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December 2009

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UMassMemorial

Laboratories

New Offerings for Drugs of Abuse [DOA] Testing in Urine

Our Clinical Toxicology Laboratory has added new drugs to its testing menu. These drugs are offered as qualitative screens, providing presumptive positive results. [When necessary confirmation testing should be conducted]. The testing is conducted on our Olympus AU400e instruments utilizing EMIT® technology. The system can be utilized for routine or STAT samples.

EMIT assays are based on competition between drug in the urine specimen and drug labeled with an enzyme, such as glucose-6-phosphate dehydrogenase (G6PDH), for antibody binding sites. Enzyme activity decreases upon binding to the antibody, so the drug concentration in the specimen can be measured in terms of enzyme activity. Active enzyme converts nicotinamide adenine dinucleotide (NAD) to NADH, resulting in an absorbance change that is measured spectrophotometrically. Endogenous serum G6PDH does not interfere because the coenzyme NAD functions only with the bacterial (*Leuconostoc mesenteroides*) enzyme employed in this assay.

The new tests include a panel [DOA5] which mimics the federal DHHS so-called “NIDA” five drugs of abuse, namely amphetamines, cannabinoids, cocaine metabolite, opiates and phencyclidine [PCP]. In addition single drug tests are available for Ecstasy [MDMA], Methaqualone, Heroin metabolite [6-acetylmorphine], Lysergic Acid Diethylamide [LSD], Oxycodone and Buprenorphine.



Photo: Kevin Vance

UMass Memorial Medical Center Laboratories

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Mnemonic	Test	Target Drug	Cutoff Concentration ng/mL
DOA5	Amphetamines	Methamphetamine	1000
	Cannabinoids	Carboxy-THC	50
	Cocaine metabolite	Benzoylcegonine	300
	Phencyclidine	Phencyclidin	25
	Opiates	Morphine	300
UMDMA	Ecstasy	MDMA	500
UOXY	Oxycodone	Oxycodone	100
UHERMTB	Heroin Metabolite	6-Acetylmorphine	10
UBUP	Buprenorphine	Buprenorphine	20
LSDUS	LSD	d-LSD	0.5
MAHAQUR	Methaqualone	Methaqualone	300

Cross Reactivity:

The specificity of these tests for the drug of interest varies by vendor. It may also vary by specific lots as vendors change antibodies and other components within the reagent kits. We are currently using Microgenics Corporation CEDIA® and DRI® reagents. The following are therefore, general guidelines.

DOA: In this panel a cutoff concentration of 300 ng/mL for opiates is utilized. This is lower than the federal DHHS cutoff of 2000 ng/mL. The reason for the difference is the federal cutoff is focused on workplace drug testing only and a desire to detect heroin users rather than individuals ingesting opioids legitimately. The opiate assay in this panel will not detect opioids such as oxycodone or hydrocodone until the urine concentrations are significant [16,000 and 1700 ng/mL, respectively]. This assay should also not be utilized for the detection of methadone, meperidine, oxymorphone, levorphanol, hydromorphone, fentanyl, buprenorphine, propoxyphene and tramadol.

The amphetamine assay demonstrates 100% cross reactivity with d-amphetamine and d-methamphetamine; 77% with MDMA and 40% with MDA. Therefore, if Ecstasy use is suspected, order UMDMA.

UMDMA: Approximately 56% cross reactivity was observed with MDA, a MDMA metabolite; 83% with MDEA, and <30% with para-methoxy amphetamine [PMA] and para-methoxymethamphetamine [PMMA]. Amphetamine, methamphetamine, pseudoephedrine, ephedrine, phentermine and phenylpropanolamine demonstrated <1% cross reactivity with this assay.



UOXY: This assay demonstrates approximately 100% cross reactivity with oxymorphone but not the nor-metabolites. Low cross reactivity has been reported with other opioids.¹

UHERMTB: This assay has high specificity. In one study² a 98% confirmation rate was obtained utilizing a screening cutoff of 10 ng/mL and a confirmation cutoff of 5 ng/mL [GCMS] [n=525]. 6-AM concentrations in these specimens ranged 5-16,923 ng/mL. All confirmed specimens also contained morphine [8-222,427 ng/mL]. **False positive** results may be obtained with high concentrations of other opioids [morphine \geq 10,000; oxycodone \geq 61,000 ng/mL] and structurally related compounds [pentazocine \geq 35,000 ng/mL]. Note: Specificity information provided in the manufacturer package inserts is not always current or accurate. For example, the package insert for this assay states an oxycodone concentration of 400,000 ng/mL produced a negative result, however, positive results have been obtained with oxycodone concentrations of 61,000 ng/mL.²

UBUP: This assay demonstrates approximately 98% cross reactivity with buprenorphine 3- β -glucuronide but <0.5% with norbuprenorphine or its conjugate.

LSDUS: This test does not demonstrate any cross reactivity with lysergic acid or iso-LSD.

MAHAQUR: This assay demonstrates low cross reactivity with hydroxy metabolites of methaqualone.

Specimen requirements:	
Collect:	random urine
Transport:	room temperature or refrigerated [refrigeration preferred if suspected heroin use to prevent loss of 6-AM]
Minimum Volume:	15 ml urine for panel 5 ml for single drug



Availability:

These tests will be orderable December 7, 2009

The future:

We are in the process of compiling a screening Pain Panel which maybe helpful for clients in Addiction Medicine/ Methadone Maintenance/Pain Management. Additional single drug tests should be available from the manufacturer within 12 months [such as fentanyl, tramadol]. If you have suggestions of what you would like to see in a panel please email Amanda.jenkins@umassmemorial.org.

For additional Information:

1. Jenkins, A.J., Poirier, J.G., and Juhascik, M.P.: "Cross Reactivity of Naloxone with Oxycodone Immunoassays: Implications for individuals taking Suboxone®". *Clin. Chem.* 55:7 doi:10.1373/clinchem. 2009. 125096. April 30, 2009 and *Clin Chem* Jul 2009 55:1434-1436.
2. Jenkins, A.J., Lavins, E.S., and Snyder, A.: Evaluation of the Cedia® heroin metabolite (6-AM) immunoassay with urine specimens from a criminal justice drug testing program. *J. Anal. Toxicol.* 28: 201-204 (2005).
3. Assay Package Inserts. Microgenics Corporation, Fremont, CA 94538. 1-800-232- 3342.

If you have questions, comments or suggestions, please contact:

Dr. Amanda Jenkins, Director of Clinical and Forensic Toxicology at 508-442-9074 or via e-mail at Amanda.Jenkins@umassmemorial.org or
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DNA sequence analysis for Ellis-van Creveld Syndrome and Weyers Acrofacial Dysostosis

The UMass Memorial Molecular Diagnostic Laboratory is offering sequencing of the EVC and EVC2 genes to detect mutations that cause Ellis-van Creveld Syndrome and Weyers acrofacial dysostosis.

Background: Ellis-van Creveld syndrome, EvC, is an inherited, autosomal recessive, chondrodysplastic dwarfism, frequently associated with congenital heart disease (60% of patients). The consistently observed clinical manifestations include short-limbed disproportionate dwarfism, shortened ribs, postaxial polydactyly and dysplastic nails and teeth. EvC syndrome is a genetically heterogeneous condition in which homozygous or compound heterozygous mutations in either the EVC or EVC2 genes can result in the disease.

EvC syndrome occurs world-wide, with cases reported in Mexico, India, Great Britain, South America, North America, Europe, in Asian and Ashkenazi population. Ellis-van Creveld syndrome is the most frequent dwarfism found in the Old Order Amish of Lancaster County, Pennsylvania (approximately 1 per 5000 births).

Sequence analysis of the EVC and EVC2 genes can confirm a clinical diagnosis of EvC, as well as to help differentiate EvC from Weyers acrofacial dysostosis, an autosomal dominant disorder sharing the hallmark manifestations of EvC, but with a less severe presentation and without cardiac abnormalities. Mutation identification can also provide useful information for carrier screening and prenatal diagnosis.

Methodology: Sequencing of genomic DNA is performed using polymerase chain reaction (PCR) amplification of 21 coding exons of the EVC gene and 22 coding exons of the EVC2 gene, including exon-intron junctions, followed by DNA sequence analysis using capillary electrophoresis.

This analysis of the EVC and EVC2 genes may result in the identification and reporting of genetic variants of uncertain clinical significance.

Requirements: The UMass Memorial Molecular Diagnostics Test Requisition and Consent should be used and sent with the sample. Copies of this requisition may be obtained from Customer Service at 800-476-4431. Specimen requirement is 3ml of blood in a lavender top (EDTA) tube, sent to the laboratory either at room temperature or refrigerated (not frozen).

If you have comments, questions, or suggestions, please contact:

Dr. Edward Ginns at 508-856-8134, or via email at Edward.Ginns@umassmed.edu or

Dr. Marzena Galdzicka at 508-856-4384 or via email at Marzena.Galdzicka@umassmed.edu



References:

1. Galdzicka M., S. Patnala, M.G. Hirshman, J-F. Cai., H. Nitowsky, J.A Egeland and E.I. Ginns. "A new gene, EVC2, is mutated in Ellis-van Creveld Syndrome". *Molecular Genetics and Metabolism* 77: 291-295, 2002.
2. Galdzicka M, Jabs E.W, Ginns E.I. "An EVC2 mutation is associated with Weyers acrofacial dysostosis and severe EvC syndrome in the same pedigree". *Am. J. Hum. Genet.* 1763, 2006.
3. Galdzicka M, England JA., Ginns EI. "EVC and EVC2 and Ellis-van Creveld Syndrome". 2nd edition of CJ Epstein, RP Erickson, A Wynshaw-Boris (eds.) *Inborn Errors of Development*; Oxford University Press, New York, 2008.
4. Ruiz-Perez VL, Ide SE, Strom TM, Lorenz B, Wilson D, Woods K, King L, Francomano C, Freisinger P, Spranger S, Marino B, Dallapiccola B, Wright M, Meitinger T, Polymeropoulos MH, Goodship J. "Mutations in a new gene in Ellis-van Creveld syndrome and Weyers acrofacial dysostosis". *Nat Genet.* 24(3):283-6, 2000. Erratum in: *Nat Genet.* 25(1):125, 2000.
5. Ruiz-Perez VL, Tompson SW, Blair HJ, Espinoza-Valdez C, Lapunzina P, Silva EO, Hamel B, Gibbs JL, Young ID, Wright MJ, Goodship JA. "Mutations in two nonhomologous genes in a head-to-head configuration cause Ellis-van Creveld syndrome". *Am J Hum Genet.* ;72(3):728-32, 2003.

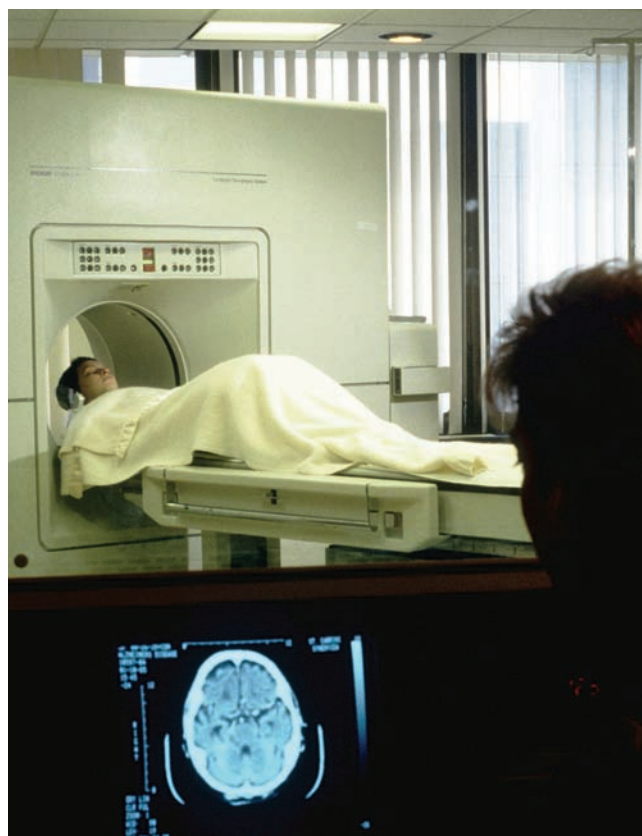
Arthritis



- I. **Definition.** Arthritis is defined by **inflammation of one or more joints** and is usually associated with pain and structural changes in the affected joints. The clinician should be aware of signs and symptoms that may be suggestive of more serious underlying pathology.
- II. **Overview.** There are four basic principles when considering the clinical approach to arthritis.
 - A. The **history** and **physical examination**, combined with **synovial fluid analysis** and **radiograph studies** when appropriate, are the most important components of the evaluation.
 - B. **Joint symptomatology** can be caused by disorders that do not primarily involve the joint. Distinguishing articular from periarticular, soft tissue, neurologic, psychogenic, and referred symptoms is central to the initial evaluation.
 - C. The clinician must **differentiate localized processes from systemic disease**.
 - D. **Rheumatologic disorders**, including monarticular and polyarticular arthritis, are usually diagnosed on the basis of the complete clinical picture, and seldom on the basis of a single diagnostic test.

III. Differential diagnosis

- A. **Monarticular and polyarticular arthritis** can be broadly separated into structural and inflammatory disorders.
 1. **Structural disorders** are characterized by mechanical/ degenerative, metabolic, or traumatic disruption of the joint. Osteoarthritis and meniscal and ligamentous injuries are common examples of mechanical/ degenerative conditions.
 2. **Inflammatory arthritis** is characterized by inflammation of the synovial lining and may also involve the subsynovium or the synovial attachments to the joint capsule. These can be localized disorders, such as infection and crystal-induced disease [e.g., gout, calcium pyrophosphate disease (CPPD), hydroxyapatite], or systemic illnesses. In some of the systemic conditions, such as rheumatoid arthritis and seronegative spondyloarthropathies, the joint manifestations are a major part of the illness, whereas in other systemic conditions, such as systemic lupus erythematosus and sarcoidosis, the arthritic aspects of the disease usually have only a comparatively minor role in the disease process.



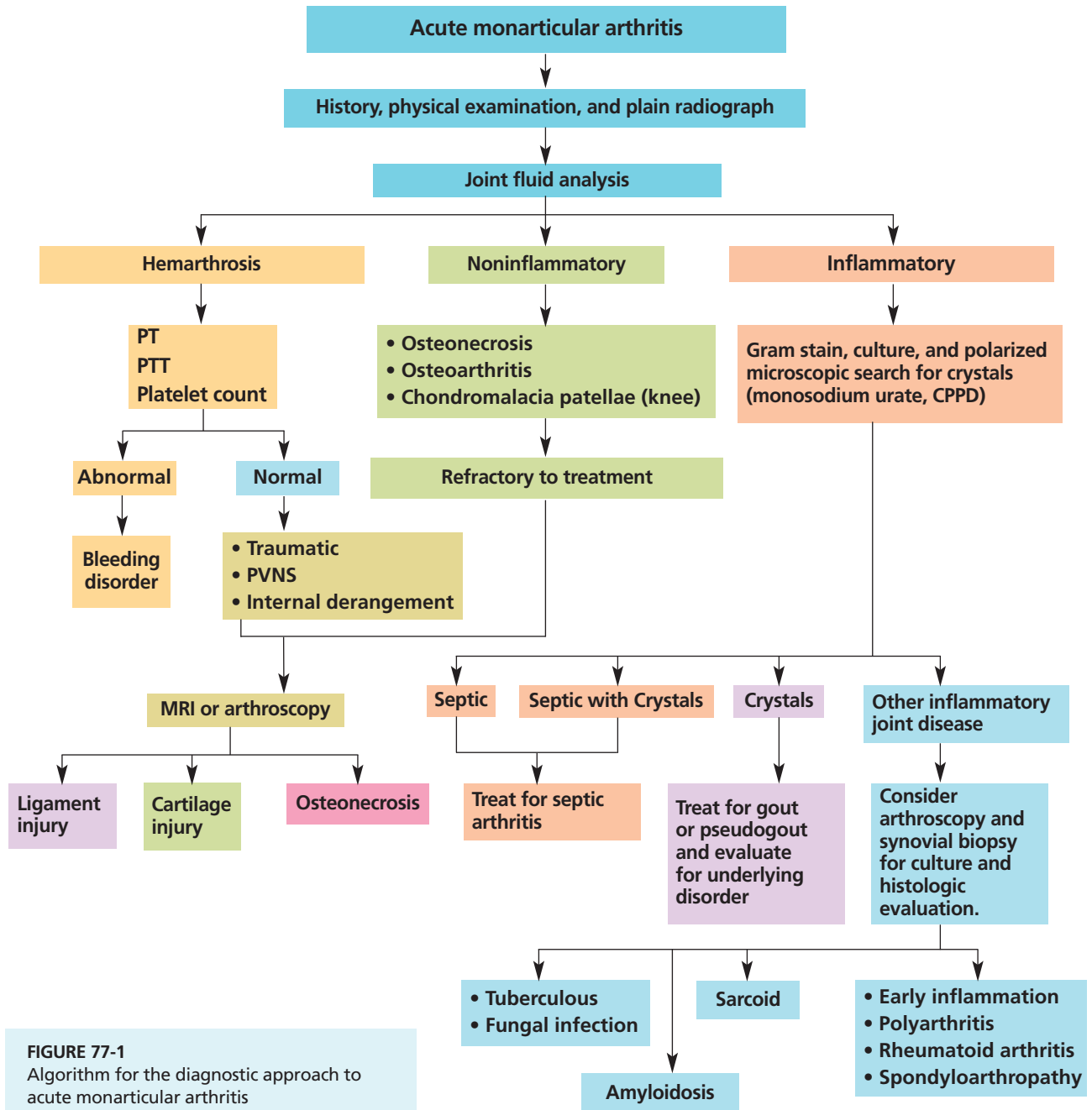
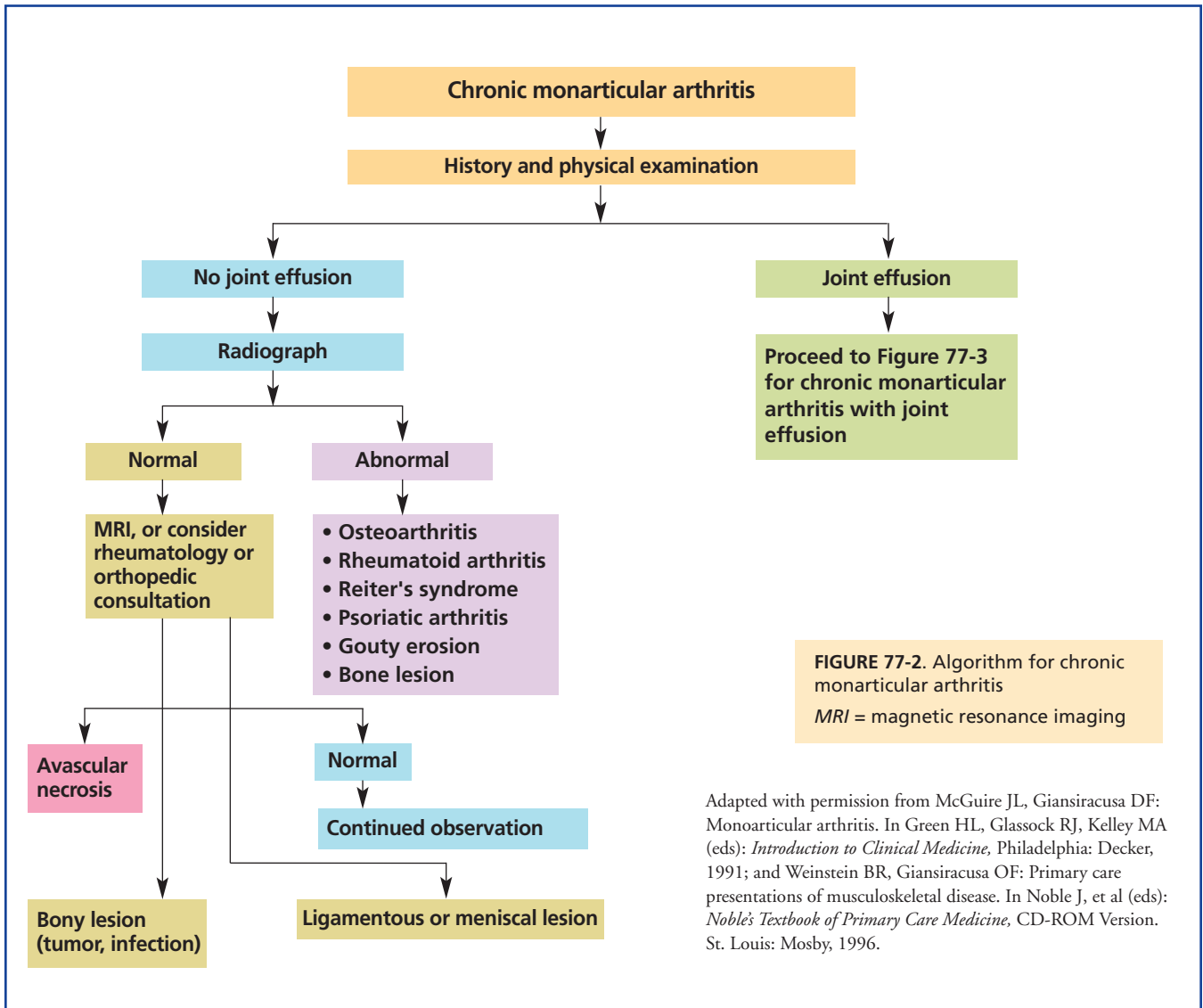


FIGURE 77-1
 Algorithm for the diagnostic approach to acute monarticular arthritis
 CPD = calcium pyrophosphate dihydrate
 MRI = magnetic resonance imaging
 PT = prothrombin time
 PTT = partial thromboplastin time
 PVNS = pigmented villonodular tenosynovitis

Adapted with permission from McGuire JL, Giansiracusa OF: Monoarticular arthritis. In Green HL, Glasscock RJ, Kelley MA (eds): *Introduction to Clinical Medicine*. Philadelphia: Decker, 1991; and Weinstein SR, Giansiracusa OF: *Primary care presentations of musculoskeletal disease*. In Noble J, et al (eds): *Noble's Textbook of Primary Care Medicine*, CD-ROM Version. 8t. Louis: Mosby, 1996.



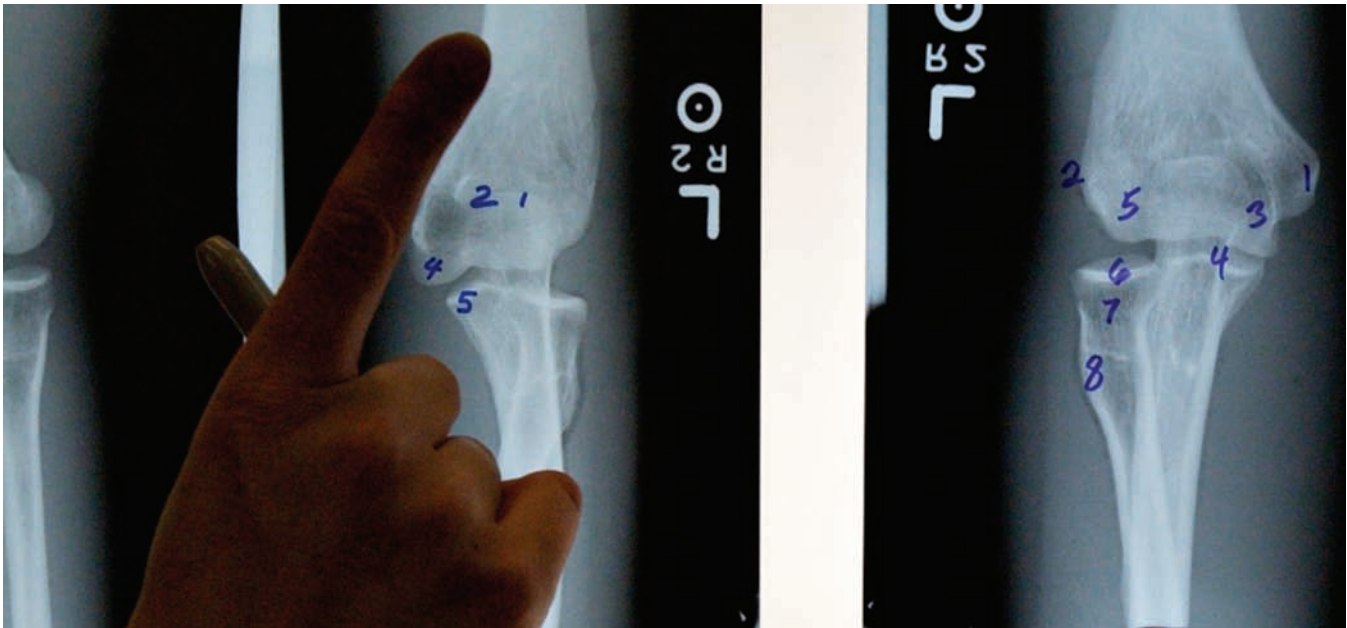
Joint effusion

↓

Proceed to Figure 77-3 for chronic monarticular arthritis with joint effusion

FIGURE 77-2. Algorithm for chronic monarticular arthritis
MRI = magnetic resonance imaging

Adapted with permission from McGuire JL, Giansiracusa DF: Monoarticular arthritis. In Green HL, Glasscock RJ, Kelley MA (eds): *Introduction to Clinical Medicine*, Philadelphia: Decker, 1991; and Weinstein BR, Giansiracusa OF: Primary care presentations of musculoskeletal disease. In Noble J, et al (eds): *Noble's Textbook of Primary Care Medicine*, CD-ROM Version. St. Louis: Mosby, 1996.



B. Acute monarticular arthritis (Figure 77-1)

1. **Differential diagnosis:** The differential diagnosis of acute monarticular arthritis is narrow.
 - a. Infectious arthritis, including causes such as staphylococcal, gono-coccal, and Gram-negative organisms (immunosuppressed patients)
 - b. Crystal-induced diseases
 - c. Traumatic arthritis
 - d. Chronic monarticular or polyarticular arthritis with acute presentation
2. **Synovial fluid:** In addition to the history and physical examination, analysis of the synovial fluid is usually the most helpful diagnostic test (see VB).

C. Chronic monarticular arthritis (Figures 77-2 and 77-3).

The differential diagnosis for chronic monarticular arthritis is broad and includes most of the disorders that cause acute monarticular and polyarticular arthritis. The initial approach consists of trying to distinguish between **inflammatory and noninflammatory conditions**.

1. **Radiographic studies** play an important role.
2. **Magnetic resonance imaging (MRI) and arthroscopy** may be helpful modalities when the diagnosis remains elusive.

D. Polyarticular arthritis (Figure 77-4)

1. **Noninflammatory causes:** The principle noninflammatory causes include osteoarthritis (primary and secondary) and avascular necrosis. Osteoarthritis can occur secondary to other disorders, including trauma, metabolic problems (e.g., hemochromatosis, hypothyroidism), and CPPD.

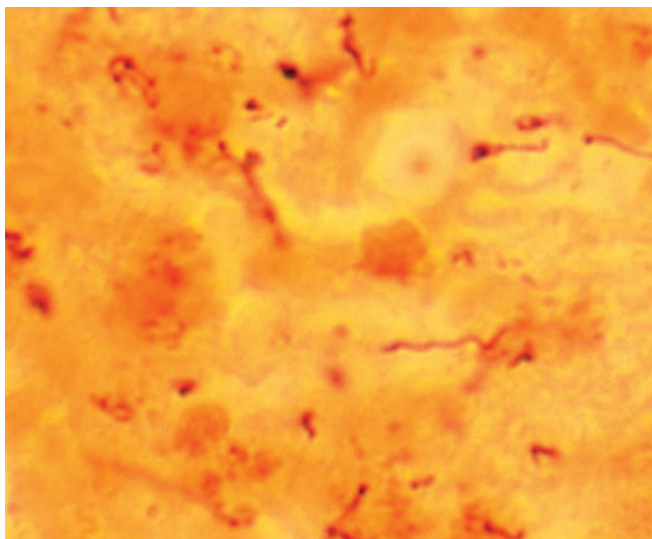


Image courtesy of Department of Health and Human Services Public Health Image Library (PHIL)

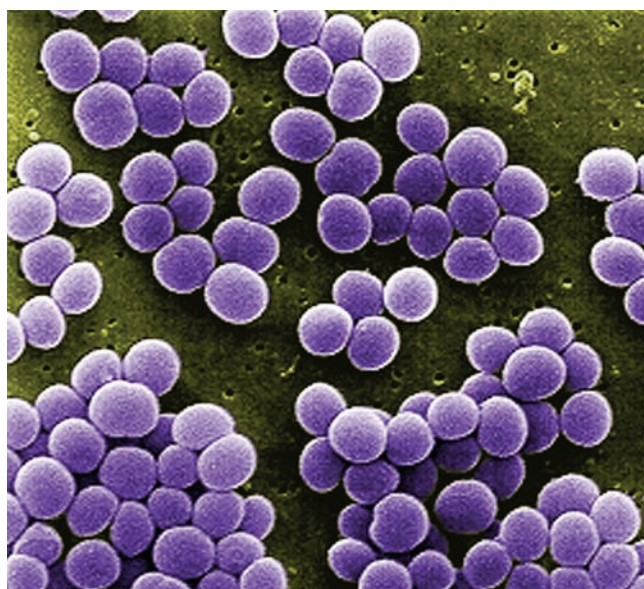


Image courtesy of Department of Health and Human Services Public Health Image Library (PHIL)

2. **Inflammatory causes:** Inflammatory polyarthritides include diseases that tend to affect many joints, those that involve just a few joints (pauciarticular), and those that have a special predilection to also involve the axial skeleton.

IV. Clinical Evaluation

A. History

1. Differentiate **articular from nonarticular disorders**.
2. Elicit the **acuity and tempo of symptoms** by inquiring about onset, duration, provoking and relieving factors, relationship to overuse or previous trauma, and involvement of joints in a migratory or additive fashion. Elicit a history of previous episodes.
3. Assess for **symptoms of inflammation**. The presence of “gelling” or morning stiffness, in which periods of inactivity are associated with stiffness lasting longer than 30 minutes, is characteristic of inflammatory joint problems.
4. Assess for **systemic illnesses**, especially for constitutional symptoms. The review of systems should pay particular attention to the following:
 - a. High-risk behaviors (including HN or hepatitis risk factors)
 - b. Travel to tick-inhabited regions
 - c. The presence of rashes
 - d. History of sexually transmitted disease (STD)
 - e. Prodromal symptoms (e.g., diarrhea, viral symptoms)
 - f. Ocular symptoms
 - g. Xerostomia
 - h. Drug use

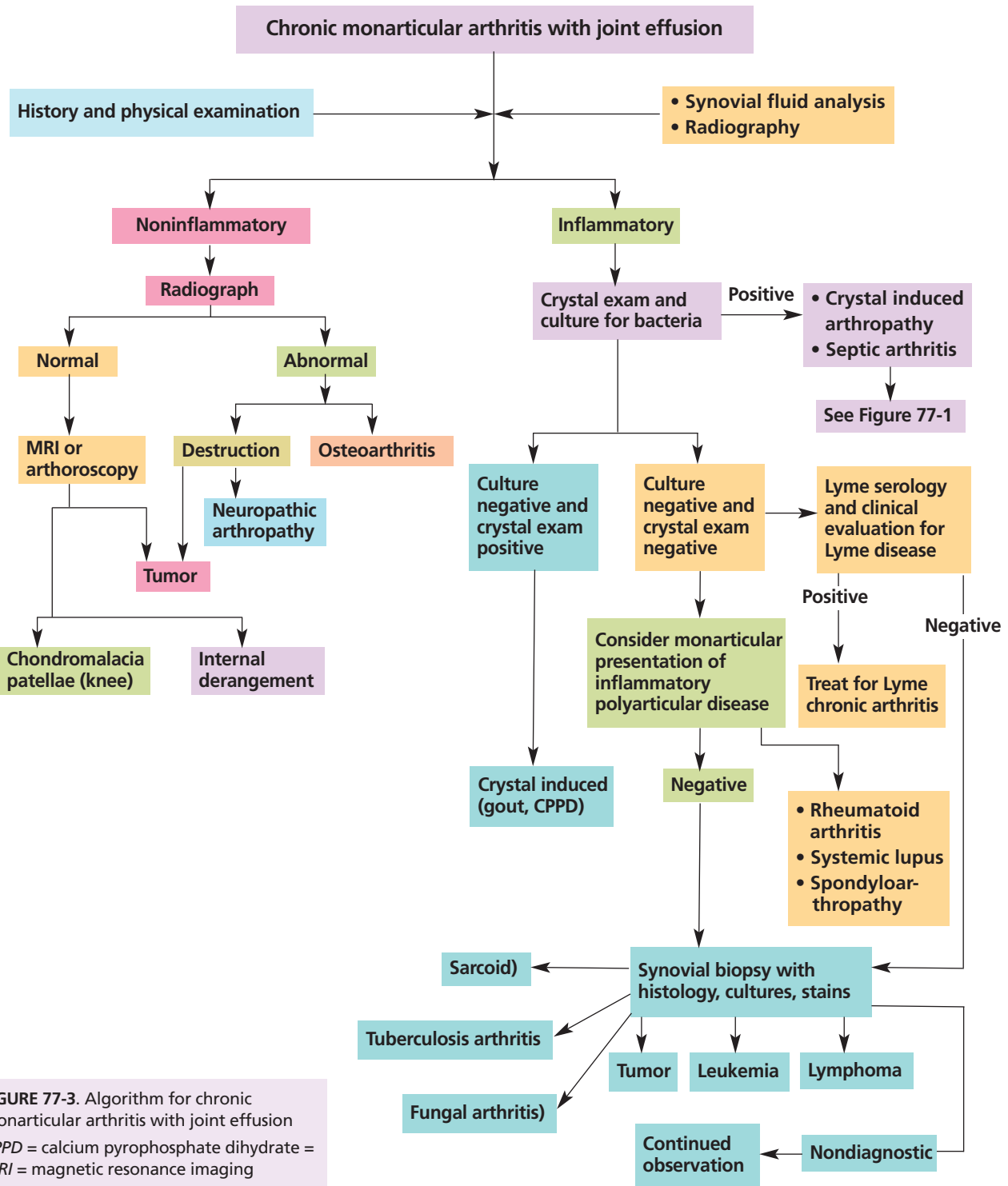
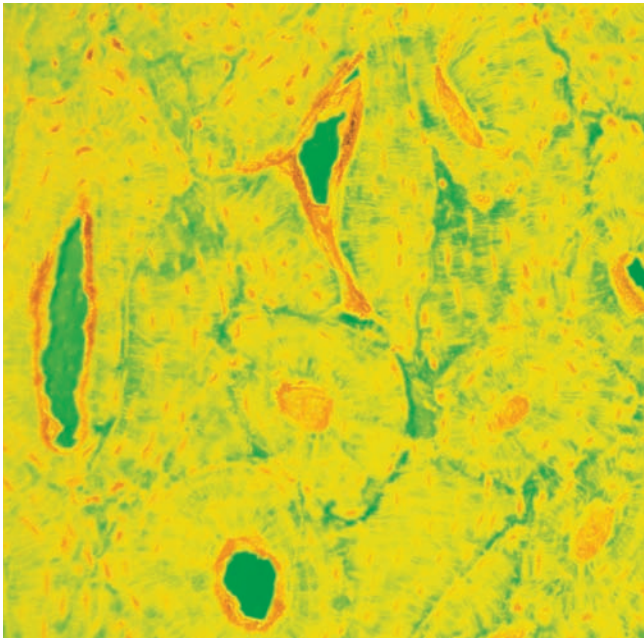


FIGURE 77-3. Algorithm for chronic monarticular arthritis with joint effusion
 CPPD = calcium pyrophosphate dihydrate =
 MRI = magnetic resonance imaging

Adapted with permission from McGuire JL, Giansiracusa OF: Monoarticular arthritis. In Green HL, Glasscock RJ, Kelley MA (eds): *Introduction to Clinical Medicine*. Philadelphia: Decker, 1991; and Weinstein BR, Giansiracusa OF: Primary care presentations of musculoskeletal disease. In Noble J, et al (eds): *Noble's Textbook of Primary Care Medicine*, CD-ROM Version. St. Louis: Mosby, 1996.



5. **Family history:** There are genetic and familial predispositions to a number of arthritic disorders, including rheumatoid arthritis, systemic lupus erythematosus, gout, osteoarthritis, and seronegative spondyloarthropathies.

B. Physical examination

1. **General principle:** When examining an affected joint, the contralateral side of the patient can serve as a reference point.

2. **Nonmusculoskeletal examination:** Look for clues to systemic illness (especially dermatologic, ocular, and neurologic), and assess for lymphadenopathy.

3. Musculoskeletal examination

a. **Inspection:** Assess posture, gait, and movement. Look for signs of inflammation by checking for erythema, warmth, and swelling. Joint deformity is associated with destructive changes within the joint. Active range of motion is limited with both articular and periarticular processes but is helpful in assessing function and mobility.

b. **Palpation** of the articular and periarticular structures can usually help differentiate joint effusions, synovial thickening, bony changes, enthesitis (i.e., inflammation at sites of ligamentous and tendinous attachments to bone, a characteristic finding in seronegative spondylarthropathies), and periarticular/soft-tissue processes. Palpating areas of maximal tenderness also helps localize the problem. The presence of crepitus suggests destructive changes within the joint.

c. **Pain with passive range of motion** characterizes articular problems, whereas in bursa and tendon disorders, pain is more prominent with active range of motion.

d. **Provocative maneuvers** that stress tendons, ligaments, and the joints for stability are useful localizing techniques and can help establish a diagnosis.

V. Diagnostic Testing

A. **Laboratory testing.** In the patient where the diagnosis remains uncertain following clinical assessment, laboratory testing may be advised. These tests will have the greatest utility in patients where a systemic illness is being considered. In such instances, a complete blood count (CBC), renal functions, urinalysis, liver chemistries, and serum calcium are considered initial studies. The composite clinical picture should guide further assessment, including rheumatologic testing [erythrocyte sedimentation rate (ESR), C-reactive protein, antibody testing, rheumatoid factor (RF), serum protein electrophoresis, Lyme titer], imaging studies (MRI, plain films of sacroiliac joints, chest radiography, and nuclear imaging), and use of the microbiology laboratory.

B. **Synovial fluid analysis** constitutes the basic initial laboratory evaluation when a joint effusion is identified:



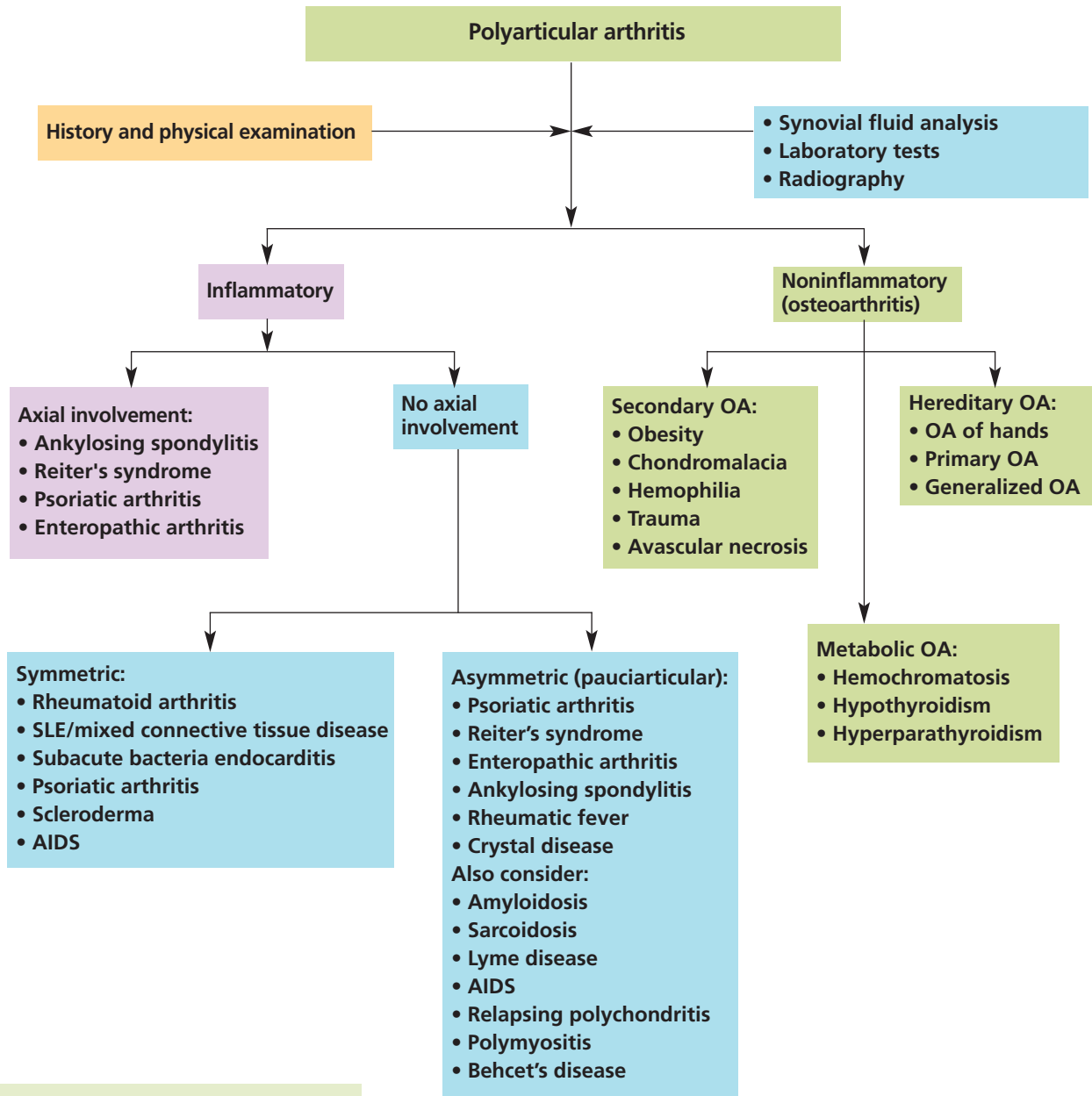


FIGURE 77-4. Differential diagnoses of polyarticular arthritis
 OA = osteoarthritis
 SLE = systemic lupus erythematosus

(Adapted with permission from Doud D: Polyarticular arthritis. In Greene HL, et al (eds): *Decision Making in Medicine*, St. Louis: Mosby, 1993; and Weinstein BR, Giansiracusa OF: Primary care presentations of musculoskeletal disease. In Noble J, et al (eds): *Noble's Textbook of Primary Care Medicine*, CD-ROM Version. St. Louis: Mosby, 1996.)

1. **Normal joint fluid** is transparent, colorless, highly viscous, and usually has < 200 white blood cells (WBCs)/mm³, with less than 25% polymorphonuclear leukocytes.
2. In **noninflammatory disorders**, such as osteoarthritis, the fluid is yellow, clear, highly viscous, and has WBC counts between 200 and 2,000, with less than 25% polymorphonuclear leukocytes.
3. In **inflammatory arthritides**, such as rheumatoid arthritis, the fluid may be translucent or opaque, yellow in color, and have reduced viscosity. The WBC count is usually between 2,000 and 100,000, with greater than 50% polymorphonuclear leukocytes. Joints that are infected with virulent bacterial organisms may

have even more inflammatory findings, with WBC counts > 100,000, mostly polymorphonuclear leukocytes.

4. **Hemorrhagic effusions** are seen with trauma, coagulopathies, neoplasms, severe osteoarthritis, and pigmented villonodular synovitis.

Reference: Bruce Weinstein and David Giansiracusa, *Guide to Diagnostic Testing*, pp 467–474

If you have questions, comments or suggestions, please contact:
Dr. L. Michael Snyder, Chairman of Hospital Laboratories
at 508-442-9280



Mass Spectrometric Applications in Clinical Diagnostics



Highlights of the Educational Symposium

UMass Memorial Clinical Laboratories first full day educational symposium, co sponsored by Waters Corporation, was held on November 9, 2009 at Amphitheater-1 at UMass Medical School, Worcester, MA was well attended by approximately 100 registrants from the New England region. Mass Spectrometric applications are fast becoming an indispensable field for medical and diagnostic professionals. A better understanding of the MS technology and its potential can help us all in our efforts in improving clinical diagnostics for the ultimate goal of better diagnosis, management and treatment of various diseases. In this symposium a group of well known experts in clinical, forensic and molecular areas, who used this technology and developed various applications, presented their experiences and recommendations and future directions.

The symposium started with a welcome address by **Dr. Guy Vallaro**, Vice President of clinical labs at **UMass Memorial Medical Center** at Worcester, MA. He reviewed briefly the relevance of Mass Spectrometric applications in different areas of clinical diagnostics and growing importance in the field. He concluded with certain prognostications about the future directions of mass spectrometric applications including standardization, hardware and various potential applications in toxicology, TDM, genetic and proteomic fields.

This was followed by **Dr. Neal Lindeman** from **Brigham and Women's Hospital, Harvard Medical School**, Boston, MA on the basics of implementation of mass spectrometry for clinical monitoring in a naïve laboratory. He discussed various details about different types of mass spectrometry, its components, principles, challenges from both technical and information systems from a clinical lab point of view. He also discussed the advantages of mass spectrometry over immunoassay for immunosuppressant monitoring.

The next speaker that followed was this was **Dr. Mat Juhascik** from the forensic toxicology laboratory at **UMass Memorial Hospital Laboratories** in Worcester, MA. He discussed in detail method development, appropriate method validation, and trouble shooting techniques of an LCMS assay using Cannabinoids test in the post mortem blood as an example. He discussed various strategies for developing a new assay from chromatography through extraction and different troubleshooting techniques for development and validation of mass spectrometric methods.

Dr. Thomas G. Rosano from **Albany Medical College and Hospital**, NY, presented an interesting discovery of value of glycolic acid measurement by GC-MS in Ethylene glycol poisoning. He discussed various medical examiner and clinical case studies in ethylene glycol poisoning and concluded that measurement of glycolic acid provides further metabolic confirmation of poisoning, especially when extended time for metabolism results in low or undetected levels of ethylene glycol.

Dr. Marc Kellogg from **Children's Hospital** Boston, MA discussed the advantages of UPLC over HPLC in the clinical laboratory environment. He described how tandem mass spectrometry can be used to overcome some key issues associated with chromatographic separation of amino acids. He also discussed the major metabolites of caffeine and described how tandem mass spectrometry can be used for quantification of caffeine and its metabolites in saliva, blood and urine.

Dr. Joseph Orsini from Newborn Screening Program New York State Dept. of Health, Wadsworth Center, Albany presented an overview of Newborn Screening utilizing Tandem Mass Spectrometry. He discussed various amino acid, organic acid, fatty acid oxidation defects and Lysosomal storage disorders including Krabbe disease. He detailed how an enzyme assay can be performed using tandem MS in the newborn screening programs and discussed some of the ethical issues associated with screening.

Dr. Marzena Galdzicka from the Molecular Diagnostics Laboratory of UMass Medical School and the Hospital Laboratories presented various mass spectrometric applications in nucleic acid testing including SNP genotyping-mutation, polymorphism detection, copy number analyses, gene expression-transcriptional profiling, quantitative methylation analysis, molecular microbial testing and the feasibility of noninvasive Prenatal Diagnostics using circulating cell-free fetal nucleic acid from maternal serum or plasma.

For further information please contact

L.V. Rao, Ph.D, Director, Core Labs, Immunology and Clinical Research and Support at 774-442-9615 or via email at Lokinendi.Rao@umassmemorial.org

Dr. Sunny Tam from the Biochemistry and Molecular Pharmacology departments of UMass Medical School, Worcester, MA discussed aspects of Biomarker Discovery and Validation of Pre-term Amniotic Fluids Using Proteomic Technology. He presented the Obstacles for biological fluid proteomic analysis with some solutions as well as some of the proteomic identification techniques such as 2D gel or iTRAQ. Also presented was a premature birth study with amniotic fluids using iTRAQ proteomic Analysis and Biomarker discovery and validation of pre-term amniotic fluids.

Dr. John P. Shocker from the Metabolic Profiling Business Development at Waters Corporation, Milford, MA, presented problems encountered in the experimental design of a biomarker study and how to overcome them. He described in detail the major analytical challenges in the biomarker discovery process and how to select the correct analytical technique to meet these challenges. He discussed various computational methods in mining the complex data sets generated by modern analytical techniques. At the end he demonstrated a software he created to analyze various lipids from different populations to introduce the applications of personalized medicine.

Many thanks for Ms. Tammy Hicks at Water Corporation, MA and Ms. Deb Moiles and Ms. Deb Faryna for excellent various symposium arrangements and Ms. Angela Nardella for symposium pictures.





UMassMemorial

Laboratories

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UMass Memorial Laboratories has opened a Patient Service Center (phlebotomy draw station) at 354 Merrimack St., Entrance C, Suite 208 Lawrence, Massachusetts.

The vision of UMass Memorial Laboratories is:

- To be a leading provider of laboratory services throughout New England, meeting the needs of patients and providers in the region, and
- To be one of the top ten academic medical center-based laboratories in the United States



Lawrence PSC

**354 Merrimack Street, Entrance C, Suite 208
Lawrence, Massachusetts**

Lawrence PSC is located at 354 Merrimack St., Lawrence, Massachusetts.

The hours are Monday through Friday 8:00am-5:00pm, closed 12:15pm-1:15pm.

The phone number at Lawrence PSC is 978-725-6650.