Orthopedics 2.0
Internet Book Version 1.0

How Regenerative Medicine will Create the Next Generation of Less Invasive Orthopedics

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First, this book is part of a two decade quest to find what I have termed, the “Unified Field Theory” of the musculoskeletal system. Physicists have long sought a single “theory of everything” that ties together all other theories into one grand explanation of the universe. I’ve sought to do the same for the musculoskeletal system, as theories abound about how to diagnose and treat joint, muscle, tendon, ligament, and spinal problems. Orthopedic surgeons have a surgical approach, family practice sports medicine practitioners another conservative approach, chiropractors their own alternative approach, physical therapists yet another.

Within chiropractic, physical therapy, and alternative medicine, there are literally hundreds of wholly different theories about what’s wrong with the body and how best to address these problems. Having studied many of these, I always found a kernel of truth and some interconnection between them. As the research in this area has become more robust in the past 20 years, many of these concepts can now be vetted by scientific observation, rather than intuitive guesses.

This book contains my own theory of how the musculoskeletal system works and a way to organize that information for both doctors and patients. The reader should note that while many of the components of this new theory are supported by rigorous scientific research, the whole package as I present it hasn’t been studied using what
doctor’s call “Level I Evidence”. This type of medical evidence means that randomized controlled trials have been performed and the treatment approach has found to be effective. Having said that, most of what we do today for patients with musculoskeletal problems is not supported by level I evidence. This includes joint arthroscopy, microfracture surgery, labral repairs, all spinal surgeries (including fusion, laminectomy, and discectomy), tenotomy, realignment surgeries (high tibial osteotomy, lateral releases), rotator cuff repair, ligament repairs, arthroscopic and surgical debridement, chiropractic adjustments, acupuncture, massage, most all physical therapy, just to name a few. These surgical and non-surgical approaches all lack the type of rigorous scientific support (Level I Evidence) that shows they are effective. In fact, when some of these procedures have been studied in controlled trials, they have often been shown to be no better than placebo surgery or no surgery (arthroscopic knee debridement is the most recent procedure shown to have no benefit).

This is an internet book. What’s that? The science behind many of these concepts would create an unwieldy publication that would be too difficult for patients to read and follow. By publishing this book on the internet, I can easily hyperlink to scientific abstracts and other references so that the reader can delve deeper into any subject, or simply read the basic explanations. In addition, my goal is to allow patients to submit questions and feedback so the book can be updated and improved. The internet allows this kind of flexibility, hence the version designation in the title (starting with version 1.0 and adding to that with each minor or major change). As the book is updated on the internet, the version number will allow the reader to know if he or she has the most updated information. Making this book better is as simple as clicking a link. To submit questions or ask for clarification requests for any part of this book, click here to send an e-mail to the author.

What is Orthopedics 2.0? In particular, orthopedics 2.0 doesn’t refer to the discipline of Orthopedic Surgery nor its successor. While orthopedic surgery may well be used as a part of Orthopedics 2.0, Ortho 2.0 has a bigger focus beyond just fixing one part of the musculoskeletal system (bone, joints, muscles, tendon, and ligaments). While the focus of this book is non-surgical, there will always be situations where the best approach is surgical. What will likely occur over the next 1-2 decades is a slow and steady movement towards less invasive orthopedic type procedures. This is identical to what’s occurred in other areas of medicine such as cardiology, with fewer more invasive open heart surgeries and more x-ray guided catheter procedures.
Ortho 2.0 represents the shift from joint salvage to repair. When the focus shifts to repair, the amount one needs to know increases exponentially. The pyramid above outlines what we use to evaluate the musculoskeletal system. While I use stem cells in daily practice, it’s important to note that helping patients is often not as simple as injecting magic stem cells. This book details the system our clinic uses to decide which procedures and therapies to apply as well as the way we look at joints, muscles, nerves, bone, tendons, and ligaments.

The problem with repairing the musculoskeletal system is its complexity. Think about your car. You know that there are critical components to keep it running. The wheels have to be aligned or the car won’t go straight and the tires will wear unevenly. The connections between the wheels, the axel, the driveshaft, and the engine have to be flexible and allow fluid movement. The engine as it turns the drive shaft has to be well oiled. As the engine cranks up to ever faster speeds, the connections had better be stable, or the whole thing will fly apart. Finally, your engine now has miles of wiring and small computers on board to monitor the whole thing and to regulate the activity of the engine, brakes, gasoline usage, and monitoring systems.

Now think about your body and its bones, joints, muscles, tendons, ligaments, and nerves. The same principles of alignment, good joint connections, stability, and sound wiring (nerves and minicomputers that impact everything from the timing of muscle firing to the information about joint position) apply. Regrettably, our surgical approach to date has too often just focused on bringing the car into the shop to replace a few worn parts, but not considering how the parts got that way. Let’s look at that analogy now as it applies to a person. If a 40 or 50 something patient that runs every day is suddenly diagnosed with right (and not left) knee arthritis, shouldn’t we ask ourselves why was only the right knee impacted? Could it be that for years the right knee was getting worn down due to poor alignment? We’d all accept that at face value that a misaligned front wheel and axel could cause the right front tire to wear faster than the left. Yet for some reason, our medical care system often ignores why one joint wore out faster. The reason? If the plan is to replace the joint, who cares? What if we wanted to save the joint? Would it matter more? Absolutely. This is the reason for the Ortho 2.0 approach and this book. When the shift is moved from replacement to repair, it matters how the joint got that way, if the joint is stable, if the surrounding muscles are firing correctly to protect the joint, whether the alignment is correct to support a healthy joint, and if the wiring is in order.
While stem cells are a great advance and represent a cutting edge tool, their use without considering all of these other things doesn't get patients where they want to be, which is a joint they can count on for many years to come. In this book, we’ll look at all of the parts of the Ortho 2.0 paradigm listed above, or as my partner coined the term, SANA. This stands for Stability, Articulation, Neuromuscular, and Alignment.
“In all of Shakespeare’s plays, no matter what tragic events occur, no matter what rises and falls, we return to stability in the end.”

Charlton Heston

Stability

What does it mean to be stable? Stable in a mechanical sense means resistance to falling part or falling down. For your body, joint stability means that the surfaces of the joint are kept in proper alignment during movement. Why is this important? When the joint surfaces uncontrollably crash into one another and can’t be kept in good alignment, the joint wears down much faster. An unstable joint literally experiences many times the wear and tear of a stable joint. Since stability in many joints is the number one determinant of whether that joint will have a long happy life or become “old” before it’s time, it’s a wonder more time isn’t spent accessing this component of joint health.
Let’s define joint stability a little further. Most of the stability that we currently focus on is called “surgical stability”. This means that a joint (either a peripheral joint like the knee or a spinal joint) is very, very unstable and unable to hold itself together at all. In these cases, surgery is often needed to stabilize the joint. Examples would be a completely torn ACL in the knee or severely damaged ligaments in the spine where a spinal cord injury is feared if the spine isn’t surgically stabilized. A surgically unstable knee may need a new cadaver or artificial ACL ligament implanted through surgery, while a surgically unstable spine may need a fusion where the spine bones (vertebra) are fused together with additional bone. There are two major types of instability, surgical and non-surgical. Surgical instability is less common than it’s more prevalent cousin, non-surgical instability. This more common type of instability often doesn’t require surgery and is characterized by small extra motions in the joint beyond the normal range. This type of instability has also been called “sub-failure” instability, since it doesn’t involve the joint falling apart. This is the focus of this chapter; a discussion of what causes sub-failure instability, how it might be helped, and why it’s so important when one considers joint repair or salvage rather than replacement. Our understanding of sub-failure instability is younger and more immature, so while we have some diagnostic tests to detect this type of instability, our understanding of what is normal and abnormal is only now coming into focus. However, this type of instability is quite real and it’s a clear long-term insidious drag on joint health. A good example of this is the research showing that replacing an ACL ligament in the knee, will lead to earlier and more significant arthritis in that knee joint. Why? While surgeons take great care to make sure the replaced ligament is identical to the torn one, there is no way to ensure the replacement ACL has exactly the same specs as the original. The new ligament can be too tight, too loose, or simply not have the identical load bearing characteristics of the original equipment.

**Sub-failure Stability 101**

There are two types of sub-failure instability; passive ligament stability and active muscular stability. Passive ligament stability keeps our joints from getting badly misaligned. Ligaments act as the living duct tape to hold our joints together. An example would be an ACL ligament in the knee, which keeps the femur bone from slipping forward on the tibia bone. Without this ligament, every
step would cause the joint to experience a potentially damaging shift. Active muscular stability is what happens when we move and represents stability fine tuning. Our joints tend to want to slip slightly out of alignment as we move, even with intact ligaments. As this happens, signals are sent to selective muscles that surround the joint to adjust and correct the alignment. Without this active system, our joints (especially ones like the shoulder that are dramatically dependent on muscle stability) would be “sloppy”.

One of my favorite authors on stability, Panjabi has a great diagram that sums up normal stability of any joint. Consider the diagram to the left, the red ball in a blue cup. It represents what happens when you move any joint. Let’s start with your finger. Move it back and forth right now. As you move it only a little bit in a back and forth motion, there isn’t much resistance to motion (the ball on the bottom of the cup). If you move it a lot (bend it all the way back), there’s more resistance to motion. In fact, it eventually stops. This is because the ligaments that surround the joint (along with the covering of the joint, called the joint capsule), prevent this excessive motion and ultimately stop the joint from moving. In the picture above, the ball represents the amount of joint movement from the center. Like any ball in a cup, the ball naturally wants to stay at the bottom. This is what happens in a functioning joint stability system, the passive elements (ligaments) and active elements (muscles) work to keep the joint aligned (ball at bottom of the cup).

In the new diagrams below, we see what happens to the ball in two conditions. One is where the ball is kept in the middle of the cup. In this case, small forces act on the joint to keep the ball in the center (the joint aligned). This is the fine tuning that our stability muscles provide. They act as constant stabilizers for the joint, keeping it in good alignment while we move. Note that the resistance of gravity tends to keep the ball in the middle of the cup, just as our muscles keep our joint in good position. This area is called the “neutral zone”, or the correct alignment of the joint.
Now consider what happens as you bend your finger back again. The resistance goes dramatically up (it gets harder to bend the finger the further you bend the finger). The diagram to the left shows what happens as the ball ascends the wall of the cup; the resistance to continued motion increases. This resistance to motion happens because the ligaments kick in to prevent catastrophic damage to the joint.

So in summary, understanding stability is a bit like a ball in a cup. Our muscles provide constant input to the joint to keep the joint alignment fine tuned as we move (the ball in the bottom of the cup or the joint in the neutral zone). When the joint moves too much, the ligaments act as the last defense to prevent joint damage from excessive motion (the ball trying to move higher in the cup).

**Examples of Stability**

The low back is a marvel of stability engineering. The spine is made from a series of blocks that stack one upon the other and protect the spinal cord and nerve roots. These interlocking blocks (vertebra) use the same stability model as described above—muscles and
ligaments. These stability models also apply to all of your joints like the knee, shoulder, hip, elbow, ankle, etc...

Now let’s look at how the spine stays stable. What happens when you place a bunch of kid’s blocks, one on top of the other? This tower of blocks gets less stable as the pile gets higher (see right). One way to stabilize this high tower of blocks would be to tape the blocks together. This would make the blocks more stable, but wouldn’t allow much motion (see left). You could use more rubbery and flexible tape than say duct tape or scotch tape, but again you’d either end up with providing too little stability (highly elastic rubbery tape that gives a lot when you stretch it) or too much (duct tape or scotch tape).

Imagine if we had to move all of the taped blocks so that they could all bend into a c-shaped curve? (see right) How stable would your blocks be then? The right kind of tape (ligaments) would likely allow this motion, but the individual blocks would start to shift against each other. This shifting could result in disaster, as the spinal cord runs in a hole inside the blocks (spinal canal) and the spinal nerves exit between the blocks through a special bony doorway (foramen). As a result, too much movement between the blocks means nerve damage or worse, a spinal cord injury. This is the dilemma of the spine, how to stack lots of blocks (about 25 high in most people) while keeping the whole thing stable and flexible. Is there a solution? Yes, the muscles provide that solution. They keep the blocks (vertebrae) aligned against one another while you bend and twist.

**Muscular Stability in the Spine**

If muscles help solve the problem of keeping the blocks aligned against each other while the spine moves, which muscles are these? For most of the spine, this special muscle is called multifidus. These small muscles travel from vertebra to vertebra to keep the spine bones in proper position against each other. In the 1990’s, some very smart scientists noted that these muscles
were smaller than usual in most patients with chronic low back pain. Since that time, atrophy (when the muscles get smaller and weaker) of these and other spinal muscles is a known cause of low back and leg pain. These muscles can be seen on almost all MRI’s and numerous research articles have correlated shrinkage of these muscles and back or leg pain, but regrettably this atrophy is almost never commented on in radiology reports. So to make sure that your spine stays aligned and isn’t constantly bumping into spinal nerves, these muscles have got to be firing on all cylinders.

**Muscular Stability in Joints**

Probably the joint most reliant on muscular stability is the shoulder. The shallow socket of the shoulder means the ball of the humerus has lots of opportunity to get out of place. The rotator cuff muscles help to keep the ball in the center of this shallow socket. Other joints rely on similar control mechanisms. Some joints have more intrinsic stability because they have deep sockets (like the hip) or are tightly bound by ligaments (like the knee). However, the goal of the muscles crossing the joint is the same, to keep it aligned while the joint moves. Despite different designs, all joints are dependent on muscles to fine tune their alignment.

**What Happens when the Muscular Stability System goes Down?**

When the muscles fail to align the joint, the joint becomes sloppy. Too much movement is allowed in all the wrong directions. This causes excessive joint wear and tear on the joint (arthritis), whether the joint is a disc in the low back or the knee joint. Obviously, the first way to help this problem would be to strengthen muscles around the joint. However, just getting the stability muscles stronger sometimes isn’t enough. Sometimes the nerves telling the muscles what to do don’t work well. In cases like this, no amount of strengthening will help until the nerve issue is addressed.
Training muscles for stability can take a lot of different forms. First, the ligaments have to be strong enough to be able to handle normal body forces. Second, the muscles have to be able to contract normally and unfettered by neuromuscular problems (see chapter 4). Finally, the muscles can be trained for coordination using one of a number of therapies (click here to see stability therapies).

A Dramatic Example of What Happens when the Spinal Stability System is Off-line

Several years ago I went for a several hour mountain bike ride. I had just read a paper that discussed how factory workers showed weaker back stabilizing muscles after being in a bent forward posture for several hours. Regrettably that day my own back would become an example of this phenomenon. Right after the ride I went to pick up my son, felt a pop in my back, and went down to the ground. It took days to recover and for the first 48 hours I was in agony. I’ve felt the same thing occur a few more times, most notably once while dead lifting weights in the morning (after sleeping in a flexed posture all night) and at other times. These are all dramatic examples of what happens when stabilizing muscles are turned off. In my case, the lumbar multifidus was turned off from the spinal nerves that supply these muscles being irritated in that flexed posture. This lead to a period of instability in that spinal segment, a time during which I was more likely to get injured. Essentially that spinal segment was left completely unprotected by the spinal muscles.

Why Physical Therapy Sometimes Fails

The past 1-2 decades of supervising rehab programs for patients has taught me that while strengthening the muscles often helps the muscular stability of the joint, sometimes it fails. Why? The muscles are controlled by nerves. While much of traditional medicine has focused on big problems in nerves that can be picked up on static imaging like MRI or on electrical tests like an EMG (Electromyogram-a test where they stick needles into the muscles...
and give you electrical shocks), the more recent research shows that a lot can go wrong with nerves that is generally invisible to these tests. This smaller amount of spinal nerve root irritation can wreck havoc with the muscles by shutting parts of them down. When a part of a muscle gets shut down, it may not be responsive to strength training. These shut down muscle areas are called trigger points. An easy way to get rid of these tight and weak areas of the muscle is trigger point massage, or direct pressure on the area. When this fails, a trigger point injection or dry needling of the spot (IMS or Intra-muscular Stimulation) is often the answer. In our experience, this can allow that part of the muscle to work again and start helping the joint to stabilize. This concept is discussed further in chapter 4.

**Hypomobility versus Hypermobility**

So far, we’ve been discussing the concept of too much movement, or hypermobility. Equally important is hypomobility, or where a joint or spinal segment doesn’t move enough in all directions or certain directions. This is what chiropractors and osteopaths have been focusing on for years. The reason we M.D.’s have given them a hard time, is that hypomobility has been traditionally hard to measure. However, there is good evidence now that this does occur. In fact, studies that specifically apply this concept (hypermobility versus hypomobility), show that patients with spinal hypermobility treated with exercise do better than patients with hypomobility. This makes sense, as if you have too much mobility you need to get the muscular stability system back on line with exercise or other treatments to restore muscle function.

However, patients with hypomobility did poorly with stability exercise. Why? They need more mobility, not more stability. This group did better with manipulation to force these segments to move.

The take home message is that if your spinal or peripheral joints don’t move normally in all directions, you have to get them to move normally or this will place more wear and tear on the certain parts of the joint. Take for
example this model of a joint and the muscles that help control that joint’s movement. We have a ball in socket type joint with a ball sitting in a shallow socket (like the shoulder). Here we’ll call them muscle A and muscle B. Both muscle A and B pull equally on the joint. When one pulls harder, the opposite muscle lengthens equally, to allow the joint to move.

So as this joint moves, the ball stays in the middle of the socket (or the neutral zone as we’ve discussed). What happens if one muscle can’t release as the other pulls? Now the joint moves too much to one side, banging into the side of the socket. This is an example of muscular hypomobility, where knots or trigger points in one muscle makes it tight and weak (more on this topic at this link). How do you fix this? You need to loosen up the tight muscle. This is discussed further in the neuromuscular section. The same thing can happens if one part of the joint capsule (the thick fibrous covering of the joint that helps to limit motion) is too tight or the ligaments that hold the joint together are too tight.

Can Strong Muscles Substitute for a Bad Ligament Stability System?

As discussed, there are two types of stability systems, the fine tuning is provided by the muscles while the ligaments prevent serious abnormal joint movements that can lead to catastrophic joint damage. If the ligaments are stretched out a little bit, but still intact, the muscles may be able to substitute and protect the joint in most situations. However, if the ligaments are stretched or damaged so that they allow bigger abnormal motions in the joint, no amount of muscular stability will help. In the end, while having stronger stability muscles may help reduce some of the wear and tear, the joint will still get into abnormal alignments that
will lead to accumulated damage. So if ligaments are stretched, it’s best to tighten them (this can often be done without surgery-see the section on prolotherapy) and if they’re completely torn, the only option may be to surgically replace the ligament.

**Micro instability: A Constant Drag on Joint Health**

It’s important to note that most sub-failure instability might not be felt as the joint giving too much in the wrong direction. These small amounts of extra motion are called micro instability and while any one or ten events might not lead to injury, they can have a big impact over long periods of time. Even an extra millimeter of motion, when repeated 10,000 times, can damage a joint. As a result, often the best way to look for these small amounts of extra motion is a good physical exam by a physician trained to look for these small amounts of extra motion. The American Association of Orthopedic Medicine is a good place to find such physicians. This group provides educational seminars for doctors interested in treating instability with injection therapy. Other types of tests are described below.

**Bone and Joint Tissues are Alive**

We’ve come to think of bone as inanimate cement. However, bone is made up of mature cells (osteoblasts) and stem cells that react to their environment. It’s well known for instance that when the cushioning cartilage in a joint wears out, the bone underneath the worn out cartilage makes itself thicker to handle the new forces. We know that people who don’t exercise or pursue non-weight bearing exercise have more brittle bones and that people who lift heavy weights have more dense bones. Bone is alive and quickly reacts to its environment. How quickly? As an example, for many years most physicians were convinced that bone spurs in the spine took years to form. This was based on the bone as dumb, inanimate cement theory. However, more recent research shows that
when the lumbar discs are injured as part of an experiment, bone spurs begin to form in the 1-2 month time frame.

The same holds true for muscles, tendons, ligaments. They all react to increased strain forces by making themselves thicker and stronger. This ability to react quickly to increased (or decreased) demands is mediated in part by adult stem cells. The switch from seeing these orthopedic tissues as inanimate filler (bone, cartilage) or pieces of inanimate duct tape (ligament, tendon) to living tissues that react, is a key concept in understanding why alignment of the joints is so important in orthopedics 2.0.

**Functional Bone Spurs?**

Bone reacts to forces. I’ve spoken to many patients over the past few years that are planning on surgery to remove bone spurs. While I can think of a few situations where this makes a lot of sense (like when a bone spur is pressing on a nerve or severely limiting range of motion). I call these non-functional bone spurs. However, in many situations, bone spurs are functional. What does this mean? Let’s take the example of bone spurs that develop in a knee. The knee has cartilage and a fibro cartilage meniscus, both of which help absorb shock. The meniscus component also acts as a spacer to help keep the joint surfaces apart. When the meniscus is healthy, it stays within the joint (see picture at left). When the meniscus gets degenerated or pieces of the meniscus are removed surgically, the meniscus starts to migrate out of the joint (see top picture to the right). Since bone is alive and reacts to these forces, the body responds by placing bone in this area to take advantage of this new meniscus position (see bottom picture to the right). This response is called a “bone spur” or “osteoaphyte”. We’ve been conditioned to believe that all bone spurs are bad. However, as you can see here, these bone spurs allow the knee to take advantage of this new meniscus position and continue to use the spacer (meniscus) to absorb shock. If we remove these bone spurs, the knee loses its ability to absorb shock and the body will just place more bone spurs in this location. I call these “functional bone spurs” in that
they serve a purpose and their removal doesn’t positively impact the joint. Since all bone spurs are a reaction to instability or joint forces, we have to be careful about removing this reactive tissue, to make sure that the joint will be better off after removal.

How do I know if I have a Stability Problem and What can I do to Help it?

How do you know if you have a stability problem? Patients often complain of popping or cracking in the spine or joints. They may at times feel sudden shifts in the spine or affected joint. For example, when performing cutting exercises, they may feel their knee “give way”. In the neck, patients may feel that by the afternoon or evening they have a “heavy head”. In other patients, there may be no perceptible sense of instability or popping/cracking in the joint, just joint pain or swelling after activity. These patients usually have smaller amounts of micro instability.

What tests can diagnose the problem?

Spine: In the spine, larger amounts of instability can be seen on either low back or neck flexion-extension x-rays. These are tests where the patient looks down and up or bends forward and backward while x-ray films are taken. Regrettably, too often, the technicians that take these films don’t push patients far enough into these positions (flexion or forward, extension or backwards). If you undergo one of these tests, make sure to push yourself in these motions (without hurting yourself). The research shows that such efforts can reduce the false negative rates of these tests (a false negative is a test that fails to show positive even though the patient has the disease). A newer form of this test is called Digital Motion X-ray (DMX). This test looks at a moving x-ray view of the spine as the patient is put through various ranges of motion. It can also be used in various peripheral joints such as knee, shoulder, elbow, etc...

Testing smaller amounts of instability in various areas can often be done at home or with a physical therapist. The links below are not meant to substitute for an experienced
medical provider or physical therapist, but more to give the patient a sense of how stability can be addressed. Home stability tests and home exercise links:

- **General Spine or core stability**
  - **Low Back:**
    - Basic
      - Transversus abdominis
      - Multifidus
    - Advanced - levels 1 and 2, levels 3-6
  - **Stability with walking**
  - **Shoulder stabilization exercises and more of a gym work-out**
  - **Ankle stability exercises, more advanced exercises**
  - **Cervical:** The basic exercises are low level strengthening for severely unstable patients.
    - Basic - Deep neck flexors level 1, level 2, level 3, level 4
    - Advanced - The focus is on neck and shoulder extensor stability. These are for neck/shoulder stability.

Peripheral Joints: For peripheral joints, the most common x-ray tests for stability are for the shoulder AC joint and the ankle. **For the shoulder, the x-rays are taken with and without the patient holding weights.** For the ankle, the nerves may be anesthetized with a numbing medicine and then the ankle turned with an x-ray taken at maximum excursion. The goal of both of these tests is to see too much movement in the joint with stress. As a result, they are often called stress radiographs (another word for x-ray).

For smaller amounts of micro instability (very common) we use high speed cameras to observe the joint under high speed loads. These cameras can often show a sudden shift in the wrong direction or a “sloppy joint”. For example, a pitcher who may have elbow instability can be imaged while throwing at speed. The video can be slowed down 5-20 times to see if the elbow bows or bends inappropriately during throwing. Another method for detecting smaller amounts of instability in the knee is a KT-1000 athrometer.
This is a machine that replicates the physical exam for small amounts of laxity in the ACL ligament.

While diagnostic tests for instability and hypomobility are just becoming popular, the best way to diagnose these problems is still history and exam. An experienced physician can compare joint motion from side to side (good side versus bad side) as well as stress the joint to look for signs of instability. As discussed above, the AAOM is a good place to look for doctors experienced in diagnosing smaller amounts of instability. Many orthopedic surgeons can also diagnose instability, but realize their focus will be on the larger amounts of instability that have been described as surgical instability.

**What therapies might help?**

Spine: The lowest level spinal stability training is using ultrasound imaging or other biofeedback devices to help patients contract their multifidus and transversus muscles in the low back. The same type of program can help with neck instability. This program is called deep neck flexor work (strengthens longus colli and longus capitus), and has research to show its efficacy in helping headaches.

The next level of difficulty for spinal stability is usually where most programs start. If this level of therapy makes things worse, then either it’s too advanced for the level of muscle atrophy or there are other issues that have to be addressed (like damaged ligaments, irritated nerves, painful joints). For the low back, the oldest such program is called Dynamic Lumbar Stability or DLS. In the neck, once patients have mastered lower level neck strengthening exercises like deep neck flexors, we have used the BTE multicervical unit with some success in many patients.

Peripheral Joints: Stability programs for the joints can be broken down by area:

Shoulder: Rotator cuff exercises can be helpful; these are very commonly prescribed and taught by most physical therapists.

Knee: Knee stability exercises are also commonly taught in many physical therapy programs. The kinesotaping programs are a good bet and combine taping to provide better proprioceptive feedback from the joint being trained. This method can also be used for other joints.
Ankle: rocker boards, B.A.P.S. boards, and other unstable platforms can be used by therapists to help the leg muscles provide more efficient stability in the ankle.

Stretched ligaments causing unstable joints can often be helped without surgery by Prolotherapy or PRP injections. This will be discussed further in the next chapter.
“The universe as we know it is a joint product of the observer and the observed”

Teilhard de Chardin

Articulation

Articulation means joint. This could be any joint like peripheral joints (knee, shoulder, hip, ankle, elbow, wrist, etc...) or the joint between two spine vertebrae the disc. While peripheral joints are generally different than a spine disc, they share more in common than not. Both spinal and peripheral joints allow motion and do so in a controlled manner. A joint has certain standard components:
Cushioning- In the peripheral joints, the cushion is usually the cartilage or meniscus. In the spine, the middle of the disc called the nucleus pulposis serves as the cushion. Both of these are not inanimate pieces of rubber, but living tissues with cells and structure. Once these components die off, these joints lose their ability to provide shock absorption.

These are found in the shoulder and hip.

**Stability-** This is provided by the joint capsule (tough outer covering of the joint) or the ligaments that help hold the joint together. In the disc, this is provided by the tough outer covering of the disc and surrounding ligaments. In addition, many joints have another element called a labrum to hold a ball in a socket.

Notice that while our entire medical care system in orthopedic surgery revolves around joints, in the SANA system, the joint is simply one part of a bigger system whose other parts are equally important or more important. What are the implications of paying too much attention to the joint? Imagine that instead of being focused on the musculoskeletal system, we were concerned about the urinary system, but instead of considering the kidney, ureter, bladder, and urethra, all we focused on was the bladder? This is too often what we do today; we focus on the joint and exclude the surrounding muscles, tendons, ligaments, and nerves.

**Imaging Divergence: or the Very Poor correlations between Structure and Function**

Every patient I have ever met wants an MRI, which is a fancy picture of the soft-tissues that’s created by powerful magnetic fields. While our practice uses MRI’s to help define pathology, what if I told you that if you placed a bet that what’s on your MRI is causing
your pain that I could give you only 1:1 odds (50/50)? Let’s start with the most pervasive musculoskeletal MRI finding of the late 20th century, knee meniscus tears. If your doctor sees a meniscus tear on your MRI, it’s a sure thing that the meniscus tear is causing your pain, right? Wrong. A recent study published in the New England Journal of Medicine showed that about 60% of patients without a history of active knee pain have meniscus tears on MRI. This study was completed by the famed Framingham heart study group. They observed two groups of middle aged to elderly patients, with one group having recent active knee pain and the other having no recent or remote history of knee pain. Turns out they both had about a 60% rate of having meniscus tears on their MRI. This study calls into question the reasoning behind likely hundreds of thousands of knee surgeries performed over the past two decades. Since many of these tears aren’t likely causing the patient’s pain, why are we operating on them?

Other studies of structural problems on imaging and their correlation to pain have been equally disappointing. Several low back studies have shown that patients with severe problems on MRI are often pain free, while other patients with severe pain often have limited structural changes on MRI of the spine.

So how do we use MRI? First, we use it to look for clues about what’s happening inside the joint. Take for example this gentleman with severe pain on the outside of his knee. The lateral or outside of the knee can hurt for many reasons. The most commonly cited would be a meniscus tear, a cartilage problem, or a lateral collateral ligament issue. Taking those one by one, as stated above, meniscus tears have recently found to be common in patients without pain, so we need to take meniscus tears with a big grain of salt now (or at least confirm they are likely causing pain before just assuming they are causing pain). A cartilage problem would mean injury or degeneration of cartilage on the end of the lateral femur or the tibia. Finally, the lateral collateral ligament is the living duct tape on the side of the knee that helps to keep it stable, so an injury there can mean chronic pain and knee instability.
This 60 some year old gentleman had years of lateral knee pain when he entered into one of our research studies. His other knee had a stem cell injection into a frayed and torn meniscus and this not only helped to restructure the meniscus, but also got rid of much of the bone bruising that was caused by the bad meniscus. However, the knee that’s the subject of this discussion didn’t respond. Our initial focus of stem cell therapy was the medial meniscus, which looked horrible on MRI. However, he continued to tell us that most of his pain was lateral (where his MRI looked great). So we looked closer.

The 3.0 T coronal MRI fat saturation sequence on the last page shows a really bad medial meniscus off on the left of the image, but the patient only has a mild ache on that side. The lateral meniscus on the right side of the image may have some subtle signal changes, but no convincing tears or problems and he is without significant arthritis on that side. He’s was also not tender over that lateral joint line. So what’s causing his lateral pain? If we look closer, we see this (picture below):

Notice the bright area in the darker bone in the blown up image to the right (in the dashed white circle). This is swelling in the bone at the lateral tibia, just above where the fibula attaches. Turns out this is exactly where he’s tender, just at the bony origin of the ligaments that hold the fibula to the tibia. On the second round of injections, we focused here, with almost complete resolution of his pain.
This example brings up an important issue. As discussed earlier, meniscus pathology is common in patients without pain, so we need to take all meniscus findings with a grain of salt (great compilation of studies commented on in the NY times at this link). Studies have shown no difference in the amount of meniscus tears seen on MRI in patients with and without knee pain. However, that doesn't mean MRI is useless, it just needs to be combined with some common sense and the other MRI (my regional investigator or physical exam).

In summary, operating solely on MRI findings is not a good idea. On the other hand, in our experience these fat saturation sequences can often help sort through multiple issues seen on knee MRI, to find the one that the best candidate for the pain generator. We’ve seen knees with cartilage problems and associated swelling in the bone on fat sat images, while other issues in the same knee have no associated increased signal (and aren’t tender on exam). So in conclusion, always trust exam first, MRI is for correlation only. Or as a old friend told me once, “If you listen to the patient he or she will always tell you what’s wrong, if you listen to the patient long enough, he or she will tell you how to treat it…”

Cutting out Pieces of the Joint: Debridement or a Slippery Slope to More Rapid Arthritis?

A common technique to help “clean up” a joint is arthroscopic debridement. The concept is that the surgeon will cut out loose pieces of cartilage. In the knee this means cutting out any torn pieces of meniscus. While this may make some sense at face value, the tissue we’re removing in debridement is made up of live cells often critical for the overall health of the joint. Two large studies have shown that this surgery in the knee produces no better results than a fake placebo surgery or physical therapy. Why? We’re removing structure from the joint. Let’s take an example. Let’s say you owned a house where one day one of the walls started to crack and fall apart (like the meniscus seen on MRI), but is still structurally sound. You can repair the wall or remove the wall. Since you don’t have the technology to repair the wall (which is what
happens in many of today’s joint surgeries—words like “repair” are actually a misnomer, they often mean “cut out”), you decide to remove it. You may get some temporary benefit from removing the wall as it was an eye sore and perhaps it makes the house flow better. However, since it’s a load bearing wall (helping to hold up the second story), things in the long run get worse. The floor on the second story starts to sag and other walls begin to crack under loads they weren’t designed to handle. Before long, it’s clear that removing the wall was a bad idea. This is exactly what happens in many of today’s joint surgeries. In the knee, we remove chunks of meniscus with each surgery, despite the fact that research has shown that doing so means that arthritis will likely develop much more quickly.

**Understanding The Body’s Repairmen - Stem Cells**

Remember that house in your neighborhood inhabited by an older person who couldn’t keep up with the maintenance? We’d all accept at face value that a house left unattended for years will weather and begin slowly to degrade and fall apart. Our joints and bodies are the same. A quick run around the block, a work-out in the gym, or just daily use will cause micro damage in any number of tissues. Left unrepaired, these areas will begin to breakdown over time (just like the unattended house down the street).

So what keeps us from falling apart after just a few years? The figure on the previous page tells the story of the opposing forces of damage vs. repair. Everything we do everyday adds small (or large) amounts of damage or “wear and tear” on our tissues. On the other side of that coin is repair. This is the mechanism that fixes the damage.

Turns out we have billions to trillions of tiny little repairmen in all tissues of our
body. These repairmen are called adult stem cells. As an example, consider an adult stem cell type called a mesenchymal stem cell (MSC). These cells live in your tissues and are called into action once damage is detected. They can act as a general contractor in the repair response, giving signals to the body to bring in the other subcontractor cell types that are needed for the repair job. They can also “di f ferent iat e” (read turn into) the final cell type needed for the repair. For example, if the repair is needed for the cartilage of your knee, they can differentiate into these cartilage cells. When we're young, while there may be a lot of abuse on the body, in general, the amount of repair capability (adult stem cell numbers and function) generally far exceeds the amount of damage we can inflict. As we age we have fewer of these stem cells around. Even when we're younger, an area can become injured so that it doesn't allow the repairmen in the door (less blood flow or there just aren't enough cells to effect a proper repair). At this point, the amount of damage starts to exceed the body’s ability to repair.

What if we could turn that equation around? What if, despite being older or even younger with an area that has too much damage for the local repair cells to handle, we could amplify repair in the area? As you might have guessed, this is a basic tenant of Ortho 2.0. The doctor's job is to increase the local repair response so that it exceeds the existing damage or wear and tear on the area. In addition, the other part of that equation applies as discussed above. The other half of the doctor's job is to reduce the local damage on the area. How is this done?

**Improving the Repair Response**

Ortho 2.0 always involves trying to improve the body’s ability to repair a damaged area. I would divide these approaches into 3 levels of sophistication:

- **Level I: Micro-injury**
- **Level II: Improving the Healing Environment**
- **Level III: Stem Cells**
Level I - Micro injury

Ever since ancient times, creating a small injury to prompt healing has been seen as a good idea. For horses, this was called “pin firing”. The technique was to take a hot poker and place it into a non-healing ligament to cause small amounts of damage to the area, which caused the body to kick up a repair response. While barbaric, it generally worked. For centuries doctors have created small injuries in a non-healing wound by “roughing” up the tissues. Physicians still use this concept today for tendons, ligaments, and joint capsules. For example, in a shoulder capsulorrhaphy a surgeon usually inserts a small catheter that heats up to prompt healing in a damaged shoulder capsule (the covering of the shoulder joint that helps control motion). Surgeons still score ligaments with scalpels and needles in the technique called percutaneous tenotomy, also to prompt a healing response. Another example is micro-fracture surgery to fix a hole in the cartilage; this is where the surgeon pokes holes in the bone to cause the cartilage to heal. Finally, the procedure known as prolotherapy is in this same category. In this procedure, rather than a mechanical injury being initiated, the physician injects a chemical irritant to cause a chemical micro-injury. All of these types of treatment rely on the same concept that we get one bite of the healing “apple” and if something fails to heal completely the first time, we can get more bites at that apple simply by causing a small injury to the area.

The big advantage to micro injury techniques is that these basic procedures are simple and often inexpensive ways to try to get an area to heal. The downside is that while many times they work well, sometimes they don’t have enough oomph to get the right type of healing or enough healing.

Level I - More on Prolotherapy

Prolotherapy is an injection technique where chemicals are injected to cause a small inflammatory healing reaction. In the 1940’s, this was a mainstream orthopedic procedure used to treat lax ligaments and spinal pain. Heck, it even had its own pharmaceutical (Sanusol). However, as the next half of the 20th century progressed, prolo fell out of favor. Why?
Some say it was linked to the bad outcome of a single injection placed where it shouldn’t be in the spinal canal. However, others place prolo’s demise on the fact that it had no sustainable medical business model. It was simply replaced by big surgical procedures that were far sexier and which had better reimbursement. We may never know, however, over the past two decades, I've seen this simple and inexpensive technique work for patients who otherwise would not have been helped. I’ve published on prolo’s ability to tighten loose spine ligaments simply through injection and others have published on the same observation in lax knee ligaments.

Level II - Improving the Healing Environment

The next level of sophistication beyond just creating a healing micro injury is making the conditions in the area more conducive to healing or “anabolic”. You may have heard this term associated with body builders that use steroids. This is not the same use here, although body builders “build” muscle, so this is why they use “anabolic” steroids (literally steroids that build”). Here the term means making an area pro-repair or better able to heal.

Creating an anabolic healing environment is not a new concept in medicine and surgery. For centuries, physicians have known that some people have better innate abilities to heal, while others have less healing capabilities. The acronym “PPP” (pios spoor protoplasm) was used in my residency training to mean a patient that due to disease or extreme old age was unable to heal after surgery. While surgeons have always known that some patients could have a compromised ability to heal, not much attention has been paid in how to make routine and otherwise healthy patients heal better. Surgeons have always understood the basics, like good nutrition, young age, high levels of fitness, good blood supply, etc… About 20 years ago that started to change in the dental community. Some dentists began experimenting with a simple concoction called “PRP” or “Platelet Rich Plasma”. The dentists used this stuff made from the patient’s own blood to help their dental implants heal.

PRP is a simple example of how we can improve the healing environment. The blood has platelets which contain growth factors that help to ramp up healing. To understand
how these platelets work, a paper cut will illustrate the basic points. When we cut ourselves, we bleed into the cut. The blood coagulates because of little cells in our blood called platelets. The job of the platelets doesn’t stop there, they go on to release certain factors that help to heal the cut.

**Growth factors** are like espresso shots for cells. A cell works at a certain pace to do its job. If we add growth factors (like those in PRP), it’s like buying all of the cells trying to repair the area a bunch of Starbucks gift cards. The cells react to the growth factors like people react to triple espresso shots, they work harder and faster. So if we use an example of a construction site, where we have a few brick layers building a new wall, if we add growth factors, our brick layers will build our wall faster.

As you might have guessed, Ortho 2.0 uses these same concepts to promote healing. The most basic level II procedure today is PRP, which can be mixed up from a patient blood sample in a bedside centrifuge or in a simple hospital or clinic based lab. PRP means that the healing platelets have been concentrated. Injecting the patient’s own blood can often accomplish the same thing, as it’s also rich in platelets. In our clinic, we also use next generation level II tools beyond PRP. These include platelet lysate or PL. In the case of PL, our advanced cell biology lab makes PRP from the patient’s blood and then breaks open the platelets to make all of the growth factors immediately available. The big advantage to using PL instead of PRP is that PRP has to be mixed up each time we want to use it, but PL can be mixed up in a larger batch and stored indefinitely in a sophisticated freezing process known as “cryo-storage”. We also use various activated, incubated platelet supernatants or next generation PRP. Think of these as PRP 2.0. To make these next generation solutions, our lab starts with PRP, activates it with various natural substances, and incubates the platelets for several hours to several days. During this time the platelets are directed to produce certain natural growth factors that are important in specific types of healing. We then take that soup of natural, growth factors and use that instead of PRP. So for example, we can inject a solution that is rich in the joint and cartilage repair **growth factor TGF-beta** by telling the patient’s platelets to produce more of that natural growth factor.
Other level II techniques are cell concentrates, such as **Bone Marrow Aspirate concentrates** (BMAC). This is whole marrow (looks like blood) that’s extracted from the bone marrow using a needle and spun down in a special centrifuge or simple hospital or clinic lab to produce a cell concentrate. While BMAC does have orthopedic type stem cells (MSC’s), they are very few in number, making up only about 1 in 100,000 of the harvested cells.

**Level III - Stem Cell Therapy or the Adding in the General Contractors of the Body**

A general contractor (GC) is the person who pulls a house together. He or she hires subcontractors like plumbers, carpenters, and electricians. The GC’s of your body are **stem cells**. So level III advanced techniques use concentrated stem cells to help repair tissues. There are a number of different types of stem cells. We’ve all heard of **embryonic stem cells**, which are taken from a growing embryo. While these cells are very potent stem cells, they also have the nasty habit of forming tumors. Cells can also be taken from a donor who’s older than an embryo, such as **cord blood stem cells** or an adult to obtain **adult stem cells**. However, while some of these cell types might be appropriate as last ditch efforts to save someone’s life, their risk of transmitting genetic disease makes them too risky for orthopedic applications. **As an example, in one study, an older rat bred to have osteoporosis donated stem cells to a young rat without the disease.** The young rat acquired osteoporosis in the bargain. Since we currently don’t possess the technology to screen donors for all inheritable diseases, the risk of using someone else’s stem cells is too high for now (in my opinion). That leaves the patient’s own stem cells (**autologous**). There are many types, but for the purposes of this orthopedics discussion, one stands out as the best candidate for our GC position—the **mesenchymal stem cell**. These cells are found in many tissues. For orthopedic applications, their ability to help coordinate the repair response as well as turn into cartilage, bone, tendon, muscle, and ligament make them ideal. Other cells such as **Very Small Embryonic Like** or **Embryonic Like Stem Cells**
(VSEL’s or ELSC’s) are also promising for orthopedic use, but not enough research has been done yet on these adult stem cell types with regard to safety to make them practical for everyday use. Also realize that there are likely hundreds of classes and sub-classes of adult stem cells that will eventually be used for therapy. Many of these may even be combined with mixtures of other non stem cells to better promote healing.

How we deliver stem cells as part of ortho 2.0 makes a big difference. While delivery into an arm vein is attractive because of the low level of expertise needed to deliver cells, studies have consistently shown that adult stem cells delivered in this fashion are trapped in the lungs (pulmonary first pass effect). Of even more concern is a recent study showing that for patients considering the use of stem cells to treat CNS disorders, only about 1 in 200,000 cells injected via an IV route reaches the brain and central nervous system (1.5-3.7% made it past the lungs, 0.295% made it to the carotid artery, and 0.0005% made it past the blood brain barrier into the brain). At this point, until these pulmonary first pass issues are worked out, credible stem cell delivery is local. This means placing cells directly into the tissue or into the arterial circulation that directly supplies the tissue. In addition, for orthopedic applications (and likely for others), it’s hyper-local, meaning that placement of cells into one part of the joint may provide results; where as non-specific placement in the joint may provide less results.

The Opposite of Healing: Apoptosis

What’s the opposite of healing? Causing apoptosis, or pre-programmed cell death without any ability to heal. For many years doctors have injected high dose steroids because they quickly bring down swelling and make the area feel better. However, study after study continue to show that these drugs at the high doses that physicians often inject (milligrams) cause local pre-programmed cell death (apoptosis). While causing a little cell injury is not necessarily a bad thing (as
discussed above), steroids work by taking away the local repair response (inflammation and swelling), and so you’re left with an injured area that can’t repair itself.

Your body can release natural steroids into an area where the inflammation dial may be turned up too high, which turns down that inflammation dial just a smidge. How much is too much steroid? While the milligrams of steroid commonly injected by doctors might not seem like much, it’s about 100,000-1,000,000 times more steroid than your body would expect to see in the area. As an example, if the amount your body uses to control joint swelling is the height of a matchbook (nanogram range), the amount most doctors have been taught to inject is the height of the Empire State Building (milligram range). Or as I like to tell patients, if we inject the much smaller nanogram dose, we’re putting in a thumbtack with a ball peen hammer, but if we inject the much larger milligram dose, it’s like putting in the same thumbtack with a sledgehammer.

If you use the ball peen hammer, there won’t be much collateral damage, but using the sledgehammer is bound to create problems. Why don’t we see more doctors injecting the smaller physiologic doses? For one reason, they just aren’t available. Steroids for injection bought from a medical supply company come only in the much bigger milligram ranges. When we use the much smaller nanogram ranges, we use a special compounding pharmacy to create the medication. Despite injecting the much smaller doses, we usually see the same results (decreased swelling). In addition, research has shown that these smaller doses can increase the good growth factors in a joint associated with repair.

Why doesn’t my joint just heal on its own? - or Inflammation has Gotten a Bum Rap

“Inflammation”, you’ve likely heard the term in a negative way. Inflammation means swelling. You’ve likely heard that too much inflammation in our arteries may the cause of heart disease. You may have heard of rare syndrome where too much inflammation after a leg or arm injury can cause serious problems (compartment syndrome, where out of control swelling in a confined space can lead to severe injury). All of this is true, but for this chapter, you have to understand that like anything, there is also a good side to inflammation. Without inflammation, we could never heal ourselves.
We’ve all had a chronically swollen joint or seen people with joints that swell. The reaction from modern medicine has been to inject high dose steroids into these joints. As stated above, since high dose steroids are potent at reducing inflammation, this may seem at first to help. However, these ultra high dose drugs also destroy the natural repair response. So we now have a joint that no longer swells, but also has no ability to heal itself.

Why does a joint stay swollen? Swelling is the result of your body marshalling the troops to heal an area. All of the cell types needed to build new tissue are in the swollen area: cells to clean up the damaged tissue (macrophages), cells to recognize any foreign and deactivate invaders (white blood cells), and stem cells to act as general contractors in managing the repair response (mesenchymal stem cells). However, your body will keep throwing inflammation at the area (swelling), if the “were done here” signal is not received from the newly formed repair tissue. As discussed above, if there aren’t enough stem cells to complete the construction project, the done signal may never be received.

An easy way to think about swelling is that it’s like the oven that bakes the cake. After all, the term “inflammation” incorporates the Greek for the word for flame. When an area stays swollen and chronically “inflamed”, it’s like low level oven heat. If you place cake batter in a 200 degree oven, you don’t end up with a baked cake, rather dried out mush. Why? The chemical reaction that “bakes” the cake needs higher heat or said another way, to be more “inflamed”. Turn up the oven to 400 degrees and you get a cake. The same holds true for a chronically swollen joint. The low heat of chronic inflammation isn’t enough to repair the tissue, so the joint stays swollen. However, using the micro injury techniques above, we can “turn up the heat” and use much higher level healing inflammation to heal the tissue (or bake the cake).

So in summary, inflammation isn’t usually a bad thing in orthopedic applications. Swelling is necessary to heal. Doing things to get rid of swelling (the Rest Ice Compression Elevation or RICE mantra) or anti-inflammatory drugs may have their place in certain rare circumstances to prevent things like compartment syndromes. However, in orthopedics 2.0, the use of drugs like high dose steroids, NSAID’s (Motrin, Ibuprofen, Aspirin, Aleve, or other Non-steroidal Anti-inflammatory Drugs) to kill the healing inflammatory response (in my opinion), is generally considered a bad idea.
Should I take Anti-inflammatory to help my joints?  
- Medications that Adversely Impact Regenerative Orthopedics

Anti-inflammatory drugs have become a mainstay of orthopedic and musculoskeletal care. While we have discussed steroid medications, what about Non-steroidal Anti-inflammatory Drugs (NSAID’s)? These are medication that affect the pathways for inflammation, most of them work by inhibiting the COX inflammation pathway (*cyclo-oxygenase*). COX drugs help control swelling, but they also cause stomach ulcers by inhibiting the enzyme that helps to protect the stomach wall. Just how dangerous are these drugs? Moore in 2002 published that the estimated risk of death due to stomach ulcer when taking NSAID’s for more than 60 days was 1 in 1,200! While this represents only a small number of people who are very sensitive to this drug class, the number are concerning. As a result of these inherent dangers, newer drugs were designed to work against COX-2 rather than COX-1 (which is more responsible for stomach wall protection), but these drugs had a new set of side effects. These drugs (like Vioxx, Bextra, and Celebrex) all come with a new cardiovascular risk (risk of sudden death by heart attack).

How do NSAID’s impact healing? Well from a 50,000 foot view, inflammation is needed to heal, so blocking inflammation may inhibit healing. Sure enough, NSAID drugs like Motrin and others have been shown to delay healing. While most of this research has focused on fracture healing, we keep patients undergoing regenerative medicine treatments off these drugs.

Other drugs are also notable for causing musculoskeletal problems. The antibiotic drug class that includes Cipro (Quinalones) has been shown to lead to tendon ruptures. Heartburn drugs like Nexium have also been linked to hip fracture risk. The upshot? Many commonly used drugs can adversely impact regenerative medicine healing. Our own cell culture data implicates cholesterol and certain blood pressure drugs as causing problems with mesenchymal stem cell growth in culture.
Which Joint is Causing the Pain?

In joints like the fingers, it’s easy to see which one likely is causing the pain. The joints are easily palpable and you can press on any one and ask the patient if it hurts. However, there are many joints in the body that aren’t so easily palpable. Take for example the hip and the SI joint. Both can refer pain to the hip/groin area. It’s very hard to get your fingers near the hip joint as it’s buried in inches of muscle. As a doctor, you can feel the back of the SI joint, but most of it is buried inside the pelvis. How do you know which one is causing what the patient reports as “hip” pain? The only way to know for sure is to “block” one or the other (inject numbing medicine to kill the pain from one). Since the accuracy of the injection is important (meaning if the doctor injects the numbing medicine into the muscles instead of the joint, you’re no closer to solving the mystery), fluoroscopy (real time x-ray imaging) or ultrasound are used to place medication. There are many other joints where it’s difficult to tell if they are the “pain generator” (the joint that causing the pain). Other examples include the facet joints in the spine, the small joints of the foot and ankle, the multiple joints of the wrist, etc...

Regrettably, we’ve seen through the years patients who have been told they need a hip replacement based on a bad hip x-ray (maybe the right hip) when the left hip looked the same, but didn’t hurt. We then block the hip, only to have the patient report minimal pain relief. We then block the SI joint, and the patient gets complete relief of pain. So the moral of this story is, before consider major surgical procedures, be absolutely sure you know which joint is causing the pain.

What are the resources to treat my Joint?

As discussed above, MRI is a good place to start as an assessment of overall joint health, but the results can sometimes be misleading. It’s important to realize that MRI’s come in different strengths and these numbers determine how much the radiologist will be able to see. Think of these as like the differences between
resolutions in digital cameras. We all know that the camera on our cell phone is not as high a resolution as a professional SLR type digital camera with a fancy lens. The same holds true for MRI. These machines have designations using Tesla strengths. For today’s purposes, if the Tesla number is below 1.5, then the MRI machine will produce pictures of a lower quality. There are a few rare exceptions, such as the newer high field open MRI’s at 1.0 Tesla, which seem to produce reasonable pictures. To get the best resolution, look for an MRI scanner with a 3.0 Tesla resolution. In any big city, for every 100 MRI’s, there may be 5 that can meet this higher resolution standard. Also realize that insurers are generally agnostic about these numbers, so you will usually pay the same co-pay and deductible regardless of the resolution of the scanner.

As discussed in this chapter, seeking out regenerative techniques can be broken down into their level of sophistication:

Level I- The non-surgical level I technique that is minimally invasive is Prolotherapy. There are two resources to find a prolotherapist in your area. www.getprolo.com is a paid advertising site for prolotherapists, while the American Association of Orthopedic Medicine (AAOM) is a professional society that lists members who use this technique. For other surgical level I techniques such as micro fracture, tenotomy, and shoulder capsulorrhaphy, the American Academy of Orthopedic Surgeons is a good bet.

Level II- PRP is becoming a widely used regenerative medicine tool. The Total Tendon Network is a good place to start. You may also have luck just googling PRP plus your city name, as many doctors using PRP have web-sites. The ICMS has also initiated a treatment registry for PRP physicians who want to collect and share data about PRP complications and outcomes. Physicians who use BMAC are a little more difficult to find, but starting with the company that manufactures the bedside centrifuge to create BMAC is likely a good bet.

Level III- The ICMS maintains a treatment registry for physicians who use stem cell therapy as part of their practice. This organization also has promulgated strict clinical and lab guidelines and certifies both labs and physicians, so this would be the best place to identify physicians in your area practicing the more sophisticated level III techniques.
“True freedom is where an individual’s thoughts and actions are in alignment with that which is true, correct, and of honor - no matter the personal price.”

Bryant H. McGill

Alignment

In most patients, there is usually more joint damage on one side than the other. Why? If someone has a genetic predisposition to arthritis and this is the only factor causing the joints to degenerate, shouldn’t all joints be affected equally? In addition, osteoarthritis is more commonly seen first in the knees and hips and less often in the ankles and elbows. Why? Again, shouldn’t we see all joints being impacted the same? The reason is clear, the wear and tear on our joints occurs unevenly, with some being impacted more than others or one side undergoing more wear than the other.
Reducing the Wear and Tear

As already discussed, increasing healing ability is only one part of the ortho 2.0 equation. The other half of this coin is reducing the wear and tear forces that destroyed the joint in the first place. As an example, placing new tires on a car with bad alignment without fixing the alignment is guaranteed to quickly wear out the tires out again. This issue is often ignored in our current quick fix treatment methodology. I've seen hundreds of patients with a specific wear pattern like the right medial meniscus, where the most salient question has never been asked. How did this knee get like this and what are we going to do to ensure that it doesn’t get this way again?

The reason the issue of specific wear and tear patterns is mostly ignored is that it’s complex. Most physicians aren’t trained to understand the biomechanics of the body. The few physical therapists that have spent years of extra study learning biomechanics are often heavily incentivized by insurance companies not to take the time needed to figure out why a part keeps failing.

Let's use a simple example to illustrate this concept. The skeleton on the left has been drawn with red force arrows going down from the hips. Let's say that for an unknown reason, slightly more force is applied to the right ride (the thicker arrow) than the left side (the thinner arrow). We take thousands of steps a day. What happens to the extra forces on the right and how does the body handle them? The right hip, knee, and ankle will all react. They will initially just shore up the bone, tendons, ligaments, cartilage, and muscle on that side. When this person is young, with many adult stem cells in these areas, he or she may not notice much. However, as the number of adult stem cells begins to decrease with aging, the damage due to wear and tear at some point will begin to overtake the ability of these areas to repair these tissues and react to the extra forces. These areas (the ones that are the most vulnerable) will begin to break down. Now if our only goal is to replace one of the right sided joints with an artificial joint, that prosthesis may wear out a bit faster on that side, but this is likely not a big issue. However, if we want to preserve that right sided joint with ortho 2.0 type procedures, we had better figure out why that joint is getting so much more wear and tear and correct that problem.
**Lordosis in the spine—or It’s All About the Curves**

**Lordosis** is the medical term for a front-back spinal curve. A healthy body in normal standing strives to use as little energy as possible. The diagram to the left shows that the neck and low back curves are counterbalanced so that we can stand up straight with a minimal of effort.

Far too little attention has traditionally been placed on these curves, but the medical and surgical community is slowly changing their position on this important issue. Researchers have completed clinical trials showing that rehabilitation efforts to restore these curves do produce positive clinical results. Spine surgeons now routinely take extra time and energy to restore or preserve the normal or natural spinal curve when performing fusions and disc replacements.

Why are these so important? The vertebra is built and oriented in such a way as to equally distribute forces between the front of each vertebra (the disc) and the back of the vertebra (the facet joints). This only happens when the normal spinal curves (lordosis) are present. On the diagram to the left, note that the green arrows show force distributed between the front (disc) and back (facet joint).

When the curve is lost (straight spine), the forces get distributed more toward the disc, which can cause it to get overloaded. We often see this on MRI’s as swelling in the vertebra around the disc (which means the disc isn’t capable of handling all of these forces and they’re getting distributed to the surrounding bone). Note the diagram to the right, which shows a loss of curve and a move of forces (large red arrows) to the front (disc). This loss of spine curve is a problem when considering stem cell therapy or any other type of regenerative procedure on the disc, as while such a procedure might be able to help keep the...
disc from failing, the same forces that caused the disc to fail in the first place (loss of the spinal curve placing too much pressure on the disc), will persist. So we believe it’s better to have the patient undergo therapy to restore the normal curve either before or during regenerative disc therapy.

What happens if the curve is too much? In that case this is called a hyper-lordosis and the weight gets distributed too much to the facet joint. These little joints are about the size of your finger joints and live in pairs at the back of the spine. There are two at each spinal level, with one on the left and one on the right. They help to control motion, so that any specific vertebra can’t get too far out of line. When they get overloaded by a big spinal curve, they can get arthritis much more quickly and show signs of wear such as cysts and hypertrophy (the body will literally make them bigger to handle the extra weight). When these joints become bigger (hypertrophy) they can place pressure on the nearby exiting spinal nerves, and cause a new set of problems.

**Forward Head, Kyphosis, and Lower Neck Joints**

As discussed above, the spine is a delicately balanced machine where the neck and low back curves are counterbalanced by the thoracic curve. As we age, our head and shoulder tend to hunch forward, as shown to the left. As this happens, in order for us to see straight again, we crank our necks back further. Because of this extra backwards pressure on the neck, the facet joints in the bottom of the neck can be compressed, causing pain and more arthritis at those levels. While performing traditional facet injections into these aggravated joints can help, it does nothing to solve the excess loads on the area, so the pain just returns. This is a great example of how Ortho 2.0 differs from pain management. The pain management approach is to put steroid anti-inflammatories into the joint to reduce pain. The Ortho 2.0 approach is to treat the joint (more likely with either low dose anti-inflammatory or regenerative techniques), but also to work on getting weight off the joints. This would be curve restoration and certain types of postural or alignment based physical therapy.
Upper Cervical Input

The upper neck is a special area with respect to alignment and posture. We've all heard about the balance system that involves the inner ear. However, normal balance also involves body position and postural alignment. Our balance system is more complex. A bit like a N.A.S.A. space ships that has triple redundant systems so that if one fails to provide enough information about balance, another system will automatically take over. This makes sense, since the loss of balance perception is not compatible with leading an active life and fending for yourself. Our balance system therefore has three inputs (as shown on the left): the inner ear, the eyes, and the upper neck joints. For many years, the focus has only been on the inner ear, but these other two systems are equally important.

What part of the upper neck seems to be the most important for balance? A number of years ago a team of researchers started killing the little nerves that took information from the C2-C3 joints. The goal was to help the pain associated with pain from these joints. While the pain got better, these patients all became very dizzy. The researchers were confused. While they knew about the inner ear being involved in balance, they didn't know about the upper neck. This misconception still exists in medicine today, with the vast majority of doctors not knowing about “cervicogenic dizziness” or dizziness coming from the neck, despite published research on the phenomenon. Based on the research, the C2-C3 area and upper neck muscles, as well as the sternocleidomastoid muscles all seem to be implicated.

So what does this have to do with alignment? Recently, some Australian researchers have determined that patients with whiplash injuries have difficulty in determining which end is up. Literally, their heads don't have the same proprioceptive ability as normal
subjects. We believe that this is due to injury of the upper neck joints, which is common in this type of injury. We also see this type of injury in patients who have injured the upper neck in the past or for some reason have chronic overload forces at C2-C3 or the high upper neck. These patients often have a head tilt. When you correct the head tilt, they feel crooked. Why? One joint is giving bad information about normal posture. This causes the patient to tilt the head, which is often noticed by the doctor while the patient is lying face up. This tilted head feels normal. The “righting reflex” then kicks in to keep the eyes and head level while standing. This causes the patient to tilt the body to one side to compensate. This then causes all sorts of problems, with arm frequently getting numb (usually on the little finger side) from thoracic outlet syndrome (the nerves in the shoulder getting pinched). In addition, notice how this can impact problems down to pelvis and even how one side of the leg/foot complex strikes the ground. As a result, in looking at alignment, one must always consider how it interfaces with the neuromuscular system (in this case proprioception from the upper neck).

Can Alignment be Impacted when the Spinal Stability System goes Off-line?

You've already learned about the stability system in the low back (multifidus muscle). Is there something similar in the neck and can it impact alignment? We asked ourselves that question about 10 years ago. One of our physical therapists was tasked with seeing of the multifidus muscle in the neck showed signs of atrophy, like in the low back. He was able to
prove (as part of his PhD thesis) that this does occur. I believe that when these small segmental stabilizers go off line, something else has to kick in to hold us together.

In the neck and shoulder, the muscles that kick in are the trapezius, levator scapula, and scalenes (and sometimes jaw muscles). These muscles were never deigned to be stability muscles, so they quickly get overloaded. As they get tighter to stabilize the neck, they also usually bring one shoulder higher.

This then leads to the same types of problems seen with the upper neck problems discussed above-thoracic outlet syndrome. In addition, the attachments of the muscles get angry, as they were never designed to handle this type of excessive loading. This is called enthesopathy and is discussed elsewhere. In addition, the upper trapezius and scalenes both have nerves nearby or traveling through them, so when they get too chronically tight, these nerves get in on the act. For the upper trapezius, the occipital nerve can get irritated, causing headaches. For the scalenes, the brachial plexus can get pinched, again leading to thoracic outlet symptoms with numbness in the little (ulnar or lower trunk of the brachial plexus distribution).

When this stability problem happens in the low back, we tend to see the big muscles that attach the pelvis to the ribs substitute for the smaller muscles. This includes the quadratus lumborum and iliocostalis lumborum, both of which originate at a common attachment point in the low back (see figure to left). This is a similar situation to the lateral and medial epicondyles of the elbow, where the muscles of the forearm attach and can cause tennis elbow or golfers elbow. This common low back attachment site is the PSIS. We consider this the tennis elbow site of the low back. This spot is the dimple (also called “Dimples of Venus”) just on either side of the upper tailbone. This is again an enthesopathy, which is better explained in a later chapter.
Realignment Surgery?

When I was in residency, one of my most and least favorite rotations was through pediatric rehabilitation. It was one of my favorites because it was fun to be around the kids, and my least because these particular kids all had severe physical deformities. The surgeons on this rotation were great heroes, often allowing these kids to walk or function better by adding an inch here, taking away an inch there, or cutting this or that tendon. These kids were so severely disabled that it simply didn’t matter that the accuracy of the surgical healing could be off by a few millimeters either way.

Fast forward 20 years and I no longer see disabled kids for a living, but patients with chronic joint and spine pain. I have seen hundreds of patients through the years who have undergone the same type of realignment surgeries, although they didn’t do so well. What’s the difference? The normal musculoskeletal system is tuned to sub-millimeter to millimeter precision. Human accuracy and surgical healing can be off, a few millimeters either way. So while it’s possible that a surgically realigned tendon, muscle, ligament, or bone might be in the perfect anatomical position, it’s more likely that it will heal “a little off”. Also, many times these surgeries are for quick fix type goals and ignore the cause of the problem. Take the example of a knee lateral release. The concept is that the patella isn’t tracking properly and is being pulled too far to the outside of its groove (or doesn’t have enough pull toward the inside). Rather than asking what biomechanical forces have caused this to occur (issues in the hip, low back, etc…) we often try to take a quick fix approach by cutting some of the quadriceps attachment and fascia on the outside. Since the patella is aligned to sub-millimeter precision and the surgery can only have accurate healing to a few millimeters, I often see the patella in misalignment after the surgery. For example, if the lateral side scars and heals too tight, the patella will be too far lateral, or if too much of the lateral side is cut, too far
medial. Add in that the same forces that were pulling the patella too far laterally are likely still there (say too little hip external rotation) and the surgery hasn't solved the cause. Since these are permanent realignments of the musculoskeletal system, rather than a quick decision, I tell patients to think long and hard before getting a procedure that can't easily be undone.

**How do I know if I have an alignment problem?**

First, I've only scratched the surface here concerning common problems with alignment. The goal was to introduce the concept, not list all things that an experienced musculoskeletal expert would see in daily practice.

At its simplest, patients with alignment problems have one sided pain or arthritis in the absence of specific trauma. For example, while they may have both knees that hurt, the right hurts much worse than the left. In addition, an MRI or x-ray of both knees shows one has much more severe arthritis than the other. This makes no sense, as they never injured the either side.

Other examples of alignment issues can often be seen when looking in a mirror or asking your friends. You may notice that one shoulder is higher than the other, or the head is slightly tilted to the right or left, or that one hip is higher. Looking at wear patterns on clothing and shoes can give more clues. For example, does one shoe wear more than the other? Does one part of the sole of one shoe wear more than the other parts? Does one part of your pants wear out faster than another? Is it easier to hold a handbag or back pack on one shoulder or the other?

When you’re active, are you dramatically stronger on one side versus the other (more than you would expect related to just being right or left handed)?

Here are some tests of postural alignment, which you can do at home:

- **Excellent test and home therapy videos from the Egoscue method**
- **Basic standing posture and alignment**
- **Shoulder:** *Floor Angel Test*-tests for tightness in the front of the shoulder leading to a protracted shoulder (forward).
- **Hip:** *Modified Thomas Test*-tests for tightness in the psoas muscle.
If I have alignment problem, what can I do about it?

The good news is that there are many therapists and practitioners who specialize in alignment. These concepts really began shortly after the turn of the century, when traditional allopathic medicine was in its infancy and unable to address what seemed like obvious problems to non-physicians. The pioneers were Moshe Feldenkrais, Ida Rolf, and Matthias Alexander. I was introduced to these geniuses when I realized that by the early 1990’s (just out of residency) these issues were still not being addressed. The concepts I’ve discussed here were not part of my training in physical medicine and rehabilitation. To remedy this deficit, I took to reading the old works of these masters to try and learn what I have never been taught as a physician.

Newer systems such as Pilates, Muscle Activation Technique, Myofascial Release, and Egoscue have added to the diversity of treatment methodologies that address various aspects of posture and alignment. In addition, curve restoration has now become a scientifically vetted medical art.

A caution, while some physical therapists have spent years learning advanced biomechanics, they are few and far between. The standard course of physical therapy education contains very little about how to identify and address common alignment problems. This is despite one of the early geniuses of muscle function actually being a physical therapist (Florence Kendall). So if you’ve tried and failed physical therapy, it’s unlikely that you actually saw a physical therapist with proper training in the art of biomechanical and alignment analysis and treatment.

• **Rolfing:** Sounds a bit like the vernacular for vomiting, but is actually the last name of the founder. The focus is on very rigorous deep massage techniques to free up area of muscle and fascial tightness to restore normal posture and alignment. Generally 10 sessions.

• **Alexander:** A turn of the century orator in a time before electric amplification of voice.

• Alexander figured out that certain head and neck positions allowed the speaker to project his or her voice better in an auditorium. This was later applied to “sick” performers to perfect their performances. This is now a system of treatment focused on head and neck alignment popular with stage and theater performers.
• **Muscle Activation Technique**: Developed by an athletic trainer, Greg Roskoph, and based on the concept that certain muscles can become less active based on injury and certain patterns of movement. The focus is balancing the moving biomechanics of the body by “turning on” these inhibited muscles.

• **Myofascial Release**: Pioneered by John Barnes, a massage therapist in Arizona, the focus is on trigger point massage to release or free up tight muscles leading to poor body alignment. There is less focus on overall body posture than in Rolfing.

• **Egoscue**: Begun by Pete Egoscue, this system focuses on activating and strengthening specific muscles with specific exercises to restore to normal body alignment and posture. This system has become popular with physical therapists wanting to increase their knowledge about biomechanics.

• **Feldenkrais**: Developed by an Israeli physicist, the focus is on alignment in simple movements.

• **Curve Restoration**: The guru’s of this now scientifically vetted field are the Harrison’s, chiropractors who have been publishing their results in peer reviewed medical journals for years. They use very specifically designed forms of special traction to restore the normal curvature. They have also designed home units so that patients can try to deal with this problem in a do it yourself program.
My countrymen should have nerves of steel, muscles of iron, and minds like thunderbolt.”

Swami Vivekanda

**Neuromuscular**

To many of us, the term “Neuromuscular” is a new term. It means both nerves and muscles and is often used to refer to the connection between the two. While the nerves in various parts of the body tell many organs what to do, the organ they direct which has the most immediate and easy to observe response is muscle. Your nerve says jump, and your muscle says “how high?”

Think of the nerves as the wires that connect the main computer (the brain) with the muscles. You think of a movement and that nerve impulse drives muscles. Information also goes the other way, from the skin, muscles, joints, ligaments, and tendons up to the brain. This information is called “proprioceptive” and allows you to finely adjust
movements to what’s going on in your environment. If you step on something unstable, you might fall. That information is quickly relayed to the spinal cord where reflexes instantly adjust your stance.

It's easy to see how the nervous system plays a big role in the type of muscular spinal stability discussed in the first chapter. As discussed above, this type of stability during movement is made possible by proprioception, which is used to provide real time feedback so that a moving joint stays in its neutral zone. For example, if the joint experiences forces that might cause it to translate or shift too much, small joint sensors detect this motion and instantly tighten muscles to counteract that abnormal motion and keep the two joint surfaces aligned (keep the joint in the neutral zone). If this didn't happen thousands of time each day, the joint would wear out much more quickly.

As discussed above, when spinal nerve irritation or compression occurs, the muscular stability system for the spine goes off line, and the spine becomes unstable. I believe the same happens in peripheral joints like the knee. If spinal nerves are irritated in the back (again, you may not feel any back pain), the muscles that help stabilize the knee in movement can go off-line or have reduced efficiency and as a result, the knee joint becomes unstable. So now, when the knee experiences abnormal forces like a shift, the wiring loop through the spine between the joint sensors and the muscles that protect the spine is impacted, causing an ever so slight delay. This delay leads to a joint that gets out of alignment more easily during motion and as a result, a joint that is more likely to become arthritic. Since this concept of muscular activation delay has already been very well documented for spinal stability (here the delay causes the vertebrae to become unstable in movement), there is no reason to believe it only applies to the spine.

One of the problems we have had as a medical community is our main and widely available test for diagnosing nerve pathology (EMG-Electromyogram/NCS-Nerve Conduction Study) is very specific for certain types of nerve injuries (such as when a nerve is wholly or partially destroyed by trauma), but not very sensitive for other types of nerve problems. In particular, many significant problems with the nerves involve small fibers (small fiber neuropathy), whereas the EMG/NCS test can't detect this type of pathology. In addition, the test has very poor sensitivity in detecting nerve irritation. While other more sensitive nerve tests (in particular QST or Quantitative Somatosensory Tests) are commonly used in research, they are not yet widely used by physicians. So in a real way,
physicians are often “flying blind” from a diagnostic testing standpoint in figuring out when nerves have certain types of problems. In other words, based on the research, a negative EMG/NCS doesn’t rule out nerve trouble. It’s a test that’s highly specific, but with poor sensitivity. The definition of a good diagnostic test is one which is highly specific and highly sensitive (capable of detecting the disease 99% of the time when it’s present, and shows negative 99% of the time when the disease isn’t there).

So in summary, I believe that even small amounts of spinal nerve irritation may not cause any noticeable back or neck pain, but can wreck havoc with the muscular stability system either in the spine itself or in the peripheral joints. Since this system protects your joints during activity, when this type of nerve problem takes muscles off line or reduces their efficiency, this will eventually lead to less protection for the joints and an earlier onset of arthritis. In addition, the diagnostic test toolbox we have available to us today doesn't include tests that are capable of detecting this type of nerve problem, hence the reason this problem often goes undiagnosed.

In addition, I believe that treating this problem is a key component of long-term joint preservation.

**Arthritis doesn’t Cause Pain, Pain Causes Arthritis**

I saw this title come across a science news feed last year. I hit me like a welcome pie in the face, as I had often suspected that something like this had been happening in my patients. The concept is simple, yet if confirmed in humans (this was a well done animal study) will change the face of orthopedics and rheumatology forever. It’s equivalent to when we doctors learned that stomach ulcers were caused by bacteria and not stress (I was taught in medical school they were due to stress).

The title is self-explanatory. The authors created an elegant animal model that showed that nerve activation in a joint leads to bad chemicals being dumped into the joint, which leads to pain and faster onset of joint arthritis. This is a reverse of what has traditionally been considered, i.e. that a joint is injured and begins to degrade and then causes pain. It’s important to stop for a moment to consider how these scientists have turned orthopedics on its head. Again, our entire orthopedic care model is based
on the concept that injury in a joint (or accumulated injuries over a long period of time) leads to arthritis in the joint, which leads to more joint breakdown and pain. This new model reverses the old paradigm so that now it’s aggravated nerves that lead to arthritis. Sound familiar? I believe this is just an extension of what we’ve been discussing here, problems with spinal nerve irritation lead to bad chemicals being dumped into a joint and a “sloppy” joint with poor stability which ultimately leads to arthritis.

I have had my own low back caused knee problems. Using this new model, my knee problems were caused by spinal nerve irritation (which I never perceived as low back pain) causing not only a sloppy knee joint (due to parts of the big stabilizer muscles being shut down by trigger points), but also bad catabolic (break down) chemicals dumping into the joint. This issue was quickly fixed not by operating on my knee or even injecting magic stem cells into the knee, but by bringing the spinal and joint stability systems back on-line by using IMS to get rid of the trigger points.

Low level Arthritis Pain versus Nerve Pain

Based upon my clinical experience and this new model of nerve related joint pain and arthritis, I would place patients into two distinct categories, what I’ll call “Neuropathic Arthritis” vs. “Classic Arthritis”. Early on in the degenerative process and for some patients who have more of a spinal component to their joint pain, patients are firmly in the “Neuropathic Arthritis” (NA) camp. These patients have severe joint pain which is often disabling or can become disabling with certain types of activity. I see these patients in the clinic, often very desperate because their joint pain is very intrusive. They are either completely disabled by their pain or they are unable to exercise at high levels. In this new model of joint pain, these patients have an active spinal nerve problem manifesting as joint pain. They are often unaware that this joint pain is linked to their spine, but if you dig enough, they will usually admit to a history of spinal problems that has either
(in their mind) been successfully treated (perhaps with a surgery many years in the past) or spinal pain that is ongoing and low level and in their mind under good control. They have usually had several unsuccessful joint surgeries, which didn’t work because while they have issues in the joint, they also have active issues in the spine which were unaddressed by their joint surgeries. Treating the spine in these patients can often make a huge difference.

The second camp is the traditional “Classic Arthritis” (CA). The CA group no longer has an active spinal component, or if they do, their joint has long since degenerated. Their pain pattern is different and matches what we know of arthritis pain. You may remember your grandparents being stiff in the morning with low level pain that becomes better with activity as the joint “warms up”. Just like gramps and granny, once these patients start moving, they generally feel better. Treating the spine in this group is often too little too late, as the joint damage is done.

It’s important to note that there are other factors at play in many of these patients, so this is a simplified discussion. For example, patients with joints that are unstable from a ligament standpoint may also have more pain when they are active and patients with bad joints due to severe trauma may have less pain as their joint warms up. Like anything in medicine, the body is a very complex machine, hence the SANA approach, which looks at all components of the musculoskeletal system.

**So what can be Done to Fix the Spinal Nerve-Joint Connection?**

Despite their being data that shows that irritated spinal nerves can cause may be associated with joint problems, most physicians have a hard time associating joint pain with a low back nerve problem. The first step in identifying a spinal nerve component as a cause of joint pain is simply a thorough neurologic exam. When I say a complete exam, I don’t mean the “can you fell this?” type neurologic exam. This careful exam is focused on comparative sensation from side to side and on the same side of multiple different types of sensation. This includes not only light touch (the can you feel this exam), but also pain sensation (pinprick), hot/cold sensation, etc... The exam also recognizes that there are multiple types of pain and nerve referral patterns, including those from spinal joints, from nerve trauma, and muscle trigger points.
If the exam shows that spinal nerve irritation may be occurring, the next step is a spine MRI. Correlations between the exam findings and the MRI are important. In addition, this correlation acknowledges that while spinal nerves can be compressed by bone spurs and herniated/bulged spinal discs, they can also be irritated by sloppy stability in the spine (see chapter 1). An MRI marker of this type of sloppy stability can be seen on MRI as multifidus muscle atrophy (see chapter 1). So even though there may be no bulging disc on the right at L5-S1 that pressing on the descending S1 spinal nerve, significant atrophy of the deep stabilizers at this level (multifidus) combined with sensation problems at the right S1 territory in the leg means that the L5-S1 segment is likely sloppy from a muscular stability standpoint.

**Treatment for Irritated Spinal Nerves**

The diagram to the left shows that the spinal discs can herniate their inner contents (nucleus pulposis), which can place pressure on spinal nerves. This is called radiculopathy (if more severe) or radiculitis (if less severe). This has also been called “sciatica”, although this is not an accurate term. Their solution was to surgically remove the herniated portion of the disc sitting on the spinal nerve. On the one hand, this was a great advance. Patients with numb and weak legs due to a bad back now had a treatment. On the other hand, it began our current move towards invasive spine treatment, a path that many have criticized.

Treatment for herniated discs remained largely surgical until studies in the 1980’s showed that a few years after the disc herniation, results for patients treated with surgery and those not treated weren’t all that different. What emerged from this research was the idea that perhaps herniated disc patients could be treated without surgery. As a result, the focus began to shift towards conservative management, which by the late 1990’s included epidural injections. This meant placing strong anti-inflammatory medications around the painful and swollen spinal nerves. At fist these injections were just adapted from pregnancy epidurals given to control the pain of labor, but later these evolved to more specific placement of medication between the disc and swollen nerve. These newer injections were called transforaminal injections (literally meaning through
the foramen-or into the hole in the spine where the spine nerve exits). Transforaminal epidural injections of anti-inflammatory seem to work better than the older injection types (interlaminar and caudal).

The conventional wisdom regarding epidural injections is that we wait until the patient has failed a significant course of medications, physical therapy, and time. While this makes some sense from a cost savings standpoint, our newer understanding that irritated spinal nerves can lead to shrunken spinal stabilizing muscles and instability, means that earlier intervention is likely warranted. I believe that preservation of these spinal stabilizers is very important and that the goal of treatment is to bring them back on-line early by calming down a swollen and irritated spinal nerves. My own example is illustrative. Several years ago I was performing heavy dead lifts from the floor, early in the morning (generally a bad idea) and felt a pop and went down on the ground. My pain was so severe that I couldn't walk easily, stand-up, move, etc... Had I stayed like this for any length of time, my spinal stabilizers would have quickly atrophied leading to months of rehab to get back into shape. Instead, I had my partner perform an immediate epidural to calm these swollen nerves and was back to weight lifting 3 days later.

What are the generation of treatments for irritated spinal nerves? Rather than big dose anti-inflammatories, we believe that the next step will be using much lower dose medications and combining these with regenerative medicine. So rather than injecting 80 mg of corticosteroid (the height of the Empire state building), injecting smaller physiologic doses of steroids and adding in level II (or even level III) regenerative medicine solutions.

**The Great Adaption Machine**

Our bodies were designed to keep moving at all costs. In a pre-industrial society, the amount to physical prowess it takes to collect, hunt, process, and consume food is great. However, in such a society, the potential for injury from a runaway animal or even a rocksless is great. The only way for us to be able to get injured and keep going was to design the musculoskeletal system to be the great adaption machine. What does this mean? At its simplest, let’s take a left foot injury. With an injured left foot, you instinctively limp on the left and transfer more weight to the right. This takes weight off the left so it can heal. This strategy works, because we were meant to heal on the run. Studies where patients are asked to bear more weight on an injured or operated area
(different from the current orthopedic healing paradigm) show that healing with weight bearing is better than extended periods without weight on the joint.

The great adaption machine also gets a good deal more complex. I have noticed that in chronically injured patients, the system is constantly rearranging forces to be able to offload certain areas. As an example is my own minor chronic neck, upper back, low back, leg problems. At times my left scapula will hurt, at other times my biceps tendon, and at others, my low back. I can feel my body rerouting forces through adaption, from one site to the next. When the neck stabilizers go off-line or when they are too taxed from my heavy weight lifting routine, the big neck muscles take over and the upper trapezius, levator scapula, scalenes, and SCM fire up. The left scapula and scalenes too overloaded? My body reroutes the forces to the front of shoulder by moving the scapula forward. If this causes the biceps too much pain, my body reroutes those forces by turning the rib cage, which causes the low back to get torqued, and so on. This complex neuromuscular response has allowed us for millennia to continue to function with injury.

For patients and medical practitioners, this adaption process can often be like peeling back layers of an onion. Again, at it’s simplest (the injured left foot analogy), since the left foot is hurting, it may come as a surprise to the patient that the right foot begins to hurt as a result of excessive use. Even more cryptic are situations where 3 or 4 steps occur in the adaption process (like my neck, shoulder blade, front of shoulder, ribs). In those situations, the patient may be completely unaware that the problems are related. In addition, physicians will often only go for the “low hanging fruit” of where it hurts today. This approach again avoids the salient question, how did all of this get this way? In addition, just treating the part that hurts will only be a temporary fix, as this part will soon be overloaded again!

**Managing Trigger Points-A Forgotten Art**

Trigger point injections (TPI’s) were first popularized by Janet Travell, M.D., one of JFK’s physicians. Janet made it into popular medical culture because JFK had a bad back that often responded well to her trigger point injections. Sometime later, a Canadian Neurologist, Chan Gunn, M.D., added a significant piece to the trigger point puzzle. Travell had noted that just using a needle
without injecting anesthetic (dry needling) seemed to work just as well. To the medical establishment of the day, this seemed like Voodoo. At the time Chinese acupuncture was unknown, so Travell largely placed her emphasis on injecting anesthetic and anti-inflammatory medications. Gunn grew up in Korea, where a more aggressive form of Korean muscle acupuncture was common, so he moved forward with Travell’s dry needling technique, substituting the much finer and less traumatic acupuncture needles for the more traumatic cutting edge injection needles used by Travel. Gunn also theorized that the muscle trigger points that Travell thought were due to overuse were more likely caused by nerve irritation.

The science of the last 20 years supports Gunn’s theory that nerve problems (autonomic and likely spinal nerve) and trigger points are closely related. By the early 1990’s, while physicians who were expert at trigger point injections were few and far between, TPI’s were used by a plurality of doctors treating musculoskeletal pain. Then something happened that often drives the course of medical care much more than efficacy or science, the reimbursement changed. Prior to the mid-1990’s, a physician could receive adequate compensation per site injected, then after the mid 1990’s the average compensation for this procedure was reduced by about 50-75%. In addition, getting compensated by insurers became more difficult. This was all it took to relegate the art of trigger point injections to the history books. Regrettably, the stock of physicians performing the procedure was wiped out. Today, because of this reimbursement collapse, finding a physician experienced in managing this type of muscle pain is like finding a needle in a haystack.

Why has the reduction in the number of physicians knowledgeable in trigger point therapy negatively impacted the quality of musculoskeletal care? I think my own personal story is important. In the late 1990’s I attended a medical conference that involved days of sitting. For an unknown reason, my left knee began to ache and swell. There was no trauma to the knee. I was literally hobbling around the office and all of my aerobic exercise came to a screeching halt. I underwent an MRI, convinced that I had somehow torn a meniscus or some cartilage. While the MRI showed the swelling and perhaps some small tears in the meniscus, it didn’t show a “smoking gun” cause for my severe pain. I went to see an orthopedic surgeon who wanted to perform a diagnostic arthroscopy, likely chop out
some meniscus, and remove a “plica”. I was desperate and convinced the MRI was missing the true cause, so I reluctantly signed on for surgery. A few days before the planned surgery, a visiting doctor from Canada was in our clinic and asked if I had tried trigger point therapy in my quadriceps muscle and low back? I said no, looking at him like he was some alien speaking in tongues. At this point I had seen the best physical therapists in town and failed all of their exercises, so I was desperate. I told my Canadian colleague that I would try anything. Turns out, this visiting physician was one of those “needles in a haystack” as he was experienced in the Gunn trigger point technique (called IMS, short for Intramuscular Stimulation). He examined my thigh muscle (quadriceps) and my low back, pulled out an acupuncture needle, and proceeded to stick this in my low back and thigh muscles. The muscles cramped suddenly as the muscles hit the trigger points (more strange than painful). After a two minute treatment, I got off the table, and walked normally for the first time in months. That night I went running for the first time in months, without a twinge. I canceled my surgery and have never looked back. I was so impressed; I learned the technique and began using it in patients.

IMS has revolutionized our practice, providing relief to patients who would only otherwise be treated by much more invasive treatments. Because of reimbursement issues (insurers don't generally cover IMS and the other form of trigger point therapy (TPI) is poorly reimbursed), the technique has remained obscure. There may be other reasons the technique has never moved to a wider physician audience, as it takes significant effort and dedication to learn how best to apply the procedure to get consistent results. At a medical conference where both traditional Chinese acupuncture and IMS were being taught, I had insight into how my medical colleagues view this complexity. After Dr. Gunn lectured about IMS, I turned to the physician sitting to the left of me and asked, “Wow, isn’t this IMS stuff great?” Her response was, “It’s too complex, you have to learn where all the muscles are, what they do, where to put the needle for each one, what to avoid...with traditional Chinese acupuncture I just look at a chart on the wall and put the needle at X marks the spot.” So while traditional Chinese acupuncture (placing a needle into the skin at specific Chinese chi points) has become popular, IMS has remained in obscurity. These past few years, IMS has finally taken a leap forward by being adopted
by various Colorado physical therapists (PT). One of our PT’s who we had trained in IMS went through the red tape to allow physical therapists to widely practice the technique after very intense coursework. As a result, IMS is now gaining more acceptance and more patients are getting access to the technique.

How did muscle trigger points in my spine and thigh cause severe knee pain and swelling? Turns out I had fractured a few little bones in the back about 10 years before the day I had my knee pain. Other than a few bouts of mild stiffness, I had never had any ongoing back pain after the fractures, just a sudden and unexplained onset of knee pain. So what’s the connection? The upper low back spinal nerves were irritated, which caused big trigger points to develop in my quadriceps thigh muscle. As this happened, large sections of that big muscle began to shut down, turning off the major stability system of the knee, which began to swell because of the extra wear and tear movements. Why didn’t my back hurt? Believe it or not, pressing on spinal nerves generally doesn’t give you back pain, it causes symptoms where the nerve innervates (the area the nerve supplies). So if I took magic fingers and pressed on the right L5 spinal nerve in your back, you would feel it in your right leg and big toe, not your back.

**Enthesopathy**

We take for granted that our muscles not only contract, but also have a function as shock absorbers, letting go in a controlled fashion. As an example, when you jump from a fence at a height of just 4 feet, your femur bone should break. Why doesn’t it? The big quadriceps muscle absorbs the shock by acting an **eccentric contraction** (controlled release). When a muscle has trigger points, the biomechanical properties change. Large sections of the muscle can lose their ability to act as active shock absorbers. We believe this leads to extra pull on the areas where the muscles attach to the bone. This causes swelling and breakdown of these areas known as **enthesopathy**.
While many physicians will recognize problems in joints, and fewer will recognize trigger points in muscles, in our experience, even fewer will recognize enthesopathy. This is a problem, as in our experience, many patients suffer from this problem. The good news is a new generation of physicians armed with PRP to treat tendons and ultrasound imaging to detect problems in those tendons, is finally beginning to address this important problem:

**Common areas of enthesopathy:**

**Head:** The back of the head where the trapezius, sternocleidomastoid, and sub-occipital muscles insert is frequently an area of muscle attachment overload. These muscles can irritate the greater and lesser occipital nerves, leading to headache.

**Upper back:** The back of the ribs where the iliocostalis and quadratus lumborum muscles insert can become inflamed and lead to back pain. We see this commonly when the multifidus stabilizers are off line.

**Lower back and pelvis:** The PSIS area is where many large low back muscles take their anchor. As already described, we consider this the tennis elbow area of the low back. Patient’s can often point to a single spot just to the side of the upper tailbone (dimples of venus).

**Knee:** The pes anserine area is a common insertion for many muscles that travel in the front of the thigh including the sartorius, gracilis, and semitendinosus. These patients have pain on the inside of the knee that is often confused with meniscus tears. We see this problem in patients who have low back issues leading to knee pain.
**Elbow:** The classic medial and lateral epicondylitis (golfer’s and tennis elbow) are attachment sites for muscles that get overloaded. We see this commonly in patients who have nerve irritation in the neck or shoulder (cervical radiculopathy or radiculitis and thoracic outlet syndrome).

### How Instability and Enthesopathy are Often Linked

Have you ever tried to stand-up in a row boat? The “ground” you’re standing on is unstable, as it seems to randomly move in unexpected ways. The amount of energy it takes to do anything, even reaching for an oar, is tremendous. The same happens when a joint is unstable.

In the case of your body, the muscles automatically kick into overdrive to try and compensate and stabilize the joint. Think about how stiff and tight your body would be standing in the row boat. When your muscles do this for long periods of time, they pull too much on their attachments and can cause enthesopathy. For example, this commonly occurs when the spinal stability system goes off line. Look at the picture to the left of just some of the spine muscles that act between segments (from one vertebra to the next). Imagine if all of these were working overtime. Where they attach would be on fire, as these attachments would be overloaded (entheopathy). This would hurt and cause local pain. This is why may spine injection based approaches often fail. We can inject medications into a specific structure like the fact joint or around the nerves, but if these tendons attached to these small segmental muscles have been damaged due to years of overuse, then nothing about placing medication in a joint or around a nerve (which are different locations than these muscle attachments) will help the pain. We also frequently see this around the SI joint and hip girdle (see Case 1: SI Joint Shirley in Chapter 5).
Multifidus Atrophy

If you have numbness, tingling, or weakness in an arm or a leg, you meet the classical definition of spinal nerve root compression called radiculopathy or radiculitis (literally in Latin, root-disease and root-swelling). Getting to a diagnosis in this instance is usually routine, as long as there is something on your exam that correlates with a mechanical compression on your MRI (bone spurs or disc pressing on a spinal nerve). However, if you have more subtle signs of nerve root irritation or nothing structural on your MRI, you’re less likely to get a diagnosis.

While you may have been told there’s nothing on your MRI of importance, the research in this area may argue otherwise. An important finding may be multifidus atrophy. If you look at your MRI, you may be able to see this problem yourself. This finding is easy to see if you think of the spinal muscles as a steak. A steak in the grocery store is a cross section of the muscles, just like the axial view of an MRI. If you find the axial view of the spine and open those images, in the low back (where this finding is commonly seen), it will look like the image to the left. The multifidus muscles will be in the back of the spine (to the bottom of an MRI image).

This area looks like a steak.

Now let’s look at the quality of that steak. A good steak has much more muscle and less fat. A bad steak has more fat than muscle. The same applies to multifidus muscle atrophy. The MRI picture to the left has both a high quality steak (less fat) and a low quality steak (more fat). The top image has very little multifidus atrophy. The muscle (or steak) in the dashed white circle has very little fat. This means the
muscle is intact and has not atrophied (become smaller). On the bottom, is the bad steak. In the dashed white circle, there is as much fat as muscle, so this multifidus is atrophied. Why is this important? Multifidus atrophy has been associated with both chronic low back pain and leg pain. Why? The muscle acts as a stabilizer of the vertebra (as discussed in the first chapter). If it gets weaker and smaller, it can’t stabilize as well and this can lead to nerves getting irritated.

Problems at the Neuromuscular Junction: Painful Dystonia

The neuromuscular junction is where the nerve talks to the muscle. As you recall, the nerve talks to the muscle to tell it what to do. The nerve says jump, and the muscle says “how high?”. Think of the neuromuscular junction as a room where there are two people talking, one the nerve and the other the muscle. If the room is quiet, the instructions barked by the nerve to the muscle can be clearly understood. If the room is very noisy or the nerve isn’t speaking clearly, the instructions may be misheard or misinterpreted by the muscle. The latter case is what happens in cervical dystonia. This is a disease at its most severe that results in a patient with spasmodic torticollis, or where a patient has his or her head turned to one side on a permanent basis. However, like all diseases, this problem comes in less severe forms, where the patient just has constantly tight muscles that won’t let go. This chronic tightness (called dystonia) naturally changes the alignment, which can lead to pain and other problems. I call this less severe form, painful dystonia. We see this in patients who have had traumatic nerve injuries (often in car crash trauma). In this case, the noise in the neuromuscular junction room is turned up, so that the nerve instructions to the muscle sound like “stay tight all the time”, even when that signal makes no sense for the conditions. This can be treated with medications like Myobloc, which work to turn down the noise in the room, so that the muscle can hear the nerve instructions. Injected in enough quantity, this medication could turn off all signals from the nerve to the muscle, but injected in smaller quantities, it only reduces the noise in the room.
Central Sensitization

Let’s say you’re in your car and all of a sudden all of the warning lights start to go off. You bring the car to the mechanic and he or she says that there may be a few things wrong here and there with the car, but the real problem is that the wiring is bad. This is central sensitization (CS) also known as Complex Regional Pain Syndrome type II, Fibromyalgia, neural sensitization, etc... In all of these conditions, it’s an injury to the pain reporting wiring of the body (the nerves and microprocessors that control them) that cause the problem. The nerves become hypersensitive to pain. This phenomenon has been extensively published, most references are for whiplash or fibromyalgia. This problem is also now being discussed as related to joint pain (as discussed earlier).

Patients with CS simply have a nervous system that’s on fire. At its early stages it may cause arthritis (see above), but as this gets worse, large areas of the body can be impacted. In addition, these areas don’t follow normal nerve pathways like dermatomes (skin areas associated with certain spinal nerves), so oftentimes many physicians without training in this area label these patients as having “non-anatomic” sensation problems. These patients as they progress, can’t tolerate physical therapy, massage, injections, acupuncture, IMS, etc... Our research group demonstrated that at an early stage, trigger points may make the sensitization problem worse. Ay later stages or when more severe nerve injury has occurred, cold sensitivity is common. For patients with traumatic CS, a cold summer’s night (about 60 degrees F) is actually painful, as that’s all it takes to active pain nerves. Think about this for a second. How cold would it have to be for a normal person you to perceive cold as pain? Below freezing? 20 below? These patients feel this at less than 60 degrees.

CS patients are generally the most difficult patients to treat. First, the pain sensitivity levels have to be brought down to a more normal level. One way to do this is medication. We have seen many medications for this type of nerve related pain come and go, Neurontin, Tegretol, Elavil (Amitriptyline), Doxepin, just to name a new. They all had the problem in that they didn’t work for most patients. However, newer nerve pain drugs are
just coming to market, with many new ones in the pipeline. The most effective drug we have seen is the newer drug Lyrica. This works well in about 6 in 10 of these patients to reduce nerve pain and “put some water on the fire”. Once this is accomplished, the next step is usually to identify the problems that caused the fire. In many patients, there were specific musculoskeletal problems that lead to other which ultimately lead to the fire getting out of control. Finding these specific problems and treating them can then start to provide relief. As an example, a patient labeled with “Fibromyalgia” may note that his right neck and shoulder began hurting first, then his right low back, then his arm and leg. Tracing the issues back to the neck would be the way to approach this patient.

**Neuromuscular Resources**

Calming down nerves through injection often requires an expert trained in x-ray guided procedures. Here are some resources:

- **ISIS (International Spinal Injection Society)**
- **ASIPP (American Society for Interventional Pain Practitioners)**

The most effective way we've seen to address chronic trigger points is either through IMS or trigger point injections. Here are some lists of where to find these “needle in the haystack” doctors and physical therapists:

- A list of Gunn IMS practitioners
- Trigger point educational group
- Physical therapists trained in IMS

Trigger points in muscles can be difficult to treat on your own, but we've seen some success with these approaches:

- Electro Therapeutic Point Stimulation (ETPS)
- TheraCane

Enthesopathy: See level 1 prolotherapy and level 2 PRP resources in chapter 2
Putting it All Together

My goal with the last chapter is to present several patient examples, from less complex to more complex, so that the reader can see how the puzzle pieces fit together and interact.

Case 1: SI Joint Shirley

Shirley is a 54 year old woman who fell and had chronic pain in the back of her hip. She had seen multiple physical therapists and chiropractors, obtaining only temporary relief. Patients like Shirley who complain of hip pain are often told they pain coming from the hip joint. An x-ray is usually taken showing some arthritis, which would be common for her age. Based on little else than the report of hip pain and the x-ray, the
patient is frequently scheduled for a hip replacement without ever confirming that it’s her hip joint that causing the pain. Isn’t hip pain always from the hip joint?

To investigate the possible cause of hip pain, let’s look at the pelvis. The picture to the right shows that the hip joint is connected to the pelvis and that the next joint up the chain is called the SI Joint (short for sacroiliac joint). You can see that these joints are close together. Pain from either joint can cause patients to complain of “hip pain”. The hip joint proper tends to cause more groin pain and patients with SI joint problems tend to have more pain in the back of the hip near the PSIS area (see diagram to the left which shows a common location of SI joint pain). Truth is, either the hip or the SI joint can cause pain in the back or front of the hip, so how do we tell which is causing the complaint of hip pain? To determine the source of the pain, we performed diagnostic numbing injections. Under x-ray guidance (fluoroscopy), we injected numbing medicine into the SI joint and hip. Injecting the hip only minimally helped her pain, while injecting her SI joint took away 70% of her pain. We had our man!

Since we had injected ultra low dose anti-inflammatories in her joint, this gave her some relief for a few weeks, but the pain returned. Now the question was, what caused her to have chronic SI joint pain? Because of her fall on the SI joint area, we suspected instability, which was confirmed on exam (see SANA pyramid to the right). What other clues lead us to believe that she might be unstable in the SI joint? The patient had tenderness throughout the muscle attachments associated with the SI joint. The attachments of the gluteal muscles and piriformis showed signs of enthesopathy. At the back of the hip (greater trochanter), she was also tender where these muscles attached. If she had just injured the joint, this didn't make sense.
However, if the joint was unstable, these muscles would be working overtime to try and help stabilize the area, thus causing the enthesopathy. So in addition to treating the SI joint, these other areas would need to be treated as well. Why? While they might go away if we fixed the stability issue in the SI joint, based on clinical experience it was more likely that they would remain as the damage at these muscle tendon attachments had been done.

So what options are there to fix an unstable SI joint? Fusing the joint surgically with screws has been used, but fusion usually just transfers forces to the joints above and below, so we wouldn’t consider this option. Most pain management physicians would either consider repeatedly injecting high dose steroids into the joint or an SI joint radiofrequency procedure. As discussed in the chapter on articulation, high dose steroids can damage the joint. Radiofrequency is where we special needles or catheters are inserted to ablate the nerves that take pain from the joint. This is covered by many insurers, but these small nerves also provide proprioceptive input to the muscles that stabilize the joint, so nuking them could mean less active muscular stability. In addition, this would only address the joint pain and not the pain she was having from the enthesopathy muscle areas. In our experience, the only treatment that would be regenerative rather than ablative (build up rather than destroy), tighten the ligaments to help the passive instability, and address the areas of muscle/tendon enthesopathy, is prolotherapy. After two prolo treatments pain was down by 75%. The next step was to address the likely muscular stability issues. The muscular stability system for the SI joint is the transversus abdominis. This is a tough muscle to teach patients to contract, as it’s the deepest stomach muscle. However, we can easily see the muscle on ultrasound imaging, so the we used this advanced imaging to help the patient have a sense of when she was contracting the muscle.

In summary, Shirley is a good case to understand how all the treatment choices and pieces fit together. Starting with identifying a pain generator, then asking why she still hurts (instability), then looking at different joint treatment options, and finally ending with a very specific rehabilitation component to address the muscular instability and weakness.
Case 2: Ankle Alice

This is a 55 yo woman who was seen for an ankle problem that began after a climbing fall last year. She had multiple ankle ligament sprains as well as bone chips in the joint from trauma. After two surgical debridements, she wasn’t much better. I have included MRI’s above that show (on the far left) a normal ankle alignment between the tibia-talus-calcaneus bones (normal ankle MRI from someone else-not the patient). With this type of normal alignment, when the patient steps down to walk, forces are generated that start at the bottom of the foot and move up through the ankle bones. I have drawn these forces here as the yellow dotted lines. Notice that these force lines are relatively straight in the normal patient MRI on the next page. The forces move from the calcaneus (heel bone) to the talus (ankle bone) to the tibia (lower leg bone). In this patient’s MRI’s in the middle and on the right (same coronal MRI slice-but different types of MRI sequences), you can see how the force line is shifted to the left. The calcaneus (heel bone) is rotated laterally (in these images-to the left) and as a result there is overload of the bones and talo-calcaneal joint (the yellow dashed line on the far right picture showing light color in the otherwise dark bone). So because the heel bone is now bent to the lateral side (left in the above pictures), the ground forces don’t hit the talus bone evenly. Instead, the lateral talus and heel bone (calcaneus) are overloaded on this MRI and the bone is so beat up, it’s actually starting to die off (the dark color in the bone on the middle picture and the light color in the bone on the right picture). So how did the ankle get this way? Regrettably, nobody ever asked this question before. Her exam revealed a loose deltoid ligament, which I’ve drawn in above in the middle picture as a red dashed line ("stretched deltoid ligament") above. Think of this as living duct tape that normally doesn’t allow the heel bone to move to the left (lateral). When this got stretched in her climbing fall, it all of a sudden allowed her heel bone to move
laterally and redistributed the forces to the lateral side of that joint. This caused her cartilage on that side to wear out more quickly and the bone to be beat up.

So in order to fix this (with whatever—we use injected mesenchymal stem cells, others might use different tools), you have to shore up the lateral sub-talar joint (for us injecting her own stem cells into that joint) as well as tighten the deltoid ligament to try and prevent the overload from happening. This example illustrates how orthopedics 2.0 is about more than quick surgical fixes, it’s about figuring out how the joint got to its current condition and then designing strategies using advanced tools (such as stem cells) to try and restore normal joint function. When we look at this versus the SANA pyramid, we see that the stability impacted both the joint (Articulation) and the Alignment.

**Unstable Mabel**

Mable is a 45 year old white female who was in a rear end car crash a few months prior to our evaluation. Immediately after the crash she noted severe and sharp right sided upper neck and head pain. When she is seen in the clinic, she’s tender over the right C1-C3 facet joints and her right upper trapezius, levator scapula, sternocleidomastoids, and scalenes are tight. She has headaches with pressure over the upper neck. What happened to Mable and why does she still hurt after several months?

The upper neck facet joints are commonly injured in rear end car crashes. In addition, the upper neck ligaments can be injured as well. Mable gets good relief once we inject low dose anti-inflammatories into the right C1-C3 facet joints, but that’s only temporary. Her 3.0 ultra high field MRI shows that there is evidence of likely ligament stretching of the ligaments that hold the head on (alar and transverse ligaments). I believe what happened to Mable is that one these structures were injured, the upper neck stability muscles went off line. There is good evidence in whiplash injured patients that these muscles atrophy. What happens next? The big neck muscles take over. These are the upper trapezius, levator scapula, sternocleidomastoids (SCM’s), and the scalenes. However, they weren’t designed to be neck stabilizers, so their attachments get overloaded, leading to enthesopthy. This is what causes pain at the back of Mabel’s head and irritates the
occipital nerves that exit near the attachment of the upper trapezius and SCM’s. This leads to chronic headache (as well as the referred pain from the injured C1-C3 joints, which are known causes of headache as well). In addition, Mabel was unable to bear weight on these injured joints, so her neck curve was lost as her body figured out how to off-load the joints.

We injected the upper cervical facet joints and a high field MRI of her upper neck showed problems in the ligaments that hold the head in place. The entheseopathy was treated by prolotherapy injections where the muscles attached to the back of the head. Once the facet joints and upper neck muscles were calmed down, Mabel was able to strengthen her neck. She also underwent curve restoration to get back the normal lordosis. Once the joint pain, entheseopathy, muscular stability, and alignment were addressed, Mabel dramatically improved.

Sensitized Sally

Sally is a 40 year old woman in a rear end car crash who developed neck and back pain and within weeks of the injury. At that point she developed severe pain and numbness in her hands and feet as well as headaches. She was diagnosed with Fibromyalgia and given pain medications. When she was first seen, she had severe tenderness everywhere. She discussed that attempts at physical therapy, massage, and trigger point injections had all caused days of severe pain. The patient was given a Quantitative Somatosensory Test which demonstrated significant central sensitization. She was placed on Lyrica to help reduce her nerve sensitization. Once that began to reduce the severe pain, it became clear that multiple joints and muscles were being impacted by the nerve issues, her posture had eroded, and the muscular stability in the low back was compromised. Once the Lyrica began to help, we addressed the neuromuscular trigger points with IMS, the postural issues with Egoscue, and injected multiple joints with low dose anti-inflammatories to reduce pain. The patient also had an epidural in the low back to reduce nerve pain and rehabilitation to get the low back
stabilizers back on line. The patient’s pain then began to reduce. However, due to the nature of the nerve injury, she will require long-term pain management.

**Catawampus Wayne**

Are you ready for a more complex biomechanical analysis? This one demonstrates how a little injury can eventually lead to bigger problems. This patient had a serious fall from a bike about three years ago. He injured his shoulder, kidney, and hip. When he was first evaluated for stem cell treatment of his hip, I was concerned about his low back. While stem cells in the hip helped the hip pain (he now walk faster through an airport), over the ensuing year he continued to develop problems in his low back and leg. He was finally was diagnosed with a cyst on his right L4-L5 facet joint, which was pressing on a spinal nerve and giving him pain down the leg. The facet joints are small joints in the back, and sometimes arthritis of the joints can result in a cyst (just like a swollen knee joint can develop a Baker’s cyst). These cysts can press on spinal nerves, so they can be a double whammy for the patient. The patient had the facet cyst treated with a steroid injection to pop the cyst. This helped some of the leg symptoms and severe nerve pain, but by the time I re-examined him, his back was pretty bad (unable to stand straight). This was impacting his work as a physician. While knowing he has a facet cyst is a good start in helping him, asking the question of how he got that way is important if this is going to be successfully treated without surgical fusion of this level. His case is a good example of the ortho 2.0 concept. Consider the ortho 2.0 pyramid to the left, in which I’ve filled in various portions.

To better explain, more discussion and pictures are needed. On his flexion-extension views, it was noted that he had the L4 vertebra slipping forward on the L5 vertebra. This forward slippage was at the same level as his facet cyst. Coincidence? Likely not. The way to understand this problem involves some ligaments in the back of the spine that act as the major duct tape that help keep the spine aligned. These ligaments are the
supraspinous and interspinous ligaments. The image to the left shows the ligaments (red lines) in the back of the spine.

So before his bike accident, these ligaments were doing their job, helping to hold the spine in alignment. The picture to the left shows that the ligaments are holding things in place. They act to make sure that all of the vertebra stay aligned when you bend forward. They also control how much each vertebra is allowed to move relative to the vertebra above or below. These ligaments are represented as the red lines in the picture to the left.

After the accident, this is what I believe happened. Note that after the bike crash, the injury and tearing of these ligaments (gap in the red lines) allow the L4 vertebra to move forward on the L5 vertebra. Since these ligaments help to hold the vertebra in alignment when he bends forward, the facet joints (above red star, there are two at each level) move more than they can tolerate. This ultimately leads to excess wear and tear of these joints. Is there another part of this puzzle?

An interesting observation as he lies prone on the table is seen to the left. What gives with the severe bulging of left abdominal wall? Further questioning of the patient reveals that he also injured his kidney in the bike accident and had surgery on the left. The scar can be seen in the picture above. An important stabilizer of the back is the transversus abdominus. This muscle was likely cut through to get at the kidney, resulting in the muscle weakness you see above on the left side (inability to hold in the abdominal contents).
Putting all of this together into the ortho 2.0 triangle, results in the analysis to the left. The transversus abdominus is a muscle that's the deepest of the abdominal wall. It attaches to the thoracodorsal fascia and pulling on this muscle on both sides helps to allow the buoyancy of the abdominal contents to assist in off loading the weight of the upper body by literally floating it on the abdominal contents. It's also a major low back stabilizer all by itself.

The picture below shows that it attaches to fascia that then attaches to the back of the vertebra on both sides (spinous process). This axial view (saw you in half view) shows that if the pull is equal on both sides, this helps to keep the vertebra straight.
However, if we cut one side of the transversus abdominus muscle (for example to get to a damaged kidney), the forces on the vertebra will be unequal, causing it to have a slight tendency to rotate (in this case to the left). This forces on the right facet joint will increase, causing more wear and tear forces on that side.

To the left is an actual axial MRI image which shows the abnormal pull to the right by the transversus abdominus muscles (orange arrows) causing extra force on the right facet joint (yellow star). This is where the facet cyst is located.

So in summary, we believe that the damage to the ligaments in the back (supraspinous and interspinous ligaments) as well as this abnormal pull of one transversus abdominus over the other, have caused the facet joints to wear out. Their response on the right (the side where we would predict the most force) is to swell to try and keep up with the wear and tear. This led to a facet cyst and then ultimately pressure on the spinal nerve. This complex example illustrates the importance of piecing together all of the parts and pieces of what caused the musculoskeletal system to fail. Many times treating patients with musculoskeletal problems is as simple as a quick fix (in this case popping a facet cyst with a facet injection), other times it takes considerably more analysis.

**Pain Generator Gerry**

Gerry is a middle aged man who had a mid-foot fusion that caused severe lateral ankle pain and grinding. A surgeon took an x-ray that showed arthritis at the talo-tibial joint and decided to replace that part of her ankle. This caused more severe lateral ankle pain and grinding. What went wrong?

Regrettably, the association between arthritis on x-ray and pain is pretty weak. Time and time again, research studies show that patients with arthritis or degenerative joints on x-ray are often asymptomatic. We have published on this issue in the low back and the more recent discovery that 60% of knee meniscus tears don’t cause pain, has been blogged on in the past. There is also a recent study showing that pain causes arthritis, and not the other way around. This means that irritated nerves in the joint and presumably
in the spine, dump bad chemicals in the joint which ultimately degrades the joint. So with all of this data showing that we shouldn't rely on x-rays or MRI's to predict where pain is coming from, why do we see physicians treating the imaging every day? In this patient’s case, an x-ray showing degeneration of her tibio-talar joint lead to that joint being replaced. The pain got worse. Why?

Let’s start with the concept of fusion. A fusion is where the surgeon places hardware (screws and plates) to make a solid structure. Bone is also usually placed in the area to literally grow the two joint surfaces into a solid mass of bone, further freezing motion. The concept began with surgeons treating bony multi-trauma. This was a great advance that allowed surgeons to artificially fix a fracture through surgery and allow the patient much more activity than placing him or her in a cast and in traction. This has been applied to degenerated joints more recently. The theory is that if the joint hurts, fusion will prevent motion in the joint which will ultimately freeze its motion and the pain. The problem with fusion is that all joints are connected. Fuse one joint and the motion that should be carried by that joint gets thrown to the next joint in line. This force transfer from the fused joint to the next joint often causes arthritis at the next overloaded joint. The poor next joint in line just wasn’t designed to take that kind of force.

Let’s look at this patient’s fluoroscope image below. The fibula has been marked and the bottom part of it outlined. The first joints to be fused in the mid-foot (the bottom area marked as “FUSED”) caused those forces to be distributed to the joint between the bottom of the fibula (represented by the red arrow going up). This caused the joint between the end of the fibula and the rest of the foot (marked in red at the end of the fibula) to become overloaded and chronically painful. It likely also caused the other joint up the chain (the tibio-talar or one that was eventually replaced) to become degenerative. However, the catch was that the degenerated tibio-talar joint wasn’t causing pain. So when that joint was replaced (the bell shaped dark colored hardware and the downward facing ‘C” shaped hardware above that) the surgeon also placed the screws to fuse the fibula to the tibia (the area marked as “FUSED”
where the screws are located). This then caused more overload of the joint below (the red arrow going down). This made the pain worse, as this was the painful joint to begin with (not the one that was replaced). In the end, both the area below the painful joint and above that joint were fused, causing extra forces from above and below to be relayed into the painful joint. The result, more pain.

Could this have been prevented? YES. The way to prevent this was to perform diagnostic numbing injections under x-ray to see which joint was causing pain. In the end, it would have been determined that the tibio-talar joint wasn’t causing much pain (therefore did not need to be replaced) and the joint between the end of the fibula and the rest of the ankle was causing pain. How do I know this? This morning, injection of this joint at the end of the fibula eliminated her severe pain with walking.

The take home message? Big surgeries in the ankle and elsewhere can have big consequences that are many times irreversible. With the Regenexx procedure, we always take care to first diagnose where the pain is coming from before deploying cells at that location. If needed, this includes diagnostic numbing injections. The same should hold true for any patient considering a joint replacement. Just because the joint looks bad on x-ray or MRI doesn’t mean it necessarily causing pain!
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Dr. Centeno is one of the few physicians in the world with extensive experience in the culture expansion of and clinical use of adult stem cells to treat orthopedic injuries. He is a founding member of the International Cellular Medicine Society as well as the Spinal Injury Foundation. His clinic incorporates a variety of revolutionary pain management techniques to bring its broad patient base relief and results. Dr. Centeno treats patients from all over the US who travel to Colorado to under-go innovative, non-surgical treatments. Dr. Centeno has chaired multiple international research based conferences. He also maintains an active research based practice, with multiple publications listed in the US National Library of Medicine. Dr. Centeno has also served as editor-in-chief of a medical research journal dedicated to traumatic injury.

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