MEDICAL SCHOOL

for Claims Examiners & Attorneys

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TABLE OF CONTENTS

MUSCULOSKELETAL DISORDERS

INTRODUCTION

Musculoskeletal pain syndromes and the accompanying physical and emotional sequelae account for one of six patient visits to physicians. Numerous others self-medicate or seek alternative health professionals for treatment and often subject themselves to expensive medical evaluations and ineffective treatments. Thus, these problems are not minor in nature and consume considerable numbers of health care dollars and associated resources along with missed time and lost productivity from the workplace. When severe, these disorders may result in loss of overall function in daily life activities, may be accompanied by mental health problems and may result in a chronic pain syndrome.

TERMINOLOGY

The musculoskeletal system can be divided into bones, joints, and supporting soft tissue structures (muscles, ligaments, tendons and bursa). Soft tissue complaints are very common disorders and dwarf those of bone and joints in terms of frequency. Although the terms commonly used for damage or irritation of muscles (fibrositis), ligaments (enthesitis), tendons (tendinitis, tendinosis or tendinopathy), and bursa (bursitis) suggests inflammation, in fact these conditions are usually non-inflammatory. Current terminology also describes soft tissue complaints involving the muscles and contiguous bony prominences that are either localized (myofascial pain syndrome or MPS) or widespread throughout the body (fibromyalgia syndrome or FMS).

SPASM, STRAIN & SPRAIN

Muscles contract and relax when instructed by nerves to make various body parts move. A MUSCLE SPASM occurs when a muscle is continually contracting due to tension or injury. When a muscle is in constant spasm, there is not enough oxygen and blood moving through it to remove waste products such as lactic acid, which produces pain as it accumulates, causing burning and cramping.

A STRAIN is an abnormal stretch of muscle usually due to fatigue or over-exertion, leading to injury and is less severe than a SPRAIN, which is abnormal stretching (Grade 1), partial tearing (Grade 2) or complete tearing (Grade 3) of ligaments (the tough fibers that hold the bones together at the joints) or tendons. Normally, sprains and strains go away spontaneously in a few days or weeks with proper rest, exercise, medication, and therapy. Unfortunately, with continued pain and spasm, there is not time for healing. The muscle only becomes weaker and shortened, more easily injured and less able to function normally.

MYOFASCIAL PAIN SYNDROME & FIBROMYLAGIA

MYOFASCIAL PAIN SYNDROME (MPS) & FIBROMYALGIA (FM)

The terms MYOFASCIAL PAIN and FIBROMYALGIA refer to syndromes of musculoskeletal discomfort.

Myofascial Pain Syndrome (myo = muscle and fascial = the covering of the muscle) or MPS refers to soft tissue tenderness and pain with a specific zone of discomfort or pain ("trigger point") within the muscle that triggers pain that refers away from the trigger point to contiguous areas.

The examiner can roll the muscle between the thumb and index finger to feel this taut band or trigger point, which then causes pain to be felt in the referral zone. This palpation is typically accompanied by a characteristic localized twitch response of the muscle. The patient's behavioral reaction to firm palpation of a trigger point is termed a positive jump sign; the patient withdraws ("jumps") or gives a verbal response such as "that's it."

Trigger points may be characterized as either active or latent. Active trigger points cause spontaneous pain at rest or with motion that stretches or overloads the muscle. There is some restricted motion of the associated muscle. Latent trigger points are quiescent and result primarily in muscle tightness and dysfunction with associated pain upon palpatory pressure.

In the experience of examiners, the described taut bands and visible muscle "twitching" are not consistently seen, but trigger points are accepted as manifestations of myofascial pain.

Common complaints with MPS include muscle spasm, tenderness, stiffness, limitation of motion, and weakness. Myofascial pain occurs equally in both sexes. X-rays and laboratory test abnormalities are not part of this condition and are either coincidental or reflect another condition.

Conversely, myofascial pain occurs frequently in inactive individuals indicating that trauma, either acute or cumulative, is not necessary for causation. Perhaps deconditioning itself can cause this condition. Still, in the presence of appropriate acute or cumulative trauma, industrial causation is reasonably medically probable as it would be for a muscle strain.

Causes of myofascial pain syndromes can therefore include acute and cumulative trauma, the physical condition and emotional state of the individual and also familial factors. In patients with a remote history of trauma and latent trigger points, MPS activation may come about with intense heat or cold, weather changes or emotional stimuli.

Myofascial pain or muscle strain defines primarily a muscle process. Referred pain from deeper structures, including bone, ligament, bursa, tendon, disc, joint and even viscera, must be excluded, especially when the pain is severe. Myofascial pain should not produce severe symptoms, so that an underlying cause should be sought in such situations. Severe pain, weakness or loss of motion should suggest another source, either organic or non-organic.

The characteristics of myofascial pain may long outlast the precipitating event, which sets up a self-generating pain cycle that is perpetuated through lack of proper treatment, sustained muscle tension,

distorted posture, pain reinforcing behavior, and failure to reduce other contributing factors such as sleep disturbance.

Fibromyalgia (FM) is a chronic pain disorder characterized by diffuse muscle soreness and stiffness associated with specific, reproducible tender points. Most individuals suffer from sleep disturbance and many from emotional dysfunction. Many patients with fibromyalgia have multiple complaints suggesting a profound constitutional disorder. They commonly have subjective complaints of joint pain, stiffness, swelling, numbness or coldness of the extremities, headaches, bowel pain and irregularity, non-refreshing sleep and fatigue. Physical examination characteristically is normal, except for the described tender points. X-rays and laboratory tests are normal.

FM is usually idiopathic (without a specific etiology). There is some thought that fibromyalgia may be triggered by physical or psychological trauma. Fibromyalgia appears to be quite common, possibly affecting 10 million Americans and an estimated 3–6% of the world population. FM prevalence varies amongst ethnic and racial groups. While it is most prevalent in women - 80-90 percent of the people who have FM are women (most commonly between the ages of 20-55) - it also occurs in men and children of all ethnic groups.

The American College of Rheumatology notes that doctors diagnose fibromyalgia based on all the patient's relevant symptoms and normal and no longer just on the number of tender places during an examination. There is no test to detect fibromyalgia. Though there is no cure, medications can reduce symptoms in some patients. Patients also may feel better with proper self-care, such as exercise and getting enough sleep.

Fibromyalgia can coexist with other musculoskeletal disorders. The presence of other conditions does not exclude the diagnosis of fibromyalgia nor should the other condition be assumed the cause. On rare occasion hypothyroidism can cause similar symptoms that will respond to thyroid replacement. Treatment of a coexisting musculoskeletal condition, however, does not usually effectively treat fibromyalgia, confirming that fibromyalgia can coexist with and is usually not caused by other musculoskeletal disorders.

Many medical disorders are associated with FM, including hepatitis C infection, HIV infection, systemic lupus erythematosus and rheumatoid arthritis, to name a few. The evaluation of FM must consider these possibilities.

Most individuals with FMS have other concurrent complaints that roughly parallel the severity of the disorder and prognosis. These can include headaches, non-anatomic paresthesias, atypical (non-cardiac) chest pain, irritable bowel syndrome (recurrent abdominal pain and constipation and/or diarrhea), difficulty concentrating and thinking, non-refreshing sleep, fatigue, temporomandibular disorder, and other less common complaints. These are not part of the FM criteria but are common complaints.

DIIFERENTIAL DIAGNOSIS

The differential diagnosis includes inflammatory and degenerative joint diseases, other connective tissue diseases, malignancy, visceral dysfunction, endocrine disorders (particularly thyroid), neurological diseases and psychological disorders. Chronic Fatigue Syndrome (CFS) and Multiple Chemical Sensitivity (MCS) patients have similar complaints to individuals with fibromyalgia, although with CFS, fatigue and cognitive problems seem to predominate, and with MCS, environmental sensitivities. With pain down a limb, a radiculopathy secondary to a herniated disk is a consideration, but similar symptoms

can be found in patients with a myofascial pain syndrome.

Once other causes have been ruled out, attention should be focused on deciding if the diagnosis of MPS or FM is reasonable. It is important to remember that people and their illnesses present as frequently complex with many factors contributing to their complaints.

Myofascial pain tends to be more acute in onset, regional in presentation, more amenable to treatment and characterized by trigger points. FM tends to be more insidious in onset, more global in presentation, more guarded in prognosis, and characterized by discrete reproducible tender points.

TREATMENT

Treatment should be circumspect, well directed and realistic. Expectations and treatment must be tempered by the knowledge that outcome is frequently frustrated by continued symptoms.

Treatment for myofascial pain and fibromyalgia is directed first towards diagnosing and treating factors such as structural abnormalities, medical conditions or emotional dysfunction. Trigger or tender points are then identified and treated directly with a number of different techniques including spray (with an icy cold vapo-coolant such as fluormethane) and stretch, massage and acupressure. Acupuncture may also be beneficial. Other techniques include needling (trigger point injections) of the affected areas either with or without a local anesthetic. Steroid injections are not recommended.

A careful and graduated physical rehabilitation program of stretching, strengthening and conditioning coincides with these treatments. A TENS (transcutaneous electrical nerve stimulation) unit may be helpful. Other treatments include gentle aerobic conditioning, psychological approaches (biofeedback, psychotherapy, cognitive-behavioral therapy) and non-opioid medications (antidepressants, anti-neuropathic agents, muscle relaxants and nonsteroidal anti-inflammatory agents).

Treatment for fibromyalgia has proved less effective than for myofascial pain in terms of long-term results. The prognosis for these patients often includes a waxing and waning course without a true cure of the condition. One author reported no more than a 20% remission rate. Treatment should be limited, goal oriented and directed toward safely alleviating symptoms while assisting the patient in coping with a chronic problem.

SUMMARY

Myofascial Pain Syndrome and Fibromyalgia continue to be regarded by some as specific and separate diseases while others consider them a continuum of the same problem. Other physicians consider either MPS or FM wastebasket terms for soft-tissue complaints while others consider them simply nonexistent. Part of this confusion stems from the lack of attention to appropriate diagnostic criteria and the absence of correlating diagnostic testing abnormalities in patients who appear to demonstrate many psychological and behavioral symptoms.

There is significant clinical information though to support that both MPS and FM are true medical entities and deserve appropriate attention and recognition. Evaluation and treatment of myofascial pain and fibromyalgia should include consistency, conviction and compassion.

Consistency in diagnosis and level of disability should be fair and based on the injured worker's true

functional capacity for work activities; conviction that these disorders do exist and are common while realizing that they should not be over treated nor should the patient's perception of disability be encouraged; and compassion that these disorders defy analysis by more sophisticated techniques but are valid sources of physical discomfort and disability.

OSTEOARTHRITIS (DJD)

INTRODUCTION

Osteoarthritis (degenerative joint disease) is the most common type of arthritis, dwarfing all other types combined. By 40 years of age, 90 percent of the population has radiologic changes of osteoarthritis, but many have no symptoms. Osteoarthritis causes symptoms in 25 percent of the population, usually becoming symptomatic after the age of 50. Osteoarthritis before the age of 40 is rare and suggests secondary causes such as genetic predisposition or prior trauma.

TYPES OF OSTEOARTHRITIS

Primary osteoarthritis is of unknown cause and represents the vast majority of cases. Secondary osteoarthritis is caused by a known event such as trauma to the joint and underlying bone (subchondral bone), developmental deformity of the normal congruity of the joint or misalignment from ligamentous instability leading to imperfect movement of one joint surface on the other or the result of past disease of the joint, such as infection or rheumatoid arthritis. Progression in secondary osteoarthritis can occur prematurely and progress more rapidly.

PATHOGENESIS

The normal joint consists of bone capped with a unique type of cartilage (hyaline cartilage) that is stronger than any synthesized substitute. Underneath the cartilage cap is subchondral bone. This bone consists of a tough outer shell (cortex) and an internal system of struts that intertwine (trabecular bone). The struts provide lightness, strength, and increased shock absorption. The forces of the joint are transmitted through the cartilage to the subchondral bone, decreasing stress and wear on the cartilage.

The perimeter of the joint is lined with synovium -- a thin sheet of cells that encompasses the entire periphery and attaches at the margins of the joint. This provides nutrition and secretes a fluid that reduces joint friction. The joint capsule also entirely surrounds the joint and is composed of a specialized layer of ligaments that form a second compartment that augments joint stability. Surrounding this entire arrangement are additional ligaments, providing further stability, tendons and muscles that attach near the joint to provide joint power and motion and bursa that act as friction pads for joint motion.

The architecture of the joint also provides joint stability and dictates the direction of joint motion. Specialized pads of fibrocartilage, and not hyaline cartilage, that are more susceptible to trauma, form menisci of the knee. The lateral meniscus is in the lateral compartment of the knee, and the medial meniscus is in the medial compartment of the knee. These attach to the margins of the knee, enhancing joint stability and by spreading stress over a larger area, reducing the wear on the joint. Dysfunction of the meniscus allows instability and more wear of the joint. Internal ligaments of the knee (cruciate ligaments) also provide stability. Tear of either the menisci or internal ligaments can lead to secondary

arthritis. Some joints, like the ball-and-socket joint of the hip, are inherently very stable. Most other joints, however, require ligamentous integrity for normal stability and motion. Damage to this normal support system of ligaments can lead to misalignment that can cause secondary osteoarthritis. Damage to the articular cartilage, especially if combined with damage to the subchondral bone, can cause secondary osteoarthritis. Diseases that affect joints, such as rheumatoid arthritis or infection, can permanently damage the joint and cause osteoarthritis.

In both primary and secondary osteoarthritis, the cartilage deteriorates because of biochemical events that degrade the normal composition and function of the cartilage. What initiates this process is unknown. Cartilage is lost irregularly, so that joint space loss on x-ray (which appears as decreased space between the ends of the bones forming the joint) is usually irregular. Bony spurs (osteophytes) form near the margins of the joints or vertebral bodies. These are a reaction to loss of cartilage and an attempt to further buttress the joint. Spurs alone, however, are common without damage to the joint or disc and cannot be considered definitive evidence of true osteoarthritis.

In more advanced cases the subchondral bone eburnates (polishes and enlarges). This is manifested on x-ray as density (sclerosis) of the subchondral bone. Bony cysts in the subchondral bone form by a mechanism not totally understood.

Of all these findings, irregular joint space narrowing or intervertebral disc narrowing is most diagnostic of osteoarthritis. As mentioned above, bony spurs are common without true damage to the joint or disc space. Without joint or disc space narrowing, spurs cannot be considered true osteoarthritis but degenerative changes that occur with aging.

Indeed, the term degenerative arthritis is a misnomer, as this suggests deterioration from aging and wear and tear. Neither is mainly responsible for primary osteoarthritis, which requires active biochemical events in the cartilage to cause cartilage deterioration.

EPIDEMIOLOGY

Osteoarthritis occurs with aging but is not caused by aging for the reasons noted above. Osteoarthritis of the fingers is more common in women, especially associated with the distal and proximal interphalangeal (finger) joints. Osteoarthritis of the thumb carpometacarpal joint (CMCJ) is much more common in women than men. Osteoarthritis of the knee is equal in men and women, but when it occurs before the age of 50, is more common in men. Osteoarthritis also occurs with certain diseases, but this is beyond the scope of this paper.

CLINICAL FINDINGS

Pain is the hallmark of symptomatic osteoarthritis and usually occurs with motion or weight bearing. Stiffness of the joint, especially after being kept in one position too long or first thing in the morning upon arising, is common but is usually rapidly relieved by motion. Joint enlargement from soft tissue proliferation around the joint, osteophytes and bony enlargement of the subchondral bone, make the joints

feel either firmly enlarged or swollen but without large amounts of fluid (effusion). Indeed, large effusions (fluid in the joint) are relatively uncommon. Crepitus (grinding and popping of the joint with motion) is caused by motion of the irregular joint surface. Pain usually begins insidiously but can be punctuated by acute episodes, sometimes accompanied by a large effusion. Most symptomatic osteoarthritis is slowly progressive, although the disease can spontaneously arrest.

The pattern of involvement is important both in terms of the type of osteoarthritis and the extent of disability. Primary osteoarthritis commonly involves select joints: The distal interphalangeal joints and proximal interphalangeal joints of the fingers, the thumb carpometacarpal joint, the acromioclavicular joint lying above the true shoulder joint, the hip, the knee and the great toe metatarsal phalangeal joint. A similar process affects the facet joints of the spine and the intervertebral discs. The discs are actually composed of fibrocartilage and are not true joints, but the same term (osteoarthritis) commonly refers to disc degeneration. Other terms, perhaps more appropriate, for disc degeneration are spondylosis and degenerative disc disease. When other joints are involved, for example the wrist, elbow, true shoulder joint (glenohumeral joint) or ankle, secondary causes should be scrupulously investigated, as these are rarely involved in primary osteoarthritis.

JOINT SPECIFIC CHARACTERISTICS

Osteoarthritis of the distal (DIP) and proximal (PIP) interphalangeal joints of the fingers can be associated with prominent nodal enlargement (Heberden's nodes of the DIP and Bouchard's nodes of the PIP joints), which is a subset of osteoarthritis that is genetically determined and is much more common in women around the menopause. In typical osteoarthritis of the fingers or with nodal osteoarthritis, decreased joint motion and fist closure is common. Joint deformities, which are usually mild, are also common, but disability is usually not severe, as pain is usually not severe. The hand has tremendous reserve function, which is why osteoarthritis typically produces less disability. Thumb carpometacarpal (CMC) joint osteoarthritis, however, is the exception. Because the thumb represents 50 percent of the function of the hand, especially for pinching and power grasping, very symptomatic osteoarthritis of this joint causes substantial disability. Bony enlargement of the thumb gives a squared-off appearance to the base of the thumb. Subluxation laterally is common in more advanced cases, although the joint is not usually hypermobile but actually has reduced motion, especially abduction and adduction.

The acromioclavicular joint above the true shoulder joint is usually enlarged by the time symptoms occur. This joint can be painful, or inferior spurs of the joint can impinge on the tendons of the shoulder (rotator cuff) leading to impingement syndrome and rotator cuff wear and tear.

Many authorities believe that osteoarthritis of the hip arises from minor developmental deformities of the joint leading to joint incongruity and excessive wear. Pain can localize to the groin, as the hip joint actually sits deep in the pelvis. Sometimes pain referred to the knee is much more prominent than hip pain, emphasizing the importance of examining both when knee pain is prominent. Pain on flexion combined with abduction and external rotation of the hip (Patrick's test, also referred to as FABERE test) is common. Reduced internal rotation is usually the first sign of limited motion.

Knee osteoarthritis is frequently associated with angulation of the knee. Most commonly the loss of the medial joint space leads to a varus deformity (knees together), and less commonly loss of the lateral joint space leads to valgus deformity (knees apart). The patellofemoral joint (kneecap) can be involved alone, but usually is involved with other compartments of the knee. Pain with motion or weight bearing is usually the first complaint. Firm or bony enlargement is common. Limited motion and joint line tenderness occur later. Joint effusions are usually small, but occasionally acute episodes are associated with large joint effusions.

Osteoarthritis of the metatarsal phalangeal joint (MTP) of the big toe can lead to enlargement and stiffness of the joint. This can cause pain on toe-off during walking or climbing.

Spinal osteoarthritis can affect all three areas of the spine, but thoracic osteoarthritis rarely causes symptoms. The disc acts as a roller bearing so that reduced forward motion in symptomatic osteoarthritis of the disc is common but can occur without pain. The facet joints are responsible for most lateral and oblique movements and extension. Both the disc spaces and facet joints are frequently tender in symptomatic osteoarthritis, and motion is frequently painful and limited. Bony overgrowth, combined with disc space and intervertebral narrowing, can cause compression of the nerves. When this occurs in the central spinal canal, it is referred to as central spinal stenosis. When this occurs in the intervertebral foramen, it is termed foraminal stenosis. Both can cause aching of the leg with standing or walking (spinal neurogenic claudication). Sometimes back pain is more prominent than leg pain, and other times it is the reverse. When a disc herniates, previously asymptomatic spinal stenosis can be converted to symptomatic spinal stenosis by loss of further space in the spinal canal or intervertebral foramen. Neurologic findings in spinal stenosis occur much less commonly, including changes in sensation and reflex, loss of muscle power and bulk and nerve root tension signs, such as straight leg raising. This makes evaluation more problematic.

DIAGNOSTIC TESTING

Osteoarthritis is not associated with blood abnormalities. Blood tests are done to exclude other diagnoses. Joint fluid, when present, shows minimal abnormalities with slightly increased white blood cell counts. X-rays remain the best tool to confirm the diagnosis. Sometimes symptoms precede x-ray changes. The most reliable sign of osteoarthritis, either of the joint or disc is actual narrowing of the joint or disc space. Other findings, such as spurring, can simply indicate aging and do not necessarily mean true osteoarthritis. MRI of both the joints and spine can detect earlier changes than x-rays. The significance of these abnormalities in correlating with a clinical diagnosis must be on a case by case basis. Important mechanical causes of osteoarthritis, however, include significant leg length discrepancy, and examination for any ligamentous or internal joint instability. A careful work and recreational history may point to a traumatic event in the past. Frequently the history, physical findings and simple laboratory testing, including blood testing, synovial fluid analysis and x-rays, yield the proper diagnosis.

DISABILITY

The location of the joint involved, the number of joints involved and the severity of pain, loss of motion and weakness determine loss of functional ability or disability. The fingers can tolerate substantial loss of motion and still function relatively well. Severe pain is less common with finger joint involvement but can occur and cause substantial disability. The thumb carpometacarpal joint, when more symptomatic, markedly reduces hand function because of its prominent contribution to power grasp and pinch. Acromioclavicular joint disability is rarely severe except for its contribution to impingement of the rotator cuff. Hip and knee osteoarthritis, when very symptomatic, are very disabling, as these are weight bearing joints. Involvement of the great toe metatarsal phalangeal joint rarely causes severe disability except when pain and stiffness significantly limit toe-off during walking and climbing. Spine involvement can be very disabling from disc or facet pain and, less commonly, spinal stenosis.

Usually osteoarthritis, except of the fingers, is limited to only one or two joints. When more than three areas are involved, the condition is termed generalized osteoarthritis. This is not uncommon. Multiple joint involvement can be confused with other diseases, and it frequently causes more disability. The same is true of disability from bilateral involvement of the hips or knees.

TREATMENT

The primary goals are to decrease pain and improve function. Education of the patient in the nature of the disease and the principles of treatment are the first priority. The patient should be strongly encouraged to increase physical activity that is not high impact, improve diet and achieve near ideal body weight. Obesity places increased stress on the joints. Generalized exercise, especially aerobic exercise, and exercises specific for the involved joint or spine, decrease pain and improve function. Generalized exercises are essential. Specific exercises increase joint mobility, reduce joint instability, and improve muscle strength. Strengthened muscles frequently reduce pain. Muscles provide stability and shock absorption for joints. For example, improvement of the rotator cuff muscles of the shoulder re-balances the muscles and can lessen impingement. Improvement of strength of the knee acts as a natural shock absorber for the knee, reducing pain. Increased muscle strength of the extensor and flexor muscles of the spine increases stability and reduces pain.

When such treatment is not enough, medication to reduce pain should start with the simplest and that associated with the least side effects. Tylenol can frequently help in milder cases. In moderate cases nonsteroidal anti-inflammatory drugs (NSAIDs) provide more relief. These medications can occasionally have serious side effects, especially in high-risk groups. Gastrointestinal side effects are common, and ulcers of the stomach and duodenum can cause serious morbidity and even mortality. In high risk groups there are prophylactic gastrointestinal protective medications that can reduce the risk. Individuals prone to kidney disease, especially diabetics, elderly individuals, those with heart failure and those on certain medications for hypertension or heart failure, are at increased risk of kidney dysfunction. These patients require more frequent monitoring. Other side effects, including liver disease, are much less common. Individuals at high risk must be monitored with blood testing frequently initially and those at low risk less frequently.

Supports and splinting can provide additional relief by resting the joint or enhancing stability. Intra-articular injections of corticosteroids can reduce symptoms up to months and sometimes even longer. Hyaluronate injections can restore normal viscoelasticity of the joint fluid and reduce pain for months.

In more advanced cases a variety of invasive techniques are available. Sometimes simple lavage (rinsing out) of the joint can provide temporary relief. This can be done with needles or at the time of arthroscopy. Arthroscopic debridement (scraping) of the joint surface, used for the knee, can provide relief for months and sometimes a year or more. Drilling holes in areas of cartilage loss can promote fibrocartilage generation, which can increase the joint space, decrease pain and improve function, sometimes for extended periods.

Certain specific procedures are available, such as removal of the acromioclavicular joint (Mumford procedure) and high tibial osteotomy to realign knee joints with primarily early medial joint compartment involvement. Arthrodesis (fusion) of finger and the thumb IPJs and the metatarsal phalangeal joint of the great toe are preferred, since joint replacements frequently break down. Still, replacement of finger and great toe joints is sometimes used, especially in individuals with primarily pain and not much loss of motion. Specialized procedures for the thumb CMCJ besides arthrodesis include implanting ligaments, (anchovy procedure) in the joint space, acting as a cushion and reducing pain. Total hip and knee joint replacements are successful in the vast majority of cases. The longevity of currently used devices is quite good. Total knee replacements frequently last indefinitely. Total hip replacements deteriorate at approximately one percent per year. Obviously, these replacements are best saved for older individuals, but they can be used very successfully even in younger individuals. Even with excellent results significant prophylactic disability must be imposed to prevent premature wear of these devices. Fusion of painful lumbar spinal discs using cages of metal in the disc space that promote bony ingrowth and pedicle screw systems to enhance fusion are new and still controversial techniques. These are the equivalent of arthrodesis (fusion) of a painful joint, except for the disc. Many remain skeptical that lumbar disc fusion will be as successful as joint fusion, and disc fusion should be approached with caution.

PREVENTION

Little is known about prevention beyond the basics. As what causes primary osteoarthritis is not known, there is no antidote. Certain common-sense rules apply: Eating a healthy diet, exercising moderately, maintaining near ideal body weight and avoiding acute injury. In the workplace proper ergonomics, rotating workers through repetitive, strenuous jobs, and workplace safety could substantially reduce the risk of osteoarthritis.

SUMMARY

Osteoarthritis is the gradual and irregular deterioration of joint hyaline cartilage or spine fibrocartilage. It is not simply wear and tear or aging but rather an active biochemical process that damages the cartilage. Although the hyaline cartilage of joints is relatively impervious to trauma, trauma can cause osteoarthritis in certain circumstances, especially when there is permanent damage to the cartilage and subchondral bone, malalignment of the bone, joint instability or illnesses that damage the joints. Although most

individuals have degenerative changes on x-ray by the age of 40, only 25 percent of the population develops symptomatic osteoarthritis, usually past the age of 50. Still, this makes osteoarthritis a very common disease, exceeding all other types of arthritis combined.

The hallmark of symptomatic osteoarthritis is pain. Other findings include bony enlargement and soft tissue swelling of the joint with large effusions the exception, limited motion, crepitus and disc and joint tenderness. The best diagnostic test remains x-ray. Narrowing of the joint or disc space indicates deterioration of the joint or disc cartilage and is the best determinant of osteoarthritis. The significance of early MRI changes of osteoarthritis remains problematic and requires matching complaints and physical findings.

Treatment is frequently successful, starting with the least risky measures, such as weight reduction, exercise and diet, and progressing up the pain ladder, using analgesic cream, supports, acetaminophen and non-steroidal anti-inflammatory drugs. Opioids should be avoided. Injections of corticosteroids and hyaluronate can reduce symptoms for months and sometimes longer. More aggressive management includes joint lavage, joint debridement and drilling of osteoarthritic areas to promote fibrocartilage growth. Surgery, most commonly total hip and knee replacement is frequently successful. Arthrodesis (fusion) of other joints, including the fingers and great toe metatarsal phalangeal joint, are usually preferred to joint replacement. The thumb carpometacarpal joint can be fused or cushioned using ligament imposition. The acromioclavicular joint can simply be removed, as it has very little function. Surgery for degenerative arthritis of the spine including using instrumentation (plates, cages and screws) is appropriate in selected cases. Prevention includes diet, exercise and maintaining ideal body weight. In the workplace avoiding prolonged, repetitive, strenuous activities by job rotation, proper ergonomics and workplace safety could substantially reduce osteoarthritis. The disability from osteoarthritis can be substantial, especially if it involves multiple joints or weight bearing joints of the lower extremities.

NECK PAIN & INJURIES

INTRODUCTION

Neck or cervical pain is ubiquitous in society and particularly common as a presenting cause of pain and disability among injured workers. It is so common that the expression "a pain in the neck" has become a figure of speech in our language. Causes for these cervical problems are numerous and include cervical spine or disc degenerative disease (spondylosis), damage to the spinal cord (myelopathy) or nerve roots (radiculopathy), muscular or myofascial syndromes and other more ominous spinal and non-spinal conditions.

EPIDEMIOLOGY

Neck pain represents less than 2% of all workplace injuries and is far less prevalent than low back pain as a cause of worker absenteeism. The one-year prevalence rate of neck pain in most industrialized countries is approximately 20%. The majority of these injuries are diagnosed as either a strain or sprain.

Certain occupations seem to have a predisposition to neck symptoms. Manual laborers have more symptoms than office workers, and the type of work seems to affect the risk. A history of twisting and bending during work as well as older age are strong risk factors. Workers who have been required to do repetitive tasks with their upper extremity as well as prolonged sitting with their head in a flexed position, such as during keyboarding, are at risk of developing mechanical neck pain. Studies have additionally pointed out the importance of non-physical or psychosocial factors such as job or life satisfaction in determining disability status and treatment outcome. X-rays cannot be used to predict symptoms or disability status.

ANATOMY

The cervical spine is significantly different in motion and function from the rest of the spine. Whereas the lumbar spine is well suited to accommodate heavy loads and provide stability, the cervical spine is better suited for mobility and is not required to transmit heavy loads. The neck is more subject to injury than any other portion of the spine because of its position and mobility.

The cervical spine is positioned on top of the relatively immobile thoracic spine. The head needs to be very mobile because it is balanced on top of the neck to bend and rotate through a wide range of motion, which maximizes our visual and auditory ability. The cervical spine also supports the muscles of the shoulder girdle and those that move the thoracic outlet during breathing.

In addition to the special articulations of the head and neck (occiput and C1) and C1 and C2 (atlanto-axial) as well as cervical vertebrae C3 through C7, other areas at risk for possible injury include the posterior facet (zygapophyseal) joints, the intervertebral discs, as well as the cervical spine muscles and their attachments.

There are seven cervical vertebrae with the upper two, the atlas (C1) and the axis (C2) being unusual in configuration while the lower five (C3 - C7) are similar in appearance. It is best to think of the cervical spine as made up of functional units: each two adjacent vertebra and their interposed tissues forming a functional unit. The bony structure and the ligaments that connect the vertebrae limit neck motion. The anterior portion of the functional unit comprises two vertebral bodies separated by the intervertebral disc which functions like a hydraulic shock absorber. The posterior portion includes the transverse processes, the posterior spinous processes and the supporting ligaments along with the articulating synovial facet (zygapophyseal) joints. These joints allow the complex movements of lateral flexion and axial rotation in addition to anteroposterior flexion and extension. When injured, the facet joints can refer pain into the neck, shoulders, and posterior head (occiput).

The eight cervical nerve roots exit through and occupy one-third of canal-like structures called the intervertebral foramina. The foramina are largest at vertebrae C2-3 and progressively decrease in size to C6-7. The first cervical root exits between the skull and the atlas (C1 vertebra) with the last cervical root (C8) exiting between vertebra C7 and T1. A C5-6 disc herniation will affect the C6 nerve root and a C6-7 herniation will affect the C7 root.

CERVICAL SPONDYLOSIS

Cervical spondylosis or degenerative disease of the cervical spine and its cartilaginous and ligamentous structures occurs over time due to cumulative and specific trauma. It is also a natural consequence of the aging process and is a common finding on x-rays of older individuals. While in the majority of cases, cervical spondylosis is not associated with significant symptoms, it can be associated with muscular and neurologic dysfunction along with pain and disability.

The discs, even in asymptomatic normal individuals, fragment, lose water content and collapse as we get older. Over time this disc degeneration leads to mechanical stress and bone formation or overgrowth at the vertebral body edges leading to bony bars (osteophytes) that can extend, impinge and encroach on nervous tissue. These osteophytes or "hard" discs are distinguished from "soft" disc protrusions or herniations that occur more commonly in young and middle-aged people.

Bony overgrowth and protrusions or collapse may contribute to spinal stenosis, a narrowing of the spinal canal or the neural foramina (the channel that the nerve roots run through). This can result in spinal cord impingement (myelopathy) or nerve root compromise (radiculopathy).

While there is some debate as to the role of cumulative occupational trauma in cervical spondylosis, it is generally accepted that an increased incidence is noted in individuals who carry heavy loads on their heads or shoulders and degenerative cervical disease is also seen in dancers and gymnasts. In an individual with a spine already compromised by spinal stenosis, repeated or severe cervical motion, particularly at the extremes of range over time will lead to cumulative trauma. Even when cumulative trauma is not specifically involved, cervical problems may develop associated with work activities and "light up" an underlying cervical degenerative (spondylosis) problem.

While cervical spondylosis is common and most often asymptomatic in the general population in middle and later years, it may also result in a variety of symptoms leading to complaints of pain and dysfunction.

CERVICAL RADICULOPATHY

Cervical radiculopathy is a pathologic process involving compromise and damage of the nerve root arising from compression secondary to disc herniation, cervical spondylosis or stenosis, tumor or trauma. Cervical radiculopathy can also present without any clear cause. It stands to reason that a similarly sized disc herniation in two individuals can result in two completely different clinical pictures. A disc herniation, when there is also congenital spinal stenosis, may be much more damaging than when no stenosis is present. One spine has more "room" to accommodate the encroaching disc herniation without severely compressing the nerve while in the individual with stenosis significant compromise may occur.

The most commonly involved nerve roots in cervical radiculopathy are caused by C5-6 or C6-7 disc herniations or spondylosis. The symptoms of cervical radiculopathy can include pain, abnormal sensations (paresthesias) and weakness. Complaints may involve the neck and upper back and/or the upper extremity. While there may be "objective" findings of reflex or strength changes in the affected limb in some individuals, the neurologic examination may be normal in others. Many patients present with a normal neurologic exam yet complain bitterly of neck and upper back or scapular discomfort with or without radiation to the arm. The pain may also be atypical and present as chest, breast, or facial pain. Complaints of headaches are common.

In addition to checking circumferential measurements for muscle atrophy, range of motion, strength, sensation and reflexes, the examiner may check neck or foraminal compression (Spurling’s maneuver) in which the head is extending backwards and to the symptomatic side and pressure is applied to the top of the head. This movement and pressure compresses or further narrows the space in the foramina for the nerve root thus eliciting symptoms.

CERVICAL MYELOPATHY

While a radiculopathy affects a nerve root, a myelopathy suggests compression and injury to the spinal cord itself. Cervical myelopathy, when not related to immediate trauma, is commonly secondary to degenerative spondylitic changes. It typically presents after the fifth decade and is often confused with other diseases. Individuals with this problem present with a slow progressive history of neurologic deterioration with remissions and exacerbations. Progressive myelopathy leads to subtle gait disturbance with lower extremity increased reflexes and tone (upper motor neuron lesion) and polyradiculopathic (lower motor neuron) changes in the upper extremities. There may be bowel and bladder dysfunction with episodes of incontinence. The differential diagnosis can include such diseases as amyotrophic lateral sclerosis (ALS) or multiple sclerosis (MS).

WHIPLASH

Whiplash injuries are based on physical pathology but are significantly affected by psychosocial factors. These factors can include such issues as coping strategies, family and work place problems, and an

adversarial compensation/litigation system. Whiplash injuries result in a collection of musculoligamentous symptoms (sprain/strain) typically involving rapid neck acceleration-deceleration and torsion often as the result of an automobile accident. The diagnosis is determined by the history provided, by the mechanism of injury, the symptoms and the physical findings on examination.

Neck pain is the most common manifestation of whiplash. The pain is typically described as dull and movement worsens aching over the back of the neck which. Sharp pain is often noted with quick movement and there are often associated complaints of stiffness and restricted neck motion. Pain may radiate to the head, upper back or shoulders. Headaches are also a common feature of whiplash with pain starting in the back of the head and moving forward. Other complaints can include visual disturbances, insomnia, dizziness, swallowing problems, weakness, and arm pain, sensations of numbness and tingling and occasionally memory and concentration problems. Whiplash sufferers can present with temporomandibular joint (TMJ) dysfunction, thoracic outlet syndrome and also with upper body myofascial pain and fibromyalgia complaints.

Common physical findings include posterior cervical tenderness and limited, painful cervical range of motion. Both cervical and upper back musculature may be tender and in "spasm." The neurological examination is usually normal. Radiologic findings will be minimal and may show straightening of the normal cervical curve secondary to muscle spasm. Pre-existing degenerative changes may be evident.

It is a mistake to discount physical injury and pain after whiplash as "compensation or litigation neurosis." There is good clinical evidence to support the physiologic basis of physical injury after whiplash. On the other hand though, it should be strongly emphasized that how an individual responds to injury and discomfort will often have more to do with psychosocial, historical and genetic factors than the physical damage itself.

OTHER CAUSES OF UPPER BODY COMPLAINTS

There are many possible causes of pain, sensory complaints and weakness that can affect the neck and upper body. This includes, but is not limited to, carpal tunnel syndrome, ulnar nerve entrapment at the elbow (tardy ulnar palsy), thoracic outlet syndrome, various tendinitis/bursitis problems, shoulder damage, neuropathy, and brachial plexus injuries. There are a host of other more ominous spinal and non-spinal conditions that deserve consideration, and which may present as cervical pain.

DIAGNOSTIC TESTING

Cervical x-rays can be helpful to define the degree of degenerative changes but must be considered with caution as cervical spondylosis is commonly found in asymptomatic individuals. Plain x-rays can reveal disc space narrowing, rheumatologic disease, destructive lesions, slippage, stenosis, fractures and instability with flexion-extension views. Magnetic resonance imaging (MRI) is noninvasive while showing good detail of neural elements. Gadolinium can be used in post-surgical spines to evaluate he presence scar tissue. When there are technical or individual reasons why the patient cannot undergo MRI testing, the most reasonable alternative is Myelography/CT scanning. Electrodiagnostic studies

(electromyography or EMG and nerve conduction velocity or NCV) may help distinguish the presence of a radiculopathy at a certain cervical root level or show evidence of peripheral nerve compromise.

GENERAL TREATMENT GUIDELINES

Initial care starts with providing patient education and assurance. Patient comfort is important and can usually be achieved with nonprescription analgesics. When such medications are insufficient, prescribed pharmaceuticals may be added along with physical methods.

Acetaminophen is the safest effective medication for acute cervical problems, but nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin and ibuprofen, are also effective although they can cause gastrointestinal or other problems. These drugs should not be taken concomitantly with regular alcohol intake.

Muscle relaxants are commonly prescribed but seem to be no more effective than NSAIDs and cause drowsiness in up to 30 percent of patients taking them.

Corticosteroids both orally or by epidural injection can be effective with cervical root irritation but have limited usefulness and need to be part of an overall rehabilitation-oriented treatment regimen.

Opioids appear no more effective than safer analgesics for managing acute neck symptoms. Opioids should be used sparingly and only for a short time in most situations.

Physical methods of treatment are numerous and include manipulation, traction, physical modalities, invasive techniques and other various therapies. Acupuncture is effective in selected individuals.

Spinal manipulation is safe and effective for patients in the first month of cervical symptoms without radiculopathy. If manipulation has not resulted in symptomatic and functional improvement after 4 weeks, the patient should be reevaluated.

Passive modalities such as heat and massage may play a limited role in acute cases but should be short term and part of a more functionally oriented neck rehabilitation program. Many cervical patients have referred pain to the upper back and extremity and should engage in a regular stretching and exercise program. Traction does appear to benefit the individual with a disc herniation but will often cause increased discomfort when the cause of symptoms is musculoskeletal or mechanical.

Patients with acute cervical problems should avoid undue irritation from activities and postures that increase stress on the neck and thus aggravate symptoms. Proper neck positioning and posture with work simplification should be taught to the patient. Debilitation should be avoided by having the patient engage in an incremental, gradually increasing program of aerobic (endurance) conditioning exercises.

Isometric exercises should be started early followed by gentle range of motion exercises. More vigorous strengthening, stretching and conditioning should follow this. The individual should be taught proper neck

care and good body mechanics. This should all be followed with a self-directed neck rehabilitation program.

Surgery may be a reasonable alternative for selected individuals when psychological issues are at a minimum and there is a good correlation between the clinical and radiologic findings.

SUMMARY

Neck or cervical pain is ubiquitous in society and particularly common as a presenting cause of pain and disability among injured workers. Causes for these cervical problems are numerous and include cervical spine or disc degenerative disease (spondylosis), damage to the spinal cord (myelopathy) or nerve roots (radiculopathy), muscular or myofascial syndromes and other more ominous spinal and non-spinal conditions. Diagnosis should start with a careful history and physical examination followed by rehabilitation oriented functional treatment program. Selected individuals benefit from surgical intervention. Injured workers who become increasingly psychologically and physically dysfunctional should be referred to the appropriate center for chronic pain management.

THORACIC OUTLET SYNDROME (TOS)

INTRODUCTION

The diagnosis and treatment of thoracic outlet syndrome (TOS) has evolved over many years surrounded by controversy and disagreement. In fact, even the existence of this syndrome as a true clinical entity has been questioned. The weight of clinical and scientific evidence though does suggest that certain individuals have symptoms and signs compatible with the diagnosis of thoracic outlet syndrome. In the great majority of these patients, a successful outcome can be achieved with a careful diagnostic workup and a conservative rehabilitation approach. Surgery is occasionally indicated in specific cases or when the rehabilitative approach has failed.

DESCRIPTION & ANATOMY

Thoracic outlet syndrome is a term describing compression of the nerves (brachial plexus) and/or the vessels (subclavian artery and vein) to the upper limb. This compression occurs in the region (thoracic outlet) beneath the clavicle (collar bone) between the neck and the shoulder. The thoracic outlet is bounded by several structures: the anterior and middle scalene muscles, the first rib, the clavicle, and, at a lower point, by the tendon of the pectoralis minor muscle.

The brachial plexus is a branching group of nerves that form from the nerve roots as they leave the spinal cord in the region of the cervical spine (neck). The brachial plexus passes anteriorly and downward, passing behind and underneath the clavicle (collar bone) where it branches into the peripheral nerves which course below and in front of the shoulder, and then down the arm to provide motor (strength) and sensory (feeling) function to the upper extremity.

As the brachial plexus and subclavian vessels pass through the thoracic outlet, there is potential for both static and dynamic compression and/or compromise. Since the thoracic outlet is a closed space, any intrusion or swelling such as from a fractured clavicle, hypertrophied or spasming muscle or tumor can lead to static compression of the structures that pass through that space. Further, in an already “tight” or compromised thoracic outlet, dynamic movements, such as holding the arm overhead and backward (hyperabduction), will put further compression on the enclosed structures and bring on symptoms. Even breathing can add compression to the thoracic outlet. Breathing is normally begun with the diaphragm, but in paradoxical or chest breathing the patient starts by elevating the upper ribs with the scalene muscles and this tightens the thoracic outlet.

SYMPTOMS

The character and pattern of symptoms will vary depending on the degree to which the nerves and/or blood vessels are compromised. The patient may complain of tingling, numbness, weakness and discomfort particularly down the inside of the arm going into the hand. There may also be swelling, paleness and coldness of the arm and hand. Other related symptoms may include headaches in the back of the head and pain in the neck, shoulder and arm. Symptoms can be brought on by overhead activities such

as hair combing, or at night when sleeping on one side which can put pressure on the structures within the thoracic outlet. Each of these maneuvers/positions causes a tightening or compression of the thoracic space. Thus the nerves and/or blood vessels may be compromised and produce the associated symptoms.

THE CONTROVERSY

Over the years TOS has taken on a negative connotation because of poor surgical results or because it has often been employed as a "wastebasket" term when the treating clinician is short on a diagnosis and unable to explain the patient's complaints. This has resulted in much controversy and disagreement among professionals along with confusion and distrust among claims examiners and patients.

TOS is commonly confused with the following clinical entities: cervical nerve root compression from a protruded disc or degenerative spine disease, stretch injuries to the brachial plexus, tumors, a myofascial pain syndrome or peripheral upper extremity nerve entrapment.

Once the diagnosis is made, too many practitioners have been quick to offer a surgical remedy. A surgical approach is at the core of the controversy precisely because it has either been too often recommended to treat clinical entities that are not TOS or else surgery has been inappropriately used in patients with TOS, with poor results in either case.

Thoracic Outlet Syndrome does exist and is usually treatable with a non-invasive physical rehabilitation approach. Surgery should be reserved for the exceptional case.

DIAGNOSIS

At the foundation of appropriate treatment for TOS, or for any pathology for that matter, is careful diagnosis. Diagnostic tests, such as EMG's or NCV's, may show non-specific abnormalities, but are often normal in patients with TOS. Therefore, these tests are used more for differential diagnosis than confirmation of the diagnosis of TOS. Specific protocols for TOS MRI scanning have become available.

After taking a thorough history, the physician or therapist will do a physical examination and then may perform a variety of thoracic outlet compression maneuvers during the physical examination to reproduce the patient's symptoms and thus help pin down the diagnosis. Thoracic outlet compression tests must be interpreted carefully, since even asymptomatic individuals can develop arm numbness, tingling, pain, and diminution of the wrist pulse with these maneuvers. It is a challenge to the clinician to determine the significance of findings on examination in light of the entire clinical picture, including consideration of non-organic, psychosocial factors of disability and dysfunction.

TREATMENT

Treatment approaches for thoracic outlet symptoms are directed toward alleviation or reduction of compression of this space. Surgery, if indicated, may involve total or partial removal of the first rib and an occasionally present extra cervical rib. One of the scalene muscles may also be removed if it felt to be entrapping or compressing the neurovascular structures. The risks with TOS surgery are significant and include brachial plexus and nerve injury, hemorrhage, infection, and pulmonary complications. Compared

with a non-surgical approach, patients receiving surgery had greater medical costs and have been found to be three to four times more likely to be work disabled. Therefore, due to often unnecessary surgery with poor results and the above-mentioned risks, surgery is not typically the preferred treatment.

More commonly and appropriately though, first-line treatment is directed towards a physical rehabilitation program, in which physical therapy plays a large role. Initial treatment emphasis is placed on weight loss, postural re-education, and shoulder girdle exercises along with stretching, strengthening, conditioning and the passage of time. Evaluation of activities of daily living and the workplace environment is a must. Physical therapy management of TOS requires accurate evaluation of the peripheral nervous system, posture, and the cervico-scapular muscles. Patients should be instructed in postural correction in sitting, standing and sleeping, stretching exercises, and strengthening exercises of the lower scapular stabilizers beginning in gravity-assisted positions to regain normal movement patterns in the cervico-scapular region. Other techniques include evaluation of joint mobility and muscular imbalance. Patient education, compliance to an exercise program, and behavioral and ergonomic modification at home and work are critical to long-term successful conservative management. Selected patients may benefit from trigger point injections or acupuncture treatments.

Short-term modalities such as heat, cold, massage, ultrasound and electrical stimulation may reduce symptoms temporarily and through pain reduction allow treatment and stretching of the effected tissues. The patient is taught to avoid postures or positions that aggravate symptoms. Patients are told to avoid drooping shoulders, to guard against the arms being above shoulder level and in a sustained position overhead. It is important to not carry heavy objects in the hand of the affected extremity or on the affected shoulder. The patient should avoid sleeping on the affected side. Biofeedback and/or relaxation training can be helpful in relaxing the involved musculature, retraining skilled hand function at the computer and improving postural awareness.

PSYCHOSOCIAL ASPECTS of TOS

Each person responds and reacts differently to discomfort. There are many individuals who for whatever reason are somatically preoccupied and/or may have underlying psychopathology or psychosocial dysfunction which seems to enhance their perception of pain and disability. Some patients become extremely angry and some display a deep sense of entitlement. Surgically removing a structural “abnormality” such as a cervical rib (a normal finding in many asymptomatic people) may be a devastating mistake in a patient whose problem is not a significant TOS, but rather emotional dysfunction, anger, entitlement or somatic preoccupation.

Patients with non-physiologic pain behavior, psychosocial dysfunction, anger or a sense of entitlement are difficult to treat. Often our best efforts lead to poor results and even more complaints of pain and disability. While difficult to treat, they need to be approached from a model of cognitive restructuring or altering thought processes. Basically, cognitive restructuring is used to change patterns of negative thoughts and self-defeating attitudes in order to generate more healthy and positive thoughts, emotions and actions. These patients tend to do better in a tightly controlled, behaviorally oriented, functional restoration program of chronic pain management.

SURGERY for TOS

If the patient is not responding to conservative physical rehabilitation treatment approaches, it is then important to differentiate between the occasional patient who should be considered for surgery and others for whom surgery would be a mistake and a potential disaster.

What patient then is a good candidate for surgery when conservative measures have failed? Surgery should be considered an option in the few select emotionally stable patients with significant neurovascular compression at the thoracic outlet. Even in some individuals with less than optimal results with conservative treatment, surgery is a mistake and will only lead to further problems. Surgery is never a "last resort," but rather only one tool to be considered for use.

SUMMARY

Thoracic Outlet Syndrome occurs with compression of the neurovascular bundle as it exits the cervical area, traverses behind the clavicle and progresses towards the upper arm. It is best treated with a rehabilitation approach but certain cases justify surgical intervention. A number of injured workers with TOS, with or without surgery, may appear considerably dysfunctional and disabled.

It is useful to find reasonable and knowledgeable physicians who are willing to take the time to provide excellence in diagnosis along with caring, but firm treatment. It is critically important for the physician to assist the patient in understanding that TOS is not life-threatening, is treatable and should not prevent return to some type of gainful employment.

Formal physical therapy should be time-limited with the patient being weaned away from medical intervention and thereafter engaged in a self-directed exercise and conditioning program. Medications should be limited to acetaminophen, aspirin, or one of the non-steroidal anti-inflammatory drugs. In an occasional patient, a tricyclic antidepressant may help with pain relief and insomnia.

SHOULDER PAIN & INJURIES

INTRODUCTION

A variety of disorders affects the shoulder and result in pain and functional disability. Shoulder injuries may result from specific acute, traumatic events or from repetitive overuse activities. A thorough history and physical examination are essential tools in constructing a differential diagnosis and treatment plan for the patient. This article will focus on the functional anatomy of the shoulder, as well as the more common conditions associated with shoulder injury.

FUNCTIONAL ANATOMY

As with other musculoskeletal disorders, a complete understanding of the anatomy and function of the shoulder is essential. The use of the term "shoulder joint" requires clarification. This term generally refers to the glenohumeral joint which is only one of the four joints that comprise the shoulder. Since all these joints are responsible for normal shoulder motion, the term shoulder girdle or shoulder complex is preferred. The shoulder complex is difficult to assess because of its many structures, complex movements and the variety of lesions that can occur either inside or outside the joints.

The glenohumeral (GH) joint is formed by the articulation of the humeral head with the glenoid cavity of the scapula. This joint is the most mobile joint in the body. It is a multiaxial ball-and-socket synovial joint, like the hip. However, the glenoid cavity is quite shallow, and mobility is obtained at the expense of stability. The glenoid labrum is an oval disc of fibrocartilage that is attached to the glenoid rim and serves to deepen the glenoid cavity, thus stabilizing the joint. This capsule and surrounding muscles and ligaments provide most of the stability.

Several muscles collectively referred to as the rotator cuff muscles (supraspinatus, infraspinatus, subscapularis and teres minor) perform the important motions of the GH joint. These four rotator muscles have a conjoined tendinous insertion that comprises the cuff. This is a common site of injury. Additionally, bursae surround this region and commonly become inflamed, resulting in a bursitis.

The acromioclavicular (AC) joint is formed by the articulation of the acromion of the scapula and the clavicle. It is a diarthrodial joint whose structural integrity arises from an intrinsic capsule, an articular disc and surrounding ligaments. Disruption of one or all of these ligaments may occur with a fall onto the lateral aspect of the shoulder. It is also a common source of symptoms caused by arthritic changes occurring within the joint.

The sternoclavicular (SC) joint is similar to the AC joint, however it is rarely involved in clinical complaints. Along with the AC joint it enables the humerus to move through 180 degrees of abduction. There is a disc between the two bony articulations, which adds significant stability. Disruption of this joint is most commonly seen in sports injuries or motor vehicle accidents.

Although the scapulothoracic (ST) "joint" is not a true joint, it functions as an integral part of the shoulder complex. It is comprised of the scapula (shoulder blade) and the muscles covering the posterior chest wall (thorax). It is important because it connects the upper extremity to the axial skeleton, and normal shoulder motion is dependent upon scapulothoracic movement. The scapula glides on the thorax during movement of the upper extremity. This motion is additive to the movement of the GH joint. After the initial 20 degrees of abduction at the GH joint, scapular motion contributes 1 degree of motion for every 2 degrees of GH motion. Several muscles act on these joints to coordinate complex movements of the shoulder. Injury to the nerve supply to these muscles, or the muscles directly will adversely affect shoulder function.

SPECIFIC INJURIES

The incidence of shoulder injuries has become increasingly prevalent in the workplace. The reason for this is unclear; however, an improved understanding of shoulder biomechanics and pathologic conditions affecting the shoulder has led health care providers to a better awareness of these disorders. The use of computers, resulting in prolonged repetitive keyboarding, may also be responsible for injuries to the shoulder as a result of cumulative microtrauma.

Fortunately, most shoulder injuries can be managed non-surgically with conservative treatment. Proper evaluation begins with a thorough history and physical examination. The presenting symptom is most often pain with or without associated weakness. A comprehensive history leads to a correct diagnosis in most cases. Determining the precipitating event and the mechanism of injury is essential in developing an accurate diagnosis. Physical examination begins with a careful visual inspection and palpation of the bony structures and soft tissues comprising the shoulder girdle. This is followed by range of motion (ROM) testing, neurological examination, special tests and examination of related areas. A systematic approach is the best way to avoid misdiagnosis.

SHOULDER PAIN ETIOLOGY: Intrinsic & Extrinsic

Causes of shoulder pain can be classified as either intrinsic or extrinsic. Intrinsic disorders arise from within the shoulder complex, while extrinsic disorders originate outside the shoulder. Intrinsic disorders of the shoulder characteristically involve complaints of pain aggravated by using the shoulder (e.g. raising the arm), localized or diffuse tenderness to palpation over the shoulder, and pain that can be elicited by active or passive motion of the arm. Intrinsic disorders can be conveniently divided into two types; overuse and traumatic injuries. The importance of the shoulder in daily work, home and recreational activities is usually taken for granted until it is injured. Overuse injuries include impingement syndrome, glenohumeral instability, degenerative arthritis, and adhesive capsulitis. Repetitive overhead activities or unaccustomed repetitive strenuous activities are frequently implicated as the cause.

Rotator cuff impingement syndrome is as a very common cause of shoulder pain. Impingement syndrome encompasses many previously used terms and may range in severity from bursitis and tendinitis to frank tendinous disruption (cuff tear). Disorders of the rotator cuff commonly occur secondary to repetitive overuse, which results in cumulative microtrauma. In addition, trauma and age-related degenerative changes can result in greater susceptibility of these tissues to injury. The rotator cuff muscles stabilize the glenohumeral (GH) joint and include the supraspinatus, infraspinatus, subscapularis and teres minor. These muscles are responsible for internal and external rotation and abduction of the arm.

In addition, these muscles depress the humeral head and oppose the strong upward pull of the deltoid muscle. The supraspinatus and infraspinatus are the most commonly involved structures in an impingement syndrome. Normally, the tendons glide smoothly in the space beneath the acromion (subacromial). Any change in the size of the subacromial compartment may also give rise to impingement signs. Common causes include bony spur formation and variations in the shape of the acromion and/or coracoid processes, which differ among individuals. Occasionally, calcification of the coracoacromial ligament may occur and can lead to impingement. There is also an area within the supraspinatus tendon that has a poor vascular supply and is susceptible to inflammation and tearing.

The subacromial bursa is located between the acromion and the rotator cuff and usually connects with the subdeltoid bursa. The purpose of the bursa is to facilitate movement and reduce shearing forces between the structures. They usually become inflamed in the early stages of an impingement syndrome, and may become chronically scarred and thickened. This results in a relative decrease in the subacromial space and may result in a worsening impingement. In patients with a rotator cuff injury, the humeral head tends to migrate upward against the acromion, resulting in pain and weakness, and propagates further insult and injury to the tendons. Weakness of the rotator cuff muscles may also occur with injury to the specific nerves innervating the muscles.

IMPINGEMENT SYNDROME

Early diagnosis of impingement syndrome is critical in preventing further progression, and in insuring a good response to conservative treatment. However, clinical signs and symptoms are often non-specific and too often diagnosis is delayed until a full-thickness tear has developed. There are usually complaints of pain localized to the anterior, lateral and superior aspects of the shoulder. Mild pain may be present at rest, but is usually exacerbated by reaching at or above shoulder level, pulling, pushing, or related activities. Physical examination typically reveals tenderness over the anterior and lateral shoulder with restricted active and/or passive range of motion. The presence of an impingement syndrome is suspected when the patient demonstrates a painful arc of motion, especially with abduction and flexion (shoulder elevation). Positive impingement signs are often associated with rotator cuff pathology. The Neer impingement test is positive when passive forward flexion of the arm above 130 degrees elicits pain. The abduction internal rotation impingement reinforcement test is performed with the arm abducted 90 degrees and bringing the forearm from neutral to internal rotation. Another positive impingement test (disappearance of pain after intrabursal injection of lidocaine) helps to support the diagnosis. A positive drop arm test, where the patient has difficulty slowly lowering the abducted arm, may also indicate a rotator cuff tear.

BICIPITAL & CALCIFIC TENDINITIS

Associated conditions such as bicipital tendinitis and calcific tendinitis may present alone or concomitantly with an impingement syndrome or glenohumeral instability. Bicipital tendinitis refers to overuse of the long head of the biceps brachii muscle. The long head of the biceps originates from the supraglenoid tubercle of the scapula and passes over the top of the humeral head to unite more distally with the short head in middle of the upper arm. The long head of the muscle moves the arm away from the body and rotates it inwardly.

The long head of the biceps is usually affected inside the bicipital groove. The inflammatory process involves the tendon and its sheath. This may occur secondary to direct trauma, overuse of the arm in an overhead position, or because of abnormalities in the soft tissues that surround the tendon. In addition, a shallow bicipital groove may lead to subluxation of the tendon.

The most common complaint is pain in the area of the bicipital groove. The pain increases with activity and abates with rest. Physical exam typically reveals tenderness over the biceps tendon. There are also provocative tests such as Speed's test (resistance to forward flexion of the arm with elbow extended and forearm supinated) and Yergason's sign (resisted supination with elbow flexed at 90 degrees) which help to confirm the diagnosis.

Most cases of bicipital tendinitis respond to non-operative treatment. Initial management includes avoiding offending movements (modified rest), NSAIDs, and gentle stretching exercises. Therapeutic ice and heat may help. Patients typically respond to a regimen of active physical therapy with progressive stretching and strengthening exercises. Local corticosteroid injections may also be beneficial.

GLENOHUMERAL INSTABILITY

Glenohumeral instability may be classified with respect to the mechanism of injury as either atraumatic or traumatic. Atraumatic instability is often associated with general ligamentous laxity and both shoulders may be affected. It is often associated with capsular laxity without disruption of the glenoid labrum or surrounding soft tissues. Instability may be unidirectional or multidirectional. Traumatic instability may be associated with either a single high-velocity trauma, often associated with fracture and/or dislocation, or microtrauma from repetitive overhead activities. Traumatic instability is also often associated with labral fraying or tearing and usually results in anterior/inferior instability. Management of instability is often dependent on the type of instability present. Patients with glenohumeral instability may also have associated impingement syndrome and bicipital tendinitis. Often the cause for these problems (especially in young adults) is the underlying instability. Instability may also be associated with numbness, particularly in the ulnar nerve distribution. Pain is often present in the posterior aspect of the glenohumeral joint. Assessment should always include evaluation for generalized ligamentous laxity. There is a wide range of variability in the degree of laxity among individuals, and therefore comparison to the uninvolved or asymptomatic shoulder is recommended. Provocative stress testing (apprehension test) can be demonstrated by gently abducting and externally rotating the patients arm. If the shoulder is ready to dislocate, the patient will likely resist further motion. Treatment is often coordinated similarly to impingement syndromes. Surgical intervention may be necessary in patients with chronic subluxation and/or dislocation who fail to respond to conservative treatment and/or who have neurologic dysfunction as a result of the instability.

DEGENERATIVE ARTHRITIS

Degenerative arthritis may develop from many years of task-specific work, or after a significant traumatic event (post-traumatic arthritis) although the actual cause may be difficult to prove. Primary osteoarthritis of the glenohumeral joint is relatively uncommon when compared with the incidence in weight-bearing joints. Arthritic changes occur more commonly in the AC joint. Pain from degenerative arthritis is usually gradual in onset and occurs with joint movement. Tenderness may be noted over the GH joint (usually

posteriorly) or the AC joint (anteriorly). Range of motion may be restricted, painful and associated with crepitus. Plain x-rays typically demonstrate joint space narrowing and/or osteophyte (bone spur) formation. Treatment usually consists of the judicious use of non-opioid analgesics, anti-inflammatory agents, and ice. Patient education is important to instruct the individual to avoid activities resulting in repetitive microtrauma (joint protection). In progressive cases where function is severely impaired, surgery, including joint replacement may be indicated.

ADHESIVE CAPSULITIS

Adhesive capsulitis is also referred to as "frozen shoulder." Its cause remains unknown. Ongoing debate as to pathogenesis has led to the proposal of several different treatment approaches. Controlled studies comparing outcome are lacking. The most remarkable pathologic change is fibrosis that can involve the articular capsule. Adhesive capsulitis may present independently or as part of a more complex clinical presentation. It often presents as a late sequelae of prolonged shoulder immobilization secondary to a variety of orthopedic and neurologic disorders affecting the arm, however it may present insidiously with no known specific precipitating event. Likewise, spontaneous resolution is known to occur. Findings on examination are limited active and passive shoulder movement, especially in internal and external rotation and abduction. Complaints of a painful arc (as described in impingement syndrome) may or may not be reported. Treatment regimens vary, however it is agreed that the best therapy is prevention. Thus, at the earliest onset, the patient should be involved in an aggressive physical therapy program. Adjuncts such as analgesics and intra-bursal or intra-articular steroid injections may also be useful. In progressive cases, passive manipulation under general anesthesia or surgery may be required. These interventions should be immediately followed by aggressive physiotherapy. Other intrinsic disorders include humeral fractures, rheumatoid arthritis, gout, pseudogout, and osteonecrosis. These will not be discussed.

Extrinsic disorders originating outside the shoulder may refer pain to the shoulder and present a diagnostic challenge. Pathology involving the cervical spine, brachial plexus, visceral disease (e.g. gallbladder disease) or neoplastic disease may be culprits. One example of a common extrinsic disorder causing shoulder pain is cervical degenerative disease and radiculopathy. Assessment of the shoulder region should always include evaluation of the cervical spine to rule out referred symptoms. Discussion of specific extrinsic disorders related to shoulder pain is outside the scope of this article. Nonetheless, the importance of a complete history and physical examination to exclude these potential causes is imperative. Definitive diagnosis and treatment of these disorders usually necessitates referral to the appropriate specialist.

DIAGNOSTIC IMAGING

Plain radiographs are the first imaging step in the diagnosis of shoulder injuries. They should always be obtained when direct trauma to the shoulder occurs (e.g. fall onto the shoulder), or when the clinical presentation and findings suggest fracture, dislocation, bony tumor invasion, bony infection or rheumatological disorder. Plain x-rays may be helpful in assessing overuse injuries but should only be obtained if the patient fails to respond to conservative management. Degenerative changes within the AC or GH joint may be seen. Calcium deposits would suggest calcific tendinitis, and a modified transcapular or outlet view will demonstrate the configuration of the acromion. Although there is no consistent radiographic finding in acute or partial rotator cuff tears, indirect evidence of chronic tears may be seen

radiographically as a narrowing of the space between the acromion and the humeral head, and sclerosis of the greater tuberosity. Additional diagnostic studies such as computed tomography (CT), CT arthrography, magnetic resonance imaging (MRI) and fluoroscopy may be indicated in conditions that do not respond favorably to treatment, and that might require surgical intervention. Because of its high soft-tissue contrast resolution and multiplanar imaging capability, MRI has been demonstrated to be an effective means of early identification of impingement syndrome and rotator cuff tears.

TREATMENT

Treatment for all these conditions is typically conservative, especially in acute and mild cases. Initial therapy should be aimed at reducing pain, inflammation, and mechanical dysfunction and improving shoulder motion. Therapy is based on activity modification, a decrease in the load placed on the shoulder, stretching and increasing the strength of the rotator cuff muscles (Codman's exercises). Oral anti-inflammatory agents and/or analgesics are frequently used to relieve discomfort and facilitate therapy. Ultrasound, heat, ice and steroid injections may be used similarly. Reinjury prevention is also an important aspect of treatment. Those patients who have a more severe clinical presentation or those who do not improve over three to four months or worsen will require further diagnostic evaluation, and more aggressive treatment. Indications for surgery are made on an individual basis and should take into account the persons age, underlying pathology, clinical course (e.g. acute vs. chronic), functional limitations, progress with conservative treatment, and obviously the patient's wishes. Specific surgical procedures will not be discussed.

SUMMARY

The incidence of shoulder pain is increasing in the workplace. The normal use of our arms is required for the vast majority of industrial and non-industrial activities, and shoulder pain can result in significant impairment and disability. The differential diagnosis of shoulder pain is quite extensive, and a comprehensive history and physical examination are important in order to establish a diagnosis and a focused treatment plan. Most conditions respond well to conservative management, however in certain instances surgical intervention may be warranted.

ELBOW & FOREARM PAIN & INJURIES

ANATOMY

The elbow is a hinge and pivotal joint that has the primary function of positioning the hand. Movements of the elbow include flexion and extension, as well as rotation - pronation (palm facing downward) and supination (palm facing upward). Normal range of motion is from 0 degrees (fully extended) to 145 degrees (fully flexed), and 75 degrees pronation to 85 degrees of supination.

The bony structures comprising the elbow include the end of the humerus in the upper arm and the proximal portion of the radius and ulna in the forearm. There are three bony articulations; the ulna and humerus (ulnohumeral), the radius and humerus (radiohumeral), and the radius and ulna (radioulnar). The ligaments around the elbow include the medial and lateral collateral ligaments, the annular ligament (which holds the radial head to the ulna), and the interosseous ligament between the radius and ulna.

Several muscles are involved with functional movement of the elbow. These include the biceps and brachialis (flexion), triceps and anconeus (extension), pronator teres, and supinator muscles. Additionally, several muscles originate at the elbow and are involved in common musculoskeletal problems associated with elbow pain. These include the wrist and finger flexor and extensor muscles.

Several neurovascular structures cross the elbow including the ulnar, radial, and median nerves, and the brachial artery and vein. The location of these structures is important when evaluating patients after traumatic injury to the elbow and additionally when considering procedural interventions near the elbow.

MEDICAL PROBLEMS

Medical problems involving the elbow area typically present with pain (most common), stiffness and/or swelling. Pain and swelling soon after an injury may be secondary to a fracture or dislocation or a tendon rupture. The patient should be evaluated for neurovascular injury, which if present will require emergent intervention. An x-ray is usually indicated when pain and swelling follow a specific trauma. A fracture or dislocation will be evident on x-ray. If the x-ray is unrevealing, a tendon rupture should be considered. When there is a bulbous swelling associated with redness and warmth at the tip of the elbow, an olecranon bursitis should be suspected. This bursitis may or may not be related to direct elbow trauma. When sudden swelling and pain are present, consideration should also be given to infection or an inflammatory condition such as gout or rheumatoid arthritis.

LATERAL & MEDIAL EPICONDYLITIS

An epicondylitis is actually not a tendinitis or inflammatory condition, but rather involves tissue degenerative or tendinosis. This condition is very common and most often develops from overuse of the forearm muscles in motions that require rotation of the forearm and movements of the wrist. Lateral epicondylitis or "tennis elbow" describes a condition characterized by lateral elbow pain. The individual

usually complains of pain over the outside of the elbow and in the upper outer forearm. The pain can be provoked when the elbow is extended, palm down while the individual attempts to extend the fingers and wrist against resistance. The pain can be debilitating and work disabling. It may be caused by direct injury or from repetitive overuse activities of the upper extremity. It is associated with recreational activities such as tennis and other racquet sports and is commonly seen in occupations that require repeated contractions of the extensor and supinator muscles. A medial epicondylitis or "golfers elbow" occurs on the inside of the elbow and can be seen in golfers and handball players. It is usually seen in occupations that require repeated contractions of the flexor and pronator muscles. Professions in which repeated contractions are performed against a resisting force are particularly prone to development of lateral and/or medial epicondylitis.

Treatment is typically directed towards eliminating activities that cause pain, the use of non-steroidal anti-inflammatory medications and/or a short course of oral steroids. A tennis elbow strap or wrist brace may be helpful and are basically used to remind the individual to avoid painful activities. The tennis elbow strap theoretically prevents excessive muscle contraction by constriction and thus changes the forces at the lateral and medical epicondyle.

In the acute stage ice may be helpful, and when chronic, the patient may prefer heat. Ultrasound to tolerance and deep friction massage may be beneficial. A carefully designed progressive stretching and strengthening program is indicated, but it is just as critical to look at ergonomic issues so as to prevent further injury and/or reinjury in both work and recreational activities. A series of no more than three steroid injections may also be beneficial.

Some patients with refractory epicondylitis may actually have a radial tunnel syndrome, which is entrapment of the posterior interosseous nerve (a branch of the radial nerve) as it passes under the supinator muscle. In these recalcitrant cases, a surgical release may be indicated.

OLECRANON BURSITIS

Posterior elbow pain is usually associated with an olecranon bursitis, which is an inflammation of the bursa between the tip of the elbow and the skin. This bursa is superficial and is prone to trauma and irritability. Patients with an acute bursitis usually have pain and swelling, which can be treated with aspiration (removing fluid with a needle and syringe) and compression along with non-steroidal anti-inflammatories. If there is redness, warmth, significant tenderness, or a nearby break in the skin, the bursa must be aspirated, and the fluid sent for culture to rule out infection. If an infection is suspected, treatment with oral antibiotics is indicated. With chronic olecranon bursitis, there may be recurrent swelling along with the sensation of tender nodules from scarring. In these cases, the physician may decide to instill a steroid after aspiration. Surgical treatment is only required with repeated recurrences or infections unresponsive to antibiotic treatment. The differential diagnosis includes fracture of the olecranon process, rheumatoid arthritis, gout or a synovial cyst of the elbow joint.

DEGENERATIVE JOINT DISEASE

Degenerative changes in the elbow are relatively rare and usually seen in patients with a history of previous trauma. It is often difficult to attribute degenerative changes in the elbow to industrial causation, however specific and/or cumulative trauma may aggravate underlying degenerative disease. Patients will usually complain of varying degrees of pain and stiffness. They may have reduced range of motion. Occasionally, a loose body may occur when a bone spur "breaks off" after an acute trauma, and this may require arthroscopic removal. Prosthetic joint replacement is reserved for severe and debilitating degenerative disease.

DISLOCATIONS

Elbow dislocations are the second most common dislocation in adults, second only to the shoulder, and are typically related to trauma. They are most common in young adults due to sport injuries (50%). Posterior dislocations are the most common (98%) with a fall on the outstretched hand as the most likely cause. Elbow dislocations rarely occur in isolation and are usually accompanied by other injuries such as a radial head fracture or damage to the radial artery or median nerve. The patient usually presents with pain, swelling and the inability to bend the elbow. An x-ray usually confirms the diagnosis. Treatment involves reduction as soon as possible followed by a posterior elbow splint. Anti-inflammatory medication may be helpful and after one to two weeks the splint is removed, and gentle range of motion exercises are initiated. Prolonged immobilization should be avoided because of the potential for loss of elbow motion. Repeat dislocation or ongoing instability is uncommon.

FRACTURES

Fractures of the distal humerus are relatively rare but may be associated with serious complications. Nondisplaced fractures can be treated with splinting and early motion. Displaced fractures require open reduction. Following an injury, the individual presents with pain, swelling, deformity and discoloration (ecchymosis) around the area.

Fractures of the olecranon are more common because of its location. The olecranon is the proximal end of the ulna and is the bony prominence at the tip of the elbow. The swelling may cause nerve compression and associated numbness distally in the fingers. Nondisplaced fractures can be treated with a splint with the elbow at 90 degrees. Most displaced fractures are best treated surgically by internal fixation with plates or a combination of wires and pins. With the more severe fractures, the elbow may lack full motion and there may be loss of strength. In addition to persistent elbow pain, the individual may develop subsequent degenerative joint arthritis.

Fractures of the radial head usually result from a fall on the outstretched hand with the arm and forearm turned in. Dislocation of the elbow is often associated with radial head fractures. These fractures may be difficult to visualize on initial x-rays and follow-up films 10 to 14 days and/or bone scan may be necessary to confirm the diagnosis. Sometimes a prominent posterior fat pad is the only radiographic evidence seen and is a result of bleeding into the joint secondary to the fracture. Treatment is conservative with a splint

for nondisplaced fractures followed by range of motion exercises. Surgery is reserved for more severe injuries. A poor outcome will result in loss of motion and instability.

SUMMARY

The elbow is very important in allowing position and functional use of our hands. Elbow and forearm pain is common and may be secondary to both vocational and avocational activities. Elbow and forearm problems are frequently seen in workers who are using a computer keyboard repeatedly and/or continuously, and in those occupations that require forceful gripping. Ergonomic assessment and activity modification may dramatically affect the clinical course and outcome. Traumatic injuries to the elbow may result in nerve or vascular injury, and/or loss of motion resulting in disability. Early diagnosis and appropriate treatment is essential.

ULNAR NERVE COMPROMISE AT THE ELBOW

INTRODUCTION

Injured workers can present with symptoms suggestive of injury to the ulnar nerve. This nerve can be entrapped or compromised at various locations along its path. This article will address ulnar nerve compression at the elbow.

ANATOMY

As the C8 and T1 nerve roots in the lower neck area leave the spinal cord and become part of the brachial plexus (located between the neck and the shoulder underneath the clavicle/collar bone), some of their fibers reform into the ulnar nerve which then courses down the arm. At the middle of the arm, as the ulnar nerve passes posteriorly and at the inside of the elbow, the nerve enters a groove formed by the medial epicondyle of the humerus and olecranon of the ulna. The ulnar nerve then continues along the medial forearm and through the wrist into the hand. You may be able to find your own ulnar nerve by feeling on the inside of the elbow. There is a depression between the two bony ridges and the ulnar nerve travels in this groove. The nerve feels like a piece of thick string or thin rope. If you tap on it ("funny-bone"), you may get a jolt-like electric shock.

FUNCTION

The ulnar nerve in the elbow area contains both sensory (feeling) and motor (movement) fibers. The sensory portion provides sensation to the medial palm and half of the fourth and the entire fifth finger. The ulnar motor function includes flexing the wrist and last two fingers toward the ulna side and spreading the fingers apart.

TERMINOLOGY

Various terms have been used in the medical literature to describe ulnar nerve compromise about the elbow. Terms utilized have included "Tardy Ulnar Palsy" (tardy meaning late, i.e. occurs years after an elbow injury, dislocation or fracture) and "Cubital Tunnel Syndrome" (from the Latin cubitum meaning elbow). In medical reports you will also see terms such as ulnar nerve compromise, compression and entrapment at the elbow. Other accepted terminology includes "Ulnar Neuropathy at the Elbow."

ETIOLOGY

The ulnar nerve at the elbow is quite superficial and is easily injured. The sensory and motor fibers that innervate the hand are more superficial and therefore more susceptible to damage than those to the forearm. The ulnar nerve is more easily traumatized with the elbow flexed as with flexion, the groove or tunnel is actually smaller.

Almost everyone has had the experience of hitting their elbow or "funny bone" and getting a jolting shock-like electrical sensation down the arm. When this happens, the ulnar nerve is hit and produces this uncomfortable feeling. Usually, hitting it just once without too much force doesn't cause any damage, but with a severe enough injury or with repeated trauma over time, the nerve can be damaged and lead to numbness, weakness and pain in the forearm and hand.

Something as simple as chronically leaning on or flexing your elbow can lead to ulnar nerve problems in some individuals. Some patients with other medical problems, i.e., diabetes, are in fact more sensitive than others to such nerve injury. Further, anatomical differences such as a narrow groove or fibrous bands can lead to certain people being more susceptible to ulnar nerve compromise about the elbow.

Additionally, approximately 16% of asymptomatic individuals have a hypermobile ulnar nerve which "pops" out of the bony ulnar groove with elbow flexion. For most of us, this "dislocating" ulnar nerve remains quiescent or asymptomatic, but it is more likely to be subject to direct or indirect trauma. For instance, an individual who leans on the elbow a lot at work may traumatize the nerve cumulatively over time.

Entrapment or compromise of the ulnar nerve at the elbow is actually the second most common compression neuropathy of the upper extremity, with only carpal tunnel syndrome presently more frequently. Injury to the ulnar nerve at the elbow represents a major disability in the work force, particularly considering the increased use of computers and the accompanying use of the elbows in a flexed posture.

Potential causes of ulnar nerve injury at the elbow are many. There may be an anomalous constricting muscle or fibrous band compressing the nerve. Previous trauma or a congenitally narrowed bony ulnar groove or a short nerve may be responsible. Space-occupying lesions such as ganglions, lipomas, bony osteophytes, rheumatoid synovitis, or hematomas may all lead to ulnar compromise at the elbow.

"Sensitive" nerves from an underlying metabolic problem an/or neuropathy (diabetes, renal failure, alcoholism, etc.) may be implicated. Habit patterns and work positions (leaning on or flexing the elbows) may be factors in the etiology of ulnar neuropathy at the elbow. Importantly, there may be a combination of these factors which together lead to symptoms.

PRESENTATION & EVALUATION

The physical findings for patients with ulnar nerve compromise at the elbow can include the following: The patient may complain of medial elbow and forearm pain. There may be tenderness and/or enlargement of the nerve as it passes behind the medial epicondyle of the humerus (ulnar groove or tunnel) where it is easily palpable.

The patient may complain of numbness, tingling and coldness in the region innervated by the ulnar nerve, especially the ring and little finger. There may be weakness and atrophy in the muscles supplied by the ulnar nerve as well. With nerve injury, there can also be a burning, hypersensitivity type of pain (causalgia).

Examination should additionally address whether there is subluxation or dislocation of the nerve with elbow flexion and whether with gentle tapping over the nerve at the elbow there is a positive Tinel's sign (reproduction of the shock-like sensation). Remember, gentle tapping should be used, as too vigorous percussion will give a positive Tinel's test even in normal individuals. Symptoms may also be reproduced by a provocative test, holding the elbow flexed for several minutes. Alternatively, elbow extension may relieve symptoms.

A staging or grading system has been devised to distinguish mild from moderate and severe ulnar nerve compromise. Such a system can help in determining outcome and for recommending treatment. Mild cases includes paresthesias (numbness and tingling) that come and go, subjective weakness and complaints of clumsiness and elbow flexion testing and/or Tinel's sign which may be positive. Moderate cases also have paresthesias that come and go, but there is measurable weakness and the elbow flexion and/or Tinel's sign are positive. In Severe cases, paresthesias are persistent, vibration perception is decreased, there is abnormal two-point discrimination along with measurable weakness and atrophy and testing for elbow flexion and Tinel's sign is positive.

In addition to these clinical findings on history and physical examination, an electrodiagnostic (electromyography/EMG and nerve conduction testing/NCV) evaluation can be extremely useful in localizing the problem to the ulnar nerve at the elbow. The electromyographer can determine that there is specific electrical slowing of nerve conduction across the ulnar nerve at the elbow along with needle EMG changes in the distal muscles specific to this diagnosis. An x-ray including special views of the cubital tunnel may be helpful in revealing bony abnormalities.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes ruling out a C8 and/or T1 cervical radiculopathy, thoracic outlet syndrome or ulnar nerve compromise at the wrist or elsewhere. Rarely, a pancoast tumor at the lung apex may compress the brachial plexus and present early as involvement of the ulnar nerve.

It is also possible that two different problems are occurring at the same time; this is termed a "double crush" syndrome. The patient may have an ulnar entrapment at the elbow along with another problem such as a lower cervical radiculopathy, a thoracic outlet syndrome or ulnar nerve entrapment at the wrist.

As an example, if the injured worker has a mild thoracic outlet syndrome along with ulnar nerve compromise at the elbow, either one alone may be sub-clinical, but together they add up to being symptomatic suggesting an ulnar nerve problem. If recognized as a dual problem, simply treatment with thoracic outlet exercises and padding the elbow to protect the ulnar nerve may be sufficient treatment and will avoid needless surgery for either.

Further, in the patient with ulnar nerve compromise at the elbow the clinician must consider whether this finding is singular and related to compromise at the elbow alone or rather only an early symptom of a more diffuse process such as a peripheral neuropathy. Recognition and treatment of an underlying metabolic dysfunction or neuropathy will lead to a more successful outcome while avoiding unnecessary

and potentially damaging surgery. Once medically stabilized, if the compression is progressive or severe, surgery may be indicated to prevent neurologic deterioration.

PREVENTION & TREATMENT

In mild cases of ulnar nerve compromise at the elbow where symptoms are predominantly sensory with little or no weakness, conservative care is successful in the majority of cases. Patients are told to wear an elbow protector (cushioned pad), to minimize working or sleeping with the elbow flexed and to discontinue leaning on the elbow.

For patients undergoing any surgical procedures or for those comatose or obtunded in intensive care units, positioning is important to prevent injury to the ulnar nerve. The shoulder should be externally rotated and elbow flexion minimized while the elbow is also protected with a soft pad.

Nonsteroidal anti-inflammatory drugs (NSAIDs) and a Transcutaneous Electrical Nerve Stimulator (TENS) can be helpful. Hand therapy for strengthening and range of motion is recommended.

In moderate to profound cases where there is progressive weakness, especially in the presence of significant electrical slowing across the elbow and severe EMG changes, surgical treatment may be warranted. There is some controversy in the medical literature about whether surgery should be limited to decompression of the nerve and release of any restricting bands versus freeing up the nerve and transposing or moving it anteriorly so it no longer gets "stretched" or damaged in the bony ulnar groove. Some surgeons recommend removing the medial epicondyle of the humerus. Of greater controversy is whether an "internal neurolysis" should be performed. In this procedure the surgeon tries to separate nerve bundles (fascicles) and release scar tissue.

If surgery for ulnar compromise at the elbow is to be carried out, the least invasive procedure is usually the best; i.e., a decompression alone is the safest and usually the best procedure. Trying to move nerves, chip off pieces of bone and separate out small nerve bundles can lead to iatrogenic (doctor caused) injury and can result in a patient who is worse off than before the procedure. This does not mean that a more involved surgical procedure is never indicated. When there is post-traumatic scarring, a space-occupying cause such as a bony ridge (osteophyte) or a subluxing nerve, then more involved surgery may be appropriate.

Surgical results depend greatly on the degree of preoperative compression and nerve damage. Those patients with muscle weakness and atrophy have the poorest outcome. With surgery, there may be immediate postoperative relief of paresthesias, but the patient should expect a gradual recovery process of weeks to months depending on the preoperative severity. Post-operative therapy and rehabilitation are recommended.

When surgery has failed and the patient’s symptoms have not responded to operative intervention, a number of possibilities exist. Consider whether enough time has gone by to allow for healing and was the original diagnosis correct and if so, was the correct procedure performed. Re-exploration and a more

involved procedure may be warranted, but the patient must be cautioned that improvement may be minimal and there is potential for symptom worsening.

Making a recommendation with regard to treatment and surgery is where the "art" of medicine and surgery comes in. The best physician is usually a well-trained and meticulous individual who doesn't always think that a more complicated and involved procedure is necessarily best and doesn't feel they are invincible and can do no harm. This "physician-healer" weighs all aspects of the patient's presentation, both physical and emotional, makes a recommendation based on all the available information, and then proceeds with caution and concern.

SUMMARY

The ulnar nerve may be compromised at the elbow for a variety of reasons. The first step is diagnosis to determine if the problem is limited to the elbow, secondary to a more widespread problem such as a neuropathy or possibly related to an entirely different cause. The history, physical examination and electrodiagnosis are important tools leading to a diagnosis.

Once diagnosed as an ulnar nerve compromise at the elbow, initial treatment should include wearing a protective pad, limited elbow flexion and preventing leaning on the elbow. An ergonomic evaluation of the workstation is appropriate. Medications and therapy may be of benefit. Surgery should be avoided if possible but if needed, a simple decompression is usually best. In certain circumstances, more involved surgery is recommended.

CARPAL TUNNEL SYNDROME

INTRODUCTION

This section will focus on the prevention, diagnosis, management and treatment of carpal tunnel syndrome. CTS is the most common peripheral neuropathy and the foremost cumulative trauma disorder (CTD) of the upper extremities. It also is one of the costliest and frequently reported workers' compensation claims. CTS results from injury to, or excessive pressure on, the median nerve at the wrist.

ANATOMY

The carpal tunnel is bounded on three sides by the carpal bones (the bottom) and by the transverse carpal ligament (the roof) on the palm side of the wrist. In addition to the median nerve, running through this tunnel are blood vessels and the finger flexor tendons.

The flexor tendons are important because they allow us to move the fingers and the hand, such as when we grasp objects. The tendons are covered by smooth, slippery synovium that allows the tendons to glide against each other as the hand is used to grasp objects.

The median nerve runs into the hand to supply sensation on the palm side to the thumb, index finger, long finger, and half of the ring finger. The nerve also supplies a branch to the muscles of the thumb, the thenar eminence. These muscles help move the thumb and are very important in moving the thumb so that you can touch each of the other fingers (called opposition).

PATHOPHYSIOLOGY

The diameter of the carpal tunnel normally allows enough space for the structures that pass through it. For whatever reason, when this confined space is compromised, the median nerve and its blood supply may become compressed and the nerve damaged.

Any condition, which causes irritation or inflammation of the tendons, can result in swelling and thickening of synovium. When this occurs, the pressure begins to increase in the carpal tunnel because the bones and ligaments that make up the tunnel are not able to stretch in response to the swelling. Increased pressure in the carpal tunnel begins to squeeze the median nerve against the transverse carpal ligament. Eventually, the pressure reaches a point when the nerve can no longer function normally. Pain and numbness in the hand begins.

INCIDENCE AND PREVALENCE

The condition can occur at any age but is most often encountered in working individuals over 30 years of age and in the general population between 50 to 60 years of age. Carpal Tunnel Syndrome affects 0.1% of the general population and as many as 15% of workers in high-risk industries such as electronic parts

assemblers, musicians, and dental hygienists. One study found the risk of CTS was almost 15 times greater for workers in jobs combining high repetitiveness and cold temperatures. It occurs three to five times more frequently in women than in men.

Although the use of highly repetitive wrist movements appears to be correlated with the development of carpal tunnel syndrome, other factors, such as medical conditions like diabetes, rheumatoid arthritis, thyroid disease, and pregnancy may be even more important. Nonetheless, the incidence rises with increasing repetition of hand use (such as typing), and by a high level of force with each motion, (such as with meat packers), in whom the incidence of carpal tunnel syndrome has been reported as high as 15%.

Approximately 25% of individuals with rheumatoid arthritis and 10% of individuals with diabetes mellitus will develop CTS during the course of their disease.

In recent years, increased numbers of workers have presented with carpal tunnel syndrome. One reason for this increase may be that automation and job specialization have fragmented workers' tasks to the point where a given job may involve only a few manipulations performed thousands of times per workday.

SYMPTOMS

The individual with a carpal tunnel syndrome typically complains of a feeling of numbness, tingling or burning pain in the distribution of the median nerve, particularly with wrist and hand activities. The symptoms may wake them at night. The complaints may actually be worse in the early morning hours or upon awaking and may be partially relieved by shaking the hands. Symptoms are particularly worsened by prolonged activities with the wrists flexed or with using the fingers and gripping fists, e.g., driving, tool handling, keyboarding, knitting, sewing, etc.

Symptoms are frequently bilateral, but usually worse in the dominant hand. Pain can extend to the forearm and shoulder or neck. The individual may complain of weakness, dropping things or clumsiness.

CAUSES

It is important for the physician to obtain a detailed medical, occupational and social history, including recreational activities and hobbies. CTS tends to run in families, so this part of the history is important too.

Any condition that compromises the median nerve at the wrist, either by compression or vascular impairment may lead to carpal tunnel syndrome. This could include:

- Distortion of the bony or ligamentous configuration (fractures, injuries, infections, etc.)
- Increasing the volume of the contents may cause compression of the median nerve (tumors, synovial inflammation, swelling during pregnancy, with rheumatoid arthritis or with fluid retention in heart or kidney failure, etc.)
- Job-related factors, including working with the wrist and hand in stressful postures, vibration, exposure to cold through air temperature, materials or tool use, excessive strength requirements,

poor gripping surfaces, excessive direct pressure on the palm of the hand, and repetitive movements of the fingers and wrists
- Avocational activities, such as home-building projects, gardening, sewing, crocheting, knitting, racket sports and throwing, etc.
- In conditions where there may be an underlying neuropathy or nerve sensitivity, such as in diabetes mellitus or kidney failure, the median nerve may be more sensitive to trauma or compression, which in the normal individual would not result in nerve damage
- CTS can be seen with other systemic factors, including endocrine disorders, various drug and environmental toxins, vascular disorders and with inflammatory disease
- Other positive important factors include obesity, older age, and lack of exercise and wrist dimensions (individuals with squarer wrists are more likely to develop CTS than those with rectangular wrists).

DIFFERENTIAL DIAGNOSIS

Not all symptoms in the distribution of the median nerve mean that the patient has a carpal tunnel syndrome. Other problems may mimic or be confused with carpal tunnel syndrome. These include cervical disc disease with radiculopathy, thoracic outlet syndrome, pronator syndrome (median nerve compression at the elbow) and ulnar nerve compromise at the elbow. Other non-neurologic conditions include epicondylitis (tennis elbow), de Quervain's tenosynovitis, myofascial pain syndrome, fibromyalgia and cumulative trauma overuse syndromes.

Symptoms, which would suggest the problem is not carpal tunnel syndrome, include pain that radiates above the shoulder, numbness with coughing or sneezing, or numbness without pain.

DIAGNOSIS, TESTS AND EXAMINATION

Diagnosis relies first on the medical history which often clearly points to a carpal tunnel syndrome. The physical exam may reveal a sensory loss in the median nerve distribution (the thumb, index and middle finger) and there may be weakness and atrophy of the median innervated hand muscles (the thenar eminence or pad of the thumb).

Tinel's sign may be positive where the wrist is gently tapped above the median nerve and a tingling sensation occurs. Phalen's test is positive when the patient complains of numbness and tingling when the wrist is maintained in a flexed position for up to 60 seconds.

X-rays of the wrists will rule out any bony abnormality. Laboratory blood tests should be done to rule out an underlying systemic disease. Electromyogram (EMG) and nerve conduction studies are highly diagnostic and are considered the gold standard in making the diagnosis. A nerve conduction study measures the time it takes for the median nerve impulse to travel across the carpal tunnel. An abnormal latency or delay in conduction of the nerve, in the presence of associated symptoms, confirms the diagnosis.

TREATMENT

Conservative treatment is indicated unless the condition is severely advanced or requires immediate resolution of symptoms. Early treatment is critical because function lost as a result of nerve damage may never be fully restored. Approximately 50% of individuals can be managed successfully without surgery.

Recurrence of symptoms, even after seemingly successful treatment, is not uncommon, particularly if the individual returns to similar activities that brought on the condition in the first place.

Conservative, non-operative treatment options include the following:

- Patient education is important, including splint care, avoiding certain wrist and hand postures and motions procedures to avoid swelling and tendon-gliding exercises.

- A neutral or slight extension position wrist splint will sometimes decrease the symptoms, especially the numbness and pain occurring at night. When the wrist is in this position, the carpal tunnel is at its maximize size so the nerve has as much room as possible.

- Diuretic agents ("water" pills) may decrease excess fluid and swelling thus relieving pressure within the carpal tunnel.

- Oral steroids or non-steroidal anti-inflammatory medications (NSAIDs) may also help control the swelling of the synovium and reduce the symptoms of carpal tunnel syndrome.

- In some studies, high doses of Vitamin B-6 have also shown some efficacy in decreasing the symptoms of carpal tunnel syndrome.

- A steroid injection into the carpal tunnel may be helpful. This medication will decrease the swelling of the synovium and may give temporary relief of symptoms.

- Activity modification and pacing at home and at work may be beneficial and avoid cumulative trauma and re-injury.

- Treating underlying systemic disease such as rheumatoid arthritis will effectively reduce carpal tunnel swelling and thus allow the median nerve adequate space.

- Removal of constricting devices bindings, bandages or jewelry is recommended.

- Rehabilitation exercises & strengthening provided by a physical or occupational therapist with special expertise in hand therapy can be of value.

- Ergonomic evaluation & modification of the work site is equally important as medical treatment alone may not be successful without addressing job related causative factors. Changes can include tool and task redesign, desk height modification, an ergonomic chair, pacing of activities and a

host of other changes to make the job safer, as well as leading to greater worker productivity. The injured worker can be a great resource for needed modifications and should be included with the employer and ergonomist in evaluating the work site.

PROGNOSTICATING SUCCESS WITH CONSERVATIVE TREATMENT

Five factors have been identified which suggest limited success with conservative, non-surgical medical management for CTS. These include age over 50, symptom duration greater than 10 months, constant paresthesias (numbness, tingling and burning), flexor tenosynovitis and a positive Phalen's test at less than 30 seconds. When none of these factors are present, two-thirds of the patients are cured with medical management. When three factors are present, there is less than a 5% likelihood of response to medical management, and with four or five factors, none of the patients are cured.

SURGICAL TREATMENT

In addition to the above criteria, surgery becomes an option when conservative measures have failed and/or the condition is severe (pain, atrophy & weakness) and progressive, or when there is a space-occupying lesion in the carpal canal.

Surgery may be open or endoscopic. At a minimum, it involves surgical release of the transverse carpal ligament. Some surgeons advocate an internal neurolysis, but there is no conclusive proof that this more expensive and extensive procedure is more effective, and there are risks with increased handling of the nerve.

Endoscopic surgical procedures have been developed and advocates claim less morbidity and a faster recovery and return to work. Critics note the cost of this new surgery and claim there is more nerve damage and other operative complications from the small incision.

An estimated 50,000 to 100,000 carpal tunnel operations are done each year. Of the patients treated surgically, approximately 90% have a good result with partial or complete symptom resolution. Another 5% experience a worsening or no change with surgery. In another 5% of cases, symptoms redevelop.

Post-surgical care emphasizes activities that control edema, maintain range of motion and restrict the formation of tendon adhesions. Treatment includes hand elevation and upper extremity exercises. The wrist is splinted in a position of slight extension for two to three weeks. Progressive hand use and exercises follow thereafter.

After surgery, office workers can be back on the job in one week; however, carpenters or athletes may be incapacitated for 4 to 6 weeks.

EXPECTED OUTCOME

The patient should have less pain with improvement in sensory and motor function. There should be a reduction in paresthesias (numbness, tingling, burning). Improved grip strength and dexterity should be demonstrated. Surgery should prevent further deterioration in nerve function.

PREVENTION

Ergonomic evaluation and redesign of the workplace and job tasks are appropriate starting points in preventing carpal tunnel syndrome. Workers should be taught proper gripping techniques and to work with the wrist straight while decreasing repetitive motions. Pacing of work activities is recommended with rest and alternate hand use. The worker should use the minimal pressure, force and speed necessary to get the job done.

Employers should evaluate whether the worker and job make a good "fit." An ergonomic workstation is both worker and job specific. It should be realized though that body habitus (size, height and shape) and physical condition (medical and aerobic) play a role in successful ongoing completion of job tasks. Lastly, workers with ongoing psychosocial problems, whether at home or on the job, may be more prone to injury. Once "injured" they may be more difficult to successfully treat and return to work. If the "true" problem is psychogenic or social, neither conservative medical care nor surgery will "fix" the injured worker and lead to gainful re-employment.

SUMMARY

Carpal tunnel syndrome or CTS is one of the most commonly reported cumulative trauma problems and in some industries appears to be approaching epidemic proportions. Prevention is the most cost-effective approach by recognizing potential problems before they occur, and through ergonomically evaluating and redesigning the job and workstation. CTS presents with discomfort, numbness and tingling in the median nerve distribution of the hand and is more commonly associated with repetitive wrist and finger use. Effective treatment includes identification and elimination of causative physical factors, diagnosis and treatment of underlying medical conditions, hand rehabilitation and possibly surgery.

HAND INJURIES

INTRODUCTION

Our hands are truly complex, active and intricate parts of our bodies, allowing for a variety of functions. They allow us to feel, grasp, perform fine movements and discriminate while displaying exquisite dexterity. The primary function of the entire upper extremity is to place the hand in a position for optimal function. Hand intricacy is attributed to the large portion of the brain dedicated to their control.

In the United States more than 16 million people each year receive emergency care for hand injuries. These common emergencies include fractures, ligamentous injuries and infections. Work related injuries can be devastating leading to pain, suffering, disability and loss of ability to compete on the open labor market.

The first treatment goal of hand injuries is to restore function followed by pain relief and cosmetic appearance. The hand and fingers tolerate injury and immobilization poorly and thus immediate and appropriate medical attention is of paramount importance as delay of treatment can have dire long-term consequences.

This article is about the wrist, hand and fingers and how they can be injured in the workplace.

TERMINOLOGY & ANATOMY

The terms "lateral" or "radial" and "medial" or "ulnar" are used to distinguish the thumb side of the hand from the little-finger side of the hand, respectively. The hand is described as having a palmar surface (the palm) and a dorsal side (the back of the hand). The thenar eminence is the prominence that the thumb muscles form on the lateral side of the hand while the hypothenar eminence is the area formed by the little-finger muscles on the medial side. Rotation of hand from neutral position to palm up position is called supination. The reverse is called pronation.

The hand and wrist are made up of 27 bones (excluding the radius and ulna), 17 articulations, and 19 intrinsic and 20 extrinsic muscles.

There are multiple joints between various bones along with fascia, tendon sheaths and tendons, blood vessels and nerves and numerous hand muscles. In addition to the small muscles of the hand itself, there are tendons from muscles extending on both the flexor and extensor surface of the forearm that control hand movement.

The wrist joint is the junction (articulation) between the forearm radial and ulna bones and the eight carpal bones (made up of two rows). The wrist moves in flexion (80^0), extension (70^0), radial deviation (20^0 thumbward) and ulnar deviation (30^0 towards the small finger). Between the carpal bones and the

phalanges of the fingers are the elongated five metacarpal bones. Distal to the metacarpals are the phalanges, two in number for the thumb and three for each of the other digits. The joints between the various bones of the hand are termed the carpometacarpal joints, metacarpal phalangeal joints and the interphalangeal joints (proximal and distal).

CUMULATIVE TRAUMA DISORDERS

Cumulative trauma disorders or CTDs involve repetitive use over time leading to microtrauma that occurs to such a degree that it overwhelms and exceeds the adaptive and healing capacity of normal tissues. There are many factors that can lead to CTDs including repeated, sustained or forceful exertions; excessive intensity or duration; localized mechanical stresses; prolonged constrained posture; extreme joint positioning; low temperatures; vibration; repetitive impact; tool and workplace design; job stress; and work organization.

Early treatment of CTDs is non-surgical and is based on reducing pain, controlling inflammation, facilitating tissue healing and monitoring activity levels. Initial treatment may include “active rest” which can be accomplished by decreasing the frequency of the work activity, modifying the job or by altering the activity by using a supportive device (i.e., splint). Anti-inflammatory medications, hand therapy and ergonomic work modifications are the mainstay of treatment. If the problem persists, a local steroid and anesthetic injection may prove beneficial. In appropriate situations that do not respond to conservative care, surgery can be an option.

Ganglion cysts are most commonly found as a rubbery swelling on the back of the wrist. They may also occur on the palmar surface of the fingers, wrist and hand. They are non-cancerous, fluid-filled cysts arising from irritated or inflamed ligaments, joint linings, or tendon sheaths. It is not unusual for the cysts to change size quickly or even disappear entirely. Ganglion cysts are not dangerous, and most do not require treatment unless they are cosmetically unsightly, painful, or interfere with hand function. Treatment can include removing the fluid with a small needle and syringe, injecting steroids, or removing the cyst surgically.

DeQuervain's stenosing tenosynovitis is an irritation and swelling of the sheath or tunnel that surrounds the thumb muscle tendons as they pass from the back of the wrist to the thumb (specifically the extensor pollicis brevis and abductor pollicis longus of the thumb). It is most common in women between the ages of 30 and 50. This disorder usually occurs after repetitive use (especially wringing) of the wrist and has also been called "washerwoman’s" sprain.

The major symptom is aching pain at the wrist and thumb, aggravated by motion, along with pain when grasping or pinching. Tenderness is elicited just distal to the radial styloid process over the site of the involved tendon sheaths. Sometimes a lump or thickening can be felt in this area. If the hand is made into a fist with the thumb "tucked in" and bent towards the little finger, the pain gets worse (Finkelstein test). This test includes passive movement at the wrist performed in all ranges of motion, and if forcible deviation of the wrist towards the ulnar side (little finger) provokes severe pain at the site of the affected tendon sheaths, then the test is considered positive.

DeQuervain's stenosing tenosynovitis is seen in workers secondary to repetitive strain or overuse and can also be seen in association with pregnancy or with an inflammatory arthritis such as rheumatoid disease. If treated early, many cases improve with rest in a splint, warm soaks, an injection with a steroid and/or taking anti-inflammatory medications. More severe cases or those that do not respond to other treatment may require surgery. Modification of the activities which caused the symptoms initially may also be required. A careful review of job functions may reveal offending activities.

Carpal Tunnel Syndrome: (see previous article) CTS is the most common peripheral neuropathy and the foremost cumulative trauma disorder (CTD) of the upper extremities. It also is one of the most costly and frequently reported Workers' Compensation claims. CTS results from injury to, or excessive pressure on, the median nerve at the wrist. The individual with a carpal tunnel syndrome typically complains of a feeling of numbness, tingling or burning pain in the distribution of the median nerve, particularly with wrist and hand activities. The symptoms may wake them at night. Symptoms are particularly worsened by prolonged activities with the wrists flexed or with using the fingers and gripping fists, e.g., driving, tool handling, keyboarding, knitting, sewing, etc. The individual may complain of weakness, dropping things or clumsiness. The gold standard for diagnosis is electrodiagnostic testing. Treatment includes rest, splinting, anti-inflammatories, injections and occasionally surgery.

HAND ARTHRITIC DISORDERS

When we talk about hand arthritis, we need to distinguish between degenerative osteoarthritis and inflammatory conditions such as rheumatoid arthritis.

Large joints such as the hips and knees receive continuous stress from walking, running, sporting activity, or injury and are more commonly affected by the wearing of cartilage (degenerative arthritis) than the hand joints. However, the joints of the hand do experience stress in everyday use, and because the hand joints are smaller, these stresses are concentrated over a smaller surface area. The high ratio of stress to surface area can cause the smooth joint cartilage to wear down over the years. As the cartilage degenerates, the underlying bone becomes exposed. When the deteriorated joint moves, bone rubs upon bone causing pain, swelling, limiting motion, and frequently causing a grinding or popping sensation. Furthermore, forms of arthritis that are caused by inflammation (rheumatoid or psoriatic arthritis) of the tissues lining the joint frequently affect the small joints of the hands and wrists to cause joint destruction.

Degenerative Joint Disease or Osteoarthritis of the hands has a genetic predisposition to primarily affect the distal finger joints (DIP), and moderately affect the proximal finger joints (PIP) and the first carpometacarpal joints (the basilar joint of the thumb), with extensive synovitis and cyst formation.

Bony enlargement of the DIP joints (Heberden's nodes) and bony overgrowth of the PIP joints (Bouchard's nodules) are present, often without significant soft tissue swelling. The MCP joints (knuckles) and wrists are usually spared in osteoarthritis. The thumb basal joint (where the thumb meets the wrist) is exposed to very high stresses with normal activities. Forces felt at the tip of the thumb are multiplied twelve times in their effect to the thumb base, thus predisposing this joint to wear.

Degenerative arthritis of the thumb basal joint is very common, especially in women, and frequently requires joint replacement. Attempts at silicone replacement of this joint have not been as successful as hoped for due to implant failure and bone destruction. Thus, the most common joint replacement procedure for the thumb base is done with natural material. The procedure is termed the ligament reconstruction-tendon interposition procedure (LRTI). This procedure uses the patient's own tendon to stabilize the thumb and resurface the joint. LRTI provides stability and pain relief. Long term results have been excellent. This has also been called the tendon roll or "anchovy" procedure because the tendon used is curled to form the new joint cushion.

Unlike with rheumatoid arthritis, in degenerative arthritis blood tests are usually normal, regardless of disease severity. Treatment can include range-of-motion exercises in warm water, intermittent splinting to prevent deformity, use of analgesics or NSAIDs, and occasional intra-articular injections of corticosteroids for acutely symptomatic joints to relieve pain and prevent limited motion. All patients should continue moving their hands as disuse frequently results in stiffness.

Rheumatoid Arthritis is not caused by work, but in a patient with this disease, the workplace activities may cause a lighting up or worsening of the already existing disease. Rheumatoid arthritis frequently causes swelling, pain, and stiffness in the wrists, as well as the small joints in the middle and at the base of the fingers.

Rheumatoid arthritis frequently causes hand deformities. Tissue lumps called rheumatoid nodules can form over the joints of the hand and wrist. The joints of the fingers and thumb can become deformed and contracted by the destruction of the supporting ligaments, so that grasping and pinching movements are not possible.

The diagnosis of hand joint problems typically involves evaluating symptoms, physical examination and the x-ray appearance of the joints. Blood testing is sometimes also helpful in the assessment process. Joint replacement surgery becomes a treatment option when significant joint destruction and/or deformity are present.

Surgical options include cleaning of the abnormal cartilage and bone, including removal of bone spurs, joint fusion and joint replacement surgery. The optimal surgical treatment of arthritis of the hand and wrist varies from patient to patient and is based on many factors. These factors include the patient's age, hand dominance, employment, level of pain, functional goals, and underlying disease.

Surgical cleaning of the joint is usually performed in cases of early "wear and tear" degenerative arthritis and painful bone spurs, or in cases of rheumatoid arthritis with large amounts of inflamed tissue. Removal of bone spurs is especially helpful when the arthritis involves the joints at the ends of the fingers (distal interphalangeal or DIP joints).

Fusion of a joint involves removing the joint and surgically "fusing" the bone ends so that the two bones effectively become one solid bone. This procedure terminates all motion at that joint and thus eliminates the pain. The benefit of fusion is pain relief, but consequences include elimination of motion at the fused joint, which can hinder function. This surgical option is reserved for patients with advanced arthritis.

Joint fusion is usually the best surgical option in patients who are younger and very active. Younger patients may not be candidates for joint replacement because of the increased stress demand on the joints which accompany higher activity levels. This increased stress demand can quickly wear out an artificial joint.

Joint replacement surgery in the hand is an excellent option for treating rheumatoid arthritis of the hand in older, low activity patients. Joint replacement surgery can provide pain relief, increase finger range of motion, and improve hand function.

COMMON HAND AND FINGER DEFORMITIES

Dupuytren's (du-pwe-TRAHNZ) Contracture is a hereditary scar-like thickening of the tissue (fascia) that lies just below the skin of the palm. Dimpling and puckering of the skin over the area eventually occur. This condition may vary from small lumps or bands to very thick bands that may eventually pull the affected finger into the palm and limit straightening of the finger into extension.

Dupuytren's contracture varies in its rate of progression from minor skin puckering for many years to rapid contracture (fixed flexed position) of the fingers. Dupuytren's contracture initially may cause only a minor painless lump in the palm of the hand near the base of the finger(s). Dupuytren's contracture most commonly affects the ring finger but can affect any and all fingers. Dupuytren's contracture can also affect one or both hands.

The precise cause of Dupuytren's contracture is not known. While the condition may not be specifically caused by work, there is a general consensus that job specific activities my light up this condition. It usually affects more men than woman, in the fifty to seventy-year age group. Dupuytren's disease is sometimes inherited, and may be associated with cigarette smoking, vascular disease, epilepsy, diabetes and alcoholism.

The treatment of Dupuytren's contracture depends on its severity.
Most patients with Dupuytren's contracture require reassurance that the problem is treatable with stretching exercises with heat application. When the palm is persistently sore with grasping, ultrasound treatments can be beneficial. Sometimes local inflammation is best relieved with a cortisone injection. Surgery is recommended if there is progressive contracture drawing the fingers into the hand. Small nodules or lumps in the palm do not need treatment until they are very large and interfere with hand function. Even with successful surgical removal, the bands may reappear or occur in other fingers.

Trigger Finger is an irritation of the digital sheath that surrounds the flexor tendons. When the tendon sheath becomes thickened or swollen it pinches the tendon and prevents it from gliding smoothly. In some cases, the tendon catches and then suddenly releases as though a "trigger" were released. The constriction is usually due to inflammation of the sheath (tenosynovitis) that commonly results from overuse or trauma to the underside of the finger. Less commonly it results from a puncture wound that leads to infection of the tendon and its sheath. Trigger finger usually occurs in the third and fourth fingers. Sometimes the swelling can be treated with rest, activity modification, nonsteroidal anti-inflammatory drugs (NSAIDs),

or steroid injections and splinting to provide a gentle stretch. The tendon sheath will then return to its normal, pain-free conditions. More severe cases may require surgery to release the tendon. This can be done as an outpatient procedure.

Mallet Finger is a flexion deformity of the terminal interphalangeal joint in which the fingertip droops and extension is not possible. This deformity may result from an extensor tendon injury or bony avulsion, causing a flexion lag at the distal interphalangeal (DIP) joint. Closed injuries may be treated with splinting that holds the DIP joint in extension and leaves the proximal interphalangeal (PIP) joint free. There is a potential to develop a Swan Neck Deformity if a mallet finger is left untreated.

A Swan-Neck Deformity involves only the fingers and presents with flexion of the knuckle (Metacarpal flexion), hyperextension of the middle (PIP) joint, and flexion of the distal (DIP) joint. It is the result of contracture of the intrinsic muscles. Although a swan-neck deformity is a characteristic finding in rheumatoid arthritis, this deformity is also seen after trauma. The inability to overcome hyperextension of the PIP joint makes finger closure impossible and can cause severe disability. Treatment for a swan-neck deformity is aimed at correcting the underlying cause when possible such as bony misalignment, rebalancing the extensor mechanism or releasing spastic intrinsic muscles.

True swan-neck deformity does not affect the thumb, which is "missing" a phalangeal joint. However, severe hyperextension of the interphalangeal joint of the thumb with flexion of the metacarpophalangeal (MCP) joint is called a "Z" Deformity. If associated with thumb instability, this deformity can interfere greatly with prehension (pinch). This deformity is usually hereditary or due to rheumatoid arthritis and can usually be corrected by interphalangeal joint fusion (arthrodesis).

A Boutonniere (buttonhole) Deformity presents with a fixed flexion of the middle (PIP) finger joint accompanied by extension of MCP and hyperextension of the distal (DIP) joint. This may result from lacerations, dislocation, fracture, osteoarthritis, or rheumatoid arthritis. Classically, the deformity is caused by disruption of the central slip attachment base of the middle phalanx extensor tendon, creating a so-called buttonholing of the proximal phalanx between the lateral bands of the extensor tendon. Surgical reconstruction after fixed deformities develop is often found to be unsatisfactory.

ACUTE HAND INJURIES

Acute hand injuries can lead to severe disability and hand dysfunction. These injuries should be taken seriously and treated aggressively. The normal course after an acute hand injury is swelling, which may rapidly lead to pain and stiffness of the fingers. The swollen, painful hand is often held straight at the knuckles (a position of metacarpophalangeal joint extension) and flexed at the finger joints (interphalangeal joint flexion). When the hand is held in this position for an extended period, the collateral ligaments rapidly contract to produce permanent contractures. Therefore, the injured hand should be splinted with the fingers at 90 degrees to the palm (MCP joints flexed, the interphalangeal joints extended) and the thumb abducted. Hand therapy with mobilization should begin as soon as possible; once hand deformities become relatively established, they cannot be significantly altered by splinting, exercise, or other non-operative treatment.

Gamekeeper's Thumb results from disruption of the ulnar collateral ligament of the thumb resulting from forced radial deviation of the MCP joint of the thumb. Gamekeeper's thumb was originally described as an occupational hazard of gamekeepers in England, who used their hands to break the necks of rabbits. When encountered now, it is usually an aftermath of a fall in which the thumb is jammed onto a hard surface. Conservative management with a splint is often satisfactory, but early surgery should be considered if there is considerable deviation or instability.

A Colles fracture of the distal radius is caused by a forced dorsiflexion of the wrist and typically occurs in individuals over 50 years of age who fall on an outstretched hand. The fracture is typically dorsally displaced and angulated.

The scapholunate ligament may be ruptured by a fall on the outstretched hand. The injury is frequently thought to be a wrist sprain; however, the pain localizes to the region of the scapholunate ligament. Stress testing the ligament by radial and ulnar deviation of the wrist while palpating the scaphoid and lunate confirms the diagnosis. A posteroanterior x-ray of the clenched fist shows abnormal widening of the gap between the scaphoid and lunate. Treatment of an acute injury involves surgical ligament repair. A chronic painful scapholunate ligament rupture requires reconstructive surgery.

Lunate Dislocation is secondary to anterior displacement of the lunate with relatively normal alignment of the rest of the carpus. Perilunate Dislocation is secondary to displacement of the rest of the carpus with relatively normal alignment of the lunate. Significant trauma to the wrist may completely disrupt the two carpal rows, also resulting in lunate dislocation (the lunate is seen lying anterior to the wrist on the lateral x-ray) or in perilunate dislocation. The result is wrist pain and numbness along the distribution of the median nerve (dorsal and palmar thumb and index and middle fingers and lateral half of the ring finger). Perilunate dislocation is often associated with fracture of the scaphoid. Lunate and perilunate dislocations are reduced with open repair and stabilization, especially of the fractured scaphoid.

Metacarpophalangeal Fractures and Joint Dislocations are characterized by pain, limited movement, and digit deformity. Some may be treated with closed reduction, but open reduction and internal fixation or surgical repair is frequently required.

A Hook of the Hamate Fracture may occur while striking the ground with a stick or making a divot playing golf. Clinically, this fracture is suspected by tenderness over the hook of hamate in the palm. It is also indicated by pain on resisted flexion of the little finger, which localizes to the region of the hamate. X-ray or CT scan of the carpal tunnel confirms the diagnosis. Treatment of the nondisplaced fracture requires open reduction and fixation, or excision, of the fragment.

Distal Phalanx Tuft Fractures result from a crush injury to the fingertip (jammed finger). A subungual hematoma indicates a nail bed laceration. Open fractures require irrigation, repair of the nail bed with fine sutures, and replacement of the nail plate to act as a splint. Large displaced fractures are held with a K-wire. Hyperesthesia frequently persists long after the fracture has healed, requiring desensitization techniques.

DEFORMITIES SECONDARY TO NERVE INJURY

In some cases after nerve injuries, recovery may be incomplete and lead to hand deformities.

An "Ape-Hand" Deformity is the result of median nerve palsy leading to wasting of the thenar eminence. The thumb falls back in line with the fingers as a result of the pull of the extensor muscles and the individual is unable to oppose or flex the thumb.

A Bishop's or Benediction Hand Deformity is due to wasting of the ulnar innervated intrinsic hand muscles. The index finger is semi-straight while the middle, ring and little fingers are flexed.

A Drop-Wrist Deformity is due to weakness or paralysis of the radial nerve innervated extensors of the wrist and fingers.

A Claw Fingers Deformity results from intrinsic muscle weakness and the over-action of the extensor muscles on the proximal phalanx of the fingers. The MCP joints are hyperextended and the PIP and DIP joints are flexed. When intrinsic muscle function is completely lost, the term "intrinsic minus" hand is used. The deformity is most often due to a combination of median and ulnar nerve damage.

KIENBÖCK'S DISEASE

Kienböck's Disease (avascular necrosis of the lunate bone) is a relatively unusual condition of unknown cause. It occurs most commonly in the dominant hand of men aged 20 to 45 years old. Patients usually have no recollection of trauma. Symptoms generally start with insidious onset of wrist pain, localized to the region of the lunate carpal bone. It is bilateral in 10% of cases and most often occurs in workers performing heavy manual labor.

Diagnosis may be accomplished at an early stage by MRI or CT and confirmed by the x-ray appearance of a sclerotic lunate, which gradually develops cystic changes and a coronal fracture, and then collapses.

Treatment is aimed at relieving pressure on the lunate by surgery to shorten the radius or lengthen the ulna. Alternative treatments attempt to revascularize the lunate. Salvage procedures are required once the lunate has collapsed and produced fixed rotation of the scaphoid and subsequent degeneration of the carpal joints. Total wrist arthrodesis is performed as a last resort to relieve pain when the condition has progressed to an advanced stage.

HAND INFECTIONS

A Paronychia (pair-oh-NICK-ee-ah) is an inflammation of the nail fold or abscess curving right around a nail bed of a finger. Simple sterile lancing of the abscess is generally enough to cure the infection without oral antibiotics. The nail will be scarred and wrinkled, or perhaps even shed, as a result of the infection. The nail will usually grow back in a couple of months without any permanent deformity. The inflammation can be bacterial or fungal and is not contagious. Chronic paronychias mainly occur in people who have

constantly wet hands, such as dairy farmers, fishermen, bar tenders and housewives. It is more likely to occur, and more difficult to clear up, in those with poor circulation, especially during the winter months.

The most common cause of a bite infection is a tooth-induced injury to the MCP joint as a result of a punch to the mouth. A small puncture wound may hide significant injury to the tendon, capsule, and cartilage of the joint. The oral flora of humans and animals contains a mixture of potential pathogens. All bite injuries are potentially dangerous and can cause significant infection. They should be debrided surgically, with the wounds left open. Systemic antibiotics should be given to prevent septic arthritis with permanent destruction of the MCP joint.

A Felon is an infection of the pulp space of a phalanx. The most common site is the distal pulp, which may be involved centrally, laterally, and apically. The septa between pulp spaces ordinarily limit the spread of infection, resulting in an abscess, which creates pressure and necrosis of adjacent tissues. The underlying bone, joint, or flexor tendons may become infected, and intense throbbing pain and a swollen pulp are present. Treatment involves prompt incision and drainage (adequately dividing the fibrous septa) and administration of appropriate antibiotics (usually a cephalosporin).

A Palm Compartment Abscess may spread between the metacarpals, from the midpalmar space to the dorsum, presenting as an infection on the dorsum of the hand. Palm abscesses present as intense throbbing pain with swelling and severe tenderness on palpation. Incision and drainage with wound cultures, with care to avoid the many important structures, are required in conjunction with appropriate antibiotic therapy.

Infections of the Tendon Sheath present with pain and swelling with tenderness along the length of the tendon sheath and pain with extension of the finger indicate infection of the flexor tendon sheath. Pus may pass from the thumb proximally through the carpal tunnel and communicate with the little finger, forming a horseshoe abscess. Acute calcific tendinitis or systemic rheumatic diseases with tendon sheath involvement can occasionally mimic tendon sheath infections. Tendon sheath infections require surgical drainage (i.e., irrigation of the tendon sheath by inserting a cannula into one end and allowing the irrigating fluid to pass along the tendon sheath to the other end). Antibiotic therapy is also required.

Herpetic Whitlow is a viral infection of the distal finger, often mistaken for a felon. Herpes simplex virus may cause intense, painful cutaneous infection. The appearance of vesicles on the volar or dorsal distal phalanx is diagnostic. Pain preceding the appearance of the vesicles may cause confusion. The condition is self-limited, and surgery is contraindicated. It can be seen in health care workers dealing with oral secretions.

SUMMARY

Our hands are important parts of our bodies allowing for a variety of functions including feeling, grasping, performing fine movements and discriminating along with allowing for exquisite dexterity. The primary function of the entire upper extremity is to place the hand in a position for optimal function. The first treatment goal of hand injuries is to restore function followed by pain relief and cosmetic appearance. The hand and fingers tolerate injury and immobilization poorly and thus immediate and appropriate medical

attention is of paramount importance as delay of treatment can have dire long-term consequences. Proper early treatment provides the injured worker the best opportunity to manage pain and regain function leading to return to home and work activities.

LOW BACK PAIN & INJURIES

INTRODUCTION

Low back problems are ubiquitous and about two-thirds of adults suffer an episode of low back pain at some. In any one year, about 50% of adults have low back symptoms with 15 to 20% of them seeking medical attention. About 2% of the U.S. workforce report back injuries each year. In working persons under the age of 45, low back problems are the most common cause of activity restriction.

The US Department of Labor estimates that 100 million workdays are lost annually because of low back pain. Across all major industries in the United States, the back is reported to be the single most common anatomic location for which workers' compensation claims are made. Claims for low back pain represent 20% to 30% of all compensable work injuries, and about one third of worker compensation costs.

In the United States, there is a wide variety in practice patterns in treating low back pain suggesting some ambiguity regarding optimal care. Some have argued that there has been excessive radiologic imaging, over-medication and too much passive care for this problem.

Acute low back pain is defined as activity intolerance due to lower back or back-related leg symptoms of less than 3 months duration. Continued symptoms thereafter are referred to as chronic low back pain.

Although back pain is among the most common of human conditions, fortunately the majority of causes of back pain are benign. Some 60% of patients recover in a week, and about 90% in 6 weeks. Advanced age, pain radiating into the leg, and psychosocial and occupational factors have all been identified as increasing the risk for developing chronic low back pain.

Strong evidence exists for forceful or heavy lifting, bending and twisting of the trunk, whole body vibration, and heavy manual work as occupational risk factors for low back disorders. Obesity and poor conditioning are risk factors as well. Static work postures, such as remaining seated or standing for long periods, may also contribute to low back pain. Jobs associated with low back pain are most commonly those associated with physical labor or material handling tasks.

In most cases of back pain, the precise origin or diagnosis remains unknown. Indeed, excluding degenerative changes, the majority of patients are given only a symptom diagnosis of low back pain, strain or sprain.

Proper treatment of low back pain involves shifting the focus of care towards increasing activity tolerance and away from treating the pain alone. This approach decreases the chance of low back pain becoming chronic.

CLASSIFICATION & ETIOLOGY

The conditions that may present as back pain generally fall into the categories of rheumatologic, neoplastic (cancerous), vascular, infectious, traumatic, metabolic, neurologic, and mechanical. Among these, the mechanical and the rheumatologic conditions are by far the most common, whereas others, including neoplastic, infectious, and vascular, are less common but the most urgent.

Low back pain may originate from many different spinal structures, including ligaments, facet joints, the vertebral periosteum (dense fibrous membrane covering the surface), the paravertebral musculature and fascia (A sheet or band of fibrous connective tissue), blood vessels, the annulus fibrosus, and spinal nerve roots.

The more common causes are related to musculoligamentous injuries and age-related degenerative changes followed by disk herniation and spinal and foraminal stenosis.

INITIAL ASSESSMENT & DIAGNOSIS

There are basically three questions to be answered when evaluating the patient presenting with low back pain. Is there underlying systemic or serious medical disease, are there psychosocial factors playing a role in the patient's presentation and is there a neurological compromise suggesting the need for more acute or surgical intervention?

The initial assessment by the physician starts with a focused medical history and physical examination directed to rule out the minority of cases with evidence of potentially serious underlying conditions. Absence of these "red flags" rules out the need for special studies during the first month of symptoms when spontaneous recovery is expected in the majority of individuals.

Serious spinal or special conditions ("red flags") can include fracture after a fall or after a vehicular accident. In the older or osteoporotic patient, fractures can occur with even minor trauma. Tumors or infections should be considered particularly if the individual with back pain is under 20 or over 50 years old. Particular concern exists when there is a past history of cancer or in individuals with a history of a disorder with a predilection for infection or hemorrhage. Constitutional symptoms such as fever or unexplained weight loss should alert the physician that an underlying serious condition might exist. Elderly patients with a new onset of back pain should raise concern.

Certain individuals are at higher risk for spinal infection such as those with recent bacterial infections, intravenous drug users, or with immune suppression (from a transplant, steroids or HIV). A possible tumor or infection should be considered in the individual who complains of severe nighttime pain or pain that worsens with lying down.

One emergency situation is when the individual presents with symptoms of a cauda equina syndrome. This occurs with severe compression on the lumbosacral nerve roots in the spinal canal. The individual presents with saddle numbness about the groin area, recent onset bowel and/or bladder dysfunction and a

severe or progressive neurologic deficit with weakness and a sensory change in the lower extremities. This condition is considered a surgical emergency.

The physician must be wary of rheumatological disorders such as ankylosing spondylitis. Non-spinal pathology can include abdominal aortic aneurysm (the patient may present with a pulsatile abdominal mass), kidney stones, low-grade urinary tract infection, pancreatitis, peptic ulcer disease, pelvic inflammatory disease, ectopic pregnancy, prostatitis, large-bowel obstruction and abdominal bleeding or tumors. The patient with hip pathology may present with buttock area pain suggesting referral from the lower back region.

Of all of the simple laboratory tests that may be of use in low back pain, the erythrocyte sedimentation rate (ESR) is the most important, but also the least specific. Not only is it generally elevated in metastatic disease, but it also elevates in infectious disorders of the spine (such as osteomyelitis, discitis, or epidural abscess). It may be the only test to be abnormal in epidural abscess, since this may present without fever or elevated white blood cell count. The ESR may also give a clue as to the inflammatory nature of the complaint in spondyloarthropathies. However an ESR does carry a relatively low sensitivity and specificity regarding individual disorders and should be interpreted cautiously. Back pain with an elevated white blood cell count, usually associated with infection, requires additional consideration as well.

Assuming the low back problem is not caused by non-spinal pathology (pelvic, abdominal, thoracic) and is not a potential serious or life-threatening spinal condition, then consideration is given to the diagnosis of either sciatica (back-related lower limb symptoms suggesting lumbosacral nerve root compromise) or nonspecific back symptoms (suggesting neither nerve root compromise or a serious underlying condition).

The great majority of patients with back pain have symptoms secondary to musculoligamentous, spinal or disc pathology. Degenerative spine disease (osteoarthritis or spondylosis) can be secondary to cumulative trauma but also is a naturally occurring process with normal aging. It is important to understand that degenerative processes of the spine are not always associated with pain. The posterior elements of the spine including the facets are not meant to be weight bearing and can be a source of pain both from faulty posture and degenerative changes. Spinal stenosis can be congenital but also related to degenerative changes combining bony overgrowth and disc protrusions.

The sacroiliac joint is an often-overlooked source of low back pain. The common referral pattern from the sacroiliac joint is to the buttock, although the posterior thigh and the groin may be involved.

Spondylolysis (a "fracture" of the pars interarticularis) leading to a spondylolisthesis (slippage of one vertebra over another) places many back tissues under stress, including the discs, facet joints, and ligaments of the spine. In the young patient, the pain may come directly from the spondylolysis bone defect. This is not likely in the older patient in whom the pain is more probably caused by over-stressed tissues.

When a nerve root is compromised from a herniated disc and/or stenosis, the patient often develops a nerve root compromise leading to a radiculopathy. Facet degenerative changes (arthropathy) can also lead to nerve root compromise. This can cause pain, numbness, weakness and tingling down the limb.

Symptoms are often worsened with sitting and the individual may report that they prefer changing positions frequently. Sitting involves lumbar flexion and especially with twisting, this increases intradiscal pressure significantly. They may also prefer lying on their side in the fetal position. Disc herniation can occur even without significant trauma because an already weakened disc can suddenly give way with something as simple as light lifting, bending over, twisting, bearing down or coughing.

MEDICAL HISTORY AND PHYSICAL EXAMINATION

The medical history serves to elicit information to make a diagnosis but importantly also serves to establish a rapport between the clinician and the patient. It also serves to provide insight into concerns, expectations, and non-physical (psychological and socioeconomic) issues that may alter the patient's response to treatment. It is important for the clinician to determine an estimate of the patient's perception of his or her functional activity tolerance.

The physical examination is guided by the medical history and includes observation of the patient, a regional back exam, neurologic screening plus testing for sciatic tension signs. The exam is mostly subjective since patient response or interpretation is required except for reflex testing and circumferential measurements for girth atrophy.

INITIAL CARE OF ACUTE LOW BACK PAIN

Initial care starts with providing patient education and assurance. Patient comfort is important and can usually be achieved with nonprescription analgesics. When such medications are insufficient, prescribed pharmaceuticals may be added along with physical methods. Activity alteration is appropriate but complete rest is to be avoided.

Patients who present without red flags should be advised that there is no evidence of a severe underlying problem and that a rapid recovery can be expected.

Acetaminophen is the safest effective medication for acute low back problems, but aspirin and various other non-steroidal anti-inflammatory drugs (NSAIDs) are also effective although they can cause gastrointestinal or other problems. These drugs should not be taken concomitantly with regular alcohol intake.

Muscle relaxants are commonly prescribed but seem to be no more effective than NSAIDs and cause drowsiness in up to 30 percent of patients taking them. Muscle relaxants may have a limited role when true muscle spasm exists. Opioids appear no more effective than other analgesics for managing acute low back symptoms. Opioids, if prescribed at all for acute back pain, should be used sparingly and only for a short time. Oral steroids are not recommended for the treatment of acute lower back pain with the exception of patients with nerve root compromise with a radiculopathy. In these patients, epidural steroid injections or nerve root blocks may also be useful to reduce swelling and inflammation. Antidepressants may be useful in patients with chronic back pain but are not helpful in the acute stages.

Physical methods of treatment are numerous and include manipulation, traction, physical modalities (ice, heat, diathermy, and ultrasound), invasive techniques and other various therapies. Physiologically, it makes more sense to use cold in the acute stages of back pain and heat in later stages.

Corsets, braces, and back belts are widely used, but there is no scientific evidence of positive effect in acute back pain. In patients with chronic and recurrent problems, there is conflicting evidence, as is the case with the preventive use of back belts in industry. Continuous use of corsets may result in weakening of the trunk muscles and should be avoided. Traction may be beneficial for herniated discs with radiculopathy but will often prove more painful for mechanical back pain.

Spinal manipulation is safe and effective for patients in the first month of acute low back symptoms without radiculopathy and may even speed recovery. For patients with symptoms lasting greater than one month, manipulation is probably safe, but its efficacy is unproved. If manipulation has not resulted in symptomatic and functional improvement after four to six weeks, it should be stopped, and the patient reevaluated. Obviously, this statement is controversial, but my bias is that manipulation is a passive treatment and that the back pain patient needs to engage in functionally oriented back rehabilitation. Manipulation should not be used in patients with neurologic deficits. Manipulation has no preventive value.

Aerobic exercise programs such as walking, biking, or swimming and trunk muscle exercises and stretching should be started early in patients with back pain once the acute episodes settles down. A stretching and fitness program should be continued indefinitely in all patients but particularly those with recurrent or chronic problems. This program should begin with supervised physical therapy but may progress to an independent gym or home program. Maintaining good physical fitness allows a more rapid recovery and a physically fit spine may be protective against recurrences.

Most patients will not require bed rest as inactivity actually can lead to potential debilitating effects. Two to four days of bed rest are reserved for patients with the most severe limitations (primarily due to leg pain from sciatica).

Prolonged bed rest actually leads to deconditioning and is counterproductive. A gradual return to normal activities is part of the recommended treatment. Patients with acute back problems should avoid undue back irritation from activities and postures that increase stress on the back and thus aggravate symptoms. Repetitive activities involving low back flexion and flexion with rotation should be minimized. Prolonged sitting is usually best avoided. Proper lifting techniques and posture with work simplification should be taught to the patient and encouraged. Ergonomic workplace modifications should be considered.

Debilitation should be avoided by having the patient engage in an incremental, gradually increasing program of aerobic (endurance) conditioning exercises such as walking, stationary biking, swimming and even light jogging. There is no evidence to indicate that back-specific exercise machines are effective for treating low back problems. Specific spinal exercises that focus on flexibility and trunk strengthening are important.

Some of the medications such as opioids together with decreased activity can lead to constipation with back pain. Appropriate diet and stool softeners can be of benefit.

An epidural steroid series may prove beneficial for nerve compression and inflammation from a protruding disc or stenosis. Selective nerve root and facet blocks may be effective in selected patients.

Surgery should be considered only when serious spinal pathology or nerve root dysfunction obviously due to a herniated disc, stenosis or spondylolisthesis is detected. Even in those conditions, non-operative treatment is often successful, and surgery should not be performed without appropriate attempts at such treatment.

Patient who present with neurologic deterioration or cauda equina syndrome remain the exception and should be addressed surgically on an emergent basis. Otherwise, surgical nerve root decompression can be considered if the sciatica is both severe and disabling, the symptoms of sciatica persist without improvement for longer than 4 weeks or with rapid and extreme progression, and there is strong physiologic evidence of dysfunction of a specific nerve root with intervertebral disc herniation confirmed at the corresponding level and side by findings on an imaging study.

Patients with acute low back pain alone, without findings of serious conditions or significant nerve root compression, rarely benefit from surgery. Even individuals with strong clinical findings of nerve root dysfunction due to disc herniation recover activity tolerance with 1 month and evidence indicates that delaying surgery for this period does not worsen outcomes. With or without an operation, more than 80 percent of patients with obvious surgical indications eventually recover. Surgery should be viewed as a way for speeding recovery of patients with obvious surgical indications, but benefits fewer than 40 percent of patients with questionable physiologic findings. Moreover, surgery increases the chance of future procedures with higher complication rates.

Work Status

Keeping the injured worker with acute back symptoms on the job is often a difficult decision for the clinician. Being off work for more than short periods of time can lead to increasing perceptions of being disabled and being unable to return to the job. Alternatively, work activities, particularly prolonged sitting and unassisted lifting can aggravate back symptoms. The best prescription for return to work should take into consideration the job demands balanced by the injured worker's functional ability. The physician, injured worker and employer should work together to arrange for appropriate work modifications that allow for safe early return to work.

SPECIAL STUDIES AND DIAGNOSTIC CONSIDERATIONS

Routine testing (laboratory tests, plain x-rays of the lumbar spine or an EMG) or imaging studies (MRI’s, CAT or bone scans) are not recommended during the first month of activity limitation due to back symptoms except when a red flag raises suspicion of a dangerous low back or non-spinal condition. If the patient’s limitations due to low back symptoms do not improve in 4 weeks, reassessment is recommended regarding further testing.

Selection of a test or special study should be based on clinical judgment considering either the emergence of a red flag or physiologic evidence of tissue insult or neurologic dysfunction. In years past, it was often stated that there was no reason to order an imaging study unless it was needed to define a lesion prior to surgery or when a progressive neurological deficit was present. In reality, patients can have significantly symptomatic disc disease without positive or abnormal neurologic findings. An MRI or CT scan can help the clinician define the cause of the problem and allow for directed rehabilitation treatment and certainly may result in greater prophylactic work restrictions if abnormalities are found.

Reliance on imaging studies to evaluate the source of low back symptoms carries a significant risk of diagnostic confusion as such studies may falsely identify a finding that was present long before the symptoms were present and may be of no clinical consequence. This is known as an incidental finding. Studies have demonstrated that even in healthy, asymptomatic individuals who undergo imaging studies, "abnormalities" are found in over one-third of non-symptomatic people tested.

Electromyography (EMG) including H-reflex testing may be useful to identify subtle focal neurologic dysfunction in patients with leg symptoms lasting longer than 3-4 weeks. Sensory evoked potentials (SEPs) may be added to the assessment if spinal stenosis or spinal cord myelopathy is suspected.

Laboratory tests such as erythrocyte sedimentation rate (ESR or sed rate), complete blood count (CBC), and urinalysis (UA) can be useful to screen for nonspecific medical disease (especially infection and tumor) of the low back. A bone scan can detect active degenerative changes, physiologic reactions to suspected spinal tumors, infection or occult fracture.

MANAGEMENT CONSIDERATIONS AFTER SPECIAL STUDIES

When special studies fail to define the exact cause of symptoms, the patient should not receive the impression that "nothing is wrong" or that the problem could be "in their head." The patient should be assured that a clinical work-up is highly successful in detecting serious conditions but does not reveal the precise cause of most low back symptoms.

Physicians can get themselves and their patients in serious trouble at times with continued passive treatment and further unnecessary tests, procedures and surgery. There is the risk of both a bad outcome from treatment (iatrogenic illness) but also the risk of the patient staying in a dysfunctional and disability-enabled mode from continued passive interventions.

Once it has been determined that the problem is not amenable to surgical intervention, then patients should be encouraged to become progressively more functional and return to normal activities of everyday life within their functional capacity.

FURTHER MANAGEMENT CONSIDERATIONS

Following diagnostic or surgical procedures, the management of most patients becomes focused on improving physical conditioning through an incrementally increasing exercise program. The goal of this program is to build activity tolerance and overcome individual limitations due to back symptoms. At this

point in treatment, symptom control methods are only an adjunct to making prescribed exercises more tolerable. The therapy prescription should start with low-stress aerobic activities followed by specific muscle conditioning. Finally, specific training to perform activities required at home and at work can begin.

Certain questions should be raised when the patient has difficulty regaining the ability to tolerate pre-injury activities. Could the patient have a serious, undetected medical condition? Are the patient's activity goals realistic? Should alternatives be sought such as a job change through vocational rehabilitation? Are there psychosocial, non-physiologic issues clouding the patient's presentation?

Some of these patients will benefit from psychological counseling to help deal with continued pain, dysfunction and the inability to return to former activity levels. Biofeedback, hypnosis and therapies targeting development of coping skills are excellent adjuncts to psychological counseling.

Some of these individuals will unfortunately progress to a chronic pain state with increasing pain complaints and dysfunction. They are difficult to evaluate and treat. They should be referred for a multidisciplinary evaluation and possibly treatment.

SUMMARY

Low back problems affect virtually everyone at some time during their life. In persons under the age of 45, low back problems are the most common cause of disability. About 90% of individuals with an acute low back problem will spontaneously recover activity tolerance within one month.

Initial care starts with providing patient education and assurance followed by the judicious use of medications and physical rehabilitation. Selection of a test or special study should be based on clinical judgment considering either the emergence of a red flag or physiologic evidence of tissue insult or neurologic dysfunction. Surgical intervention is reserved for individuals with a progressive neurological deficit or when orthopedic and neurological abnormalities in a psychologically stable individual suggest surgical remediation will result in a positive outcome.

The body has an amazing ability to heal itself with proper nutrition, exercise and a positive mental attitude. Our medicines, tests, procedures and surgeries can prove very beneficial, but they can also inadvertently inflict harm. Knowing the patient well and taking into consideration all the information in a comprehensive fashion, helps to avoid potential pitfalls and the likelihood of making a challenging problem worse.

LUMBAR SPINAL STENOSIS

INTRODUCTION & DEFINITION

Lumbar spinal stenosis is defined as a narrowing of the lower spinal canal. The radiologic diagnosis is made by an x-ray, MRI and/or CT scan. Spinal stenosis is actually very common, particularly in later adulthood and is most often asymptomatic. A finding of spinal stenosis on a radiologic study does not necessarily equate to the presence of disease or a physiologic abnormality.

Lumbar spinal stenosis becomes symptomatic when the narrowing leads to compression and compromise of the lumbar and sacral nerve roots (the cauda equina) prior to their individually exiting the spine. In addition to constriction of the nerve roots, there can also be compression of the small blood vessels (arterioles and capillaries) in this region along with congestion of the venules (veins). Additionally, the flow of spinal fluid to the nerves is restricted. Research studies have shown that abnormal blood flow and congestion are common in symptomatic individuals while those with spinal stenosis by radiologic studies without symptoms have less congestion.

SYMPTOMS

Symptoms can vary due to many factors including the degree of inflammation, spine position (symptoms often increase with back extension or bending backwards), progression of the underlying degenerative process, and from new processes such as a disc herniation. Symptoms appear worse when there has been a more rapid progression.

Spinal stenosis symptoms tend to wax and wane over time with the intensity varying from periods with relatively few complaints to periods with increasing pain and dysfunction. The patient may have alternating leg and back pain with changing physical examination findings, depending on the severity of symptoms.

The changes seen with lumbar spinal stenosis are basically the same as those seen with degenerative spine and disc disease that occurs naturally with aging. In certain individuals, though, when a certain threshold is reached and the space available is too narrow or there is new inflammation, symptoms develop. Given the large number of structures that can be involved, it is not unusual to see varying complaints in degree and over time. Pain may involve both sides or vary from side to side. For many individuals, symptoms will worsen, but more than half will stabilize or even show improvement with time. As the individual ages, radiographic studies will show a progression of changes over time. Approximately one-quarter of patients have one episode that resolves in less than six months and never have significant symptoms again.

Lumbar spinal stenosis typically occurs after the age of 60, but can occur in younger individuals, particularly those who have had prior spine trauma or problems. For example, an individual may undergo spine surgery for a disc herniation in their thirties with a good result, but years later have problems with

spinal stenosis as other factors such as inflammation or normal progressive degenerative changes add to an already compromised, but previously asymptomatic spine.

DIAGNOSTIC ASSESSMENT

The history and physical examination are most important. The individual often describes back and leg pain that increases with activity and position (leaning backward or walking uphill) with symptoms relieved somewhat by position changes (bending forward or alternating sides). The patient often describes leg heaviness, fatigue, pain, or numbness and tingling. Typically leg symptoms outweigh back complaints. In more severe cases, there may be urinary urgency or incontinence.

The physical examination is rarely dramatic and may be normal or produce subtle findings in most cases. The physician looks for muscle weakness, reflex asymmetry, sensory changes, or positive straight leg raising. If the exam is normal at rest, it should be repeated after exercise as this may bring out subtle changes. It can be particularly useful to have the patient, while standing, lean over the exam table backward in extension, as this often reproduces symptoms. Weakness and positive straight leg raising are uncommon and if found should suggest additional pathology such as a disc herniation or a synovial cyst of the facet.

The differential diagnosis includes vascular claudication (not enough blood gets to the limbs due to constriction of the arterial vessels) but this tends to be more activity-related than position dependent. One must also rule out myofascial problems, infection, disc herniations, and tumors.

Radiologic studies include plain x-rays that may show degenerative changes but are not particularly helpful except in advanced cases. CT scanning and Magnetic Resonance Imaging (MRI) are more definitive for diagnostic purposes.

TREATMENT

Treatment approaches should begin and progress in a stepwise fashion starting with physical rehabilitation and medications, progressing through chronic pain management in selected individuals and ending possibly with surgical intervention.

Certain patients with high grade focal stenosis demonstrated by myelographic block or MRI should probably be surgically decompressed early on because the operation is small (micro) while the disease is precarious and usually resistant to prolonged conservative care. Prolonged conservative care is more important in patients with spondylolisthesis (slippage of vertebrae over another), scoliosis, and multifocal disease because the operation is much more involved and hazardous.

The majority of individuals can be treated successfully with stretching, strengthening and aerobic conditioning along with proper pacing of activities. Postural training is extremely important as symptoms are worsened with a lordotic (back extended) posture and with poor body mechanics. Weight loss in overweight and deconditioned individuals is important. Some patients do better with an aquatics rehabilitation program.

The appropriate use of analgesic and anti-inflammatory medications combined with judicious use of muscle relaxants and anti-depressants can be extremely helpful. In selected patients, pain management techniques including relaxation techniques, biofeedback, and meditation may be helpful. The treatment of secondary problems such as myofascial pain or fibromyalgia may be beneficial. When there is an inflammatory component, an occasional epidural or nerve root steroid injection may be helpful.

The use of opioid medication should be avoided but may be useful briefly for flare-ups. Some individuals find a TENS unit can be helpful, and for some acupuncture can be of benefit. Some individuals with lumbar spinal stenosis seek manipulative treatment, but there should be concerned about possible negative effects in an individual with an already compromised spine. There is no really good scientific data about manipulation specifically for spinal stenosis, but if used it probably should be limited to early, milder cases.

Operative intervention should be based on the patient's quality of life and level of function. In most cases, surgery should not be considered unless a non-operative rehabilitation effort has failed or there is evidence for a high-grade focal stenosis. Surgery tends to be more effective in lumbar spinal stenosis for leg pain rather than for back symptoms. In younger patients (below the age of sixty), the individual may outlive any beneficial results from surgery and even develop new or recurrent symptoms. Prolonged deconditioning can be devastating and should be avoided. The individual who is limited to household only ambulation for greater than six weeks or is bed ridden for even shorter periods should be considered for surgery. This assumes a lack of intervening significant psychosocial factors.

The surgical risk for lumbar spinal stenosis is low with a single level micro decompression alone whereas the risk is considerable with a multi-level fusion with instrumentation. There are no single functional criteria that by itself leads to a decision to go forward with surgery. Each individual is very unique regarding the level of pain, dysfunction, and disability they will present even with similar radiographic and physical findings.

We should be reminded by teachings of Dr. William Osler who reminded us that, "It is more important to know about the patient who has the disease than about the disease the patient has." It is a terrible mistake to focus on subjective complaints or radiologic findings alone, when other psychosocial variables may have more to do with an individual's presentation and complaints. The patient with a history of psychosocial dysfunction and who is unhappy about his or her job or marriage may do poorly with surgery or even non-operative care, unless these issues are realized and addressed. The seemingly symptomatic elderly patient may have radiologic evidence of lumbar spinal stenosis but are the symptoms of back pain truly related to the radiologic findings or are the complaints a function of depression, loneliness and the need for attention.

A few years ago, a neighbor asked me to see his elderly father. This gentleman had terrible degenerative disease and spinal stenosis and had already been scheduled for back surgery. His symptoms were managed, though, with a short course of medication, physical therapy, psychological care, and involvement in a senior activity center. He did have rather severe degenerative lumbar spine stenosis, but his problem was a combination of poor posture, myofascial pain, depression and loneliness.

Surgery involves decompression directed at the cauda equina and nerve roots. This type of surgery can lead to spinal instability and in certain cases a spinal fusion with instrumentation may be needed. A surgical fusion is a more complicated and costly procedure with greater risks and higher morbidity. Over time instability may develop above and below the fusion. There is no guarantee of a successful fusion and post-operative complications may cause increased complaints and disability.

On the other hand, severe restriction of mobility in the patient can have serious constitutional effects. If the patient remains unable to ambulate for weeks or longer despite liberal analgesia, root or epidural blocks, and encouragement, these serious debilitating effects must be weighed against the surgical risks. A surgical evaluation will usually be able to determine pre-operatively the number of levels needing decompression, whether a fusion may be anticipated and over how many levels. The surgeon may also feel there is no operable option. Deformity, instability and osteoporosis may be such that there is no reasonable "fixing" of the problem with surgery.

Studies have suggested a fairly good success rate (75% or greater) in carefully selected cases, particularly with leg pain. The long-term results deteriorate over time. Patients may develop a similar problem at another level or recurrence at a non-fused level. The "half-life" of good results seems to be about 8-10 years.

SUMMARY

Lumbar spinal stenosis is a narrowing in the lumbar bony canal and/or foramen which becomes increasingly more prevalent as we grow older. In the majority of individuals, it is asymptomatic, but when there is compression and compromise of the nerve roots, symptoms may develop. Complaints typically include back and leg pain along with fatigue, numbness and tingling. Treatment includes both medications and physical rehabilitation techniques, but surgery may be indicated in selected cases.

HIP PAIN & INJURIES

INTRODUCTION

Hip pain is a common complaint in both a primary medical and orthopedic practice. Determining the source of hip pain is often challenging as hip problems may occur as a result of direct injury, cumulative trauma or secondary to lighting up an underlying degenerative process. Hip dysfunction presents both with localized and referred pain complaints and commonly results in abnormal gait patterns.

HIP STABILITY AND ANATOMY

The hip is a large, deep "ball-and-socket" synovial joint. This joint is composed of two parts: the hip socket referred to as the acetabulum; and the "ball" which is the head of the femur (thighbone). The deep acetabulum receives the head of the femur to form the hip joint. As compared to the shoulder, the hip joint has gained stability at the expense of some loss of motion. The strength of the hip joint results largely from the shape of the articular surfaces and in the ligaments, rather than from the associated musculature. In general, the major ligaments have a common action in limiting internal rotation of the femur.

Muscles attach near the neck of the femur and exert forces that translate into leg movement. There is a cushioning bursa, the trochanteric bursa that protects the tissues lateral to the hip. When the bursa becomes inflamed, it is termed a trochanteric bursitis.

The nerve supply to the hip comes from the femoral, sciatic and obturator nerves. The sciatic nerve is made of a medial peroneal nerve and a lateral tibial nerve division. The peroneal division is more susceptible to injury with hip dislocations or during total hip replacement. When damaged, the patient may present with a foot drop (dorsi-flexor weakness). The blood supply to the femoral head is derived primarily from two arteries that encircle it. These vessels run upward along the neck of the femur and make them susceptible to disruption when the neck of the femur is fractured.

BIOMECHANICS & GAIT

The hip is subject to considerable stress over a lifetime from movement, weight-bearing and repetitive impact. Any derangement in the smooth gliding of the joint surfaces can produce abnormal stress that can initiate a progressive deterioration of cartilage and subsequently the joint. The hip moves in a combination of three basic planes, flexion and extension, abduction and adduction (side-to-side) and external and internal rotation.

The muscles that hold us erect and allow walking and stair climbing exert considerable forces across the hip in the range of 3 to 6 times body weight. Thus a 25-pound weight gain may seem like 150 additional pounds to the hip. It is easy to recognize that degenerative changes (osteoarthritis) of the hip may be symptomatic much earlier and to a greater extent in obese patients.

The lower extremities are dedicated to the important tasks of weight bearing and ambulation. A gait abnormality is usually a consequence of pain, weakness, and/or a difference in leg length. The hips play an important role in normal walking and running and thus gait is usually significantly affected by hip pathology.

There are two phases in the normal gait cycle: stance phase when the foot is on the ground; and swing phase, when it is moving forward. Most hip problems become apparent in stance phase. The individual with hip pathology will typically shorten the stance phase and will lean over the affected hip. The gait will be characterized as antalgic (painful). During the swing phase, the pelvis may not rotate normally around the painful and stiff hip joint.

Reducing the overall load or increasing the surface area over which the load acts can decrease stress on the hip. Canes, crutches, and walkers decrease overall load and are therefore a common part of the non-surgical treatment for arthritis of the hip. Properly used, a cane will significantly improve walking distance and comfort.

HISTORY AND PHYSICAL EXAMINATION

The patient may present with specific complaints of hip pain, but often the complaints are more diffuse in nature involving the buttocks, groin and knee. Complaints commonly involve anterior groin pain, lateral thigh pain, posterior gluteal (buttock) pain and referred pain to the knee. Pain may also be referred from other areas such as the low back or may be due to non-muscular causes.

VARIOUS CAUSES OF HIP PAIN ARE AS FOLLOWS:

Musculoskeletal

- Avascular Necrosis (Osteonecrosis)
- Inflammatory Arthritis
- Osteoarthritis
- Bursitis
- Myositis
- Stress Fractures
- Traumatic Fracture
- Pathologic Fracture
- Post-Surgical
- Metabolic Conditions

Other Orthopedic Conditions

- Spinal Stenosis
- Degenerative Disk Disease
- Facet Joint Arthropathy
- Knee Osteoarthritis

Non-Muscular Conditions

- Hernias
- Aneurysms
- Atherosclerosis
- Meralgia Paresthetica
- Thrombophlebitis
- Cellulitis
- Abdominal Disease
- Retroperitoneal Pathology

Since the hip begins as a stable joint, problems are usually related to either specific or cumulative trauma, either of which may progress to osteoarthritis. A thorough history is essential in revealing evidence for a specific trauma versus progressive and cumulative problems over time, especially if industrial causation is an issue. The clinician should describe the mechanism of injury if known, and the nature, location, duration and intensity of symptoms. Additional clues such as the time of day when it occurs or is worse, how the symptoms respond to change in position and activity and whether there are associated swelling or other constitutional symptoms can narrow the differential diagnosis. Other issues that should be addressed involve body type, posture and body mechanics, occupation and leisure activities

The location of the symptoms may help the examiner better understand the potential source of the complaints. Pain over the anterior and lateral aspects of the thigh may be referred form the L2 or L3 nerve roots. Pain into the knee may be referred from L4 or L5 nerve roots or from the hip joint. Pain over the lateral hip area may be secondary to a trochanteric bursitis and pain posteriorly can result from a piriformis syndrome or ischial bursitis.

Observation is the first step of the examination. The patient who is leaning backward while sitting may have decreased hip flexion. For the individual leaning to one side, there may be an ischial bursitis, sacroiliac dysfunction or referred pain from the low back. The examiner may gather further information by observing the patient changing positions, as there may be changes in facial expression or other evidence of pain behavior. When the patient goes from a sitting to a standing position, the observer watches for difficulty in moving or weight bearing on the limb. With walking the individual may demonstrate a short stance phase or other gait deviations due to hip pain.

Closer inspection and palpation may demonstrate areas of localized atrophy or swelling, tenderness, discoloration or wounds, and/or temperature or skin changes. One should look for changes in symmetry between the two sides or any leg length discrepancies. The examiner should note loss of motion, weakness, and sensory or reflex changes.

OSTEOARTHRITIS

Osteoarthritis (OA) or degenerative joint disease of the hip occurs most commonly in patients over 50 years old. In the early stages progressive hip joint stiffness develops and the patient complains of pain with movement. Osteoarthritis may start as a primary degenerative process or it may develop secondary

to a variety of childhood hip disorders. It may also be the result of some other inciting factor during adult life such as trauma, infection, or a metabolic or endocrine disorder. Recognized predisposing factors include congenital abnormalities (hip dysplasia), slipped capital femoral epiphysis, unrecognized avascular necrosis (from steroids, etc.) and age, but interestingly not obesity.

The pain is often unilateral. Early complaints can include morning stiffness and pain with weight bearing. It is usually described as dull and aching and it may subside initially with rest and the application of heat. Hip pain may be referred to the groin and/or knee. In fact, some patients present with groin or knee pain as their primary complaint.

As the OA progresses, the patient will complain of pain at rest. They will begin to notice night pain and soon there will be a noticeable limp followed by constant pain regardless of the activity. Internal rotation is usually the first motion to be restricted.

From a physical examination standpoint, the patient may have pain on internal rotation and extension as an early sign of osteoarthritis. Pain with motion of the leg may differentiate hip pain from other pelvic pain. Radiologic studies will typically reveal degenerative changes. Initial conservative treatment includes non-steroidal medications, localized heat, and hip strengthening exercises. As symptoms progress one should consider the use of a cane, which is properly used in the hand opposite the most affected hip.

In advanced cases, surgery may be indicated. Surgical options include osteotomy, fusion, or hip replacement. Osteotomy is the surgical realignment of one or both joint surfaces. A correctable deformity if treated may delay further deterioration of the hip as well as other affected joints such as the knee and ankle. Hip osteotomies are not common in the United States.

A hip fusion involves fusing the femur to the pelvis, but this results in loss of motion. The spine provides the flexibility required for walking and sitting. Fusion is commonly offered to highly symptomatic young patients where the goal is to delay a hip replacement until the individual is older. There are problems though with a hip fusion including increased risk of femur fracture and accelerated degenerative disease of the knee and spine.

Total hip replacement is a surgical procedure for replacing the hip joint. During the procedure, the head of the femur (ball) and the acetabulum (socket) of the hip joint are removed and replaced with smooth artificial surfaces. These artificial pieces are implanted into healthy portions of the pelvis and femur and affixed with bone cement (there are cementless varieties too). In some cases, only one of the two components (socket or stem) may be fixed with cement and the other is cementless. This is called a "Hybrid" hip prosthesis.

A total hip replacement (THR) is usually performed for a severe arthritic condition. The operation is sometimes performed for other problems such as hip fractures (especially in the elderly) or for aseptic necrosis. Most patients who have artificial hips are over 55 years of age, but the operation is occasionally performed on younger adults. Circumstances vary, but generally patients are considered for total hip replacements if:

- Pain is severe enough to restrict work, recreational activities, and/or activities of daily living.
- Pain cannot be adequately managed conservatively by medications, the use of a cane or other assistive devices, and/or activity modification.
- Significant stiffness of the hip.
- X-rays demonstrate advanced arthritis, avascular necrosis or other significant problems.

A THR will provide pain relief in 90 to 95 percent of patients. It allows most patients to carry out normal activities of daily living. The artificial hip may allow return to active sports or heavy labor. Most patients with stiff hips before surgery will regain near-normal motion, and nearly all have improved motion. The most common complications are not directly related to the hip and do not usually affect the result of the operation. These include blood clots in the leg, urinary infections or difficulty urinating and blood clots in the lung. Complications that affect the hip are less common, but in these cases, the operation may not be as successful. This can include a leg length difference, stiffness, dislocation of hip (ball pops out of socket) and joint infection. A few of the complications, such as infection or dislocation, may require re-operation. Infected artificial hips sometimes have to be removed, leaving a short (by one to three inches), somewhat weak leg, but one that is usually reasonably comfortable and one on which you can walk with the aid of a cane or crutches.

Success rates for hip replacement surgery range from 90 to 95 percent up to 10 years. The major long-term problem is loosening of the prosthesis. This occurs either because the cement crumbles (as old mortar in a brick building) or because the bone melts away (resorbs) from the cement. By 10 years, 25 percent of all artificial hips will look loose on x-ray. Somewhat less than half of these (about 5% to 10% of all artificial hips) will be painful and require revision. Loosening is in part related to how heavy and how active the patient is. It is for this reason that orthopedists do not typically operate on very obese patients or young, active patients. Loose, painful artificial hips can usually, but not always, be replaced. The results of a second operation are not as good as the first, and the risks of complications are higher.

INFLAMMATORY ARTHRITIS

Rheumatoid arthritis (RA) is a chronic, inflammatory disease with bilateral and often symmetric involvement of joints. While RA occurs at all ages, it is more common with advancing age with the incidence peaking between the fourth to sixth decades. Women are twice as often affected than men. Rheumatoid arthritis of the hip is usually a late manifestation of more widespread disease. An x-ray will show joint space narrowing and osteoporosis about the hip area. Patients with RA will often have various other findings not involving the joints including rheumatoid nodules, neuropathy, pericarditis (inflammation about the heart), splenomegaly (enlarged spleen) and lymphadenopathy (enlarged lymph nodes). Other types of inflammatory arthritis include ankylosing spondylitis and psoriatic arthritis. Occasionally, gout will involve the hip. Joint infection is another form of inflammatory arthritis and if not treated early with antibiotics, may result in severe hip damage.

TROCHANTERIC BURSITIS

The bones, ligaments, tendons and muscles are kept from rubbing each other by four small fluid filled sacs call bursae. These bursae are susceptible to an inflammatory response from direct or indirect trauma.

The hip or trochanteric bursa is located between the hip and the overlying muscles and skin. A trochanteric bursitis usually affects the lateral aspect of thigh. It is often associated with osteoarthritis and trauma. Bursitis is usually treated with NSAIDs, ice, physical therapy modalities, and possible activity modification. Local corticosteroid injections may help cure or alleviate bursitis in the more refractory cases.

AVASCULAR NECROSIS

Avascular necrosis, aseptic necrosis or osteonecrosis of the femoral head is presumed secondary to a local vascular insufficiency. In the initial stages only the femoral head is involved and the acetabular cartilage remains normal. The degree of femoral head collapse is variable. Over time, movements of the irregular joint surfaces cause degenerative changes in the acetabular cartilage as well. Alcohol abuse and corticosteroid treatments are the most common identifiable factors associated with avascular necrosis of the hip. Metabolic and hematological (blood) abnormalities may be involved as well. Patients on renal dialysis and those with systemic lupus erythematosus are at an increased risk. Traumatic avascular necrosis can occur after hip fracture or dislocation (more commonly in older patients). There may be little pain early in the disease and only subtle physical findings may be present such as loss of hip internal rotation. Acute pain often follows the collapse of the femoral head followed by pain and symptoms of degenerative arthritis. Plain x-rays may appear normal in early avascular necrosis, but as the disease progresses, the femoral head appears sclerotic and loses its spherical shape. A bone scan late in the disease is almost always positive because of new bone formation. An MRI is a sensitive indicator of early avascular necrosis. Short of a total hip replacement, a core decompression surgery is effective in some cases. The theory behind this approach is that drilling one or more channels into the bone will relieve congestion and thus improve circulation to the area.

HIP JOINT EFFUSIONS

An effusion refers to fluid in the hip joint. It is commonly seen secondary to intracapsular fractures, toxic synovitis of the hip, septic arthritis, osteomyelitis and Legg Perthes disease in children. The person suffering from a painful hip joint effusion is most comfortable when seated with the painful thigh lightly abducted and externally rotated at the hip joint. This orientation reduces tension in synovial membrane to a minimum because it maximizes encapsulation of the femoral head by the acetabular cavity and labrum. The most sensitive indicator of a hip joint effusion in an x-ray is the presence of an inferolateral displacement of femoral head from the acetabular cavity.

HETEROTOPIC OSSIFICATION

Heterotopic ossification may present with signs of localized inflammation or pain and with elevated skin temperature. It tends to occur after spinal cord injury, head injury, burns and bruising injuries. X-rays will show soft tissue bone formation (ossification), but this usually does not appear in the first month. A bone scan may reveal increased isotopic intake by the second week. Blood tests will show an elevated alkaline phosphatase which correlates with the bone scan abnormality. Non-operative treatment includes non-steroidals such as Indomethacin. Etidronate (a medication) is used to delay mineralization and is effective in heterotopic ossification. Radiation therapy is also an option. Operative resection is delayed until the

heterotopic bone has matured (about 12 months). The main disadvantage of early resection is risk of recurrence. Bone scans and alkaline phosphatase levels may not be helpful in predicting maturity of the ossification.

HIP FRACTURES AND DISLOCATIONS

High-impact injuries to the hip region, most often seen in automobile accidents and falls, can cause severe hip and pelvic fractures. Hip fractures in the elderly are most often secondary to falls with associated osteoporosis. Pathological fractures may be seen with metastatic disease. Fracture types usually include intertrochanteric (fracture occurs between greater and lesser trochanters) and subtrochanteric (between lesser trochanter and 5 cm distal) and femoral neck fractures.

Hip dislocations are typically caused by high-energy trauma, usually from motor-vehicle accidents. They occur most frequently in young patients. There are three types: anterior, posterior and central acetabular fracture dislocations. Clinical findings in patients with posterior hip dislocations include internal rotation of the hip, whereas patients with anterior dislocations will have external rotation of the hip. If this is not the case, the examiner should be suspicious of a femoral neck or shaft fracture or a same-sided knee dislocation. The diagnosis is confirmed by an x-ray. With posterior dislocations the head will migrate superiorly while with anterior dislocations the head will migrate inferiorly or medially. Complications after a dislocation can include heterotopic ossification, avascular necrosis and sciatic nerve injury.

DIFFERENTIAL DIAGNOSIS

There are a number of structures near the hip region that can present with pain and need to be considered in the differential diagnosis of hip problems.

Ischial bursitis is associated with activities that involve prolonged sitting. It can be seen with bicycle riding. It presents with sharp, abrupt pain upon sitting. Patients report that it hurts to stand on tiptoes. The ischial tuberosity may be painful on rectal exam. The differential diagnosis includes lumbar spinal pain, thrombophlebitis and sacroiliitis. The treatment includes rest, NSAIDs and injections (difficult into this bursa).

Iliopectineal bursitis involves inflammation of psoas bursa. Pain may present in the pelvis, groin and thigh. It may cause a hernia-like bulge. The patient will have pain on extension of the hip or with attempted flexion against resistance. There is not much pain on rotation. It may be associated with rheumatoid arthritis.

Hip pain may be referred to the groin area, but symptoms may be primary from this area as well. The patient may have a groin ligament strain or inguinal or femoral hernia. Many of the muscles about the hip area and buttocks may develop myofascial pain and associated trigger points and may mimic hip pain and also sciatica.

SUMMARY

Injured workers can develop work disabling hip problems as a result of direct injury, related to cumulative trauma or secondary to lighting up an underlying degenerative process. Hip dysfunction presents both with localized and referred pain complaints and commonly results in an abnormal gait. A careful history, comprehensive physical examination and appropriate diagnostic studies can narrow the differential diagnosis, lead to more directed treatment and improved functional outcomes. Treatment approaches include anti-inflammatories, the early use of modalities followed by stretching and strengthening exercises. Assistive devices such as canes may be useful in selective cases. Surgery may be warranted with fractures and with severe degenerative disease.

KNEE PAIN & INJURIES

INTRODUCTION

The knee is commonly injured in all age groups. Knee injuries typically occur with sports and exercise, but also occur as a result of work-related activities. Knee injuries can frequently lead to significant limitations and lost time from work. Prompt diagnosis and treatment of these injuries is of paramount importance in order to prevent and/or reduce long term disability.

The knee is the largest joint in the body. It basically functions as a hinge joint, similar to a door hinge and it provides a fairly wide range of motion, especially in flexion. By permitting bending of the lower extremity, the knee is very important for performing many daily activities and for efficient walking.

The knee is susceptible to trauma because it is not protected by layers of muscle or fat and is located at the end of two long lever arms; the proximal end of the tibia (the shin bone) and the distal end of the femur (thigh bone). The patient often gives a history of a twisting episode followed by swelling, clicking, popping, grinding, giving way (buckling) or locking.

There are many areas around the knee that can be injured, but the most commonly affected structures are the cartilage (menisci) and ligaments. The ligaments are found on each side of the joint and there are two other ligaments that cross inside the knee, known as cruciates (anterior and posterior).

Although many problems about the knee can be treated conservatively with early mobilization, ice, physical therapy, and NSAIDs (non-steroidal anti-inflammatory drugs), some conditions, such as meniscus tears usually will require arthroscopic surgery.

ANATOMY

A better understanding of knee problems starts with the anatomy and how the different parts work together to maintain normal function. The medial side of the knee is on the inside of the leg and faces the other knee while the lateral side is on the outside. The important parts of the knee include bones, ligaments, tendons, and cartilage.

The knee is composed of three bones (femur, tibia and patella). The femur is the large bone of the thigh. The tibia is the large bone of the lower leg (the fibula is the small bone just lateral to the tibia and is not part of the knee mechanism). The femur and the tibia meet to form the knee and the joint is protected in front by the moveable patella (kneecap). The patella is aligned to improve the power of the large thigh muscle (quadriceps) on the front of the leg. The underside of the patella is covered with cartilage, which is the smooth covering of joint surfaces. This slippery surface helps the patella glide in a special groove on the front of the femur. By displacing the quadriceps anteriorly, it increases the muscle's mechanical advantage by 25%. The patella has the thickest cartilage of any bone in the body. It experiences significant loads during activities such as stair climbing, running and jumping.

Since the hips are wider apart than the knees, the femurs point inward towards the tibia thus creating a valgus angle ("knock-kneed" if extreme). Since the quadriceps lies along the femur, when it contracts, it pulls the patella laterally. This displacement laterally is counteracted by the inside or medial fibers of the quadriceps (vastus medialis). Any imbalance of these forces can lead to a pre-arthritic condition, chondromalacia patellae (*chonro* means "cartilage" and *malacia* means "softening"). The knee is composed of two articulations (where two bones come together), the tibiofemoral (tibia & femur) and the patellofemoral (patella & femur). The tibiofemoral joint is formed by two large bulbous areas from the femur (femoral condyles) which rest on the relatively flat tibial plateau. This joint is inherently unstable with potential for movement in four directions (Flexion-extension, side-to-side, external-internal rotation and anterior-posterior). This movement can vary from individual to individual and is stabilized by muscles and ligaments.

The joint is cushioned by cartilage that covers the ends of the tibia and femur as well as the underside of the patella. This white, shiny material has a rubbery consistency. The function of cartilage is to absorb shock and provide an extremely smooth surface to facilitate motion. There is cartilage essentially everywhere the two bony surfaces come together (articulate). Two structures called menisci sit between the femur and the tibia. The menisci (lateral and medial) are pads of cartilage that further cushion the joint, acting as shock absorbers between the bones. They also help with knee stability.

The knee has very little bony stability and behaves more like a round ball (femur) on a flat surface (tibia). Because there is no inherent bony stability, the ligaments of the knee are very important. The ligaments, all taken together, are probably the most important structures controlling stability of the knee. There are two pairs of major ligaments (medial and lateral collateral ligaments, anterior and posterior cruciate ligaments) and many minor or capsular ligaments that stabilize the knee joint.

The medial and lateral collateral ligaments lie on either side of the knee and prevent excessive side-to-side displacement of the tibia relative to the femur. The anterior and posterior cruciate ligaments lie within the knee (intra-articular) in the midline. The posterior cruciate is the larger of the two and acts like a gate hinge and also prevents posterior displacement of the tibia on the femur. The anterior cruciate ligament prevents anterior displacement and excessive internal rotation of the tibia on the femur.

Ligament injuries usually occur in pairs. Once there is compromise of the ligaments, there will be excessive movement of the knee. Increased laxity leads to excessive shear stress resulting in accelerated erosion of the meniscal surfaces and increased synovial fluid (joint swelling) production due to irritation of synovial tissues (synovitis).

Finally, the extensor or quadriceps mechanism sits in front of the knee joint. This includes the patella and the patellar tendon that connects the patella to the tibia. This tendon covers the patella and continues proximally as the quadriceps tendon (the quadriceps muscle covers the front of the thigh). The patella fits into a groove called the patellofemoral groove on the front of the distal femur. The way in which the patella fits into this groove, and slides as the knee bends, can affect the overall function of the knee. The patella functions like a fulcrum, and increases the force exerted by the quadriceps muscles as the knee straightens. When the quadriceps muscle contract, the knee straightens. When the quadriceps relaxes, the

knee bends. The quadriceps or extensor mechanism is the motor that drives the knee joint and allows us to walk.

INJURY HISTORY

The history of injury usually provides us with the likely diagnosis, and we rely on the physical examination and diagnostic tests primarily to confirm or refute our initial impressions. It is extremely important to note the mechanism of injury. Appropriate questions include: What activity was the individual engaged in? What type of surface was the patient walking/running on and what type of shoes were worn? What direction(s) was force sustained? Was there a direct trauma or specific injury or did symptoms progress over time from cumulative effects? Has it been recurrent and/or progressive? Is there a history of a twisting episode? Do the patient report swelling, clicking, popping, grinding, giving way (buckling) or locking? What are the patient's functional limits (walking, squatting, kneeling, stair climbing, etc.)? Is there a problem with sitting? Is there stiffness upon arising in the morning or after sitting?

PHYSICAL EXAMINATION

The physical examination starts out with observation of the patient during walking, sitting and moving about the exam room. It is helpful to ask the patient to point to the spot where and when it hurts the most. The physician or therapist is looking for gait deviations, alignment problems, bruising, swelling, temperature changes, muscle wasting, kneecap (patella) grinding, ligament instability, and/or loss of motion. There are also several provocation tests to evaluate for meniscal and/or ligamentous injury. The Apprehension sign involves passive lateral movement of the patella. When positive, this will result in a reflex contraction of the quadriceps muscle, thus indicating lateral instability of the patella. Collateral ligament testing provides force to test for laxity of the medial and lateral collateral ligaments. A positive Lachman’s test and/or Anterior Draw sign indicate a potential tear of the ACL. The McMurray maneuver and/or Apley test are used to assess for meniscal injuries. Specific descriptions of these tests are beyond the scope of this article.

RADIOLOGIC STUDIES

X-rays do not show ligament or cartilage damage but do show fractures and degenerative changes. A Magnetic Resonance Imaging (MRI) scan can be helpful but is not necessary unless the physician feels strongly that the outcome of the scan will contribute to or change the proposed course of treatment. It is strongly recommended that clinicians match clinical signs and symptoms with MRI findings since there is some suggestion in the literature that abnormal MRI findings such as meniscal tears are found even in asymptomatic people.

DIFFERENTIAL DIAGNOSIS

Patients who present with knee complaints do not necessarily have a knee problem. Hip pathology typically refers pain to the groin and knee. Patients with lumbar disc disease (radiculopathy) or femoral nerve compromise may have knee pain as a primary presenting complaint. When an individual has pathology in the back, hip, ankle or foot, improper body mechanics and gait may result in abnormal forces

on the knee eventually leading to true knee problems. The treatment needs to be directed at the initial offending problem. There are medical problems that may involve the knee. Gout while most commonly found in the toes, foot and ankle is also found in the knee. Pseudogout is found predominantly in the knees and wrists and hydroxyapatite deposition may involve the knee, although it is more common in the shoulder. Detailed descriptions of these conditions are beyond the scope of this article.

BURSITIS OF THE KNEE

The bones, ligaments, tendons and muscles are kept from rubbing each other by four small fluid filled sacs call bursae. These bursae are susceptible to an inflammatory response from direct or indirect trauma. The prepatellar bursa is the most commonly affected area (housemaid's knee) and may present with a significant degree of swelling. Traumatic prepatellar bursitis may be caused by acute injury such as a fall directly on the patella or by recurrent minor injuries. The infrapatellar and deep patellar bursae are infrequently affected. The fourth bursa (pes anserine) is located over the medial tibial area and is rarely affected with bursitis and is a diagnosis of exclusion. Bursitis conditions occurring about the knee are usually treated with NSAIDs, ice, physical therapy modalities, and possible activity modification. The treatment of knee bursitis can include aspiration and steroid injection. When infection is present, incision and drainage may be required when the patient fails to respond to non-surgical treatment.

MENISCAL INJURY

The meniscus is a commonly injured structure in the knee. The injury can occur in any age group. In younger people, the meniscus is fairly tough, and tears usually occur as a result of a fairly forceful twisting injury. The meniscus grows weaker with age, and in the working age population, meniscal tears can occur as a result of a fairly minor injury, even from the up and down motion of squatting. There are various specific types of meniscal tears (bucket handle, horizontal, longitudinal, oblique, radial, flap) or the tear can be a degenerative type tear where a portion of the meniscus is frayed and torn in multiple directions. A meniscal tear most often involves the medial meniscus.

The patient may give a history of knee injury associated with a rotational component. They usually present with complaints of a catching, locking or giving way sensation. There may be mild to moderate knee joint swelling. Thigh atrophy may be present. Pain is the most common problem caused by a torn meniscus. It may be felt along the joint line where the meniscus is located or may be vaguer and involve the whole knee. If the torn portion of the meniscus is large enough, locking may occur. The fragment of meniscus gets caught in the hinge mechanism of the knee and will not allow the leg to straighten completely. There are long term effects of a torn meniscus as well. The constant rubbing of the torn meniscus on the articular cartilage may cause wear and tear on the surface, leading to arthritis of the joint.

Initial treatment for a torn meniscus usually is directed towards reducing the pain and swelling in the knee. If the symptoms continue, arthroscopic surgery may be required to either remove the torn portion of the meniscus or to repair the tear. Repair of the meniscus is not possible in most cases. Young people with relatively recent meniscal tears are the most likely candidates for repair. Degenerative type tears in older people are not repairable.

The menisci provide a larger area of contact between the femur and tibia which provides cushioning and stability. A torn meniscus that cannot resist peripheral displacement will no longer provide a protective function. With the meniscus removed there is an increase in shear force that may cause premature degeneration of cartilage.

ANTERIOR CRUCIATE LIGAMENT INJURIES (ACL)

In the intact knee, the ACL provides 85% of the restraint to anterior displacement. The ACL is commonly damaged in a forceful valgus-external rotation injury. This type of injury is frequently associated with damage to the medial collateral ligament and medial supporting structures. Acute disruption is commonly associated with an audible sound, hemarthrosis with blood in the joint, guarded or painful range of motion and profound disability. MRI scanning is effective in defining ACL lesions. Functional bracing may be of benefit when there is low-grade laxity and under mild load conditions. Up to 70% of patients complains of "giving way" even with bracing.

If left untreated, the torn ACL leads to anterior laxity, rotatory instability, meniscal tears and progressive degenerative changes. Reinjury is common after resuming previous athletic activity level. Individuals with an isolated ligament injury who are willing to moderate the activity will find non-operative treatment to be satisfactory in the majority of cases. The ACL is the more commonly injured than the PCL.

POSTERIOR CRUCIATE LIGAMENT INJURIES (PCL)

The major cause of injury to the PCL, like the ACL, is probably sports related. This injury occurs when the knee is forcefully twisted or hyperextended. The PCL controls how far backward the tibia moves in relation to the femur. If the tibia moves too far the PCL can rupture. This tearing of the ligament results in a feeling of instability in the knee. The PCL may not be the only ligament injured when the knee is injured violently, such as in a car wreck or blow to the upper tibia from the front. It is common to see several ligaments in addition to the PCL injured during a knee dislocation.

The symptoms following a tear of the PCL are variable. Since the PCL is really outside of the knee joint, swelling from torn blood vessels in the ligament may not fill the joint with blood (in contrast to the ACL tear). The instability caused by the torn ligament leads to a feeling of insecurity and giving way of the knee, especially when trying to change direction on the knee.

The pain and swelling from the initial injury will usually resolve after 2 to 4 weeks, but the instability remains. The symptom of instability, and the inability for the patient to trust the knee for support is what requires treatment. There is clinical evidence that long term instability leads to early arthritis of the knee.

The history and physical examination is probably the most important tool in diagnosing a ruptured or a deficient PCL. The physician assesses by examination if the tibia moves too far back on the femur. X-rays of the knee to rule out a fracture may also be ordered. Since ligaments and tendons do not show up on x-rays, the most accurate test without actually looking into the knee, is the MRI scan. In some cases, arthroscopy may be used to make the definitive diagnosis if there is a question about what is causing the

problem. The vast majority of PCL tears are diagnosed without resorting to surgery, and arthroscopy is usually reserved to treat the problems.

The first goal of treatment for a posterior cruciate ligament injury is to limit swelling and pain. Immediately after this type of injury, ice is a good first aid treatment. A long-leg brace and use of crutches may help protect the knee from further. An anti-inflammatory medication may be prescribed, and physical therapy is then begun. Less severe tears are treated with a progressive rehabilitation program. Patients involved in high level activities, like cutting, pivoting, or working on unlevel surfaces may require a dynamic knee brace before returning to work or sports.

The key to conservative treatment is to provide long term control of swelling, giving way, and pain. If these areas can be managed, surgery may not be needed. However, the individual may also need to modify recreational and/ occupational activities. Specific physical therapy modalities to help control pain and inflammation can include electrical stimulation, cold packs/wraps, and cold whirlpool. In addition, the individual should be instructed to apply the RICE (rest, ice, compression, and elevation) principle several times a day as a home supplement to supervised visits. As swelling and pain decrease, the patient will move through a gradual and progressive rehabilitation program. Treatment will focus on restoring normal movement of the knee, developing control in the quadriceps muscle group, and applying functional exercises that simulate normal activity. This will progress to higher level agility, endurance training, and cutting or pivoting activities based on specific return to sport or work needs. To help improve stability a brace may be suggested.

Surgery may be suggested if the symptoms of instability are not controlled by a brace and a rehabilitation program. Most surgeons now favor arthroscopic reconstruction of the PCL using a piece of tendon or ligament to replace the torn PCL. After surgery, a physical therapy rehabilitation program should be instituted which usually takes about 6 months after surgery to achieve the best results.

COLLATERAL LIGAMENT INJURIES

The Collateral Ligaments are commonly injured structures in the knee. These injuries can occur in many ways. The injury usually involves a significant force, such as a fall while skiing or a direct force to the side of the leg.

The collateral ligaments limit side to side motion of the knee. If these ligaments are stretched too far, they may tear. The tear may occur near the ends or in the middle of the ligament, such as in this tear of the medial collateral ligament. If the force is great enough other ligaments may also be injured. The most common combination is a tear of the medial collateral ligament and a tear of the anterior cruciate ligament. The lateral collateral ligament on the other side of the knee can also be torn, but it is less commonly injured.

An injury violent enough to tear one of the collateral ligaments usually causes significant damage to the surrounding soft tissues. There may be bleeding and/or swelling of the tissues, and perhaps inside the knee joint (causing an effusion). The knee typically is stiff and painful. As the initial stiffness and pain reside there may be a feeling of instability, and the knee may buckle.

The initial physical examination usually gives a very good indication of what ligaments have been torn in and around the knee. X-rays may be required to rule out the possibility that bony damage has occurred as well. Stress x-rays (plain x-rays taken with someone attempting to open the side of the joint that is suspected to be unstable) may confirm that one of the collateral ligaments has been torn. The x-rays will show a widening of the joint space on that side if instability is present. An MRI scan may be ordered, primarily when there is evidence that multiple injuries have occurred, including injury to the meniscus and possibly, the anterior cruciate ligament.

Most injuries to the collateral ligaments will heal with simple immobilization in a cast or brace for 4-6 weeks. An isolated injury to one of the collateral ligaments infrequently requires surgical repair or reconstruction. The initial treatment for a collateral ligament injury focuses on decreasing inflammation. Rest and anti-inflammatory medications can help accomplish this. As the ligament heals, a physical therapy program will help in decreasing pain and inflammation, improving motion, and regaining strength. Physical therapy treatment modalities can include electrical stimulation, cold packs/wraps, and ultrasound. Initial rest is followed by range of motion and by progressive stretching and strengthening.

PATELLOFEMORAL PROBLEMS

Patellofemoral pain accounts for 25% of all knee injuries and is very common in runners. Nearly all patellofemoral disorders can be traced to some underlying predisposition of malalignment of the knee extensor mechanism. This can be the result of soft tissue inflexibility, muscle imbalances or bony malalignment of the lower extremity. Problems develop when the patella suffers wear and tear. The underlying cartilage begins to soften (Chondromalacia Patellae). Degeneration can develop from generalized wear and tear from use over time, but problems can also arise from excessive tightness of the muscles on the front or outside of the thigh. Patients can have early patellofemoral problems with normal x-rays and even without evidence of chondromalacia.

One of the more common problems is an abnormality in the way the patella moves as the knee is bent or straightened. This usually results from either a muscle or structural imbalance and is referred to as Patellofemoral Syndrome. The patella normally moves along a path that is controlled by the quadriceps muscle located on the front of the thigh. If the inside part of this muscle group is weak, a muscle imbalance occurs, and the patella will stray from its normal path. In time this faulty movement can cause wear and tear of the cartilage. The femoral condyle is a protrusion on the outside part of the knee that normally acts as a buttress for the patella, keeping it in the groove. In cases where the femoral condyle is too small, the patella has a tendency to slip outward.

Despite these imbalances, some people never have significant knee pain. Others experience vague pain in the knee that is difficult to localize. Pain may be felt along the inside edge of the patella, though this is not always the case. Typical complaints include pain when walking down stairs or hills, keeping the knee bent for long periods as in the car or theater or with pressure to the patella. The knee may feel as if it gives out on occasion, but this is thought to be a reflex response to the pain and not an indication of instability.

Diagnosis begins with a complete history the knee problem followed by an examination of the knee, including the patella. While an x-ray can help determine is the patella is abnormally worn, it may not be

helpful in determining if the patella is potentially tracking properly as this is primarily a clinical diagnosis. In the later stages, arthritis may be seen between the patella and femur. An MRI scan can help rule out other conditions such as a torn cartilage if this is suspected.

The initial treatment for patellar dysfunction begins by decreasing inflammation in the knee. Rest, anti-inflammatory medication and physical therapy can help decrease the pain, inflammation and swelling. If these measures fail to provide lasting improvement, surgery may be indicated. A lateral release may be performed to relieve pressure on the articular cartilage, allowing the patella to shift back to a normal position. In this operation, the tight ligaments on the outside (lateral side) of the patella are cut to allow the patella to slide more towards the center of the femoral groove. These ligaments eventually heal with scar tissue that fills in the gap created by the surgery. In cases of severe malalignment, a lateral release alone may not be enough. The tendons on the inside edge of the knee (the medial side) may have to be tightened. In very severe cases, such as in a dislocating patella, the attachment of the patellar tendon may also have to be moved.

KNEE OSTEOARTHRITIS

The knee is the most common joint to be involved in osteoarthritis and it is a common problem for many people over 50. Osteoarthritis is sometimes referred to as degenerative, or wear and tear arthritis. Osteoarthritis may result from an injury to the knee earlier in life. Fractures involving the joint surfaces, instability from ligament tears, and meniscal injuries can all cause abnormal wear of the knee joint. Not all cases of osteoarthritis are related to prior injury, however. Research has shown that some people are prone to develop osteoarthritis, and this tendency may be genetic. Obesity has been shown to be a causative (and not just associated) risk factor. The medial compartment of knee is most commonly affected when obesity is a factor. The problem is more common in females. Treatment is geared towards walking, weight loss, wraps to minimize effusion, ice/heat, NSAIDs and eventually may lead to knee replacement.

The main problem in osteoarthritis is degeneration of the cartilage that covers the joint. This results in areas of the joint where bone rubs against bone. Bone spurs may form around the joint as the body attempts to repair itself. Osteoarthritis develops slowly over several years. The symptoms of osteoarthritis are pain, swelling, and stiffness. The pain is usually worse during and after activity. Early in the course of the disease, pain may be absent or minimal while walking, but after sitting for several minutes the knee becomes stiff and painful. As the condition progresses, pain can interfere with even simple daily activities. In the late stages, the pain can be continuous and even affect sleep patterns.

The diagnosis can usually be made on the basis of the initial history and examination. X-rays are very helpful in the diagnosis and will be the only special test required in the majority of cases. Treatment is directed at decreasing the symptoms and slowing the progression. The first goal is to help reduce pain. Acetaminophen or an anti-inflammatory medication should be used in combination with physical therapy. The patient is encouraged to keep the muscles around the knee flexible and strong and to avoid unnecessary trauma. A cane may be helpful to unload weightbearing. It should be used in the opposite hand to provide a wider base of support. A good pair of shoes may help reduce impact. Avoiding excessive stair climbing or prolonged standing and walking on hard surfaces is also recommended.

If the symptoms persist or progress, a steroid injection may be used to bring the inflammation under better control and ease the pain. Steroids are very powerful anti-inflammatory medications but may have secondary effects that limit usefulness in the treatment of osteoarthritis. The major drawback of intra-articular injections is that it may actually speed the process of degeneration with repetitive use. Most physicians avoid multiple injections unless the joint is already in the end stages of degeneration.

Arthroscopy is sometimes useful in the treatment of osteoarthritis of the knee. Looking directly at the articular cartilage surfaces of the knee is the most accurate way of determining how advanced the osteoarthritis is. Arthroscopy also allows the surgeon to debride the knee joint. Debridement essentially consists of cleaning out the joint of all debris and loose fragments. The areas of the knee joint that are badly worn may be roughened to promote the growth of new cartilage. If successful, arthroscopy usually affords temporary relief of symptoms for somewhere between 6 months to 2 years.

Osteoarthritis usually affects the inside half (medial compartment) of the knee more often than the outside (lateral compartment). This can lead to the lower extremity becoming slightly bowlegged, or in medical terms, a genu varum deformity. The result is that the weight bearing line of the lower extremity moves more medially (towards the medial compartment of the knee). The end result is that there is more pressure on the medial joint surfaces, which leads to more pain and faster degeneration. There is a knee brace available for medial compartment osteoarthritis.

In some cases, realigning the angles in the lower extremity can result in shifting the weight-bearing line to the lateral compartment of the knee. This, presumably, places the majority of the weight-bearing force into a healthier compartment. The result is to reduce the pain and delay the progression of the degeneration of the medial compartment. The procedure to realign the angles of the lower extremity is called a proximal tibial osteotomy. In this procedure a wedge of bone is removed from the lateral side of the upper tibia. This converts the extremity from being bow-legged to knock-kneed. This procedure is not always successful, and generally will reduce pain, but not eliminate it. The advantage to this approach is that very active people still have their own knee joint, and once the bone heals there are no restrictions to activity. The proximal tibial osteotomy in the best of circumstances is probably only temporary. It is usually done in hopes of postponing the need for a total knee replacement.

The ultimate treatment for advanced osteoarthritis of the knee is to replace the joint surfaces with an artificial knee joint. The decision to proceed with a total knee replacement is usually only considered in people over the age of 60, although younger patients sometimes require the surgery simply because no other acceptable solution is available to treat their condition. The main reason that orthopedic surgeons are reluctant to perform the surgery on younger individuals, is that the younger the patient, the more likely the artificial joint will fail. Replacing the knee the second and third time is much harder and much less likely to succeed.

Artificial knee joints last about 12 years in the elderly population. Younger patients are more active and place more stress on the artificial joint and this can lead to loosening and premature failure. Obviously, younger patients are also more likely to outlive their artificial joint and will almost surely require a revision at some point down the road.

KNEE INJURES IN THE WORKPLACE

Most work-related knee injuries can be diagnosed on the basis of the specific mechanism of injury. A detailed account of the injury, including the nature of the injury and initial impairment is critical in validating the industrial claim. The development of MRI scanning and arthroscopy has resulted in a dramatic change in advancing our ability to diagnose and treat knee injuries in injured workers. This has led to accelerated healing with earlier return to the workplace.

Several studies have examined the causal relationship between osteoarthritis (OA) of the knee and occupational activity. Although most of these studies are either occupation specific (e.g. carpet layers, roofers, coal miners) they generally form similar SUMMARYs. The majority of studies propose that osteoarthritis of the knee is more commonly seen in those occupations which require frequent or repetitive squatting, kneeling or climbing. In one study, the increase in risk associated with kneeling or squatting appeared to be more marked in subjects whose jobs entailed heavy lifting, however the size of the study prohibited a direct correlation. (Cooper C. Ann Rheum Dis 1994 Feb;53(2):90-93)

Another study reviewed the literature on the risk of knee disorders in connection with kneeling or squatting work and heavy physical work. They concluded that all the studies demonstrated a significantly increased prevalence of knee osteoarthritis for subjects with kneeling or squatting work, and four of eight studies demonstrated a significantly increased prevalence in those performing heavy physical work. All the studies on bursitis showed an increased prevalence of bursitis in subjects with kneeling work. Occupational exposure could not be sufficiently documented as the cause of meniscal lesions and chondromalacia.

A study at Stanford University, examined the roles of physical activity (both work and leisure), obesity, and a history of significant knee injury in the development of OA. They concluded that the obesity, significant knee injury and long-term heavy physical activity are important risk factors for the development of OA of the knee.

SUMMARY

The knee is the largest joint in the body and basically functions as a hinge joint providing a fairly wide range of motion. By permitting bending of the lower extremity, the knee is very important for performing many daily activities and for efficient walking. The knee is commonly injured in all age groups and injuries can frequently lead to significant limitations and lost time from work. Prompt diagnosis and treatment of these injuries is of paramount importance in order to prevent and/or reduce long term disability.

The knee is susceptible to trauma because of its location and because it is not protected by layers of muscle or fat. The patient often gives a history of a twisting episode followed by swelling, clicking, popping, grinding, giving way (buckling) or locking. There are many areas around the knee that can be injured, but the most commonly affected structures are the cartilage (menisci) and ligaments. The ligaments are found on each side of the joint and there are two other ligaments that cross inside the knee, known as cruciates (anterior and posterior).

Industrial exposure may contribute to knee pathology and therefore disability through specific injury but also secondary to cumulative trauma. Although many problems about the knee can be treated conservatively with early mobilization, ice, physical therapy, and non-steroidal anti-inflammatory drugs, some conditions will require surgery.

ANKLE & FOOT INJURIES

INTRODUCTION

We may ignore and take for granted our feet and ankles either by wearing poorly designed footwear or by engaging in harmful activities, but we realize soon enough if they have suffered abuse or are injured. Foot and ankle problems are actually very common, and it is not unusual to hear someone say, "oh my aching feet."

This part of our body is critical for providing a stable platform of support in standing, to assist with efficient forward propulsion of the body, to adapt to changes in uneven terrain and to attenuate impact loading (shock absorption). The disorders of the ankle and foot are difficult to describe due to their complexity. The ankle and foot are composed of more than 28 bones. The complexity is further reflected by the presence of numerous muscles, tendons, ligaments, fascial structures, nerves and blood vessels as well as skin, hair and nails. The ankle and foot are subject to numerous static disorders and multiple potential traumatic events.

ANATOMY OF THE ANKLE & FOOT

The ankle is a complex mechanism. It is a hinge joint. What we normally think of as the ankle is actually made up of two joints: the first, the true ankle joint is composed of 3 bones. The tibia projects downward to form the medial malleolus (the inside, or medial, portion of the ankle), the fibula projects downward to form the lateral malleolus (the lateral or outside portion of the ankle) and the talus coming up from the foot underneath.

The true ankle joint is responsible for the up and down motion of the foot. Beneath the true ankle joint is the second part of the ankle, the subtalar joint, which consists of the talus on top and calcaneus on the bottom. The subtalar joint allows side to side motion of the foot. The ends of the bones in these joints are covered by articular cartilage.

The foot itself is composed of 14 phalangeal, 5 metatarsal, 7 tarsal and various accessory bones. The posterior portion of the foot includes two of the largest bones of the foot, the calcaneus and the talus. The calcaneus (heel bone) rests beneath the talus (ankle bone) and forms the posterior segment of the foot. The anterior segment is best thought of as having a medial component (mostly concerned with propulsion) and a lateral component (chiefly serves for balance).

The ligaments of the ankle are crucial for support. The deltoid ligament (medial collateral ligament) on the inside of the ankle is a strong triangular band that provides medial stability (prevents eversion). Turning the ankle out (eversion) will injure this ligament and/or cause an avulsion "chip" fracture of the distal tibia (medial malleolus). More commonly injured is the lateral collateral ligament (on the outside of

the ankle) with the foot pointing down (plantar flexion) and turning in (inversion injury). The lateral collateral ligament is actually made of three separate ligaments.

EXAMINATION OF THE ANKLE & FOOT

The examination starts with inspecting the ankle and foot with the patient standing, walking and sitting. Any deformity, swelling, temperature change or discoloration is noted. Changes in the skin, hair pattern or nails is observed. The peripheral pulses are checked. Ankle and foot range of motion and any associated discomfort is recorded. Any areas of tenderness are documented. Strength and sensation are checked. A side-to-side rocking motion tests the subtalar joint and the midtarsal joints are moved to note motion in adduction and abduction.

ACHILLES TENDINITIS / TENDINOSIS

While there are many causes of posterior ankle pain, there are two distinct conditions that may affect the Achilles tendon, tendinitis and tendinosis, each having a distinct prognosis. Tendinitis involves a peritendinous inflammation which does not generally progress to degenerative tendinosis or rupture. It often results from over exercise, especially in runners. Tightness of the heel cord (contracture) will exacerbate the patient's symptoms. On exam, the patient generally does not have a strength deficit and does not have tenderness to deep palpation. Treatment includes local heat or ultrasound and heel cord stretching in mild cases. For moderate cases, a short period of rest followed by gradual mobilization with temporary avoidance of repetitive activities such as running may be helpful. Steroid injections into or around the Achilles tendon may provoke rupture and should generally be avoided. Tendinosis is a degenerative process that can occur without signs of inflammation and may be related to region of diminished blood supply just above the tendon insertion. Often the tendinosis remains subclinical until it presents as a rupture. The differential diagnosis includes systemic conditions such as gout. On examination, deep palpation will elicit tenderness. There may be palpable nodularity in the tender aspect of the tendon and weakness is evidenced by inability to raise up on the toes. X-rays may show calcification within the Achilles tendon, which may indicate a more proximal tendinosis.

ANKLE SPRAINS & STRAINS

The terms sprains and strains are often used interchangeably but they actually represent different types of injuries. Strains involve a pull, bruise, or tear in a muscle or tendon and occurs from improper or overuse of a muscle. A sprain occurs when a joint has been twisted or wrenched severely enough to tear or damage supporting ligaments (fibrous tissue that surrounds the joint, adding additional strength and stability). A sprain generally requires a longer healing period and is considered a more serious injury than a strain.

Ankle sprains may be the most common acute orthopedic injury. It's not unusual to hear from a patient that this has happened before, sometimes on multiple occasions. Sports or work activities requiring a lot of climbing, stopping, starting and twisting motions have a high incidence of ankle injuries. Caution should be taken when the terrain is uneven or there are potholes. The injured person usually presents with a history of their ankle "turning." Sometimes a "pop" or "snap" is felt or heard. Difficulty walking follows

the incident and in a short time the ankle swells, sometimes so excessively that the individual is sure that it's broken.

The typical ankle sprain results when the foot turns in (inversion) and results in a stretching or tearing of the small ligaments that attach bone to bone on the outside or lateral aspect of the ankle (lateral collateral ligament).

Sprains of the medial ankle, or Deltoid ligament, are less common and involve eversion injuries where the foot turns severely outward. In fact, an injury to the ankle that forcefully everts the ankle will usually cause bony damage rather than spraining the ligament.

Lateral ankle sprains (lateral collateral ligament) are classified into three grades. A grade 1 sprain is a partial tear of the anterior talofibular ligament (ATF). A grade 2 sprain is usually a tear of the ATF, but the calcaneofibular ligament (CF) remains intact. A grade 3 sprain is a tear of both of these ligaments. The physical examination almost always shows swelling and discoloration (black and blue) over the outside part of the joint. Frequently, there is instability noted when the heel structures are moved forward and back as the leg is stabilized (the drawer test). Range of motion in the ankle can be limited secondary to pain. X-rays are essential even though sprains are usually negative because fractures must be ruled out, as the treatment would be different with some unstable fractures requiring surgical internal fixation.

Although the treatment varies from place to place, for any acute ankle sprain, the best initial treatment includes protection, rest, ice, compression, non-steroidal anti-inflammatory medication and elevation. If the sprain is minor (grade 1), these measures will suffice, although additional bracing or taping of the ankle gives much more support and allows the patient more confidence while ambulating or exercising.

The more severe sprains (grades 2 - 3), that have greater instability and tearing of the ligaments, should be placed in a walking cast for three weeks to allow the injured structures to heal. These casts can be non-removable fiberglass or the removable variety. Following this three-week period, the patient should engage in exercises and two to three weeks of physical therapy to strengthen the ankle and reduce swelling. During this period, the ankle is often taped or braced by the physician, therapist, or trainer.

Surgical intervention for more severe (Grade 3) ankle ligamentous injuries is controversial with some authors advocating non-operative treatment while others propose surgical intervention. A detailed discussion of this controversy is beyond the scope of this article but in general, the non-athlete is best treated conservatively.

In the long run, the very best treatment of ankle sprains is prevention. This means that during activities that have a high incidence of ankle injuries the individual should wear appropriate footwear, a brace or get the ankle taped. Hightop gym shoes or boot can effectively provide ankle support. Braces are relatively inexpensive and easy to use. Taping works well, but requires someone available who is well trained to apply it appropriately.

HEEL PAIN

The most common cause of deep pain on the bottom surface of the heel is *plantar fasciitis* (inflammation of the plantar fascia). The plantar fascia is a broad band of fibrous tissue that runs along the bottom surface of the foot from the heel to the toes. It is just below the skin and subcutaneous fat. It helps to secure the arch. Long standing inflammation causes the deposition of calcium at the point where the inserts into the heel bone. This results in the appearance of a bony heel spur on x-ray. The spur itself is usually not the source of the pain.

Symptoms can include sharp pain localized to the bottom and/or inside margin of the heel often worse on arising in the morning and after rest. The pain is often aggravated by prolonged weightbearing and ambulation and may severely limit activities. It is most common in middle-aged and overweight adults. Causes can include excessive load on the foot from obesity, excessive flattening of the arch on weight bearing, a tight plantar fascia and over *pronation* of the foot (a complex motion including outward rotation of the heel and inward rotation of the ankle).

Treatment can include application of ice to the heel area after prolonged activity along with wearing supportive shoes with a stiff heal counter (the part of the shoe which wraps around the heel) and a good arch. A well-made running or walking shoe is a good example. Sometimes a shoe with a moderately high heel will relieve pressure on the fascia. Stretching exercises for the calf muscles will also help stretch the plantar fascia. This should not be attempted when the heel is sore. Anti-inflammatory medication can be helpful also as can a localized steroid injection. Physical therapy modalities such as localized heat or the application of ultrasound may be beneficial along with taping and padding to relieve strain on the plantar fascia. An orthotic such as a shoe insert may help control foot function. Surgical release of the plantar fascia and excision of the heel spur is rarely required.

Other causes of heel pain include various types of arthritis, trauma to the heel, a calcaneal stress fracture, inflammation of the tendons around the heel, a heel neuroma (benign tumors of the nerves around the heel), abnormality in the shape of the heel bone and a foreign body such as a splinter in the heel.

FOREFOOT PAIN

There are a number of possible causes of forefoot pain including stress fractures, Morton’s neuroma (benign tumor of a nerve running between the metatarsals), metatarsalgia (painful and inflammation of the metatarsal bones and their soft tissue sheath), capsulitis (painful and inflammation of the joints between the metatarsal bones and toes), tendonitis (inflammation of the tendons which course along the top of the foot), dislocation of a joint between a metatarsal and a toe (metatarsal-phalangeal joint), severe plantar callus (callus on bottom of the foot) or bursitis (an inflamed fluid-filled sac often between a bone and an area of pressure).

STRESS FRACTURES

A stress fracture is a break in a bone cause by repetitive stress. It may occur in any bone, but is quite common in the metatarsal bones of the foot. There is often no recollection of injury. The patient may simply develop a painful forefoot after some activity such a walking, sports, or stooping down onto the ball of the foot. A small crack develops in the cortex (outer shell) of the bone. Without proper treatment this may progress to a through and through fracture of the bone. The second and third metatarsals are the most commonly affected. Metatarsal stress fracture may not become apparent on x-rays until a few weeks after the injury. The individual will complain of sharp pain in the forefoot, aggravated by walking and there will be tenderness to pressure on the top surface of a metatarsal bone along with diffuse swelling of the skin over the forefoot. Causes of stress fractures include decreased density of the bones from osteoporosis and from unusual stress on a metatarsal due to malposition or another forefoot deformity (e.g. bunion). The problem can be related to obesity, abnormal foot structure or mechanics (e.g. flatfoot) and to increased levels of activity, especially without proper conditioning.

Evaluation includes x-rays and possibly a bone scan. Initial treatment includes icing to reduce swelling, limiting weight bearing, applying orthopedic taping and padding to relieve stress from the metatarsals, and using a special surgical/trauma shoe with a rigid sole. In some cases, a plaster cast may be necessary. Medication can be prescribed for pain relief and to reduce inflammation. Physical therapy may be helpful.

METATARSALGIA

Metatarsalgia is essentially pain and tenderness of the bottom surface (plantar) of the metatarsal bones (this is commonly referred to as the ball of the foot) that bear a disproportionate amount of the body weight. In the normal foot the metatarsal heads bear one-half of a person’s body weight when stepping on the front or ball of the foot. Any foot and ankle problem that concentrates weight bearing on the metatarsal heads may lead to metatarsalgia. The finding of tenderness at the area of the metatarsal heads makes the diagnosis. Treatment can include weight reduction and heel cord stretching if a heel cord contracture is present. A metatarsal pad or bar and/or orthotics can reduce forefoot pressure and transfer weight bearing to longitudinal and metatarsal arches and can lower the heel to reduce metatarsal head pressure. Rocker bottom shoes can reduce forefoot motion and pressures and a carefully placed metatarsal pad proximal to painful metatarsal head can relieve discomfort.

PES CAVUS & PES PLANUS

Pes cavus, also termed clawfoot or hollow foot, is essentially a foot with a very high arch. This can be totally asymptomatic but when symptoms do exist the individual experiences pressure on the metatarsal heads or unusual fatigue when walking or standing. Mild cases can be treated with properly fitted shoes with a low heel and a metatarsal bar. Stretching exercises are recommended. Surgery should be avoided if at all possible and should only be considered in severe cases. Pes planus, or flat feet, as known as "fallen arches" is usually congenital and is caused by ligamentous laxity. Treatment usually involves arch supports or orthotics.

CORNS OR CALLUSES

Corns or calluses are hyperkeratosis of the skin. This is a thickening of the surface layer of the skin in response to pressure. Calluses usually form on the ball of the foot, the heel, and the underside of the big toe (hallux). They may, however, form over any bony prominence. Corns usually form on the toes, where the bone is prominent and presses the skin against the shoe, ground, or other bones. As a corn becomes thick the tissues under the corn are subject to increased irritation. There may be a deep-seated nucleation. This is like a core where the corn is thickest and most painful. As corns become inflamed, there is pain and sometimes swelling and redness. Common places where corns form are the top surface of the toe, at the tip of the toe, and between the toes.

Symptoms can include a hard growth usually on the toes or ball of the foot and pain on weight bearing that is relieved by rest. The problem is more common in women than men. Causes include thin soled and high-heeled shoes, mal-alignment of the metatarsal bone, abnormalities of gait, flat feet and high arched feet, excessively long metatarsal bone, obesity, bony prominences, loss of the fat pad on the underside of the foot and a short Achilles tendon.

Sometimes a shoe can be too loose with the foot sliding forward with each step. Corns can also form from prolonged walking on a downward slope. Preventive treatment includes avoiding shoes that are too tight or loose and buying supportive shoes with an extra depth toe box (the part of the shoe over the toes) and a cushioned insole. It is also important to not apply socks or stockings tightly around the toes. A pumice stone or other abrasive can be used to reduce the thickness of the corn. Non-medicated pads can be around the corn to relieve pressure. Caution should be exercised with corn removing solutions as they contain acid and sometimes increase discomfort. Diabetics and others with diminished circulation should never use them. The orthopedic foot specialist or podiatrist may carefully debride (pare down) the corn and any deep-seated core it may have. It should be stressed that this provides only temporary relief, if the pressure continues after treatment. In selected cases crooked or deformed toes can be surgically straightened or the surgeon can remove bony prominences.

Complications that can result from corns include development of a bursitis (formation of a painful inflamed fluid-filled sac beneath the corn), infection, development of an ulcer or open area that forms within the corn. This may even extend down to bone. Because of the serious consequences of infection, diabetics and those with diminished circulation should always seek professional help. Discomfort of the back, hips, knees, legs, or feet may occur due to changes in posture and/or gait in an attempt to "protect" the painful callus. Other conditions which can resemble corns include warts, various tumors of the skin and subcutaneous (below the skin) tissues or reaction to a foreign body (e.g. splinter).

BUNIONS

Bunions are one of the most common deformities of the forefoot. There is a displacement of the first metatarsal bone toward the mid-line of the body and a simultaneous displacement of the great toe away from the mid-line (and toward the smaller toes). This causes a prominence of bone on the inside (medial) margin of the forefoot and this is termed a bunion. With continued drifting of the great toe *(hallux)* toward

the smaller toes, it may come to rest under (occasionally over) the second toe. The incidence of bunions is much higher in women than men. This is thought to be due to shoe fashion. There are genetic factors and certain predisposing abnormalities of foot function. Wearing narrow toed and high-heeled shoes can greatly accelerate the formation of a bunion. Symptoms include redness, swelling, or pain along the inside margin of the foot just behind the great toe. There is usually moderate to severe discomfort at the bunion when wearing shoes, particularly if tight fitting. A painful callus may develop over the bunion. There may be a painful corn on the adjacent sides of the first and second toes and there may be irritation caused by overlapping of the first and second toes. Additionally there may be stiffness and discomfort in the joint between the great toe and the first metatarsal. There may be a fluid filled cyst or bursa between the skin and the "bunion bone". Skin over the bunion may break down causing an ulceration that can become infected.

Bunions may be caused by a number of factors including an abnormality in foot function, particularly a pronated foot. This is probably the most important and common causative factor. There may be a family history of bunions. Narrow toed dress shoes and high heels may contribute to the formation of a bunion. Bunions are common in patients with Rheumatoid and Psoriatic arthritis. The individual may have a genetic or neuromuscular disease resulting in muscle imbalance. A limb length inequality can cause a bunion on the longer limb. Bunions may form when there is generalized laxity of the ligaments. Bunions may also form when there has been trauma to or surgery on the soft tissue structures around the great toe (first metatarsal-phalangeal) joint.

The individual can self-treat by applying a commercial non-medicated bunion pad around the bony prominence and wear shoes with a wide and deep toe box. The person should avoid all high-heeled shoes. For a bunion that becomes painful, red, and swollen, the individual may elevate the foot and apply ice for about 20 minutes every hour. For persistent or increasing symptoms a orthopedic foot specialist or podiatrist should be consulted. The practitioner may apply special pads and dressings to protect the bunion from shoe pressure. Injecting a steroid and local anesthetic around the bunion may help to reduce inflammation. This is especially useful if there is an associated bursitis. Another approach is to apply various splints or digital orthotics to reposition the great toe joint. Specially made shoes may be recommended. Finally, bunion surgery may be recommended.

Other causes of pain at the great toe joint include arthritis of the first metatarsal-phalangeal joint, injury to the soft tissue structures around the first metatarsal-phalangeal joint and fracture of the great toe or first metatarsal. Tight shoes may cause pain at an otherwise normal joint. Sesamoiditis, an inflammation of one or both small bones that rest in tendons under the first metatarsal-phalangeal joint, should also be considered in the differential diagnosis.

INGROWN TOENAILS

Ingrown toenails are one the more common foot problems. They can be very painful with people limiting their activity to keep off their sore feet. Ingrown toenails are caused by impingement of the skin along the margins of the nail by the nail plate. Some ingrown toenails are chronic with repeated episodes of pain and infection. Pain can be present without infection and occasionally infection is present without pain. The usual signs of infection include redness, swelling, increased warmth, and pain. Pain is felt along the

margin(s) of the toenail and is aggravated by wearing shoes, particularly those with narrow toes. The area may be sensitive to any pressure, even the weight of the bedclothes. There may be signs of infection with drainage of pus or a watery discharge tinged with blood.

Trauma, improper trimming of toenails, tight fitting shoes that compress the toes together and hose or socks that are too tight can cause the problem. There may be an abnormally shaped nail plate or other toenail deformities (e.g. excessively thick nail plate). Treatment starts with prevention by cutting the toenails straight across and leaving them slightly longer then the end of the toe and to avoid tight fitting footwear. Soaking the foot in a basin of warm water two or three times a day can help discomfort. An infected ingrown nail requires prompt professional attention. Treatment can include antibiotics and trimming a small spicule of nail to relieve the pressure. *Callus* (dead skin) accumulated in the nail groove can be removed. Routine ingrown toenail care may need to be done periodically. There may be a need to elevate the end of the nail plate to prevent impingement on the soft tissues or to surgically drain an infection or completely remove a deformed toenail so it will not grow back.

SINUS TARSI

The sinus tarsi is the depression found on the lateral side of the tarsus and is distal to and on the same level as the lateral malleolus. In the Sinus Tarsi Syndrome there is pain and tenderness on the lateral side of the hindfoot originating from the area of the sinus tarsus.

TARSAL TUNNEL SYNDROME

The Tarsal Tunnel Syndrome is a compression syndrome of the tibial nerve in the tarsal tunnel. The tarsal tunnel is formed by the flexor retinaculum behind and distal to the medial malleolus. Patients note pain, paresthesias, and in some cases atrophy of foot intrinsic muscles. Pain will radiate along the plantar side of the foot. A positive Tinel's sign can often be elicited behind medial malleolus. Manual compression for 30 seconds may reproduce symptoms. The differential diagnosis includes stress fractures, inflammatory arthritides, plantar fasciitis, herniated disk, peripheral neuropathy, a ganglia or neoplasm. Electrodiagnostic testing may reveal a prolonged distal motor latency and needle EMG abnormalities in the associated innervated muscle. An MRI may be used to identify ganglia or extrinsic masses and the specific site of compression. Conservative treatment includes removing external causes such as poorly fitting shoes, a medial arch support or orthotic device, non-steroidal medication and local steroids. Operative decompression is considered for patients with space occupying lesions.

ANKLE & FOOT FRACTURES

Fractures and fracture dislocations result from forces applied in various combinations and degrees that produce excessive motion or compression. From practical standpoint, the examining physician needs to analyze the accident to determine the direction of the causative force and the degree of instability that has resulted. Fracture reduction is accomplished by various means of applying force in the opposite direction. Surgery may be required. Post reduction and surgical treatment usually involves some type of immobilization (traction, cast, brace, strapping, etc.) for a period of time depending upon the stability of

the fracture and the healing time for the particularly individual. Rehabilitation follows with heat, massage and exercises to restore function.

While it is beyond the scope of this article to discuss the numerous and varied types of fractures involving the ankle and foot, a few of the more common work-related fractures will be discussed. A crush injury of the calcaneus (heel fracture) is particularly painful and disabling. The usual mechanism of injury is a fall from a height. The extent of damage and resultant disability will vary considerably with the fall distance, landing surface, and the patient's height, weight and occupation. There may be an associated lower spine compression fracture as well. Calcaneal fractures involve many months of immobilization and non-weight bearing is usually required during healing. With more involved fractures, an ankle fusion may be needed. Tarsal fractures and dislocation, other than those involving the calcaneus are less common. Fractures of the neck of the talus and partial or complete dislocation of this bone, are the next most common tarsal fractures.

Direct or indirect forces may cause fractures of the metatarsal shaft. Metatarsal stress fractures are discussed elsewhere in this article. Treatment for undisplaced fractures is a walking boot with a soft or spongy sole. In grossly displaced fractures surgery is usually required. Immobilization is required post-operatively to allow adequate fracture union.

Fracture phalanges are often caused by stubbing or crush injuries of the toes. Treatment includes careful cleansing of any damaged tissue with careful wrapping with a protective gauze dressing. Walking may be permitted if pressure on the toe can be avoided.

The most common dislocation is the first metatarsophalangeal joint (base of the big toe). Traction and direct plantar pressure with flexion of the phalanx readily reduce it. The joint needs protection for a period of time from excessive plantar pressure and a metatarsal bar in the shoe may be helpful.

FUNGAL INFECTIONS OR ATHLETE'S FOOT

Athlete's foot is a fungal infection characterized by itchy, scaly lesions between the toes that frequently leads to fissure formation. Secondary infections are not uncommon. Prevention relies on proper hygiene. Once the foot is infected, medication is available for treatment.

MEDICAL CONDITIONS & THE FOOT

Patients with peripheral vascular disease may develop increasing loss of distal blood flow resulting in cool feet with loss of sensation. The feet are more prone to trauma and skin breakdown. The patient with rheumatoid arthritis is particularly prone to degenerative changes, abnormal biomechanics and resultant pain and deformity. The patient with diabetes is prone to vascular disease and neuropathy with associated decreased sensation. The feet may be more prone to injury, yet with lack of pain from decreased sensation the individual my not realize that there is skin breakdown and infection developing. Toes may become necrotic, ulcers may develop and not heal in a timely manner and in the worse case, amputation may be necessary. For all of these medical conditions, prevention is the key to successful treatment with emphasis

on careful observation for skin changes or deformity, cleanliness, proper footwear and pacing activities to avoid overuse or abuse to the feet. Medications specific to the disease are also important.

SUMMARY

We may take our ankles and feet for granted, but they let us know when they have been abused or traumatized. In fact, ankle and foot problems are very common. Work related trauma whether on a cumulative or specific injury basis deserves immediate attention and medical care. The general podiatrist shares the care of ankle and foot problems with the family physician, internist, and sports injury specialist, physiatrist or general orthopedist in most cases. More complex case with difficult problems may require the podiatrist or orthopedist foot specialist to render care. While most ankle and foot problems respond to conservative care, surgery may be needed in certain cases. Proper treatment always starts with prevention by wearing proper footwear, avoiding abuse and being careful during work and sports activities.

CHRONIC PAIN DISORDERS

Many pain specialists recommend that the term "chronic pain" should be referred to as "persistent pain" – which can be continuous or recurrent and of sufficient duration and intensity to adversely affect a person's well-being, level of function, and quality of life.

Acute pain is characterized as being of recent onset, transient, and usually from an identifiable cause.

Chronic (or persistent) pain can be described as ongoing or recurrent pain, lasting beyond the usual course of acute illness or injury healing, more than 3 to 6 months, and which adversely affects the individual's well-being. Another definition for chronic or persistent pain is pain that continues when it should not. The International Association for the Study of Pain defines pain as a negative sensory and emotional experience. As such, the definition recognizes the important role of processes in the nervous system and brain (both neurological and psychological) in the experience of pain. Therefore, the nervous system and brain are very important therapeutic targets.

Chronic pain is classified by pathophysiology (the functional changes associated with, or resulting from disease or injury) as nociceptive (due to ongoing tissue injury) or neuropathic (resulting from damage to the nervous system – the brain, spinal cord, or peripheral nerves). The experience of pain can be due to either nociceptive and neuropathic changes or both -- but it is *always combined with the brain's reaction* to incoming information, including the brain's interpretation of what that incoming information means, what the brain learns as a result, and the responses it generates.

Central pain syndrome is a neurological condition caused by a process that specifically affects the central nervous system (CNS), which includes the brain, brainstem, and spinal cord. The disorder occurs in people who have or who have experienced strokes, multiple sclerosis, Parkinson's disease, brain tumors, limb amputations, brain injuries, or spinal cord injuries. It may develop months or years after injury or damage to the CNS. This also includes conditions such as chronic headaches, fibromyalgia, and Complex Regional Pain Syndrome (CRPS). In central pain syndromes, pain feels as though it is emanating from a specific place in the body, but the sensation is actually being generated by the nervous system and brain.

NEUROPATHIC PAIN

Neuropathic pain refers to pain related to the nervous system and can be divided into two basic categories: peripheral (nerves) and central (brain and spinal cord) etiologies. Common peripheral neuropathic conditions include polyneuropathy from diabetes or induced by chemotherapy, radicular pain (from injury to a nerve root), and postsurgical chronic neuropathic pain. Central neuropathic pain conditions include multiple sclerosis, poststroke pain, spinal cord injury–related pain, postherpetic neuralgia, complex regional pain syndrome, and trigeminal neuralgia.

The clinical presentation of neuropathic pain commonly includes descriptions of burning, pins and needles (paresthesia), tingling, numbness, electric shocks/shooting, crawling, itching, and intolerance to temperature. In more advanced cases, patients may describe pain arising from stimuli that are not usually painful (termed allodynia) or pain from normally painful stimuli that is out of proportion to what would be expected (termed hyperalgesia).

Multidisciplinary conservative care and nonopioid medications (tricyclic antidepressants, serotonin norepinephrine reuptake inhibitors, gabapentanoids, topicals, and transdermal substances) are recommended as firstline therapy; combination therapy (firstline medications) and tramadol and tapentadol are recommended as secondline; serotonin-specific reuptake inhibitors/anticonvulsants/NMDA antagonists and interventional therapies as third-line; neurostimulation as a fourth-line treatment; low-dose opioids (no greater than 90 morphine equivalent units) are fifth-line; and finally, targeted drug delivery is the last-line therapy for patients with refractory pain.

COMPLEX REGIONAL PAIN SYNDROME (CRPS)

CRPS DEFINITION

Complex regional pain syndrome (CRPS) is a chronic pain condition defined as a disorder of regions of the body characterized by spontaneous and evoked pain that is disproportionate in time or degree to the usual course of any known trauma or other lesion. The pain is not restricted to a specific nerve territory or dermatome and usually has a distal extremity predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings. The syndrome shows variable progression over time.

CRPS DIAGNOSTIC CRITERIA

Clinical diagnostic criteria for complex regional pain syndrome[1] are as follows:

- Continuing pain, which is disproportionate to any inciting event
- Must report at least one **symptom** in *three* of the *four* following categories:
    a. Sensory: Reports of hyperalgesia and/or allodynia
    b. Vasomotor: Reports of temperature asymmetry and/or skin color asymmetry
    c. Sudomotor/Edema: Reports of edema and/or sweating changes and/or sweating asymmetry
    d. Motor/Trophic: Reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)
- Must display at least *one* **sign*** at time of evaluation in *two* or *more* of the following categories
    a. Sensory: Evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement)
    b. Vasomotor: Evidence of temperature asymmetry and/or skin color changes and/or asymmetry
    c. Sudomotor/Edema: Evidence of edema and/or seating changes and/or sweating asymmetry
    d. Motor/Trophic: Evidence of decreased range of motion and/or motor dysfunction (weakness, tremor dystonia) and/or trophic changes (hair, nail, skin)
- There is no other diagnosis that better explains the signs and symptoms

*A sign is counted only if it is observed at time of diagnosis.

[1] Harden RN, Oaklander AL, Burton AW, et al. (Pain Med 2013; 14(2): 180-229)

Subtypes of complex regional pain syndrome (CRPS)

- CRPS I (old name: Reflex Sympathetic Dystrophy)
- CRPS II (old name: Causalgia): Involves definitive evidence of a nerve lesion
- CRPS-NOS* (not otherwise specified): partially meets CRPS criteria; not better explained by any other condition

*This subtype was added to capture any patients previously diagnosed with CRPS who did not meet the full criteria.

Onset of Symptoms:

From a specific insult, typical symptoms occur within the first few weeks of initiating event but certainly during 3-4 months after the initiating insult. Variable and evolving symptoms described above may occur later either due to natural evolution or though related to treatment for an original injury including surgery or additional insult secondary to compensatory trauma or misuse. Sometimes the diagnosis is made late because the symptoms were not recognized earlier by the treating physician(s).

CRPS Types:

"Warm CRPS" is associated with a warm, red, and edematous extremity, whereas "cold CRPS" presents with a cold, dusky, sweaty extremity. Acute CRPS is more often associated with a warm CRPS presentation, whereas chronic CRPS is more often characterized by a cold CRPS presentation.

Prognosis:

For acute CRPS, 74% of diagnosed CRPS cases resolved with relatively conservative care.

TREATMENT

The selection of a treatment approach depends on the severity of symptoms and the degree of disability. Of paramount importance is that a successful treatment outcome for CRPS depends on a coordinated functional restoration interdisciplinary team approach. Building a therapeutic alliance between the patient and the treatment team is of critical importance.

Since pain and limb dysfunction are the major early complaints, pain control, education, physical rehabilitation and emotional stabilization are the main treatment objectives. Coexisting problems such as depression, sleep disturbance, anxiety, fear of reinjury, and generalized physical deconditioning should be evaluated and treated.

Therapeutic approaches include physical rehabilitation (i.e., physical and occupational therapy), psychological care including cognitive behavioral therapy (CBT), relaxation training, medication

management, and a variety of techniques that, directly or indirectly, are aimed at blocking or interrupting chronic changes to an overactive nervous system and in some cases decreasing sympathetic hyperactivity. Patients are encouraged to use the affected limb. Treatment is more successful if started early rather than later in the disease process.

While physicians and therapists have many tools in their treatment armamentarium, the single most important treatment for these patients is education and learning how to manage their chronic pain condition. Patients who can learn about the cause and meaning of their pain are able to make better choices regarding the use of their extremity which may improve the natural history of the disease process.

Medication Management

Medications may include treatment with oral, transdermal, topical agents, drug delivery patches, parenteral infusions, and injections. This may include steroids, anti-inflammatories, antidepressants, vasodilators, anti-spasm medications, and anticonvulsants (i.e., gabapentin) type medications. The use of opioids is frowned upon, but they still may have a place in recalcitrant cases. No single oral medication or injection is specifically approved by the FDA (Food and Drug Administration) for CRPS. There is no curative therapy and no single medication or combination of medications is proven to modify the disease.

Interventional Procedures

Usual procedures employed in diagnosis and treatment typically include stellate ganglion along with thoracic and lumbar sympathetic block. Sympathetic blockade may provide a useful adjunct to aggressive medical therapy, but it should not be considered as a sensitive or specific test for the diagnosis. Some physicians advocate lidocaine infusions or use of intravenous ketamine.

Each of these procedures is designed to alter the function of the nervous system temporarily. During this temporary alteration, patients are evaluated to see if pain, function and evidence of sympathetic dysfunction have been positively influenced. Frequently, patients will receive several of the above procedures as a trial to determine which, if any of them should be integrated into the multidisciplinary management plan.

The goal of each of these techniques is to provide a temporary but effective pause in neuronal hyperactivity, a contributor to the pain, thereby allowing the rehabilitative care to restore more normal healing and function to the affected tissues. If following a trial of the different procedures mentioned above, the treating physician feels that a significant benefit has been gained, then repeated administration of these procedures over a period of six to twelve weeks (and in some cases longer) is viewed as appropriate.

The desired outcome from each procedure should be as long a period of effective decreased pain and improved functional capacity as possible. The majority of practitioners recognizes a response profile of several days to a week as being optimal for the initial intensive treatment and would be willing to repeat these blocks on a weekly or perhaps twice weekly basis in order to facilitate the rehabilitation process. One should recognize, however, that sympathetic blocks on their own infrequently, if ever, “cure” patients.

These blocks and procedures should be viewed as any other medical treatment; an effective form of temporary palliation and a useful tool to help the patient with the remainder of the multidisciplinary management provided by physical rehabilitation and psychological services.

Patients may have a tendency to view the medical components of treatment as curative alone and it is the job of the therapy team to reinforce the rehabilitative and psychological components as being critical, while the medical interventions are primarily palliative. This continued de-emphasis of the medical components of treatment will help to prevent patients from viewing the locus of control with regards to their ongoing improvement in this disease as being outside of themselves or within physician control. In other words, the patient should be responsible and in charge of their rehabilitation and pain management.

More aggressive medical strategies employed include the use of spinal cord stimulation (SCS) or peripheral nerve stimulation (PNS).

There have been documented cases of clear beneficial effects from spinal cord stimulation (SCS) wherein small electrodes are placed in the epidural space outside the spinal cord to deliver microelectrical currents to the descending portions of the spinal cord. These currents induce activity in the patient's own intrinsic pain modulating system. Before permanent implantation, a trial period is warranted. Permanent implantation should depend on objective evidence of benefit including improvement in functional capacity and in the overall rehabilitation program in order to justify the risk and expense associated with chronic implantation. Additionally, these modalities should be utilized only by physicians experienced with these techniques in a multidisciplinary setting. Patients who receive these therapies should be selected by careful medical and psychological screening.

All medical therapies whether conservative or spinal cord stimulation need to be presented to the patient and reinforced as approaches that are used to provide a "window of opportunity" for functional restoration therapy where they can be aggressively and intensively rehabilitated.

Education

An education program is important for the patient so her or she can understand what has happened and what can be done about it. Since treatment often involves a 24-hour a day, seven days a week effort, the patient must be empowered to be able to provide self-treatment and gain confidence.

Physical Rehabilitation

The ultimate goal of therapy is to reduce pain and improve function of the patient's affected area. Physical rehabilitation can be detrimental if not applied appropriately.

Many therapists still limit treatment from concerns regarding causing further injury to the affected extremity due to the often-reported high levels of pain, color and swelling changes with use and movement.

This section discusses a variety of treatment modalities used in physical and occupational therapy. Treatment modalities can be passive or active, but overall, the direction of treatment should be toward individual self-management.

The physical therapy evaluation starts with an assessment of appearance along with active and passive range of motion and measurement of swelling. A related soft tissues assessment, including that for myofascial trigger points, should also be included. The therapist also evaluates strength, sensation and pain response, coordination, dexterity, temperature changes and functional use ability.

Passive treatments include splinting, paraffin, massage, electrical stimulation, ultrasound, contrast baths and edema control treatments.

Active treatments include various desensitization techniques, active exercise or functional use, stress loading (scrubbing and carrying), normalizing compensatory movement patterns, and flare management techniques including pacing, cognitive behavioral therapy and relaxation techniques.

Active treatments are essential in all cases and the benefits of passive treatments are person specific. A functional restoration program (FRP) combines these active treatments with psychological care and medical/medication management.

Treatment is directed toward pain relief, desensitization, edema reduction, normalization of tone and sensation, proper posturing and positioning, range of motion and stretching to maintain and improve flexibility, stress loading, and strengthening. In more severe cases, splinting and bracing may be utilized. Prolonged splinting or bracing should be avoided and may contribute to development of other compensatory problems.

Treatment is a team effort with adequate analgesia provided. Treatment in severe cases usually starts slowly with edema relieving techniques, gentle desensitization and the use of passive modalities followed by gentle flexibility and strengthening exercises. As the patient improves, treatment consists of more aggressive range-of-motion exercises, stress loading, strengthening, and general aerobic conditioning.

While the importance of maximizing functional use of the affected limb cannot be overstated, it is just as important to realize that some individuals with residual symptoms of CRPS, will need to learn proper pacing of activities and avoidance of pain inciting events. The individual will need to learn skills to perform some functions in an alternate and less symptom provoking manner. Additional skills such as diaphragmatic breathing, relaxation techniques, imagery, and special mind-body exercises such as Tai Chi or Feldenkrais may be beneficial.

Normalization of use and functional rehabilitation comprise the final stages of therapy. This stage may include work hardening, vocational rehabilitation or retraining, and workplace modification. Patients may need weeks to several months to progress through this stage.

Fear & Avoidance Therapy

Fear of re-injury, fear of movement (kinesiophobia) and avoidance due to increased pain levels is a common barrier in returning to normal life, work or recreational activities after an injury. Driven by fear of further pain or the threat of further damage, many people with CRPS increasingly restrict activities and begin to exhibit a maladaptive avoidance response.

One of the first steps is education on the detrimental effects of guarding and disuse and the importance of movement in treatment of CRPS.

Flare Management

Flare-ups (an increase from the normal baseline level of pain) can cause both physical and emotional reactions. Physical reactions include guarding the limb, avoiding activity, tightening of muscles, holding the breath, stomach and chest tightening and nausea. When these physical reactions occur, the pain level worsens and propagates the cycle of flare-ups.

Flare-up management consists of learning a new set of active tools that can assist people with CRPS to feel more in control of their symptoms and be able to push themselves harder to meet their physical goals. There are multiple physical and cognitive tools that can be effective for patient with CRPS. These tools overlap widely and both physical and cognitive tools shoulder be used in order to allow the best flare management success.

Flare management tools include but are not limited to relaxation breathing with focus on decreasing guarding, light movement, yoga, tai chi, mindfulness-based stress reduction, cognitive behavior therapy practice, distraction, guided imagery, positive self-talk and pacing. Breathing and relaxation techniques are the foundations of active pain management skills and are taught early in treatment. Instruction on correct breathing and relaxation is imperative as the patient begins the painful rehabilitative process.

Pacing

Pacing is used as a flare management tool, but also as a way of performing activities during the day. Many people believe that pacing means being less productive, but in reality, the overall goal is to best manage the symptoms of CRPS and to become as productive as possible. Learning how to pace can be very frustrating as there are many ways to pace tasks and there are multiple non-physical barriers including old ways of doing things, thoughts of what someone should be able to do and pushing to be able to meet others' expectations. Many people with CRPS become very frustrated due to a drastically different tolerance level and inability to perform even the simplest of activities due to high levels of pain.

Aerobic Conditioning, Strengthening and Stretching

These approaches vary widely but are an important part of CRPS treatment. They are all geared initially to physical reactivation and use of the limb to the extent possible and within reason but provide considerable value to non-affected other body parts. Physical activity not only increases general health

but appears to provide pain reduction benefits possibly through endorphin release but also serves to utilize time and keep the individual occupied while having the potential for socialization in a group setting (i.e., walking, at a fitness center, etc.). An exercise program focused on the non-affected areas can provide both endorphin release, but also a way to pace the exercise program and give the affected areas a break, while still staying active.

Functional Activities

Functional activity training consists of activities that increase the ability to use the affected extremity in daily, work or recreational activities. These can include activities of daily living such as grooming or dressing, household activities such as cooking or cleaning or activities such as driving or grocery shopping. These tasks may be avoided completely or may be completed by compensating in different ways or performed on a modified basis with difficulty. In CRPS of an upper extremity, functional activities with the affected extremity can become minimal to non-existent and often the non-affected extremity is over-used. In CRPS of the lower extremity, any task that requires weight-bearing through that extremity is typically avoided or a compensatory movement is performed.

Desensitization

Symptoms of CRPS commonly include increased sensitivity to both noxious and non-noxious stimuli. Desensitization is simply finding ways to decrease or de-sensitize the over-excited somatic pain response. Desensitization techniques are aimed at normalizing this over-active response with a variety of different sensations.

Contrast Baths

Contrast Baths are the immersion of a body part alternately in cold and hot water. Contrast Baths can be used for different purposes in treatment of CRPS. Many people find relief from contrast baths and use them as a flare management tool. In these cases, the temperatures are kept at comfortable ranges. Others use contrast baths for desensitization as the temperature changes can be quite painful for the affected extremity. In this case, the water should be at a temperature that is just outside of the comfortable range. Even when used for desensitization, very cold water is not usually recommended if the limb is typically cold.

Paraffin

Paraffin can be used as a warm wax bath in which the body part is dipped into multiple times and then covered with a plastic bag and towel or covering like an oven mitt. While some people with CRPS cannot tolerate the feel of the wax on their skin or the warm temperature, many others find great relief in the warming properties. In the latter case, it is recommended to use paraffin before exercising the limb to allow for easier movement. Many people use paraffin as a way to control joint swelling or to help decrease contractures.

Electrical Stimulation

Electrical Stimulation is a common tool, although clinical experience suggests that patients either find good benefit from the tool from a flare management perspective or are highly flared due to hypersensitivity, even at a proximal site.

Splinting

Splinting is often discussed as a treatment option in CRPS, although this tool is limited in its actual clinical use. Splinting can be used for protection or guarding or to prevent or reduce contractures. Using this treatment for protection or guarding should only be done in the early stages only and on a limited basis, as movement should be encouraged, not restricted.

Edema Management

Edema is a common sign of CRPS and can vary greatly between people diagnosed with this disease. Edema in the early stages of CRPS should be addressed with edema management garments (such as Isotoner gloves, Jobst garments, or Coban wrap) and active range of motion. Self- retrograde massage can be used if tolerated but should not be performed by the therapist unless there is a strong trust that the practitioner will stop treatment if the patient can no longer tolerate it.

In the later stages of CRPS, changing levels of edema are common with physical activity and especially during times of flare-ups. This edema often recedes to its normal level after the activity or when the flare-up calms down. If this is the case, the edema should not be a limiting factor when performing physical activity.

Stress Loading: Scrubbing & Carrying

The stress loading protocol is a widely used rehabilitation tool in treatment and management of CRPS. The protocol involves stressful use of the affected extremity with minimal joint range of motion. Stress loading is comprised of two components: scrubbing and carrying. Each activity engages the affected extremity in consistent weight bearing activities within a small range of movement for gradually increasing periods of time. Scrubbing consists of applying a constant force through the affected area while the limb is moved back and forth as if scrubbing the floor.

Guarding/Postural Re-training

Due to the high levels of pain in CRPS, guarding and weight-bearing avoidance are common and are a typical initial reaction to the onset of symptoms. For an upper extremity, the limb is often held close to the body in an internally rotated and adducted position with the elbow and fingers flexed and the shoulder girdle elevated. For a lower extremity, weight-bearing is avoided as much as possible, even in the sitting position. Keeping the limb in a guarded position can be calming and allows the limb to feel protected. It can also keep the limb from being accidentally bumped or jostled. Unfortunately, this guarded positioning

not only leads to avoidance of use of the limb but can also cause other musculoskeletal secondary issues such as muscle length changes and joint or soft tissue contractures.

Postural training and positioning can minimize protective guarding, promote balance and facilitate improved functional use of the extremity. Postural instruction and exercise assist in placing the affected extremity in a correct position to facilitate normal movement patterns and proper muscle retraining.

Gait training is another important part of postural retraining for an affected lower extremity. Whether weight-bearing through the limb is entirely avoided or the weight is placed on only one part of the foot, gait, or the walking mechanics, become highly irregular. A normal gait pattern is typically painful and therefore avoided with multiple compensations. This can lead to patterns of weakness in the low back, hip, knee and ankle musculature. Gait training begins by determining not only what areas of the foot are being avoided, but also the tolerance to weight-bearing through these areas. Subtle changes in the weight-bearing through the foot are encouraged and complemented with weight-bearing exercises. Depending on the tolerance, weight-bearing exercises may first need to be performed in a more controlled setting, such as standing, before the gait pattern can be altered.

Brain Retraining Techniques

Although the pathophysiology of CRPS is not well understood, peripheral and central changes have been observed and altered central representation of perceptual, motor, and autonomic systems have been implicated.

A brain-focused motor and sensory exercise program can help re-develop healthy nerve connections and brain organization. Certain pathways in the brain are activated when the brain needs to recognize a body part (sensory) and before and during a movement of that body part (motor). The goal of these treatments is to reorganize the brain and its pathways to diminish pain and sensitivity.

Graded motor imagery is a set of rehabilitation processes used to treat pain and movement problems related to an altered nervous system. The graded motor imagery program for patients with CRPS consists of limb laterality training, imagined hand movements and then mirror box therapy, in that order.

Conclusions Regarding Physical Restorative Therapies

Information about physical rehabilitation techniques for people with CRPS continues to grow and develop, although further research studies are needed to determine efficacy in both early and late CRPS. A trained therapist can be helpful in directing the rehabilitation efforts and determining the plan for using the above-mentioned treatment tools. Overall, an active and patient-directed approach is important along with setting realistic short- and long-term goals.

Psychological Treatment

Patients with chronic pain problems benefit from psychological services offered in conjunction with physical rehabilitation and medical management techniques. Regardless of the individual's prior psychosocial history, it is common to struggle emotionally when dealing with chronic illness and pain.

Psychological services may include counseling for the patient and significant others, as well as a variety of techniques for pain control and reduction. This can include cognitive behavioral and acceptance and mindfulness-based interventions, biofeedback, stress reduction, meditation, relaxation training and hypnosis. Services should be time limited, goal oriented, and coordinated as part of a multidisciplinary or interdisciplinary treatment approach.

"Multidisciplinary" approaches include treatment directed by one clinician with multiple disciplines included such as physical and occupational therapy, pain psychology, relaxation therapy, medical management, vocational rehabilitation, and nursing education. Multidisciplinary treatment plans commonly use disciplines at different sites. In contrast, an "interdisciplinary" approach may utilize the same disciplines as mentioned above but is more collaborative and structured. Care is delivered in one facility, where therapists can better communicate and adjust care. These programs are usually structured, outpatient, day programs, multiple hours per week, for weeks at a time and include both individual and group therapies.

Functional Restoration & Multi- and Interdisciplinary Pain Management

The most effective approach for many CRPS patients involves a functional restoration multidisciplinary or interdisciplinary chronic pain program. These programs are cost-effective and involve an individualized, but highly structured, medication optimization, behavioral/psychological rehabilitation and physical conditioning program in a group setting.

Individuals engage in stretching, strengthening, aerobic conditioning and desensitization techniques, while learning behavioral and psychological approaches to better manage pain along with educational activities and work simulation.

Dependency on the doctor and therapist is discouraged and the program is geared towards healthy behaviors and return to leisure and work activities. The group setting provides friendship among patients and encourages mutual support.

HEADACHES: EVALUATION & TREATMENT

INTRODUCTION

Headache is ubiquitous in society and one of the most common presenting complaints to physicians. It is a major cause of decreased productivity and sickness absence from work. Greater than two-thirds of people suffer from at least one headache a year, and chronic recurrent headaches are experienced in 36% of women and 19% of men. Headaches can be debilitating, and frequently cause alarm and fear, but they are rarely (.004%) a symptom of underlying serious disease. There is no consensus on what constitutes ideal headache management. Various conventional and alternative health care practitioners and self-help remedies share the burden of treatment.

HEADACHE DIAGNOSIS

The diagnosis of headache by a doctor, in contrast to many other medical conditions, depends almost entirely on the medical history, as objective physical findings are rarely present. The astute physician will best be able to help the headache sufferer by taking a clear history and thus be able to distinguish some sort of headache pattern. Important questions include age of onset, frequency and duration of each headache, location, and quality, time of onset, associated phenomena, and aggravating or relieving factors.

On physical examination, the physician is interested in first getting a general sense of whether the patient is mentally alert and looks well or ill overall, followed by a thorough, general and neurological examination. Because there is no way to measure the quality and severity of another person's headache, it is important for the physician to also gauge the nature and extent of psychological and social factors which may be impacting on the patient's presentation and perception of pain. Medications and other therapies will be of little benefit if the headache is used as a means to get attention, drugs or avoid working.

TESTS

An accurate medical history usually suffices in the majority of patients, but when there is diagnostic difficulty or a suggestion of a serious illness, further testing becomes mandatory. These can include blood tests, lumbar puncture, EEG, radiologic studies (x-rays, CT, MRI), and radioisotope scanning. The actual differential diagnosis can be quite extensive.

OMINOUS WARNING SIGNALS

The sudden onset of a new severe headache that appears "out of the blue" may suggest a hemorrhage (subarachnoid) or meningitis. A progressively worsening headache may be a danger signal of increased intracranial pressure on the brain (brain tumors, subdural hematoma). Headaches brought on or worsened by exertion (straining, coughing, sexual activity) may indicate a vascular headache, but also may indicate a hemorrhage (subarachnoid) or increased intracranial pressure. Another warning signal are associated

symptoms, such as drowsiness or confusion, a change in neurologic findings, or medical illness such as fever, chills, or diffuse aching pains. Since most headaches start before middle age, the onset of a first headache after age 50 should be investigated further until proven benign. Finally, an abnormality on physical examination is uncommon with most headaches, and further consideration is therefore warranted.

TENSION-TYPE HEADACHES

Tension-type headaches usually start episodically, associated with stress, fatigue, or such activities as prolonged reading, but they may progress into a chronic condition. They represent the great majority (about 90%) of all headaches. Women represent 75% of tension-type headache sufferers, and 40% of patients have a family history of headaches. These headaches are usually described as dull, diffuse, and persistent, while varying in intensity throughout the day. Patients often describe a feeling of pressure, tightness or heaviness in a band around the head. About 10% of these individuals also have migraine, which confuses the presentation.

Tension-type headaches can occur related to expected or actual stressors, or unpleasant events. The headaches may last hours or days and are considered episodic rather than chronic if they occur less than fifteen times in a month.

Depression and anxiety are commonly found associated with tension-type headaches. The cause of these headaches is unclear, but there may be a relationship to psychological stress or excessive muscle contraction. Treatment includes a combination of psychological, physical, and medication approaches. Stress reduction and relaxation techniques, meditation, biofeedback, stretching, exercise, and conditioning can be helpful. Effective medications can include simple, over-the-counter analgesics and prescription anti-depressants.

MIGRAINE HEADACHES

Migraine, although common, is still poorly understood, with treatment often depending upon trial and error. It is not a single entity and has two major and multiple less common variants. Migraine is present in 12-15% of adults and 2-3 times more common in women than men. Migraine predominantly affects adults below the age of 40.

The two major variants include migraine without (approximately two-thirds) and with (one-third) aura (flashing lights, shimmering zigzagging lines, and areas of lost vision). Migraine sufferers typically go through four distinct phases that blend together during an attack. Up to 50% of individuals suffer from some sort of premonitory symptoms or prodrome. Symptoms develop slowly and precede the migraine attack and can include heightened or dulled perception, sudden mood changes, irritability or withdrawal, cravings for certain foods (particularly sweets), excessive yawning, or speech difficulties. These symptoms may not be marked or even realized by the patient. Phase 2, or the aura, include visual disturbances, unilateral weakness and/or pins and needles in the hands, and difficulty speaking. The aura usually proceeds the headache by 60 minutes or less.

Phase 3 is the headache itself. It is usually severe, pulsatile, and typically on one side of the head only, but it can be on both sides. The headache is frequently aggravated by routine physical activity and frequently accompanied by nausea and vomiting or intolerance to noise or light and is aggravated by movement. The migraine sufferer typically, therefore, prefers a quiet, dark room to rest during the headache, which typically lasts between 2 to 72 hours.

Phase 4 is the postsyndrome, lasting up to 24 hours, after the headache has subsided, during which the individuals feels drained or washed-out, with tired achy muscles. Some patients may actually experience a period of euphoria after the headache has disappeared.

The cause of migraine remains a mystery, but there does appear to be a strong genetic influence. There appears to be a relationship between cerebral blood flow and migraine, with the symptoms of migraine a result of constriction of the cranial blood vessels and the headache then caused by subsequent blood vessel enlargement or dilatation. Scientists now believe, though, that there is not a single factor that can be blamed for migraines, but rather a complex interplay of many biochemical and neurologic changes, which interact to trigger and sustain the headache.

Migraine is characterized by attacks separated by symptom-free intervals. The physical and neurologic examination is normal. Migraine therapy can be divided into acute treatment of the attack and preventive treatment. Precipitating factors (triggers) should be avoided. Acute treatment includes over-the-counter analgesics and stronger prescription pain medication. Abortive medications are best used early in a migraine attack. At present the most effective drugs for interrupting a migraine attack are the triptans. All the acute treatments for migraine are more effective if followed by a short rest period or a nap.

Migraine trigger avoidance strategies are important. Factors commonly associated with triggering migraine attacks include loud noise, strong smells, flashing or fluorescent lights, missing or delayed meals, mental stress, changes in sleep patterns, menstruation and certain foods such as citrus fruits, cheese, chocolate, and alcohol.

For frequent migraine sufferers, prophylactic medication can be useful to reduce the frequency of attacks. The most commonly used drugs are NSAIDs, the beta-blockers (e.g. Propranolol), the calcium channel antagonists (e.g. Verapamil), and anti-depressant medications (e.g. Amitriptyline). Side effects can be a problem from these medications.

Botulinum toxin injections can be effective for migraine headaches.

Among the non-drug treatments, behavioral therapies such as relaxation and biofeedback can be effective. Some reports have stated that acupuncture is effective in certain cases.

The exact cause of migraine is still unknown but appears to be related to both vascular and neurogenic components. Migraine symptoms in many individuals cease or diminish into late adult life and after menopause in women.

CLUSTER HEADACHES

These less common headaches (0.5% of males and less than 0.1% of females) tend to be devastatingly severe and lead to progressive emotional and physical decompensation. They typically begin in the 20 to 40 age range and do not have a genetic predisposition.

Cluster headaches are characterized by excruciating pain that lasts 15-90 minutes, located behind or around one eye, although pain can radiate to the temple, jaw, nose, chin, and teeth. There is often tearing on the same side, eye redness (conjunctival injection), and nasal congestion. The eyelid may droop, the pupil may decrease in size, there may be sweating and facial flushing. In contrast to migraine, there is no aura, no visual or sensory symptoms, and nausea and vomiting are unusual.

Attacks occur during specific periods or "clusters" which last from 2-12 weeks, more commonly occurring in the spring or autumn, separated by remissions lasting at least 14 days but usually several months.

While in a cluster period, the patient may suffer one or more attacks daily, often at the same time of day.

Attacks regularly begin during sleep but can occur during the day. Cluster headaches develop into a crescendo and are so severe that sufferers are unable to lie down and remain still and typically pace about. Cluster headaches may be provoked by external stimuli, such as alcohol, cold or heat air blowing onto the face, excitement, sleep, and vasodilators found in foods and medications.

Treatment with 100% oxygen inhalation can be effective for an acute attack within 10-15 minutes in 60-70% of cases. Injection of sumatriptan or dihydroergotamine is presently the most effective treatment of cluster headaches. Other drugs used in prophylactic treatment include methysergide, methylergonovine, ergonovine maleate, lithium, and calcium antagonists.

The cause of cluster headaches is unclear but appears to involve a mixture of vascular and neurogenic mechanisms possibly related to a vulnerable carotid artery affected by neural influences. Cluster headaches, like other vascular headaches, tend to decrease in frequency and intensity as the individual gets older.

POST-TRAUMATIC HEADACHE / POST-CONCUSSION SYNDROME

This syndrome is found post mild to moderate closed head injury and also after flexion-extension (whiplash) injuries. The primary symptoms, usually strikingly consistent from patient to patient, include one or more of the following: 1) headache, neck and shoulder pain; 2) sleep disturbance; 3) cognitive abnormalities; 4) mood and personality changes; and 5) dizziness.

Post-traumatic headache is a common complaint in individuals after head trauma. In most cases the patient notes the onset of headache within twenty-four hours of injury, but in some cases, it may not be apparent for several days to weeks. The headache usually consists of a bilateral pressure-like sensation located in the back or front of the head, although it can be one-sided. While most patients complain of a constant

headache, it can also wax and wane in intensity and severity. Exercise, bending over, coughing or rapid movement of the head usually increases complaints. Symptoms decrease with rest, relaxation and sleep. Patients may complain of migraine-like symptoms such as nausea and vomiting plus light sensitivity (photophobia). A brain injury can be the initiating factor for chronic migraine and cluster headaches.

Headaches represent the most common symptoms in patients after a closed head injury and usually persist for more than two months in 60% of patients. There does not appear to be any correlation with the degree of injury or period of unconsciousness. With a usual lack of "objective" findings, there is occasional skepticism regarding the validity of the injured workers complaints, but malingering is rare, and the majority of patients have truly valid symptoms. When there is cognitive dysfunction, treatment includes reassurance, cognitive retraining, psychosocial readjustment, communication, leisure skills training, physical rehabilitation and vocational counseling/rehabilitation. Specific treatment for post-traumatic headaches can include appropriate education, medications, biofeedback, TENS, acupuncture, stress management training along with physical rehabilitation and conditioning.

DRUG REBOUND HEADACHES

Some drugs commonly used in the treatment of headaches, particularly if used improperly or chronically, may actually cause headaches. These can include analgesics like aspirin, acetaminophen, benzodiazepines, barbiturates, various opioids (although they should mostly be avoided in treating headaches) or ergotamine (Cafergot). Headache analgesic abuse is much more common in females, and the majority of abusers are 30-50 years of age. While it is more common in migraine sufferers, daily drug use ("abuse") can also be found in individuals with tension-type headaches.

A drug rebound headache typically is present on waking and varies in intensity but lasts throughout the day. It is often described as mild to moderate, dull, bilateral, frontal-occipital, or diffuse. Migraine attacks may be superimposed upon the drug rebound headache. Rebound headache sufferers typically self-medicate throughout the day, and any pain relief is usually transient and rarely complete.

Treatment involves rapid withdrawal of non-opioid medications (opioids should be withdrawn slowly secondary to problems with dependence). Inpatient hospital care may be needed to deal with withdrawal symptoms, both physical and emotional. Upon drug discontinuance, a severe headache is to be expected, lasting 3-6 days.

Once withdrawal is complete, a thorough headache reassessment is appropriate, along with counseling regarding future medication use. Prophylactic medications are useful in decreasing any rebound headache effects and are found to be most effective for the underlying headache phenomenon once the drug rebound agents are discontinued. Non-medical treatments, such as relaxation, biofeedback, and meditation, should be encouraged.

SUMMARY

Headache is the most common pain complaint and one of the most common presenting complaints to physicians. It is a major cause of decreased productivity and absence from work. Headaches can be debilitating, and frequently cause alarm and fear, but they are rarely a symptom of underlying serious disease. The diagnosis of headache, in contrast to many other medical conditions, depends almost entirely on the medical history, as objective physical findings are rarely present. It is important to consider psychological and social issues as well.

Certain warning signals such as the sudden onset of a new severe headache, a progressively worsening headache and headaches brought on or increased by exertion deserve immediate medical attention. Symptoms such as drowsiness or confusion, a change in neurologic findings or medical illness such as fever, chills, or diffuse aching pains warrants further attention.

Tension-type headaches are the most common (90%) and usually start episodically, associated with stress, fatigue, or such activities as prolonged reading, but they may progress into a chronic condition. Depression and anxiety are commonly found associated with tension-type headaches. The cause of these headaches is unclear, but there may be a relationship to psychological stress or excessive muscle contraction. Treatment includes a combination of psychological, physical, and medication approaches.

Migraine headaches typically start with premonitory symptoms or a prodrome (sudden mood changes, cravings for certain foods, excessive yawning, or speech difficulties). This stage is followed by the aura (visual disturbances, unilateral weakness and/or pins and needles in the hands) followed by the headache itself. It is usually severe, pulsatile, and typically on one side of the head only, but it can be on both sides. The headache is frequently aggravated by routine physical activity and frequently accompanied by nausea and vomiting or intolerance to noise or light and is aggravated by movement. There is a period after the headache has subsided, during which the individual feels drained or washed-out sometimes followed by a period of euphoria.

Migraine treatment includes avoidance of triggers plus the use of over-the-counter analgesics, triptans, and, when appropriate, stronger prescription pain medication. A vasoconstrictor may be useful. Behavioral therapies are recommended. Botulinum toxin may be effective in selected cases.

Cluster headaches are rare but tend to be severe and lead to progressive emotional and physical decompensation. Drug rebound headaches may be secondary to the very drugs used to treat headache and should be suspected in individuals with daily and increasing drug use ("abuse"). Inpatient hospital care may be needed to deal with withdrawal symptoms, both physical and emotional.

In the post-traumatic headache/post-concussion syndrome symptoms include headache, neck and shoulder pain; sleep disturbance; cognitive abnormalities; mood and personality changes; and dizziness. Post-traumatic headache is a common complaint in individuals after head trauma. When there is cognitive dysfunction, treatment includes reassurance, cognitive retraining, psychosocial readjustment, communication, leisure skills training, physical rehabilitation and vocational counseling/rehabilitation.

Specific treatment for post-traumatic headaches can include appropriate education, medications, biofeedback, TENS, acupuncture, stress management training along with physical rehabilitation and conditioning.