Musculoskeletal Complaints
The approach to a patient’s musculoskeletal complaint is a standardized, often sequential search for what can and what cannot be managed by the examining doctor. There is always an ultimate decision: rule in or rule out referable conditions.

The crucial decision with acute traumatic pain is to rule out fracture (and its complications such as neural or vascular damage), dislocation, and gross instability.

The crucial decision with nontraumatic pain is to rule out tumors, inflammatory arthritides, infections, or visceral referral.

There appears to be a misinterpretation regarding the amount of information necessary to make diagnostic or management decisions. One error is to think of all joints as distinctly different because the names of structures, disorders, or orthopaedic tests are different for each joint. Another error is to make the assumption that the joint operates as an independent contractor without accountability to other joints. The first error leads to an overspecialization effort that often leaves the doctor unwilling to attack the vast amount of individual information for each joint. The second error leads the examiner to an approach that excludes important information that may contribute to the diagnosis of a patient’s complaint. Each is an error in extremes: the first is that too much knowledge is assumed necessary; the second assumes that too little baseline information is needed for making diagnostic and treatment decisions.

A general approach to evaluation of any joint (and surrounding structures) utilizes the perspective that a joint is a joint. Although a specific joint may function differently because of its bony configuration, structurally, it is composed generally of the same tissues. Most joint regions have bone, ligaments, a capsule, cartilage and synovium, surrounding tendons and muscles, associated bursae, blood vessels, nerves, fat, and skin. All of these structures may be injured by compression or stretch. Compression may lead to fracture in bones or neural dysfunction in nerves. Stretch leads to varying degrees of tendon/muscle, ligament/capsule, neural/vascular, or bone/epiphyseal damage ranging from minor disruption to full rupture. Joints can be further divided into weight-bearing and non-weight bearing. Non-weight-bearing joints may be transformed into weight-bearing joints through various positions such as handstands or falls with the upper extremity, hyperextension of the spine, or any axial compression force to the joint. Weight-bearing joints are generally more susceptible to chronic degeneration and osteoarthritis.

Bones and joints are also susceptible to nonmechanical processes that involve seeding of infection or cancer as well as the development of primary cancer and the immunologically based rheumatoid and connective tissue disorders. Clues to rheumatoid and seronegative arthritides include a pattern of involvement with a specific predilection to a joint or groups of joints coupled with laboratory investigation.

The approach to evaluation of a neuromusculoskeletal complaint is also directed by a knowledge of common conditions affecting specific structures (regardless of the specific names). Following is a list of these structures and the disorders or conditions most often encountered with each:

- **bone**
  - tumor, primary or metastatic
  - osteochondrosis/apophysitis
  - fracture
  - osteopenia (osteoporosis)
  - osteomyelitis
soft tissue
1. muscle
   - strain or rupture
   - trigger points
   - atrophy
   - myositic ossificans
   - muscular dystrophy
   - rhabdomyositis
2. tendon
   - tendinitis
   - tendinosis
   - paratenonitis
   - rupture
3. ligament
   - sprain or rupture
4. bursa
   - bursitis
5. fascia
   - myofascitis

joint
- arthritis
- subluxation/fixation (chiropractic)
- synovitis
- infection
- joint mice
- dislocation/subluxation (medical)

GENERAL STRATEGY

History
Clarify the onset.
- Is the complaint traumatic?
- Is there a history of overuse?
- Is the onset insidious?

Clarify the type of complaint.
- Is the complaint one of pain, numbness or tingling, stiffness, looseness, crepitus, locking, or a combination of complaints?
- Localize the complaint to anterior, posterior, medial, or lateral if applicable.

Clarify the mechanism if traumatic (for extremities see Table 1–1).
- If there was a fall onto a specific region or structure within that region, consider fracture, dislocation, or contusion.
- Determine whether there was an excessive valgus or varus force, internal or external rotation, or flexion or extension. Consider ligament/capsule or muscle/tendon.
- If there was sudden axial traction to the joint, consider sprain or subluxation.
- If there was axial compression to the joint, consider fracture or synovitis.

Determine whether the mechanism is one of overuse.
- In what position does the patient work?
- Does the patient perform a repetitive movement at work or during sports activities? Consider muscle strain, tendinitis, trigger points, or peripheral nerve entrapment.

If insidious, determine the following:
- Are there associated systemic signs of fever, malaise/fatigue, lymphadenopathy, multiple affected areas, etc?
- Are there local signs of inflammation including swelling, heat, or redness?
- Is there local deformity?
- Is there associated weakness, numbness, tingling, or other associated neurologic dysfunction?

Determine whether the patient has a current or past history or diagnosis of his or her complaint or other related disorders.
- Are there associated spinal complaints or radiation from the spine? Consider subluxation, nerve root entrapment, or compression.
- Does the patient have a diagnosis of another arthritide, systemic disorder such as diabetes, or past history of cancer?
- Does the patient have “visceral” complaints such as abdominal or chest pain, fever, weight loss, or other complaints?

Evaluation
- With trauma, palpate for points of tenderness and test for neurovascular status distal to the site of injury; obtain plain films to rule out the possibility of fracture/dislocation.
- Palpate for swelling, masses, and warmth.
- Determine whether swelling is present and if so, whether it is intra- or extra-articular. If extra-articular, attempt to differentiate between bursal versus vascular inflammation.
- If deformity or mass is evident, attempt to differentiate between soft versus bony tissue. The most common soft-tissue causes would include lipomas, neuromas, and ganglions (or other cyts), or fascial herniation.
### Table 1–1

<table>
<thead>
<tr>
<th>Joint-Specific Injury Mechanism</th>
<th>Possible Structure(s) Damaged</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shoulder</strong></td>
<td></td>
</tr>
<tr>
<td>Fall on an outstretched arm (extended elbow)</td>
<td>Rotator cuff tear</td>
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<tr>
<td></td>
<td>Glenoid labrum tear</td>
</tr>
<tr>
<td></td>
<td>Posterior dislocation</td>
</tr>
<tr>
<td></td>
<td>Clavicular fracture</td>
</tr>
<tr>
<td>Arm forced into abduction/external rotation</td>
<td>Anterior dislocation</td>
</tr>
<tr>
<td>Blow to the shoulder area</td>
<td>Anterior musculature strain</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
</tr>
<tr>
<td></td>
<td>Acromioclavicular separation</td>
</tr>
<tr>
<td></td>
<td>Dislocation</td>
</tr>
<tr>
<td>Fall onto top of shoulder</td>
<td>Shoulder pointer</td>
</tr>
<tr>
<td></td>
<td>Acromioclavicular separation</td>
</tr>
<tr>
<td></td>
<td>Distal clavicular fracture</td>
</tr>
<tr>
<td></td>
<td>Plexus injury</td>
</tr>
<tr>
<td></td>
<td>Medical subluxation</td>
</tr>
<tr>
<td>Traction injury to arm</td>
<td></td>
</tr>
<tr>
<td><strong>Elbow</strong></td>
<td></td>
</tr>
<tr>
<td>Direct fall on tip of elbow or fall on hand with elbow flexed</td>
<td>Olecranon fracture</td>
</tr>
<tr>
<td>Fall on hand with extended elbow</td>
<td>Radial head fracture</td>
</tr>
<tr>
<td>Hyperextension injury to elbow</td>
<td>Elbow dislocation</td>
</tr>
<tr>
<td></td>
<td>Supracondylar fracture in children</td>
</tr>
<tr>
<td></td>
<td>Capitellum fracture</td>
</tr>
<tr>
<td></td>
<td>Avulsion of medial epicondyle</td>
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<tr>
<td></td>
<td>Medial collateral ligament sprain or rupture</td>
</tr>
<tr>
<td></td>
<td>Radial head subluxation</td>
</tr>
<tr>
<td>Severe valgus stress</td>
<td></td>
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<tr>
<td></td>
<td>Sudden traction of forearm</td>
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<tr>
<td><strong>Wrist/hand</strong></td>
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<tr>
<td>Fall on dorsiflexed hand</td>
<td>Navicular fracture</td>
</tr>
<tr>
<td></td>
<td>Epiphyseal and torus fractures in children</td>
</tr>
<tr>
<td></td>
<td>Carpal dislocation, or instability</td>
</tr>
<tr>
<td></td>
<td>Gamekeeper’s thumb (ulnar collateral ligament damage)</td>
</tr>
<tr>
<td></td>
<td>Bennett’s fracture</td>
</tr>
<tr>
<td>Hyperextension or abduction of thumb</td>
<td>Dislocation</td>
</tr>
<tr>
<td>Axial compression of thumb</td>
<td></td>
</tr>
<tr>
<td>Hyperextension of finger</td>
<td>Volar plate injury</td>
</tr>
<tr>
<td></td>
<td>Jersey finger (rupture of flexor digitorum profundus)</td>
</tr>
<tr>
<td></td>
<td>Dislocation</td>
</tr>
<tr>
<td></td>
<td>Avulsion of central slip</td>
</tr>
<tr>
<td></td>
<td>Mallet finger (rupture of extensor tendon)</td>
</tr>
<tr>
<td></td>
<td>Collateral ligament or volar plate injury</td>
</tr>
<tr>
<td></td>
<td>Capsular irritation</td>
</tr>
<tr>
<td></td>
<td>Fracture</td>
</tr>
<tr>
<td>Hyperflexion of finger</td>
<td></td>
</tr>
<tr>
<td>Valgus/varus stress injury to finger</td>
<td></td>
</tr>
<tr>
<td>Axial compression</td>
<td></td>
</tr>
<tr>
<td><strong>Hip</strong></td>
<td></td>
</tr>
<tr>
<td>Fall on hip</td>
<td>Fracture</td>
</tr>
<tr>
<td></td>
<td>Syme’s</td>
</tr>
</tbody>
</table>
With no history of trauma or overuse, consider the use of special imaging, including MRI or CT; bone scan for cancer seeding screen or for stress fracture; electrodagnostic studies if persistent neurological findings are present; laboratory if systemic findings are present; or synovial fluid analysis if swelling is present or if an arthritide is suspected but in need of differentiation (see Table 1–2).

Palpate and challenge the ligaments and capsule of the joint.

Challenge the musculotendinous attachments with stretch, contraction, and a combination of contraction in a stretched position.

Measure the functional capacities of the region involved; determine any associated biomechanical faults that may be contributing to the problem.

### Table 1–1

**Joint-Specific Injury Mechanism (continued)**

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Possible Structure(s) Damaged</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hip (continued)</strong></td>
<td></td>
</tr>
<tr>
<td>Fall on hip</td>
<td>Hip pointer</td>
</tr>
<tr>
<td>Blow to flexed, adducted hip</td>
<td>Trochanteric bursitis</td>
</tr>
<tr>
<td>Blow to flexed, adducted hip</td>
<td>Posterior dislocation</td>
</tr>
<tr>
<td><strong>Knee</strong></td>
<td></td>
</tr>
<tr>
<td>Hyperextension</td>
<td>Anterior cruciate ligament tear</td>
</tr>
<tr>
<td>Sudden deceleration</td>
<td>Anterior cruciate ligament tear</td>
</tr>
<tr>
<td>Blow to a flexed knee at proximal tibia or hyperflexion</td>
<td>Posterior cruciate ligament tear</td>
</tr>
<tr>
<td>Blow to anterior knee/patella</td>
<td>Imitation of plica</td>
</tr>
<tr>
<td>Valgus force</td>
<td>Patellar fracture</td>
</tr>
<tr>
<td></td>
<td>Bursitis</td>
</tr>
<tr>
<td></td>
<td>Infrapatellar fat pad irritation</td>
</tr>
<tr>
<td></td>
<td>Medial collateral ligament tear</td>
</tr>
<tr>
<td></td>
<td>Pes anserine strain</td>
</tr>
<tr>
<td></td>
<td>Meniscus</td>
</tr>
<tr>
<td></td>
<td>Anterior cruciate ligament, meniscus, medial collateral ligament</td>
</tr>
<tr>
<td>Rotational injury with foot fixed on ground</td>
<td></td>
</tr>
<tr>
<td>Rotational injury with a valgus force</td>
<td></td>
</tr>
<tr>
<td><strong>Foot/ankle</strong></td>
<td></td>
</tr>
<tr>
<td>Plantarflexion, inversion of ankle</td>
<td>Ankle sprain with possible associated bifurcate ligament damage, fracture, or peroneal tendon snapping from torn retinaculum</td>
</tr>
<tr>
<td>Eversion injury to ankle</td>
<td>Deltoid ligament sprain or rupture</td>
</tr>
<tr>
<td>Eversion injury to ankle</td>
<td>Fracture</td>
</tr>
<tr>
<td>Hyperextension of great toe</td>
<td>Dislocation</td>
</tr>
<tr>
<td>Landing on heels</td>
<td>Turf-toe injury to capsular ligaments</td>
</tr>
<tr>
<td></td>
<td>Fat pad imitation</td>
</tr>
<tr>
<td></td>
<td>Ankle or tibial fracture</td>
</tr>
</tbody>
</table>

**Management**

- Refer fracture/dislocation, infection, and tumors for orthopaedic management.
- Refer or comanage rheumatoid and connective tissue disorders.
- If the problem is one of instability without ligament rupture, stabilize the joint through an appropriate exercise program using a brace initially, if necessary, to assist.
- If the problem is weakness, strengthen the associated muscle.
- Functionally retrain the individual for a return to daily activities and occupational or sport requirements.
- Use manipulation/mobilization for articular dysfunction.
# Synovial Fluid Examination

<table>
<thead>
<tr>
<th>Type</th>
<th>Examples</th>
<th>Color</th>
<th>Clarity</th>
<th>WBC (per μL)</th>
<th>PMNs</th>
<th>Culture</th>
<th>Glucose</th>
<th>Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>Clear</td>
<td>Transparent</td>
<td>&lt;200</td>
<td>&lt;25%</td>
<td>Negative</td>
<td>Nearly = to serum</td>
<td>&lt;1.5</td>
</tr>
<tr>
<td>Group I (Noninflammatory)</td>
<td></td>
<td>Yellow</td>
<td>Transparent</td>
<td>200–300</td>
<td>&lt;25%</td>
<td>Negative</td>
<td>Nearly = to serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>DJD</td>
<td>Trauma</td>
<td>Yellow</td>
<td>Transparent</td>
<td>200–300</td>
<td>&lt;25%</td>
<td>Negative</td>
<td>Nearly = to serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Osteochondritis dissecans</td>
<td>PVS</td>
<td>Yellow</td>
<td>Transparent</td>
<td>200–300</td>
<td>&lt;25%</td>
<td>Negative</td>
<td>Nearly = to serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Osteochondromatosis</td>
<td>Neuropathic arthropathy</td>
<td>Yellow</td>
<td>Transparent</td>
<td>200–300</td>
<td>&lt;25%</td>
<td>Negative</td>
<td>Nearly = to serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Group II (Inflammatory)</td>
<td>RA (Active crystal-induced) (gout, pseudo-gout)</td>
<td>Yellow to opalescent</td>
<td>Transparent to opaque</td>
<td>3,000–50,000</td>
<td>50% or more</td>
<td>Negative</td>
<td>&gt; 25, lower than serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Active crystal-induced</td>
<td>Seronegatives (AS, Reiter's psoriatic)</td>
<td>Yellow to opalescent</td>
<td>Transparent to opaque</td>
<td>3,000–50,000</td>
<td>50% or more</td>
<td>Negative</td>
<td>&gt; 25, lower than serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Enteropathic (IBD)</td>
<td>Rheumatic fever</td>
<td>Yellow to opalescent</td>
<td>Transparent to opaque</td>
<td>3,000–50,000</td>
<td>50% or more</td>
<td>Negative</td>
<td>&gt; 25, lower than serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>SLE</td>
<td>Scleroderma</td>
<td>Yellow to opalescent</td>
<td>Transparent to opaque</td>
<td>3,000–50,000</td>
<td>50% or more</td>
<td>Negative</td>
<td>&gt; 25, lower than serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Myotic infection</td>
<td>Yellow to green</td>
<td>Opaque</td>
<td>&gt; 50,000</td>
<td>75% or more</td>
<td>Usually positive</td>
<td>&lt; 25, much lower than serum</td>
<td>Often &gt; 3.5</td>
</tr>
<tr>
<td>Group III (Purulent)</td>
<td>Pyogenic bacterial infection</td>
<td>Yellow to green</td>
<td>Opaque</td>
<td>&gt; 50,000</td>
<td>75% or more</td>
<td>Usually positive</td>
<td>&lt; 25, much lower than serum</td>
<td>Often &gt; 3.5</td>
</tr>
</tbody>
</table>

Note: Joint aspiration findings for hemorrhagic causes, including hemophilia, trauma (with or without fracture), neuropathic arthropathy, PVS, and benign neoplasms (e.g., hemangioma) are dominated by blood in the joint.

Legend: WBC = white blood cell; PMN = polymorphonuclear leukocytes; PVS = pigmented villonodular synovitis; IBD = inflammatory bowel disease (includes ulcerative colitis and regional enteritis).
**HISTORY**

A mnemonic approach to the patient’s complaints may be helpful in organizing the vast number of possibilities. Beginning with a description of the patient’s complaint, a list of common causes may be attached. WIRS Pain is a mnemonic for weakness, instability, restricted movement, surface complaints, and pain.

**Weakness**

Weakness may be due to pain inhibition, muscle strain, or neurologic interruption at the myoneural junction, peripheral nerve, nerve root, or spinal cord and above. Weakness may be a misinterpretation by the patient when instability or a “loose” joint is present or the patient has stiffness that must be overcome by increased muscular activity.

**Instability**

Instability is due to either traumatic damage to ligamentous or muscular support or due to the inherent looseness found in some individuals’ joints. This inherent looseness is usually global and can be identified in other joints or acquired as a result of repetitive overstretch positioning. Instability is most apparent when the joint is positioned so that muscles have less mechanical advantage (e.g., overhead shoulder positions) or when a quick movement demand is faster than the reaction time for the corresponding muscles (cutting or rotating knee movements).

**Restricted Movement**

Restricted movement may be due to pain, muscle spasm, stretching of soft tissue contracture, or mechanical blockage by osteophytes, joint mice, fracture, or effusion.

**Surface Complaints**

Superficial complaints include skin lesions, cuts/abrasions, swelling, and a patient’s subjective sense of numbness or paresthesias.

**Pain**

Pain is nonspecific; however, the cause usually will be revealed by combining a history of trauma, overuse, or insidious onset with associated complaints and significant examination findings. It is important to determine local pain versus referred pain. Following are some guidelines:

- Referred pain from scleratogenous sources: Scleratogenous pain presents as a nondermatomal pattern with no hard neurologic findings such as significant decrease in myotomal strength or deep tendon reflex changes. Although the term is used broadly, here we are referring mainly to facet- and disc-generated pain.
- Referred pain from visceral sources: In most cases a historical screening of patients will reveal primary or secondary visceral complaints. It is important to know the classic referral zones, such as scapular/shoulder pain with cholelithiasis and medial arm pain with cardiac ischemia.
- Bone pain: Bone pain is deep pain, commonly worse in the evening. Trauma may indicate an underlying fracture requiring radiographic evaluation. An overuse history may be suggestive of a stress fracture requiring a radiographic evaluation. If results of the radiograph are negative, but a stress fracture is still suspected, a bone scan is warranted.

A careful history will usually indicate the diagnosis or, at the very least, narrow down the possibilities to two or three. Physical examination and imaging studies more often are used as a confirmation of one’s suspicion(s).

Generalizing a history approach allows the doctor to address any complaint regardless of region. Generally speaking, damage to structures locally is due to (1) exceeding the tensile stress of ligaments, capsule, muscles, and tendons; (2) compression of bone; (3) demineralization of bone; or (4) intrinsic destructive processes involving arthritides (e.g., pannus formation with rheumatoid arthritis [RA], crystal deposition with gout or pseudogout), infections, or cancer. Although the first two categories are almost always the result of trauma or overuse, the latter two are more commonly insidious. Traumatic and overuse disorders are classically local with regard to signs and symptoms, whereas arthritides and cancer are often either generalized or stereotypical based on the type.

Suspicion of specific structures is based on a basic knowledge of what causes damage to any similar structure regardless of which region or joint is involved. Ligament or capsular injury is often the result of excessive force on the opposite side of the ligament/capsule. For example, a valgus stress (outside to inside force) to the knee will cause an injury to the medial collateral ligament; a varus force, the lateral collateral ligament. Although more dramatically evident in an acute injury, it must be remembered that low-level, chronic stresses are often the cause of ligamentous or capsular sprain. Muscle injury can be divided into stretch injury and contraction injury. Often when ligaments are damaged, muscle/tendon groups are also involved. Muscle/tendons often act as static stabilizers simply because when they cross the joint they are in the way when outside forces stretch that joint. Additionally, muscles will often contract in an attempt to protect the joint and either incur damage or impose more damage to the joint. This occurs especially when a joint is in extension (such as the knee and elbow) or in neutral (such as the wrist and ankle). Contraction injury is divided into concentric and eccentric. Usually an overexertion problem, concentric injury often occurs when too heavy a weight is lifted or a sudden explosive muscle activity is required. Concentric injury occurs as the muscle is shortening. Eccentric injury occurs while the muscle is lengthening. Although eccentric injury may occur with lifting, this pattern is frequently seen with overuse or repetitive activity and/or injuries that challenge the decelerator or stabilizer role of the muscle.
Tendons are susceptible primarily to overstrain from a sudden, forceful muscle contraction or from overuse. Occasionally, direct trauma may damage or inflame the tendon or its sheath. Rheumatoid and connective tissue disorders can also affect the synovial lining or paratenon. Sometimes the use of various terminologies in the description of tendon disorders is confusing. Newer terminology replacing older nomenclature causes some of this difficulty, coupled with new theories as to the types of tendon pathology that occur related to its structure and function. Following is an updated list:

- **Paratenonitis**—This term is replacing tenosynovitis, tenovaginitis, and peritendinitis. It is characterized by inflammation of only the paratenon (lined by synovium or not). Clinical signs are swelling, pain, crepitation along the tendon, local tenderness, and warmth.

- **Tendinitis**—Now used in place of strain or tear of a tendon. This term refers to symptomatic degeneration of a tendon with vascular disruption and an inflammatory repair response. Stages include: acute, < 2 weeks; sub-acute, 4–6 weeks; and chronic, ≥ 6 weeks. Three subgroups include: (1) purely inflammatory with acute hemorrhage and tearing; (2) inflammation that is in addition to preexisting degeneration, and (3) calcification and tendinosis that is chronic.

- **Tendinosis**—The newer term used to indicate intratendinous degeneration due to atrophy (due to aging, microtrauma, vascular compromise, etc.). This is considered noninflammatory with hypocellularity, variable vascular ingrowth, local necrosis, and/or calcification, with accompanying fiber disorientation. Palpable nodules can be found, such as in the Achilles, with or without tenderness.

- **Paratenonitis with tendinosis**—This describes a paratenon inflammation associated with intratendinous degeneration. Unlike tendinosis, this combination of pathologies presents clinically with a possible palpable tendon nodule, with accompanying signs of swelling and inflammation.

Bursae are protective cushions placed strategically at points of friction, particularly between muscle/tendon and bone. Although there are standard bursae in most individuals, adventitious bursae may develop at sites of repetitive friction in individuals performing specific activities. Bursae may be deep or superficial. Superficial bursae are susceptible to direct traumatic forces. Deep bursae are more susceptible to compression by bone or soft tissue structures. Compression is often position specific such as during overhead movements with the shoulder. Bursitis may be secondary to other soft tissue involvement such as calcific tendinitis.

When musculoskeletal pain does not have an obvious mechanical or traumatic cause, a search is initiated for myofascial disorders, arthritides, psychologic factors, connective tissue disorders, cancer, and infection (see Table 1–3).

Arthritis has a "geriatric" connotation, yet it may affect any age group. The term simply means that the joint is affected. Generally, arthritis is due to degeneration or destruction that is age-related or trauma related, infectious, inflammatory, and/or autoimmune. Based on the cause, arthritis may present as a monoarthropathy (i.e., single joint), oligoarthropathy (2–4 joints), or as a polyarthropathy (≥ 5 joints). When a single joint is involved, gout (first toe), infectious (direct infection or indirect spreading from another source such as gonococcal), or trauma should be considered. When multiple joints are involved a distinction in thinking occurs differentiating degenerative, inflammatory (primarily rheumatoid and rheumatoid variants), and crystalline induced (primarily gout, pseudo-gout, amyloidosis, etc.). Seronegatives and enteropathic arthropathies tend to be oligoarticular, whereas RA and LE tend to involve more joints.

Considering arthritis as a cause of joint pain, there are several other general factors that when considered separately and then clustered together provide a good tool for narrowing the large list of possibilities. The sequence of how these factors are considered may change given the presentation of the patient, yet the discussion will begin with age. There are very few arthritides that affect the young. Primarily, juvenile rheumatoid arthritis or arthritis secondary to other diseases would be considered. For the young to middle-aged adult, primarily inflammatory and/or autoimmune arthritides are considered, including:

- Seronegative arthritides (i.e., negative for rheumatoid factor) including ankylosing spondylitis (AS), Reiter’s, and psoriatic
- Rheumatoid arthritis (RA)
- Scleroderma
- Lupus erythematosis (LE)
- Osteitis condensins ili
- Synoviochondrometaplasia

For onset in the senior, the primary considerations include:

- Degenerative joint disease; osteoarthritis (OA)
- Diffuse idiopathic skeletal hyperostosis (DISH)
- Hypertrophic osteoarthropathy
- Gout
- Pseudogout; calcium pyrophosphate dihydrate (CCPD) deposition disease

Considering gender, males are more prone toward AS, Reiter’s, gout, hypertrophic osteoarthropathy, and secondary OA. Females are more prone toward juvenile and adult RA, LE, scleroderma, and osteitis condensins ili, as
## TABLE 1–3

<table>
<thead>
<tr>
<th>Type</th>
<th>Features</th>
<th>Management Issues</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Degenerative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary Osteoarthritis</td>
<td>Age of Onset: Generally &gt; 45 y/o</td>
<td>Management in early and middle stages should include strengthening around involved joints. If weight-bearing joint, begin with non-weight-bearing and progress cautiously to weight-bearing if possible. Maintenance of normal joint motion and function may be facilitated by adjusting/manipulation or mobilization. Dietary approaches include glucosamine and chondroitin sulfate. Medical management may include recommendations for NSAIDs, in particular, COX-2 inhibitors. Some medical specialists may recommend viscosupplementation (injection of hyaluronic acid into the degenerative joint). This is of questionable value. In some joints, joint replacement is necessary.</td>
</tr>
<tr>
<td></td>
<td>Gender Predominance: Ratio of female to male = 10:1</td>
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<tr>
<td></td>
<td>Common Joints Involved: Hips, knees, SI joint, AC joint, first MCP, first MC trapezium, DIP joints of hands</td>
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<td></td>
<td>Often initially asymptomatic, gradual increase in joint stiffness and pain. Deformity may be apparent (e.g., Heberden's nodes in hands). May eventually lead to joint subluxation and instability.</td>
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<td></td>
<td>Radiologically: The distribution is asymmetric, with non-uniform loss of joint space, osteophyte formation, subchondral sclerosis (eburnation), subchondral cysts.</td>
<td></td>
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<tr>
<td>Secondary Osteoarthritis</td>
<td>Age of Onset: &gt; 25 y/o</td>
<td>Management in early and middle stages should include strengthening around involved joints. If weight-bearing, begin with non-weightbearing and progress cautiously to weight-bearing if possible. Maintenance of normal joint motion and function may be facilitated by adjusting/manipulation or mobilization. Dietary approaches include glucosamine and chondroitin sulfate. Medical management may include recommendations for NSAIDs, in particular, COX II inhibitors. Some medical specialists may recommend viscosupplementation (injection of hyaluronic acid into the degenerative joint). This is of questionable value. In some joints, joint replacement is necessary.</td>
</tr>
<tr>
<td></td>
<td>Gender Predominance: Equal</td>
<td></td>
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<tr>
<td></td>
<td>Common Joints Involved: GH, AC, SI, hip, elbow, knee, foot, hand</td>
<td></td>
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<tr>
<td></td>
<td>Cause is secondary to other disorders or diseases/injuries such as trauma, septic or inflammatory arthritis, slipped epiphyses, degenerative joint disease, avascular necrosis, osteopenia, and osteoarthritis. Similar radiographic presentation.</td>
<td></td>
</tr>
<tr>
<td>Erosive Osteoarthritis</td>
<td>Age of Onset: 40–50 y/o</td>
<td>Management in early and middle stages should include strengthening around involved joints. If weight bearing begin with non-weight-bearing and progress cautiously to weight-bearing if possible. Dietary approaches include glucosamine and chondroitin sulfate. Medical management may include recommendations for NSAIDs, in particular, COX II inhibitors. In addition, the following anti-inflammatory medications may be suggested:</td>
</tr>
<tr>
<td></td>
<td>Gender Predominance: Female</td>
<td></td>
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<tr>
<td></td>
<td>Common Joints Involved: Interphalangeal joints of hand</td>
<td>■ DMARDs — Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet are highly effective.</td>
</tr>
<tr>
<td></td>
<td>Inflammatory variant of DJD characterized by cartilage degeneration and synovial proliferation. Acute episodes that appear similar to inflammatory/synovial arthritis, chronically may evolve to subluxation and development of Heberden's nodes. Radiologically similar to OA with additional finding of central erosions.</td>
<td></td>
</tr>
<tr>
<td>Degenerative Spine Disease</td>
<td>Age of Onset: &gt; 30 y/o</td>
<td>Management in early and middle stages should include strengthening of the spinal muscles with a focus on abdominal strengthening, and extensor strengthening and stretching. The three-joint complex model stresses the need to consider the interrelationship of facets, zygapophyseal joints, and intervertebral disc in the progression of DJD of the spine. Maintenance of normal joint motion and function may be facilitated by adjusting or manipulation or mobilization. Dietary approaches include an anti-inflammatory dietary regimen and use of glucosamine and chondroitin sulfate. Medical management may include recommendations for NSAIDs, in particular, COX II inhibitors for pain management.</td>
</tr>
<tr>
<td></td>
<td>Gender Predominance: Equal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Common Joints Involved: Specific spinal involvement at C5–C7, T2–T5, T10–T12, L4–S1 with additional involvement of uncovertebral, costovertebral, discotorrhea, and apophyseal (facet) joint involvement</td>
<td></td>
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</table>
### General Approach to Musculoskeletal Complaints

**TABLE 1–3**

<table>
<thead>
<tr>
<th>Type</th>
<th>Age of Onset</th>
<th>Gender Predominance</th>
<th>Common Joints Involved</th>
<th>Features</th>
<th>Management Issues</th>
</tr>
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<tbody>
<tr>
<td>Diffuse idiopathic skeletal hyperostosis (DISH) (synonym: ankylosing hyperostosis, Forestier’s disease)</td>
<td>50 y/o and older</td>
<td>Male</td>
<td>Spine, predominantly T7-T11 (calcification of anterior longitudinal ligament) with 30% peripheral joint involvement</td>
<td>Found in 25% of men and 15% of women &gt; 50 y/o (common). May be asymptomatic, when symptoms present, complaints involving the spine; some peripheral joints; may include abnormal &quot;flowing wax&quot; appearance of spine and occasional complaints involving the Achilles tendon, extensor wad of wrist/forearm, plantar fascia, and quadriceps tendon (may find entheseophytes at corresponding sites); about one quarter of patients have diabetes. Radiographically diffuse, thickening of the hyperostosis primarily along the anterolateral aspect of the spine. 50% of patients also have ossification of the PLL, especially in the cervical spine.</td>
<td>Management in early and middle stages should include strengthening of the spinal muscles with a focus on abdominal strengthening and extensor strengthening and stretching. Dietary approaches include an anti-inflammatory dietary regimen and use of glucosamine and chondroitin sulfate, yet DISH appears to follow its own course of progression specific to the individual but generally always progressive. Medical management may include recommendations for NSAIDs, in particular, COX-2 inhibitors for pain management.</td>
</tr>
<tr>
<td>Neuropathic (Neurotrophic) Arthropathy</td>
<td>Variable</td>
<td>Variable</td>
<td>Knee, hip, ankle, spine, shoulder, elbow, wrist, foot</td>
<td>Variable upper motor and lower motor lesions cause a combination of loss of proprioception and pain perception leading to joint destruction. Conditions include syringomyelia, diabetes, tabes dorsalis, multiple sclerosis, Charcot-Marie-Tooth disease, prolonged use of intra-articular corticosteroids, porphyria, and leprosy among others. A somewhat separate but related cause is spinal cord damage resulting in paraplegia or quadriplegia which results in unusually asymptomatic bony ankylosis. Radiographically neuropathic arthropathy is seen as joint collapse, pseudarthrosis, fragmentation, and deformity.</td>
<td>Treatment is directed toward the primary disease. If in weightbearing joints, mechanical assistance is often required. In severe cases, amputation is necessary.</td>
</tr>
<tr>
<td>Synovochondrometaplasia (idiopathic synovial osteochondromatosis)</td>
<td>30–50 y/o</td>
<td>Male, female ratio = 3:1</td>
<td>Knee, hip, ankle, elbow, wrist</td>
<td>Synovochondrometaplasia, as the name implies, is a synovial metaplasia that results in the formation of cartilage that then forms loose bodies in the joint. This process is usually idiopathic but may be the result of trauma. The patient will report increasing pain, swelling, crepitation, and locking due to the loose bodies. Radiographically the loose bodies can be seen if radiopaque. Sometimes erosion may occur as in the &quot;apple-core&quot; deformity of the hip.</td>
<td>Synovectomy for most patients. Joint replacement may be recommended for older patients.</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Positive for Rheumatoid Factor (Seropositive)</td>
<td>25–35 y/o</td>
<td>Hand, foot, wrist, knee, elbow, GH joint, AC joint, and cervical spine (atlantoaxial)</td>
<td>Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting, soft-tissue therapy, and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies.</td>
<td></td>
</tr>
<tr>
<td>Type</td>
<td>Features</td>
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| RA (continued)  | A symmetric, bilateral, polyarticular disorder of the synovial membrane resulting in joint pain, swelling and destruction. Also involved are ligaments, tendons, and bursae. The diagnostic criteria includes: Deformities such as Boutonniere, swan-neck, phalangeal deviation, and arthritis mutilans; morning stiffness that lasts longer than one hour, specific swelling of several joints (including the PIP, MCP, and wrist), rheumatoid nodules, positive for rheumatoid factor, and radiographic evidence that includes erosions or periarticular osteopenia or both in hands or wrists or both. Need four or more of the above for at least 6 weeks. Additional symptoms may include fatigue, anorexia, weight loss, and muscular pain or stiffness. Special concern is for atlanto-axial instability due to ligament erosion and a resulting risk of excessive movement leading to spinal cord compression. | Incorporate an "anti-inflammatory" diet regimen (see Table 1–9). Medical management includes:  
- NSAIDs—COX-1 inhibitors (e.g., ibuprofen, naproxen) or COX-2 inhibitors (e.g., Vioxx, Celebrex)  
- DMARDs—Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet are highly effective.  
- Biologic agents—Reduce the production of tissue necrosis factor (TNF) (e.g., Embrel and Remicade are usually given together with methotrexate). May be administered as infusion therapy.  
- Corticosteroids—Rarely needed. |
| Juvenile Chronic Arthritis | Age of Onset—5–10 y/o  
Gender Predominance—Variable based on specific disorder  
Common Joints Involved—Hand, foot, wrist, knee, elbow, hip, and cervical spine  
Several types including:  
- Juvenile-onset adult RA—same findings as RA  
- Still’s disease—more of a systemic disease  
- Juvenile-onset seronegative arthropathies—see each disorder  
Radiographically similar with the possible addition of growth disturbances of bone and epiphyseal compression fractures | Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Medical management includes:  
- NSAIDs—COX-1 inhibitors (e.g., ibuprofen, naproxen) or COX-2 inhibitors (e.g., Vioxx, Celebrex)  
- DMARDs—Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet are highly effective.  
- Biologic agents—Reduce the production of tissue necrosis factor (TNF) (e.g., Embrel and Remicade are usually given together with methotrexate). May be administered as infusion therapy.  
- Corticosteroids—Rarely needed. |
| Ankylosing Spondylitis (AS) | Age of Onset—15–35 y/o  
Gender Predominance—Male to female ratio = 4:1 to 10:1  
Common Joints Involved—SI joint, thoracolumbar spine, cervical spine, symphysis pubis, hip, shoulder, and heel  
Complaints often begin with SI pain and progress to low back and thoracic stiffness. Eventually there may be a decrease in chest expansion. Periphera joint involvement occurs in approximately 50%, as does radiating pain to the lower extremity. Areas of concern include enthesitis (20% of cases), axial insufficiency, aneurysms, pulmonary fibrosis, pleuritis, IBD, and amyloidosis. Laboratory findings include an increased ESR during active phase, negative for RA and E factors, HLA-B27 positive in 80% (positive in 6–8% of normal population). Radiographically there are classic signs, including symmetrical involvement of SI joints, ligament calcification, and marginal syndesmophytes, eventually leading to “trolley-track” sign and bamboo spine. | Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Medical management includes:  
- NSAIDs—COX-1 inhibitors (e.g., ibuprofen, naproxen) or COX-2 inhibitors (e.g., Vioxx, Celebrex)  
- DMARDs—Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet are highly effective.  
- Biologic agents—Reduce the production of tissue necrosis factor (TNF) (e.g., Embrel and Remicade are usually given together with methotrexate). May be administered as infusion therapy.  
- Corticosteroids—Rarely needed. |
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<tbody>
<tr>
<td><strong>Reiter's Syndrome</strong></td>
<td>Age of Onset: 15–35 y/o</td>
<td>Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Medical management includes:</td>
</tr>
<tr>
<td>Gender Predominance: Male to female ratio = 5:1 to 50:1 depending upon study</td>
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<td>■ NSAIDs—COX-1 (e.g., ibuprofen, naproxen) or COX-2 inhibitors (e.g., Vioxx, Celebrex)</td>
</tr>
<tr>
<td>Common Joints Involved: SI joint, foot, heel, ankle, knee, hip, spine, more rarely the upper extremity</td>
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<td>■ DMARDs—Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet highly effective.</td>
</tr>
<tr>
<td>Urethritis and other eye complaints often following a STD or gastrointestinal infection. Keratitis, keratoderma, and keratosis of nails may be found. Systemic findings may include fever, weight loss, thrombocytopenia or amyloidosis. Lab findings may include positive HLA-B27 (75%), leukocytosis, anemia, and elevated ESR. Radiographically, SI joint is prominent, anterior avulsion instability, nonmarginal syndesmophytes. Similar to psoriatic arthritis, a single digit may be involved (sausage fingers) and enthesopathies are common as in AS. Monitor for aortic regurgitation in chronic cases.</td>
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<td>■ Biologic agents—Reduce the production of tissue necrosis factor (TNF) (e.g., Enbrel and Remicade are usually given together with methotrexate). May be administered as infusion therapy.</td>
</tr>
<tr>
<td>Psoriatic</td>
<td>Age of Onset: 20–50 y/o</td>
<td>Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. When arthritis is present, cyclosporine, methotrexate, and acitretin are used. Methotrexate is associated with hepatic toxicity; cyclosporine associated with hypertension and nephrotoxicity; and acitretin is associated with the elevated serum lipids, mucocutaneous toxicity, and hepatotoxicity. New drugs are being marketed that, although highly promising, are extremely expensive. These drugs are part of a new class of medications called biologic response modifiers or “biologics.” The mechanism for these new drugs is either to block and reduce abnormal T-lymphocyte activity or the inflammatory response. Examples are alefacept and etanercept. (The “cept” ending is an indication of the drug’s effect, which is fusion of a receptor to the Fc portion of human IgG.)</td>
</tr>
<tr>
<td>Gender Predominance: Generally equal</td>
<td>Gender Predominance: Generally equal</td>
<td>■ NSAIDs—COX-1 (e.g., ibuprofen, naproxen) or COX-2 inhibitors (e.g., Vioxx, Celebrex)</td>
</tr>
<tr>
<td>Common Joints Involved: Hand, foot, SI joint, thoracolumbar spine, and cervical spine</td>
<td>Common Joints Involved: Hand, foot, SI joint, thoracolumbar spine, and cervical spine</td>
<td>■ DMARDs—Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet highly effective.</td>
</tr>
<tr>
<td>Only about 5% of those with skin disease have the joint involvement. There are various patterns, yet many times the proximal and distal IP joints are involved. A deforming type may lead to ankylosis mutilans. In addition to having scaling patches of skin (psoriasis) on the extensor surfaces of the knees and elbows, patients may also have nail changes, including pitting, disfigurement, and splitting in some cases. Hypertension occurs arthritic joint. Other skin lesions may occur in the hands and feet. Lab includes HLA-B27 antigen (90% of cases), mild anemia, elevated ESR during active periods, occasionally elevated uric acid levels. Radiographically, involvement of the hands is similar to RA. In addition, one digit may be involved (sausage fingers) and tracector pitting and proliferation. ivory phalanx) occur in the spine, nonmarginal syndesmophytes may be seen.</td>
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<td>■ Biologic agents—Reduce the production of tissue necrosis factor (TNF) (e.g., Enbrel and Remicade are usually given together with methotrexate). May be administered as infusion therapy.</td>
</tr>
<tr>
<td>Enteropathic (associated with inflammatory bowel disease (IBD))</td>
<td>Enteropathic (associated with inflammatory bowel disease (IBD))</td>
<td>Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Use anti-inflammatory approaches in diet and supplement recommendations and physiotherapy management.</td>
</tr>
<tr>
<td>Age of Onset: Variable</td>
<td>Age of Onset: Variable</td>
<td>■ NSAIDs—COX-1 (e.g., ibuprofen, naproxen) or COX-2 inhibitors (e.g., Vioxx, Celebrex)</td>
</tr>
<tr>
<td>Gender Predominance: Variable</td>
<td>Gender Predominance: Variable</td>
<td>■ DMARDs—Disease-modifying antirheumatic drugs (e.g., methotrexate [Rheumatrex and Trexal], hydrochloroquine [Plaquenil], and leflunomide [Arava]). These are toxic and may take weeks to months to work, yet highly effective.</td>
</tr>
<tr>
<td>Common Joints Involved: SI joint and spine, occasionally peripheral joint involvement</td>
<td>Common Joints Involved: SI joint and spine, occasionally peripheral joint involvement</td>
<td>■ Biologic agents—Reduce the production of tissue necrosis factor (TNF) (e.g., Enbrel and Remicade are usually given together with methotrexate)</td>
</tr>
<tr>
<td>Many inflammatory disorders affecting the GI tract may result in an arthritis similar to the seronegative arthritides. Disorders include Crohn’s, ulcerative colitis, Whipple’s disease, and infections, including Salmonella, Shigella, and Yersinia. Intestinal bypass surgery may also be related. The frequency of IBD and AS is about 15%. Laboratory reveals HLA-B27 in 90% of those with IBD and arthritis. Radiographic findings are similar to AS including SI involvement and the spine.</td>
<td>Many inflammatory disorders affecting the GI tract may result in an arthritis similar to the seronegative arthritides. Disorders include Crohn’s, ulcerative colitis, Whipple’s disease, and infections, including Salmonella, Shigella, and Yersinia. Intestinal bypass surgery may also be related. The frequency of IBD and AS is about 15%. Laboratory reveals HLA-B27 in 90% of those with IBD and arthritis. Radiographic findings are similar to AS including SI involvement and the spine.</td>
<td>■ Corticosteroids</td>
</tr>
</tbody>
</table>

### Selected Arthritic Disorders (continued)

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<tr>
<th>Type</th>
<th>Features</th>
<th>Management Issues</th>
</tr>
</thead>
</table>
| **Systemic Lupus Erythematosus (SLE)** | **Age of Onset** — 20–45 y/o  
**Gender Predominance** — Female more than male  
**Common Joints Involved** — Hand and osteonecrosis, specifically of femur (head and condyles) and sometimes shoulder (humeral head)  
A systemic autoimmune disorder characterized by multi-system involvement resulting in generalized findings such as fever, anorexia, weight loss, malaise, and weakness. Visceral inflammation occurs. Skin affects include rashes (e.g., butterfly malar rash). Polyarthralgia is common. Like many patients with autoimmune mediated conditions, tendons are weakened and may rupture. Laboratory reveals anemia with leukopenia and plasma protein abnormalities (protein electrophoresis usually ordered due to globulin increase). Antinuclear antibody and LE cells present. A false-positive sylphils test may occur. Radiographically a symmetric, nonerosive yet deforming arthropathy is seen. Osteonecrosis may be seen due to the disease or due to treatment (controversial). | Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Protection of the skin includes avoiding sunlight, and it is important when exposed to use a sunblock with SPF 15 or higher. Primary treatment is prednisone for joint pain, cutaneous lesions, and renal and CNS involvement. Other medical therapies include antibiotics (hydroxychloroquine) and NSAIc. Infections common due to immunosuppression and is the cause of death in one-third of cases. Blacks and Hispanics fair worse. Pericarditis is found in 25% of patients. Also screening for renal function is important to determine disease activity. |
| **Scleroderma (progressive systemic sclerosis)** | **Age of Onset** — 20–30 y/o  
**Gender Predominance** — Female more than male  
**Common Joints Involved** — Hand, wrist, foot, ribs, and more rarely the spine  
There are two types of this collagen-vascular disease: one with systemic involvement (progressive) and one without (localized). Scleroderma is characterized by involvement of multiple organs including skin, heart, lungs, kidneys, GI tract, and musculoskeletal system; therefore, signs and symptoms are quite variable. Muscle weakness, including dysphagia; Raynaud’s phenomenon; hyperpigmentation; telangiectasias, and thickening and thickening of the skin of the face, hands, and feet. Laboratory findings include an elevated ESR (60–70%), positive RF (20–40%), positive ANA (DS–96%), and a high protein level in synovial fluid. Radiographically there are periarticular and subcutaneous calcifications including paraspinal, phalangeal tuft, and superior rib erosions. | Management is for various aspects of the disease. Following are combinations of medical and conservative approaches:  
**Raynauds**  
- Calcium channel blockers  
- Peripheral adrenergic blockers  
- Protective measures against cold, cessation of smoking, and decreased use of caffeine and other sympathomimetics  
**Renal**  
- Initially ACE inhibitors; may lead to dialysis or kidney transplant  
**Pulmonary hypertension**  
- May require oxygen or lung transplant in serious cases  
**Esophageal reflux**  
- Avoid large meals and a recumbent position after meals  
- Avoid sympathomimetic substances and certain foods  
- H2 inhibitors and/or proton-pump inhibitors  
**Arthralgias**  
- NSAIDs |
| **Dermatomyositis and Polymyositis** | **Age of Onset** — 5–10 y/o and again at 20–50 y/o  
**Gender Predominance** — Female to male ratio = 2:1  
**Common Joints Involved** — Soft tissues primarily of the thigh, leg, and arm  
Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Protection of the skin with SPF 15 or higher is important. Provide physical therapy to keep muscle stretch and strength. | Given that some of the therapies employed by chiropractic are mechanical, including adjusting soft-tissue therapy, and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. Protection of the skin with SPF 15 or higher is important. Provide physical therapy to keep muscle stretch and strength. |
### Table 1–3

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<tr>
<td><strong>Dermatomyositis and Polymyositis (continued)</strong></td>
<td>Dermatomyositis affects skin and muscle, whereas, polymyositis affects primarily muscle. The effect is inflammation and degeneration of striated muscle with a laying down of sheet-like calcifications in soft tissue. About half of patients have arthritis while one-third have Raynaud phenomenon. Disability occurs due to progressive symmetric, proximal muscle weakness. Laboratory findings include CRP elevations and elevations in urinary creatinine levels. EMG reveals a proximal myopathy as does muscle biopsy. Radiographically, there is soft tissue atrophy coupled with sheet-like soft tissue calcifications and sometimes ossification. Like other inflammatory conditions, there is phalangeal tuft resorption.</td>
<td>If dysphagia is present, speech therapy should be employed. Inflammatory aspect may be managed medically with prednisone, immunosuppressive therapy such as methotrexate or azathioprine. Approximately 50% go into remission in 5 years, with an approximate 75% 8-year survival. Those who do not remiss remain on therapy.</td>
</tr>
</tbody>
</table>
| **Mixed Connective Tissue Disease** | **Age of Onset** — 20–50 y/o  
**Gender Predominance** — Female more than male  
**Common Joints Involved** — Hand, wrist, and foot | Caution with rheumatoid conditions is unpredictable flare-ups. Given that some of the therapies employed by chiropractic are mechanical, including adjusting, soft-tissue therapy, and physiotherapy, it is important to keep in mind that this is an inflammatory condition and can be exacerbated by these therapies. The medical management approach would include those for the underlying disorders. See management under each. |
| **Hypertrophic Osteoarthropathy** (Marie-Bamberger syndrome or pulmonary osteoarthropathy) | **Age of Onset** — 40–60 y/o  
**Gender Predominance** — Primarily male  
**Common Joints Involved** — Fingers (clawing), periostitis in tibia, fibula, radius, and ulna | Identification of the underlying disorder directs appropriate treatment measures. In some cases, the hypertrophic osteoarthropathy may improve or disappear with effective care. This may include chemotherapy for tumors or antibiotic therapy for chronic pulmonary infection. In some cases, vagotomy or percutaneous vagal blockade is necessary for symptomatic relief. NSAIDs and similar agents are used initially for symptom control. |
| **Osteitis Condensans Illi** | **Age of Onset** — 20–40 y/o  
**Gender Predominance** — Female to male ratio = 9:1  
**Common Joints Involved** — Sacroiliac | When symptomatic, management includes anti-inflammatory approaches. Generally, self-resolving. |
<table>
<thead>
<tr>
<th>Type</th>
<th>Features</th>
<th>Management Issues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteitis Pubis</td>
<td><strong>Age of Onset</strong> — Varies, but with females during the reproductive years. <strong>Gender Predominance</strong> — Female predominant. <strong>Common Joints Involved</strong> — Symphysis pubis. An inflammatory process secondary to trauma, pelvic surgery, or childbirth. This is particularly true if complicated by infection. Possibly due to intraosseous venous congestion and/or infection. Radiographically appears as joint space widening, subchondral sclerosis, localized osteoporosis, and joint erosions.</td>
<td>Initial management may be with NSAIDs and rest. More severe cases may require oral or locally injected corticosteroids. In rare cases, arthrodesis is necessary.</td>
</tr>
<tr>
<td>Metabolic Crystal Deposition</td>
<td><strong>Gout</strong></td>
<td><strong>Age of Onset</strong> — &gt;30 y/o (in females, postmenopausal). <strong>Gender Predominance</strong> — Male. <strong>Common Joints Involved</strong> — First MTP joint of foot, feet, ankle, and knees. First attack often sudden and nocturnal, affecting the first MCP joint of foot. This may follow excess alcohol or meat intake. Fever is common during the acute attack. The joint is red and swollen. Dequamation and pruritis after the acute attack are common. Tophi (calcium urate deposits) appear after several attacks of gout and are found behind the ears, olecranon, prepatellar bursa, hands, and feet. There is a dramatic response to NSAIDs or colchicines during the acute attack. Those with gout should be evaluated for associated conditions including alcoholism, various nephropathies, myeloproliferative disorders, hypertension, and insulin resistance. Occurrence in 2nd and 3rd decade indicates hereditary disorders such as hypoxanthine-guanine phosphoribosyltransferase deficiency (hypoxanthemia) or gout. Dietary modification includes attention to hydration, low-purine diet (avoid meat, yeast, alcohol, beans, legumes), alcohol avoidance, lose weight. Medication avoidance includes hyperuricemic meds such as thiazide and loop diuretics, also avoid low-dose aspirin and niacin. Medical approach includes acute attacks; NSAIDs or corticosteroids, or colchicines (for inflammation). Long-term approach incorporates drugs to reduce serum uric acid and decrease tophi deposits; primarily allopurinol (xanthine-oxidase inhibitor) and probenecid. Low-dose colchicines are acceptable as prophylaxis for first 6 months.</td>
</tr>
<tr>
<td></td>
<td><strong>Calcium Pyrophosphate Deposition Disease (CCPD)</strong></td>
<td><strong>Age of Onset</strong> — &gt;50 y/o. <strong>Gender Predominance</strong> — Generally equal, dependent on cause. <strong>Common Joints Involved</strong> — Knee, symphysis pubis, hand, wrist, hip, shoulder, elbow, spine. CCPD crystal deposition in soft tissue occurs due to trauma, several metabolic diseases, and other causes. The general term is pseudogout. It is associated with metabolic disorders that include hemochromatosis, hyperparathyroidism, ochronosis, diabetes, hypothyroidism, Wilson’s disease, among others. There are various sub-types such as pseudogout, pseudohypermuritic arthritis, and pseudogenerative joint disease. The most common pseudo-gout may occur similar to gout, however, it occurs at a later age in most instances. May be asymptomatic or symptomatic. When symptomatic, pain and swelling occur. Joint aspiration reveals pyrophosphate crystals in synovial fluid. ESR is elevated during acute attacks. Radiographic changes include soft tissue swelling, radiolucent spots (urate crystals) are evident.</td>
</tr>
</tbody>
</table>

---

**TABLE 1–3**

**Selected Arthritic Disorders (continued)**

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<table>
<thead>
<tr>
<th>Table 1-3</th>
<th>Selected Arthritic Disorders (continued)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type</strong></td>
<td><strong>Features</strong></td>
</tr>
<tr>
<td>Hydroxyapatite Deposition Disease</td>
<td>Age of Onset — 40–70 y/o</td>
</tr>
<tr>
<td>Gender Predominance — Equal</td>
<td>Common Joints Involved — Shoulder, hip, cervical spine</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>Age of Onset — 20–40 y/o</td>
</tr>
<tr>
<td>Gender Predominance — Equal</td>
<td>Common Joints Involved — Hands, wrists, and feet</td>
</tr>
<tr>
<td>Hemochromatosis</td>
<td>Age of Onset — 40–60 y/o</td>
</tr>
<tr>
<td>Gender Predominance — Male to female ratio = 10:1 to 20:1</td>
<td>Common Joints Involved — Hip, knee, shoulder, wrist, hand</td>
</tr>
<tr>
<td>Alkaptonuria (Ochronosis)</td>
<td>Age of Onset — 30–40 y/o (present at birth though)</td>
</tr>
<tr>
<td>Gender Predominance — Equal</td>
<td>Common Joints Involved — Spine, hip and knee</td>
</tr>
</tbody>
</table>
### Selected Arthritic Disorders (continued)

<table>
<thead>
<tr>
<th>Type</th>
<th>Features</th>
<th>Management Issues</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alkaptonuria (Ochronosis)</strong> (continued)</td>
<td>not usually seen until age 20), discoloration of urine and ochronotic arthropathy that includes acute exacerbations of articular pain in the spine. Cartilage of nose and ears may appear brown but not blue on transillumination. Renal and prostate stones are common. Laboratory findings include urine that turns black on standing homogenously and a urine. Radiographically, there is accelerated DJD of the spine with eventual bamboo spine, often beginning with narrowing of the interpediculate ligament.</td>
<td></td>
</tr>
</tbody>
</table>
| **Pigmented Villonodular Synovitis (PVS)** | **Age of Onset** — 20–40 y/o  
**Gender Predominance** — Male (slight)  
**Common Joints Involved** — Knee, hip, elbow, ankle | Treatment is complete synovectomy. Irradiation has also been used in some patients. | A synovial proliferative disorder of unknown origin, although 50% of individuals report a history of trauma, usually occurs in one joint. In the hand or foot, a tendon involvement is termed giant cell tumor. Slowly developing joint pain with associated swelling, tenderness, and warmth. Aspiration may reveal hemorrhage. Radiographically, a “popcorn” appearance is seen with minimal preservation of joint space. Cystic erosions with hemorrhagic joint effusion can be seen. MRI is diagnostic. |
| **Hemophilic Arthropathy** | **Age of Onset** — 2–3 y/o  
**Gender Predominance** — Male  
**Common Joints Involved** — Knee and elbow commonly affected, although most other appendicular joints can be affected | At the first sign of hemarthrosis, infusion of factor VIII or IX is initiated. The involved joint is kept in as much extension as possible while NSAIDs and local icing are used for pain control. COX-2 inhibitors are preferred for chronic scenarios associated with hypertrophied synovium. Synovectomy, either open or arthroscopic, may be recommended. |
| **Infectious** | Arthritis may be secondary to bacterial, fungal, or viral infections, causing infiltration of synovial or periarticular tissues. | Antibiotic therapy is often given parenterally, and joint aspiration is performed as well as lavage and débridement dependent on degree of involvement. For fungal infections, amphotericin B and similar medications are needed. |
| | There are a number of risk factors for joint infections including: older age (over half of cases in patients over 60 years of age), joint surgery, intravenous drug use, alcoholism, diabetes, immunosuppressive illnesses or use of immunosuppressive medications, malignancy, chronic disease of the liver, lung, or kidney, skin infections, or malignancy. For acute arthritis, the most common bacterial cause is adult boil Neisseria gonorrhoeae. Others include Staphylococcus aureus, Streptococcus, and some gram-negative such as Enterobacter, Pseudomonas aeruginosa, and Salmonella marcescens. For chronic arthritis, primary causes include mycobacterium and fungi. | |

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### TABLE 1–3

<table>
<thead>
<tr>
<th>Type</th>
<th>Features</th>
<th>Management Issues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious (continued)</td>
<td>Early clinical signs are pain, swelling, and warmth. Radiographic changes with bacterial causes include early soft tissue and synovial swelling. After about 2 weeks, joint space narrowing and erosions begin to appear. Inflammatory markers will be elevated, with a WBC count with a decrease in viscosity and glucose. Gram-staining and culture will usually reveal the causative organism.</td>
<td></td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>Recurrent, migratory joint pain involving the knee and other large joints is seen in patients with familial hyperlipidemia. The arthritis can present as temporary inflammatory synovitis or periarthritis may cause myalgias or polymyalgia.</td>
<td>Management is with NSAIDs or similar approaches for the acute management. Long-term management may involve the use of HMG CoA reductase inhibitors; however, these may cause myalgias or polymyalgia.</td>
</tr>
</tbody>
</table>
examples. Further distinction can be made from a pattern of joint involvement. For example, OA tends to affect large joints such as the knee and hip, with eventual involvement in the hands (specifically first metacarpal phalangeal and distal interphalangeal joints), whereas RA tends to affect the metacarpal and proximal interphalangeal joints of the hand first and then larger joints. Gout tends to affect the first toe and knees primarily. The seronegatives tend to affect the sacroiliac joint or spine with possible affect in peripheral joints. Finally, associated systemic signs may help relate the arthritis to disorders such as LE, scleroderma, enteropathic arthritides (i.e., arthritis associated with inflammatory bowel disease), and so on.

Assembling and applying this information, if a middle-aged female presented with a polyarthropathy that included the hands but not the spine, without other systemic involvement, RA would be high on the list of differentials. If a young to middle-aged male presented with sacroiliac pain, no spinal pain, and involvement of a finger, Reiter’s or AS would be high on the list of differentials.

A review article by Margaretten et al evaluated the ability of a clinician to determine whether their adult patient has septic arthritis. The history indicators would be whether the patient is diabetic, has rheumatoid arthritis, HIV infection, skin infection, has had joint surgery, or a hip or knee prosthesis. Joint pain, a history of joint swelling, and fever were the only findings that occurred in more than 50% of patients. Other findings such as night sweats and rigors were inconsistent and often not found with septic arthritis. However, the addition of synovial fluid analysis for WBCs and percentage of polymorphonuclear cells from arthrocentesis were needed to confirm the likelihood prior to Gram stain and culture test results.

### Examination

#### Acute Traumatic Injury

An approach to acute injury evaluation initially focuses on neurovascular status distal to and local to the injury site. These neurovascular injuries often are secondary to fracture. Motor assessments with active and active resisted attempts evaluate both muscle and neural integrity. Sensory testing incorporates the use of a pin in an attempt to test pain perception and a paper clip for testing two-point discrimination in the fingers. Palpation of pulses is useful in determining major vascular injury. Although these tests are more applicable to extremity injury, injury to the spine requires the same diligent search for an intact neurovascular system. With these conditions reasonably eliminated, the specific sequence one uses is less important than the fact that the approach is comprehensive.

#### General Approach

However complex the orthopaedic evaluation may become, the basics remain the same regardless of which joints and/or surrounding structures are involved (Table 1–4). Basically, orthopedic testing attempts to (1) reproduce a patient’s complaint (i.e., elicit pain, provoke numbness/tingling, or reproduce popping or clicking); (2) reveal laxity; (3) demonstrate weakness; or (4) demonstrate restriction (orthopaedic evaluation, in the context of a chiropractor, also includes accessory motion evaluation at a joint). The possible caveats to these attempts are that

<table>
<thead>
<tr>
<th>Condition</th>
<th>Active ROM</th>
<th>Passive ROM</th>
<th>Resisted Movement</th>
<th>Key Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthritis/capsulitis</td>
<td>Painful at limit of range</td>
<td>Painful at limit of range</td>
<td>Usually painless within range of motion</td>
<td>Often specific capsular pattern of one or two restricted movement patterns</td>
</tr>
<tr>
<td>Tendinitis</td>
<td>Variable</td>
<td>Pain on stretch</td>
<td>Painful, especially if contracted in stretched position</td>
<td>Insertion of tendon is often tender or slightly proximal to insertion</td>
</tr>
<tr>
<td>Tendinosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tendon rupture</td>
<td>None</td>
<td>Full, painless</td>
<td>Weak, painless</td>
<td>Note displaced muscle belly</td>
</tr>
<tr>
<td>Ligament sprain</td>
<td>Decreased, limited by pain</td>
<td>Pain on stability challenge</td>
<td>Painless if full rupture, painful if partial</td>
<td>Overpressure laxity may indicate degree of damage</td>
</tr>
<tr>
<td>Muscle strain</td>
<td>Painful, often midrange</td>
<td>Passive stretch may increase</td>
<td>If resistance is sufficient, pain is produced</td>
<td>Check with resistance throughout full range of movement</td>
</tr>
<tr>
<td>Intraarticular body</td>
<td>Sudden onset of pain in a specific range of motion</td>
<td>Sudden onset of pain in a specific range of motion is also possible</td>
<td>Usually painless</td>
<td>An “arc” of pain with a “catching” or blockage is highly suggestive</td>
</tr>
<tr>
<td>Acute bursitis (deep)</td>
<td>Painful in most directions</td>
<td>Empty end-feel is often present</td>
<td>Isometric testing is often painful</td>
<td>Positional relief is less common than with muscle/tendon injury</td>
</tr>
</tbody>
</table>

Key: ROM: range of motion
pain may be due to many factors and is therefore non-specific (localization and injury pattern help better define); laxity may be normal for an individual (especially if bilateral) or pathologic; weakness may be due to reflex inhibition caused by pain (relatively nonspecific), laxity, muscle injury, or neurologic damage; and restriction to movement may be due to soft tissue or bony blockage.

The mechanics of orthopaedic tests have similarities regardless of any assigned name. Testing involves one of three approaches: (1) stretch, (2) compress, or (3) contract. When performing a named orthopaedic test, reflection on what is the intended use coupled with the understanding of what other structures may be challenged is imperative to appreciate and interpret fully the variety of patient responses possible. Although a test is designed to stretch a ligament, also stretched are muscles, tendons, and nerves. The same maneuver may elicit a positive response through compression of tissues. For example, a valgus force stretches the medial knee yet compresses the lateral knee. Although not the intended response, any pain response to a maneuver may provide important information if simple biomechanics are kept in mind.

Another general principle is that similar structures are tested similarly (Table 1–5).

<table>
<thead>
<tr>
<th>Structure</th>
<th>Initial Evaluation</th>
<th>Specific Imaging Evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone</td>
<td>Radiograph</td>
<td>MRI or CT for metastasis (nonspecific)</td>
</tr>
<tr>
<td>Tumor</td>
<td>Palpation, radiograph</td>
<td>Possible bone scan</td>
</tr>
<tr>
<td>Osteochondrosis/apophysitis</td>
<td>Palpation, radiograph</td>
<td>CT or possibly MRI</td>
</tr>
<tr>
<td>Fracture</td>
<td>Palpation, percussion, tuning fork, radiograph</td>
<td>Bone scan, SPECT scan, quantified CT, dual-energy absorptiometry</td>
</tr>
<tr>
<td>Stress fracture</td>
<td>Palpation, percussion, radiograph</td>
<td>Quantified CT, dual-energy absorptiometry</td>
</tr>
<tr>
<td>Osteopenia (osteoporosis)</td>
<td>Radiograph</td>
<td>MRI</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td>Radiograph</td>
<td>MRI</td>
</tr>
<tr>
<td>Soft Tissue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strain or rupture</td>
<td>Active resistance</td>
<td>For rupture, sonography, or MRI</td>
</tr>
<tr>
<td>Trigger points</td>
<td>Palpation</td>
<td>None</td>
</tr>
<tr>
<td>Atrophy</td>
<td>Observation</td>
<td>Electrodiagnostic studies</td>
</tr>
<tr>
<td>Myositis ossificans</td>
<td>Palpation, radiograph</td>
<td>CT</td>
</tr>
<tr>
<td>Muscular dystrophy</td>
<td>Muscle testing, LDH on lab</td>
<td>Electrodiagnostic studies</td>
</tr>
<tr>
<td>Tendon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tendinitis/tendinosis</td>
<td>Stretch and contraction</td>
<td>Sonography</td>
</tr>
<tr>
<td>Paratenonitis</td>
<td>Stretch</td>
<td>Sonography or MRI</td>
</tr>
<tr>
<td>Rupture</td>
<td>Lack of passive tension effect</td>
<td>Sonography or MRI</td>
</tr>
<tr>
<td>Ligament</td>
<td>Stability testing</td>
<td>MRI</td>
</tr>
<tr>
<td>Sprain or rupture</td>
<td>Palpation</td>
<td>MRI or bursography</td>
</tr>
<tr>
<td>Bursa</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bursitis</td>
<td>Palpation</td>
<td>MRI or bursography</td>
</tr>
<tr>
<td>Fascia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myofascitis</td>
<td>Palpation</td>
<td>None</td>
</tr>
<tr>
<td>Joint</td>
<td>Characteristic joint involvement, laboratory findings including rheumatoid factor, HLA-B27, ANA, and radiographic characteristics</td>
<td>CT for bone, MRI for soft tissue involvement</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Palpation, indirect radiographic findings</td>
<td>CT for facet joints (research only)</td>
</tr>
<tr>
<td>Subluxation/fixation (chiropractic)</td>
<td>Capsular pattern of restriction</td>
<td>MRI, joint aspiration</td>
</tr>
<tr>
<td>Synovitis</td>
<td>Restricted ROM, radiograph</td>
<td>CT or MRI</td>
</tr>
<tr>
<td>Joint mice</td>
<td>Observation and radiograph</td>
<td>CT</td>
</tr>
<tr>
<td>Dislocation/subluxation (medical)</td>
<td>Observation and radiograph</td>
<td>CT</td>
</tr>
</tbody>
</table>

Key: MRI, magnetic resonance imaging; CT, computed tomography; LDH, lactate dehydrogenase; HLA, human leukocyte antigens; ANA, antinuclear antibodies; ROM, range of motion; SPECT, single photon emission computed tomography.
trigger points. In two studies Shah et al.4, 5 evaluated the work serves as a road map for investigation. Of promise was conducted by Chen et al.6 These reactive trigger points than in the controls. Another study tonin, and norepinephrine. All levels were higher in the sis factor, and inflammatory fractions of interleukin, sero-

... substance P, calcitonin gene-related peptide, tumor necro-

to a normal control match. Using a microdialysis needle biochemical environment of a trigger point as compared to a normal control match. Their work serves as a road map for investigation. New studies help clarify the etiology and diagnosis of trigger points. In two studies Shah et al.4, 5 evaluated the biochemical environment of a trigger point as compared to a normal control match. Using a microdialysis needle and sampling B21 (an acupuncture point and myofascial trigger point) they were able to measure pH, bradykinin, substance P, calcitonin gene-related peptide, tumor necrosis factor, and inflammatory fractions of interleukin, serotonin, and norepinephrine. All levels were higher in the active trigger points than in the controls. Another study of promise was conducted by Chen et al.6 These researchers attempted to investigate the presence of taut bands of myofascial trigger points (MTP) using magnetic resonance elastography (MRE). There are some concerns regarding this study, but preliminary interpretation suggests that this technology may be able to identify and distinguish these MTP bands.

The reliability of soft tissue palpation has been evaluated for the spine and the extremities. In general, it is evident that soft tissue palpation findings are not as reliable as bony palpation among examiners. When specific sites in the extremities are exposure through specific positioning, however, the reliability may increase.7

Although orthopaedic testing is the standard for orthopaedists, more involved investigations are usually added by the chiropractor and/or manual therapist. The first is based on the work of Cyriax,8 which emphasizes the “feel” of soft tissue palpation, especially at end-range. Combined with this end-range determination, a selective tension approach is incorporated using the responses to active, active resisted, and passive movements to differentiate between contractile (muscle/tendon) and noncontractile (ligament/capsule and bursa) tissue. Another approach is to challenge specifically each joint to determine fixation or hypermobility. Finally, a functional approach to movement as proposed by Janda9 and Lewit10 is often used. This approach addresses the quality of movement and the “postural” tendencies toward imbalance of strength and flexibility of muscles.

Selective Tension Approach
Cyriax8 divided the quality of passive end-range at a joint into normal and abnormal. Some normal end-feels include the following:

- Soft tissue approximation—This is a soft end-feel that occurs when a muscle opposes another muscle, for example, when the calf muscles hit the hamstrings or the forearm hits the biceps on flexion.

- Muscular—This is an elastic end-feel that occurs when a muscle is stretched to its end-range. This occurs with straight leg raising with the hamstrings.

- Bone-on-bone or cartilaginous—This occurs when the joint anatomically stops, as occurs with elbow extension.

- Capsular—This occurs with a tight, slightly elastic feel such as occurs with full hip rotation. It is due to the elastic tension that develops in the joint capsule when stretched.

Abnormal end-feels include the following:

- Spasm—When muscle spasm is present, pain will prevent full range of motion.

- Springy block or rebound—This occurs when there is a mechanical blockage such as a torn meniscus in the knee or labrum in the shoulder. The end-range occurs before a full range of motion is attained.

- Empty—This occurs when there is an acute painful process such as a bursitis. The patient prevents movement to end-range.
Many examiners probably sense these different end-range palpation findings. They have not categorized them, yet interpret them intuitively.

Some examiners will equate timing of the onset of pain on passive testing with staging of injury as follows:

- Loose—This end-feel is indicative of capsular or ligamentous damage and is in essence the end-feel that is found with a positive ligament stability test.

- Pain felt before end-range is considered an acute process that would obviate the application of vigorous therapy.

- Pain felt at the same time as end-range is indicative of a subacute process and would be amenable to gentle stretching and mobilization.

- Pain felt after end-range is indicative of a chronic process that may respond to aggressive stretching and manipulation.

By taking the patient through passive range of motion (PROM) and active range of motion (AROM) and testing resisted motion, a clearer idea of contractile versus noncontractile tissue involvement may be appreciated (Figure 1–1 and Table 1–5). It should be evident that contractile tissue may be painful with either stretch or midrange contraction. If both findings are present, they should be present in opposite directions (e.g., contraction into flexion hurts anteriorly while passive extension does also). If end-range stretch is not painful but contraction at end-range is, the tendon of the involved muscle is likely involved. If pain is not found with active movement but passive movement into end-range causes pain, noncontractile tissue is probably involved. Active movement should not affect most noncontractile tissue unless it is compressed. This is more likely to occur at end-range. Cyriax's selective tension approach is a logical attempt to localize the involved tissue, yet until recently it has remained unchallenged. One study demonstrated a high interexaminer reliability using these methods. The interexaminer agreement was 90.5% with a kappa statistic of 0.875.11

An extension of the selective tension approach is to determine the effect of mild isometric contractions on restricted range of motion. If a patient provides a mild resistance for several seconds to the agonist and antagonist pattern of restriction (e.g., flexion/extension) and repeats this several times followed by an attempt at stretch by the examiner, a distinction between soft tissue or bony blockage to movement may be determined. For example, if a patient presented with a restriction to abduction of the shoulder, repetitive, reciprocal contraction (minimal contraction for 5 to 6 seconds) into abduction and adduction several times will increase the available range if soft tissue is the cause (Exhibit 1–1). Bony blockage from OA, fracture, or a torn labrum will result in little or no increase in motion with the same procedure.

**Functional Approach**

Traditional muscle evaluation involves a test of muscle strength only. Janda9 and Lewit10 and others12 advocate an approach that takes into account not only the quantity of contraction (strength) but also the quality of movement. There is a recognized natural imbalance in muscle strength. Not all muscles are created equal. It is known that small muscles are often phasic, required to react quickly to changes in the environment, whereas larger muscles are often tonic, posturally assigned. Certain movement patterns are biased. For example, supination is stronger than pronation and internal rotation of the shoulder is stronger than external rotation. This bias is in large part due to the size or number (or both) of muscles used in the movement pattern. Strength is also positionally dependent. Certain positions place at a disadvantage some muscles of a synergistic group.

There is another perspective with regard to muscle weakness and tightness that may affect evaluation and eventually management. An observation by Janda9 and Lewit10 is that there are crossed and layered patterns of weakness and tightness. For example, in the low back it is not uncommon to find a pattern of anterior weakness in the abdominal muscles associated with posterior tightness of the erector spinae (sagittal pattern). A vertical pattern is illustrated by the association of the tight erector spinae's being sandwiched between weak gluteal muscles inferiorly and weak lower trapezius muscles superiorly. These two planes create a "crossed" pattern whereby tightness of the erector spinae is associated with tightness of the iliopsoas, and weakness of the abdominal muscles is associated with weakness of the gluteal muscles. This pattern is relatively consistent throughout the body and is a reflection of two concepts: (1) muscles that function to resist the effects of gravity (postural muscles) have a tendency to become tight in sedentary people, and (2) muscles that function more dynamically are underused and become weak and prone to injury. Additionally, muscles that cross more than one joint are prone to tightness. For example, the rectus femoris, which crosses the hip and knee, is prone toward tightness, whereas the medialis obliquus, which does not cross a joint and is primarily a "dynamic" muscle, is often weak.

With the above concepts in mind, Lewit and Janda have focused on an observation of quality of movement with an emphasis on the timing and recruitment during a movement pattern. Often these two concepts overlap when the timing of the movement is a reflection of recruitment. For example, hip extension in a lying position requires a timing of contraction beginning with the
Musculoskeletal Complaints

Patients present with a musculoligamentous injury.

1. Passive range of motion (PROM) normal?
   - Yes
   - No
     2. AROM and PROM increased; resisted movement is painless and strong?  
        Yes
        - Consider hypermobility.
        No
        3. Active range of motion (AROM) lost and resisted movement weak and painless.
           Yes
           - Consider tendon rupture or neural compromise.
           No
           4. Pain on resisted movement, especially in a fully stretched position?
              Yes
              - Consider tendinitis or tenosynovitis.
              No
              5. AROM is painful; pain is increased with resisted movement at midrange?
                 Yes
                 - Consider muscle strain.
                 No
                 6. Ligament challenge reveals abnormal movement (may be painful or painless)?
                    Yes
                    - Ligament sprain.
                    No
                    7. PROM is decreased and painful?
                       Yes
                       - Equal restriction in both AROM and PROM; however, resisted motion in available range is painless and strong?
                          Yes
                          - Consider impingement or an intraarticular loose body.
                          No
                          8. Painful arc with AROM or PROM with a specific pattern of movement?
                             Yes
                             - Consider tendon rupture or neural compromise.
                             No
                             9. PROM increased with post-isometric relaxation attempt?
                                Yes
                                - Consider muscle splinting as cause of decreased ROM.
                                No
                                10. Consider bony blockage often due to advanced degenerative joint changes.
                                    No
                                    11. Consider ligament or capsular sprain or joint subluxation/fixation.
                                        Yes
                                        - Consider muscle strain.
                                        No
                                        12. No injury evident or patient is insincere or uncooperative. Repeat testing.
                                            No
                                            13. If multiple muscles are painful, consider vascular compromise, fibromyalgia, or psychologic problems.
                                                Yes
                                                - Consider hypermobility.
                                                No
                                                14. PROM is normal; PROM is painful at end-range?
                                                    Yes
                                                    - Consider impingement or an intraarticular loose body.
                                                    No
                                                    15. Consider ligament or capsular sprain or joint subluxation/fixation.
                                                        Yes
                                                        - Consider tendon rupture or neural compromise.
                                                        No
                                                        16. Consider tendinitis or tenosynovitis.
                                                            Yes
                                                            - Consider hypermobility.
                                                            No
                                                            17. Consider bony blockage often due to advanced degenerative joint changes.
                                                                Yes
                                                                - Consider impingement or an intraarticular loose body.
                                                                No
                                                                18. Consider muscle splinting as cause of decreased ROM.
                                                                    Yes
                                                                    - Consider hypermobility.
                                                                    No
                                                                    19. Consider tendinitis or tenosynovitis.
                                                                        Yes
                                                                        - Consider hypermobility.
                                                                        No
                                                                        20. Consider bony blockage often due to advanced degenerative joint changes.
                                                                            Yes
                                                                            - Consider impingement or an intraarticular loose body.
                                                                            No
                                                                            21. Consider tendon rupture or neural compromise.
                                                                                Yes
                                                                                - Consider hypermobility.
                                                                                No
                                                                                22. Consider tendinitis or tenosynovitis.
                                                                                    Yes
                                                                                    - Consider hypermobility.
                                                                                    No
EXHIBIT 1-1

Postisometric Relaxation, Propioceptive Neuromuscular Facilitation (PNF) Hold and Relax, and PNF Contract and Relax

Postisometric Relaxation
- Stretch the affected muscle to patient tolerance.
- Maintain the stretch position while the patient isometrically contracts the muscle for 6 to 10 seconds at a 25% effort against the doctor’s resistance.
- Instruct the patient to relax fully (taking in a deep breath and letting it out may help).
- Attempt a further stretch of the muscle with the patient relaxed.
- Repeat this procedure five or six times or until no further stretch seems possible (whichever comes first).

PNF Hold-Relax
- This technique is very similar to a postisometric relaxation approach; however, classically the patient attempts a maximum contraction of either the agonist or antagonist.
- Caution must be used with maximal contractions. The author prefers to start with a postisometric approach using a 25% contraction before proceeding to more forceful resistance.

PNF Contract-Relax
- This is a full isotonic contraction followed by a stretch into a new position.
- There are several variations of this technique. A popular one is called CRAC (contract-relax-antagonist-contract).

Accessory Motion
One of the indicators for manipulation or adjusting is blockage of accessory motion. Accessory motion is that subtle amount of bone-on-bone movement that is not under voluntary control. For example, although the humerus moves on the glenoid during abduction, there is a degree of movement measured in millimeters that is necessary yet not under the control of the shoulder abductor muscles. Determining whether accessory motion is available involves placing the joint in a specific position and attempting passively to move one bone on another. If the end-feel is springy, then joint play is available. If there is a perceived restriction, however, movement at the joint may be restricted. It is important to distinguish between the end-range descriptions of Cyriax and the end-feel of accessory motion. Cyriax is referring to the end-range of an extremity or spinal movement such as flexion, extension, abduction, or adduction. Accessory motion is palpated at the joint both with the joint in a neutral or open-packed position and also with a coupled movement pattern taken to end-range actively and passively. The joint would not be restricted by the tension of the capsule or muscle with the neutral position method. The active and passive techniques take advantage of the end-range position to determine whether the accompanying accessory motion is, in fact, occurring. There are specific guidelines for both assessment and application of treatment to accessory motion barriers.

Radiography and Special Imaging
When making choices regarding the need for radiographs or special imaging, it is important to keep one major question in mind: Is there a reasonably high expectation that the information provided by the study will dictate or alter the type of treatment or dictate whether medical referral is needed? If the answer is no, it is important to delay ordering expensive, unnecessary studies at that given time. As time passes, the answer to the question may change. Some secondary issues with regard to further testing are as follows:

- What are the risks to the patient?
- What is the cost? Are there less expensive methods of arriving at the same diagnosis?
The decision for the use of radiographs is based on relative risk. Patients often can be categorized into high- and low-risk groups by combining history and examination data. Many groups have developed similar standards for absolute or relative indications for the need for radiographs.\(^\text{15-17}\) Generally, for patients with joint pain, the following are some suggested indicators:

- significant trauma
- suspicion of cancer (unexplained weight loss, prior history of cancer, patients over age 50 years)
- suspicion of infection (fever of unknown origin above 100°F and/or chills, use of intravenous drugs, recent urinary tract infection)
- chronic corticosteroid use
- drug or alcohol abuse
- neuromotor deficits
- scoliosis
- history of surgery to the involved region
- laboratory indicators such as significantly elevated erythrocyte sedimentation rate, alkaline phosphatase, positive rheumatoid factor, monoclonal spiking on electrophoresis
- dermopathy suggestive of psoriasis, Reiter’s syndrome, melanoma, and the like
- lymphadenopathy
- patients unresponsive to 1 month of conservative care
- medicolegal requirements or concerns

Choice of imaging is based on the sensitivity and specificity of a given imaging tool, the cost, and the availability (see Table 1–5). In general:

- Radiography—Signs of many conditions, including cancer, fracture, infection, osteoporosis, and degeneration, often are visible. The degree of sensitivity is quite low with early disease, however.
- Magnetic resonance imaging (MRI) is extremely valuable in evaluating soft tissue such as tendons, ligaments, and discs. In evaluating the volume of tumor or infection involvement, MRI is also valuable. Spinal cord processes such as multiple sclerosis or syringomyelia are well visualized on MRI (Table 1–6).
- When attempting further to clarify the degree of bony spinal stenosis, the extent of fracture, or other bony processes, computed tomography (CT) is often a sensitive tool—better than MRI in many cases. Recent cerebrovascular events and some tumors are well visualized with CT.
- When the search is for stress fracture, metastasis to bone, or avascular necrosis, bone scans often provide valuable information.

When determining the degree of osteoporosis in a patient, dual x-ray radiographic absorptiometry is more sensitive than standard radiography.

In late 2007, a set of diagnostic imaging practice guidelines for musculoskeletal complaints in adults was released.\(^\text{18}\) The guidelines were the result of years of research including an extensive literature search, an external review by 12 chiropractic specialists for external review, and finally, a two-round modified Delphi process involving 149 international experts. The agreement on recommendations was quite high (approximately 85%). Generally, these guidelines are intended to inform clinicians as to the best scientific evidence currently available, and are intended to be used in conjunction with sound clinical judgment and experience. The hope is that in addition to identifying patients in need of further diagnostic workup, unnecessary use of radiographs, and therefore the time and cost for health care will be reduced, while maintaining or improving patient care.

A new approach being studied and utilized by physical therapists internationally is rehabilitative ultrasound imaging (RUSI).\(^\text{19-26}\) The concept is to use diagnostic ultrasonography (US) to evaluate morphological changes while therapy is being applied. By determining the best position for stretch or for the effects of care, RUSI is being advocated as an important adjunct to musculoskeletal management. For example, RUSI would be used to measure the cross-sectional area of a muscle or tendon.

**MANAGEMENT**

Conservative management of a musculoskeletal problem is based on several broad principles.

- Initial management involves a greater degree of passive care with a transition into active care dominance over time.
- The goals for patient management vary based on the acuteness of the problem.
- Rehabilitation progresses in a sequence: passive motion to active motion to active resisted motion (begins with isometrics and progresses to isotonics) to functional training.

Although traditionally it was the doctor’s role to be active and the patient’s to be passive with treatment, it is becoming clear that there is a point at which role switching is necessary. When a patient has acute pain, the goal is to reduce the pain and assist healing. Many of the treatment methods used with acute pain employ procedures that are doctor dependent. As the patient progressively improves, there should be a focus on the patient’s active participation in restoring normal function. Nelson\(^\text{27}\) has outlined some criteria for passive care (Figure 1–2). These
### Magnetic Resonance Imaging for the Chiropractor

<table>
<thead>
<tr>
<th>MRI Better Than CT</th>
<th>MRI Equal to CT</th>
<th>CT Better Than MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI of the Head</td>
<td>MRI of the Head</td>
<td>MRI of the Head</td>
</tr>
<tr>
<td>Severe headaches</td>
<td>Hydrocephalus</td>
<td>Fracture of the skull base</td>
</tr>
<tr>
<td>Visual disturbance</td>
<td>Brain atrophy</td>
<td>Fracture of the calvaria</td>
</tr>
<tr>
<td>Sensory-neural hearing loss</td>
<td></td>
<td>Cholesteatoma of inner ear</td>
</tr>
<tr>
<td>Primary brain tumor</td>
<td></td>
<td>Intracranial hemorrhage 1–3 days old</td>
</tr>
<tr>
<td>Metastatic brain tumor</td>
<td></td>
<td>Cerebral infarction 1–3 days old</td>
</tr>
<tr>
<td>Intracranial infection</td>
<td></td>
<td>Intracranial calcifications</td>
</tr>
<tr>
<td>Age-related OIS disease</td>
<td></td>
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<tr>
<td>Multiple sclerosis</td>
<td></td>
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<tr>
<td>Dementia</td>
<td></td>
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<tr>
<td>Chronic subdural hematoma</td>
<td></td>
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<tr>
<td>Posttraumatic evaluation of the brain</td>
<td></td>
<td></td>
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<tr>
<td>Intracranial hemorrhage older than 3 days</td>
<td></td>
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<tr>
<td>Cerebral infarction older than 3 days</td>
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<tbody>
<tr>
<td>Tumors or masses at the level of the foramen magnum</td>
<td>Spinal stenosis</td>
<td>Occult fracture of a vertebra</td>
</tr>
<tr>
<td>Chiari I malformation</td>
<td></td>
<td>Complex fracture of a vertebra</td>
</tr>
<tr>
<td>Cervical or thoracic herniated disc</td>
<td></td>
<td>Bony foraminal encroachment</td>
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<tr>
<td>Posttraumatic syrinx</td>
<td></td>
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<tr>
<td>Syringomyelia</td>
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<tr>
<td>Acquired immunodeficiency syndrome-related myelopathy</td>
<td></td>
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<tr>
<td>Multiple sclerosis of the spinal cord</td>
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<td></td>
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<tr>
<td>Posttraumatic epidural hematoma</td>
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<tr>
<td>Epidural metastatic disease</td>
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<tr>
<td>Epidural abscess</td>
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</tbody>
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<table>
<thead>
<tr>
<th>MRI of the Lumbar Spine</th>
<th>MRI of the Lumbar Spine</th>
<th>MRI of the Lumbar Spine</th>
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</thead>
<tbody>
<tr>
<td>Small lumbar herniation</td>
<td>Large lumbar herniation</td>
<td>Occult fracture</td>
</tr>
<tr>
<td>Foraminal herniation</td>
<td>Spinal stenosis</td>
<td>Hypertrophic bony overgrowth or spurring</td>
</tr>
<tr>
<td>Interruption of the posterior longitudinal ligament</td>
<td></td>
<td>Bony foraminal encroachment</td>
</tr>
<tr>
<td>Root sleeve compression</td>
<td>Spinal stenosis</td>
<td>Spondylolisthesis</td>
</tr>
<tr>
<td>Postoperative scar versus recurrent lumbar herniation (with gadolinium)</td>
<td></td>
<td>Evaluation of posterior element fusion</td>
</tr>
</tbody>
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<thead>
<tr>
<th>MRI of the Shoulder</th>
<th>MRI of the Shoulder</th>
<th>MRI of the Shoulder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posttraumatic bone bruise</td>
<td>Rotator cuff tear</td>
<td>Subtle-glenoid labrum tear</td>
</tr>
<tr>
<td>Arteriovenous malformation of the head</td>
<td></td>
<td>Evaluation of the gelenohumeral ligaments</td>
</tr>
<tr>
<td>Impingement syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lipoma (or soft tissue mass)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brachial plexus tumor</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>MRI of the Knee</th>
<th>MRI of the Knee</th>
<th>MRI of the Knee</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posttraumatic bone bruise</td>
<td>Meniscal tear</td>
<td>Evaluation of the meniscus following previous meniscectomy</td>
</tr>
<tr>
<td>Osteochondritis dissecans</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior cruciate ligament tear</td>
<td></td>
<td></td>
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<tr>
<td>Posterior cruciate ligament tear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collateral ligament tear</td>
<td></td>
<td></td>
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<tr>
<td>Patellar tendon abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td></td>
<td>Evaluation of the articular cartilage</td>
</tr>
<tr>
<td>Tumor</td>
<td></td>
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</tr>
</tbody>
</table>

Source: Courtesy of Murray Solomon, M.D., Redwood City, California.
Musculoskeletal Complaints

Evidence of hypomobility or hypermobility?

Presence of active inflammation or acute pain?

Evidence of deconditioned soft tissue, reduced endurance, or compromised balance/proprioception?

Manage for:
- Hypomobility to increase ROM (D)
- Hypermobility for stabilization (E)

Go to active care algorithm (Figure 1–5)

Consider:
- Potential chronic inflammatory disorders or pain behavior. Discharge or refer.

Discharge

Anchors:
- (A) — Passive care criteria: History of recent trauma, acute condition or flare up, inflammation, or dependency behavior
- (B) — Care for inflammation: PRICE, high-voltage galvanic stimulation, ultrasound, NSAIDs, contrast baths, therapeutic heat
- (C) — Care for pain: Mobilization, manipulation, acupuncture, trigger-point therapy, TENS, interferential stimulation, NSAIDs, protection, cryotherapy, heat
- (D) — Care for hypomobility: Passive stretch, assisted stretch, mobilization, manipulation, soft tissue massage
- (E) — Care for hypermobility: Taping, elastic support, brace, splint, cast, surgical repair, begin active stabilization

General Approach to Musculoskeletal Complaints

1. Care for inflammation might include the traditional approach of protection, rest, ice, and, if appropriate, compression and elevation. Modalities that are available include high-voltage galvanic stimulation, ultrasound, therapeutic heat, contrast baths, and nonsteroidal anti-inflammatory drugs (NSAIDs), or enzyme alternatives.

2. Options for care for pain include manipulation, mobilization, trigger-point therapy, transcutaneous electrical nerve stimulation (TENS), interferential stimulation, ice, cryotherapy, acupuncture, and NSAIDs (Table 1–7).

3. Care for hypomobility includes various forms of stretch, manipulation, mobilization, and soft tissue approaches such as myofascial release techniques.

4. Care for hypermobility includes protection with taping, casts, splints, or various braces.

Numerous techniques for stretching and soft tissue pain control are used. Exhibits 1–2 through 1–4 outline many of these approaches, including rhythmic stabilization, postsisometric relaxation, proprioceptive neuro-muscular facilitation (PNF) hold-relax and contract-relax techniques, cross-friction massage, spray and stretch, and myofascial release techniques (MRT or active resistive technique [ART])28, and Graston technique.

Recommendations for the frequency of manual therapy generally have been outlined by the Mercy Guidelines (Figures 1–3 and 1–4).29 A brief summary follows:

- If the condition is acute (≤6 weeks) and uncomplicated (no red flags indicating referral), there may be an initial trial treatment phase of 2 weeks at a frequency of three to five times per week.
- At 2 weeks the case is reevaluated (unless there is progressive worsening); if improving, the patient is given an education program regarding activities of daily living (ADL) and a graduated program of exercise and stretching, with treatment continuing for up to 8 weeks depending on the patient’s progress; if not improved, a 2-week trial with a different treatment plan is suggested.
- If after the second 2-week trial the patient has not improved, consultation or referral is suggested.
- Cases that will likely have a prolonged recovery include those with symptoms lasting longer than 8 days, severe pain, more than four previous episodes, or preexisting structural or pathologic conditions.

Active care criteria include decreasing pain and inflammation and an improvement in range of motion and joint mobility (Figures 1–5 and 1–6). There is a phase where passive and active care coexist. During this stage,

---

**TABLE 1–7**

<table>
<thead>
<tr>
<th>Physiotherapy Approaches for Musculoskeletal Complaints</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Various Transcutaneous Electrical Nerve Stimulation (TENS) Approaches</strong></td>
</tr>
<tr>
<td><strong>Type</strong></td>
</tr>
<tr>
<td>High Frequency</td>
</tr>
<tr>
<td>Low Frequency</td>
</tr>
<tr>
<td>Brief and Intense</td>
</tr>
<tr>
<td>Galvanic: 1–5</td>
</tr>
</tbody>
</table>

**Pulsed High-Volt Galvanic**

<table>
<thead>
<tr>
<th>Treatment Effect</th>
<th>Mode</th>
<th>Frequency (pps)</th>
<th>Type of Stimulation</th>
<th>Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduce edema</td>
<td>Continuous</td>
<td>High: 80–100</td>
<td>Sensory</td>
<td>20</td>
</tr>
<tr>
<td>Reduce muscle spasm</td>
<td>Continuous</td>
<td>High: 80–120</td>
<td>Sensory/motor</td>
<td>20–30</td>
</tr>
<tr>
<td>Autonomic response</td>
<td>Uninterrupted/Pulsed</td>
<td>High: 80–100</td>
<td>Sensory</td>
<td>20–30</td>
</tr>
<tr>
<td>Neuro/hormonal</td>
<td>Uninterrupted/Pulsed</td>
<td>Low: 2–5</td>
<td>MOTOR (twitch)</td>
<td>30–60</td>
</tr>
<tr>
<td>Muscle pumping</td>
<td>Surged: 1/3</td>
<td>Low: 30–50</td>
<td>MOTOR</td>
<td>20–30</td>
</tr>
</tbody>
</table>
Rhythmic Stabilization

A variation of hold-relax, this technique uses a reciprocal contraction of the agonist and the antagonist following the approach outlined below:

■ Stretch the involved muscle to patient tolerance.
■ Use a physician’s contact on both sides of a joint.
■ Ask the patient to contract with a 25% contraction in the direction of agonist contraction for 5 to 8 seconds.
■ Without resting, ask the patient to contract into the opposite direction for 5 to 8 seconds.
■ Repeat this procedure five or six times.
■ Ask the patient to relax.
■ Stretch into new position.
■ Repeat the above five or six times or until no more stretch appears available (whichever comes first).

Cross-Friction Massage and Spray and Stretch

Cross-Friction Massage

Cross-friction massage is a technique popularized by Cyriax. The rationale behind its use is somewhat dependent on the patient’s presenting phase of injury. For example, in subacute injury the intent is to align collagen for stronger scar formation. With chronic conditions the cross-friction approach is used to break up adhesions and increase blood supply. A secondary effect of cross-friction massage is a pressure anesthesia, which occurs after a couple minutes of application. There are several suggestions for the proper use of cross-friction massage:

■ It appears to be most effective with tendons and ligaments.
■ The tendon or ligament should be placed under slight tension (by stretching the involved structure) while cross-friction is performed.
■ The contact is skin on skin with no lotion.
■ The pressure is applied as a transverse motion (90° to the involved structure).
■ Monitoring the patient every 2 minutes for a total of 6 to 9 minutes is recommended.
■ Prior to application, some practitioners recommended ice; others recommend moist heat for approximately 5 minutes.
■ Treatment is given every other day for 1 to 2 weeks (up to 4 weeks maximum).

Spray (Cold) and Stretch

Although the technique of using fluoromethane spray for stretching muscles was popularized by Travell and Simons, concerns over damage to the ozone layer and increasing unavailability of the spray have led to a return to the use of ice. With the use of either tool, the technique of application has several common protocol components:

■ The muscle being stretched is placed in a position of mild to moderate stretch. Maintain this stretch while applying the cold stimulus.
■ The cold stimulus is applied in a series of linear strokes to the skin overlying the muscle and its associated pain referral zone. This is applied in the direction of pain referral.
■ Gradually increase the stretch while applying the cold.
■ Following the stretch, the skin should be briefly rewarmed with a moist heat pack.
■ The muscle should then be put through a full range of motion, passively and then actively (this is an attempt to avoid posttreatment soreness).
Several techniques have been developed and popularized under different technique names. Most techniques involve a stripping motion of a muscle. A combination of these techniques is found with MRT (or ART) as proposed by Leahy and Mock. These techniques are best used when a muscle is determined to be dysfunctional. This is accomplished through a combination of palpation, range of motion (ROM) findings, and muscle testing. This technique is not intended for acute injury (within 24 to 36 hours) or for ligaments and tendons that respond better to cross-friction massage. In essence, this is an extension of other myofascial or trigger-point approaches.

Skin lotion should be used when possible. Following is a summary of this approach. There are four levels. Use the highest level that patient tolerance permits.

**Level 4**
- Place the muscle in its shortest position.
- Apply a firm contact to the muscle just distal to the site of palpable adhesion.
- Ask the patient to move the limb actively through an antagonist pattern (if the joint is in extension, the patient flexes), elongating the muscle.
- Always maintain a fixed contact on the patient so that the adhesions are forced under the contact point.

**Level 3**
- Place the muscle in its shortest position.
- Apply a firm contact to muscle just distal to the site of palpable adhesion.
- Passively move the limb through an antagonist pattern, elongating the muscle.
- Always maintain a fixed contact on the patient so that the adhesions are forced under the contact point.

**Level 2**
- Place the muscle in a stretched position (creating tension).
- Apply muscle-stripping massage (along the direction of muscle fibers) using a broad contact, concentrating on areas of adhesion.

**Level 1**
- Place the muscle in a neutral position (no tension).
- Apply muscle-stripping massage, concentrating on areas of adhesion.

Treatment usually involves several passes over the muscle, treatment every other day, and resolution within the first few treatments.

Adjunctive care involves prescription of exercises for the involved muscle, starting with facilitation.
**Figure 1–3  Acute/Uncomplicated Cases—Algorithm**

Annotations

(A)—In general, more aggressive in-office intervention may be necessary early.

(B)—Promotion of rest, elevation, active rest, and remobilization as needed.

(C)—Supportive maintenance care is inappropriate.

(D)—Complicated case factors may include radicular pain, anomalies, etc.

Supportive care using passive procedures (including CMT) may be necessary.

Subacute Episode
Symptoms > 6, < 16 weeks

Chronic Episode
Symptoms > 16 weeks

— Passive care including CMT for exacerbations only, prn
— Supervised rehabilitation and lifestyle changes

Discharge or referral

Subacute Episode
Symptoms > 6, < 16 weeks

— Passive care including CMT not generally to exceed twice weekly
— Active care, dissuasion of pain behavior, education, exercises, and/or rehabilitation
— Supportive care inappropriate

Supportive care using passive procedures (including CMT) may be necessary

Is patient insincere or noncompliant with care/treatment?

Discharge or referral

Is patient insincere or noncompliant with care/treatment?

Continue to preepisode status 6–16 weeks therapy goal (A)

Discharge and/or elective care

May not return to preinjury status. Consider declaration of maximum therapeutic benefit. (A)

Musculoskeletal Complaints

Evidence of joint hypermobility?

Manage for:
Support to prevent tissue damage and stabilization exercises (B) (4 to 16 weeks)

Evidence of improved mobility?

Go to Active Exercise Algorithm (Figure 1–6)

Evaluate for potential permanent joint changes and adjust long-term goals

Evidence of joint hypomobility?

Manage with:
Active assisted ROM, PNF, active ROM, and/or continued passive ROM (2 to 8 weeks)

Evidence of improved stability?

Go to Active Exercise Algorithm

Evidence of deconditioned soft tissue?

Reduce endurance capacity?

Proprioception or balance deficits

Discharge

Go to Active Exercise Algorithm

Manage with:
High repetition w/resistance; interval/circuit training; cardiovascular training; to treatment goal

Manage with:
Balance board; gymnastic balls; agility drills; plyometric training; job/sport skills training; to treatment goal

An annotations
- **Active care criteria:** Decreasing pain and inflammation, tolerance to increasing activity, improvement in joint motion, and favorable response to passive care
- **Stabilization exercises:** Includes isometric and limited-arc dynamic efforts


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General Approach to Musculoskeletal Complaints

Figure 1–6 Active Care: Exercise—Algorithm

1. Musculoskeletal condition responsive to passive and/or active management

2. Patient tolerates: Multiple-angle isometrics for 2- to 4-week trial?
   - No → Go to Box 2 or Discharge
   - Yes → Consider: One 1- to 3-week trial of active ROM and pain control
     Assess for compliance
     Retest for tolerance
     Go to Box 2 or Discharge

3. Patient tolerates: Manual resistance through pain-free ROM?
   - No → Go to Box 2 or Discharge
   - Yes → Consider: Additional 1 to 3 weeks of active ROM and isometrics
     Assess for compliance
     Retest for tolerance
     Go to Box 2 or Discharge

4. Patient tolerates: Short-arc dynamic exercise
   - Yes → Go to Box 2 or Discharge
   - No → Consider: Additional 1 to 3 weeks of manual resistance through ROM
     Assess for compliance
     Retest for tolerance
     Go to Box 4 or Discharge

5. Patient tolerates: Extended ROM, moderate resistance, 2–4 sets/6–12 reps, multijoint motions, and progressive resistance?
   - Yes → Go to Box 2 or Discharge
   - No → Consider: Additional 1 to 3 weeks of short-arc dynamic exercise
     Assess for compliance
     Retest for tolerance
     Go to Box 6 or Discharge

6. Patient tolerates: Heavy resistance, 3–5 sets/5–8 reps, work hardening?
   - Yes → Go to Box 2 or Discharge
   - No → Consider: Additional 1 to 3 weeks of extended ROM, moderate resistance
     Assess for compliance
     Retest for tolerance
     Go to Box 8 or Discharge

7. Consider: Additional 1 to 3 weeks of extended ROM, moderate resistance
   Assess for compliance
   Retest for tolerance
   Go to Box 8 or Discharge

8. Consider: Additional 1 to 3 weeks of short-arc dynamic exercise
   Assess for compliance
   Retest for tolerance
   Go to Box 6 or Discharge

9. Consider: Additional 1 to 3 weeks of active ROM and pain control
   Assess for compliance
   Retest for tolerance
   Go to Box 2 or Discharge

10. Consider: Additional 1 to 3 weeks of manual resistance through ROM
    Assess for compliance
    Retest for tolerance
    Go to Box 4 or Discharge

11. Consider: Additional 1 to 3 weeks of short-arc dynamic exercise
    Assess for compliance
    Retest for tolerance
    Go to Box 6 or Discharge

12. Discharge or go to Box 11 Active Care Algorithm (Figure 1–5)

isometrics performed in limited arcs are helpful initiators and facilitators for a progressive exercise program. Progressing through a graded program involves setting criteria for passing each stage. The most common criteria are range of motion, strength levels, and performance without pain.

Active care elements include training to increase range of motion, strengthening primary and secondary stabilizers of a given joint or region, increasing the endurance capabilities of the muscles, proprioceptively training for balance and reaction time, and finally, functionally training for a specific sport or occupational task. Each element involves different training strategies (Table 1–8 and Exhibits 1–5 through 1–7).

**Strength and Endurance**

Strengthening begins with facilitation. This is accomplished either through isometrics performed at every 20° to 30° or rhythmic stabilization using elastic tubing, performing very fast, short-arc movements for 60 seconds or until fatigue or pain limits further performance. Strengthening may then progress to holding end-range isometrics with elastic tubing for several seconds, and slowly releasing through the eccentric (negative) contraction. In some cases, these end-range isometrics may be performed against gravity only first. If these elements are strong and pain free, progressing to full-arc isotonics using weights or elastic tubing may be introduced. It is best to begin with three to five sets of high repetitions (12 to 20) using 50% to 70% of maximum weight. After 1 to 3 weeks of this training, progression through a more vigorous strengthening program may be determined by the daily adjustable progressive resistance exercise (DAPRE) approach31 (although the exercises are performed every other day). This is a pyramid approach using lower weight with more repetitions and progressing through sets to higher weight and fewer repetitions. The last number of repetitions performed determines the working weight for the next workout.

**Proprioceptive Training**

Proprioceptive training incorporates various balance devices such as wobble boards, giant exercise balls, and minitrampolines. The intention is to have the body part react to changing support as quickly as possible and to integrate the rest of the body in this attempt.

**Functional Training**

Functional training is based on the requirements of a given sport or occupational activity and requires a knowledge of the biomechanics involved. Various PNF techniques may be employed. Simulated task performance is another approach for occupational retraining.

**Nutritional Support**

The nutritional support needed for musculoskeletal healing is based on recommendations made by Gerber.32

- In the inflammatory phase of healing, proteolytic enzymes, bioflavinoids, and vitamins C and E are recommended. Bromelain in doses of 1200 mg/d of

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**TABLE 1–8**

<table>
<thead>
<tr>
<th>Set</th>
<th>Weight</th>
<th>Repetitions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1/2 working weight</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>3/4 working weight</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>Full working weight</td>
<td>Maximum</td>
</tr>
<tr>
<td>4</td>
<td>Adjusted working weight (based on 3rd set)</td>
<td>Maximum</td>
</tr>
</tbody>
</table>

- Use the following table to determine working weight for 4th set (Based on 3rd set number of repetitions)
- Full working weight (3rd set) of the next training session is based on number of repetitions performed in the 4th set

<table>
<thead>
<tr>
<th>No. of Repetitions</th>
<th>For 4th Set</th>
<th>Next Session Full Working Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–2</td>
<td>Decrease 5–10 lb</td>
<td>Decrease 5–10 lb</td>
</tr>
<tr>
<td>3–4</td>
<td>Decrease 0–5 lb</td>
<td>The same</td>
</tr>
<tr>
<td>5–6</td>
<td>The same</td>
<td>Increase 5–10 lb</td>
</tr>
<tr>
<td>7–10</td>
<td>Increase 5–10 lb</td>
<td>Increase 5–15 lb</td>
</tr>
<tr>
<td>11 or more</td>
<td>Increase 10–15 lb</td>
<td>Increase 10–20 lb</td>
</tr>
</tbody>
</table>
**Eccentric Exercise Protocols**

**General Comments**
- Eccentrics are usually begun in the subacute phase of healing.
- Although there is some disagreement, the initial phase begins somewhere between 3 and 7 days after injury depending on severity.
- The superiority of eccentrics over concentrics occurs only during the first 19 days postinjury.
- A load of up to 20% above a one-repetition maximum is considered safe.
- It is suggested by the literature to perform between 3 and 20 repetitions with a three-set maximum; two to three times per week. Two times per week will probably prevent delayed-onset muscle soreness (DOMS).
- Rest periods are not as important due to the low oxygen demand. Somewhere between 30 seconds to 1 minute is sufficient.
- Training begins with slow progressing to faster repetitions.
- Two concerns are chance of overload injury and DOMS.
- Generally, there are three phases of training. An example for the lower extremity follows:
  1. Two-leg concentric/eccentric training is followed by two-leg concentric/injured leg eccentric work.
  2. Slow, submaximal, single-leg eccentrics are performed. The first two phases are usually completed in 3 weeks or less.
  3. Functional eccentrics are performed in preparation for plyometrics. This phase usually takes 2 to 3 weeks to complete.

**Functional Eccentrics for the Lower Extremity**
A sample of a functional eccentric program would include the following:
1. One-leg step-up onto 12-inch stool; noninvolved leg steps up first, down last
2. One-leg step-up; involved leg steps up first, down last
3. Repeat with 18-inch step height
4. Slow quarter squats
5. Rapid quarter squats
6. Slow parallel squats
7. Rapid parallel squats

**Curwin and Stanish**
1. Static stretching for 15–30 seconds is repeated three to five times.
2. Eccentric exercise is begun with gravity or light weights. For the first 2 days they are performed slowly. During days 3–5 they are performed at moderate speed. On days 6 and 7 the exercises are performed quickly. Three sets of 10 are performed.
3. After the eccentric phase, a repeat of the static stretching phase is performed.
4. Follow with 5–10 minutes of icing.

Curwin and Stanish feel that there should be some pain felt in the third set. If not, the resistance should be increased slightly. If pain is felt in the first two sets, weight should be decreased slightly.
EXHIBIT 1–6

Advanced Training Approaches

Russian Stimulation Protocol

- Place one electrode over the muscle and one over the associated nerve root.
- Use a 2500-Hz carrier wave; modulate at 50 pulses per second.
- Increase intensity to patient tolerance.
- Use 10-second maximum contraction with 50-second rest periods equaling 10 contractions in 10 minutes.
- Use three to five treatments per week for 5 to 7 weeks for a total of 23 to 35 treatments (2-day rest period per week).
- Protocol is used one time per year, best at night and not before or after strenuous exercise.

Plyometrics for the Lower Extremity

- Plyometrics are advanced exercises used only under the following conditions:
  1. Strength and flexibility are preinjury.
  2. Static stability is demonstrated with the following:
     - single-leg stance
     - single-leg quarter squat
     - single-leg half squat
   All can be performed for 30 seconds with eyes open and closed.

- A plyometric workout should be sport specific and include the following (general conditioning):
  1. Warm-up for 10 to 20 minutes
  2. Low-intensity drills; 3 to 5 exercises; 10 to 20 repetitions
  3. Moderate-intensity drills: 3 to 4 exercises; 5 to 8 repetitions
  4. High-intensity drills: 2 to 3 exercises; 10 to 20 repetitions

- A plyometric protocol begins with horizontal and progresses to vertical movements.
- Horizontal progression is as follows:
  1. Double-leg forward hopping in a straight line
  2. Side-to-side hopping, double leg
  3. Combination of side-to-side and forward hopping
  4. Follow with single-leg progression following the above sequence

- Vertical progression is as follows:
  1. Jump from the floor to a box and back down, starting with 6 in box and progressing to 12, then 18, then 20.
  2. Jump in a line using boxes of variable height.

Never use plyometrics for an athlete with quadriceps or patellar tendinitis.
**EXHIBIT 1–7**

**Classic Elastic Tubing Protocol**

**Facilitation**
A fast midrange movement is performed for 30 to 60 seconds or until painful. The number of sets is determined by the overall status of the patient. When this can be performed pain free for 2 or 3 days, move on to the next phase.

**Strength**
A slow full-range movement is performed and held for an isometric contraction of up to 30 seconds at end-range. This is followed by a slow eccentric phase (at least twice as long as the concentric). Rest for 10 seconds and perform again for up to 10 repetitions (pain or fatigue dependent). When performed for 2 or 3 days pain free, move to next phase.

**Endurance**
A fast full-range movement is performed at the rate of one per second. This may be performed for 50 to 60 seconds or until pain or fatigue is felt. Several sets may be performed with resting phases of 30 seconds. When this is possible for 2 or 3 days pain free, the patient has the option of progressing to pulley or free-weight exercise.

- The thickness of the tubing and the length determine the resistance.
- Thicker, shorter tubing is more resistant and requires more patient effort.
- Resistance increases throughout the concentric contraction and decreases through the eccentric phase.

**Variations of Elastic Tubing Exercise Protocols**
- Currently, short-arc, fast repetitions are used for stabilization. May be used every 20° or so or may focus on position of instability (e.g., 20° to 30° knee flexion for anterior cruciate ligament tears).
- Eccentric focus only for tendinitis. For example, place knee in final position of flexion or extension and resist tubing while lengthening the muscle. For example, extend knee, apply tubing behind, and gradually allow tubing to overcome resistance; end position of knee flexion.
- Sports cord training
- Closed-chain exercise: squats or seated foot dragging
- Functional PNF diagonal pattern training

*Note: Always ice after any of the above exercises.*

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2400 μg potency taken between meals for several days may be beneficial.

- In the proliferative phase, arginine, glycine, proline, vitamins A and C, pantothenic acid, and zinc may be of benefit. Connective tissue repair may be aided with glycosaminoglycans, manganese, and chondroitin sulfate.
- Fracture healing may be enhanced with adequate dietary calcium, vitamin D, phosphorus, and magnesium; microcrystalline hydroxyapatite may also be of benefit (6 to 8 g/d).

**The Anti-Inflammatory Diet**

Linoleic acid from n-6 fatty acids is converted to arachadonic acid and through several steps into prostaglandin-E2 (PGE2) and other pro-inflammatory eicosanoids. It may be that many chronic diseases and cancers are in part maintained or supported by this pro-inflammatory environment. Breast cancer is one example. One study indicates that women with high n6 fatty acid intake and a low n3 fatty acid intake were more prone to develop breast cancer. N-6 fatty acids are found in high amounts in most seeds (and their oils) as well as in grains and their manufactured varieties (e.g., flours, pasta, cereal, chips, desserts). Meats, dairy fats, and shellfish are also converted to arachadonic acid which is converted to PGE2. There is a belief that a ratio of n6 to n3 fatty acids should be close to 1:1 for humans to maintain an anti-inflammatory internal environment. Ratios in the modern diet can be as high as 10–30:1. If a diet consisted of less grain and seed and more vegetables, fruit, and fish, the
A proper balance of n6/n3 would be maintained. It is possible to buy eggs and beef that have a higher n3 content. ω-3 fatty acids may also be supplemented in the form of fish oil (obtainable from eicosapentaenoic acid and docosahexanoic acid (EPA/DHA) and alpha-linolenic acid (ALA) from flaxseed oil. Typical doses for EPA/DHA are 1 to 2 grams per day and for ALA, 2 grams per day.

A popular theory regarding the cause of many diseases that appear autoimmune is that both molecular mimicry and a “leaky gut” are factors in establishing an autoimmune response in the body. The molecular mimicry theory is an extrapolation of the viral mimicry theory that is believed to be the mechanism by which some diseases such as multiple sclerosis are activated. The dietary version suggests that, in genetically susceptible individuals, certain undigested food particles can mimic human protein, such as collagen, and elicit an autoimmune response. For example, bovine serum albumin (BSA) found in cow’s milk may be considered by the body as an antigen, and given that it is similar in sequence to human collagen may cause an autoimmune reaction. Another example is glycine-rich protein found in grains and legumes, which has a similar protein sequence to connective tissue. Other suspected agents include wheat germ agglutinin (WGA) found in wheat, phytohaemagglutinin (PHA) found in kidney beans, and peanut lectin (PNA) found in peanuts.

In addition to digestion, the gastrointestinal system, specifically the intestinal lining, provides an immune defense for the body. Immune defense is accomplished partly by the intestinal mucosa that acts as a physical barrier, by intestinal secretions (e.g., secretory IgA antibodies), and via intramural lymphocytes. One suggested test for the leaky gut syndrome is the lactulose-manitol test.

Dietary support for providing an intact and functioning intestinal lining includes (see Table 1–9):

- Glutamine—acts as a fuel source for intestinal cell maintenance and repair
- Vitamins C and E, lipoic acid, zinc, and ginkgo biloba—acts as antioxidants, protecting the mucosal lining from free-radical damage
- DGL (deglycyrrhizinated licorice)—thought to increase cell wall integrity of mucosal cells
- NAG (N-acetyl glucosamine)—helps to heal extracellular tissue and may decrease binding of some lectins
- Probiotics—believed to counteract harmful bacteria
- Hydrochloric acid and digestive enzymes—believed by some that if food particles are digested with the assistance of supplemental HCL and digestive enzymes, the less likely it will be that antigenic responses will occur.

Modification of the Inflammatory Response

Aspirin, non-steroid anti-inflammatory agents (NSAIDs), and cyclooxygenase inhibitors (COX) block the cyclooxygenase enzyme that converts arachidonic acid to prostaglandin E-2, decreasing or blocking inflammation. Also, corticosteroids inhibit phospholipase A2, which inhibits arachidonic acid release from phospholipids in the cell membrane. Cell-signaling molecules, which stimulate genes, induce the expression of the COX enzyme. Aspirin, NSAIDs, and corticosteroids inhibit binding of cell-signaling molecules such as NFκB, which reduces inflammation (see Exhibit 1–8). Conversely, NFκB activation induces COX-2 activation, which leads to inflammation. The expression of the coding gene for COX-2 for the production of prostaglandins is transcriptionally regulated by NFκB. It is in the cytoplasm and is bound to its inhibitor. Free radicals release NFκB from the inhibitor, which then moves into the nucleus to activate genes responsible for COX-2 activation.

Green tea polyphenols, resveratrol from red wine, vitamins C and E, curcumin, and glutathione reduce the activation of NFκB. It is possible that carotenoids and flavonoids also have similar actions. Also, the anti-inflammatory omega-3 fatty acids are found in green vegetables, most fish, wild game, grass-fed meat, and EPA/DHA fish oil.

Table 1–10 presents a general nutritional approach to tendinosis and other soft tissue injury.
<table>
<thead>
<tr>
<th>Substance</th>
<th>How Might It Work?</th>
<th>Dosage</th>
<th>Special Instructions</th>
<th>Contraindications and Possible Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucosamine sulfate</td>
<td>Stimulates the rebuilding of damaged cartilage</td>
<td>500 mg three times per day</td>
<td>Take with meals. Take 6–8 weeks to determine effect</td>
<td>No contraindications. May cause some gastrointestinal upset. Does not interfere with other anti-inflammation drugs. Some products processed with sodium chloride; use caution with patients who are hypertensive</td>
</tr>
<tr>
<td>Boswellia</td>
<td>Decreases inflammation</td>
<td>150 mg three times per day (for example, if extract contains 37.5% boswellic acids, need 400 mg of extract taken three times per day)</td>
<td>Take for 8 to 12 weeks</td>
<td>None at recommended dosage. Avoid Equisetum palustre, another species of horsetail that contains toxic alkaloids. Occasional gastrointestinal upset. Some caution about patients with manic-depression switching from depression to a manic episode. Apparently safe in pregnancy.</td>
</tr>
<tr>
<td>Horsetail</td>
<td>Decreases inflammation</td>
<td>Taken as a tea at 1–4 g/day; tincture would be taken as 2–6 ml three times per day</td>
<td>None</td>
<td>None. Avoid Equisetum palustre, another species of horsetail that contains toxic alkaloids. Occasional gastrointestinal upset. Some caution about patients with manic-depression switching from depression to a manic episode. Apparently safe in pregnancy.</td>
</tr>
<tr>
<td>SAM (S-adenosylmethionine)</td>
<td>Possibly raises levels of dopamine</td>
<td>1600 mg/day</td>
<td>None</td>
<td>None. Serum creatinine levels may increase in patients with renal insufficiency. Occasional gastrointestinal upset. Some caution about patients with manic-depression switching from depression to a manic episode. Apparently safe in pregnancy.</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Antioxidant</td>
<td>100–300 IU/day</td>
<td>None</td>
<td>None. Occasional gastrointestinal upset. Some caution about patients with manic-depression switching from depression to a manic episode. Apparently safe in pregnancy.</td>
</tr>
<tr>
<td>Niacinamide</td>
<td>Form of vitamin B3, may relieve symptoms and increase mobility</td>
<td>250 mg of niacinamide or nicotinamide 4–16 times/day</td>
<td>Improvement may take 3–4 months of supplementation</td>
<td>None. Occasional gastrointestinal upset. Some caution about patients with manic-depression switching from depression to a manic episode. Apparently safe in pregnancy.</td>
</tr>
<tr>
<td>Vitamin C, iron (glycinate), and alpha-ketoglutaric acid</td>
<td>Required for hydroxylation of L-proline to L-hydroxyproline needed for quality collagen production</td>
<td>Vitamin C — 3000–6000 mg/day in divided dosages</td>
<td>None</td>
<td>Use caution with high dosages of vitamin C; it may lead to diarrhea or urinary tract irritation in some people.</td>
</tr>
</tbody>
</table>

Note: These substances have not been approved by the Food and Drug Administration for the treatment of this disorder.

Chondroitin sulfate is occasionally suggested for OA. Probably same effect or less than glucosamine sulfate. If taken, typical level is 400 mg three times per day.
EXHIBIT 1–8

Contributors and Mediators of Inflammation: A Simplified Presentation

Possible Inhibitors of Inflammation

- Antioxidants (Vitamins E, A, C, phytochemicals)
- NSAIDs, Aspirin, COX inhibitors
- Tumeric, ginger, bioflavinoids, boswellia

Pro-Inflammatory

- Free Radicals
- Meats, dairy fats, shellfish, etc.
- Omega-6 Fatty Acids (Grains, “seed” oils)

Inhibition

Arachadonic Acid

Conversion

Linoleic Acid

Corticosteroids

Inhibition

Phospholipase A2

DGLA

Cyclooxygenase Enzyme

Cell Membrane Phospholipids

Prostaglandin E2

Thromboxane A2

Inhibition

From arachadonic acid

Prostaglandin E1

Thromboxane A1

Prostaglandin E3

Thromboxane A3

Release of pro-inflammatory cascade including histamine, serotonin, etc.

Legend:

COX = cyclooxygenase
EPA = eicosapentaenoic
DHA = docosahexaenoic
DGLA = dihomo-gamma-linolenic acid
NSAIDS = non-steroidal anti-inflammatory drugs

Fish oil

DHA  EPA

Alpha-linolenic acid

Omega-3 Fatty Acids (Leafy greens, flaxseed oil, grasses)

Anti-inflammatory mediators: reduce inflammation, pain, and thrombosis

General Nutritional Approach to Tendinitis/Tendinosis and Other Soft Tissue Injury

<table>
<thead>
<tr>
<th>Substance(s)</th>
<th>Recommended Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proteolytic enzymes (trypsin, chymotrypsin, bromelain)</td>
<td>Bromelain at 1200 mg/day of 2400 mcu (milk clotting units) in divided doses in between meals is at the high end of recommended dose. This is for acute inflammatory phase only (for several days only). Contraindicated in patients with bleeding tendencies (or peptic ulcer), systemic infection, or allergy to source product such as pineapple, pork, beef, or papaya.</td>
</tr>
<tr>
<td>Bioflavinoids (quercetin, hesperidine, rutin, etc.)</td>
<td>600–1800 mg/day. Often taken at 200 mg every 2 waking hours. Taken before peak of inflammatory phase.</td>
</tr>
<tr>
<td>Herbs—Boswellia, ginger, tumeric, cayenne</td>
<td>Boswellia—400 mg, ginger—300 mg, tumeric—200 mg, cayenne—50 mg taken every 2 waking hours during inflammatory phase.</td>
</tr>
</tbody>
</table>

APPENDIX 1–1

Web Resources

General Orthopedics and Arthritis
National Institute of Arthritis and Musculoskeletal and Skin Diseases
(877) 220-4267; www.niams.nih.gov
The Arthritis Society
(800) 321-1433; www.arthritis.ca
American Academy of Orthopaedic Surgeons
(800) 824-BONES (26637); www.aaos.org

Autoimmune Disorders
American Autoimmune Related Disease Association
(800) 598-4668; http://www.aarda.org

APPENDIX 1–2

Consensus Document for the Operationally Defined Use of I.C.D. Codes
Palmer Chiropractic College West Clinics
The use of the International Classification of Disease (ICD) Codes is varied due to:
- lack of agreement on the definitions and criteria for a given diagnosis
- lack of a specific code for a specific disorder
- unintentional, inappropriate use due to lack of training in the use of ICD codes
- intentional, inappropriate use to provide a patient representation that is more conducive to reimbursement by an insurance company

The lack of consistency is often based on a lack of understanding regarding the use of codes. Following are some general guidelines regarding the appropriate use of ICD codes based on code format:

- Code the primary diagnosis first followed by secondary, tertiary, and so on. Code any coexisting conditions that affect treatment.
- Code to the highest degree of specificity. Use the fourth of fifth digit when available.
- Qualify each diagnosis with a description of whether acute, chronic, traumatic, non-traumatic, and include severity of pain when appropriate.
- Codes identified as NEC (not elsewhere classifiable) are to be used when there is lack of information to be more specific in the diagnosis.
- Codes identified as NOS (not otherwise specified) indicates the code is unspecified, and although you have enough information, there is no apparent clear match. If possible a more specific code should be investigated (search alphabetical index).
- Pay attention to the main category title and description for each major code number and note includes and exclude lists.
- Pay attention to codes requiring an additional code when (1) more information gives a more complete picture of the patient, and (2) when an underlying disease must be coded first.
- Code only known diagnoses; do not code “rule-out” diagnoses or diagnoses documented in chart as “probable” or “suspected.”

In an effort to reduce variation, the Palmer-West clinicians have agreed to a narrowing of the number of ICD codes used and the criteria needed for a given code or group of codes. The reduction in the number of codes used is based on patient presentations commonly seen. This distillation does not exclude the use of less common codes when needed. The focus on more commonly seen presentations, associated codes and their operational definitions will assist in:

- the education of students in the use of ICD codes
- the transfer of cases between clinicians
Musculoskeletal Complaints

Communicating a patient’s specific diagnosis to an outside agency
Research efforts designed to follow the outcome of treatment on a specific group of patients must have a clear set of criteria to define a given diagnosis. Standardization of ICD usage is crucial to this endeavor.

Realizing that, to some degree, the designation of a given code or group of codes to a given patient presentation is somewhat arbitrary (due to lack of clear definition in the ICD classification), the agreement on the operational use of the code(s) will help present a consistent impression of a patient to clinicians, interns, instructors, and outside agencies.

Although open to some interpretation, the ICD codes do clearly set apart codes that are exclusively used for musculoskeletal and neurological conditions as broad categories. Not always clearly defined, but implied, is the use of some codes as adjunct codes requiring another code as a primary code. Also, not clearly defined, however, implied is the use of some codes only when radiographic or special imaging confirmation is available.

Below are some broad classification categories based on general codes and the patient presentation restriction for its use:

<table>
<thead>
<tr>
<th>ICD-9-CM Codes</th>
<th>Patient Presentation</th>
</tr>
</thead>
</table>
| 739—Nonallopathic lesion (requires 4th digit) (AKA subluxation, somatic dysfunction, segmental dysfunction) | - May be used as a primary code if patient is asymptomatic and indications of subluxation are determined from chiropractic evaluation.  
- Should be used in conjunction with a separate primary code when chiropractic findings indicate SMT for that area |
| 839—Other and ill-defined dislocations (requires 4th & 5th digits as well as specific segments) | - Used exclusively to designate subluxation in Medicare patients |
| 846 & 847—Sprains/Strains of SI Joint and other Unspecified Areas of Back | - Used when there is a history of trauma (which also includes an onset of movement induced back pain)  
- May be used in presentations that include trauma and patient has pain radiation into leg (referred) and no supportive neurological findings are present |
| 723 & 724—Other Disorders of Cervical Spine and Unspecified Disorders of the Back (requires 4th digit) | - May be used when no evidence of sprain/strain, nerve root or spinal cord involvement is present  
- Used when cause is primarily myofascial  
- Used when the above criteria are met and patient has referred pain into the leg (no hard neurologic evidence of nerve root involvement)  
- A subcode of 724.6 is used for instability or chronic sprain/strain of low back or SI  
- A subcode of 724.8 will be used for facet syndrome which requires injury and/or reproduction of back or back/leg symptoms with hyperextension |
| 905.7—Late Effect of Sprain/Strain without Mention of Tendon Injury | - Preferable code for patients with recurrent or chronic pain as a result of a clear injury such as motor vehicle, sport, or occupationally related  
- Must have associated code for original sp/st |
| 907.3—Late Effect of Nerve Root, Spinal Plexus, and Other Nerves of Trunk | - Used when persistent nerve damage is evident indicated by an affected DTR, myotome, or dermatome  
- Must have associated code for original nerve injury |
The following codes require radiographic or special study confirmation in addition to clinical information for their use.

- 720.0—ankylosing spondylitis
- 721—spondylitis (with and without myelopathy)
- 722—intervertebral disc disorders (includes disc compression of nerve roots or spinal cord and Schmorl's nodes)
- 723.0—spinal stenosis of cervical region
- 724.0—spinal stenosis of thoracic and lumbar regions
- 737.30—idiopathic scoliosis
- 738—degenerative spondylolisthesis
- 738.4—acquired spondylolisthesis
- 756.11—congenital spondylolisthesis
- 756.12—congenital spondylolisthesis
- 805—fracture of spine without spinal cord injury

**APPENDIX 1–3**

**References**


