PART

I

Musculoskeletal Complaints

CHAPTER

1

General Approach to Musculoskeletal Complaints

CONTEXT

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** **Joint-Specific Injury Mechanism**

Mechanism	Possible Structure(s) Damaged
Shoulder	
Fall on an outstretched arm (extended elbow)	Rotator cuff tear
	Glenoid labrum tear
	Posterior dislocation
	Clavicular fracture
Arm forced into abduction/external rotation	Anterior dislocation
	Anterior musculature strain
Blow to the shoulder area	Fracture
	Acromioclavicular separation
	Dislocation
Fall onto top of shoulder	Shoulder pointer
	Acromioclavicular separation
	Distal clavicular fracture
Traction injury to arm	Plexus injury
	Medical subluxation
Elbow	
Direct fall on tip of elbow or fall on hand with elbow flexed	Olecranon fracture
Fall on hand with extended elbow	Radial head fracture
Hyperextension injury to elbow	Elbow dislocation
	Supracondylar fracture in children
Severe valgus stress	Capitellum fracture
	Avulsion of medial epicondyle
	Medial collateral ligament sprain or rupture
Sudden traction of forearm	Radial head subluxation
Wrist/hand	
Fall on dorsiflexed hand	Navicular fracture
	Epiphyseal and torus fractures in children
	Carpal dislocation, or instability
Hyperextension or abduction of thumb	Gamekeeper's thumb (ulnar collateral ligament damage)
Axial compression of thumb	Bennett's fracture
	Dislocation
Hyperextension of finger	Volar plate injury
	Jersey finger (rupture of flexor digitorum profundus)
	Dislocation
Hyperflexion of finger	Avulsion of central slip
	Mallet finger (rupture of extensor tendon)
Valgus/varus stress injury to finger	Collateral ligament or volar plate injury
Axial compression	Capsular irritation
	Fracture
Hip	
Fall on hip	Fracture
	Synovitis

(continued)

TABLE 1–1 **Joint-Specific Injury Mechanism (continued)**

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

- 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; electrodiagnostic 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.

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.

TABLE 1–2 Synovial Fluid Examination

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

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 muscularactivity.

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.[1] 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 intratendinosis 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 monoarthopathy (i.e., single joint), oligoarthopathy (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.

When 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 illi
- 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 illi, as

TABLE 1–3 Selected Arthritic Disorders

Type	Features	Management Issues
Degenerative		
Primary Osteoarthritis	**Age of Onset**—Generally > 45 y/o **Gender Predominance**—Ratio of female to male = 10:1 **Common Joints Involved**—Hips, knees, SI joint, AC joint, first MCP, first MC trapezium, DIP joints of hands Often initially asymptomatic; gradual increase in joint stiffness and pain. Deformity may be apparent (e.g., Herberden's nodes in hands). May eventually lead to joint subluxation and instability. Radiolographically: The distribution is asymmetric, with non-uniform loss of joint space, osteophyte formation, subchondral sclerosis (eburnation), subchondral cysts.	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.
Secondary Osteoarthritis	**Age of Onset**— > 25 y/o **Gender Predominance**—Equal **Common Joints Involved**—GH, AC, SI, hip, elbow, knee, foot, hand Cause is secondary to other disorders or diseases/injuries such as trauma, septic or inflammatory arthritis, slipped epiphyses, dysplasias, fracture/dislocation, avascular necrosis, ochronosis, and acromegaly. Similar radiographic presentation.	Management in early and middle stages should include strengthening around involved joints. If weightbearing, 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.
Erosive Osteoarthritis	**Age of Onset**—40–50 y/o **Gender Predominance**—Female **Common Joints Involved**—Interphalangeal joints of hand 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 Herberden's nodes. Radiologically similar to OA with additional finding of central erosions.	Management in early and middle stages should include strengthening around involved joints. If weightbearing, begin with non-weightbearing 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: ■ 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 usually given together with methotrexate)
Degenerative Spine Disease	**Age of Onset**— > 30 y/o **Gender Predominance**—Equal **Common Joints Involved**—Specific spinal involvement at C5-C7, T2-T5, T10-T12, L4-S1 with additional involvement of uncovertebral, costovertebral, discovertebral, and apophyseal (facet) joint involvement Range from asymptomatic to severely symptomatic with pain and stiffness. Radiographic to clinical correlation is poor. May contribute to IVF narrowing and spinal stenosis. Radiographic findings include disc space narrowing, hypertrophy of smaller joints such as facets and costovertebral, synovial cysts, Schmorl's nodes, and intradiscal vacuum phenomena are common. In middle stages, joint and capsular laxity may lead to subluxation and listhesis.	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 joint and invertervebral disc joints 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.

TABLE 1–3 Selected Arthritic Disorders (*continued*)

Type	Features	Management Issues
Diffuse Idiopathic Skeletal Hyperostosis (DISH) (synonyms: ankylosing hyperostosis, Forestier's disease)	**Age of Onset**—50 y/o and older **Gender Predominance**—Male **Common Joints Involved**—Spine; predominantly T7-T11 (calcification of anterior longitudinal ligament) with 30% peripheral joint involvement Found in 25% of men and 15% of women > 50 y/o (common). May be asymptomatic; when symptomatic, similar complaints associated with DJD such as stiffness and pain; 20% of patients report dsyphagia; occasional complaints involving the Achilles tendon, extensor wad of wrist/forearm, plantar fascia, and quadriceps tendon (may find enthesmophytes at corresponding sites); about a quarter of patients have diabetes. Radiographically: Diffuse, thick, hyperostosis primarily along the anterolateral aspect of spine ("flowing wax" appearance); 50% of patients also have ossification of the PLL, especially in the cervical spine.	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.
Neuorpathic (Neurotrophic) Arthropathy	**Age of Onset**—Variable **Gender Predominance**—Variable **Common Joints Involved**—Knee, hip, ankle, spine, shoulder, elbow, wrist, foot 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, Charco-Marie-Tooth disease, prolonged used of intra-articular corticosteroids, pernicious anemia, and leprosy, among others. A somewhat separate but related cause is spinal cord damage resulting in paraplegia or quadriplegia which results in usually asymptomatic bony ankylosis. Radiographically neuropathic arthropathy is seen as joint collapse, pseudoarthrosis, fragmentation, and deformity.	Treatment is directed toward the primary disease. If in weightbearing joints, mechanical assistance is often required. In severe cases, amputation is necessary.
Synoviochondrometaplasia (idiopathic synovial osteochondromatosis)	**Age of Onset**—30–50 y/o **Gender Predominance**—Male to female ratio = 3:1 **Common Joints Involved**—Knee, hip, ankle, elbow, wrist Synoviochondrometaplasia, 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, crepitus, and locking due to the loose bodies. Radiographically the loose bodies can be seen if radiopaque. Sometimes erosion may occur as in the "apple-core" deformity of the hip.	Synovectomy for most patients. Joint replacement may be recommended for older patients.
Inflammatory Positive for Rheumatoid Factor (Seropositive)		
Rheumatoid Arthritis (RA)	**Age of Onset**—25–55 y/o **Gender Predominance**—Female to male ratio = 2/3:1 **Common Joints Involved**—Hand, foot, wrist, knee, elbow, GH joint, AC joint, and cervical spine (atlantoaxial)	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.

TABLE 1–3 Selected Arthritic Disorders (continued)

Type	Features	Management Issues
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 mutalins; morning stiffness that lasts longer than one hour, specific swelling of several joints (including the PIP joints, MCP joint, 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/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) ■ Corticosteroids ■ 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 usually given together with methotrexate). May be administered as infusion therapy.
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, heel, 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 of 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.
Negative for Rheumatoid Factor (Seronegative)		
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. Peripheral joint involvement occurs in approximately 50% as does radiating pain to the lower extremity. Areas of concern include iritis (20% of cases), aortic insufficiency, aneurysms, pulmonary fibrosis, pleuritis, IBD, and amyloidosis. Laboratory findings include an increased ESR during active phases, negative for RA and LE factors; HLA B-27, positive in 80% (positive in 6–8% of general population). Radiographically there are classic signs, including symmetrical involvement of the SI joints, ligament calcification, and marginal syndesmophytes, eventually leading to "trolley-track" sign, and bamboo spine.	Caution with rheumatoid conditions is unpredictable are flare-ups. Given that some of the therapies employed by chiropractic is 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

TABLE 1–3 Selected Arthritic Disorders (continued)

Type	Features	Management Issues
Reiter's Syndrome	**Age of Onset**—15–35 y/o **Gender Predominance**—Male to female ratio = 5:1 to 50:1 depending upon study **Common Joints Involved**—SI joint, foot, heel, ankle, knee, hip, spine; more rarely the upper extremity Urethritis and other eye complaints often following a STD or gastrointestinal infection. Kerratitis, kerratoderma, and keratosis of nails may be found. Systemic findings may include fever, weight loss, thrombophlebitis, or amyloidosis. Lab findings may include positive HLA-B27 (75%), leukocytosis, anemia, and elevated ESR. Radiographically SI joint is prominent, antlanto-axial instability, nonmarginal syndesmophytes. Similar to psoriatic arthritis, a single digit may be involved (sausage finger) and enthesopathies are common as in AS. Monitor for aortic regurgitation in chronic cases.	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 (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
Psoriatic	**Age of Onset**—20–50 y/o **Gender Predominance**—Generally equal **Common Joints Involved**—Hand, foot, SI joint, thoracolumbar spine, and cervical spine 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 arthritis mutilans. In addition to possibly having scaly patches of skin (psoriasis) on the extensor surfaces of the knees and elbows, patients may also have nail changes, including pitting, discoloration, and splintering. In some cases hyperostosis occurs at the SC joint. Other skin lesions may occur in the hands and feet. Lab includes HLA-B27 antigen (60% of cases), mild anemia, elevated ESR during active periods, occasionally elevated uric acid levels. Radiographically the involvement of the hands is similar to RA. In addition, one digit is often affected (sausage finger) and tuft resorption and proliferation (ivory phalanx) occur. In the spine, nonmarginal syndesmophytes may be seen.	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 elevated serum lipids, mucocutaneous toxicity, and teratogenicity. New drugs are being marketed that, although highly promising, are extremely expensive. These drugs are part of a new class of medications called immune modulators (also known as biological 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 drugs effect, which is fusion of a receptor to the Fc portion of human IgGI.)
Enteropathic (associated with inflammatory bowel disease [IBD])	**Age of Onset**—Variable **Gender Predominance**—Variable **Common Joints Involved**—SI joint and spine; occasionally peripheral joint involvement 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.	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. Medical management includes: ■ NSAIDs—COX-1 (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) ■ Corticosteroids

TABLE 1–3 Selected Arthritic Disorders (continued)

Type	Features	Management Issues
Systemic Lupus Erythematosis (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). Polyarthritis is common. Like many patients with autoimmune rheumatoid conditions, tendons are weakened and may rupture. Laboratory reveals anemia with leucopenia and plasma protein abnormalities (protein electrophoresis usually ordered due to globulin increase). Antinuclear antibody and LE cells present. A false-positive syphilis test may occur. Radiographically a symmetric, nonerosive yet deforming arthropathy is seen. Osteonecrosis may be seen due to the disease or due to treatment (corticosteroids).	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 predisone for joint pain, cutaneous lesions, and renal and CNS involvement. Other medical therapies include antimalarials (hydroxychloroquine) and NSAIDs. Infection is common due to immunosupression 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; vitiligo and telangiectasias; and thickening and tightening of the skin of the face, hands, and feet. Laboratory findings include an elevated ESR (60–70%), positive RF (20–40%), positive ANA (35–96%), 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: Raynaud's ■ Calcium channel blockers ■ Peripheral adrenergic blockers ■ Protective measures against cold, cessation of smoking, and decreased use of caffeine and other sympathomatics 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 sympathomametic substances and certain foods ■ H2 inhibitors and/or proton-pump inhibitors Arthralgias ■ NSAIDs 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.
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.

TABLE **1–3** **Selected Arthritic Disorders (continued)**

Type	Features	Management Issues
Dermatomyositis and Polymyositis (*continued*)	Dermatomyositis affects skin and muscle, whereas, polymyositis affects primarily muscle. The affect 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's phenomenon. Disability occurs due to progressive symmetric, proximal muscle weakness. Laboratory findings include CPK 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.	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.
Mixed Connective Tissue Disease	**Age of Onset**—20–50 y/o **Gender Predominance**—Female more than male **Common Joints Involved**—Hand, wrist, and foot This group of conditions is an overlap of several specific diseases such as RA, SLE, dermatomyositis, and scleroderma. Laboratory findings include specific findings for each disorder and presence of ribonuclease-sensitive extractable nuclear antigen. Radiographic findings are those for each disorder and include joint destruction with marginal erosions and soft tissue calcification.	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 (clubbing); periostitis in tibia, fibula, radius, and ulna There is a triad of peripheral arthritis with clubbing of the fingers and periostitis of the distal long bones. This process appears to be secondary to processes in thorax or abdomen, most commonly, bronchogenic carcinoma, and seems to be neurovascular due to vagus nerve dysfunction. Patients often have signs only of the underlying disorder. Radiographic findings include the digital clubbing and long bone symmetrical periostitis.	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 Condensins Illi	**Age of Onset**—20–40 y/o **Gender Predominance**—Female to male ratio = 9:1 **Common Joints Involved**—Sacroiliac This bilateral disorder affects females probably through a combination of ligament laxity (hormonally induced) and mechanical stresses that lead to sclerotic changes in the iliac subchondral bone. The process is often asymptomatic. When symptomatic, may present as low back pain with or without leg pain; however, caution must be taken when attempting to relate the radiographic changes to symptoms. The condition, if symptomatic, appears to be primarily self-limiting.	When symptomatic, management includes anti-inflammatory approaches. Generally, self-resolving.

TABLE **1–3** **Selected Arthritic Disorders (continued)**

Type	Features	Management Issues
Osteitis Pubis	**Age of Onset**—Varies, but with females during the reproductive years **Gender Predominance**—Female predominant **Common Joints Involved**—Symphysis pubes 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.	Initial management may be with NSAIDs and rest. More severe cases may require oral or locally injected corticosteroids. In rare cases, arthrodesis is necessary.
Metabolic Crystal Deposition		
Gout	**Age of Onset**—>30 y/o (in females, postmenopausal) **Gender Predominance**—Male **Common Joints Involved**—First MTP joint of foot, feet, ankle, and knees First attack is 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. Desquamation 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 bursae, 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 phosphoriboxyltransferase deficiency. Hyperuricemia is common, especially during acute attacks; joint aspiration reveals calcium urate crystals. Radiographically, joint destruction with soft-tissue swelling and radioluscent spots (urate crystals) are evident.	For those with single attack, lifestyle modification first. Diet—attention to hydration, low-purine diet (avoid meat, yeasts including beer/alcohol, beans, legumes), alcohol avoidance, lose weight. Medication avoidance—hyperuricemic meds such as thiazide and loop diuretics; also avoid low-dose aspirin and niacin. Medical approach—acute attacks; NSAIDs or corticosteroids, or colchicines (for inflammation); long-term approach incorporates drugs to reduce serum uric acid and decrease tophi deposits; primarily allopurinal (xanthine-oxidase inhibitor) and probenecid. Low-dose colchicines are acceptable as a prophylaxis for first 6 months.
Calcium Pyrophosphate Deposition Disease (CCPD)	**Age of Onset**—>50 y/o **Gender Predominance**—Generally equal dependent on cause **Common Joints Involved**—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 *chondromcalcinosis* 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, pseudorheumatoid arthritis, and pseudodegenerative joint disease. The most common, pseudo-gout, may appear 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. Calcification of intra and extra-articular structures with eventual articular destruction and fragmentation.	Management may include joint aspiration, short-term use of NSAIDs or colchicines, or corticosteroid injection during acute attacks. For recurrent attacks, low-dose colchicines have been used as has corticosteroids. Antimalarial medications have also been used in some cases. Radioactive synovectomy has also been performed on some patients.

TABLE **1–3** **Selected Arthritic Disorders (continued)**

Type	Features	Management Issues
Hydroxyapatite Deposition Disease	**Age of Onset**—40–70 y/o **Gender Predominance**—Equal **Common Joints Involved**—Shoulder, hip, cervical spine This idiopathic process results in calcium (hydroxiapatite) deposition in tendons, bursae, and other periarticular soft tissue. In the spine this may include nucleus pulposis calcification. Technically not an arthritis, pain is felt around joints. It is believed that symptoms develop as the process resolves (inflammation) rather than during the deposition process. Radiographically, soft tissue opacities are seen around the joint.	Management may include joint aspiration, short term use of NSAIDs or colchicines, or corticosteroid injection during acute attacks. For recurrent attacks, low-dose colchicines has been used as has corticosteroids. Pulsed ultrasound using iontophoresis may be of benefit.
Other		
Sarcoidosis	**Age of Onset**—20–40 y/o **Gender Predominance**—Equal **Common Joints Involved**—Hands, wrists, and feet This is systemic disease that produces noncaseating granulomas. It is more common in Scandanavian and Black populations. Generalized symptoms/signs predominate with low-grade fever, rash, lymphadenopathy, malaise, fatigue, arthralgias, and iritis. A sub-group of patients have Lofgren's syndrome. Laboratory findings include a reverse A/G ratio, elevated ESR, hypercalcemia, and a positive Kviem test. Skeletal involvement occurs in 15% of patients. Radiographically, granulomas are seen in the perihylar region of the lungs with infiltrates and fibrosis. In joints there may be circumscribed, lytic, intraosseous lesions.	Arthritis is managed similar to rheumatoid arthritis, incorporating corticosteroids in severe cases or colchicine as is used with gout. Unknown whether an anti-inflammatory diet is helpful.
Hemochromatosis	**Age of Onset**—40–60 y/o **Gender Predominance** —Male to female ratio = 10:1 to 20:1 **Common Joints Involved** —Hip, knee, shoulder, wrist, hand Rare disorder involving deposition of iron into various tissues. Triad includes bronze skin, liver cirrhosis, and diabetes mellitus. When joints are affected there may be pain, stiffness, and swelling; usually bilaterally; however, may begin in a single joint. Laboratory includes elevated ESR and serum iron, increased saturation of plasma iron binding protein transferring, and liver biopsy findings. Radiographically, usually bilateral involvement with osteoporosis, CPPD crystal deposition (50% of patients), and involvement of MCP joints.	Weekly phlebotomies is the treatment approach to prolong lives.
Alkaptonuria (Ochronosis)	**Age of Onse**—30–40 y/o (present at birth though) **Gender Predominance**—Equal **Common Joints Involved**—Spine, hip, and knee An hereditary disorder of tyrosine characterized by absence of homogentisic acid oxidase leading to deposition in tissues throughout the body. The accumulation of homogentisic acid oxidizes to form a black pigment. Ochronosis (brown-black pigmentation in connective tissue	Homogentisic acid accumulation could theoretically be controlled through dietary restrictions of phenylalanine and tyrosine, yet the long-term results have not proven this to be an effective treatment. Similarly, theoretically ascorbic acid supplementation could could block oxidation of homogentisic acid, but the effectiveness of this approach has not been confirmed.

TABLE 1–3 **Selected Arthritic Disorders (continued)**

Type	Features	Management Issues
Alkaptonuria (Ochronosis) (*continued*)	not usually seen until age 20), discoloration of urine and ochronotic arthropathy that includes acute exacerbations of arthritic pain in the spine. Cartilage of nose and ears may appear brown but blue on transillumination. Renal and prostate stones are common. Laboratory findings include urine that turns black on standing, homogentisic acid in urine. Radiographically, there is accelerated DJD of the spine with eventual bamboo spine, often beginning with calcification of the interspinous ligament.	
Pigmented Villonodular Synovitis (PVS)	**Age of Onset**—20–40 y/o **Gender Predominance**—Male (slight) **Common Joints Involved**—Knee, hip, elbow, ankle 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 initial preservation of joint space. Cystic erosions with hemorrhagic joint effusion can be seen. MRI is diagnostic.	Treatment is complete synovectomy. Irradiation has also been used in some patients.
Hemophillic 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 Hemophilia is a group of disorders that share a problem with clotting factors and result in dysfunctional blood coagulation. The result is bleeding throughout the body, manifested externally as bruising, and prolonged bleeding, such as nose bleeds. Within joints, bleeding occurs and gradually causes changes that include swelling, contractures, fibrosis, and joint destruction. Due to the age of onset, radiographic findings include epiphyseal overgrowth, accelerated skeletal maturation, and radioluscent joint effusions. At some point the joint has a similar appearance radiographically to juvenile rheumatoid arthritis.	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.**	
	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 in adults is Neisseria gonorrheae. Others include Staphylococcus aureua, streptococci, and some gram-negative such as Enterobacter, Psuedomonas auroginosa, and Serratia marcescens. For chronic arthritis, primary causes include mycobacterium and fungi.	Antibiotic therapy is often given parenterally, and joint aspiration is performed as well as lavage and debridement dependent on degree of involvement. For fungal infections, amphotericin B and similar medications are needed.

TABLE **1–3** **Selected Arthritic Disorders (continued)**

Type	Features	Management Issues
Infectious (*continued*)	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. With chronic causes, joint space is preserved longer. Synovial fluid analysis will reveal a high WBC count with a decrease in viscosity and glucose. Gram staining and culture will usually reveal the causative organism.	
Hyperlipidemia	Recurrent, migratory join pain involving the knee and other large joints is seen in patients with familial hypercholesterolemia. The appearance is that of inflammatory process with fever, swelling, tenderness, and warmth. The onset is generally acute. The joint pain may not be in the joint itself but represent periarthritis or peritendinitis given that there is usually no joint damage. Xanthomas have been reported in the Achilles tendon, patellar tendon, and extensor tendons of the hands and feet. Diagnostic suspicion is high with a finding of familial hypercholesterolemia on laboratory evaluation coupled with the onset of recurrent migratory arthritis. Radiographs may show osteopenia and bone cysts.	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 polymysitis.

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[2] 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). Generally, orthopaedic 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 **1–4** **Selective Tension Approach**

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

Key: ROM: range of motion

pain may be due to many factors and is therefore nonspecific (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 1–5 General Approach Based on Structure

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

Key: MRI, magnetic resonance imaging; CT, computed tomography; LDH, lactate dehydrogenase; HLA, human leukocyte antigen; ANA, antinuclear antibodies; ROM, range of motion; SPECT, single photon emission computed tomography.

- Ligaments/capsules—Use direct palpation (if possible) and perform a stress test that usually involves stabilizing one bone while moving the neighboring bone on it (for example, drawer testing of the shoulder, knee, and ankle). In essence, motion palpation of a joint is the same as many ligament stability tests, yet the intent is different; locate restrictions, not instability.
- Tendons—Use direct palpation and stretch into end-range (contraction at end-range stretch may also be used).
- Muscles—Use direct palpation and contraction (although traditionally used to detect weakness, the main focus is to determine reproduction of a patient's complaint).
- Nerves—Tapping (i.e., Tinel's) and compression are direct tests for superficial nerves; indirect tests include motor and sensory evaluation of specific peripheral nerves, nerve plexus, nerve root, or central nervous system (CNS) involvement including muscle tests, deep tendon reflex testing, and sensory testing with a pin/brush or pinwheel.

Palpation is a valuable tool when accessing superficial tissues. Accessibility is limited, based on the joint and its location. The fingers and toes are thin accessible structures, whereas the hip and shoulder are not. Direct palpation of ligaments and tendons may reveal tenderness. Muscles may also be palpated for tenderness and possible associated referred patterns of pain. These trigger points have been mapped by Travell and Simons.[3] 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 exposed 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 Janda[9] and Lewit[10] 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

Cyriax[8] 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.

- 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.

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:

- 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. Janda[9] and Lewit[10] and others[12] 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 Janda[9] and Lewit[10] 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 toward 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

Figure 1–1 Assessment of Musculoligamentous Injury—Algorithm

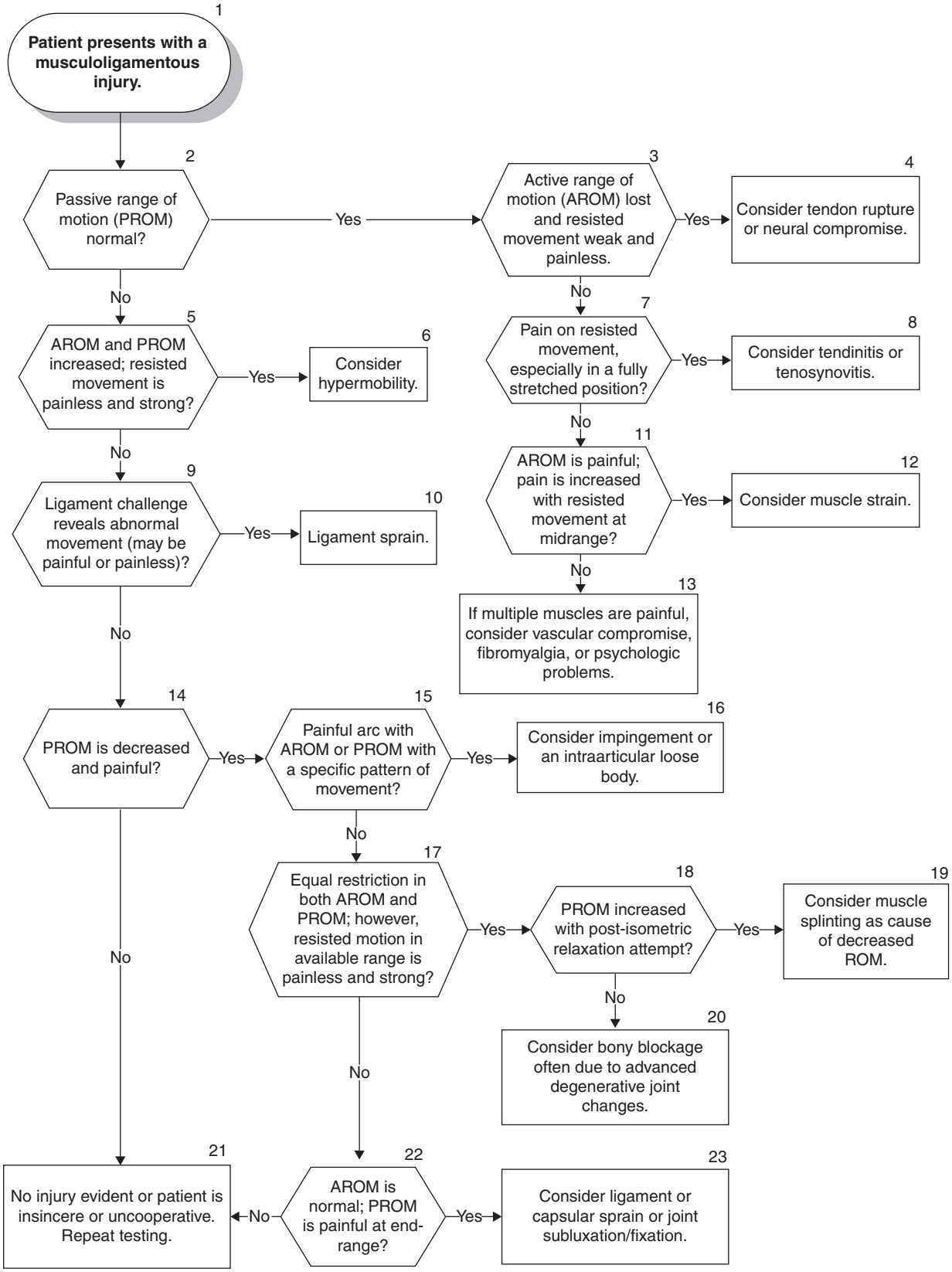

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EXHIBIT 1–1

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 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).

hamstrings. This is followed by gluteal contraction, then erector spinae contraction. If the hamstrings or gluteals do not participate, the erector spinae contract, causing a weak contraction and a lordotic/compressive load to the low back. In the neck, flexion may reveal an imbalance in movement. If the patient's jaw juts forward at the beginning of the pattern, weak neck flexors with associated "strong" sternocleidomastoids are indicated.

Accessory Motion

One of the indicators for manipulation or adjusting is blockage of accessory motion.[13] 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[8] 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.

Specific patterns of extremity and spinal movement are coupled with specific accessory motion so that restrictions in active movement may be indirectly an indicator of dysfunction of the accompanying accessory motion.[14]

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?

- What are the legal ramifications if the study or studies are or are not performed?

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.[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.[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).[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.

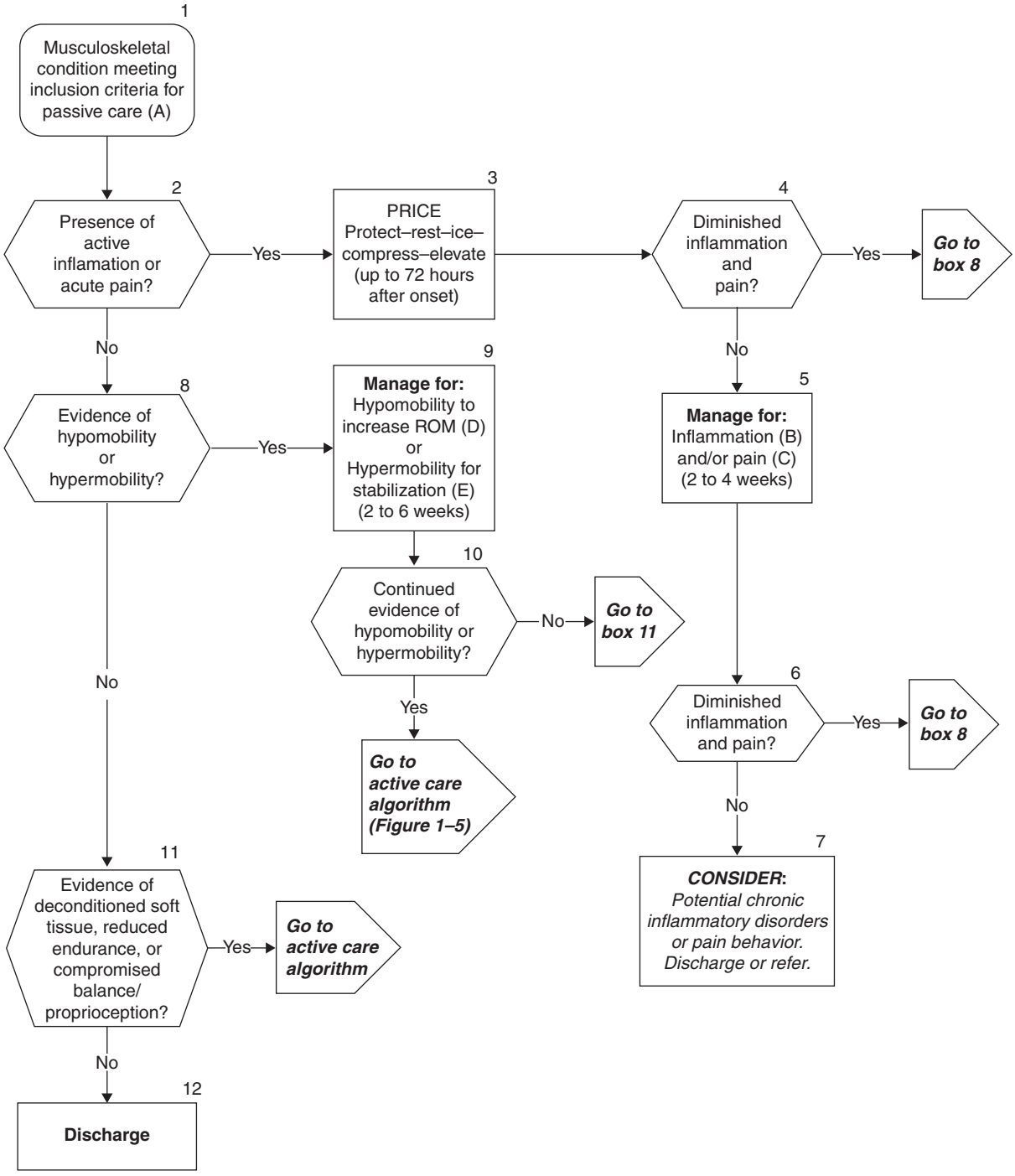
Although traditionally it was the doctor's role to be active and the patient's to be passive with treatment, it is becoming clear that there is a point at which role switching is necessary. When a patient has acute pain, the goal is to reduce the pain and assist healing. Many of the treatment methods 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[27] has outlined some criteria for passive care (Figure 1–2). These

TABLE **1–6** **Magnetic Resonance Imaging for the Chiropractor**

MRI Better Than CT	MRI Equal to CT	CT Better Than MRI
MRI of the Head		
Severe headaches	Hydrocephalus	Fracture of the calvaria
Visual disturbance	Brain atrophy	Fracture of the skull base
Sensory-neural hearing loss		Cholesteatoma of inner ear
Primary brain tumor		Intracranial hemorrhage 1–3 days old
Metastatic brain tumor		Cerebral infarction 1–3 days old
Intracranial infection		Intracranial calcifications
Age-related CNS disease		
Multiple sclerosis		
Dementia		
Chronic subdural hematoma		
Posttraumatic evaluation of the brain		
Intracranial hemorrhage older than 3 days		
Cerebral infarction older than 3 days		
MRI of the Cervical and Thoracic Spines		
Tumors or masses at the level of the foramen magnum	Spinal stenosis	Occult fracture of a vertebra
Chiari I malformation		Complex fracture of a vertebra
Cervical or thoracic herniated disc		Bony foraminal encroachment
Posttraumatic syrinx		
Core or conus tumor		
Acquired immunodeficiency syndrome-related myelopathy		
Multiple sclerosis of the spinal cord		
Posttraumatic epidural hematoma		
Epidural metastatic disease		
Epidural abscess		
MRI of the Lumbar Spine		
Small lumbar herniation	Large lumbar herniation	Occult fracture
Foraminal herniation	Spinal stenosis	Hypertrophic bony overgrowth or spurring
Interruption of the posterior longitudinal ligament		Bony foraminal encroachment
Root sleeve compression		Spondylolysis
Postoperative scar versus recurrent lumbar herniation (with gadolinium)		Evaluation of posterior element fusion
MRI of the Shoulder		
Posttraumatic bone bruise	Rotator cuff tear	Subtle glenoid labrum tear
Avascular necrosis of humeral head		Evaluation of the glenohumeral ligaments
Impingement syndrome		
Lipoma (or soft tissue mass)		
Tumor		
Brachial plexus tumor		
MRI of the Knee		
Posttraumatic bone bruise	Meniscal tear	Evaluation of the meniscus following previous meniscectomy
Osteochondritis dissecans		Evaluation of the articular cartilage
Anterior cruciate ligament tear		
Posterior cruciate ligament tear		
Collateral ligament tear		
Patellar tendon abnormalities		
Infection		
Tumor		

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

Figure 1–2 Passive Care Management—Algorithm



Annotations

(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

Source: Reprinted from D. L. Nelson, *Topics in Clinical Chiropractic,* Vol. 1, No. 4, p. 75, © 1994, Aspen Publishers, Inc.

include a history of recent trauma, acute condition or flare up, inflammation, or dependency behavior. There are generally four types of care that may overlap, as follows:

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, postisometric relaxation, proprioceptive neuromuscular 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** **Physiotherapy Approaches for Musculoskeletal Complaints**

Various Transcutaneous Electrical Nerve Stimulation (TENS) Approaches

Type	Pulse Width (pps)	Pulse Rate	Amplitude	Treatment Time
High Frequency	High:75–100	<200	Increase to discomfort	20 min.–24 hrs
Low Frequency	Low:Below 10	200–300	Increase to elicit strong rhythmic contractions	30–60 min.
Brief and Intense	TENS–150	150	Increase to titanic contraction	15 min.
	Galvanic:1–5			
Burst mode	50–100	75–100	Increase to strong but comfortable contraction	20–60 min.

Pulsed High-Volt Galvanic

Treatment Effect	Mode	Frequency (pps)	Type of Stimulation	Time (min)
Reduce edema	Continuous	High:80–100	Sensory	20
Reduce muscle spasm	Continuous	High:80–120	Sensory/motor	20–30
Autonomic response	Uninterrupted/Pulsed	High:80–100	Sensory	20–30
Neuro/hormonal	Uninterrupted/Pulsed	Low:2–5	Motor (twitch)	30–60
Muscle pumping	Surged 1:3	Low:30–50	Motor	20–30

EXHIBIT 1–2

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).

EXHIBIT 1–3

Cross-Friction Massage and Spray and Stretch

Cross-Friction Massage

Cross-friction massage is a technique popularized by Cyriax.[8] 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,[3] 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).

EXHIBIT 1–4

Myofascial Release Techniques (MRTs)

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.[28] 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.

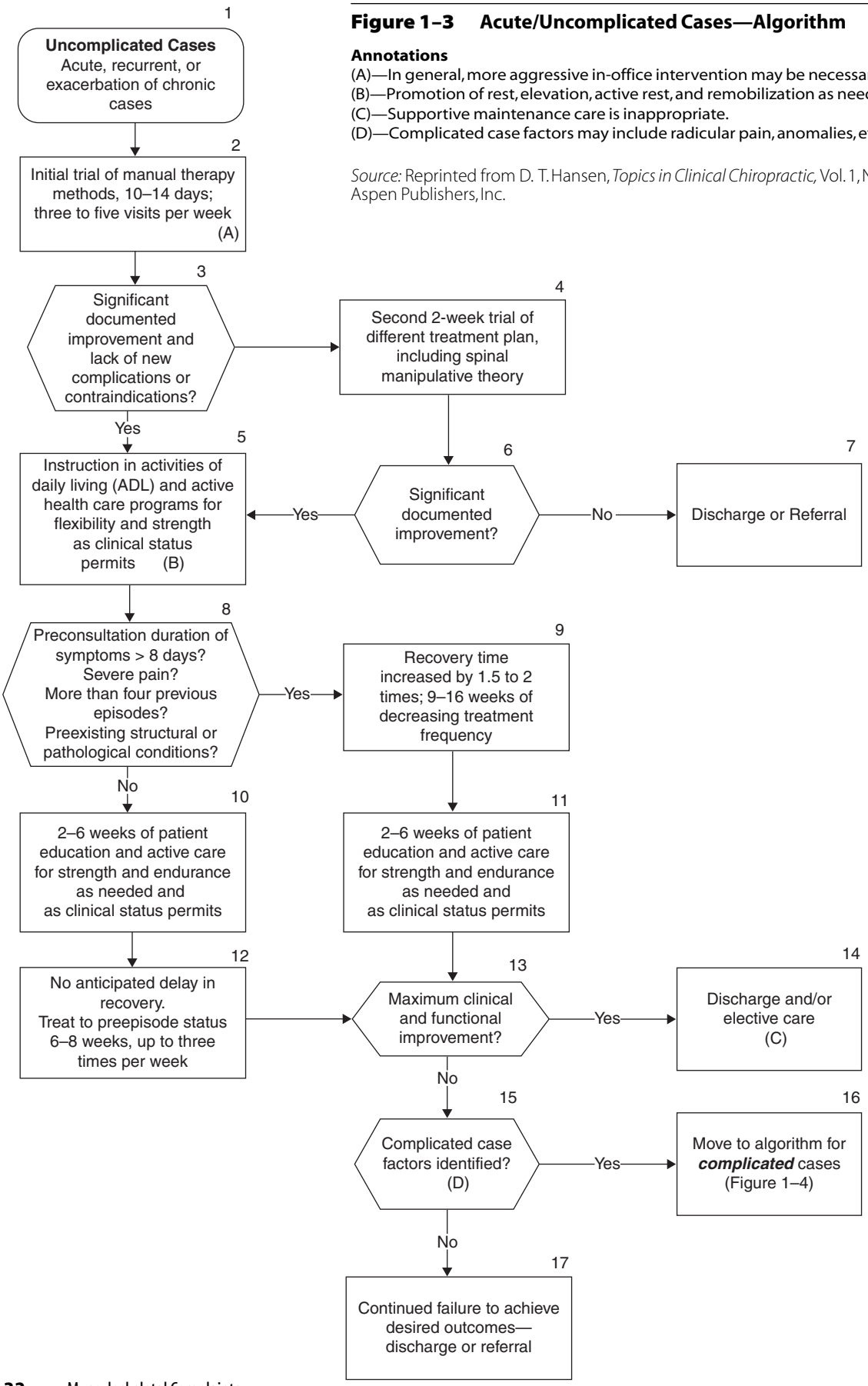


Figure 1–3 Acute/Uncomplicated Cases—Algorithm

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.

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Figure 1–4 Subacute/Chronic Complicated Cases—Algorithm

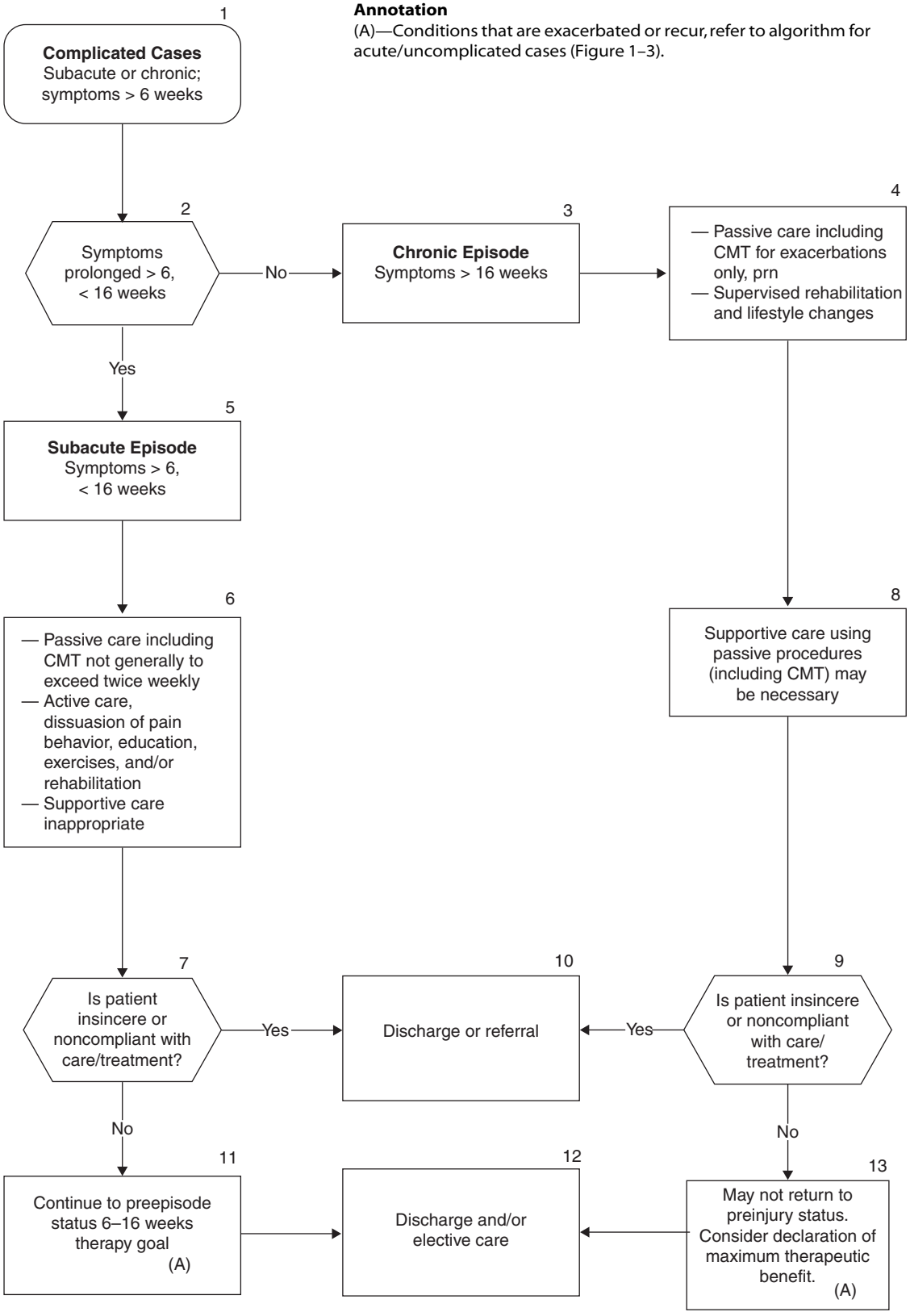

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Figure 1–5 Active Care Management—Algorithm

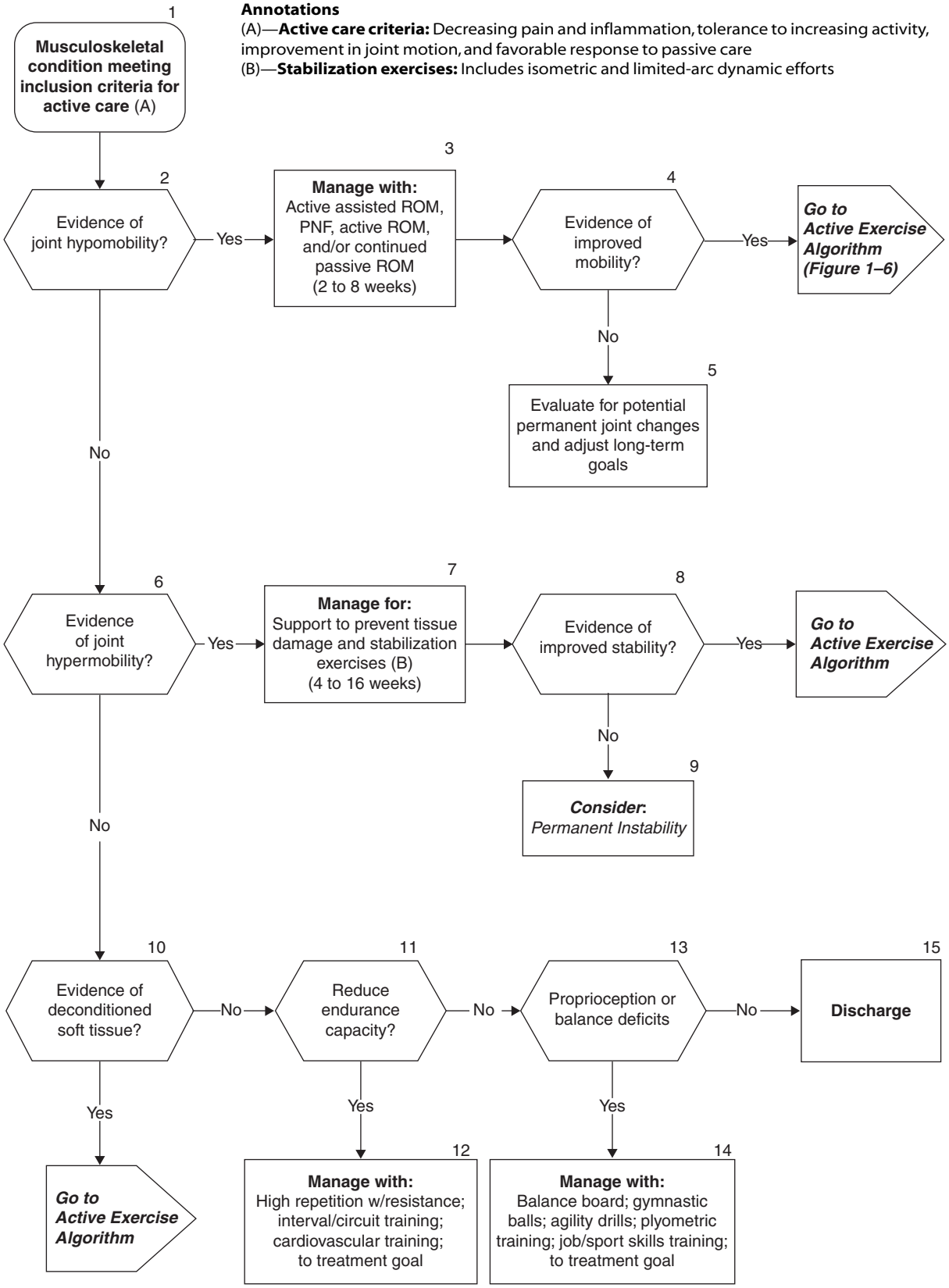

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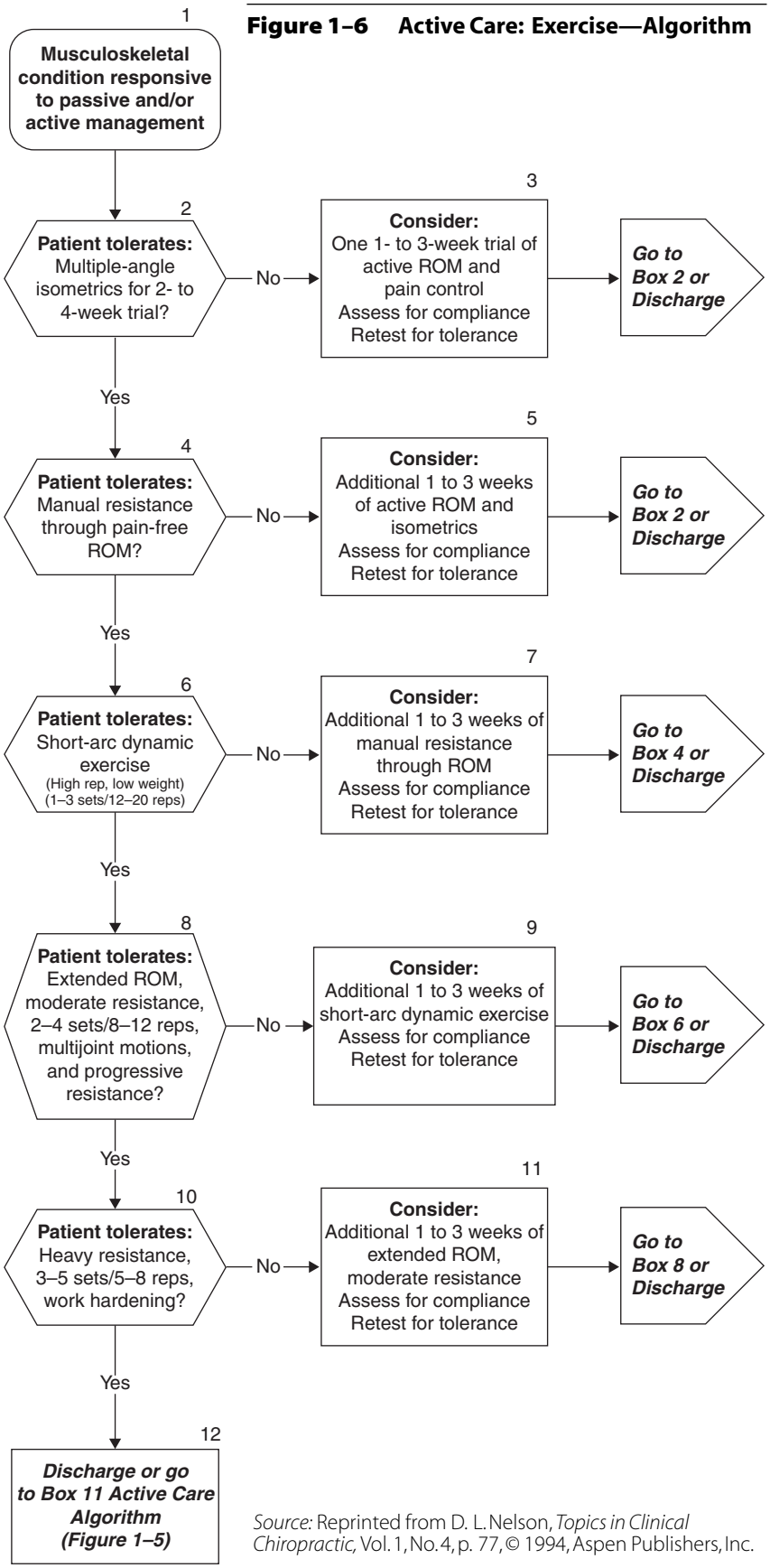
Figure 1–6 Active Care: Exercise—Algorithm



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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) approach[31] (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

TABLE **1–8** **The Daily Adjustable Progressive Resistance Exercise (DAPRE) Approach**

Set	Weight	Repetitions
1	1/2 working weight	10
2	3/4 working weight	6
3	Full working weight	Maximum
4	Adjusted working weight (based on 3rd set)	Maximum

- 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

No. of Repetitions	For 4th Set	Next Session Full Working Weight
0–2	Decrease 5–10 lb	Decrease 5–10 lb
3–4	Decrease 0–5 lb	The same
5–6	The same	Increase 5–10 lb
7–10	Increase 5–10 lb	Increase 5–15 lb
11 or more	Increase 10–15 lb	Increase 10–20 lb

EXHIBIT 1–5

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[30] Eccentric Protocol for Tendinitis/Tendinosis

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.

2400µ 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.[33] 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

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 eicosa-pentacnoic 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.[34] 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 lection (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 billoba—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-inflammatories (NSAIDs), and cyclooxygenase inhibitors (COX) block the cyclooxygenase enzyme that converts arachodonic acid to prostaglandin E-2, decreasing or blocking inflammation. Also, corticosteroids inhibit phospholipase A2, which inhibits arachidonic acid release from phosopholipids 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 NFκ-B, which reduces inflammation (see Exhibit 1–8). Conversely, NFκ-B 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 NFκ-B. It is in the cytoplasm and is bound to its inhibitor. Free radicals release NFκ-B 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 NFκ-B. It is possible that caretonoids 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 **1–9** **Nutritional Support for Osteoarthritis**

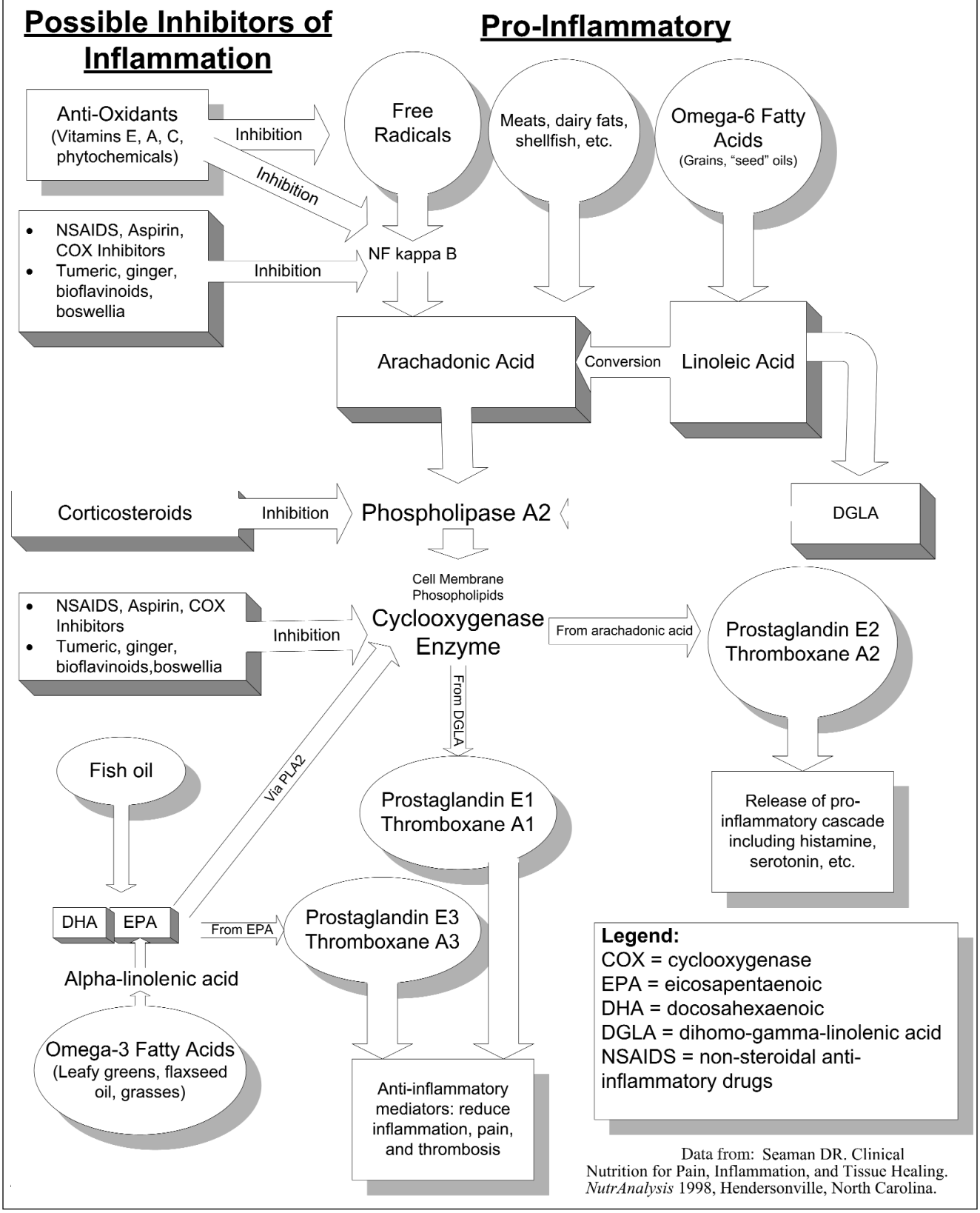
Substance	How Might It Work?	Dosage	Special Instructions	Contraindications and Possible Side Effects
Glucosamine sulfate	Stimulates the rebuilding of damaged cartilage	500 mg three times per day	Take with meals Take 6–8 weeks to determine effect	No contraindications May cause some gastrointestinal upset Does not interfere with other anti-inflammatory drugs Some products processed with sodium chloride; use caution with patients who are hypertensive
Boswellia	Decreases inflammation	150 mg three times per day (for example, if extract contains 37.5% boswellic acids, need 400 mg of extract taken three times/day)	Take for 8 to 12 weeks	None at recommended dosage
Horsetail	Decreases inflammation	Taken as a tea at 1–4 g/day; tincture would be taken as 2–6 ml three times per day	None	Avoid *Equisetum palustre,* another species of horsetail that contains toxic alkaloids
SAM (S-adensyl-methionine)	Possibly raises levels of dopamine	1600 mg/day	None	Occasional gastrointestinal upset Some caution about patients with manic-depression switching from depression to a manic episode Apparently safe in pregnancy
Vitamin E	Antioxidant	100–300 IU/day	None	None as recommended
Niacinamide	Form of vitamin B_3; may relieve symptoms and increase mobility	250 mg of niacinamide or nicotinamide 4–16 times/day	Improvement may take 3–4 months of supplementation	None at recommended dosage Rare liver problems at several thousand milligrams per day
Vitamin C, iron (glycinate), and alpha-ketoglutaris acid	Required for hydroxylation of L-proline to L-hydroxyproline needed for quality collagen production	Vitamin C—3000–6000 mg/day in divided dosages Iron (glycinate)—8–12 mg/day in divided dosages Alpha-ketoglutaric acid—15 mg/day in divided dosages	None	Use caution with high dosages of vitamin C; it may lead to diarrhea or urinary tract irritation in some people

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



Data from: Seaman DR. Clinical Nutrition for Pain, Inflammation, and Tissue Healing. *NutrAnalysis* 1998, Hendersonville, North Carolina.

TABLE 1–10 **General Nutritional Approach to Tendinitis/Tendinosis and Other Soft Tissue Injury**

Substance(s)	Recommended Amount
Proteolytic enzymes (trypsin, chymotrypsin, bromelain)	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.
Bioflavinoids (quercetin, hesperidine, rutin, etc.)	600–1800 mg/day. Often taken at 200 mg every 2 waking hours. Taken before peak of inflammatory phase.
Herbs—Boswellia, ginger, tumeric, cayenne	Boswellia—400 mg, ginger—300 mg, tumeric—200 mg, cayenne—50 mg taken every 2 waking hours during inflammatory phase.

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

- 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:

ICD-9-CM Codes	Patient Presentation
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 sydrome 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—spondylosis (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—aquired spondylolysis
- 756.11—congenital spondylolysis
- 756.12—congenital spondylolisthesis
- 805—fracture of spine without spinal cord injury

APPENDIX 1–3

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