Test Definition: MET
Methemoglobin and Sulfhemoglobin, B

Overview

Useful For
Diagnosing methemoglobinemia and sulfhemoglobinemia
Identifying cyanosis due to other causes, such as congenital heart disease

Profile Information

<table>
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<tr>
<th>Test ID</th>
<th>Reporting Name</th>
<th>Available Separately</th>
<th>Always Performed</th>
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<tr>
<td>METH</td>
<td>Methemoglobin, B</td>
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<tr>
<td>SULF</td>
<td>Sulfhemoglobin, B</td>
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Method Name
Spectrophotometry (SP)

NY State Available
Yes

Specimen

Specimen Type
Whole Blood EDTA

Specimen Required
Specimen must arrive within 72 hours of draw.

Container/Tube: Lavender top (EDTA)

Specimen Volume: Full tube

Additional Information: Patient's age is required.

Forms
If not ordering electronically, complete, print, and send a Benign Hematology Test Request Form (T755) with the specimen.

Specimen Minimum Volume
1 mL

Reject Due To

| Gross hemolysis | Reject |

Specimen Stability Information
Clinical and Interpretive

Clinical Information

Methemoglobin:

When iron in hemoglobin is oxidized from the normal divalent state to a trivalent state, the resulting brownish pigment is methemoglobin. Methemoglobin cannot combine reversibly with oxygen and is associated with cyanosis.

Methemoglobinemia, with or without sulfhemoglobinemia, is most commonly encountered as a result of administration of medications such as phenacetin, phenazopyridine, sulfonamides, local anesthetics, dapsone, or following ingestion of nitrates or nitrites. Congenital methemoglobinemias are rare. They are either due to:

-Deficiency of methemoglobin reductase (also called cytochrome B5 reductase or diaphorase) in erythrocytes, an autosomal recessive disorder.

-One of several intrinsic structural disorders of hemoglobin, called methemoglobin-M, all of which are inherited in the autosomal dominant mode.

Methemoglobinemia responds to treatment with methylene blue or ascorbic acid.

Sulfhemoglobin:

Sulfhemoglobin cannot combine with oxygen. Sulfhemoglobinemia is associated with cyanosis and often accompanies drug-induced methemoglobinemia. Sulfhemoglobinemia can be due to exposure to trinitrotoluene or zinc ethylene bisdithiocarbamate (a fungicide), or by ingestion of therapeutic doses of flutamide.

In contrast to methemoglobinemia, sulfhemoglobinemia persists until the erythrocytes containing it are destroyed. Therefore, blood level of sulfhemoglobin declines gradually over a period of weeks.

Patients with sulfhemoglobinemia often also have methemoglobinemia. There is no specific treatment for sulfhemoglobinemia. Therapy is directed at reversing the methemoglobinemia, if present.

Reference Values

METHEMOGLOBIN

0-11 months: not established

> or =1 year: 0.0-1.5% of total hemoglobin

SULFHEMOGLOBIN

0-11 months: not established

> or =1 year: 0.0-0.4% of total hemoglobin
Interpretation

In congenital methemoglobinemia, the methemoglobinemia concentration in blood is about 15% to 20% of total hemoglobin. Such patients are mildly cyanotic and asymptomatic.

In acquired (toxic) methemoglobinemia, the concentration may be much higher. Symptoms may be severe when methemoglobin is >40% of hemoglobin. Very high concentrations (>70%) may be fatal.

Cautions

Methemoglobin is unstable and is reduced to hemoglobin at a rate of about 40% per day at 0 to 4 degrees C.

A normal methemoglobin value obtained with stored or shipped specimens does not exclude prior mild methemoglobinemia. However, significant methemoglobinemia will still be demonstrable.

Sulfhemoglobin is stable and does not change in stored or shipped specimens.

Clinical Reference


Performance

Method Description

Methemoglobin:

The normal absorption spectrum of oxyhemoglobin has very little optical density above 600 nm. The absorption spectrum of methemoglobin exhibits a small, characteristic peak at 630 nm. This peak is abolished as methemoglobin is converted to cyanmethemoglobin upon addition of potassium cyanide, and the drop in optical density is proportional to methemoglobin concentration.

Sulfhemoglobin:

The normal absorption spectrum of oxyhemoglobin has very little optical density above 600 nm. However, if certain poorly defined hemoglobin denaturation products are present in a hemolysate, there is a broad elevation of the absorption curve in the range of 600 nm to 620 nm. This "sulfhemoglobin" plateau is not affected by treatment with cyanide. Sulfhemoglobin is not available, nor can it be prepared, in a pure form for preparation of a sulfhemoglobin standard. In calculating sulfhemoglobin concentration, the factor for sulfhemoglobin quantitation is based on studies of Carrico, et al (1978). (Evelyn KA, Malloy HT: Microdetermination of oxyhemoglobin, methemoglobin, and sulfhemoglobin in a single sample of blood. J Biol Chem 1938;126:655-662; Carrico RJ, Peisach J, Alben JO: The preparation and some physical properties of sulfhemoglobin. J Analyt Biochem 1978;253:2386-2391; Fairbanks VF, Klee GG: Biochemical aspects of hematology. In Teitz Textbook of Clinical Chemistry. Edited by CA Burtis, ER Ashwood, WB Saunders Company, 1999, pp 1676-1678)

PDF Report

No

Day(s) and Time(s) Test Performed

Monday through Saturday; Continuously

Analytic Time

Same day/1 day
Test Definition: MET
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Maximum Laboratory Time
3 days

Specimen Retention Time
7 days

Performing Laboratory Location
Rochester

Fees and Codes

Fees
- Authorized users can sign in to Test Prices for detailed fee information.
- Clients without access to Test Prices can contact Customer Service 24 hours a day, seven days a week.
- Prospective clients should contact their Regional Manager. For assistance, contact Customer Service.

Test Classification
See Individual Test IDs

CPT Code Information
83050-Methemoglobin
83060-Sulfhemoglobin

LOINC® Information

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<tr>
<th>Test ID</th>
<th>Test Order Name</th>
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