to meet iron requirements.</td>
</tr>
<tr>
<td></td>
<td>• Recommend one feeding per day of foods rich in vitamin C (fruits and vegetables) to improve iron absorption.</td>
</tr>
<tr>
<td>9 to 12 months</td>
<td>• Begin introducing plain pureed meats. Provide appropriate counseling for vegetarian families.</td>
</tr>
<tr>
<td></td>
<td>• Discourage introduction of cow milk before 12 months of age.</td>
</tr>
<tr>
<td>1 to 5 years</td>
<td>• Encourage iron-rich foods.</td>
</tr>
<tr>
<td>≥ 6 years</td>
<td>• Limit milk consumption to 24 oz/d.</td>
</tr>
<tr>
<td></td>
<td>• Encourage iron-rich foods and foods rich in vitamin C to improve iron absorption, especially in menstruating females.</td>
</tr>
</tbody>
</table>

### Table 3. Diet Counseling to Prevent Iron Deficiency in Children
years and screening menstruating females annually. The Centers for Disease Control and Prevention recommend annual screening of adolescent females if their risk is increased (eg, excessive menstrual blood loss, low iron intake, previous diagnosis of iron deficiency); otherwise, anemia should be screened for every 5 to 10 years.

In the appropriate clinical setting, an abnormally low Hgb or Hct combined with a dietary history of low iron intake strongly suggests iron deficiency anemia. Further laboratory testing, such as measurement of serum ferritin, will help to confirm the diagnosis, but in most cases is not necessary. Response to a therapeutic trial of supplemental iron is considered clinically diagnostic. If a child who has a normal diet that contains adequate servings of iron-rich foods is anemic, additional evaluation may be indicated to look for blood loss (eg, occult rectal bleeding).

Presumptive iron deficiency is treated with oral iron salts, most commonly over-the-counter ferrous sulfate, which is inexpensive and relatively well absorbed. Dosages are calculated for elemental iron: children receive 3 to 6 mg/kg per day (qd or tid), and adolescents receive 60 mg/dose (qd or bid). If the iron deficiency is nutritional, the response to iron typically is rapid. Parenteral iron can be given if oral iron is not tolerated; intramuscular iron injections usually are not appropriate. Erythrocyte transfusion should be used only if the anemia is causing severe cardiovascular compromise; hypervolemia and cardiac dilatation may result from rapid correction of the anemia.

After 1 month of therapy, the Hgb measurement should be repeated. An increase of 1 g/dL (10 g/L) or greater confirms the diagnosis of iron deficiency anemia. No improvement in Hgb should prompt further evaluation of the anemia with additional laboratory tests, including MCV, RDW, and serum ferritin, and a search for possible sources of blood loss. Iron therapy should be continued for an additional 2 to 3 months after Hgb has returned to a normal level, and Hgb should be remeasured approximately 6 months after discontinuation of iron therapy.

The evidence is clear that early diagnosis and adequate treatment of iron deficiency are critical to prevention or reversal of any negative medical or behavioral effects. As advocates for children, pediatricians must screen for this common nutritional deficiency actively and accurately. Hgb and Hct are the most readily available and cost-effective screening tests, but newer tests that detect iron deficiency before the onset of anemia (eg, CHr) are being studied prospectively in healthy infants and may gain widespread acceptance, particularly as the prevalence of iron deficiency anemia decreases.

Suggested Reading
Fairbanks VF. Laboratory testing for iron status. Hosp Pract. 1991;26S:17–24
Looker AC, Dallman PR, Carroll MD, Gunter EW, Johnson CL. Prevalence of iron deficiency in the United States. JAMA. 1997;277:973–976
EXCERPT FROM AAP CPG ON IRON DEFICIENCY

Until the controversies are settled, the cur-
cumstances and evaluated through well-
rently available evidence supports the
1. Term, healthy infants have suffi-
and specific to the diagnosis. In addi-
the AAP, the World Health Organiza-
ion for Pediatric Gastroenterology, Hepatology and
also support the use of the
measurement of TRF1 as a screening test
once the method has been validated and
normal values for infants and toddlers
have been established.

Another step to improve the current
screening system is to use technology-
icted reminders for screening and
follow-up of infants and toddlers with a
diagnosis of ID/IDA. Reminders could
be incorporated into electronic health
records, and there should be docu-
mentation that Hb concentrations have
returned to the normal range. The effi-
cacy of any program for minimizing ID
and IDA should be tracked scien-
tifically and evaluated through well-
planned surveillance programs.

SUMMARY

Given that iron is the world’s most
common single-nutrient deficiency
and there is some evidence of adverse
effects of both ID and IDA on cognitive
and behavioral development, it is im-
portant to minimize ID and IDA in in-
fants and toddlers without waiting for unequivo-
cal evidence. Controversies
remain regarding the timing and
methods used for screening for ID/IDA
as well as regarding the use of iron
supplements to prevent ID/IDA. Al-
though further study is required to
generate higher levels of evidence to
settle these controversies, the cur-
rently available evidence supports the
following recommendations.

1. Term, healthy infants have suffi-
cient iron for at least the first 4
months of life. Human milk
contains very little iron. Exclusively
breast-fed infants are at increasing
risk of ID after 4 completed months
of age. Therefore, at 4 months of
age, breastfed infants should be
supplemented with 1 mg/kg per
day of oral iron beginning at 4
months of age until appropriate
iron-containing complementary
foods (including iron-fortified
cereals) are intro-
duced in the diet
(see Table 3). For partially
breastfed infants, the pro-
portion of human milk versus for-
mula is uncertain; therefore, begin-
ing at 4 months of age, partially
breastfed infants (more than half of their
daily feedings as human milk) who are
not receiving iron-containing
complementary foods should also
receive 1 mg/kg per day of
supplemental iron.

2. For formula-fed infants, the iron
needs for the first 12 months of life
can be met by a standard infant for-
mula (iron content: 10–12 mg/L) and
the introduction of iron-containing
complementary foods after 4 to 6
months of age, including iron-fortified
cereals (Table 3). Whole milk should
not be used before 12 completed
months of age.

3. The iron intake between 6 and 12
months of age should be 11 mg/day.
When infants are given comple-
mentary foods, red meat and vegetables
with higher iron content should be
introduced early (Table 3). To aug-
ment the iron supply, liquid iron
supplements are appropriate if
iron needs are not being met by the
intake of formula and complemen-
tary foods.

4. Toddlers 1 through 3 years of age
should have an iron intake of 7 mg/
day. This would be best delivered by
eating red meats, cereals fortified
with iron, vegetables that contain
iron, and fruits with vitamin C, which
augments the absorption of
iron (Tables 3 and 4). For toddlers
not receiving this iron intake, liquid
supplements are suitable for chil-
dren 12 through 36 months of age,
and chewable multivitamins can be
used for children 3 years and older.

5. All preterm infants should have an
iron intake of at least 2 mg/kg per
day through 12 months of age,
which is the amount of iron sup-
plied by iron-fortified formulas. Pre-
term infants fed human milk should
receive an iron supplement of 2
mg/kg per day by 1 month of age,
and this should be continued until
the infant is weaned to iron-fortified
formula or begins eating complemen-
tary foods that supply the 2 mg/kg of
iron. An exception to this practice
would include infants who have re-
ceived an iron load from multiple
transfusions of packed red blood
cells.

6. Universal screening for anemia
should be performed at approxi-
mately 12 months of age with deter-
mination of Hb concentration and
an assessment of risk factors asso-
ciated with ID/IDA. These risk fac-
tors would include low socioeco-
omic status (especially children of
Mexican American descent (Table
1)), a history of prematurity or low
birth weight, exposure to lead, ex-
clusive breastfeeding beyond 4
months of age without supplemen-
tal iron, and weaning to whole milk
or complementary foods that do not
include iron-fortified cereals or
foods naturally rich in iron (Table 3). Additional risk factors are the feeding problems, poor growth, and inadequate nutrition typically seen in infants with special health care needs. For infants and toddlers (1–3 years of age), additional screening can be performed at any time if there is a risk of ID/IDA, including inadequate dietary iron intake.

7. If the Hb level is less than 11.0 mg/dL at 12 months of age, then further evaluation for IDA is required to establish it as a cause of anemia. If there is a high risk of dietary ID as described in point 6 above, then further testing for ID should be performed, given the potential adverse effects on neurodevelopmental outcomes. Additional screening tests for ID or IDA should include measurement of:
   - SF and CRP levels; or
   - CHr concentration.

8. If a child has mild anemia (Hb level of 10–11 mg/d) and can be closely monitored, an alternative method of diagnosis would be to document a 1 g/dL increase in plasma Hb concentration after 1 month of appropriate iron-replacement therapy, especially if the history indicates that the diet is likely to be iron deficient.

9. Use of the TfR1 assay as screening for ID is promising, and the AAP supports the development of TfR1 standards for use of this assay in infants and children.

10. If IDA (or any anemia) or ID has been confirmed by history and laboratory evidence, a means of carefully tracking and following infants and toddlers with a diagnosis of ID/IDA should be implemented. Electronic health records could be used not only to generate reminder messages to screen for IDA and ID at 12 months of age but also to document that IDA and ID have been adequately treated once diagnosed.

ADDENDUM

Development of This Report

This report was written by the primary authors after extensive review of the literature using PubMed, previous AAP reports, Cochrane reviews, and reports from other groups.1,6,7,48,77

The report was also submitted to the following sections and committees of the AAP that were asked to comment on the manuscript: Committee on Fetuses and Newborns (COFHN); Committee on Psychosocial Aspects of Child and Family Health (COPACH); Section on Administration and Practice Management (SOPAM); Section on Developmental and Behavioral Pediatrics (SODBP); Section on Gastroenterology, Hepatology, and Nutrition (SOGHN); Section on Hematology and Oncology (SOHO); and Section on Breast Feeding (SOBr).

Additional comments were sought from the Centers for Disease Control and Prevention (CDC), the Department of Agriculture (WIC), the National Institutes of Health (NIH), and the Food and Drug Administration (FDA), because these governmental agencies were involved in the development of the statement and will necessarily deal with its impact. As it was developed it was extensively reviewed and revised by members of the AAP Committee on Nutrition, who unanimously approved this clinical report. It is openly acknowledged that where the highest levels of evidence are absent, the opinions and suggestions of members of the Committee on Nutrition as well as other groups consulted for this statement were taken into consideration in developing this clinical report.

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Detection of Lead Poisoning

How is Lead Poisoning Detected?

Lead poisoning usually is detected by measuring the level in blood. Many screening procedures use capillary blood as point-of-care testing. While finger-prick samples are appropriate for screening tests, all elevated capillary levels should have confirmation with a venous blood draw since capillary tests can yield frequent false positives. An elevated capillary lead level indicates that lead is in the child’s environment even if the venous level is low, however, so primary prevention and education should be initiated.

The current Bright Futures/AAP Periodicity Schedule recommends a risk assessment at the following well-child visits: 6 months, 9 months, 12 months, 18 months, 24 months, and at 3, 4, 5 and 6 years of age. The recommendation is to do a risk assessment, and do a blood lead level test only if the risk assessment comes back positive. According to the AAP and CDC, universal screens or blood lead level tests are not recommended anymore except for high prevalence areas with increased risk factors as described in a 2012 CDC report, such as older housing.

Most initial blood lead level tests are now performed as a result of a positive risk assessment or parental concern rather than because children have symptoms that suggest lead poisoning. However, children of all ages who are recent immigrants, refugees, or adoptees are more likely to have elevated blood lead levels and should be screened at the earliest opportunity. Additionally, children who live in lower socioeconomic areas may be at higher risk.

Medicaid-eligible patients also tend to be at higher risk for lead exposure because many live in these lower socioeconomic areas. Many state Medicaid/Early and Periodic Screening, Diagnostic, and Treatment programs require a universal blood lead level test at the 12-month and 24-month visits, no matter the prevalence of elevated levels based on factors such as where the patient lives. While these programs have been directed to transition their requirements to be more in line with prevalence data targeted screening, most states have not completed these efforts. For more information, see the CMCS Information Bulletin, Target Lead Screening Plans or the CDC Guide For States Interested in Transitioning to Targeted Blood Lead Screening for Medicaid-Eligible Children. Pediatricians should ask their state Medicaid office to determine which requirements they should follow when it comes Medicaid-eligible patients. This information is available for most states on the CDC Web site. However, pediatricians may err on the side of caution and obtain a lead level in Medicaid patients and those who they feel are at high risk.

Because of lead’s effects on the developing fetus, some states have developed lead screening guidelines for pregnant women. The CDC recently published guidelines on the screening of pregnant women for lead, medical and environmental management, and follow-up of mothers and infants when maternal lead levels are at least 5 micrograms per deciliter. Care of the infant includes measuring cord or neonatal blood lead to establish a baseline. Once the child is born, the guideline calls for an interruption of breastfeeding only if the maternal blood lead level is 40 micrograms per deciliter or more. Above this level, women should pump and discard their milk until after their blood lead level decreases below that benchmark.

For families using tap water to reconstitute infant formula or juice, or where there has been local concern, tap water testing may be recommended. To help determine whether a home’s water might contain lead, parents can call the EPA Safe Drinking Water Hotline at (800) 426-4791 or their local health department to find out about water testing. Well water should be tested for lead when the well is new and tested again when a pregnant woman, infant or child less than 18 years of age moves into the home. For a discussion about using well water to feed infants, see the AAP policy statement on drinking water from private wells. Most water filters remove lead.

When is Diagnostic Testing Warranted?

Some experienced clinicians measure the blood lead level in children with growth retardation, speech or language dysfunction, anemia, and attentional or behavioral disorders, especially if the parents have a specific concern about lead or about health effects from environmental chemicals. However, elevated blood lead levels that continue into school age are unusual, even if peak blood lead level at 2 years of age was high and the child’s home exposures have not been addressed. Therefore, a relatively low blood lead level in a school-aged child does not rule out earlier lead poisoning. If the question of current lead poisoning arises, however, the only reliable way to make a diagnosis is with blood lead measurement. Hair or urine lead levels give no useful information and should not be performed.

Resources

CDC's Childhood Lead Poisoning Prevention Program
CDC Low Level Exposure Harms Children: A Renewed Call for Primary Prevention
Pediatric Environmental Health Specialty Unit
CMCS Targeted Lead Screening Plans
CMS Guide for States Interested in Transitioning to Targeted Blood Lead Screening for Medicaid-Eligible Children
Blood Lead Levels in Children: What Parents Need to KnowWhere We Stand: Lead Screening
**2016 Maryland Guidelines for the Assessment and Management of Childhood Lead Exposure**

For Children 6 Months to 72 Months of Age

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### Table 1: Guidelines for Blood Lead Level Testing in Children 6 Months to 72 Months of Age (COMAR 10.11.04, as of 3/28/2016)

<table>
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</table>

**For ALL children born on or after 1/1/15, OR on Medicaid, OR ever lived in a 2004 At-Risk Zip code***

**For children born before 1/1/15, AND not on Medicaid, AND never lived in a 2004 At-Risk ZIP code***

<table>
<thead>
<tr>
<th>Age Range</th>
<th>6 Months</th>
<th>9 Months</th>
<th>12 Months</th>
<th>15 Months</th>
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</table>

**Table 2: Schedule for Confirmatory Venous Sample after Initial Capillary Test**

**Table 3: Abbreviated Clinical Guidance for Management of Lead in Children Ages 6 Months to 72 Months (Full Guidelines in Table 5)**

#### Blood Lead Level

**Follow-up testing**

- < 5 mcg/dL: On schedule
  - Table 1
  - Management includes screening and testing on schedule.
  - Continue education for prevention.

- 5 – 9 mcg/dL: 3 months
  - Table 4
  - All of above AND: Investigate for exposure source in environment and notify health department.

- 10 – 44 mcg/dL: 1 month
  - Table 4
  - Consult Table 5

**Indications for Testing**

- Parental/guardian request
- Possible lead exposure or symptoms of lead poisoning, either from health history, development assessment, physical exam or newly positive item on Lead Risk Assessment Questionnaire. (Questions can be found in the Lead Risk Assessment Questionnaire section of this document)
- Follow-up testing on a previously elevated Blood Lead Level (Table 4)
- Missed screening: If 12 month test was indicated and no proof of test, then perform as soon as possible after 12 months and then again at 24 months. If 24 month test was indicated and no proof of test, then perform test as soon as possible.
- For more information about lead testing of pregnant and breastfeeding women, see: [http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf](http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf)

**Seasonal variation of Blood Lead Levels exists, greater exposure in the summer months may necessitate more frequent follow-up.***

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**Table 4: Schedule for Follow-up Venous Blood Lead Testing after Blood Lead Level ≥ 5 mcg/dL**

<table>
<thead>
<tr>
<th>Blood Lead Level</th>
<th>Early follow-up testing (2-4 tests after identification)</th>
<th>Later follow-up testing after blood lead level declining</th>
</tr>
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<tbody>
<tr>
<td>5 – 9 mcg/dL</td>
<td>1 – 3 months***</td>
<td>6 – 9 months</td>
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<tr>
<td>10 – 19 mcg/dL</td>
<td>1 – 3 months***</td>
<td>3 – 6 months</td>
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<tr>
<td>20 – 24 mcg/dL</td>
<td>1 – 3 months***</td>
<td>1 – 3 months</td>
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<tr>
<td>25 – 44 mcg/dL</td>
<td>2 weeks – 1 month</td>
<td>1 month</td>
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<tr>
<td>≥ 45 mcg/dL</td>
<td>As Soon As Possible!</td>
<td>As Soon As Possible, based on treatment plan</td>
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*See back of chart for list of 2004 At-Risk ZIP codes*
Use validated developmental screen for levels 10 – 19 mcg/dL, such as Ages and Stages Questionnaire (ASQ). Refer children as appropriate for follow-up blood lead testing in Table 4.

Initial confirmed blood lead of 5 – 9 mcg/dL may not require home environmental investigation. Contact LHD for more guidance.

Includes discussion of pica and lead sources including house paints (before 1978), ceramics, paint on old furniture, soil, foreign travel, traditional folk medicines, certain imported items (candies, food, jewelry, toys, cosmetics, pottery), and parental occupations that can bring home lead dust and debris.

Follow-up blood lead monitoring should be considered. Children with BLL over 20 mcg/dL should be evaluated in consultation with an experienced clinician, specialist, or Local Health Department regarding further evaluation.

Use validated developmental screen for levels 10 – 19 mcg/dL, such as Ages and Stages Questionnaire (ASQ). Refer children as appropriate for further evaluation. Children with BLL over 20 mcg/dL should be evaluated in consultation with an experienced clinician, specialist, or Local Health Department regarding further evaluation.

A Notice of Defect is a written notice that tells the landlord that there is chipping, peeling paint or structural defect in the home that is in need of repair. A Notice of Defect may also tell the landlord that a ‘Person at Risk’ (a child under the age of six or a pregnant woman) has a lead level of 10 or above and that repairs need to be made in the home.

A Notice of Defect must be sent by certified mail, return receipt (be certain to retain a copy of the return receipt) and the rental property owner has 30 days to repair the listed defects. It is illegal for a property owner to evict a tenant or raise the rent for reporting problems and/or defects in the home or that a child has been poisoned by lead. A rental property owner CAN evict a tenant if they fail to make timely rental payments. To download a copy of the Notice of Defect form, visit: http://www.mde.state.md.us/programs/Land/Documents/LeadPamphlets/LeadPamphletMDENoticeOfTenantsRights.pdf

For more information or assistance with filing a Notice of Defect, contact the Maryland Department of the Environment, Lead Poisoning Prevention Program or the Green & Healthy Homes Initiative.

Lead Risk Assessment Questionnaire Screening Questions:
1. Lives in or regularly visits a house/building built before 1978 with peeling or chipping paint, recent/ongoing renovation or remodeling?
2. Ever lived outside the United States or recently arrived from a foreign country?
3. Sibling, housemate/playmate being followed or treated for lead poisoning?
4. If born before 1/1/2015, lives in a 2004 "at risk" zip code? Use validated developmental screen for levels 10 – 19 mcg/dL, such as Ages and Stages Questionnaire (ASQ). Refer children as appropriate for further evaluation. Children with BLL over 20 mcg/dL should be evaluated in consultation with an experienced clinician, specialist, or Local Health Department regarding further evaluation.
5. Frequently puts things in his/her mouth such as toys, jewelry, or keys, eats non-food items (pica)?
6. Contact with an adult whose job or hobby involves exposure to lead?
7. Lives near an active lead smelter, battery recycling plant, other lead-related industry, or road where soil and dust may be contaminated with lead?
8. Uses products from other countries such as health remedies, spices, or food, or store or serve food in leaded crystal, pottery or pewter?

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<th>Confirmed Blood Lead Level (mcg/dL)</th>
<th>&lt; 5</th>
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Clinical Resources
Mid-Atlantic Center for Children's Health & the Environment
Pediatric Environmental Health Specialty Unit
866-622-2431
kidsandenvironment@georgetown.edu
www.pehsu.org/region3.html

Mt. Washington Pediatric Hospital
Lead Treatment Program
410-367-2222
www.mwph.org

Maryland Poison Control
800-222-1222

Regulatory Programs and Resources
Maryland Department of Health and Mental Hygiene
677-703-3266
dhhm.envhealth@maryland.gov
http://phoa.mdh.maryland.gov/OEHPP/Pages/Lead.aspx

Maryland Department of the Environment
Lead Poisoning Prevention Program
410-337-3825/800-776-2706
http://www.mde.state.md.us/programs/Land/LeadPoisoningPrevention/Pages/index.aspx

Local Health Departments
http://mdkids.state.maryland.us/PAGES/DEPARTMENTS.ASPX

Centers for Disease Control and Prevention
www.cdc.gov/nceh/lead

Green & Healthy Homes Initiative
410-534-6447
800-370-5223
www.greenandhealthyhomes.org/

2004 Maryland Childhood Lead Poisoning Targeting Plan At Risk Areas by ZIP Code

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</table>

1 Refer to information about confirmation of capillary tests in Table 2.
2 Includes discussion of pica and lead sources including house paints (before 1978), ceramics, paint on old furniture, soil, foreign travel, traditional folk medicines, certain imported items (candies, food, jewelry, toys, cosmetics, pottery), and parental occupations that can bring home lead dust and debris (e.g. painting, construction, battery reclamation, ceramics, furniture refinishers, radiator repair).
3 Exposure/environmental history to identify potential lead sources. (see screening questions). Consider Notice of Defect (information at night) for child living in pre-1978 rental property.
4 Initial confirmed blood lead of 5 – 9 mcg/dL may not require home environmental investigation. Contact LHD for more guidance.
5 Refer to schedule of follow-up blood lead testing in Table 4.
6 Contact LHD for more information about care coordination for blood lead levels of 5 – 9 mcg/dL.
SCREENING INFORMATION

District of Columbia law requires that all children under the age of six be screened for lead. This “universal screening” requirement is in place because close to 90% of the District’s housing was built prior to 1978, the year lead paint was banned for use in the nation’s housing stock. This means that an overwhelming majority of the children living in the nation’s capital are at risk of exposure to lead paint. Exposure can occur when paint is not well maintained, and it can also occur when renovation or maintenance work is not conducted in a lead-safe manner.

It does not take much exposure to lead for harmful health consequences to occur, and the younger the child, the more potentially serious the health hazard becomes. Pregnant women are also a special risk group, because the lead they are exposed to goes right to the fetus. Adults are also at risk of health consequences when they are exposed to lead, but it generally takes a much heavier dose of lead exposure for harm to occur to an adult than it does for a child. Bottom line: there is no safe level of lead exposure, and the key to prevent harm is to prevent exposure.

District law requires that all children who live here must get screened as follows:

- Between the ages of 6 months and 14 months of age, and again
- Between the ages of 22 and 26 months of age.
- If a child who lives in the District has not been screened at these ages, they must be screened at least once before they are 6 years old.
- District law also requires that all children must be screened before entering daycare, pre-school, or kindergarten. This can be done at any of the above-listed opportunities.

In addition to the above, children should be screened again whenever there is a possibility they have been recently exposed to lead.

If a child is found to have an elevated blood lead level (consisting of 10 micrograms of lead per deciliter of blood or more, expressed as 10 µg/dL or greater), the District Government will provide case management assistance. This consists of a case manager meeting with the child’s family to answer questions the family may have and to provide helpful recommendations to assist the family to prevent further potential harm.

Case management assistance that the District Department of the Environment (DDOE) provides also involves what is called an “environmental investigation.” This is basically a lead inspection of the child’s home and other places that could be where the child got exposed to lead. Once the DDOE investigation is complete, the family will receive a “risk assessment report” which will detail where lead hazards were found. If lead-based paint hazards are found in the child’s home, the property owner will receive instructions about next steps that are needed to eliminate those hazards.
Screening for Lead Exposure

A. Local Requirements

DC has long required screening for lead at both the 12 and 24 month visit. Maryland has required a similar venous/capillary lead test at 12 and 24 months, regardless of where the patient lives since 2015. The clinic has point-of-care lead testing. If POC ("fingerstick" or capillary) lead is abnormal, it should be confirmed via venipuncture sample sent to the lab. Virginia still has no requirement for universal screening, but it is prudent to screen these children as well.

B. A Basic Personal-Risk Questionnaire*

<table>
<thead>
<tr>
<th>_Yes</th>
<th>_No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Does your child live in or regularly visit a house or child care facility built before 1950?</td>
<td></td>
</tr>
<tr>
<td>_Yes</td>
<td>_No</td>
</tr>
<tr>
<td>2. Does your child live in or regularly visit a house or child care facility built before 1978 that is being or has recently been renovated or remodeled (within the last 6 months)?</td>
<td></td>
</tr>
<tr>
<td>_Yes</td>
<td>_No</td>
</tr>
<tr>
<td>3. Does your child have a sibling or playmate who has or did have lead poisoning?</td>
<td></td>
</tr>
</tbody>
</table>

* Adapted from the Centers for Disease Control and Prevention. The state or local health department may recommend alternative or additional questions based on local conditions.
* If the answers to the questions are "no," a screening test is not required, although the provider should explain why the questions were asked to reinforce anticipatory guidance. If the answer to any question is "yes" or "not sure," a screening test should be considered.
* Note that all Medicaid-eligible children must be screened both at 1 and 2 years of age.

C. A Detailed Clinical Evaluation of the Child with Lead Exposure

**Medical history**
- Symptoms
- Developmental history
- Mouthing activities; Pica
- Previous blood lead levels
- Family history of lead poisoning

**Nutritional history**
- Take a dietary history, specifically iron intake
- Access to imported food, cosmetics, folk remedies?
- Food prepared/imported in pottery or metal containers?
- History of food stamps or participation in WIC?

**Environmental history**
- What is the age and condition of the residence/other structure in which the child spends time?
- Is there evidence of chewed or peeling paint on woodwork, furniture, or toys?
- How long has the family lived at their residence?
• Have there been recent renovations or repairs to the house?
• Does the home contain vinyl blinds made overseas and purchased before 1997?
• Are the windows new?
• Are there other sites at which the child spends significant amounts of time?
• What is the condition of indoor play areas?
• Do outdoor play areas contain bare soil that may be contaminated?
• How does the family attempt to control dust and dirt?
• Does the child exhibit much hand-to-mouth activity or eating of non-edible objects (pica)?
• Are the child's hands washed before meals and snacks?
• What are the occupations and hobbies of adult household members? (Fishing, working with ceramics or stained glass, and hunting increase risk of lead exposure.)
• Are painted materials or unusual materials burned in household fireplaces?

C. Recommended Follow-up Services, According to Diagnostic BLL

<table>
<thead>
<tr>
<th>BLL (µg/dL)</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;10</td>
<td>No action required</td>
</tr>
</tbody>
</table>
| 10-14      | - Obtain a confirmatory venous BLL within 1 month.  
- If still within this range, provide education to decrease blood lead exposure  
- Repeat BLL test within 3 months |
| 15-19      | - Obtain a confirmatory venous BLL within 1 month.  
- If still within this range, take environmental history and provide education to decrease exposure.  
- Repeat venous BLL test within 2 months.  

Proceed according to actions for 20–44 µg/dL if:  
• The follow-up blood lead concentration is still in this range at least 3 months after initial test; or  
• Blood lead concentration increases |
| 20-44      | Obtain a confirmatory venous BLL within 1 week.  

If still within this range:  
• Conduct a complete history & physical (including an environmental and nutritional assessment)  
• Provide education to decrease blood lead exposure and to decrease lead absorption  
• Refer the patient to the local health department for case management that should include a detailed environmental investigation with lead hazard reduction and appropriate referrals for support services.  
• Check CBC and iron stores  
• Neurodevelopmental monitoring  
• Abdominal radiography (if particulate lead ingestion is suspected) |
| 45-69      | Obtain a confirmatory venous BLL within 2 days.  

If still within this range:  
• Begin chelation after consulting clinicians experienced in lead toxicity treatment  
• Conduct a complete history & physical (including an environmental and nutritional assessment) |
• Provide education to decrease blood lead exposure and to decrease lead absorption
• Refer the patient to the local health department for case management that should include a
detailed environmental investigation with lead hazard reduction and appropriate referrals for
support services.
• Check CBC and iron stores
• Check free EP or ZPP
• Neurodevelopmental monitoring
• Abdominal radiography (if particulate lead ingestion is suspected)

<table>
<thead>
<tr>
<th>≥ 70</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Obtain a confirmatory BLL immediately.</td>
</tr>
<tr>
<td>- Hospitalize the patient.</td>
</tr>
<tr>
<td>- The rest of the management should be as for children with BLL 45-69 µg/dL.</td>
</tr>
</tbody>
</table>

### D. Some Prevention Strategies to Decrease Lead Exposure

<table>
<thead>
<tr>
<th>Source</th>
<th>Prevention Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paint</td>
<td>Identify and abate</td>
</tr>
<tr>
<td>Dust</td>
<td>Wet mop, remove blinds</td>
</tr>
<tr>
<td>Soil</td>
<td>Restrict play in area, plant ground cover, wash hands frequently</td>
</tr>
<tr>
<td>Drinking Water</td>
<td>Flush cold water pipes by running the water until it becomes as cold as it will get</td>
</tr>
<tr>
<td>Parental occupations</td>
<td>Remove work clothing at work; wash work clothes separately</td>
</tr>
<tr>
<td>Hand-to-mouth activity (or pica)</td>
<td>Frequent hand washing; minimize food on floor</td>
</tr>
<tr>
<td>Inadequate nutrition</td>
<td>Adequate intake of calcium, iron, vitamin C</td>
</tr>
<tr>
<td>Some toys, crayons, folk remedies, old cookware</td>
<td>Avoid use</td>
</tr>
</tbody>
</table>

### Screening for Tuberculosis

**A Basic Risk-Assessment Questionnaire**

1) Was your child born outside the United States (Latin America, Africa, Asia)?
2) Has your child traveled outside the United States?
3) Has your child been exposed to anyone with TB?
4) Does your child have close contact with a person who has had a positive TB skin test result?
5) Does your child spend time with anyone who has been in jail or a shelter, uses illegal drugs, or has HIV?
6) Has your child drunk raw milk or eaten unpasteurized cheese?
7) Does your child have a household member who was born outside the U.S.?
8) Does your child have a household member who has traveled outside the U.S.?
Screening for Dyslipidemia (See Extra Credit “Expert Panel”)

A. Recommendations for Lipid Assessment (based on Table 9-5 from article)

<table>
<thead>
<tr>
<th>Age</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth- 2yrs</td>
<td>No lipid screening</td>
</tr>
<tr>
<td>2-8yrs</td>
<td>Selective screening with Fasting Lipid Profile (FLP) of children with close FamHx lipid disorders ± premature heart disease OR personal RFs for CVD (see Table C)</td>
</tr>
<tr>
<td>9-11yrs</td>
<td>Universal Screening x 1 with Non-fasting Lipid Profile (NLP) of Non HDL-C</td>
</tr>
<tr>
<td>12-16yrs</td>
<td>Selective screening with FLP if new knowledge of +FamHx or personal RFs</td>
</tr>
<tr>
<td>17-21yrs</td>
<td>Universal Screening x 1 with NLP of Non HDL-C</td>
</tr>
</tbody>
</table>

B. Lipid & Lipoprotein Concentration (mg/dL) Cutoffs (based on Table 9-1 from article)

<table>
<thead>
<tr>
<th>Category</th>
<th>Acceptable</th>
<th>Borderline</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>&lt;170</td>
<td>170-199</td>
<td>≥ 200</td>
</tr>
<tr>
<td>LDL-C</td>
<td>&lt;110 (0-9 years: &lt; 75)</td>
<td>110-129</td>
<td>≥ 130</td>
</tr>
<tr>
<td>Non HDL-C*</td>
<td>&lt;120</td>
<td>120-144</td>
<td>≥ 145</td>
</tr>
<tr>
<td>TG</td>
<td>&lt;75 (0-9 years: &lt; 75)</td>
<td>75-99</td>
<td>≥ 100</td>
</tr>
<tr>
<td>HDL-C</td>
<td>&gt;45</td>
<td>40-45</td>
<td>&lt;40</td>
</tr>
</tbody>
</table>

* Non HDL-C = TC- HDL-C

C. Risk Factors for Cardiovascular Disease (based on Tables 9-6, 9-7 from article)

**HIGH LEVEL RISK FACTORS & CONDITIONS**
- Parent/grandparent history of CV disease < 55 year in males, < 65 year in females:
  - coronary atherosclerosis, MI, peripheral vascular disease, or cerebrovascular disease
- BMI > 97th percentile (> 95th %ile still moderate risk)
- Diabetes Mellitus, type 1 or type 2
- Hypertension
- Current smoker
- Chronic renal disease/end-stage renal disease
- s/p any solid organ transplant
- History of Kawasaki’s disease with coronary aneurysms (regressed aneurysms moderate risk)

**MODERATE LEVEL RISK FACTORS & CONDITIONS**
- Pre-diabetes: impaired fasting glucose ≥100; impaired glucose tolerance 2hr
- Polycystic Ovarian Syndrome
- Chronic Inflammatory Disease (SLE or JRA)
- HIV Infection
- Nephrotic syndrome

**POTENTIAL RISK FACTORS & CONDITIONS**
- h/o cancer or congenital heart disease
- Passive smoker
- Unknown Family History
TB Elimination
Tuberculin Skin Testing

What is it?
The Mantoux tuberculin skin test (TST) is the standard method of determining whether a person is infected with *Mycobacterium tuberculosis*. Reliable administration and reading of the TST requires standardization of procedures, training, supervision, and practice.

How is the TST Administered?
The TST is performed by injecting 0.1 ml of tuberculin purified protein derivative (PPD) into the inner surface of the forearm. The injection should be made with a tuberculin syringe, with the needle bevel facing upward. The TST is an intradermal injection. When placed correctly, the injection should produce a pale elevation of the skin (a wheal) 6 to 10 mm in diameter.

How is the TST Read?
The skin test reaction should be read between 48 and 72 hours after administration. A patient who does not return within 72 hours will need to be rescheduled for another skin test.

The reaction should be measured in millimeters of the induration (palpable, raised, hardened area or swelling). The reader should not measure erythema (redness). The diameter of the indurated area should be measured across the forearm (perpendicular to the long axis).

How Are TST Reactions Interpreted?
Skin test interpretation depends on two factors:
- Measurement in millimeters of the induration
- Person’s risk of being infected with TB and of progression to disease if infected

Classification of the Tuberculin Skin Test Reaction

- **An induration of 5 or more millimeters** is considered positive in
  - HIV-infected persons
  - A recent contact of a person with TB disease
  - Persons with fibrotic changes on chest radiograph consistent with prior TB
  - Patients with organ transplants
  - Persons who are immunosuppressed for other reasons (e.g., taking the equivalent of >15 mg/day of prednisone for 1 month or longer, taking TNF-α antagonists)

- **An induration of 10 or more millimeters** is considered positive in
  - Recent immigrants (< 5 years) from high-prevalence countries
  - Injection drug users
  - Residents and employees of high-risk congregate settings
  - Mycobacteriology laboratory personnel
  - Persons with clinical conditions that place them at high risk
  - Children < 4 years of age
  - Infants, children, and adolescents exposed to adults in high-risk categories

- **An induration of 15 or more millimeters** is considered positive in any person, including persons with no known risk factors for TB. However, targeted skin testing programs should only be conducted among high-risk groups.

What Are False-Positive Reactions?
Some persons may react to the TST even though they are not infected with *M. tuberculosis*. The causes of these false-positive reactions may include, but are not limited to, the following:

- Infection with nontuberculosis mycobacteria
- Previous BCG vaccination
- Incorrect method of TST administration
- Incorrect interpretation of reaction
- Incorrect bottle of antigen used
What Are False-Negative Reactions?

Some persons may not react to the TST even though they are infected with *M. tuberculosis*. The reasons for these false-negative reactions may include, but are not limited to, the following:

- Cutaneous anergy (anergy is the inability to react to skin tests because of a weakened immune system)
- Recent TB infection (within 8-10 weeks of exposure)
- Very old TB infection (many years)
- Very young age (less than 6 months old)
- Recent live-virus vaccination (e.g., measles and smallpox)
- Overwhelming TB disease
- Some viral illnesses (e.g., measles and chicken pox)
- Incorrect method of TST administration
- Incorrect interpretation of reaction

Why is Two-Step Testing Conducted?

Two-step testing is useful for the initial skin testing of adults who are going to be retested periodically, such as health care workers or nursing home residents. This two-step approach can reduce the likelihood that a boosted reaction to a subsequent TST will be misinterpreted as a recent infection.

Can TSTs Be Given To Persons Receiving Vaccinations?

Vaccination with live viruses may interfere with TST reactions. For persons scheduled to receive a TST, testing should be done as follows:

- Either on the same day as vaccination with live-virus vaccine or 4-6 weeks after the administration of the live-virus vaccine
- At least one month after smallpox vaccination

Additional Information


http://www.cdc.gov/tb
Health Maintenance II Quiz

1. For each blood test, list the AAP recommendations for risk assessment and actual testing:
   a. H/H:
   b. Pb:
   c. TB:
   d. Lipids:

2. What are the TWO most common age-groups for nutritional iron deficiency and what is the etiology in each of these age groups?

3. Fill in the parameters (Normal, Increased, or Decreased) for the hematologic and biochemical markers for iron deficiency anemia. Which marker is the first to be abnormal?
   - Hemoglobin ____________________________
   - MCV ____________________________
   - RDW ____________________________
   - Reticulocytes ____________________________
   - Serum Ferritin ____________________________
   - Serum iron ____________________________
   - TIBC ____________________________
   - TFN saturation ____________________________

4. Match the action with the blood lead levels (more than one correct answer):
   A. < 10 µg/dL  1. Referral to local health department: _________________
   B. 10-14 µg/dL  2. Immediate hospitalization: ________________________
   C. 15-19 µg/dL  3. Chelation indicated: _____________________________
   D. 20-45 µg/dL  4. Detailed environmental history: ____________________
   E. 45-69 µg/dL
   F. > 70 µg/dL

5. Match the following populations with the criteria for positive TST:
   a. Child with no risk factors: __________________________
   b. Healthy child < 4 years: ____________________________
   c. Dr. Jones patient on high-dose steroids: ________________

6. Flashforward: How can you code for these screening tests? Which garners the most RVUs?
   a. Newborn Screen: _______________________________________
   b. Hgb/Hct: ______________________________________________
   c. Tuberculin Skin Test: ___________________________________
Health Maintenance II Mini-Cases

Case 1: You are seeing the 3 year old son of an Egyptian military attaché for a well child visit. What screening would you perform? Consider the entire periodicity schedule.

Case 2: A 12 month old child has a blood lead level of 14 µg/dL that is 14 µg/dL on follow-up testing. Name 4 specific steps the parents can take to decrease lead exposure in their child.

Case 3: A mother asks about screening her 18 month old and 5 year boys for “cholesterol” after their 38 year old father undergoes a coronary catheterization for angina. What do you tell her and what screening will you order?

Bonus: What is the rationale for universal screening at 9-11yrs?
Case 4: You screen a 12mo with PMHx of Hemoglobin C trait on his newborn screen and obtain the CBC below. He was recently weaned from breast milk to whole milk, and mother reports that he takes 32-40oz per day. He still prefers Gerber purees—mostly fruits and vegetables—to table foods. Lead screening was normal. **What is your working diagnosis? What additional testing do you want, if any? What interventions, if any will you take today?**

<table>
<thead>
<tr>
<th>CBC W/Diff</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>10.3</td>
</tr>
<tr>
<td>RBC</td>
<td>4.85</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>9.6 (L)</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>29.0 (L)</td>
</tr>
<tr>
<td>MCV</td>
<td>59.9 (L)</td>
</tr>
<tr>
<td>MCH</td>
<td>19.9 (L)</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.1</td>
</tr>
<tr>
<td>RDW CV</td>
<td>25.0 (H)</td>
</tr>
<tr>
<td>Platelets</td>
<td>730 (H)</td>
</tr>
<tr>
<td>MPV</td>
<td>7.3</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>32.4 (L)</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>53.9 (H)</td>
</tr>
<tr>
<td>Monocytes</td>
<td>7.2</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>6.4</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.1 (L)</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>3.4</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>5.6 (H)</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.7</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.7 (H)</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differential Manual</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eosinophils</td>
<td>6 (H)</td>
</tr>
<tr>
<td>Basophils</td>
<td>1</td>
</tr>
<tr>
<td>Lymphocytes Atypical</td>
<td>2</td>
</tr>
<tr>
<td>Anisocytosis</td>
<td>2+</td>
</tr>
<tr>
<td>Poikilocytosis</td>
<td>1+</td>
</tr>
<tr>
<td>Microcytes</td>
<td>3+</td>
</tr>
<tr>
<td>Hypochromia</td>
<td>2+</td>
</tr>
<tr>
<td>Target Cells</td>
<td>1+</td>
</tr>
<tr>
<td>Schistocytes</td>
<td>RARE</td>
</tr>
<tr>
<td>Neutrophils Segmented</td>
<td>33</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>53</td>
</tr>
<tr>
<td>Monocytes</td>
<td>5</td>
</tr>
</tbody>
</table>
1. You are seeing a 1-year-old patient in your clinic for a health supervision visit. You explain the recommended screening tests for this visit to the medical student who accompanies you.

**Of the following, the MOST appropriate recommended screening test at this visit is:**
A. blood lead concentration by fingerstick  
B. blood lead concentration by venipuncture  
C. complete blood count with differential count  
D. serum ferritin  
E. serum iron

2. A medical student notes on rounds that a 2-year-old girl admitted for pneumonia has a complete blood count (CBC) that includes a hematocrit of 35% (0.35), hemoglobin of 11.5 g/dL (115.0 g/L), mean corpuscular volume of 68.0 fl, and platelet and white blood cell counts that are normal for age. During the bedside encounter with the child’s mother, you advise her to start the child on a multivitamin with iron and have her primary care physician obtain another CBC in a month or so. The medical student asks why you recommended iron supplementation when the child has a normal hematocrit.

**Of the following, the BEST reason to prescribe supplemental iron therapy for this child at this time is to prevent**
A. diminished cognitive abilities  
B. fatigue  
C. rapid progression to anemia  
D. recurrent infections  
E. short stature

3. As you are leaving the supermarket, the cashier tells you that she is worried because her child recently had a positive tuberculin skin test. She had to take him to the health department for skin testing because he had been in contact with her father, who recently was diagnosed with active pulmonary tuberculosis. They told her that the boy’s skin test was positive at "25," but his chest radiograph was normal. She is concerned because the doctor told her that the case is a little unusual because of the type of tuberculosis her father has. She asked the physician at the health department to write it down, and she hands you a paper that says "INH resistant." The mother asks you what type of medication her boy should receive.

**Of the following, the MOST appropriate antituberculous agent to prescribe for this boy is**
A. ciprofloxacin  
B. ethambutol  
C. isoniazid  
D. pyrazinamide  
E. rifampin

4. A family has just relocated to your community, and you are evaluating their 12-year-old son for the first time this afternoon. Family history reveals that the boy’s father and grandmother had premature cardiovascular disease. The boy’s parents are concerned about his risk of heart disease.

**Of the following, the MOST important next step in this child’s evaluation is:**
A. Echocardiography  
B. Electrocardiography  
C. Fasting lipoprotein analysis  
D. Random cholesterol measurement  
E. Referral to the cardiology clinic