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Communiqué

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Mayo Medical Laboratories' Supplemental Newborn Screening Program

Newborn Screening

State-mandated newborn screening was initiated in the early 1960s for the identification of infants affected with phenylketonuria (PKU). Shortly thereafter screening programs expanded to include additional genetic and non-genetic conditions. State-mandated newborn screening programs are not uniform across the United States. Only PKU and congenital hypothyroidism are included in all screening programs. The goal of newborn screening is the presymptomatic diagnosis of disorders for which early treatment can prevent or reduce morbidity and mortality. A newborn screening test should be simple and affordable and permit the analysis of dried blood spots.

The first newborn screening test was developed by Dr. Robert Guthrie to detect elevated levels of phenylalanine using a bacterial inhibition assay, best known as the Guthrie Test.¹ While still in use in a few laboratories, most testing is done today using immuno- and enzymatic assays. In the 1980s, tandem mass spectrometry (MS/MS) was initially introduced in clinical laboratories for the selective screening for inborn errors of fatty acid and organic acid metabolism. Over the last decade, this technology has been applied to newborn screening because it is amenable to population-wide testing for a large number of disorders of fatty acid, organic acid and amino acid metabolism (Table 1).² However, screening laboratories' lack of experience with this drastically different analytical platform and the degree of complexity of result interpretation has prevented the implementation of MS/MS in most state-mandated newborn screening programs. Currently, only a few state and private screening laboratories take full advantage of MS/MS, while others limit its use to screening for the most common fatty acid oxidation disorder, medium-chain acyl-CoA dehydrogenase (MCAD) deficiency.³

Tandem Mass Spectrometry

Tandem mass spectrometers allow the rapid analysis of individual compounds in complex mixtures. Two mass spectrometers are coupled, separated only by a collision cell, all within one instrument. The first (MS1) or second analyzer (MS2) can be set either to scan a mass range or to select one or more individual ions of a specific mass-to-charge ratio (m/z). The collision cell is utilized to further breakdown the ions by use of a neutral gas (eg, nitrogen) and to transmit them to MS2 (Figure 1).⁴ This design enables four different analytical or scan modes, three of which are being used by Mayo's Biochemical Genetics Laboratory for the purpose of newborn screening.

For acylcarnitine profiling, MS1 scans a defined mass range and MS2 is set to transmit only fragment ions with a specific m/z value following collision-activated fragmentation. In this mode, the data system then correlates each detected ion to its respective precursor scanned in MS1 (precursor or parent ion scan). In a neutral loss experiment MS1 and MS2 are both scanned at the same rate with a constant m/z difference. The resulting spectrum includes only those compounds, among precursor ions, that fragment with a common neutral loss (a behavior indicating that they belong to a family of structurally related compounds). This scan is used for the generation of amino acid profiles.

The concentrations of a few, selected amino acids and acylcarnitine species are more accurately measured when taking advantage of an MS/MS analysis in selected reaction monitoring (SRM) mode, where the selection of a parent ion in MS1 is followed by a similar process for a specific fragment ion in MS2. The resulting signal corresponds exclusively to the transition from parent to product ion, a process virtually free of any interference irrespective of the specimen analyzed.

Table 1. Disorders detectable by amino acid and acylcarnitine analyses using MS/MS.

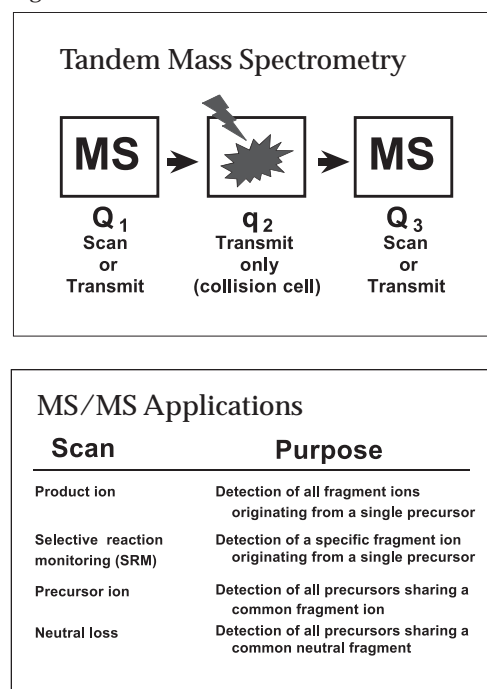
Amino Acid Metabolism	Fatty Acid Metabolism	Organic Acid Metabolism
Argininemia	Short-chain acyl-CoA dehydrogenase (SCAD) deficiency	Glutaric Acidemia Type I (GA-1)
Argininosuccinic aciduria	Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency	Isovaleric Acidemia
Citrullinemia	Mitochondrial trifunctional protein (TFP) deficiency	3-Ketothiolase deficiency
Homocystinuria	Long-chain 3-hydroxy acyl-CoA dehydrogenase (LCHAD) deficiency	3-Methylcrotonyl-CoA carboxylase deficiency
Maple syrup urine disease (MSUD)	Very long-chain acyl-CoA dehydrogenase (VLCAD) deficiency	3-Methylglutaconyl-CoA hydratase deficiency
Phenylketonuria (and other hyperphenylalaninemias)	Carnitine palmitoyl-transferase deficiency type II (CPT-2)	Multiple carboxylase deficiency
Tyrosinemia type I	Carnitine-acylcarnitine translocase (CACT) deficiency	Methylmalonic Acidemias
Tyrosinemia type II	Glutaric acidemia type II (GA-2; multiple acyl-CoA dehydrogenase deficiency)	Propionic Acidemia
	2,4-Dienoyl-CoA reductase deficiency	
	3-Hydroxy-3-methylglutaryl-CoA (HMG-CoA) lyase deficiency	

Newborn Screening and Mayo's Biochemical Genetics Laboratory

Historically, testing for inborn errors of metabolism (IEM) has been provided predominantly by research laboratories, each offering analyses only for disorders in line with their scientific interest. With increasing awareness of genetics in medicine and approximately one thousand IEMs identified to date, Clinical Biochemical Genetics is now recognized as a laboratory discipline concerned with the evaluation and diagnosis of patients and families with inherited metabolic disease, monitoring of treatment, and distinguishing heterozygous carriers from non-carriers by metabolite and enzymatic analysis of physiological fluids and tissues.

The Biochemical Genetics Laboratory at Mayo is an interdisciplinary group of laboratorians, geneticists, and pediatricians. The mission of the Biochemical Genetics Laboratory is to provide biochemical testing and result interpretation of the highest quality for the diagnosis, study, and clinical care of patients with inborn errors of metabolism, high risk of cardiac disease, malabsorption, and malnutrition disorders. The Biochemical Genetics Laboratory routinely performs qualitative detection and quantitative determination of diagnostic markers based on a variety of manual, automated, and chromatographic methods, particularly high-performance liquid chromatography (HPLC), gas chromatography/mass spectrometry (GC/MS), and MS/MS.

Figure 1.



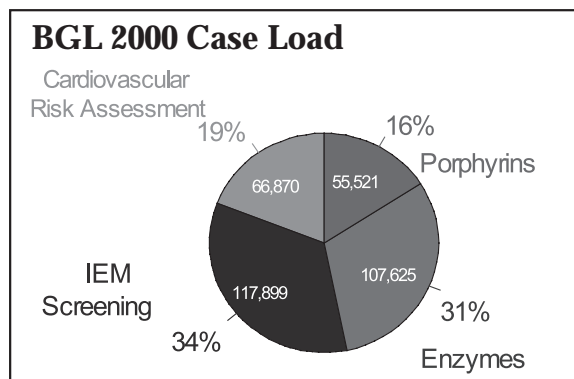
In 2000, a total of 345,787 tests were performed using over 160 different procedures, the majority of tests aimed to the biochemical diagnosis of IEM. Several of these procedures are screening tests, such as the analyses of amino acids, organic acids and acylcarnitines, and are performed on specimens from patients presenting with symptoms reminiscent of an IEM (high-risk screening), postmortem samples obtained primarily from children that have died suddenly without apparent reason (postmortem screening), as well as for the purpose of prenatal diagnosis. During the year 2000, Mayo made biochemical diagnoses in approximately 1,300 cases with 67 different IEMs (Table 2)(Figure 2).

While there have always been discrepancies between the various state programs, the inconsistent integration of MS/MS has widened the gap. Currently, state programs range from screening for only 3 disorders in some states to more than 30 in others. With proven expertise, Mayo Medical Laboratories (MML) bridges the gaps in state screening programs by offering [Supplemental Newborn Screen, Blood Spot \(#82594\)](#). MML formed a collaborative agreement with Neo Gen Screening, Inc. to offer this supplemental newborn screening. MML will use reference values and procedures developed by Neo Gen Screening, an independent laboratory with experience in high-throughput newborn screening using MS/MS.

Table 2. Diagnosed disorders in 1999 and 2000 divided according to general categories.

	1999	2000
Amino acid metabolism	63	74
Carbohydrate metabolism	262	235
Congenital disorders of glycosylation	20	12
Cholesterol biosynthesis	3	4
Mitochondrial energy metabolism (primary lactic acidemias)	16	41
Lysosomal metabolism and storage	248	345
Lipoprotein metabolism disorders	107	94
Fatty acid metabolism (mitochondrial β -oxidation oxidation) and carnitine homeostasis	63	74
Organic acid disorders	90	135
Peroxisomal biogenesis and metabolism	64	80
Porphyryn metabolism	132	193
Purine and pyrimidine metabolism	5	10
Total	1073	1297

Figure 2



Neo Gen Screening has analyzed blood samples from over one million infants.

MML offers the additional benefit of a full spectrum of follow-up testing. Although the accuracy of MS/MS in the identification of a detectable disorder is significant, it remains a screening test that requires further testing to confirm a disorder (usually on a sample other than a blood spot).

Limitations of Newborn Screening

The reliability of MS/MS screening for some fatty acid β -oxidation disorders, ie, trifunctional protein (TFP) deficiency, has not been proven conclusively, based on the prospective analysis of a significant number of cases (Table 3).⁵ In addition, the possibility of identifying patients with poorly defined disorders

(such as short-chain acyl-CoA dehydrogenase [SCAD] deficiency) or those for which no effective long-term treatment is available (ie, neonatal onset carnitine-palmitoyltransferase type II [CPT-II] deficiency) has been used as an argument against the expansion of newborn screening programs. However, an early diagnosis of these conditions is beneficial because it allows for proper genetic counseling including future reproductive decision-making, is provided at no added cost, and could greatly contain the scope and cost of the diagnostic work-up of an acutely ill newborn.⁶

Future Expansion of Newborn Screening Programs

The Biochemical Genetics Laboratory is continuously developing new methods, which allow for either more accurate diagnoses or further expansion of the spectrum of identifiable disorders. This includes the testing of newborn blood spots.

Congenital Adrenal Hyperplasia (CAH)

Screening for CAH is in place in 25 states at this time. The remaining states do not screen for this inherited endocrinopathy because the available immunoassays (ie, fluorescent immunoassay) for the determination of 17α -hydroxy progesterone (17OHP) have a poor positive predictive value causing approximately 200 false positives for each truly abnormal result. This translates into a significant financial burden on the health care system and, perhaps more importantly, unnecessary blood draws for the predominantly premature babies and emotional stress for the involved families.

To overcome this unfavorable situation, the Biochemical Genetics Laboratory in collaboration with Mayo's Endocrine Laboratory recently developed a new MS/MS based method for the determination of 17OHP and other steroids.⁷ Retrospective testing of more than 200 blood spots indicates an improvement of the positive predictive value from 0.5% to higher than 80%.

Congenital Disorders of Glycosylation (CDG)

CDG are a rapidly growing group of disorders, the first of which was described in 1980. Mayo's Biochemical Genetics Laboratory has been involved in the testing of children with symptoms suggestive of one of these disorders for many years by isoelectrofocusing of transferrin isoforms. In 2000, an MS-based new test was developed by the Biochemical Genetics Laboratory that led to dramatic improvements in sample volume requirements (1 mL serum vs. 5 mL) and turnaround time (3 days vs. 24 hours).⁸

Table 3. Tandem mass spectrometry in newborn screening for fatty acid b-oxidation disorders*

Disorder	Effectiveness of early treatment ^a	Effectiveness of MS/MS ^b
<u>Disorders of membrane-bound enzymes</u>		
Carnitine transport defect	+++	+/-
Long-chain fatty acid transport defect	+ §	-
CPT-I deficiency (liver)	+	(+)
CACT deficiency	+	+
CPT-II deficiency (neonatal onset)	-	+
CPT-II deficiency (late onset)	+	+
VLCAD deficiency	+	(+)
ETF-QO deficiency (GA2)		
Early onset (+/- congenital anomalies)	-	+
Late onset	+	+
LCHAD deficiency	+	(+)
TFP deficiency (α, β)	+ §	(+)
<u>Disorders of mitochondrial matrix enzymes</u>		
MCAD deficiency	+++	+
SCAD deficiency	+	+
Functional SCAD deficiency	?	+
ETF deficiency (α, β, GA2)		
Early onset (+/- congenital anomalies)	-	+
Late onset	+	+
Riboflavin responsive form(s) (GA2)	+++	(+)
SCHAD deficiency (muscle)	+	-
SCHAD deficiency (fibroblasts)	+	
SCHAD deficiency (liver)	+ §	
M/SCHAD deficiency	+ §	(+)
MCKAT deficiency	+	(+)
SKAT deficiency	+++	(+)
2,4-Dienoyl-CoA reductase deficiency	?	(+)
HMG-CoA synthase deficiency	+	-
HMG-CoA lyase deficiency	+	+

* from: Rinaldo P, Matern D. Disorders of fatty acid transport and mitochondrial oxidation: challenges and dilemmas of metabolic evaluation. *Genet Med.* 2000;2:338-44

Legend for Table 3

Abbreviations are as follows, in alphabetical order: CPT, carnitine palmitoyltransferase; ETF-QO, electron transfer flavoprotein ubiquinone-oxidoreductase; GA2, glutaric acidemia type II; HMG, 3-hydroxy 3-methylglutaryl; LCHAD, long-chain L-3-hydroxy acyl-CoA dehydrogenase; MCAD, medium-chain acyl-CoA dehydrogenase; MCKAT, medium-chain 3-ketoacyl-CoA thiolase; M/SCHAD, medium/short chain L-3-hydroxy acyl-CoA dehydrogenase; SCAD, short-chain acyl-CoA dehydrogenase; SCHAD, short-chain L-3-hydroxy acyl-CoA dehydrogenase; TFP, trifunctional protein; VLCAD, very long-chain acyl-CoA dehydrogenase.

^aEffectiveness of treatment: +++, demonstrated; +, dietary and preventive measures, -, no effective treatment; §, liver transplantation; ?, insufficient information.

^bEffectiveness of MS/MS: +, demonstrated in blood spots; (+), expected to be effective, but not yet conclusively demonstrated; -, not effective.

This new method now has been applied to the analysis of dried blood spots with the potential to be used for newborn screening. Some may question the rationale for inclusion of CDG in newborn screening programs since only CDG type Ib is treatable by oral supplementation with mannose. However, the extremely variable, and for the most part nonspecific phenotype, combined with the novelty of the disorders,

typically leads to a vast number of unnecessary diagnostic procedures for the patient and most produce no diagnosis. As outlined above, the identification of affected patients in the newborn period would provide for early treatment of patients with CDG type Ib and the availability of a diagnosis, genetic counseling, and prenatal diagnosis for all affected families.

Before the analyses for steroids and transferrin isoforms will be included in MML's Supplemental Newborn Screening Program, the Biochemical Genetics Laboratory will validate these new methods in IRB approved prospective studies involving neonates born at Mayo Clinic Rochester. Completion of these studies is anticipated for the end of 2001 followed by rapid availability to our clients' newborns.

Mayo Medical Laboratories' Supplemental Newborn Screening

Mayo Medical Laboratories' Supplemental Newborn Screen, Blood Spot (#82594) provides screening for the disorders listed in Table 1. The test is performed on a blood spot filter paper specimen. Please see the New Test Announcement in this issue for further information about this test. Additional questions about supplemental newborn screening can be directed to our 24-hour, toll-free number: 800-533-1710.

References:

1. Guthrie R: The introduction of newborn screening for phenylketonuria. A personal history. *Eur J Pediatr* 155: S4-5, 1996
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Method Change for Carbohydrate Antigen 19-9

A new method has been implemented for [Carbohydrate Antigen 19-9 \(CA 19-9\), Serum #9288](#). The new method has better linearity and does not have high-dose hook problems, which caused the previous method to potentially produce falsely low values when measuring specimens with very high concentrations of CA 19-9.

This new method correlates well with the previous method ($r = 0.99$) and the upper reference value remains the same (<40 U/mL); however, the new method may result in 20 to 45% higher patient values. Comparison with the previous method showed slopes varying from 1.2X higher (for values <50 U/mL) to 1.45X higher (for high values). For this reason, specimens that were submitted to MML between 4/9/01 and 7/9/01 have been held frozen and, if requested, we can reanalyze a previous specimen by this new method in order to reestablish a baseline. Please contact MML to request this service. There will be no additional charge.

Uroporphyrinogen III Synthase Test Changes

Following a normal value study, the reference values for [Uroporphyrinogen III Synthase \(Co-Synthase\)\(Upg III S\), Erythrocytes #80288](#) have been adjusted. The analytic time has also changed, increasing from 2 days to 7 days with the implementation of a confirmatory step.

Over 99% of the orders for this test are made in error. Upg III S identifies patients with an extremely rare, congenital condition that becomes apparent in early infancy. Most of the orders for this test are actually submitted to rule out acute intermittent porphyria (AIP). AIP is much more common and typically onsets during puberty or later. The correct test to order to rule out AIP is [Aminolevulinic Acid Dehydratase \(ALA-D\) and Porphobilinogen Deaminase \(PBG-D\)\(Uroporphyrinogen Synthase \[Upg-S\]\), Erythrocytes, #9625](#).

To reduce the number of inappropriate orders for this test, MML is contacting the ordering physician when a request for this test is received. The physician is contacted by fax and asked to confirm via fax or telephone that this is the correct test. If MML does not receive confirmation, the physician is telephoned.

New Reference Value:

≥ 75 Relative Units

Previous Reference Value:

≥ 40 Relative Units

Ask



US

Q: What is *Staphylococcus lugdunensis* and why is it important to test for it?

A: *Staphylococcus lugdunensis* is increasingly identified as a cause of serious infections. Recently, Mayo reported two distinct clinical situations where the identification of *S lugdunensis* infection is critical: staphylococcal endocarditis and staphylococcal prosthetic joint infection (PJI).^{1,2} To avert negative outcomes, physicians and laboratorians must be familiar with this organism and initiate a rapid approach to verify infection by *S lugdunensis*.

Staphylococci are gram positive, spherical (cocci) bacteria that cause 20-35% of the cases of native valve infective endocarditis in non-intravenous drug users.² *Staphylococcus aureus* organisms are coagulase-positive (ie, the organisms produce coagulase, an enzyme that binds plasma fibrinogen resulting in clot formation). *S aureus* is the primary coagulase-positive species found in native valve infective endocarditis.²

S lugdunensis and *Staphylococcus epidermidis* are coagulase-negative staphylococci. While *S epidermidis* is the most frequently seen coagulase-negative species in infections of prosthetic joints and native heart valves, *S lugdunensis* can also cause these infections.² *S lugdunensis* is more aggressive and associated with higher mortality than *S epidermidis*.² It is therefore important to quickly identify which of these infecting organisms are present. Identification of *S lugdunensis* can be a problem as some isolates of *S lugdunensis* produce a clumping factor that can result in a positive slide (short) coagulase test, leading to misidentification as *S aureus*.^{1,2} The tube coagulase (long) test should be used to confirm a positive slide coagulase test.¹

Presence of the *mecA* gene is considered the gold standard for identifying methicillin (oxacillin) resistance. In a recent study at Mayo, 40 isolates of *S lugdunensis* were evaluated for oxacillin resistance. (Poster presentation—American Society for Microbiology, 2001) Because the *mecA* gene was not detected in any of the isolates in the study, *S lugdunensis* would be considered susceptible to oxacillin. However, as defined by the National Committee for Clinical Laboratory Standards (NCCLS), the minimum inhibitory concentration (MIC) for coagulase-negative staphylococci to be classified as resistant to oxacillin is $>0.25 \mu\text{g}/\text{mL}$. Of the 40 *S lugdunensis* isolates tested, 35 had MICs high enough to be reported as oxacillin resistant.

The NCCLS recommendations for oxacillin breakpoints for coagulase-negative staphylococci may not be appropriate for *S lugdunensis*.² When the results of the Mayo study are interpreted using the *S aureus* breakpoints (resistance defined as oxacillin MIC $>2 \mu\text{g}/\text{mL}$), only 2 of the 40 *S lugdunensis* isolates would be identified as oxacillin resistant.

When a laboratory detects the presence of coagulase-negative staphylococci, the investigation must go beyond an assumption of contamination and rule out or document a true coagulase-negative staphylococcal infection; the identification of the staphylococcus as *S lugdunensis* can be helpful in this situation. The current process at Mayo is to rule out *S lugdunensis* whenever coagulase-negative staphylococci are isolated from sterile body sites specimens.

MML recommends staphylococcal species identification by [Organism Referred for Identification, Aerobic Bacteria #9221](#). Depending on the organism submitted (staphylococcal or otherwise), 1 or more of the following methods are used: conventional biochemical analysis, fluorescent antibody staining, carbon source utilization, and sequencing nucleic acids of 16S ribosomal RNA (rRNA). When requesting susceptibility studies prior to initiating or changing treatment, MML recommends [Antimicrobial Susceptibility, Aerobic Bacteria \(MIC\) #8073](#).

References

1. Sampathkumar P, Osmon DR, Cockerill FR III: Prosthetic Joint Infection Due to *Staphylococcus lugdunensis*. *Mayo Clinic Proceedings* 75:511-512, 2000
2. Patel R, Piper KE, Rouse MS, Uhl JR, Cockerill FR, Steckelberg JM. Frequency of isolation of *Staphylococcus lugdunensis* among staphylococcal isolates causing endocarditis: a 20-year experience. *Journal of Clinical Microbiology* 38:4262-3, 2000

Abstracts of Interest

A Long-term Retrospective Study of Young Women With Essential Thrombocythemia

Ayalew Tefferi, MD; Rafael Fonseca, MD; Denise L. Pereira, MD; and H. Clark Hoagland, MD

- **Objective:** Objective: To describe presenting clinical manifestations, long-term disease complications, prognostic indicators, and outcome of pregnancy for women younger than 50 years with essential thrombocythemia.
- **Patients and Methods:** We retrospectively reviewed the records of all patients with essential thrombocythemia evaluated at Mayo Clinic, Rochester, Minn, between 1969 and 1991 and identified 74 young women (median age, 35 years; range, 18-48 years) with essential thrombocythemia. The diagnosis was based on previously established criteria. Median follow-up was 9.2 years (range, 0.2-26.2 years).
- **Results:** Overall survival was similar to that of an age- and sex-matched control population. Thrombotic events (except superficial thrombophlebitis) occurred at and after diagnosis in 11 patients (15%) and 13 patients (18%), respectively. A history of thrombosis at diagnosis was significantly associated with recurrent thrombosis ($P=.03$). A platelet count higher than $1500 \times 10^9/L$ at diagnosis was significantly associated with gastrointestinal tract bleeding and subsequent development of venous (but not arterial) thrombosis ($P=.04$). Major hemorrhagic events occurred in only 3 patients (4%) after diagnosis. Only 1 patient developed acute leukemia. Thirty-four pregnancies occurred in 18 patients. Of these, 17 (50%) resulted in live births. Of the 17 patients with unsuccessful pregnancies, 14 had spontaneous abortions, 1 had an ectopic pregnancy, and 2 had elective abortions. Preconception platelet count, thrombotic history, or specific therapy was not useful in predicting pregnancy outcome.
- **Conclusion:** Young women with essential thrombocythemia can expect long survival with a low incidence of life-threatening thrombohemorrhagic complications or acute leukemia. There is an increased incidence of first-trimester miscarriages that may not be influenced by specific therapy.

Mayo Clinic Proceedings 76:22-28, 2001

Meeting Calendar

Interactive Satellite Program . . .

October 23, 2001

Advances in Wound Healing: Diabetes and Geriatrics
Steven J. Kavros, D.P.M.

November 6, 2001

An Update on HIV
Zelalem Temesgen, M.D.

Upcoming Education Conferences . . .

September 12-13, 2001

Integration Through Community Laboratory Insourcing
Chateau Sonesta, New Orleans, Louisiana
Course Director: Rodney Forsman
Presented by Mayo Medical Laboratories

September 28-29, 2001

Practical Spirometry
Chicago, Illinois
Course Director: Paul Scanlon, M.D.
Presented by Mayo Pulmonary Services

November 1-2, 2001

Practical Spirometry
Rochester, Minnesota
Course Director: Paul Scanlon, M.D.
Presented by Mayo Pulmonary Services



For additional information regarding the above programs, please contact the Mayo Reference Services Education Office at 1-800-533-1710.

Change in Chlamydia Screening Recommendations

Chlamydia trachomatis infection is the most common reportable sexually transmitted disease (STD) in the United States. The infection rate with this organism among 15-19 year olds is 6-fold higher than the rate among 25-34 year olds. The national infection rate for *C trachomatis* is estimated to be 3 million cases annually. Left untreated, complications such as pelvic inflammatory disease (in females) and infertility (in both sexes) may occur. Pregnant women infected with *C trachomatis* may experience preterm delivery and postpartum endometritis, while their infants are at risk of perinatal transmission.

Recently, the United States Preventive Services Task Force published new *C trachomatis* screening recommendations. (U.S. Preventive Services Task Force Update: Screening Chlamydial Infection. 2001 Release. Available from <http://www.ahcpr.gov/clinic/uspstf/uspshlm.htm>) The task force noted that chlamydial infections are asymptomatic in a large percentage of infected individuals. The panel determined that routine screening for chlamydial infection in women who are sexually active and 25 years or younger reduces the incidence of pelvic inflammatory disease (PID). Based in part on the results of a study that demonstrated a greater than 50% reduction in the incidence of PID (study group infection prevalence of 7%), the panel strongly recommends routine screening of all sexually active females in this age group. Additionally, the panel recommends routine screening of asymptomatic pregnant women (age 25 years or younger) and others determined to be at increased risk.

MML utilizes a ligase chain reaction method for detection of *C trachomatis*, [Chlamydia trachomatis Detection by Nucleic Acid Amplification #81665](#). The ligase chain reaction (LCR, Abbott Laboratories) became available in 1996 for the laboratory diagnosis of both *C trachomatis* and *Neisseria gonorrhoeae* infections. Separate tests for both organisms can be performed using a single swab (endocervix or urethra) or a urine specimen. Both tests are FDA approved. The performance characteristics of LCR for *C trachomatis* exceed culture results. The sensitivity for the detection of *C trachomatis* in urine specimens from males and females is 94% compared with 60% for culture techniques.

Communiqué

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