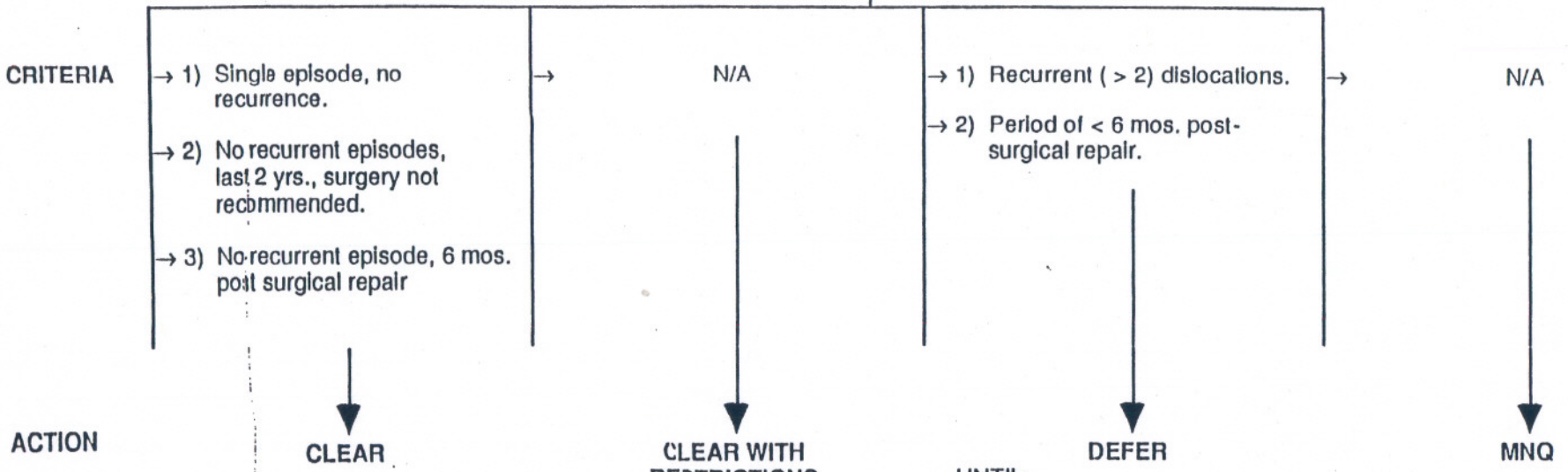
DISLOCATION OF SHOULDER (831); RECURRENT (718.31)



MEDICAL INFORMATION NEEDED: Generic Information;

Orthopedist evaluation if recurrent.

5/4/93

SHOULDER: ROTATOR CUFF INJURY (726.1) AND REPAIR (83.63) IMPINGEMENT SYNDROME AND REPAIR (726.2)

CRITERIA	→ 1) Single episode, asymptomatic 1 yr. → 2) Surgical repair 6 mos. post, no disability. → 3) Recurrent symptoms of impingement, activity related and avoidable. → 4) Surgical repair of either cuff or impingement with residual stable impairment.	→ N/A	→ 1) Symptomatic with ADLs. → 2) Surgery advised, or < 6 mos. post surgery.	→ N/A
ACTION	**CLEAR**	**CLEAR WITH RESTRICTIONS**	**DEFER**	**MNQ**
RESTRICT-IONS/DEFER			UNTIL: 1) Meets "Clear" criteria. 2) Post surgery 6 mos.	
RATIONALE				

MEDICAL INFORMATION NEEDED: Generic Information;

Orthopedist evaluation for recurrent or chronic.

5/4/93

BACK PAIN (847); MUSCLE STRAIN (847); SPRAIN (847); SCOLIOSIS (737.43); NECK PAIN (723.1) (excludes radiculopathy, osteoarthritis, or any more specific diagnosis)

CRITERIA	→ Single episode resolved occasional mild episodes relieved with non-narcotic analgesic; no Hx radicular involvement (pain below knee, numbness or tingling see ORTHO-16) (app. verbal Hx OK).	→ N/A	→ Frequent and/or severe episodes	→ N/A
ACTION	**CLEAR**	**CLEAR WITH RESTRICTIONS**	**DEFER**	**MNQ**
RESTRICT-IONS/DEFER			UNTIL: Orthopedic Evaluation. Until < 150% of ideal body weight and meets "Clear criteria". Successfully managed (exercise, wt. loss, etc.). Physical abilities letter.	
RATIONALE	Acute low back pain is usually due to muscle strain, tear, or sprain. These tend to resolve, but can become chronic.		* See weight guideline	

MEDICAL INFORMATION NEEDED: Generic information;

R/O Hx radicular involvement if any symptoms within 4 years (see ORTHO-16 if any signs or symptoms of radicular involovement)

7/17/95

DEGENERATIVE DISC DISEASE

Includes Herniated, Prolapsed, and Ruptured Inter Vertebral Discs.
Includes Micordiscectomy, Disectomy, Laminectomy, and Disc Fusion.
Includes Spinal Stenosis.

INFORMATION REQUIRED *Any history*

All Applicants:

- Report of Medical Examination to include the following:
  - Date of diagnosis
  - Description of symptoms at time of diagnosis
  - Current status to include description of current symptoms
  - Treatment history to include medications, physical therapy, surgery, etc For medications, include past and current use.
  - History of recurrence(s)
  - Limitations or restrictions of ADLs
  - Recommendations for follow-up over the next 3 years
- Specialist Evaluation (Orthopedist or Neurologist) if initial diagnosis, symptoms, or surgery *within* the past 3 years; to include the information above.

If Applicable:

- If done, copy of MRI report and/or other radiographic or diagnostic studies
- Discharge summary for all related surgeries and hospitalizations.

CLEARANCE CRITERIA	REVIEWER	GUIDANCE
1. History of prolapsed, herniated, or ruptured inter vertebral disc; includes lumbar, thoracic, and cervical discs. 2. No symptoms, or effective management of *minor* symptoms, for *at least* the past 1 year. 3. Symptoms, past or current, do not include night pain, pain at rest, or other constitutional symptoms, e.g., fever, chills, weight loss. 4. Symptom management does not require the use of muscle relaxants, e.g., Flexeril, Robaxin, or the use of narcotics. 5. No residual neurologic deficits or dysfunction, e.g., paresthesias or radiculopathies. 6. History does not include multiple injuries or symptom recurrences. 7. Fully functional, i.e., no limitations or restrictions of ADLs. 8. If treatment includes mirodicsectomy, discectomy, or laminectomy, post surgery *greater than* 1 year. 9. If treatment includes disc reconstruction or internal fixation, post surgery *greater than* 2 years. 10. If treatment includes lumbar fusion, post surgery *greater than* 3 years. 11. No associated spinal stenosis.		
Meets clearance criteria 1 - 11, AND • If *minor* symptoms present, effective management of symptoms requires no, or intermittent, use of over-the-counter pain medication, e.g., aspirin, acetaminophen, NSAIDs, etc.	**RN**	**CLEAR**
Does not meet clearance criteria due to one or more of the following: • Ineffective management of *minor* symptoms during the past 1 year. • Status post micordicsectomy, discectomy, or laminectomy *less than* 1 year. • Status post disc reconstruction or internal fixation, *less than* 2 years. • Status post lumbar fusion *less than* 3 years.	**RN**	**DEFER**

(continued on next page)

Does not meet clearance criteria due to one or more of the following: • Effective management of symptoms requires *daily use* of over-the-counter pain medication, e.g., aspirin, acetaminophen, NSAIDs, etc. • Symptom management requires the use of muscle relaxants, e.g., Flexeril, Robaxin, or the use of narcotics. • Residual neurologic deficits or dysfunction, e.g., paresthesias or radiculopathies. • History includes multiple injuries or symptom recurrences. • Not fully functional, i.e., limitations or restrictions of ADLs. • Associated spinal stenosis.	**MED ADVISOR**	Risk varies - assess based on detailed history.
Does not meet clearance criteria due to one or more of the following: • Symptoms, past or current, include night pain, pain at rest, or other constitutional symptoms, e.g., fever, chills, weight loss.	**MED ADVISOR**	**DEFER/MNQ** Deferral/MNQ letter requires review by screening manager.

DIAGNOSTIC CODES

722.2 Herniated, Ruptured or Prolapsed Inter Vertebral Disc

Cross Reference ICD.9 CM

NOTES AND INSTRUCTIONS FOR REVIEWERS

Reviewers to Consider:

- NA

COMMENTS

Goetz: Textbook of Clinical Neurology, 2nd ed., Copyright © 2003 Elsevier

Epidemiology and Risk Factors: Vertebral disc degenerative changes are a universal accompaniment of aging. Teenagers rarely develop symptomatic disc herniation. The peak incidence of symptoms occurs between the ages of 30 and 50. Patients often describe the onset of low back pain, usually remittent and without specific features in their twenties, perhaps after identifiable trauma, and the onset of more specific symptoms that leads to the diagnosis of disc herniation is often not preceded by further trauma. Probably the accumulation of degenerative changes to the annulus and the preservation of the expansile gelatinous nucleus, overlapping with a period of life when job and sports-related activities increase the amount of mechanical stress on the body, account for this peak in the incidence of disease. The incidence then falls off in the older population, probably due to the lack of mobility of the desiccated disc and the relative lack of physical activity. Women and men are affected approximately equally.

There is a tendency toward disc herniation in some families, such as those with congenital spinal anomalies, including fused and malformed vertebrae and lumbar spinal stenosis due to short pedicles. Patients with increased weight and tall stature are at increased risk for this condition. Also, acquired spinal disorders, such as common degenerative arthritis and ankylosing spondylitis, predispose to disc degeneration. Various behaviors that increase risk include sedentary occupations, physical inactivity, motor vehicle use, vibration, and smoking. In younger women, pregnancy and delivery are associated with lumbosacral herniation, and new symptoms of cervical disc herniation may occur in part because of the bending and lifting involved in child rearing.

Clinical Features and Associated Disorders: The most common site of disc herniation in the cervical region is the C6–C7 level, followed by C5–C6, C7–T1, and C4–C5. Patients typically develop some local pain in the neck that radiates to the shoulders or the interscapular region. In the most common lateral herniations, radicular symptoms ensue. These symptoms include pain in the shoulder and arm, which may follow a dermatomal pattern but more typically is deep and aching and only roughly corresponds to the involved dermatome. At the cervical levels, the roots emerge laterally to exit through the neural foramina *above* the correspondingly numbered vertebral bodies. Because the spinal cord and bony vertebral levels are roughly aligned in the neck, the level of herniation corresponds to the level of root irritation. Hence, C6–C7 herniation affects the C7 root. Pain may be exacerbated by coughing or straining. Numbness is more likely to supply reliable localizing information than pain. Compression of the C6 root typically causes numbness in the thumb and

index finger, and compression of the C7 root typically involves the index and middle fingers. When compression is severe, myotomal weakness, reflex loss, and, with time, fasciculations and atrophy may ensue. With C6 compression, the biceps, brachioradialis, pronator teres, and radial wrist extensors may be weak, and the brachioradialis and biceps reflexes may be diminished or lost. With C7 weakness, the wrist and finger extensors and the triceps are typically weak. The triceps reflex may also be diminished or lost. With C8 compression, there is often interscapular pain and pain in the medial aspect of the arm and hand with weakness of the hand intrinsic muscles. The finger flexor reflex may be lost. Lesions above C6 are less common and are associated with correspondingly more proximal sensory symptoms and weakness. Lesions of the C5 root may cause shoulder pain and pain and numbness in the lateral aspect of the upper arm. Many muscles can be used to test the C5 root, including the infraspinatus, supraspinatus, deltoid, biceps, and supinator. Lesions above this level may cause neck pain and sensory loss in the neck, supraclavicular area (C3), and acromioclavicular area (C4) of the shoulder. Lesions involving the spinal cord or roots above C4 may paralyze the diaphragm and cause respiratory compromise.

In the lumbosacral region, the most common site of herniation is the L5–S1 level, followed by the L4–L5 level and then higher levels. Symptoms of lumbosacral herniation often follow lifting or twisting injuries, or they may result from accumulated low-level trauma. Pain typically occurs in the parasacral area and radiates to the buttocks. Below C8, the roots exit through the neural foramina *below* the correspondingly numbered vertebral bodies. In patients with the most common posterolateral herniation, dermatomal radicular pain typically occurs at the level below the emerging root, which usually escapes entrapment above the protruding disc. Hence, L5–S1 herniation affects the S1 root. With posterolateral L5–S1 herniations and S1 root entrapment, the pain radiates to the posterior aspect of the thigh and, especially when the root is stretched, into the posterolateral lower leg, lateral heel, and sole. This pattern can be demonstrated by straight-leg raising, in which the smaller the angle of elevation required to elicit pain, the greater the suggestion that root compression is responsible. Characteristic pain on elevation of the opposite leg may be even stronger evidence of root compression. Some patients with symptoms that are exacerbated by root traction avoid full weight bearing on the heel of the involved side, standing with the knee flexed and the heel off the floor. When pain is less severe, symptoms may be elicited by having the patient walk on the heels. Numbness is felt in the posterolateral leg, lateral aspect of the heel, and the sole of the foot. The gastrocnemius and hamstrings may be weak, and the ankle jerk may be diminished or lost. More lateral herniation of the L5–S1 disc or herniation of the L4–L5 disc may entrap the L5 root. Here the pain may be similar, with adjustment of the findings to fit the L5 dermatome and myotome. Numbness is most marked on the dorsum of the foot. Weak muscles include the foot elevators (tibialis anterior group), everters (peronei), and invertors (tibialis posterior), and the toe extensors (extensor hallucis longus). Herniations at higher levels in the lumbosacral region cause pain and deficits that correspond to the roots involved.

In addition to these radicular syndromes, patients with central herniations in the cervical or thoracic region may develop pain and acute myelopathic symptoms with spasticity and quadriparesis or paraparesis, sensory loss at or below the segmental dermatome of the lesion, hyperactive reflexes, and Babinski's signs. Soon after an acute lesion develops, the reflexes may diminish because of spinal shock. Patients with lumbosacral central herniation may develop acute compression of the cauda equina. This causes radicular pain, paresthesias, and sensory loss referable to multiple bilateral roots, bilateral leg weakness, and loss of the lower extremity reflexes. Bowel and bladder dysfunction may occur early. When subtle, this dysfunction may be limited to asymptomatic bladder retention noted only on postvoid catheterization. When dysfunction is more severe, there may be perianal and perineal sensory loss, loss of anal tone and reflexes (the reflex anal sphincter constriction due to perianal skin stimulation or anal wink and the bulbocavernosus reflex), and fecal and urinary retention and incontinence. Degenerative herniations in the thoracic region are uncommon, and symptoms and findings at these levels should raise a suspicion of other underlying lesions, such as tumor or abscess. Disc herniations at this level may cause radiating dermatomal pain resulting from root compression; more frequently, they progress to spinal cord compression.

Spinal Stenosis: The symptom most suggestive of lumbar spinal stenosis is neurogenic claudication. Low back pain radiates to the buttocks and thighs and may extend more distally along the lumbosacral dermatomes. This pain is brought on by walking. Unlike vascular claudication, rest in the upright position does not relieve the pain, but rest while seated or forward bending, such as leaning on a shopping cart, may provide relief. Pain is exacerbated by spinal extension, such as downhill walking. When spinal stenosis is severe, patients bend forward while walking. Symptoms and signs may be either mechanical, due to bone, ligament, and joint involvement, or radicular, due to compromise of the lateral recesses or neural foramina. Proximal compression resulting from root entrapment may increase the vulnerability of nerves to dysfunction due to distal entrapment. This double crush phenomenon is presumed to be a result of disturbed axoplasmic flow and disrupted architecture of the neurofilaments. Therefore, when surgical repair of a distal entrapment fails to provide the expected relief, a contributing radiculopathy resulting from degenerative disc disease should be considered.

Differential Diagnosis: Disc herniations must be differentiated from other causes of acute and chronic neck, back, and extremity pain; radiculopathy; and myelopathy. Malignant and benign tumors affecting the spine; infection; epidural hematoma; various arthritides, including rheumatoid arthritis, ankylosing spondylitis, and Reiter's syndrome; and other spondyloarthropathies may present with similar early symptoms and signs. Various anomalies, such as conjoined spinal roots and multiple roots emerging through a single foramen, may also be confused with disc disease. Degenerative arthritis of the spine can cause symptoms by many mechanisms, including disc herniation, and the various lesions that are causing symptoms in a particular person should be differentiated as clearly as possible to allow directed therapy.

valuation: A careful history and physical examination are critical in the evaluation of disc herniation. It has been well established with ll imaging modalities that asymptomatic patients have a high incidence of anatomical lesions. To detect clinically relevant illness properly, it is therefore essential to establish the closest possible clinical correlation of the symptoms and signs with the anatomical findings of the various imaging studies. The initial history should screen for problems that raise a suspicion of severe underlying disease. All patients should be questioned about trauma, cancer, infections, recent fever, and the use of anticoagulant medications. The underlying family history and risk factors for tumor, infection, hematoma, and various disorders that predispose to disc disease should be sought. The physical examination, likewise, is undertaken to seek evidence of other severe underlying disease and to localize and classify the pain and any deficits as mechanical, radicular, or myelopathic. It is most important to immediately establish the presence of major deficits that demand rapid diagnosis and treatment. These include the cauda equina or conus syndrome, acute or progressive myelopathy, and severe radicular motor deficits. If, on the other hand, the findings are consistent with a ruptured disc and either no deficit or a mild to moderate one, it is reasonable to temporize before pursuing a workup to evaluate the cause thoroughly. If plain radiographs of the affected area reveal no evidence of unexpected lesions, conservative therapy for disc herniation may be tried before further imaging is performed. This approach is justified by the good prognosis for spontaneous recovery of patients with acute radiculopathy with mild to moderate deficits. When the clinical examination leaves doubt about the localization of the lesion, electromyography (EMG) can supplement the diagnosis of radiculopathies and suggest other localizations, such as plexopathies and neuropathies. EMG is more sensitive if it is delayed until at least 10 to 14 days after the onset of a new deficit.

The tests available for imaging include plain radiographs, computed tomography (CT), myelography with or without CT, and magnetic resonance imaging (MRI). X-ray studies can be used to screen for unexpected infection, tumor, or deformity of the bony spine. Radiographs cannot show the neural tissues or the disc itself, but loss of disc space height and other degenerative changes may provide some indirect diagnostic information. Interpretation of plain radiographs must be tempered by an awareness of the high frequency of degenerative findings in asymptomatic populations. Plain radiographs taken under conditions of flexion and extension can also be used to assess spinal stability. Myelography is invasive, indirect, and nonspecific; however, it retains certain advantages in the era of MRI. It can visualize the entire length of the spine and best defines the root sleeves. Although myelography alone cannot distinguish between osteophytes and a herniated disc compromising a foramen, when combined with CT, it provides the best visualization of lateral pathology and small osteophytes. It is now most commonly used to answer specific questions that remain after the MRI examination. CT is superior to MRI in distinguishing soft tissue from bone. MRI has emerged as the preferred imaging choice in most cases. It demonstrates bone nd soft tissues directly, easily allows multiplanar visualization, and is suited to the visualization of multiple levels. The high contrast of epidural fat and the cerebrospinal fluid (CSF)-filled thecal sac allows accurate assessment of subtle compression in most cases. Lumbar spinal stenosis is evaluated by CT or MRI. MRI best demonstrates the relationship of the bony and neural structures. CT best demonstrates lateral recess stenosis. Although the dimensions of the bony canal can be used as guidelines, diagnosis must ultimately be based on the correlation of stenosis with the clinical findings. The transverse interfacet dimension should be greater than 16 mm. A dimension of less than 10 mm indicates severe stenosis. An anteroposterior dimension of less than 12 mm suggests stenosis; however, this finding is less sensitive in patients with symptomatic disease. A lateral recess of 3 mm or less suggests stenosis.

Management: The crucial initial step in management of patients with disc herniation syndrome is to identify those lesions that merit further evaluation and immediate therapy. In the remaining cases, the good prognosis for early recovery justifies a trial of conservative therapy before definitive imaging is done. Conservative therapy includes rest in a position of comfort followed by early remobilization, gentle exercises, and analgesics for pain as needed. Nonsteroidal anti-inflammatory agents probably provide little relief in most cases. For severe pain, judicious time-limited use of narcotics should be considered. Oral and epidural corticosteroids can be helpful. Many other modalities are available, but there are few reliable data about their effectiveness in populations: medical and physical measures (e.g., ice, heat, massage, and ultrasound) that address secondary muscle spasm, transcutaneous electrical nerve stimulation, acupuncture, exercise, and traction. If improvement within the initial 4 to 6 weeks is not satisfactory, it is helpful to confirm the diagnosis by imaging. This may provide a diagnosis of an unsuspected condition, localization for epidural steroid injection, or information about suitability for eventual surgery.

Clear indications for surgery include the presence of acute myelopathy, cauda equina syndrome, severe or progressive motor deficits, and intractable pain. When conservative measures fail to provide a satisfactory response within 6 to 12 weeks, surgery should also be considered. Studies comparing the outcome of surgical therapy with conservative care suggest that early recovery occurs more often with surgery. Although the benefits of surgery are lost with prolonged follow-up periods, it is important to point out that in an often cited study, patients in the conservative therapy group who had not responded to this therapy received surgery. Newer microsurgical techniques allow shorter hospitalization and rehabilitation periods but have not been shown to improve long-term outcome. The success rate of chymopapain chemonucleolysis has not reached that of surgery in most hands, and this treatment carries significant risks. Percutaneous ucleotomy has also been disappointing and should not be pursued given the current level of experience. For patients with lumbar spinal stenosis, initial therapy is symptomatic, with analgesics, pain-modulating medications, and physical and occupational therapy. When significant disability and pain remain despite conservative measures, referral for surgical decompression should be considered.

Prognosis and Future Perspectives: The prognosis for the relief of pain and a full functional recovery is good. With bed rest alone, Weber found that 70 percent of patients experienced decreased pain and improved function within 4 weeks, and 60 percent had returned

to work. Seventy percent were functionally unrestricted at 1 year. With selective surgery, 90 percent of patients should have a good functional recovery within a year. Patients with psychosocial problems tend to do worse with either therapy, but those with appropriate indications respond better to surgery. Sensory dysfunction does not recover as fully as motor function, and a large proportion of patients retain some sensory deficits. Patients in whom relapse occurs should be re-evaluated for new lesions that are potentially addressable by surgery; however, the success rate of surgery declines with follow-up procedures, and a significant proportion of patients with disc herniation experience relapse with chronic low back pain.

Reviewers: Peter Moskovitz, M.D. (Orthopedics)
3 Washington Circle, #404, Washington, D.C. 20037
Phone: 202-333-2820 Fax: 202-833-14110 Email: pamosk@aol.com