Clinical

Isoniazid (INH) is a mainstay of treatment for tuberculosis. However, some strains of *Mycobacterium tuberculosis* may be resistant to INH and are candidates for alternative antimycobacterial agents.

The National Committee on Clinical Laboratory Standards (NCCLS) defines critical concentrations of antimycobacterial agents to be tested in order to permit standardized interpretation of *Mycobacterium tuberculosis* complex susceptibility test results. When low-level resistance to isoniazid (0.1 µg/mL using the BACTEC MGIT 960) is detected, NCCLS recommends additional testing of isoniazid at a higher concentration (0.4 µg/mL using the BACTEC MGIT 960).

Useful For

Second-tier susceptibility testing of *Mycobacterium tuberculosis* complex isolates that demonstrate resistance to INH at 0.1 µg/mL.

Interpretation

- *Mycobacterium tuberculosis* complex isolates are reported as susceptible or resistant to INH at 0.4 µg/mL.
- Some experts believe that patients infected with strains exhibiting resistance to low levels of INH (0.1 µg/mL), but not exhibiting resistance at 0.4 µg/mL, may benefit from continuing therapy with INH. A specialist in the treatment of tuberculosis should be consulted concerning the appropriate therapeutic regimen and dosages.

References


Method

The BACTEC MGIT 960 instrument is a commercial incubation system cleared by the US Food and Drug Administration (FDA) for susceptibility testing of *Mycobacterium tuberculosis* complex. The MGIT 960 performs rapid, qualitative susceptibility testing of *Mycobacterium tuberculosis* complex isolates growing in pure culture for drugs at specified concentrations. The method is based on the production and measurement of fluorescence within a Mycobacterial Growth Indicator Tube (MGIT) in the presence of actively growing *Mycobacterium tuberculosis* complex isolates. Low or undetectable levels of fluorescence in the presence of critical drug concentrations suggest a lack of *Mycobacterium tuberculosis* growth and susceptibility to the drug at the tested concentration. Increased fluorescence suggests active growth of *Mycobacterium tuberculosis* and resistance to the drug at the tested concentration. (NCCLS: Susceptibility Testing of Mycobacteria, Nocardiae, and Other Aerobic Actinomycetes; Approved Standard. NCCLS document M24-A [ISBN 1-56238-550-3]. NCCLS, Wayne, PA, 2003)
**Test Title:** Antimicrobial Susceptibility, *Mycobacterium tuberculosis* Complex, High Concentration Isoniazid #83676

**Specimen Required:** Send *Mycobacterium tuberculosis* complex isolate on Middlebrook 7-H10 agar slant or other appropriate media. Organism must be in pure culture, actively growing, placed in a proper mailing container (agar plates are not acceptable), and labeled as an etiologic agent. See “Antimicrobial Susceptibility Tests” and “Infectious Material” in Special Instructions.

**Note:**
1. **Specimen source and organism identification is required** on request form for processing.
2. If ordering electronically, please complete and submit a “MayoConnect Additional Test Information Form” (Supply T357 or see Special Instructions) with the specimen. If not ordering electronically, please complete and submit a “Microbiology Request Form” (Supply T244) with the specimen.

**Reference Values:** Results are reported as susceptible or resistant.

**Analytic Time:** 3-35 days

**Days Set Up:** Monday through Saturday; a.m.

**CPT Code:**
- 87188
- 87116/Culture (if appropriate)
- 87118/Identification (if appropriate)
- 87158/Identification other methods (if appropriate)
- 87149/Identification by nucleic acid probe (if appropriate)
- 87176/Tissue processing (if appropriate)
Antimicrobial Susceptibility, *Mycobacterium tuberculosis* Complex, High Concentration Streptomycin

**Clinical**

Streptomycin is one of a group of agents used to treat tuberculosis. However, some strains of *Mycobacterium tuberculosis* may be resistant to streptomycin and are candidates for alternative antitubercular agents.

The National Committee on Clinical Laboratory Standards (NCCLS) defines critical concentrations of antitubercular agents to be tested in order to permit standardized interpretation of *Mycobacterium tuberculosis* complex susceptibility test results. When resistance to low levels of streptomycin (1.0 µg/mL using the BACTEC MGIT 960) is detected, NCCLS recommends additional testing of streptomycin at a higher concentration (4.0 µg/mL using the BACTEC MGIT 960).

**Useful For**

Second-tier susceptibility testing of *Mycobacterium tuberculosis* complex isolates that demonstrate resistance to streptomycin at 1.0 µg/mL.

**Interpretation**

- *Mycobacterium tuberculosis* complex isolates are reported as susceptible or resistant to streptomycin at 4.0 µg/mL.
- Some experts believe that patients infected with strains exhibiting resistance to low levels of streptomycin (1.0 µg/mL), but not exhibiting resistance at 4.0 µg/mL, may benefit from continuing therapy with streptomycin. A specialist in the treatment of tuberculosis should be consulted concerning the appropriate therapeutic regimen and dosages.

**References**


**Method**

The BACTEC MGIT 960 instrument is a commercial incubation system cleared by the US Food and Drug Administration (FDA) for susceptibility testing of *Mycobacterium tuberculosis* complex. The MGIT 960 performs rapid, qualitative susceptibility testing of *Mycobacterium tuberculosis* complex isolates growing in pure culture for the drugs at critical concentrations. The method is based on the production and measurement of fluorescence within a Mycobacterial Growth Indicator Tube (MGIT) in the presence of actively growing *Mycobacterium tuberculosis* complex isolates. Low or undetectable levels of fluorescence in the presence of critical drug concentrations suggest a lack of *Mycobacterium tuberculosis* growth and susceptibility to the drug at the tested concentration. Increased fluorescence suggests active growth of *Mycobacterium tuberculosis* and resistance to the drug at the tested concentration. (NCCLS: Susceptibility Testing of Myobacteria, Nocardiae, and other Aerobic Actinomycetes; Approved Standard. NCCLS document M24-A [ISBN 1-56238-550-3]. NCCLS, Wayne, PA, 2003)
Specimen Required: Send *Mycobacterium tuberculosis* complex isolate on Middlebrook 7-H10 agar slant or other appropriate media. Organism must be in pure culture, actively growing, placed in a proper mailing container (agar plates are not acceptable), and labeled as an etiologic agent. See “Antimicrobial Susceptibility Tests” and “Infectious Material” in Special Instructions.

Note: 1. Specimen source and organism identification is required on request form for processing.
2. If ordering electronically, please complete and submit a “MayoConnect Additional Test Information Form” (Supply T357 or see Special Instructions) with the specimen. If not ordering electronically, please complete and submit a “Microbiology Request Form” (Supply T244) with the specimen.

Reference Values: Results are reported as susceptible or resistant.

Analytic Time: 3-35 days

Days Set Up: Monday through Saturday; a.m.

CPT Code:

87188
87116/Culture (if appropriate)
87118/Identification (if appropriate)
87158/Identification other methods (if appropriate)
87149/Identification by nucleic acid probe (if appropriate)
87176/Tissue processing (if appropriate)


**Arbovirus Antibody Panel IgG and IgM, Serum #83267**

### Profile Information

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<tr>
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<td>West Equine Enceph Ab, IgM, S</td>
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### Clinical

**California (LaCrosse) virus:**

California (LaCrosse) virus is a member of bunyaviridae and is one of the arthropod-borne encephalitides. It is transmitted by various *Aedes* and *Culex* mosquitoes and is found in such intermediate hosts as the rabbit, squirrel, chipmunk, and field mouse. California meningoencephalitis is usually mild and occurs in late summer. Ninety percent of infections are seen in children less than 15 years of age, usually from rural areas. The incubation period is estimated to be 7 days and acute illness lasts 10 days or less in most instances. Typically, the first symptoms are nonspecific, last 1-3 days, and are followed by the appearance of central nervous system (CNS) signs and symptoms such as stiff neck, lethargy, and seizures, which usually abate within 1 week. Symptomatic infection is almost never recognized in those over 18 years old. The most important sequelae of California virus encephalitis is epilepsy, which occurs in about 10% of children; almost always in patients who have had seizures during the acute illness. A few patients (estimated 2%) have persistent paresis. Learning disabilities or other objective cognitive deficits have been reported in a small proportion (no more than 2%) of patients. Learning performance and behavior of most recovered patients are not distinguishable from comparison groups in these same areas.

**Eastern Equine Encephalitis:**

Eastern equine encephalitis (EEE) is within the alphavirus group. It is a low prevalence cause of human disease in the eastern and Gulf Coast states. EEE is maintained by a cycle of mosquito/wild bird transmission, peaking in the summer and early fall, when man may become an adventitious host. The most common clinically apparent manifestation is a mild undifferentiated febrile illness, usually with headache. CNS involvement is demonstrated in only a minority of infected individuals, it is more abrupt and more severe than with other arboviruses, with children being more susceptible to severe disease. Fatality rates are approximately 70%.

**St. Louis Encephalitis:**

Areas of outbreaks of St. Louis encephalitis since 1933 have involved the western United States, Texas, the Ohio-Mississippi Valley, and Florida. The vector of transmission is the mosquito. Peak incidence occurs in summer and early autumn. Disease onset is characterized by generalized malaise, fever, chills, headache, drowsiness, nausea, and sore throat or cough followed in 1-4 days by meningeal and neurologic signs. The severity of illness increases with advancing age; persons over 60 years have the highest frequency of encephalitis. Symptoms of irritability, sleeplessness, depression, memory loss, and headaches can last up to 3 years.

*Continued on next page.*
Arbovirus Antibody Panel IgG and IgM, Serum

Test Title: Arbovirus Antibody Panel IgG and IgM, Serum #83267

Clinical (continued)

Western Equine Encephalitis:
The virus that causes western equine encephalitis (WEE) is widely distributed throughout the United States and Canada; disease occurs almost exclusively in the western states and Canadian provinces. The relative absence of the disease in the eastern United States probably reflects a paucity of the vector mosquito species, *Culex tarsalis*, and possibly a lower pathogenicity of local virus strains. The disease usually begins suddenly with malaise, fever, and headache, often with nausea and vomiting. Vertigo, photophobia, sore throat, respiratory symptoms, abdominal pain, and myalgia are also common. Over a few days, the headache intensifies; drowsiness and restlessness may merge into a coma in severe cases. In infants and children, the onset may be more abrupt than for adults. WEE should be suspected in any case of febrile CNS disease from an endemic area. Infants are highly susceptible to CNS disease and about 20% of cases are under 1 year of age. There is an excess of males with WEE clinical encephalitis, averaging about twice the number of infections detected in females. After recovery from the acute disease, patients may require from several months to 2 years to overcome the fatigue, headache, and irritability. Infants and children are at higher risk of permanent brain damage after recovery than adults.

Infections with arboviruses can occur at any age. The age distribution depends on the degree of exposure to the particular transmitting arthropod relating to age, sex, and occupational, vocational and recreational habits of the individuals. Once humans have been infected, the severity of the host response may be influenced by age. WEE tends to produce the most severe clinical infections in young persons and St. Louis encephalitis in older persons. Serious California (LaCrosse) virus infections primarily involve children, especially boys. Adult males exposed to California viruses have high prevalence rates of antibody but usually show no serious illness. Infection among males is primarily due to working conditions and sports activities taking place where the vector is present.

Useful For Aiding the diagnosis of arboviral (California [LaCrosse], St. Louis, eastern equine, and western equine virus) encephalitis

Interpretation

- In patients infected with these or related viruses, IgG antibody is generally detectable with in 1-3 weeks of onset, peaking within 1 to 2 months, and declining slowly thereafter.
- IgM class antibody is also reliably detected within 1-3 weeks of onset, peaking and rapidly declining within 3 months.
- A single serum specimen IgG ≥1:10 indicates exposure to the virus.
- Results from a single serum specimen can differentiate early (acute) infection from past infection with immunity if IgM is positive (suggests acute infection).
- A 4-fold or greater rise in IgG antibody titer in acute and convalescent sera indicate recent infection.
- In the United States, it is unusual for any patient to show positive reactions to more than 1 of the arboviral antigens, although WEE and EEE antigens will show a noticeable cross-reactivity.

Cautions

- All results must be correlated with clinical history and other data available to the attending physician.
- Specimens drawn within the first 2 weeks after onset are variably negative for IgG antibody and should not be used to exclude the diagnosis of arboviral disease. If arboviral infection is suspected, a second specimen should be obtained and tested 10-21 days later.
- Since cross-reactivity with dengue fever virus does occur with St. Louis encephalitis antigen and, therefore, cannot be differentiated further. The specific virus responsible for such a titer may be deduced by the travel history of the patient, along with available medical and epidemiological data, unless the virus can be isolated.
- EEE and WEE viruses show some cross-reactivity; however, antibody response to the infecting virus is typically at least 8-fold higher.
Test Title: Arbovirus Antibody Panel IgG and IgM, Serum
#83267

References

Method
Indirect immunofluorescence.


Specimen Required: Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 0.5 mL of serum refrigerated in a screw-capped, round-bottom, plastic vial. Forward promptly.

Reference Values:
IgG: <1:10
IgM: <1:10
See “Virology” in Special Instructions for additional interpretive information.

Analytic Time: Same day/1 day
Days Set Up: Monday through Friday; 9 a.m.
CPT Code:
86651/x2 CaliVirus (La Crosse) Ab, IgG & IgM
86652/x2 East Equine Enceph Ab, IgG & IgM
86653/x2 St. Louis Enceph Ab, IgG & IgM
86654/x2 West Equine Enceph Ab, IgG & IgM
California Virus (LaCrosse) IgG and IgM, Serum #83153

Profile Information

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Clinical

California virus (LaCrosse) is a member of Bunyaviridae and is one of the arthropod-borne encephalitides. It is transmitted by various *Aedes* and *Culex* mosquitoes and is found in such intermediate hosts as the rabbit, chipmunk, and field mouse. California meningoencephalitis is usually mild and occurs in late summer. Ninety percent of infections are seen in children under 15 years of age, usually from rural areas. Incubation period is estimated to be 7 days and acute illness lasts 10 days or less in most instances. Typically, the first symptoms are nonspecific, last 1-3 days, and are followed by the appearance of central nervous system signs and symptoms such as stiff neck, lethargy, and seizures, which usually abate within 1 week. Symptomatic infection is almost never recognized in those over 18 years old. The most important sequelae of California virus encephalitis is epilepsy, which occurs in about 10% of children; almost always in patients who have had seizures during the acute illness. A few patients (estimated 2%) have persistent paresis. Learning disabilities or other objective cognitive deficits have been reported in a small proportion (no more than 2%) of patients. Learning performance and behavior of most recovered patients are not distinguishable from comparison groups in these same areas.

Useful For

Aiding the diagnosis of California virus (LaCrosse)

Interpretation

- In patients infected with these or related viruses, IgG antibody is generally detectable within 1-3 weeks of onset, peaking within 1-2 months, and declining slowly thereafter.
- IgM class antibody is also reliably detected within 1-3 weeks of onset, peaking and rapidly declining within 3 months.
- Single serum specimen IgG ≥1:10 indicates exposure to the virus.
- Results from a single serum specimen can differentiate early (acute) infection from past infection with immunity if IgM is positive (suggests acute infection).
- A 4-fold or greater rise in IgG antibody titer in acute and convalescent sera indicate recent infection.
- Infections with arboviruses can occur at any age. The age distribution depends on the degree of exposure to the particular transmitting arthropod relating to age and sex, as well as the occupational, vocational, and recreational habits of the individuals. Once humans have been infected, the severity of the host response may be influenced by age: serious LaCrosse infections primarily involve children, especially boys. Adult males exposed to LaCrosse have high prevalence rates of antibody but usually show no serious illness. Infection among males is primarily due to working conditions and sports activity taking place where the vector is present.

Cautions

- All results must be correlated with clinical history and other data available to the attending physician.
- Samples drawn within the first 2 weeks after onset are variably negative for IgG antibody and should not be used to exclude the diagnosis of arboviral disease. If arboviral infection is suspected, a second sample should be obtained and tested 10-21 days later.
- Since cross-reactivity with dengue fever virus does occur with St. Louis encephalitis antigen and therefore, cannot be differentiated further. The specific virus responsible for such a titer may be deduced by the travel history of the patient, along with available medical and epidemiological data, unless the virus can be isolated.
- Usually, when an infection with an arbovirus is suspected, it is too late to isolate the virus or obtain serum specimens to detect a rise of antibody titer.
Test Title: California Virus (LaCrosse) IgG and IgM, Serum #83153

References

Method
Indirect immunofluorescence.


Specimen Required: Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 0.5 mL of serum refrigerated in a screw-capped, round-bottom, plastic vial. Forward promptly.

Reference Values: IgG: <1:10
IgM: <1:10
See “Virology” in Special Instructions for additional interpretive information.

Analytic Time: Same day/1 day
Days Set Up: Monday through Friday; 9 a.m.
CPT Code: 86651/x2
Eastern Equine Encephalitis Antibody, IgG and IgM, Serum #83155

Clinical
Eastern equine encephalitis (EEE) is within the Alphavirus group. It is a low-prevalence cause of human disease in the eastern and Gulf Coast states. EEE is maintained by a cycle of mosquito/wild bird transmission, peaking in the summer and early fall, when man may become an adventitious host. The most common clinically apparent manifestation is a mild undifferentiated febrile illness, usually with headache. Central nervous system involvement is demonstrated in only a minority of infected individuals; it is more abrupt and more severe with EEE than other arboviruses, with children being more susceptible to severe disease. Fatality rates are approximately 70% for EEE.

Useful For
Aiding in the diagnosis of eastern equine encephalitis

Interpretation
- In patients infected with this virus, IgG antibody is generally detectable within 1-3 weeks of onset, peaking within 1-2 months, and declining slowly thereafter.
- IgM class antibody is also reliably detected within 1-3 weeks of onset, peaking and rapidly declining within 3 months.
- Single serum specimen IgG ≥1:10 indicates exposure to the virus.
- Results from a single serum specimen can differentiate early (acute) infection from past infection with immunity if IgM is positive (suggests acute infection).
- A 4-fold or greater rise in IgG antibody titer in acute and convalescent sera indicate recent infection.
- In the United States it is unusual for any patient to show positive reactions to more than 1 of the arboviral antigens, although western equine encephalitis (WEE) and EEE antigens will show a noticeable cross-reactivity.
- Infections can occur at any age. The age distribution depends on the degree of exposure to the particular transmitting arthropod relating to age and sex, as well as the occupational, vocational, and recreational habits of the individuals. Once humans have been infected, the severity of the host response may be influenced by age. Infection among males is primarily due to work conditions and sports activity taking place where the vector is present.

Cautions
- All results must be correlated with clinical history and other data available to the attending physician.
- Samples drawn within the first 2 weeks after onset are variably negative for IgG antibody and should not be used to exclude the diagnosis of arboviral disease. If arboviral infection is suspected, a second sample should be obtained and tested 10-21 days later.
- Since cross-reactivity with dengue fever virus does occur with St. Louis encephalitis antigen and therefore, cannot be differentiated further. The specific virus responsible for such a titer may be deduced by the travel history of the patient, along with available medical and epidemiological data, unless the virus can be isolated.
- EEE and WEE viruses show some cross-reactivity; however, antibody response to the infecting virus is typically at least 8-fold higher.
- Usually, when an infection with an arbovirus is suspected, it is too late to isolate the virus or obtain serum specimens to detect a rise of antibody titer.
Test Title: Eastern Equine Encephalitis Antibody, IgG and IgM, Serum
#83155

References

Method
Indirect immunofluorescence.


Specimen Required: Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 0.5 mL of serum refrigerated in a screw-capped, round-bottom, plastic vial. Forward promptly.

Reference Values:
IgG: <1:10
IgM: <1:10
See "Virology" in Special Instructions for additional interpretive information.

Analytic Time: Same day/1 day
Days Set Up: Monday through Friday; 9 a.m.
CPT Code: 86652/x2
**Helicobacter pylori Antibody, IgG, IgM, and IgA, Serum #84407**

With the introduction of this profile, MML has also introduced #84408 Helicobacter pylori Antibodies, IgM, Serum and #84409 Helicobacter pylori Antibodies, IgA, Serum (see MayoAccess for information about these tests). Additionally, #80668 Helicobacter pylori Antibodies, IgG, Serum now reports quantitative results. The new reference values for #80668 (IgG) are as follows:

- Index <0.75 (negative)
- Index 0.75-0.99 (equivocal)
- Index ≥1.00 (positive)

### Clinical

*Helicobacter pylori* is a spiral-shaped, gram-negative bacillus that has been associated with gastritis, gastric and duodenal ulcers, and gastric malignancies.

*Helicobacter pylori* is found worldwide. In Caucasian populations in the United States and other industrialized countries, *Helicobacter pylori* infection is infrequent in childhood. Prevalence increases 0.5-2% with each year of age, reaching about 50% in those 60 or older. Prevalence rates appear to be higher in blacks and Hispanics than in whites. In a random population of 200 apparently healthy blood donors tested for *Helicobacter pylori* IgG antibody, the positive rate was 27.5% with an equivocal rate of 5.5%.

The diagnosis of *Helicobacter pylori* gastrointestinal disease is supported by the presence of serum antibodies IgG, IgM, or IgA to the organism. Screening patients for the presence of antibodies to *Helicobacter pylori* is a convenient, noninvasive means for assessing whether gastrointestinal symptoms may be related to *Helicobacter pylori* infection.

Because serology may lack specificity, additional noninvasive tests can be used to confirm *Helicobacter pylori* infection including the urease breath test (#81590 “Helicobacter pylori Breath Test”) or stool antigen test for *Helicobacter pylori* (#81806 “Helicobacter Pylori Antigen, Feces”). The gold standard for diagnosis of *Helicobacter pylori* disease is a biopsy of infected tissue and evaluating the tissue by Gram, silver, Giemsa, or acridine orange stains, or by immunofluorescence or immunoperoxidase methods, rapid urease testing, and/or culture.

### Usefor

Screening for *Helicobacter pylori* infection

### Interpretation

Patients with *Helicobacter pylori* infection nearly always develop antibodies of the IgG class and less frequently develop antibodies of the IgA class. IgM antibodies may be produced shortly after the onset of infection. Levels of IgM antibodies should decrease after successful treatment, but may again increase if recurrence or relapse of infection occurs.

### Cautions

- These assays should be performed only on patients with gastrointestinal symptoms because of the large percentage of *Helicobacter pylori*-colonized individuals, especially in older age groups (estimated to be 40-60% of asymptomatic Caucasians older than 60 years).
- The serologic results must be interpreted in light of the clinical signs and symptoms of the patient.
**Test Title:** Helicobacter pylori Antibody, IgG, IgM, and IgA, Serum

**#84407**

**References**

**Method**

**IgG:**
The VIDAS HPY assay is an enzyme-linked fluorescent immunoassay (ELFA) performed in an automated VIDAS instrument. All assay steps and temperatures are controlled by the instrument. A pipette tip like disposable device, the Solid Phase Receptacle (SPR), serves as the solid phase as well as a pipettor for the assay. Reagents for the assay are in the sealed HPY Reagent Strips. After preliminary wash and specimen dilution steps, the specimen is cycled in and out of the SPR for a specified time. IgG antibodies to *Helicobacter pylori* present in the specimen will bind to the *Helicobacter pylori* antigen coating the interior of the SPR. The conjugated enzyme catalyzes the hydrolysis of this substance into a fluorescent product (4-methylumbelliferone); the fluorescence is measured at 450 nm. The optical scanner in the instrument measures the intensity of fluorescence. When the VIDAS HPY assay is completed, the results are analyzed automatically by the computer, a test value is generated, and a report is printed for each specimen. (Cockerill FR, Jones MF, Beito EB, et al: An evaluation of the VIDAS *Helicobacter pylori* IgG assay. Abstr Gen Meet Am Soc Microbiol 2002 May:20-23)

**IgM and IgA:**
Partially purified *Helicobacter pylori* antigens are immobilized on the wells of a microwell plate. Diluted serum is added to the wells. IgM or IgA antibodies, specific to *Helicobacter pylori*, if present, bind to the antigen on the microwells. Excess antibodies are washed away with buffer. Antihuman IgM or IgA enzyme conjugate is added to the wells. The conjugate binds to the antigen-antibody complex on the plate. The excess enzyme conjugate is washed away and color is developed by the addition of an enzyme substrate. The intensity of the color corresponds directly to the amount of antibody present. (Package insert: GAP[R]-IgM, BIOAMERICA, INC., Newport Beach, CA, 2001)

**Specimen Required:** Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 0.6 mL of serum refrigerated in a screw-capped, round-bottom, plastic vial. Forward promptly.

**Reference Values:**

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<td>IgM</td>
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**Analytic Time:** Same day/1 day

**Days Set Up:** Monday through Saturday; 10 a.m.

**CPT Code:** 86677/x3
New Test ANNOUNCEMENT
A Mayo Reference Services Publication

Hepatitis B Surface Antigen (HBsAg) for Cadaveric or Hemolyzed Specimens, Serum #83626

Profile Information

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Reflex Algorithm

All positive HBsAg enzyme immunoassay (EIA) results are confirmed by HBsAg neutralization at an additional charge.

Clinical

Hepatitis B virus (HBV) is endemic throughout the world. The infection is spreading primarily through percutaneous contact with infected blood products (e.g., blood transfusion, sharing of needles by intravenous drug addicts). The virus is also found in various human body fluids, and it is known to be spread through oral and genital contacts. HBV can be transmitted from mother to child during delivery through contact with blood and vaginal secretions, but it is not commonly transmitted transplacentally.

Hepatitis B surface antigen (HBsAg) is the first serologic marker appearing in the serum at 6-16 weeks following HBV infection. In acute infection, HBsAg usually disappears in 1-2 months after the onset of symptoms. Persistence of HBsAg for >6 months indicates development of either a chronic carrier or chronic HBV infection.

Useful For

- Testing cadaveric and hemolyzed blood specimens for HBsAg; FDA-licensed for use with hemolyzed specimens
- Diagnosis of acute, recent (<6 month duration), or chronic hepatitis B infection; determination of chronic hepatitis B carrier status

Interpretation

- A positive result (positive screening and confirmed positive by neutralization test; see Method Section) is indicative of acute or chronic HBV infection, or chronic HBV carrier state.
- A positive (confirmed) neutralization test result is considered the definitive test result for HBsAg. Specimens that are positive by the screening test but negative (not confirmed) by the neutralization test are likely to contain cross-reactive antibodies from other infectious or immunologic disorders. These nonconfirmed HBsAg screening test results should be interpreted in conjunction with test results of other HBV serological markers (e.g., anti-HBs antibody, anti-HBc total antibody).
- The presence of HBsAg is frequently associated with HBV infectivity, especially when accompanied by the presence of HBe antigen and/or HBV DNA.

Cautions

- This test is not useful during the “window period” of acute HBV infection, (i.e., after disappearance of HBsAg and prior to appearance of anti-HBs antibody). Testing for acute HBV infection should also include anti-HBc IgM antibody.
- Positive HBsAg test results should be reported by the health care provider to the state Department of Health, as required by law in some states.
- Performance characteristics have not been established for the following specimen characteristics:
  - Lipemic
  - Containing particulate matter
Test Title: Hepatitis B Surface Antigen (HBsAg) for Cadaveric or Hemolyzed Specimens, Serum #83626

References

Method
Specimens are first screened by the Genetics System HBsAg 3.0 EIA. All positive results are confirmed by the Genetics System HBsAg Confirmatory Assay 3.0 (HBsAg Neutralization) at an additional charge.

EIA:
Wells of a microwell strip plate that are coated with mouse monoclonal antibody to HBsAg are incubated with patient serum or plasma, appropriate controls and mouse monoclonal anti-HBs peroxidase conjugate. HBsAg, if present, is bound to the solid-phase antibody and simultaneously bound by the anti-HBs conjugate. After aspiration and washing, working chromogen solution is added to the wells. After incubation, a blue or blue-green color develops in proportion to the amount of HBsAg bound to the bead.

The enzyme reaction is stopped by the addition of acid resulting in a color change to yellow. The absorbance values of controls and specimens are determined using a spectrophotometer with wavelength set at 450 nm. Specimens giving absorbance values equal to or greater than the absorbance value of the negative control mean plus a factor are considered initially reactive for HBsAg. Those giving absorbance values less than the negative control mean plus a factor are considered negative. (Package Insert: Genetics Systems HBsAg 3.0 EIA. Bio-Rad Laboratories, Redman, WA)

Neutralization:
The Genetic Systems HBsAg Confirmatory Assay 3.0 uses the principle of specific antibody neutralization to confirm the presence of HBsAg. The confirmatory reagent (human antibody to HBsAg) is incubated with the specimen in solution. If HBsAg is present in the specimen, it will be bound by the confirmatory reagent. The treated specimen is reassayed using the Genetics Systems HBsAg 3.0 EIA kit. The neutralized HBsAg is subsequently blocked from binding to the antibody-coated wells. This results in a reduction of signal when compared to the nonneutralized specimen in which negative control is used in place of confirmatory reagent. By definition, a specimen is confirmed as positive if the reduction in signal of the neutralized specimen is at least 50% and the nonneutralized control generates a signal greater than or equal to the assay cutoff. (Package Insert: Genetics Systems HBsAg Confirmatory Assay 3.0, Bio-Rad Laboratories, Redman, WA)

Specimen Required:
Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 2.0 mL of serum frozen in plastic vial.

Reference Values:
Negative

Analytic Time:
Same day/1 day

Days Set Up:
Monday through Friday; 8 a.m., 11 a.m., 3:30 p.m.
Saturday and Sunday; 9 a.m.

CPT Code:
87340/ HBsAg
87341/ HBsAg neutralization (if appropriate)
Human Immunodeficiency Virus (HIV) Types 1 and 2
Antibodies for Cadaveric or Hemolyzed Specimens, Serum
#83628

Clinical
Epidemiological data indicate that acquired immunodeficiency syndrome (AIDS) is caused by at least 2 types of human immunodeficiency virus (HIV). The first virus, HIV-1, has been isolated from patients with AIDS, AIDS-related complex, and asymptomatic infected individuals at high risk for AIDS. HIV-1 is transmitted by sexual contact, exposure to infected blood or blood products, or from an infected mother to her fetus or infant. A second HIV virus, HIV-2, was isolated from patients in West Africa in 1986. HIV-2 appears to be endemic only in West Africa, but it also has been identified in individuals who have lived in West Africa or had sexual relations with individuals from that geographic region. HIV-2 is similar to HIV-1 in its morphology, overall genomic structure, and its ability to cause AIDS.

Antibodies against HIV-1 and HIV-2 are usually not detected until 6-12 weeks following exposure and are almost always detected by 12 months. They may fall into undetectable levels in the terminal stage of AIDS.

Useful For
Screening cadaveric and hemolyzed blood specimens for HIV-1 or HIV-2; FDA-licensed for testing on hemolyzed blood

Interpretation
• A reactive result must be followed by supplemental testing (i.e., #9190 “Human Immunodeficiency Virus-1 [HIV-1] Antibody, Western Blot Assay, Serum”).
• A negative result should be interpreted with caution if patient has a history of high-risk behavior or has clinical symptoms suggestive of HIV infection.
• Anti-HIV-1/HIV-2 EIA does not differentiate between anti-HIV-1 and anti-HIV-2 antibody reactivity.
• If anti-HIV-1/HIV-2 is reactive and the HIV-1 Western blot is indeterminate or negative, consider the possibility of HIV-2 infection.

Cautions
• This test is for screening purposes only.
• Test should not be ordered without informed consent.
• Not useful for distinguishing active infection vs. passive transfer of maternal anti-HIV-1/2 antibody in infants in the postnatal period (up to 18 months of age).
• The predictive value of a positive or negative EIA result is highly dependent in the prevalence of HIV infection in the population tested.
• A reactive test result (when supplemented by a positive Western blot) should be reported by the attending physician to the State Department of Health, as required by law in many states.
• Performance characteristics have not been established for the following specimen characteristics:
  – Containing particulate matter
References


Method

The Genetics Systems HIV-1/HIV-2 PLUS O EIA is based on the principle of direct antibody sandwich technique. Microwell strip plates (solid phase) are coated with purified HIV antigens: gp190 and p24 recombinant proteins derived from HIV-1, gp36 peptide representing the immunodominant region of the HIV-2 transmembrane glycoprotein, and a synthetic polypeptide mimicking an artificial (not encoded by any existing virus) HIV-1 group O specific epitope.

Clinical serum or plasma samples and assay controls are added to the plate with specimen diluent containing a dye that changes color from purple to blue when combined with a specimen sample or control. The wells are incubated and then washed. Following addition of a colored conjugate solution (green) that contains the peroxidase-conjugated HIV-1 and HIV-2 antigens, the wells are incubated again. If anti-HIV-1 and/or anti HIV-2 antibody is present, it will not bind to the antigen coated on the well and to the peroxidase-conjugated antigens in the conjugate solution. The antigen-antibody-antigen complexes remain bound to the well during a subsequent wash step that will remove any unbound materials. Working TMP Solution is added to the plate wells and allowed to incubate. A blue or blue-green color develops in proportion to the amount of HIV antibody present in the sample. Color development is stopped by the addition of acid which changes the blue-green color to yellow. The optical absorbance of specimens and controls is determined spectrophotometrically at a wavelength of 450 nm. (Package Insert: Genetics Systems HIV-1/HIV-2 PLUS O EIA. Bio-Rad Laboratories, Redman, WA)

Specimen Required: Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 1.0 mL of serum frozen in a screw-capped, round-bottom, plastic vial.

Reference Values: Negative

Analytic Time: Same day/1 day

Days Set Up: Monday through Friday; 8 a.m., 11 a.m., 3:30 p.m.
Saturday and Sunday; 9 a.m.

CPT Code: 86703
St. Louis Encephalitis Antibody, IgG and IgM, Serum
#83154

Profile Information

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Clinical

Onset is characterized by generalized malaise, fever, chilliness, headache, drowsiness, nausea, and sore throat or cough followed in 1-4 days by the meningeal and neurologic signs. The severity of illness increases with advancing age; persons over 60 years have the highest frequency of encephalitis. Symptoms of irritability, sleeplessness, depression, memory loss, and headaches can last up to 3 years. Areas of outbreaks since 1933 have involved the western United States, Texas, the Ohio-Mississippi Valley and Florida. The vector of transmission is the mosquito. Peak incidence of St. Louis encephalitis is associated with summer and early autumn.

Useful For

Aiding in the diagnosis of St. Louis encephalitis

Interpretation

• In patients with this virus, IgG antibody is generally detectable within 1-3 weeks of onset, peaking within 1-2 months, and declining slowly thereafter.
• IgM class antibody is also reliably detected within 1-3 weeks of onset, peaking and rapidly declining within 3 months.
• Single serum specimen IgG ≥1:10 indicates exposure to the virus.
• Results from a single serum specimen can differentiate early (acute) infection from past infection with immunity if IgM is positive (suggests acute infection).
• A 4-fold or greater rise in IgG antibody titer in acute and convalescent sera indicate recent infection.
• Infections with St. Louis encephalitis can occur at any age. The age distribution depends on the degree of exposure to the particular transmitting arthropod relating to age and sex, as well as the occupational, vocational, and recreational habits of the individuals. Once humans have been infected, the severity of the host response may be influenced by age: St. Louis encephalitis tends to produce the most severe clinical infections in older persons. Infection among males is primarily due to working conditions and sports activity taking place where the vector is present.

Cautions

• All results must be correlated with clinical history and other data available to the attending physician.
• Samples drawn within the first 2 weeks after onset are variably negative for IgG antibody and should not be used to exclude the diagnosis of St. Louis encephalitis. If St. Louis encephalitis is suspected, a second sample should be obtained and tested 10-21 days later.
• Since cross-reactivity with dengue fever virus does occur with St. Louis encephalitis antigen and, therefore, cannot be differentiated further. The specific virus responsible for such a titer may be deduced by the travel history of the patient, along with available medical and epidemiological data, unless the virus can be isolated.
• Usually, when an infection with an arbovirus is suspected, it is too late to isolate the virus or obtain serum specimens to detect a rise of antibody titer.
Test Title: St. Louis Encephalitis Antibody, IgG and IgM, Serum
#83154

References

Method
Indirect immunofluorescence.


Specimen Required: Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 0.5 mL of serum refrigerated in a screw-capped, round-bottom, plastic vial. Forward promptly.

Reference Values:
IgG: <1:10
IgM: <1:10
See "Virology" in Special Instructions for additional interpretive information.

Analytic Time: Same day/1 day
Days Set Up: Monday through Friday; 9 a.m.
CPT Code: 86653/x2
Western Equine Encephalitis Antibody, IgG and IgM, Serum #83156

**Profile Information**

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<td>West Equine Enceph Ab, IgM, S</td>
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**Clinical**

The virus that causes western equine encephalitis (WEE) is widely distributed throughout the United States and Canada; disease occurs almost exclusively in the western states and Canadian provinces. The relative absence of the disease in the eastern United States probably reflects a paucity of the vector mosquito species, *Culex tarsalis*, and possibly a lower pathogenicity of local virus strains. The disease usually begins suddenly with malaise, fever, and headache, often with nausea and vomiting. Vertigo, photophobia, sore throat, respiratory symptoms, abdominal pain and myalgia are also common. Over a few days, the headache intensifies; drowsiness and restlessness may merge into a coma in severe cases. In infants and children, the onset may be more abrupt than for adults. WEE should be suspected in any case of febrile central nervous system disease from an endemic area. Infants are highly susceptible to central nervous system disease and about 20% of cases are under 1 year of age. There is an excess of males with WEE clinical encephalitis, averaging about twice the number of infections detected in females. After recovery from acute disease, patients may require from several months to 2 years to overcome the fatigue, headache and irritability. Infants and children are at higher risk of permanent brain damage after recovery than adults.

**Useful For**

Aiding the diagnosis of western equine encephalitis

**Interpretation**

- In patients infected with this virus, IgG antibody is generally detectable within 1-3 weeks of onset, peaking within 1-2 months, and declining slowly thereafter.
- IgM class antibody is also reliably detected within 1-3 weeks of onset, peaking and rapidly declining within 3 months.
- Single serum specimen IgG ≥1:10 indicates exposure to the virus.
- Results from a single serum specimen can differentiate early (acute) infection from past infection with immunity if IgM is positive (suggests acute infection).
- A 4-fold or greater rise in IgG antibody titer in acute and convalescent sera indicate recent infection.
- In the United States it is unusual for any patient to show positive reactions to more than 1 of the arboviral antigens, although WEE and eastern equine encephalitis (EEE) antigens will show a noticeable cross-reactivity.
- Infections with arboviruses can occur at any age. The age distribution depends on the degree of exposure to the particular transmitting arthropod relating to age and sex, as well as the occupational, vocational, and recreational habits of the individuals. Once humans have been infected, the severity of the host response may be influenced by age: WEE tends to produce the most severe clinical infections in young persons. Infection in males is primarily due to working conditions and sports activity taking place where the vector is present.

**Cautions**

- All results must be correlated with the clinical history and other data available to the attending physician.
- Samples drawn within the first 2 weeks after onset are variably negative for IgG antibody and should not be used to exclude the diagnosis of arboviral disease. If arboviral infection is suspected, a second sample should be obtained and tested 10-21 days later.
- Since cross-reactivity with dengue fever virus does occur with St. Louis encephalitis antigen and therefore, cannot be differentiated further. The specific virus responsible for such a titer may be deduced by the travel history of the patient, along with available medical and epidemiological data, unless the virus can be isolated.

—Continued on next page.
Test Title: Western Equine Encephalitis Antibody, IgG and IgM, Serum 
#83156

Cautions
(continued)
• EEE and WEE viruses show some cross-reactivity; however, antibody response to the infecting virus is typically at least 8-fold higher.
• Usually, when an infection with an arbovirus is suspected, it is too late to isolate the virus or obtain serum specimens to detect a rise of antibody titer.

References

Method
Indirect immunofluorescence.

Specimen Required: Draw blood in a plain, red-top tube(s) or a serum gel tube(s). Spin down and send 0.5 mL of serum refrigerated in a screw-capped, round-bottom, plastic vial. Forward promptly.

Reference Values:
IgG: <1:10
IgM: <1:10
See “Virology” in Special Instructions for additional interpretive information.

Analytic Time: Same day/1 day
Days Set Up: Monday through Friday; 9 a.m.
CPT Code: 86654/x2
Wilson Disease (WD) is an autosomal recessive disorder that results in the body’s inability to excrete excess copper due to mutations in the \textit{ATP7B} gene. Copper is an essential mineral that comes from the diet and is necessary for good health. The amount of copper required by the human body is minimal; however, the regulation of its quantity is important. Individuals with WD lack the necessary enzyme that facilitates clearance of copper from the liver to bile. As a result, copper accumulates first in the liver and gradually in other organs, particularly the brain, but also in the kidney, bones, and cornea (Kayser-Fleischer ring). Another characteristic finding in WD is failure of copper incorporation into the protein ceruloplasmin (CP), resulting in a low CP level. Since this protein accounts for 95\% of serum copper, affected patients also present with abnormally low serum copper level.

WD affects approximately 1 in 30,000 people worldwide. Approximately 1 in 90 individuals carries a gene for WD. If both parents carry the gene, there is a 25\% risk that a child will inherit both disease genes and be affected with WD. Individuals who are heterozygous for WD do not show clinical symptoms.

The main clinical features of WD are hepatic and neurologic. Age of onset ranges from 3 years to more than 50 years of age, but affected patients typically present in their early teens or 20s. The pattern and degree of liver involvement is variable. Some patients present with hepatomegaly, fever, and other nonspecific symptoms that mimic common disorders such as viral hepatitis and infectious mononucleosis. Other patients may initially present with rapidly progressive hepatic failure with evidence of cirrhosis and overwhelming hepatocyte necrosis. Liver disease is the most common presenting feature in children, typically occurring at 10-13 years of age.

While hepatic manifestations predominate in the pediatric age group, neurologic manifestations occur in later stages of the disease, usually between the ages of 15-30, and reflect the extrahepatic accumulation of copper after the liver has been saturated. About half of these patients have experienced behavioral abnormalities or changes with deterioration of school performance preceding their neurological symptoms. The symptoms may initially be subtle but will progress to Parkinsonian symptoms due to pathologic involvement of basal ganglia presenting with tremor, lack of motor coordination, drooling, dysarthria, dystonia, and spasticity. Because of pseudobulbar palsy, dysphagia occurs with high risk of aspiration. Psychiatric manifestations such as subtle mood changes, depression, and inappropriate social behavior are reported in approximately 20\% of individuals with WD.

Treatment with chelating agents or zinc, along with a copper-restricted diet, is effective if started early. If not treated, WD can cause liver failure, severe brain damage, and even death. In advanced cases, liver transplant may be necessary. Usually, individuals with WD are diagnosed after severe neurologic or liver dysfunction occurs. This is unfortunate because, while treatment can be helpful for some advanced patients, it is not curative. Treatment is very effective, however, if diagnosis occurs before the onset of life-threatening symptoms. In fact, asymptomatic patients who have been diagnosed and treated do not experience liver or neurologic dysfunction. The WD screening assay, which determines the level of ceruloplasmin, allows presymptomatic detection of affected patients.

\textbf{Useful For} Presymptomatic screening for Wilson disease after 3 months of age
Test Title: Wilson Disease Screening, Blood Spot
#83696

Interpretation
- Reports are in text form only. A report for a normal screening result will be reported as screen negative.
- An abnormal result is not sufficient to conclusively establish a diagnosis of WD. Follow-up tests, including serum copper (#8612 “Copper, Serum”); ceruloplasmin (#8364 “Ceruloplasmin, Serum”); urine 24-hour copper measurement (#8590 “Copper, Urine”); or molecular genetic analyses, are required. Liver biopsy may also be required for confirmation.

Cautions
- Ceruloplasmin levels are physiologically low in the neonatal period. This test is not recommended for newborns.
- Values of ceruloplasmin vary considerably from patient to patient and may be in the normal range in some patients with WD. Approximately 5% of WD patients were reported to have normal ceruloplasmin values.
- Ceruloplasmin levels are affected by infections (ceruloplasmin is a late acute-phase reactant) and liver function.
- Birth control pills and pregnancy increase ceruloplasmin levels.

References

Method
WD screening involves quantitation of ceruloplasmin (CP) levels in dried blood spots. Blood from a finger prick is dripped onto a filter paper collection card. The CP is eluted from the dried filter paper blood spot in a 96-well plate that has been coated with anti-CP antibody. After elution, the blood spots and elution buffer are removed from the wells and the plate is washed. A secondary anti-CP antibody, which is conjugated to horseradish peroxidase (HRP), is added and allowed to incubate. The plate is washed again before adding tetramethylbenzidine (TMB). The TMB reacts with the HRP bound to the CP-antibody complexes affixed to the plate, causing the solutions in the wells to turn teal. The reaction is stopped with the addition of hydrochloric acid, which turns the solutions yellow. This yellow color is detected at 450 nm by a plate reader. A standard curve of absorbance versus the CP concentration is constructed and used to determine CP content of control and patient blood spots. (Hahn SH, Lee SY, Jang YJ, et al: Pilot study of mass screening for Wilson's disease in Korea. Mol Genet Metab 2002 Jun;76(2):133-136)

Specimen Required: 3 blood spots. Let blood dry on the “Wilson Disease Screening Card” (Supply T532) at ambient temperature in a horizontal position for 3 hours. Please complete page 2 of the “Wilson Disease Screening Card.”

Note: 1. Do not expose specimen to heat or direct sunlight.
2. Do not stack wet specimens.

Reference Values: An interpretive report will be issued.

Analytic Time: 2 days
Days Set Up: Varies
CPT Code: 83520
## 2005 Education Calendar

### Interactive Satellite Programs . . .

**HIV Update**  
**February 22, 2005**  
**Presenter:** Joseph D. Yao, MD  
**Moderator:** Robert M. Kisabeth, MD

**Hypertension: New Developments in Laboratory Testing & Genomics**  
**March 8, 2005**  
**Presenter:** Stephen T. Turner, MD and Vincent J. Canzanello, MD  
**Moderator:** Sandra J. Taler, MD

**Obesity Management**  
**April 12, 2005**  
**Presenter:** Maria L. Collazo-Clavell, MD  
**Moderator:** Robert M. Kisabeth, MD

**Drug Cautions in the Elderly**  
**May 10, 2005**  
**Presenter:** David G. Bell, MD  
**Moderator:** Robert M. Kisabeth, MD

**Congestive Heart Failure**  
**June 1, 2005**  
**Presenter:** Allan S. Jaffe, MD  
**Moderator:** Robert M. Kisabeth, MD

**Laboratory and Clinical Collaboration in the Diagnosis and Management of Thyroid Disease**  
**June 14, 2005**  
**Presenter:** Bryan McIver, MBChB, PhD  
**Moderator:** Robert M. Kisabeth, MD

**Alzheimer's: An Update on Treatment and Research**  
**September 6, 2005**  
**Presenter:** Ronald C. Petersen, MD, PhD  
**Moderator:** Robert M. Kisabeth, MD

**Genomics & Proteomics – An Update**  
**November 1, 2005**  
**Presenter:** David B. Schowalter, MD, PhD  
**Moderator:** Robert M. Kisabeth, MD

**Liver Diseases You Shouldn't Miss**  
**December 13, 2005**  
**Presenter:** David J. Brandhagen, MD  
**Moderator:** Robert M. Kisabeth, MD

### Upcoming Education Conferences . . .

**Advanced Spirometry**  
**February 23-24, 2005**  
Mayo Clinic, Siebens Building • Rochester, Minnesota

**Practical Spirometry**  
**March 15-16, 2005**  
Mayo Clinic, Siebens Building • Rochester, Minnesota

**Phlebotomy Conference 2005: Dispelling the Myths**  
**March 17-18, 2005**  
Mayo Clinic, Siebens Building • Rochester, Minnesota

**Quality Phlebotomy: Back to the Basics**  
**April 2005**  
Location to be determined • Chicago, Illinois

**12th International Surgical Pathology Symposium**  
**May 3-6, 2005**  
Sofitel Victoria Hotel • Warsaw, Poland

**Integration Through Community Laboratory Insourcing**  
**May 18-20, 2005**  
Sofitel Philadelphia • Philadelphia, Pennsylvania

**4th Biennial Symposium – Pulmonary Pathology Society**  
**June 15-17, 2005**  
L’Imperial Palace • Annecy, France

**Coagulation Testing Quality: Lessons and Issues from Quality Assessment, Standardization and Improvement Programs & Studies**  
**June 15-17, 2005**  
The Kahler Grand Hotel • Rochester, Minnesota

**How the Practice of Medicine Informs Technology**  
**July 23, 2005**  
Rosen Centre Hotel • Orlando, Florida

**Practical Surgical Pathology**  
**September 29-October 1, 2005**  
Mayo Clinic, Siebens Building • Rochester, Minnesota

**Quality Phlebotomy: Back to the Basics**  
**October 2005**  
Location to be determined • Los Angeles, California

**Practical Spirometry**  
**November 17-18, 2005**  
Mayo Clinic, Siebens Building • Rochester, Minnesota

**Real-Time PCR for the Clinical Laboratory**  
**November 17-18, 2005**  
Mayo Clinic, Siebens Building • Rochester, Minnesota

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FOR MORE INFORMATION on these continuing medical education programs, please contact: Mayo Reference Services Education Department at 800-533-1710 or 507-284-3156. Visit us under “Education” at www.mayoreferenceservices.org.