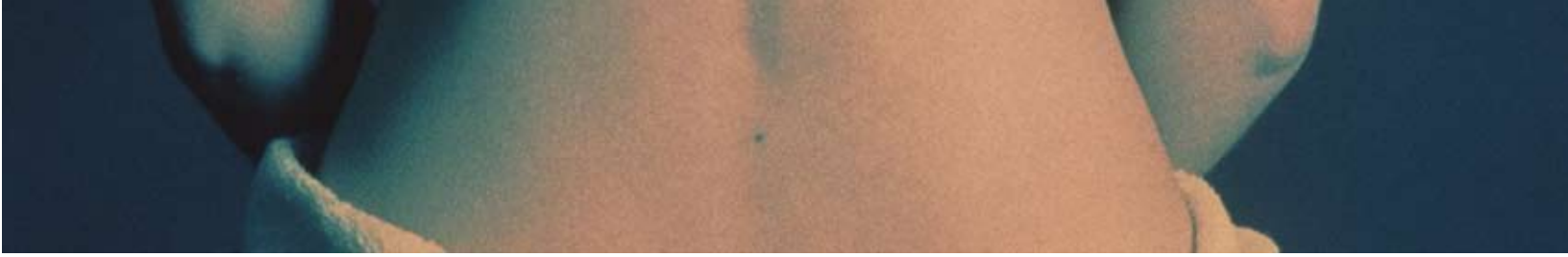

Unraveling the Complexities of Back Pain Through More Specific Examination and Diagnosis

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Back Pain: We're Still in the "Dark Ages"

Typically, discussions of back pain focus on its prevalence in society or its economic impact. Data concerning back pain have been collected for thousands of years. There are references to back pain in the code of King Hammurabi of Babylon (1955–1912 B.C.), as well as in Edwin Smith's Egyptian papyrus writings, which may date back even earlier.[1,2] These constructs are the underpinnings of today's laws pertaining to work-related injuries, malpractice, and even managed care.

While legal and societal issues concerning back pain have continued to evolve, our understanding of back pain has not. With all that modern science and healthcare have to offer, how can something as simple as back pain continue to be such a mystery? To some extent, we are still working from a scroll of papyrus. If we consider how greatly science has progressed in the last fifty years in terms of the understanding and treatment of many illnesses or diseases, perhaps it is time to change how we assess, diagnose, and treat back pain.

Pinpointing the Problem: Taking Back Pain Seriously

Back pain is not a disease or an injury, it is a symptom! Diabetes is a disease. A fracture is an injury. Yet whenever we speak of back pain, it is categorized as a disease or injury. When patients complain of chest pain, for example, healthcare providers go to great lengths to determine the specific cause of the pain. They tailor a treatment focused on correcting the underlying problem. Unfortunately, this is not the case when patients complain of back pain.

Although there are clinicians who perform thorough and detailed examinations, most patients relate that their examinations are brief at best. In fact, there are patients who have been treated for back pain, yet have never had a clinician look at, touch, nor closely examine their back to identify the cause(s) for their pain.

A typical examination of the back involves having patients bend forward while the clinician checks their reflexes (patellar and Achilles), assesses their strength through dorsiflexion of the foot and sensation on the dorsum of the foot, performs a straight-leg raising test (SLR), and then reviews or orders an X-ray or MRI. This examination is inadequate to determine the underlying cause of a patient's back pain. From this limited vantage point, however, treatment is offered in the form of NSAIDS and muscle relaxants; physical therapy or manipulation; and exercise or rest.

We need to take chronic back pain seriously. We need to take additional time to examine our patients and diagnose the underlying etiology(ies) with the same precision expected for a cardiac episode. We've known that potentially life-threatening conditions may be associated with acute back pain, yet we believed that the risk of mortality from chronic back pain was not as great. Recent literature shows that chronic pain can kill,[3] or at least adversely affect one's life span. For this reason, we must rely upon our learned skills and consider all the variables, including how and where a problem occurred. And we need to examine the physical manifestations of a problem to arrive at a working clinical diagnosis. Our most important "tools" are the patient's history and physical examination.

Arriving at a Diagnosis: Suggestions for Performing a More Specific Examination

The body is a complex machine with an internal monitoring system. There are various proprioceptive protection mechanisms that help to prevent

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further injury in the case of a malfunction. The body's first response to a problem with a movable segment is to stabilize the area involved. The body produces muscle spasms to restrict movement and prevent further injury.

With this enlightened view of back pain, our first step should be to look at the back itself. Visually examine the exposed back, palpate, and identify the muscle(s) involved. These muscle spasms might be the first indication of what the problem entails. Because pain is highly subjective and because routine clinical findings and imaging studies are frequently unremarkable, it's often difficult to identify the problem. So, when a patient demonstrates a profound spasm of a muscle that is not voluntarily controlled, you have identified criteria that support the patient's complaints.

Digital photography is a useful tool in documenting the existence of muscle spasms, and provides a graphic explanation of the clinical findings to the patient. Comparison of pre- and post-treatment photographs, can be invaluable in assessing outcomes. This is especially important in documenting the existence of a problem, such as a work-related injury where the veracity of the patient may be questioned, or when one is considering prescribing controlled substances in the absence of obvious pathology.

Identifying Common Pain Generators

There are many potential pain generators in the back. Common pain generators include disc pathologies (herniations, tears, and other degenerative lesions), nerve root compression (such as with foraminal stenosis associated with a disc herniation), muscle spasms, injury to ligaments, and inf ammation or other pathologies involving facet and SI joints.

One widespread misconception among patients and clinicians is that back pain results only from disc herniations or radiculopathy ("slipped discs" and "pinched nerves," from the patient's perspective). While nerve root compression usually results in significant pain, as well as sensory and motor complaints, the condition is not always present. Although disc bulges and herniations may be present, they are not always symptomatic. [4, 5, 6] Patients frequently relate that their back pain is caused by three bulging discs, yet they have no loss of function, they are without antalgic posturing, and they demonstrate minimal examination findings. If a patient were truly experiencing pathological problems with three discs (there are five in the lumbar spine), one would expect a more severe or dramatic presentation.

Muscle spasms are the third potential pain generator in the back. The muscle pain can be in the belly of the muscle itself, its tendon, or at the origin and insertion of the tendon (enthesis) as in an enthesitis or enthesopathy. What is the most effective means of identifying and quantifying this type of pain generator? Palpation! The next time you see a patient with back pain, plan to have an anatomical chart of the muscles or an anatomy textbook on hand so that you may precisely identify the muscles in spasm. In the case of the quadratus lumborum, which typically is one of the first muscles involved, you can palpate along the iliac crest, lateral to medial. When you reach the insertion site, a focal pain is likely to be elicited. You have just identified that an enthesitis of the muscle is likely resulting in pain lateral to the spine. The muscle then travels medial and cephalad, with the origin along the twelfth rib and the lateral processes of the upper lumbar vertebra.

Just as muscles can be strained (muscle strains are usually self-limiting and heal with or without conservative intervention), ligaments can be sprained or torn, and subsequently painful. In this instance, they can be considered primary pain generators. In the case where aberrant biomechanics exist as a result of spasm or other pathology, inf ammation can occur, with a secondary enthesitis. A common example involves the iliolumbar ligament. The iliolumbar ligament, extending from the inferior aspect of the lateral process of L5 to the medial aspect of the iliac

crest, is easily palpated in most patients. Ligaments can be considered the fourth potential generator.

Inf ammation or other pathology involving a facet joint(s) represents another cause of back pain. These articulations between the posterior aspects of adjacent vertebrae (also known as apophyseal joints) have thin, loose, articular capsules, as well as a synovial membrane and menisci. Just like the knee, the facet joint and its components can be directly injured, resulting in pain. Aberrations from normal biomechanics and degenerative or arthritic processes can lead to facet-mediated pain. Loss of the normal intervertebral disc height, as characterized by spodylosis seen on imaging studies, may be "inert" from the standpoint of pain associated with the disc, but is likely to adversely affect the facet joint, resulting in facet-mediated pain.

Facet joints are hidden deep under several layers of muscles. In practice, part of what is palpated involves the multifidus muscles directly over the facet joints. Local guarding or splinting of the multifidus muscles is likely when the facet is an active pain generator. There will be pain and tenderness on local palpation of these muscles as well as the facet joints when facets are involved. To adequately assess the facet joints, palpatory examination must be done when the patient is in a standing position (weight-bearing), such as bending over the examination table, and again in a prone position (non-weight-bearing). Some structures are more readily assessed in certain positions than in others. Furthermore, the characteristics of muscle spasms may be altered in non-weight-bearing versus weight-bearing positions, improving the clinical yield of the examination.

Palpation remains a valuable component for the identification of facet-joint pain and other pain generators. However, there are other considerations in the determination of facet involvement. During range of motion, extension can result in imbrication of the facet joints, resulting in increased pain. Forward f exion would stretch the capsule, also potentially increasing pain. Although pain and limited motion may be indicative of a problem, they do not necessarily provide insight into the source of the problem itself. While there are general rules to observe, varying the manner in which range of motion is assessed can be of significant value. For example, one may differentiate a facet problem at L5-S1 from one in the sacroiliac joint (SI) by performing Adam's or belt tests (remembering that dorsolumbar f exion first occurs in the hips, then SI joints, followed by the successive lumbar segments). An increase in pain and reduction of range of motion may be indicative of an L5-S1 pathology. A decrease in pain coupled with an increase in ability to bend forward when stabilizing the pelvis is suggestive of SI involvement.

The SI joint is another potential pain generator in the back. Patients with SI pain typically demonstrate an abnormal gait and tend to favor the asymptomatic side, as long as the pathology is not bilateral. When the SI joint is inf amed, as in sacroiliitis, restriction in the translational movement typically occurs. This can be assessed actively by having the patient stand and elevate one knee at a time, and relying upon palpation to evaluate translational movement in the SI joints. Provocative orthopedic maneuvers should be used next to confirm or rule out suspected SI-mediated pain as well as any other musculoskeletal back problems. While there are many maneuvers to consider for the SI joint, a personal favorite is simply to have the patient repeat f exion and extension when sitting. In the seated position the SI joint and hips are stabilized. The patient with sacroiliitis may demonstrate a greater degree and ease of f exion with less pain when seated. As this test does not appear to have been described in the literature before, I would not mind if we call this "Glick's Test."

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Orthopedic and Neurological Testing

Provocative orthopedic and neurological testing are essentials tools for helping to further differentiate the causes of pain. It may come as a surprise that the popular SLR, also known as Lasague's sign (the patient lies supine, knee extended, and the examiner flexes the thigh by grasping the heel), is, according to John Mazion, "at best equivocal." How could the most popular orthopedic test for the low back be equivocal? The elicitation of pain on leg raising suggests a problem, without pinpointing where the problem lies. The SLR test can also be profoundly negative in the presence of many back problems. In order to lend value to the test, one can add other maneuvers to help increase the diagnostic yield.

Although there are dozens of potential orthopedic tests, let's start by reviewing some of the basics. First, make note of the angle of the leg when pain starts, and then note the quality of the pain, including guarding or grimacing by the patient. Repeating the SLR and keeping your hand under the lumbosacral region, pay close attention to the movement above your hand when the patient reports the onset of pain (Goldwaith's). Palpating or feeling what is moving when the pain is elicited permits differentiation between hip, SI joint, lumbosacral, and lumbar problems.

At this point, especially if you suspect SI involvement, try the bilateral leg-raise test. When raising the two legs together, less movement will occur in the SI, resulting in less pain and a greater degree of movement. If there is pain radiating into the extremity during the SLR, lower the extremity to just below the point at which the pain occurred, then slightly dorsiflex at the foot (Braggard's). Re-creation of the radiating pain suggests root tension, and therefore a radiculopathy. Since a similar response can be elicited with a tight hamstring, repeat the procedure, dorsiflexing only the great toe (Sicard's). This response is more likely to be positive with a radiculopathy and not with hamstring tightness. Especially if a tight hamstring is suspect, continue raising the leg, only flex the knee. After the leg pain abates, apply pressure on the hamstring. Pain suggests hamstring tightness. The absence of hamstring pain upon palpation, but the elicitation of pain when applying pressure to the popliteal fossa, is highly suggestive of nerve root compression (Bowstring).

I would also suggest performing a very useful, although rarely used test—the deep tendon reflex (DTR). We perform the patellar reflex, primarily to assess the function of L4 (technically, L3 and L4), and the Achilles reflex to assess the function of S1 (technically, S1 and S2). As the most common level of root compromise in the low back is often said to be L5, there is a specific L5 DTR involving the biceps femoris. Both the long and short heads can be tested, but it is difficult to perform this test with the patient seated. The reflex can be elicited with the patient lying prone with the knee flexed. This is the same position the patient would assume during palpatory examination of the back, or other provocative testing, such as Yeoman's (hyperextension of the leg with the patient lying prone, knee flexed, while the sacrum is stabilized, eliciting SI Pain), and Hibb's (lateral rotation of the leg, knee bent, while the pelvis is stablized, eliciting hip or SI pain).

There are maneuvers that are more diagnostic for symptomatic disc herniations. Two that are most helpful are accomplished by first asking the patient to hold his or her legs elevated after performing the bilateral leg-raise test. Next, ask the patient to raise and lower his/her legs (Leg Lowering and Milgram's). These tests are comparable to a Valsalva's maneuver (increase in intrathecal pressure) suggesting a disc herniation.

It would be worthwhile for even the most seasoned clinician to review a text of neurological and orthopedic testing. The out-of-print book by John Mazion[7] remains a standard, as does a more recent work by Joseph Cipriano.[8] Even the Egyptian papyrus writings of Edwin Smith describe an orthopedic maneuver: "If thou examinest (a man having) a sprain in a vertebra of his spinal

column, thou shouldst say to him, "Extend now thy two legs (and) contract them both again." When he extends them both, he contracts them both immediately because of the pain he causes in the vertebra of his spinal column in which he suffers." While the diagnosis in this instance was a "sprain in a vertebra," the preferred method of treatment was to "prostrate" (straighten or manipulate) the patient. This was the preferred method of diagnosis and treatment as early as 1700 B.C., and remains an effective consideration today.

Early Treatment Considerations

Having collected this additional information from your examination, the next step is to apply your clinical experience and deductive reasoning. There should be multiple features of the examination that point arrows in the same direction. You are now armed with the information to provide, or at least recommend, a treatment focused at addressing the pain generator, not the pain. To review, the pain generators we have discussed are: disc, nerve root, muscle spasms, ligaments, facet joints, and the SI joint. The list is actually longer, and complicating matters further, pain generators typically are not mutually exclusive. Any problem or pathology in the low back is going to result in muscle guarding of some sort. Patients with a disc compressing a nerve root resulting in radiculopathy are also going to have pain attributed to muscles and their tendons, especially the enthesis. The best example of this is the quadratus lumborum (QL) muscle spasm. In this case, the radiculopathy is primary, and the QL spasm secondary.

Trust your judgment and initiate treatment. Just remember that the golden rule of back pain is to start conservatively. Consider simple treatments first and apply those with increased complexity and risk only after conservative management proves ineffective. While improvement in the clinical picture of the patient serves to confirm your diagnosis, lack of improvement suggests that the initial impression was incorrect or incomplete, or that the prescribed treatment was ineffective. If progress is limited or not recognized, it is best to attempt another course of treatment. There is no clinical justification to continue any treatment that has failed to show clinical benefit.

Exacerbations in the patient's clinical picture may also prove invaluable in refining the differential diagnosis. For example, therapy that involves traction may aggravate a suspect disc lesion. Since we know that reduced axial load can be beneficial for a disc herniation, we may suspect that other structures, such as a facet pathology, are involved. Traction may also aggravate a symptomatic disc tear. Another example of failed treatment is the lack of an anesthetic effect from a selective injection, such as an intra-articular facet block. This would suggest that the facet joint was not a pain generator. There is much clinical value in a failed treatment.

Obtaining and Integrating Additional Clinical Information

Still unsure about the underlying cause of the pain? Armed with a more specific differential diagnosis, you can consider additional testing. This may involve imaging studies such as x-rays, MRI, or CT. Other important clinical tools typically involve functional studies as well., the best-known being electromyography (EMG) in conjunction with nerve conduction studies (NCVs). Somatosensory-evoked potential studies (SEPs), can also be of great value, though their use may be more controversial.

Having a background in research and a desire to "build a better mouse trap," I generally prefer to use SEPs because they can be invaluable in identifying minor and/or sub-clinical neuropathies. They can be more helpful than EMGs in identifying radiculopathies that are primarily sensory and pre-ganglionic. Furthermore, they do not have the time constraints associated with EMGs because they are quite reactive, capable of providing a real-time snapshot of nerve function.

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patients demonstrating nerve root inf ammation of varying degrees without evidence of compression. SEP results would demonstrate slowed latencies without evidence of amplitude attenuation, which would otherwise suggest compression. There is one caution here. In order for the SEP to have a diagnostic yield that is root-level-specific, the study would have to include the evaluation of segmental nerves (those having a primary innervation of one particular nerve root) or dermatomes (areas of the skin having innervations corresponding to a particular root level). Studies of mixed nerves, such as the tibial or common peroneal, are not root-level-specific. Without additional testing they are more diagnostic of a myelopathy or the involvement of a peripheral nerve.

When the results of additional testing are combined with the findings of the clinical examination, you are in a good position to determine a treatment focused on eliminating the pain generator(s). While imaging studies may not reveal evidence of nerve root inf ammation in the absence of other structural pathology, SEPs can do so reliably. Once the SEP has identified the nerve root damage, interventional therapies can be introduced. For example, in the case of a significant or moderate latency prolongation of a segmental nerve, a transforaminal epidural steroid injection could be considered. If the latency prolongation is mild and there is clinical evidence of facet involvement at the affected level, an intra-articular facet block might be warranted. Evidence of facet involvement without any sign of nerve root compromise on exam or SEP would suggest a medial branch block (considered a diagnostic block) as the procedure of choice. If the medical branch block is successful, an ablation (typically by radio frequency) could be considered.

A clinical misconception about EMG/NCVs is that a normal study rules out the presence of nerve root involvement. A positive EMG finding can provide valuable information about a pathology. A negative finding rules out involvement of the muscle, myoneural junction, and the portion of the nerve that innervates that muscle, but not necessarily rule out the entire root or a condition that is primarily sensory.

Practical Considerations

Some of the most dramatic resolutions of chronic back pain come through cooperative interdisciplinary treatment. Using the SI joint as an example, in the case of sacroiliitis, manipulation would likely be very painful and could even aggravate the problem. Even if manipulation or certain forms of therapy provide short-term benefit, the problem often recurs. If an interventionalist treats the same problem with an intra-articular SI injection, the anesthetic effect may confirm the diagnosis. In addition, the anti-inf ammatory medication would likely reduce the inf ammation. However, the problem tends to recur. On the other hand, manipulation or mobilization of the joint immediately following injection and while the anesthetic is present would not likely be painful, and could more effectively work to restore normal biomechanical movement within the joint. Thus, healing can be more effective if the aggravating pathology (aberrant function) has been corrected. Cooperative treatment is far more beneficial to the patient than treatments provided independently.

It is essential to monitor the patient's progress during the course of treatment and to be alert to changes in the clinical picture. When pain associated with radiculopathy subsides, only to reappear in the form of a less severe sacroiliitis, you should not be surprised. The patient may not voluntarily relate or recognize the change in his or her clinical picture, leaving you to assume your treatment was not effective, when it very well may have been. Hopefully, there will be an alteration in the clinical picture to coincide with the appearance of a secondary issue. In the event that secondary issues present themselves, the practitioner's skill is essential in determining the order of treatment.

In terms of radiculopathies, just because

patients have pain radiating into their posterior legs, their knees or otherwise, the disc lesions seen on their MRI are not necessarily symptomatic. Radiating pain can be myotonic, attributed to increased resting tone of a muscle innervated by the nerve that is compromised, or it can be nothing more than a spasm of the hamstrings. Muscle guarding in the back, such as guarding of the quadratus lumborum, tends to cause the ilium to tilt forward, placing increased resting tone on muscles in the extremity, as well as on the piriformis.

Mechanical back pain, especially sacroiliitis, results in muscle guarding that includes placing increased tone on the piriformis. The piriformis can entrap the sciatic nerve (usually the peroneal division) as it exits inferior, resulting in functional or secondary entrapment of the sciatic nerve. This means that a patient with sciatica can be suffering from a problem not even in the lumbar spine, such as peroneal nerve entrapment at the piriformis secondary to sacroiliitis. There is a method using an SEP to help confirm such a condition. In this case, the common peroneal nerve demonstrates prolonged latencies, without evidence of a nerve root problem seen in the evaluation of other nerves tested. Relying upon the SEP in a manner similar to which it is used during intra-operative monitoring, one can repeat the study, placing the patient's lower extremity in a position of antalgia with respect to the piriformis (external rotation of the leg with knee f exed), and then seeing the SEP improve or normalize. For such cases, we focus treatment on the sacroiliitis, and the peroneal neuropathy/piriformis syndrome heals by itself.

There are other peripheral entrapments that can affect the lower extremities as well. In the absence of a back pathology, these entrapments might be more evident. However, when they are concomitant with a mechanical back pathology, there may be a tendency to suspect a radiculopathy. While tarsal tunnel syndrome (compression of the tibial nerve in the tarsal tunnel) is likely the best known, Pecina and his coauthors cite at least 12 others.[9] Similar overlapping symptoms can occur in patients with other neuropathic pain syndromes, such as the sequela associated with a diabetic peripheral neuropathy. It is essential to have a thorough understanding of the patient's relevant clinical history to unravel concomitant or complicating problems.

I regularly see patients who have been diagnosed with a low back problem, where failed treatment was focused on the sciatic distribution, primarily L4, L5, and S1. On many occasions these patients demonstrate problems more cephalad, with symptoms manifesting more caudally in the form of muscle spasms, especially when the erector spinae muscles (iliocostalis, longissmus, spinalis, and even the quadratus lumborum) are involved. Hands-on physical examination skills are essential to the differential diagnosis in this scenario.

Patients can experience pathologies extending from T12-L3 as well. In the case of radiculopathy, pain or other sensory symptoms may extend in the abdomen, groin, and anterior thigh. Differential diagnosis of upper lumbar pathologies may represent more of a clinical challenge, because lower lumbar segments tend to be the focus of most orthopedic maneuvers.

This discussion would not be complete without mentioning referred pain. It is well known that diseases of the internal organs may be accompanied by referred pain, hyperesthesia, and tenderness in somatic areas with common root-level innervation. As one may expect, patients experiencing abdominal pain may demonstrate musculoskeletal problems in the lower thoracic and thoracolumbar spine (the segments from which the sympathetic innervation of the upper abdominal organs arises). Patients can experience various degrees of discomfort in tissues in the pelvic area, such as vulvodynia, coccydynia, dyspareunia, and chronic prostatitis, which likewise may manifest in the lower back. The hypothesis that there is a connection between abdominal/pelvic pain and back problems has received varying support, although it is in accord with

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experimental models and clinical studies concerning viscerosomatic ref exes. The stimulation of receptors in some trigger areas of the viscera (via afferent nerves to the spinal cord) might spread to the corresponding dermatomes and myotomes, causing alterations in skin sensitivity, segmental spinal tenderness, and intervertebral aberrations. In such cases, the back pain could be regarded as referred pain accompanied by alterations in somatic structures, accomplished via viscerosomatic ref exes. Viscerosomatic or somatovisceral ref exes may also be activated by nerve root irritation at the intervertebral foramen, involving both somatic and visceral afferent fibers, or by trigger areas in cutaneous and subcutaneous tissues, muscles, tendons, or ligaments. Although patients with these symptoms may represent a minority, viscerosomatic or somatovisceral pathologies remain an essential differential diagnosis, especially in patients whose treatment outcomes have been less than desirable.

Conclusion

Acknowledging that back pain is the symptom, not the disease is the first step toward realizing that many patients suffer from chronic back pain because of the clinician's failure to diagnose the underlying cause of their pain. Given the diversity of treatment options among the disciplines, it is highly likely that by working together we will be able to arrive at a more specific diagnosis of underlying pain generators. If we succeed, we will be able to treat many more patients more effectively.

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