EDITORIAL

THOUGHT PROCESSES: STABILITY, LIGAMENTS, TENSION, PAIN & PROLOTHERAPY

Most of us see a proportion of patients whose spines we consider 'unstable'. In the Journal we have read papers on 'spinal instability' and we know there is no satisfactory objective way of defining the problem in many of these patients. For those of us who use the ligament-strengthening treatment of prolotherapy, there are many critical questions to be asked.

In one type of unstable back, a history is given of pain induced or exacerbated by prolonged standing or sitting, and relieved by activity, the so-called "Theatre-Cocktail Party Syndrome" (Cyriax, Barbor). Clinical experience suggests strongly that this group of patients does well with prolotherapy. It is felt by some therefore that patients with unstable backs have ligament laxity with or without secondary muscular trigger points and pain.

Another type of low back instability is recurrent episodes of acute back pain associated with unguarded movements of the trunk. This also is considered by some to be due to lack of ligamentous mechanical support. This group benefits from prolotherapy, but seemingly less so than the group with the "Theatre-Cocktail Party Syndrome".

There is little consensus about which physical signs indicate low back instability. The osteopathic concepts of physiological and non-physiological somatic dysfunction imply that the soft tissue supports of the sacroiliac joints may under certain circumstances decompensate allowing a true subluxation of the sacroiliac joint. Others consider recurrent pelvic asymmetry or "asymlocation" to be significant. Whether this apparent asymmetry is actually displacement of bones or due to changes in the overlying soft tissues has been called into question (Lewit).

The sitting and standing flexion sign (or variations of these tests) are widely used in assessment of pelvic ring mechanics, but their reliability and significance have not been established. Presumably they demonstrate abnormal motion of the sacroiliac joint, but this does not necessarily translate into joint instability.

Various techniques exist to isolate some of the large low back ligaments and to test their sensitivity to stretch. If stretching the ligament reproduces the patient's pain, it is felt by some that this a sign of ligament pain (and laxity).

For those with above average manual skills, passive motion palpation of the pelvic ring joints and the vertebral segments gives direct evidence of relative joint hyper- or hypomobility (Lee, Hesch, Kidd). This probably says more about the stability of the joints than does assessment of symmetry and active motion testing. However these techniques suffer from three major limitations: one is that the needed skills are highly subjective, difficult, and time-consuming to acquire; another is that interexaminer reliability is so poor that they are of little value in research; a third is that although changes in motion characteristics can be detected at the time of the examination, they may not represent the stability of the joint in everyday life. This is where the modulating effects of muscle activity on ligament tension come into play (secondary hypermobility). Changes in posture and recruitment of different supporting muscles may change the patterns of joint stability entirely.

Another method exists of diagnosing low back instability. This rests on the assumption first proposed by Hackett that if chronic pain emanates from a ligament trigger point (always at the fibro-osseous junction), there exists "ligament laxity" at that point. The best evidence to support this view is that repeat injections of a proliferating solution frequently do indeed abolish the pain and the trigger points.

A number of difficulties arise however when the theory of "ligament laxity" is examined more carefully. The first is the question of whether pain at a ligament trigger point is always, usually, or only sometimes due to unresolved injury. In ligaments supporting central joints this is particularly difficult to determine, but examination of peripheral joints may provide some theoretical answers.

It should first be noted that joint instability by itself does not necessarily mean that lax ligaments supporting a joint produce pain even when stretched to a certain degree. A weakened anterior cruciate ligament can be demonstrated to be lax, without any pain being provoked when it is stretched. Clinical experience certainly suggests that lax ligaments may render the joint and its supporting ligaments more vulnerable to injury, but with everyday usage they are generally painless.

Not only are lax ligaments not necessarily painful, but also healthy ligaments may produce pain under certain circumstances. This may be easily demonstrated in a finger interphalangeal joint. If a proximal interphalangeal joint is gently abducted and tension put on the opposite collateral ligament for a minute or so, pain will gradually develop. Even when the tension is taken off, some pain will remain and a repetition of the strain will induce the same pain more quickly than before. In other words, the ligament has been sensitised by prolonged tension to more readily produce pain.

In most peripheral joints, active muscle contraction does not place abnormal tension on ligaments. In central joints however this almost certainly does occur. How often it occurs is open to conjecture, but abnormal postural patterns due to injury, illness, degenerative processes or emotional stress could not help but put prolonged abnormal, albeit mild tension on certain central ligaments. This may be the cause of at least some of the ligamentous pain blamed on ligamentous laxity.

Another cause of ligament pain is heightened sympathetic nervous system tone. Ligaments in the lumbar and pelvic regions are richly supplied with sympathetic nervous system efferent fibres. Sympathetic efferent fibres may activate primary afferent fibres or potentiate inflammatory processes by release of neuropeptides and catecholamines (Willard). This may explain the sudden (within days) lasting response to low back prolotherapy in some patients. Almost certainly this is a 'neural therapy' effect from the local anaesthetic which is always mixed into proliferant solutions.

Ligament pain does therefore not necessarily mean ligament laxity. And ligament laxity does not necessarily cause pain. Evidence of pain coming from a ligament is therefore not of itself an adequate indication for prolotherapy.

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