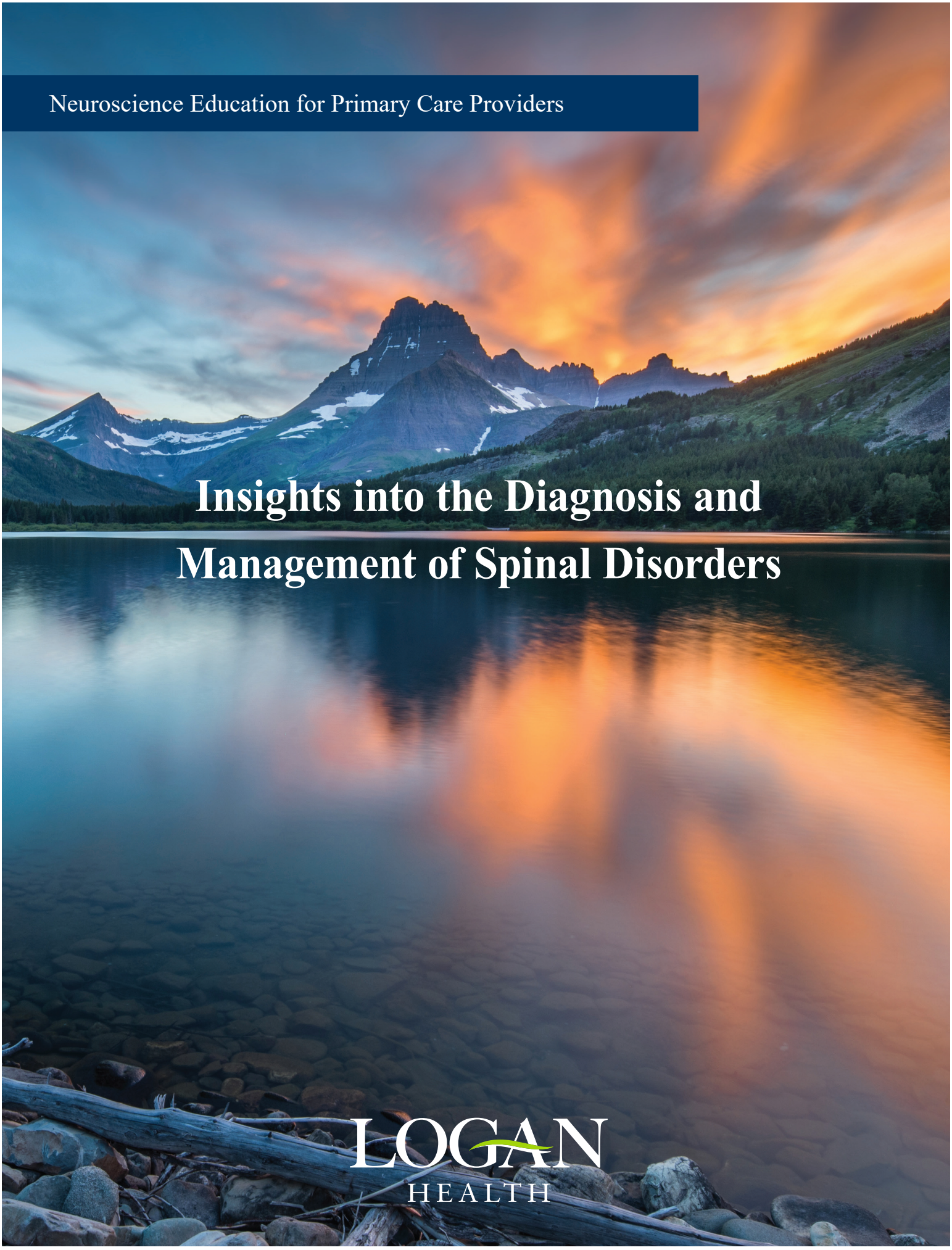
Neuroscience Education for Primary Care Providers
Insights into the Diagnosis and Management of Spinal Disorders
LOGAN
HEALTH

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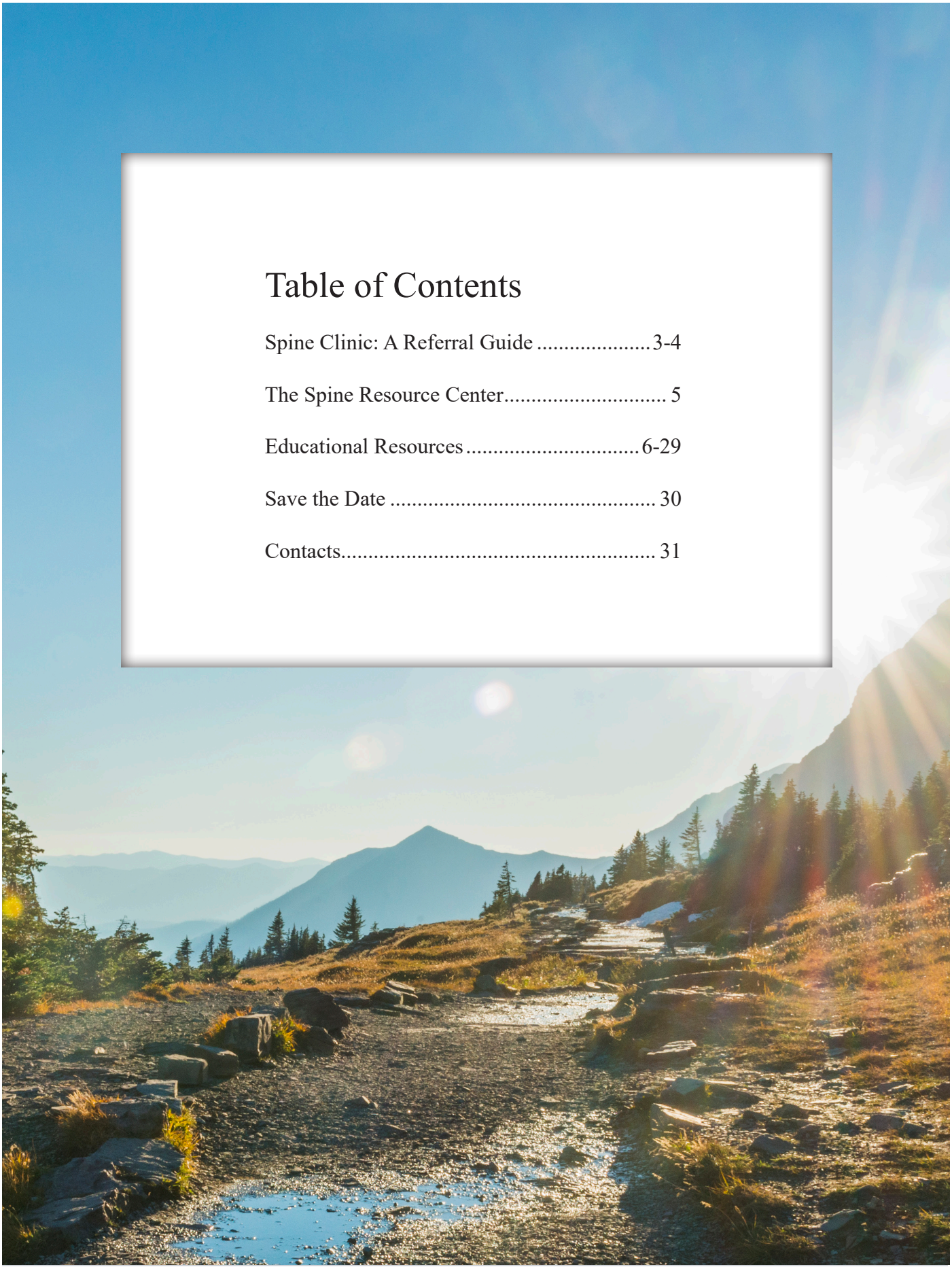


Spine Clinic: A Referral Guide

Delivering Spine Care through Collaboration

Optimizing patient experience and care starts well before the first appointment at the spine clinic. So much today is about setting and meeting patient expectations. To start with, patients can be divided into several major categories:

1. Patients with back or neck pain alone.

2. Patients with back or neck pain with radiculopathy.

3. Each of these categories is further divided as to onset into acute (0-6 weeks), subacute (6 weeks-6 months) and chronic conditions (greater than 6 months).

4. Mechanism of injury also plays a role. In reality most patients with operative spine problems are not related to a specific event.

5. Patients with a work related injury represent a unique challenge for the surgeon. Their workup in general should proceed through an occupational health expert and physical medicine specialist. Urgent surgical consultations are triggered by patients with neurological deficits.

6. The optimal patient for surgical consultation is one with back/neck pain with radiculopathy with a duration which is acute or sub-acute. This is in part due to the nature of surgical intervention. Surgical treatment of chronic conditions generally is far less successful as is surgical treatment for back and neck pain alone. The exceptions being in patients with spinal instability.

7. The art of spine surgery is in the identification of the pain generator. Identification of the pain generator then allows for targeted surgical or interventional therapies. As a referring healthcare expert you can play a role in identifying the pain generator and in setting your patients' expectation that a surgical solution maybe one of the answers to their problem. Some of the most disappointed patients are the one's "sent for surgery" whom leave the surgeons' office with no surgery recommendation. Surgery should be explained as one of the therapies which may help the patient. In the general population about 1 out of every 25 patients whom complain of neck or back issues maybe a surgical candidate (ie. 5/100). The vast majority will not. This is why in part, entry criteria into the SPORT trial (spine patient outcomes research trial) required at least 6 weeks of active symptoms for enrollment. Most acute spine problems resolve on their own.

8. The optimal patient for the spine clinic is one with acute/subacute neck or back pain with radiculopathy. These are the patients with the most favorable surgical results. The optimal work up for these patients include an MRI of the symptomatic area plus dynamic load bearing plain xrays (standing flexion/extension images). Dynamic images are essential in part on understanding the pain generator and in part whether fusion is necessary.

Secondary Considerations:

1. It is extremely helpful to send over a brief cover letter along with the patient including pertinent medical history (i.e.cardiac status, anticoagulation status, reason for consultation and treatments tried to date). Special considerations to the past medical history is whether the patient has been seen previously by a spine surgeon who recommended, "No surgery." These are patients whom are best discussed by direct communication prior to consultation.

2. If the patient has multiple medical co-morbidities which will need to be settled prior to surgery, please proceed with the workup in anticipation of the surgery. One of the contraindications for spine surgery is too high of a risk benefit ratio.

3. Prior to sending to the surgeon a general discussion should include whether a patient would have an interest in a surgical intervention. If the patient is not, surgical consultation will be frustrating for both parties.

4. The most important consideration is communication. We are here as a resource. We have access to your imaging. We encourage communication with all referring healthcare providers.

We encourage your calls and look forward to discussing your patients.

The Spine Resource Center (SRC)

Advanced and Comprehensive Spinal Disorder Diagnosis and Management

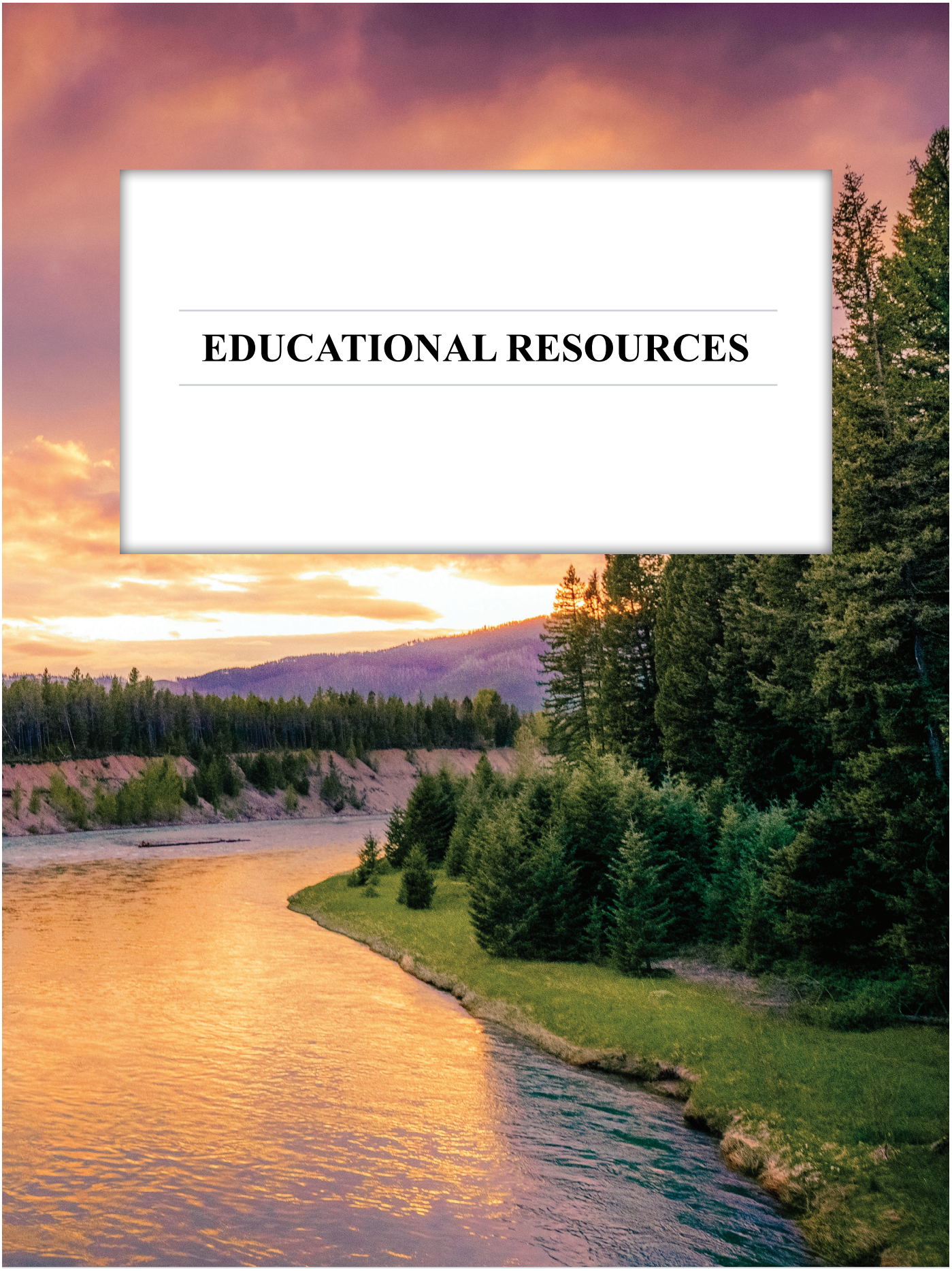
Spinal pain is one of the most common ailments, sending patients to health care providers on a global scale. Numerous treatment pathways exist for patients. These options can often be confusing, costly and time-consuming. Patient treatments for the same problem can be highly variable depending on the point of entry into health care. The vast majority of patients with new, recurrent or exacerbation of their spine problems will not need surgery or invasive interventions.

Navigating healthcare can be challenging for patients. The Spine Resource Center was created to optimize the patient experience for patients with spinal problems. Housed in the Department of Physical Medicine & Rehab (PM&R), it serves as a primary entry point for patients with new or recurrent spinal disorders. The process starts with an evaluation of the patient's signs, symptoms, and performed testing. Optimizing the clinical data optimizes the patient's clinical experience. Due diligence up front can also result in a fast track appointment with a surgeon when appropriate.

During the initial appointment, a thorough history and neurological examination are performed. The patient is evaluated and sent for focus neuro-spine imaging and or neurophysiological testing. The combination of neurological examination and ancillary testing provides guidance in the formulation of a treatment plan. Treatment plans can include physical therapies, interventional injection therapies, musculoskeletal manipulation, or referral for surgical evaluation. Patient cases are discussed by a multiple disciplinary team of spine care providers which includes the PM&R providers, neurological surgeons, and pain management specialists. The SRC providers can call upon the whole of the Neuroscience and Spine service line (Neurology, Neurological Surgery, Physical Medicine and Rehab, and Wellness and Pain Management) for consultation, case discussion and expedited intervention when needed.

The Spine Resource Center can be accessed at (406) 758-7035.

One call opens the door to advanced and comprehensive spine care in your community.



EDUCATIONAL RESOURCES

Effectively Diagnosing and Treating Back Pain

By Austin Johnston, DO

Symptoms of low back, buttock, thigh and knee pain can leave many patients, as well as their health care practitioners, frustrated with multiple diagnoses and treatment plans that provide minimal relief. Uncontrolled back pain can lead to a quandary of other health risks, including severe functional decline. The Physical Medicine & Rehab (PM&R) department at Logan Health specializes in efficiently identifying the etiology of these symptoms, and effectively treating with appropriate modalities. Often, the difficulty in obtaining an accurate diagnosis is amplified by the presence of multiple possible pain generators. This article hopes to provide useful advice for the effective evaluation and treatment of patients who present with lumbar and proximal leg pain.

Radiographic hip osteoarthritis (HOA) is present in 27 percent of adults greater than 45 years of age, and one third of these patients are symptomatic.[1] It is well documented that half of all patients with HOA suffer from low back pain.[2] When lumbar spinal stenosis (LSS) and radiographic HOA appear in conjunction, it is known as hip-spine syndrome. This syndrome was first described by Offierski and MacNab in a retrospective study in 1983.[3] Due to the overlapping nature of symptoms, conducting a thorough history and structured physical exam is critical in obtaining appropriate diagnostic modalities and providing effective treatment in a timely fashion.

Groin pain is perhaps the most recognized complaint with HOA. Evidence suggests that groin pain is both sensitive (84.3 percent) and specific (70.3 percent) for patients with HOA. Patients with this complaint are seven times more likely to have an isolated hip or hip-spine disorder versus an isolated spine disorder.[1] Some health practitioners believe the location of pain in HOA may refer to the groin, thigh and knee, but does not radiate into the lower leg. Khan et al found that 47 percent of patients with isolated HOA reported pain radiating below the knee.[4] The most common pain referral patterns were in the gluteal region, anterior thigh, anterior knee, posterior knee, anterior shin, and calf.[4] These symptoms could easily be mistaken for a lumbar radiculopathy. If groin, anterior thigh, and buttock pain persists in the absence of radiographic HOA, labral pathology of the hip joint should be considered. More than a few labral tears are misdiagnosed as a radiculopathy, with symptoms continuing on average for two years before the primary pain generator is discovered.[2] If labral pathology is present, the patient may describe the "C" sign (cupping hand over hip) or the "restaurant sign" described as the inability to sit still for prolonged periods.[2] Finally, if groin pain persists with no clear evidence of hip pathology one may consider other less likely diagnoses. One recent small retrospective study found that patients with sacroiliac joint dysfunction (SIJD), lumbar spinal canal stenosis, and lumbar disc herniation may also present with groin pain, with SIJD having the highest prevalence at 46.5 percent.[5]

Hip osteoarthritis typically presents with antalgic gait (unilateral shortened stance phase), restricted internal rotation, and painful rotation.[1,4,6] The hip osteoarthritis workup should begin with evaluation for diagnostic criteria provided by the American College of Rheumatology.[6] At PM&R we accurately evaluate hip range of motion restrictions with precise measurements, assess for contractures and identify the presence of localizing symptoms using targeted provocative maneuvers. For example, cam and pincer impingement may be detected with the anteroposterior and posteroinferior impingement test.[1] Pain or locking during internal rotation in patients with prior hip arthroplasty may indicate psoas tendon impingement.[2] The FABER exam (flexion, abduction, external rotation) may indicate trochanteric bursitis or sacroiliac joint pathology depending on the location of the pain.

Provocative maneuvers for LSS are less reliable, but remain useful for developing a comprehensive differential. Radicular symptoms may be uncovered with the straight leg test or femoral stretch test. A steppage gait may be noted with L4-L5 nerve root involvement and may be coupled with slapping of the forefoot against the ground at heel strike.[2]

A broad-based gait or a Trendelenburg (waddling) gait may be noticed with gluteal muscle weakness or L4, L5, S1 nerve root involvement. Muscle wasting may be appreciated upon visual inspection with weakness and sensory deficits following a specific myotomal and dermatomal distribution. These findings often correspond to a specific nerve root which may also present with an asymmetrically depressed reflex. Examiners should remain vigilant for hyperreflexia, clonus, and Babinski sign which may indicate upper motor neuron involvement and myelopathy. In patients with a history of arthroplasty, a leg length discrepancy may be the culprit and bilateral lower extremity lengths should be measured.

Based on the findings in the history and physical exam, initial imaging for the hip should include anteroposterior standing pelvis radiographs. In the presence of HOA, we often see femoral or acetabular osteophytes, subchondral cysts, and joint space narrowing on weight bearing views.[1] Subchondral lucency in the femoral head is indicative of osteonecrosis.[1] Evidence for cam or pincer impingement include a bony prominence near the anterolateral head and neck junction, anterior over coverage, acetabular retroversion, coxa profunda, and protrusion acetabula.[1] If there is suspicion for labral pathology, MR arthrogram is the preferred method for evaluation.

Initial spine imaging in our clinic includes anteroposterior plain radiographs with lateral extension and flexion views to evaluate for vertebral instability. Oblique views are obtained to evaluate for facet arthropathy. If clinical evaluation suggests leg length discrepancy, a CT bone length study may be obtained for definitive measurement. Practitioners within the PM&R department are well versed in the evaluation and treatment of scoliosis and a 36 inch upright radiograph is frequently obtained for accurate assessment for compensatory curves. For advanced neuroimaging, the presence of nerve root or central cord impingement on physical exam warrants magnetic resonance imaging. If hardware or implantable devices prohibit the use of MRI, a CT myelogram is the alternative method of choice to evaluate for stenosis. Within PM&R we have the capacity to perform electrodiagnostic testing. Electrodiagnostic studies may confirm axonal damage in the distribution of a particular nerve root or may provide evidence for peripheral nerve entrapment. These studies have good utility in distinguishing spinal stenosis from peripheral nerve compression, or diabetic peripheral polyneuropathy, but normal findings do not rule out degenerative lumbar spinal stenosis.[7]

With the initiation of therapy, pain and function should be logged consistently and accurately at each visit. The practitioner should establish a goal of at least 30 percent pain reduction (two point improvement on a 0-10 numeric pain rating scale) coupled with functional improvement assessed by the Oswestry or Roland Morris questionnaire.[6,8] Health practitioners within the PM&R department are experts in optimizing functional independence and work closely with local therapy centers to provide appropriate, precise exercise prescriptions to maximize potential. For conservative management of HOA, T'ai Chi is a first line recommendation due to strong evidence for efficacy with fall prevention and modest efficacy with reducing pain and improving function.[6] Typical conservative treatment options for lumbar spinal stenosis include massage, acupuncture, epidural steroid injection, cognitive behavioral therapy, lumbosacral corset, and aquatic therapy.[8] Conservative pharmacological management begins with acetaminophen or NSAID's. Other adjunctive medication such as gabapentin, tramadol and duloxetine may be added if pain and function remain uncontrolled. Duloxetine is FDA approved for osteoarthritic pain.[6,8] Epidural and intra-articular steroid injections can prove to be both diagnostic and therapeutic for lumbar spinal stenosis and hip osteoarthritis. We often use this diagnostic approach in conjunction with physical therapy exercise programs to optimize functional outcomes.

In regards to hip osteoarthritis, Devin et al allude to several studies providing evidence that patients who experience at least 50 percent pain relief following intra-articular hip injection are likely to have a successful outcome following total hip arthroplasty. They also strongly advise intra-articular injections of local anesthetic at the time of

arthrogram to efficiently diagnose the hip as the primary pain generator. If there is no relief following intra-articular hip injection, one should proceed to workup spine pathology.[3] It is imperative that any hip flexion contracture detected on physical exam is addressed with physical therapy or surgical intervention prior to the patient undergoing surgery for hip osteoarthritis or lumbar spinal stenosis. Several studies have suggested improvement in pain and function using the visual analog scale and the Oswestry Disability Index focusing on lumbar back pain following total hip arthroplasty.[2] These results are however, somewhat unpredictable.

Discovering the primary etiology of pain referred to the low back, buttock, thigh and knee is no easy task. PM&R is well suited to effectively manage a range of musculoskeletal and neurological issues that accompany low back pain patients by utilizing a comprehensive, holistic approach. It is comforting to know that symptomatic lumbar spinal stenosis patients may remain neurologically intact and stable for many years, and many will improve over time. The natural history of lumbar spinal stenosis can be favorable in 30 to 50 percent of patients, and catastrophic neurological decline is rare.[9,10] LSS symptoms are frequently episodic, and we strive to empower the patient to self-manage acute exacerbations. Many patients achieve satisfactory relief and maintenance of function by utilizing a detailed therapeutic prescription to achieve an effective routine home exercise program. Maintenance of function is the overarching goal in developing a management plan for all of our patients.

The PM&R department at Logan Health understands that if patient function is not improved, despite reduction in pain, successful outcomes may never be achieved. We house a dedicated team of individuals who work cohesively with local therapists and our neurosurgical colleagues to provide lasting benefit for our patients.

References:

1 Devin C, McCullough K, Morris B, Yates A, Kang J. Hip-spine Syndrome. J Am Acad Orthop Sure. 2012; 20:434-442

2 Defroda S, Daniels A, Deren M. Differentiating Radiculopathy from Lower Extremity Arthropathy. The Am J Med. 2016;129:1124.e1-1124.e7

3 Offierski CM, MacNab I. Hip-spine Syndrome. Spine. 1983;8(3):316-321.

4 Khan AM, McLoughlin E, Giannakas K, Hutchinson C, Andrew JG. Hip osteoarthritis: where is the pain? Ann R Coll Sure Engl. 2004; 86(2):119-121

5 Kurosawa D, Murakami E, Aizawa T. Groin pain associated with sacroiliac joint dysfunction and lumbar disorders. J Clin Neuro.2017;161:104-109

6 Weiner D, Fang M, Gentili A, Kochersberger G, et al. Deconstructing Chronic Low Back Pain in the Older Adult - Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment. Part I: Hip Osteoarthritis. Pain Medicine. 2015;16:886-897

7 Adamova B, Vohanka S, Dusek L. Differential diagnostics in patients with mild lumbar spinal stenosis: The contributions and limits of various tests. Eur Spin J 2003;12(2):190-196

8 Fritz J, Rundell S, Dougherty P, Gentili A, et al. Deconstructing Low Back Pain in the Older Adult – Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment PartVI: Lumbar Spinal Stenosis. Pain Medicine.2016;17:501-510

9 Benoist M. The natural history of lumbar degenerative spinal stenosis. J Bone Spine 2002;69:450-7.

10 Tsutsumimoto T, Shimogata M, Yui M, Ohta H, Misawa H. Then natural history of asymptomatic lumbar canal stenosis in patients undergoing surgery for cervical myelopathy. J Bone Joint Surg Br 2012;94:378-84.

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The C-Spine Shoulder Paradox

By Justin Shobe, PA-C

Cervical spine (c-spine) complaints commonly blur with shoulder complaints in the outpatient setting. Differentiation between c-spine, peripheral nerve and musculoskeletal pathology challenges even the most skillful providers. Complicating the situation further, multiple pathologies may coexist. For example, one study found up to 24 percent of cervical radiculopathy patients also demonstrate shoulder impingement.[1] An attentive history and physical exam with provocative tests can help begin to localize pathology. This also helps differentiate between radicular pain and radiculopathy – a radicular pain pattern with motor and/or sensory deficits. Clearly a supplemental role for diagnostic testing exists, however, despite technological advances, there is no gold standard for cervical radiculopathy diagnosis. All modalities have their limitations.[2] Decisions should be weighed heavily when pursuing advanced testing due to the inherent cost (i.e. MRI) and discomfort (i.e. electromyography) imposed by these tests. Furthermore, specialized testing availability can pose a problem, especially in rural Montana. Physical Medicine & Rehab (PM&R) provides a function-focused environment aimed at distinguishing the cause(s) of pain and neurological deficits, and what therapies or assistive devices might provide a safe and efficient return to daily function. This discussion outlines our diagnostic approach to patients with neck, shoulder and upper extremity complaints.

Anatomy

The complexity of the shoulder provides ample opportunities for degeneration and injury. Its joints include the acromioclavicular, glenohumeral, sternoclavicular and scapulothoracic joints. The shoulder is stabilized by the bony anatomy, labrum, rotator cuff and the capsuloligamentous complex.[3]

The cervical spine has eight sets of spinal nerves exiting amongst seven cervical vertebrae. The motor and sensory branches of spinal nerves travel via the brachial plexus to their innervation sites in the upper extremities. The encapsulated bony joints, called facets, articulate between each vertebral body. Intervertebral discs lie between each vertebral body, providing shock absorption. The lower c-spine (C5-C7) has additional posterolateral stabilization provided by the uncinate process. As we age, this articulation can become arthritic, contributing to stenosis and nerve impingement. C1 and C2 are unique in their bony anatomy and articulation. Their stability depends on an intact ligamentous complex. This articulation allows approximately 50 percent of the rotation of the head.[3]

Subjective

Patients with shoulder pathology may present with a trauma history. Dull, aching, pinpoint pain is usually more suggestive of shoulder or local musculoskeletal pathology. Pain with motion such as arm abduction and lifting overhead favors a shoulder origin as well.[3]

Cervical radicular symptoms typically do not arise from a traumatic history.[4] Painful tingling, electric-type pain and burning imply nerve irritation or injury. Pain often relieves when the ipsilateral shoulder is

raised overhead. When nerve pain is suspected, we must then work to localize the abnormality – is it a central, spinal nerve or peripheral nerve problem? Not all pain complaints follow a dermatomal pattern; in fact, one study found only 30 percent of patients with cervical radiculopathy traced out a dermatomal pattern corresponding with the level of injury.[5]

Weakness without pain should always prompt concern for nerve injury, including diffuse motor neuron diseases. Complaints of apraxia, gait disturbance, hyperreflexia and loss of bowel or bladder function warrant urgent MRI (or available/appropriate alternative) of the brain and c-spine in search of an upper motor neuron lesion manifesting as myelopathic symptoms.

Objective

Approximately 90 percent of patients with active radiculopathy present with arm pain.[3] A c-spine and shoulder exam should both be considered for this complaint. Shoulder exams should include close observation, palpation, passive and active range of motion (ROM). Limited ROM is far more common in shoulder pathology compared to cervical radiculopathy.[3] The drop arm test provides 97 percent specificity for subacromial or rotator cuff disease. Testing each of the components of the rotator cuff, biceps, serratus and rhomboids helps in isolating weakness. Provocative testing should include the empty can test (for supraspinatus tears), Neer's (for signs of impingement), Yergason's and Speed's test (for biceps tendinopathy).[2]

Cervical spine inspection begins by observing the posture of the head and neck. Patients will often sit with their head tilted to the contralateral side of their symptoms. Neck ROM and palpation help localize tenderness. Strength testing of the cervical myotomes, sensation testing of dermatomes and looking for unilateral reflex changes all provide key information to identifying the location and degree of neuropathology. Co-occurrence of shoulder pathology or generalized pain can cloud this exam. Provocative tests usually include Spurling's, Valsalva's and upper limb tension test (ULTT). Despite widespread use of provocative tests such as Spurling's, Rubenstein et al. determined a serious lack of consensus on testing technique and inferences thereof.[2] Still, their study found high sensitivity for radiculopathy on the upper limb tension test (ULTT) and high specificity with Spurling's and Valsalva's maneuvers, when performed accurately.

Gumina et al. developed a very simple test to distinguish neck and shoulder pain called the 'arm squeeze test.' Clinicians manually provide pressure to the middle third of the humerus. A positive test produces intense local pain, and suggests spinal nerve root compression with 96 percent specificity, and 96 percent sensitivity.[6] This test remains to be validated.

Once again, exam signs of ataxia, apraxia, spastic gait, hyperreflexia, a positive Hoffman's or clonus in the lower extremities should trigger urgent MRI (or available/appropriate alternative) and referral to neuroscience specialties for suspicion of myelopathy.

Diagnostics

Imaging should be ordered for confirmation of diagnoses suggested by history and exam. Shoulder pathology with positive provocative testing warrants plain x-ray of anterior-posterior (AP), axillary and scapular "Y" views. If history and exam are suspicious for rotator cuff tear, MRI arthrography is the most sensitive and specific test.[7] De Jesus et al. report MRI and ultrasound provide equal sensitivity and specificity for full thickness rotator cuff tears.[8] Patients generally tolerate ultrasound better, and it requires far less time and cost. In addition, ultrasound can examine muscles in action and observe dynamic changes.[9] Injections constitute another important diagnostic avenue, helping localize pain and pathology based on symptom relief.

Symptoms of radiculopathy with positive provocative tests (Spurling's) should prompt an initial workup including AP and lateral x-rays with a lateral flexion/extension view as well. This helps determine the stability with motion within the spine. Oblique views can help identify foraminal stenosis. When physical exam demonstrates motor or sensory deficits, MRI is the most informative modality, if available. CT myelography yields less sensitivity, but provides an MRI alternative. Electromyography/Nerve Conduction Studies (EMG/NCS) provides very specific evidence of pathology. Therefore, EMG/NCS is best used to confirm radiculopathy, not to rule it out. Likewise, selective nerve root blocks (SNRB) and transforaminal epidural steroid injections (TFESI) can be used to localize the origin of radicular symptoms based on symptom relief.

Delineating pain sources in the neck and upper extremities requires knowledge of regional anatomy and proficiency in obtaining a history and physical exam, particularly provocative testing. Judicious use of imaging helps solidify appropriate treatments. These clinical measures, alongside open communication with the appropriate specialists potentiate a streamlined, cost effective, compassionate experience for the patient that limits their pain and functional loss. As providers at Logan Health and throughout northwest Montana, this should remain our highest aspiration.

References:

1. Date ES, Gray LA. Electrodiagnostic evidence for cervical radiculopathy and suprascapular neuropathy in shoulder pain. Electromyogr Clin Neurophysiol. 1996;36:333-339.
2. Rubenstein SM, Pool JJ, van Tulder MW, Riphagen II, de Vet HC. A systematic review of the diagnostic accuracy of provocative tests of the neck for diagnosing cervical radiculopathy. Eur Spine J. 2007 Mar;16(3):307-19.
3. Bokshan et al. An Evidence-based approach to differentiating the cause of shoulder and cervical spine pain. Amer J of Med. 2016;129(9):913-8.
4. Rao R. Neck pain and cervical radiculopathy and cervical myelopathy. J Bone Joint Surg Amer. 2002;84:1872-1881
5. Murphy DR, Hurwitz EL, Gerrard JK, Clary R. Pain patterns and descriptions in patients with radicular pain: Does the pain necessarily follow a specific dermatome? Chiro and Osteo. 2009;17:9.
6. Gumina S, Carbone S, Albino P, Gurzi M, Postacchini F. Arm squeeze test: a new clinical test to distinguish neck from shoulder pain. Eur Spine J. 2013;22(7):1558-63.
7. Throckmorton TQ, Kraemer P, Kuhn JE et al. Differentiating cervical spine and shoulder pathology: common disorders and key points of evaluation and treatment. Instr Course Lect. 2013; 63: 401-408.
8. De Jesus JO, Parker L, Frangos Aj, Nazarian LN. Accuracy of MRI, MR arthrography, and ultrasound in the diagnosis of rotator cuff tears: a meta-analysis. AJR Am J Roentgenol. 2009;192(6):1701-7.
9. McMahon C, Yablon C. The Shoulder. Diagnostic Ultrasound. Elsevier. 5th ed. 2018;24.

*Article reprinted from SYNERGIES 2017

Injections for Pain Relief

By Amy Tangadahl, PA-C

Injections are an important tool in every clinician's toolkit; both for the workup and the ultimate objective of finding pain relief for the patient. Many types can be done in-office by qualified providers and may include the utilization of ultrasound. Other types of injection may be completed by interventional radiology or pain management, under visualization by fluoroscopy. In this brief summary, several different injections will be discussed, including indications and goals of those commonly used on a daily basis in a neurosurgical clinical practice.

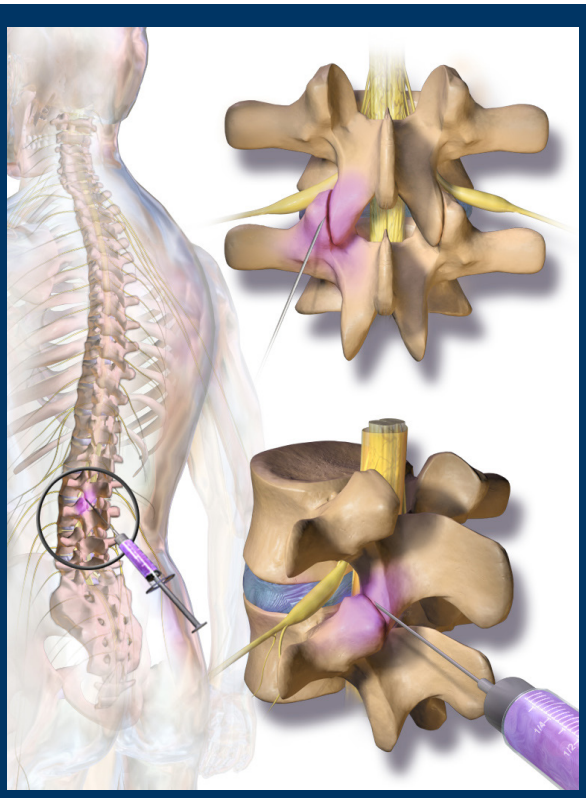
A trigger point injection is a good place to start. This common injection is done in-office by a practitioner who is comfortable with the technique. Although most commonly done without, occasionally ultrasound can be used. A trigger point is a tender area of muscle or fascia with noted sensitivity to direct palpation.

The indication for this injection is replication of the patient's pain with direct palpation to the area. The desire is to alleviate the spasm to allow the muscle/fascia to relax. The injection itself is commonly a combination of anesthetic and steroid. If the majority of the patient's pain is relieved immediately after administration of the local anesthetic, this would point toward diagnostic, meaning the trigger point is the patient's pain generator. The steroid will hopefully give longer lasting relief through decrease of inflammation. This is generally utilized through a combination of physical therapy to eliminate further muscle spasm or fascial tension.

Two important terms have been discussed and should be defined. The anesthetic is the diagnostic portion of the injection. If significant pain relief is achieved, this points towards a diagnosis. An injection done with only anesthetic is diagnostic, while one with both anesthetic and steroid, is designed as diagnostic and therapeutic. The second term to explain is pain generator. The aim of the injection types covered in this article, are to determine the patient's pain generator so progress may be made to the next stage in the treatment plan, if necessary.

The following injections summarized are ordered within the departments of interventional radiology or pain management and will be done through the use of fluoroscopy – medical imaging with use of a continuous x-ray image.

Arm or leg pain assumed to be indicative of radicular pain can be further narrowed to a particular spinal nerve root through a selective nerve root block (SNRB). The indication for a SNRB is the combination of limb pain following a precise dermatomal distribution, correlating with radiographic findings of compression of the same nerve root on MRI or CT myelogram. If the majority of the patient's pain is relieved the day of the injection, it can be inferred that this particular nerve root is causing the majority of the patient's pain. On occasion, the



A cervical, thoracic or lumbar facet joint injection involves injecting a small amount of local anesthetic and/or steroid medication, which can anesthetize the facet joints and block the pain.

steroid portion can give long enough relief that a disc herniation may be able to heal itself. However, this is usually only possible within the first three months after injury and without evidence of neurologic deficit.

Radicular pain and myofascial pain have now been covered, which leads us to the surgical nemesis of neck and back pain. Neck and back pain are caused by such a multitude of differing sources, it is very important to find a pain generator that is amenable to surgical intervention if possible. This can be done through injecting the facet blocks. A facet block is indicated if there are findings of instability on flexion and extension x-rays showing a spondylolisthesis, or possibly a facet joint effusion noted on MRI. This particular injection bathes the facet joint, once again, with a combination of anesthetic and steroid. The provider is looking for significant improvement during the anesthetic phase of the injection. The steroid portion also may give the patient longer relief, and if surgical intervention is deemed an option, a spinal fusion may be prolonged through use of other conservative treatments such as physical therapy. Prolonging intervention is appropriate as long as there are no neurologic deficits noted.

Finally, it is important to discuss medial branch blocks. Medial branch blocks are very similar to facet blocks, as their indicators are the same, but they differ in the particular location of injection. Each spinal nerve root gives off a medial branch, which innervates into the facet joint at the levels above and below. For example, the L4 nerve root radiates a medial branch, which innervates into the L3-4 and the L4-5 facet joints. Therefore, a L4-5 medial branch block will require blockage of both the L4 and L5 medial branches. Frequently, this injection is done with only local anesthetic to determine the percentage of relief the patient receives from the diagnostic portion. The injection will be repeated a second time. If significant pain relief is achieved, the patient will proceed to the next step, a rhizotomy. A rhizotomy procedure is actual burning of the nerve through heat or cold depending on indications. This can give much longer standing relief and is an excellent option for those patients who are not adequate surgical candidates, but have clear symptom such as dynamic spondylolisthesis. It is important to note, that this procedure can cause increased instability of the spine due to the denervation of the facet stabilizers. Therefore these injections should be ordered with caution for the cervical spine.

The prior injections noted are just a few of the common diagnostic and therapeutic types utilized for identification of pain generators, when making a plan for surgical intervention. Some of the other common diagnostic and therapeutic injections include shoulder, knee, sacroiliac and elbow joints; occipital, supraorbital, median, cluneal, genitofemoral, ilioinguinal/hypogastric and suprascapular nerves; and finally, trochanteric and ischial bursae. These are all used in the enduring and elusive hunt for the diagnosis of pain generators. As technology and methodology continue to advance, non-surgical methods such as injections will preserve a larger patient population who brave their ongoing struggle with neck and back pain.

*Article reprinted from SYNERGIES 2017

Ouch...My Neck Hurts! Evaluation and Treatment

By Mark Haven, PA-C

There are three types of neck pain: Acute pain that lasts less than six weeks; subacute pain that lasts six weeks to six months; and chronic pain that lasts more than six months.

Therapy modalities

- Physical therapy
- Physical Medicine & Rehab
- Chiropractor
- Acupuncture
- Medications
- Injections
- Ice, heat, TENS unit, stretching
- Neurosurgery

Medication options

- OTC pain relievers/NSAIDS
- Oral steroids/prescription NSAIDS
- Muscle relaxers
- Gabapentin or Lyrica for neuropathic pain
- Narcotics (avoid)

Axial neck pain

- Cervical strain
- Cervical facet mediated pain
- Whiplash
- Myofascial pain
- Discogenic pain (radiograph dx)

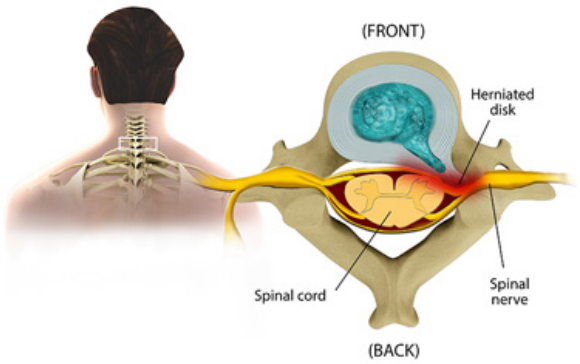
Treatment goals are to reduce pain, muscle irritability and spasm. Most mild to moderate cases see improvement in first two to three weeks with conservative therapy.

There is no role for surgical intervention in the vast majority of patients with isolated axial neck pain. Routine cervical collar immobilization should be discouraged as regular use can delay improvements.

Predominant extremity pain

- Cervical radiculopathy, may have degree of neck pain

Treatment options include conservative therapy and diagnosis with EMG. If conservative therapy is not successful, or if findings of muscle weakness or decreased reflexes surface, referral to neurosurgery may be appropriate.



Neurologic dysfunction

- Cervical myelopathy - degenerative changes narrowing the spinal canal resulting in spinal cord injury or dysfunction.

Symptoms can include a degree of neck pain or radicular symptoms, gait disturbance or difficulty with hand dexterity. Sometimes, the patient will have no pain.

Treatment for neurologic dysfunction generally requires referral to neurosurgery.

*Article reprinted from SYNERGIES 2018

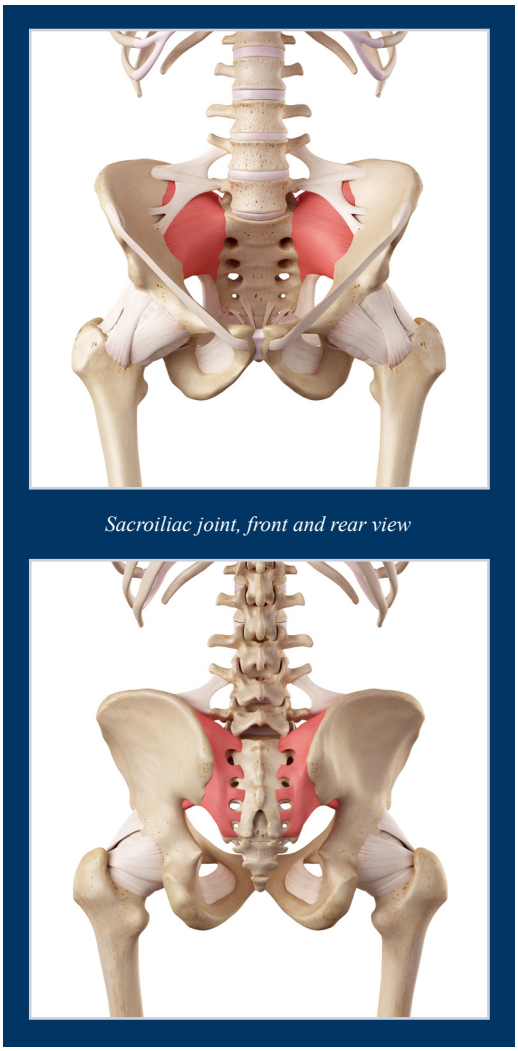
Sacroiliac Joint: An Elusive Pain Generator

By Amy Tangedahl, PA-C

The sacroiliac joint – we all know it's location, but exact function, referral pattern and kinesiology is a bit more elusive. It's no knee, elbow, hip or even complex shoulder joint. All that being said, it seems to be a frequent component in the overall pain picture of many of our patients.

I recently saw a very common patient to all of our practices. A 77-year-old male, very active outdoorsman, with low back and left leg pain. His symptoms have been slow and progressive in onset, but have been really bothering him for the past six weeks. He has attempted multiple conservative treatment options including OTC anti-inflammatories, Medrol Dose Pack, narcotics, neuropathic medications, ice, heat and chiropractic treatment. He has been evaluated by urgent care. His symptoms are diffusely distributed across his low back and into his left lateral and anterior thigh to his knee. His symptoms are made worse with sitting and better with bending forward. So far, this is a pretty average lumbar spondylosis and possible radicular picture. Then I open his imaging studies.

This poor gentleman has severe degenerative disease throughout the entire spine including degenerative disc disease, facet arthropathy, central and foraminal stenosis, disc bulges where he has discs left, and a degenerative scoliosis. The man sitting in front of me does not match with the images I'm looking at. He's got a little bit of back pain and his leg hurts, and I have nowhere to shake a needle at! More specifically, he has something going on at every level of his spine which is a diagnostic nightmare for trying to identify a pain generator. His symptoms do correlate with an L3 distribution, but guess what, this is one of the few areas he does not have compression. His exam is not helpful in delineating anything further without any focal neurologic deficit. The only thing noted is general lack of range of motion in his hips without significant pain and tenderness to palpation over the left sacroiliac joint. I started him on an anti-inflammatory and sent him for



Sacroiliac joint, front and rear view

a left lower extremity nerve conduction study and left sacroiliac joint injection. The patient followed up after the SI joint injection. He reported 90% improvement in his overall pain picture. He only reported occasional tingling into his left knee. What a success! While I feel he still has a component of potential L3 radiculopathy he is not currently having significant symptoms so we canceled the EMG study and I referred him back to physiatry for continued conservative management.

The lessons learned here are that occasionally inflammation in the sacroiliac joint can mimic lumbar radiculopathy and occasionally the sacroiliac joint can be a bigger component of a patient's overall pain picture.

The sacroiliac joint (SIJ) transmits the forces from the limbs through the pelvis to the spine and vice versa. It is innervated by a combination of the L4-S1 nerve roots with some input from the superior gluteal nerve. Secondary to this innervation as well as geometrical force irritation and inflammation can manifest as low back, hip, leg, pelvic or gluteal pain, to name a few. It is a common injury in unilateral athletes such as cross-country skiers and rowers as well as pregnant patients. Anecdotally, it is common in patients with a leg-length discrepancy, total hip or knee replacement or some other gait disturbance that causes a pelvic imbalance. Physical exam is rarely satisfying in terms of finding a solid diagnosis. The exam tests recommended are FABER, compression provocative test, thigh thrust, distraction provocative and gaenslen provocative. However, the most diagnostic test is what happens after an anesthetic injection.

We have very successful treatment options here in the valley for sacroiliitis. Initially, the injection can be both diagnostic as well as therapeutic through the steroid phase. The injections can be done by both physiatry and Dr. Kneeland at Logan Health Wellness & Pain Management. The next step is to attempt to correct the imbalance through physical therapy, orthotics, gait training, etc. If this step fails, Dr. Kneeland does a rhizotomy, or nerve burning, procedure for those nerves that innervate the SIJ. Finally, if all else has failed, there are options for SIJ fusion.

In conclusion, through the complex process of diagnosing patient's pain generators, I have found that the most diagnostic in-office test is direct palpation to the SIJ followed by an injection. Frequently this may not be curative for the patient's overall picture it does seem to improve the patient's function. As I tell my patients the old adage from long ago is true… The spine bone is connected to the hip bone.

*Article reprinted from SYNERGIES 2018

Fascia: A Target to Relieve Pain Using Osteopathic Manipulative Treatment

By Jennifer Jamrog, DO

Osteopathic Manipulative Medicine (OMM) is a branch of medicine that incorporates the belief that our body has self-healing capabilities. Its structure and function are interrelated and in order to achieve optimal health, it is important to incorporate the physical body, mind, and spiritual health into a patient's care. This model of care aligns with the mission of Logan Health Wellness & Pain Management's focus on interdisciplinary care.

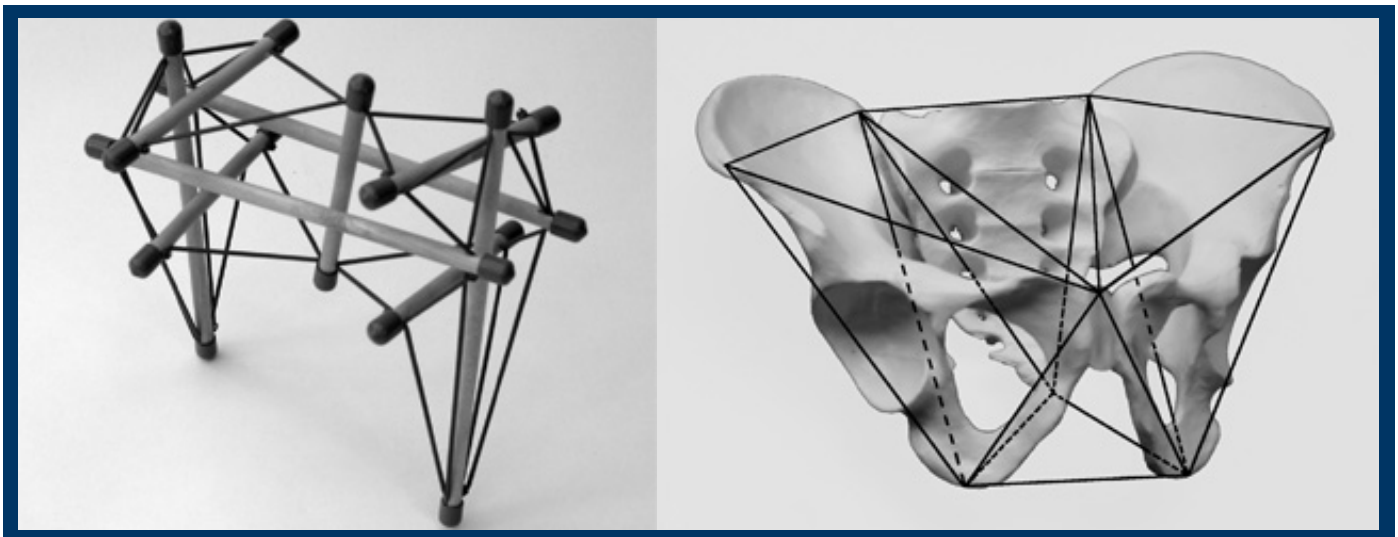
Some osteopathic physicians practice Osteopathic Manipulative Treatment (OMT), which is hands-on care that can help the body restore normal function. The physician will use his or her hands to diagnose and treat using various modalities that can address the musculoskeletal, lymphatic, nervous, cardiovascular, respiratory, and gastrointestinal systems. OMT can help many different patient populations with many different conditions. It can be gentle enough to treat a newborn baby or an acutely ill patient in the hospital, and can also be strong enough to treat an elite athlete. OMT is often used to soften and relax soft tissues, improve anatomical symmetry in the body, and improve pliability within the fascia, ligaments and tendons. In addition, it can improve removal of metabolic waste and supports the body's natural healing ability.

Tensegrity (tensional-intergrity) is defined as "the characteristic property of a stable three-dimensional structure consisting of members under tension that are contiguous and members under compression that are not."

Historically, the properties of fascia have been neglected scientifically. However, recently there has been an increasing interest in fascia by the research community. Fascia is a connective tissue made of irregular wavy collagen, fibroblasts and extracellular matrix. The fascia provides the tensegrity matrix for the whole body. It wraps around all other tissues as one spiraling membrane. In this manner, it both differentiates and integrates all of the structures in the body. Fascia manifests as a complex multilayered and multidimensional network of expansive sheets, enclosed containers and specialized attachments. It surrounds our organs and holds them in place, surrounds our blood vessels, nerves, bones, muscles, muscle groups, and each individual muscle fiber.[1,5] It spiderwebs through our subcutaneous fat in order to keep it arranged in an organized fashion. Dura mater, which is made of fascia, encloses our brain and spinal cord that contain the cerebral spinal fluid. The dura harnesses these structures to the skull, vertebrae and sacrum.

Myofasical release is an osteopathic manipulative technique that I often use in my practice. Many of the other traditional OMT techniques including muscle energy, counterstrain, soft tissue, lymphatic and cranial osteopathy can also address fascial restrictions and improve the fluid permeability from one body compartment to another. There is a belief among manual therapists that we have an embryological drive toward health and by removing mechanical obstructions in our bodies, we can achieve more optimal health. In addition, after physically removing myofascial restrictions, lymphatic outflow can be normalized and anatomical alignment can be improved.[1,4]

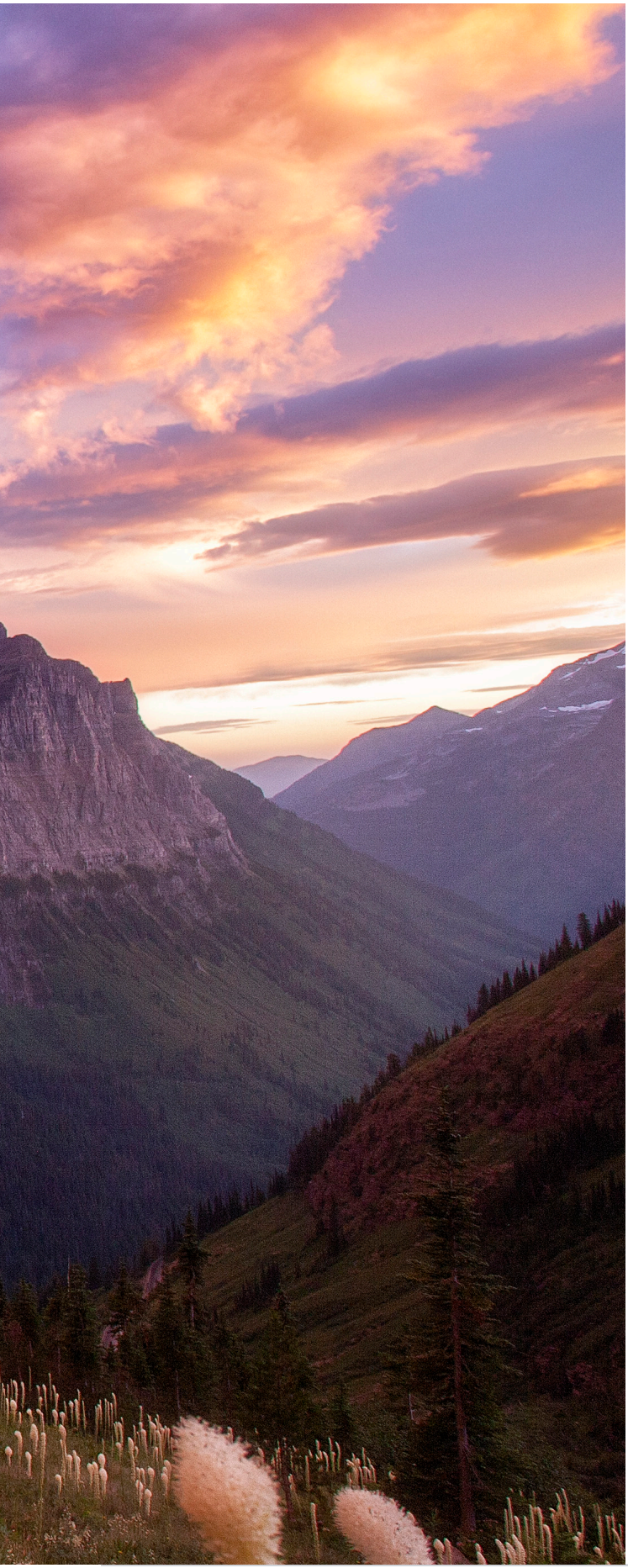
The Fascia Research Congress, formed in 2007, has been engaged in discovering the unique properties of fascia. Via histological study, there has been a recent discovery of contractile cells, free nerve endings, and mechanoreceptors within the fascial fabric.[1] Fascial planes have numerous connections that can transmit tension from one structure to a distant structure and all of the parts in between. Fascial creep refers to the time-dependent tendency of fascia to deform as the result of the application of a constant tension. Fascia has the property that if you stretch it hard and fast, its tension



Tensegrity model: If one of the cables in this model is stretched or cut, it will affect the shape (integrity) of the whole structure. This model can be applied to the human body to illustrate how an injury in one region can cause pain or asymmetry in a body part distant from the original site of injury. If the sticks represent our bones, and the cables represent our fascia, muscles tendons and ligaments, we can see how an injury or surgery could create compensation patterns that can change our bodies structural alignment, and potentially create pain compromising our functional abilities.

will increase markedly. If fascia is stretched slowly, it will respond with less tension and will deform to the shape of least resistance. In addition, we have discovered that when a fascial fibroblast is stretched the water inside is expelled toward the extracellular matrix.[2] When the tension is withdrawn, fibroblasts return to their original size and reabsorbs the water. The movement of water to the outside of the fibroblasts increases the stiffness of the fascia.[2] Constant excessive tension on fascia can lead to the extracelluar shift of water, which can lead to fibrosis. This "dried out" fascia, however, can remain functional depending on the ability of the cells to produce the appropriate extra-cellular matrix.[2] Also, fascia can direct the flow of lymph and blood to various areas via the contractile myo-fibroblasts which is involved in tissue healing.[5]

Fascia in the human body can develop restrictions in many different ways. Poor posture, surgery, inactivity, repetitive motion, and physical trauma, can all contribute to the development of fascial restrictions. In addition to OMT, many yoga poses address these fascial connections. However, many patients with chronic pain are deconditioned and these poses must be modified in order to avoid injury. Our clinic offers several movement classes that can complement OMT. Physical therapy and home exercises can also improve outcomes when used concomitantly with OMT. The benefit of OMT continues well after the treatment has ended. Often, patients feel less pain after receiving OMT, allowing them an opportunity to participate in gentle exercise or stretching, which can enhance lymphatic drainage of metabolic waste. It is well understood that when patients with low back pain or osteoarthritis stop moving, the disease progresses and pain worsens. I tell patients that my goal is not for them to passively receive OMT several times a year for the rest of their lives. My goal is to empower them to be comfortable within their bodies so that they can autonomously use movement to promote their own healing. I am happy to be part of the team at Logan Health Wellness & Pain Management to help patients in their quest to find pain relief, increased flexibility, movement potential and most importantly, improved quality of life.



References:

1. Bordoni B, Zanier E. “Understanding Fibroblasts in Order to Comprehend the Osteopathic Treatment of the Fascia.” Evidence-Based Complementary and Alternative Medicine. 2015; Article ID 860934
2. H. M. Langevin, M. Nedergaard, and A. K. Howe, “Cellular control of connective tissue matrix tension,” Journal of Cellular Biochemistry, vol. 114, no. 8, pp. 1714–1719, 2013.
3. M. E. Kramp, “Combined manual therapy techniques for the treatment of women with infertility: a case series,” Journal of the American Osteopathic Association, vol. 112, no. 10, pp. 680–684, 2012.
4. H. Chaudhry, R. Schleip, Z. Ji, B. Bukiet, M. Maney, and T. Findley, “Three-dimensional mathematical model for deformation of human fasciae in manual therapy,” Journal of the American Osteopathic Association, vol. 108, no. 8, pp. 379–390, 2008.
5. B. Bordoni and E. Zanier, “Skin, fascias, and scars: symptoms and systemic connections,” Journal of Multidisciplinary Healthcare, vol. 7, pp. 11–24, 2013.

*Article reprinted from SYNERGIES 2017

Ouch...My Back Hurts! Evaluation and Treatment

By Mark Haven, PA-C

Evaluation of low back pain without sciatic pain

Simple back pain with no sciatica pain occurs in 60 percent of patients. Patients are typically under age 50 with no signs of systemic disease and no history of cancer. Pain will usually improve with four to six weeks of conservative care. If no improvement is seen after six weeks, obtain plain film and erythrocyte sedimentation rate (ESR).

Complicated back pain without radiculopathy occurs in 37 percent of patients. Patients are typically over age 50, show signs of systemic factors including fever, weight loss, history of prior cancer, hematuria, adenopathy or injection drug use.

After obtaining plain film and ESR, consider MRI or CT if either are abnormal. Close follow-up is warranted, especially in a patient with known cancer and new back pain or in patients with IV drug use, fever and back pain.

Evaluation of low back pain with sciatic pain

Radiculopathy occurs in three percent of patients with low back pain. Symptoms of radiculopathy may appear without bladder or bilateral findings. May also have systemic signs noted in complicated back pain.

Treatment and diagnosis includes obtaining plain films, and, in some cases, ESR. If findings are normal, continue with conservative care for four to six weeks. If no improvement is noted at six weeks, request MRI or CT. Patient meets criteria for subacute low back pain, if no improvement after 12 weeks.

Urgent back pain usually occurs in less than one percent of patients. Exam findings may include acute radiculopathy with urinary retention, saddle anesthesia or bilateral neurologic symptoms. Patients may also exhibit progressive motor weakness or systemic symptoms and risk factors.

Urgent back pain requires an immediate consultation and MRI or CT to evaluate for cord or cauda equina compression.

*Article reprinted from SYNERGIES 2018

Therapy modalities

- Physical therapy
- Physical Medicine & Rehab
- Chiropractor
- Acupuncture
- Medications
- Injections
- Ice, heat, TENS unit, stretching
- Neurosurgery

Medication options

- OTC pain relievers/NSAIDS
- Oral steroids/prescription NSAIDS
- Muscle relaxers
- Gabapentin or Lyrica for neuropathic pain
- Narcotics (avoid)

What is Electromyography?

By Donald Stone, MD

For many patients and even some referring providers, electromyography (EMG) is a black box – a study viewed in terms of its inputs and outputs (or transfer characteristics), without any knowledge of its internal workings. The term EMG has at times applied to the entire electrical study and at times only the needle exam. Logan Health has had a longstanding commitment to providing comprehensive EMG services and making the process accessible to both providers and patients.

Today, EMG can best be thought of as part of a larger electrodiagnostic (EDX) medicine evaluation. At the core of the EDX evaluation are nerve conduction studies (NCS), electrical stimulation and recording from peripheral nerves, and needle EMG, and electrode evaluation of muscle electrical properties. The EDX consultant selects from a growing menu of tests to best assess nerve and muscle function and answer the clinical question.

Like all medical evaluations, the EDX assessment is only of value viewed in the clinical context. The studies should be considered an extension of the clinical assessment and do not replace a careful history and examination. Because there are hundreds of muscles and numerous nerves that can be studied, the EDX evaluation must be individualized after review of the history and laboratory data, and a directed neurologic examination and limited differential diagnosis are achieved.

There are several potential goals for the EDX evaluation. Unquestionably, the most important goal is to localize the site of the neuromuscular disorder. EMG/NCS and ancillary tests are powerful in determining if a process involves motor neurons, nerve roots, plexus, peripheral nerves, neuromuscular junction or muscle. Some electrodiagnostic abnormalities can indirectly point to a central nervous system disorder. By isolating the level at which the neuromuscular system is affected, EDX can readily distinguish the weakness caused by motor neuron degeneration in amyotrophic lateral sclerosis (Lou Gehrig's disease) or from the primary muscle weakness seen in thyroid disease, for instance.

Anatomic localization has obvious implications when nerve surgery is being contemplated, and numbness of the index finger could be due to either carpal tunnel syndrome or C6 nerve root compression from a herniated disc. At each level of the neuromuscular system, the anatomic patterns may inform us about the underlying nature of the disorder. As an example, damage to individual nerves may reflect nerve entrapment (internally, like carpal tunnel syndrome) or compression (externally, like peroneal nerve injury at the fibular neck) or a focal lack of blood supply (vasculitis). In contrast, diffuse damage to peripheral nerves can point to a systemic disorder like diabetes or toxins like alcohol overuse. Patterns of muscle involvement can help point to a hereditary rather than acquired muscle disorder.

Electrodiagnostic abnormalities can also tell us about the physiology of neuromuscular disorders, providing clues about the underlying diagnosis. In peripheral nerve disorders, for instance, preferential slowing of nerve velocities over loss of response amplitudes can tell us that the nerve coating, or myelin, is primarily affected rather than the axon. This finding has major implications for diagnosis and treatment, as there are far fewer peripheral nerve disorders primarily affecting myelin, with the many of them having specific treatments directed at the immune system. Testing of the neuromuscular junction can reliably isolate the problem to the nerve or muscle side of the connection, pointing strongly to the culprit disease.

In spite of the great usefulness of EMG/NCS, there are some important limitations. While excellent at determining the site, physiology and chronicity of many neuromuscular disorders, they often do not determine an underlying cause and other means of evaluation are often required to arrive at a specific diagnosis. In spite of this, studies indicate that EMG/NCS do significantly limit the required scope of additional testing. EDX studies can be uncomfortable for patients, though the vast majority of patients rate the discomfort as mild. Finally, it is resource intensive for both time and finances.

With these issues in mind, the most useful EDX results are achieved when the goals for testing are most clear. Questions posed in neuroanatomic terms are best answered by the EDX physicians, as the testing is geared toward a neuroanatomic answer. More reliable EDX test results are achieved in patients with more consistent motor and sensory complaints. As with the bedside neurologic exams, more pronounced motor findings result in more reliable abnormalities than subtle sensory signs. Expect that in many circumstances, ancillary testing will be required to delineate an underlying cause or to direct treatment for the problem (e.g. MRI for nerve root compression). A clear understanding of these issues allows for a meaningful EDX evaluation and helps to open the black box.

*Article reprinted from SYNERGIES 2017

Compression Fracture Challenges

By Justin Shobe, PA-C, MCHS

When exploring treatment literature of spine compression fractures, contradictory results abound. This can create a frustrating decision-making process for practitioners treating patients with suspected acute compression deformity in the spine. At Physical Medicine & Rehab (PM&R), we provide consultation and management of a broad scope of spine complaints. This includes managing patients with compression fractures. In these cases, we work to optimize the use of diagnostics, medication, bracing and outpatient therapies. We also focus heavily on safety with activities of daily living and prevention of additional injuries. If these patients demonstrate neurologic deficits, we promptly refer to our neurosurgery partners within Logan Health Neuroscience & Spine. If compression fractures go untreated, further deconditioning can occur, increasing morbidity and mortality rates due to increased risk of pneumonia, deep vein thrombosis and pulmonary embolism, among others.[1] The preparation of this article involved the review of several updated meta-analyses and consensus statements with the purpose of creating a lucid approach to acute compression fractures. This paper will provide a brief overview of the most common causes and presentations of compression fracture, and the most accepted management approaches.

Anatomy: In most humans, the spine consists of 7 cervical, 12 thoracic, and 5 lumbar vertebrae. Each vertebra transfers the load of the body's mass along the bony column of vertebrae. The vertebral bodies in the thoracolumbar spine includes 3 vertical columns (anterior, middle, posterior). Intervertebral discs separate the vertebral bodies, longitudinal ligaments are found on the anterior and posterior side of the column. The vertebral arches consisting of the pedicles and laminae create the passageway through which the spinal cord and spinal nerves travel, bathed in cerebrospinal fluid. Spinous processes are often palpable through the skin, and the transverse processes projecting laterally to provide important sites for muscle attachment, and in the case of the thoracic vertebrae, attachment sites for the ribs. Facet joints provide articulation between adjacent spinal levels. The spinal cord terminates in most people near the L1 level. The spinal nerves continue to traverse distally beyond the end of the cord, exiting the canal at their respective openings called neural foramen.[2]

History & Physical: Compressive injuries to the vertebrae themselves typically occur in the setting of trauma or pathological weakening of the bone. Osteoporosis is often blamed as a diagnosis that predisposes patients to compression fracture, but other causes for weakening should also be considered, such as malignancy and infection. Patients often complain of a sudden onset of pain near the site of compression fracture. Common mechanisms include forceful forward spinal flexion, lifting heavy objects while flexed forward, falling, coughing, and motor vehicle crashes. Pain can remain well localized, worsening with palpation and gentle manual percussion to the respective spinous processes and improved by gentle extension of the spine. Pain can also radiate into the paraspinous muscles, anterior abdomen, legs and the sacroiliac joint regions. Occasionally the patient's pain does not match the location of the fracture demonstrated on imaging, which often suggests injury to the adjacent soft tissue or nearby neurologic structures. "Red flags" are discussed in the table below. These warrant immediate magnetic resonance imaging (MRI) or computed tomography (CT) if MRI is unavailable, and surgical consultation.[3]

Diagnostics: Wedge shaped compression fractures most commonly involve the anterior column of the lower thoracic and upper lumbar vertebrae. Initial imaging for suspected fracture usually includes standing anterior-posterior and lateral x-rays. Fractures with greater than 50% loss in vertebral height or excessive kyphosis are generally considered unstable, warranting immediate surgical consultation.[4]

Computed tomography (CT) provides a detailed evaluation of the deformity, and may demonstrate fractures not identified on x-rays. MRI should be performed on patients with concern for neurologic compromise, such as motor or sensory deficits in the lower extremities. Spinal cord injury must be considered

in the setting of cervical and thoracic spine fractures. In addition to the 'red flags' described above, signs of upper motor neuron injury including brisk reflexes, ankle clonus and ataxia would warrant MRI cord survey to evaluate for cord injury. MRI can also provide valuable insight when evaluating the age of a fracture, particularly short tau inversion recovery (STIR) sequence images, which helps suppress the signal from fat. In compression fracture evaluation, injured vertebral endplates demonstrating hyperintensity on STIR sequence suggest inflammatory/edematous changes associated with recent or ongoing injury (bone marrow edema usually resolves in 1-3 months).[5] It should be noted that many compression fractures are found incidentally, with no correlating pain or acute findings on STIR sequence. MRI can help in estimating age of fractures when multiple deformities are present on x-ray. Obtaining an MRI may prove difficult in patients with severe pain and/or severe kyphotic deformity. The utility of this information should be considered carefully. In cases of lumbosacral and sacroiliac pain, attention should also be paid to the sacral ala and sacrum for possible insufficiency fracture.

Red Flags	Potential Underlying Condition
History of cancer, unexplained weight loss, immunosuppression, urinary tract infection, IV drug use, prolonged use of corticosteroids, back pain not improved with conservative management, fever.	Cancer or infection
History of significant trauma, minor fall, heavy lift in elderly or potentially osteoporotic patient, prolonged use of steroids.	Spinal Fracture
Cauda equina syndrome (acute urinary retention/overflow incontinence, loss of anal sphincter tone or fecal incontinence, saddle paresthesia/anesthesia) Global/progressive motor weakness in lower limbs.	Spinal Cord/Lumbar Nerve Root Compression

Adapted from Bigos et al.[3]

Malignancies and endocrine pathologies should be considered in the differential diagnosis, especially in cases of non-traumatic or widespread fractures. Initial screening should include a complete blood count (CBC), comprehensive metabolic panel (with attention to alkaline phosphatase and Calcium), prostate-specific antigen testing (in middle-aged and older men) and erythrocyte sedimentation rate (ESR), and parathyroid hormone (PTH).[6] Bone mineral density testing, such as dual energy x-ray absorptiometry (DEXA), can help diagnose and monitor osteopenia and osteoporosis. If DEXA has not been performed in the prior year preceding a compressive spine injury, this study can provide valuable information when determining the treatment route.[7]

TREATMENTS

Surgery: Surgical consultation should occur immediately in the setting of neurologic deficits and suspected instability. A thorough neurologic exam remains a critical component of the initial evaluation of possible compression fracture. Corticosteroids are typically started immediately as well.[6,8] In the absence of neurologic deficits or evidence of fracture instability, conservative treatments can be initiated, and steroids generally avoided.

Medications: In the absence of individual contraindications, non steroidal antiinflammatory drugs (NSAIDS) and acetaminophen serve as first line medications for treatment of compression fracture pain. Nasal calcitonin is also indicated for acute compression fractures, especially in the setting of an osteoporosis diagnosis. Calcitonin has primarily been shown to be effective in the acute setting, with no benefit to chronic pain complaints. Thus, initiation of calcitonin should occur within 10 days of the injury, and should be limited to 4 weeks.[9] A 2006 study suggested that calcitonin was superior to bisphosphonates for acute compression fracture pain management, and, because of its lower cost, calcitonin was preferred.[9] Still, bisphosphonates have been shown helpful for pain reduction in the setting

of severe pain associated with pathologic fractures.[10] Teriparatide (a recombinant form of parathyroid hormone) has been shown to decrease risk for chronic back pain in patients with compression fracture, as well as reducing the risk of additional fractures due to its propagation of bone mineralization.[11]

Loss of physical function can quickly lead to deterioration of mental health. Patients with severe pain related to a compression fracture, particularly in the geriatric population, are at 40% higher risk of developing depression like symptoms compared to their age related norms.[12] Thus providers must revisit mental health in these patients and provide adequate treatments (mental health specialty referrals, antidepressant medications, etc.) to avoid missing this important sequelae.

Opioids such as tramadol and hydrocodone can play an important role in the acute and subacute setting of compression fracture management, specifically in patients with symptoms refractory to the above mentioned pharmaceutical options. The patient's clinical picture including cognitive status, fall risk, gastrointestinal health, and respiratory status should again be considered carefully prior to administration. It is important to ensure the patient has a bowel medication regimen available if opioids are initiated.

Bracing: Bracing remains controversial in the literature.[13] Many providers universally employ bracing to reduce forward flexion and risk for further compression. When fitted appropriately, this can afford remarkable pain relief. Several prefabricated models are available. When possible, multiple prefabricated models should be trialed in the clinic to provide optimum comfort and fit. Braces often fit poorly, usually due to body habitus, lack of bracing education, or discomfort. These scenarios typically cause more harm than good, further increasing cost burden as well. Poorly fitted bracing systems can contribute to skin breakdown, worsened mechanics, myofascial pain, and worsening respiratory disease, among others.[14] Compliance is also predictably poor in these scenarios.

For patients who benefit from bracing, the literature recommends wearing during upright activities, and avoiding use while in a reclined or supine position of comfort. Tapering brace use can typically occur once they demonstrate spinal stability imaging, usually around the 6-8 week post-injury mark. This allows them to gradually increase the reliance on their musculature to provide support. Even in cases with ongoing pain, bracing is still typically limited to three months.[15] If pain remains severe after three months, vertebral augmentation and/or surgery should be considered.

In summary, due to the lack of high quality studies, the Academy of Orthopedic Surgeons (AAOS) cited, "A lack of convincing evidence to suggest for or against the use of bracing" in their 2010 recommendations.[13,16] At PM&R we recommend the use of bracing only if pain is improved for a period up to three months.

Minimally Invasive Techniques: Vertebroplasty (VP) and balloon kyphoplasty (BK) may be appropriate for patients who have failed conservative therapy for three months.[12,17] Failure typically indicates inadequate pain response to oral NSAIDs and narcotics, or contraindications to these medications.[18] These procedures may be considered sooner in the setting of painful fractures associated with metastatic disease. VP involves injecting a cement polymer into the fractured vertebral body. BK implements a balloon to expand the volume of the fractured segment, followed by infiltration with cement polymer.[12,18,19] Due to improved spinal deformity and increased vertebral height attributed to BK, the 2010 AAOS statement recommended for kyphoplasty and against VP in compression fracture patients who were neurologically intact.[13,19] Patients with painful compression deformities associated with metastatic disease may qualify for percutaneous thermal ablation when medical management provides insufficient relief.[20]

Physical Therapy: Once pain is stabilized, we typically initiate a physical therapy program aimed at promoting extension based activities, retraining mechanics for activities of daily living, and preventing additional injury. Studies have supported this practice, demonstrating improved pain levels in participating patients.[21] Despite the seemingly logical benefits of promoting range of motion and strength training, it should be noted that the 2010 AAOS treatment recommendations again were

"unable to recommend for or against physical therapy" regimens in the treatment of compression fracture due to the conflicting evidence and lack of quality studies.[13]

Conclusion: Compression fractures can cause chronic back pain at the site of injury. They can also alter the mechanics of the entire spine, causing changes in mobility and mechanics distant from the injury. Compensatory mechanisms often follow, further worsening the patient's ability to stand, walk, breathe effectively and change positions. Superimposed medical problems can arise rather quickly in the setting of decreased activity and inspiratory effort.[1,12] Thus, treating these patients appropriately can help potentially improve quality of life and maintenance of independence, as well as decreasing healthcare costs. We only recommend bracing when it improves pain and promotes activity tolerance. Alongside our neurosurgical counterparts, PM&R provides up-to-date diagnostic and therapeutic regimens in order to provide the optimal outcome in compression fracture patients. We look forward to continuing to employ best practices to best serve the Flathead Valley and beyond.

References:

1. Radvany, M.G., Murphy, K.J., Millward, S.F. et al. Research reporting standards for percutaneous vertebral augmentation. J Vasc Interv Radiol. 2009; 20: 1279–1286.
2. Martini FH, Nath JL, Bartholomew EF. Fundamentals of Anatomy and Physiology. 10th ed. San Fransisco: Pearson; 2015.
3. Bigos SJ, Bowyer OR, Braen GR, et al. Acute low back problems in adults. Clinical Practice Guideline No .14. AHCPR Publication No 95-0642. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, US Dept of Health and Human Services. December 1994.
4. Alexandru D, So W. Evaluation and management of vertebral compression fractures. The Permanente journal. 2012;16:46-51.
5. Piazzolla, A., Solarino, G., Lamartina, C. et al. Vertebral bone marrow edema (VBME) in conservatively treated acute vertebral compression fractures (VCFs): evolution and clinical correlations. Spine (Phila Pa 1976). 2015; 40: E842–E848.
6. Sherman AL. (2018, Oct 13). Lumbar compression fracture. Medscape. Retrieved September 29, 2019 from Medscape.
7. Wong C.C., McGirt M.J. Vertebral compression fractures: A review of currentmanagement and multimodal therapy. J. Multidiscip. Healthc. 2013;6:205–214. doi: 10.2147/jmdh.S31659.
8. Klimo, P. Jr. and Schmidt, M.H. Surgical management of spinal metastases. Oncologist. 2004; 9: 188–196.
9. Laroche M, Cantogrel S, Jamard B, et al. Comparison of the analgesic efficacy of pamidronate and synthetic human calcitonin in osteoporotic vertebral fractures: a double-blind controlled study. Clin Rheumatol 2006;25(5):683–686.
10. Lopez-Olivo, M.A., Shah, N.A., Pratt, G., Risser, J.M., Symanski, E., and Suarez-Almazor, M.E. Bisphosphonates in the treatment of patients with lung cancer and metastatic bone disease: a systematic review and meta-analysis. Support Care Cancer. 2012; 20: 2985–2998.
11. Suk KS, Lee HM, Moon SH, et al. At least one cyclic teriparatide administration can be helpful to delay initial onset of a new osteoporotic vertebral compression fracture. Yonsei Med J 2014;55(6):1576–1583.
12. Mazanec DJ, Podichetty VK, Mompoint A, Potnis A. Vertebral compression fractures: manage aggressively to prevent sequelae. Cleve Clin J Med 2003;70(2):147–156
13. McGuire R. AAOS Clinical practice guideline: the treatment of symptoatic osteoporotic spinal compression fractures. J Am Acad Orthop Surg. 2011;19(3):183–184.
14. Hoshino M, Tsujio T, Terai H, et al. Impact of initial conservative treatment interventions on the outcomes of patients with osteoporotic vertebral fractures. Spine (Phila Pa 1976) 2013;38(11):E641–E648.
15. Chang V, Holly LT. Bracing for thoracolumbar fractures. Neurosurg Focus 2014;37(1):E3
16. Esses SI, McGuire R, Jenkins J, et al. The treatment of symptomatic osteoporotic spinal compression fractures. J Am Acad Orthop Surg 2011;19(3):176–182.
17. Venmans A, Klazen CA, Lohle PN, Mali WP, van Rooij WJ. Natural history of pain in patients with conservatively treated osteoporotic vertebral compression fractures: results from VERTOSII. AJNR AM J Neuroradiol 2012;33:519-21.
18. Shah LM et al. ACR Appropriateness Criteria Management of Vertebral Compression Fractures. J Am Coll Radiol. 2018 Nov;15(11S):S347-S364.
19. Zhao, D.H., Chen, K., Zhu, J., Yang, X., Dong, F., and Wang, W.B. Postoperative functional evaluation of percutaneous vertebroplasty compared with percutaneous kyphoplasty for vertebral compression fractures. Am J Ther. 2016; 23: e1381–e1390.
20. Georgy, B.A.) Metastatic spinal lesions: state-of-the-art treatment options and future trends. AJNR Am J Neuroradiol. 2008; 29: 1605–1611.
21. Giangregorio LM, Macintyre NJ, Thabane L, Skidmore CJ, Papaioannou A. Exercise for improving outcomes after osteoporotic vertebral fracture. Cochrane Database Syst Rev 2013;1 CD008618.

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