Dynamic Chiropractic

CHIROPRACTIC (GENERAL)

Paradoxical Terminology: The Instability of Stabilization!

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The topic of instability seems to have increased in popularity over the past decade. I believe the reason for this is that there are different clinical definitions of instability. Furthermore, there are different schools of thought on how to stabilize instability, paradoxically complicating the subject. I will discuss two schools of thought on this topic, which I find confuses practitioners, in an attempt to improve professional communication.

Instability Defined

Ashton-Miller defined instability as a condition in which a relatively small load applied to a system causes a marked, perhaps catastrophic displacement.[1] Panjabi hypothesizes that this occurs in joints when the "neutral zone," or physiological limits, becomes increased.[2] What is the cause of such a condition with regard to the locomotor system? What are the consequences?

Anatomical Instability

The first school of thought regarding instability is that which has been utilized in the classic orthopedic, or pathanatomical, setting. Here we find increased joint laxity, either congenital (as in conditions like Ehlers-Danlos syndrome) or stress-strain induced deformity and failure of stabilizing ligaments, with resulting joint hypermobility. A fracture, marked degenerative conditions or various neoplasms clearly can also alter joint stability and therefore fall under the pathomorphological model. The resulting hypermobility, such as increased translation, distraction and/or rotation, dangerously increases the moment and risks further injurious failure during joint loading.

Failure of ligaments can occur with a sudden overload or gradually via the phenomenon of creep. Creep occurs as a prolonged load transforms a connective tissue's properties from elastic to plastic. We know that ligamentous tissue has a certain tensile strength and can typically return to its normal length after a refractory period of decreased stress. However, this is only true with moderate deformity of up to about four percent of the tissue's length. Deformity of more than four percent will generally lead to microfailure or macrofailure, leaving the ligament permanently deformed. Further lengthening can lead to greater deformity such as a partial or complete rupture.[3]

Instability from ligamentous laxity can come from two different sources. The first is the obvious instability caused by the loss of a ligament that tethers one bone to the adjoining bone. We would find this utilizing the classic orthopedic tests such as stability testing for collateral ligaments or Drawer's testing for cruciate ligaments. The second source is caused by the compromised proprioception stemming from the stretch receptors of the damaged ligamentous tissue. Why? Because abnormally increased joint distraction, shearing or torsion must occur before joint stretch receptors of the lax

tissues are sufficiently stimulated to provoke activation of the local stabilizing musculature. This minor lapse in stretch afferentation can compromise reaction times of stabilizing muscles that surround and protect the joint.

Depending on the degree of damage, surgical repair may be the only option in cases of this type of instability. However, rehabilitation measures can be very effective to sufficiently stabilize the patient and delay or avoid surgery. We see such instances of instability in the knee, where the collateral or cruciate ligaments have been significantly compromised.

I call the above definition anatomic or morphological instability because it has been diagnosed and treated by orthopedic surgeons for quite a long time. In addition, it is a simple concept in that it is based on the relative simplicity of pathomorphology.

Functional Instability

The second model of instability is more complex than the first because it deals with the relatively complex issue of pathophysiology. This model is certainly less well documented in the literature because it is relatively new and because performing scientific validation is no simple task. Curiously, the therapeutic aspect, called active stabilization, is better documented than the instability it is designed to correct.[4] In this model, instability is defined as the inability of the locomotor system to maintain optimal ranges of motion, or neutral zone, to protect soft tissues from overload, pain and injury. In contrast to the first model, significant pathomorphology is not a requisite here. As such, I call this a functional instability.

Functional instability can be based on several disparate or integrated factors. These may include faulty proprioception; joint dysfunction or subluxation; muscle imbalance; weakness; fatigability; speed of muscular reaction; postural compromise; quality of the connective tissues; cerebellar or other CNS dysfunction; and residual faulty movement patterns from past injury (see Janda's centralization phenomenon as discussed in the November 1, 1999 issue of DC).[5] In addition, instability can result from a host of illnesses or disease processes that compromise any of the organ systems that are vital to optimal locomotor system function.

In this model of instability, the patient has lost the ability to maintain optimal posture, to move in a manner that protects the joints from end range overload and maintains balance and proper levels of strength and conditioning. Any of these problems endanger the joints and soft tissue, increasing the risk of pain syndromes and sudden injury and/or disability.[5]

The assessment and treatment of instability is extremely complex, far more than simply finding an elongated, torn or ruptured ligament. Generally speaking, it involves a deep understanding of locomotor system functions and skills that can assess each of the potentially causative factors outlined above.

The goal of such assessment is to find key links that can be altered to restore functional stabilization. For example, a patient may have a right sacroiliac subluxation or dysfunction that reduces the joint's mobility and causes compromised hypermobility of the neighboring L5-S1 facets. There may be an imbalance of the lumbopelvic musculature, such as weakness of the abdominal musculature, increasing the lumbar lordosis.

Where is the pain generator in this instance? The overloaded L5-S1 facets could logically be considered a reasonable location of pain. In this instance, manipulation of the right S/I and lumbopelvic stabilization would be a logical clinical approach. How would the patient respond with just the S/I adjustment and without the stabilization exercises? Perhaps the patient would respond well. However, I have found better and longer lasting results if I add a corrective stabilization exercise such as sensorimotor exercises, including proprioceptive training, pelvic tilting tracks and postural training.

This is a very simple example of a functional stabilization approach. Other beneficial techniques may include muscle-balancing techniques, gait training, ergonomic alterations, and a host of other schemes to arrive at your goal of reduced symptoms and improved locomotor stability.[6,7,8]

Incidentally, Bogduk notes concerns for the topic of instability because it has attracted so much attention yet has little scientific basis for its valid diagnosis. He appropriately warns against abuse of the term to explain any pain aggravated by movement.[9] Indeed, we have much to learn that will allow us more appropriately to establish guidelines in the assessment, diagnosis and management of instability and stabilization.

Because of paradoxical stabilization terms, be careful to clarify your meaning when discussing one of these issues with health care providers. One clinician may interpret you to refer to an obvious surgical case, while another assumes conservative measures are in order.

It is often said that the world is becoming a smaller place. It seems as though vocabulary space is becoming a bit cramped also. This is certainly the case for the paradoxical terminology of instability and stabilization, where the pathomorphologists and pathophysiologists have each claimed common ground.

References

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