Letters to the editors

Control subjects in whiplash studies

Sterling et al. (2002) studied a group of chronically distressed whiplash patients, known to score high on somatization scales on the SCL-90-R (Moog et al. 2002), and compared them to a control group consisting of healthy volunteers. They find that whiplash patients report more pain with maneuvers that involve the upper limb and shoulder region. Sterling et al. decide to label this as ‘hyperalgesic responses’, later referred to as central hypersensitivity. There is no gold standard for the identification of this phenomenon, and no known site for its origin. Koelbaek Johansen et al. (1999) have previously made the same assumption that if one finds that chronically distressed patients give a different pain interpretation and response to a physical stimulus, then there must be some hypersensitivity of a central origin. Perhaps there is, but is it because of physical derangement, injury, or simply psychological distress? As in previous works of this type (Koelbaek-Johansen et al. 1999), Sterling et al. have failed to adequately control for the fact that whiplash subjects are psychologically distressed (Ferrari 2001).

There is a body of literature showing that if one selects healthy subjects, then make them anxious and distressed, physical stimuli will be perceived differently and recorded as a more-noxious stimulus than when the same subjects are not distressed (Levine et al. 1982; Barsky 1986, 1992; Robin et al. 1987; Barsky et al. 1988). Clearly, the healthy subjects of these experiments have no disorder prior to the distress and yet will appear to have the very phenomenon Sterling et al. reproduced. Moog et al. (2002) have recently shown that the group of whiplash subjects reporting pain with noxious stimuli used affective pain descriptors more frequently than those who did not report stimulus-induced pain, and had significantly higher affective but not sensory pain descriptor scores on the short form McGill Pain Questionnaire. It is clear that the presence of psychological distress may be just as valid an explanation for the findings of Sterling et al. (2002). The study design used by Sterling et al. cannot test for the cause or mechanism of central sensitization or hypersensitivity, as their control group was wholly inappropriate to address this important question.

We suggest, that the standard in this type of research has already been set and should be followed carefully (Carragee et al. 2000). Carragee et al. accomplished this in a study examining for responses to a noxious stimulus in chronic spinal pain. To explain, Carragee et al. wanted to examine the responses to the noxious stimulus of discography (known to be painful to some extent even in healthy subjects). Discography tends to be painful in chronic low back pain patients, and the question is whether or not the physical cause of the back pain is the cause of the response to discography, or whether the psychological distress of back pain patients is the predictor of the discography response, independent of chronic pain. Carragee et al. chose three groups as control subjects: (1) healthy subjects with no low back pain and no psychological distress, (2) subjects with chronic neck pain, but no low back pain, and (3) subjects with chronic anxiety or other chronic psychological distress, but no pain. As expected, the chronic low back pain patients reported more severe and diffuse pain with the injection much more often than the healthy subjects. What was unexpected was the 43% of the subjects with chronic neck pain but no low back pain also reported more severe and diffuse low back pain with the lumbar disc injection. This could mean that chronic neck pain patients with no low back pain share some common lesions with chronic low back pain patients. To eliminate the factor of chronic pain, one looks at the response of the psychologically distressed subjects without pain: 83% had a response just like that of the chronic low back pain patients. Thus, the independent predictor of the painful response to lumbar discography is not the presence or absence of chronic pain, nor necessarily the presence or absence of tissue pathology. It is the presence or absence of psychological distress that causes what should be a mildly painful stimulus to be registered as more severe and more diffuse (i.e. symptom amplification). That is what the chronic low back pain, chronic neck pain, and psychologically distressed subjects without chronic pain have in common. How does one interpret the results of Sterling et al.? Certainly, without a proper control group included in the study it is difficult.

Finally, one must once and for all put Elvey to rest, as well as the 19th century notion of nervous (brachial) irritation as a cause for the inexplicable (Ferrari et al. 2002). While it is understandable that we wish to provide some logical explanation for symptoms to patients, we do not believe that labelling subjective, non-specific symptoms as brachial plexus irritation is helpful. Diagnosing any brachial plexus lesion using non-objective data but rather on the basis of symptoms and/or manoeuvres which reproduce those symptoms is only valid and objective if research has already shown the symptoms...
to be specific, and the physical examination manoeuvres to correlate with other objective measures (i.e. not relying on the symptoms themselves) as a gold standard for the diagnosis. Surely, if we have no objective measurements to make in these patients, it does not mean one should discount their symptoms, but one should not be so pressed for a diagnosis that one confuses those rare cases of objectively provable thoracic outlet syndrome and brachial plexopathy with patients whose symptoms remain unexplained, or for which there may be many possible explanations yet to be defined.

The brachial plexus test of Elvey was developed by first manipulating cadaver’s arms until one could find a manoeuvre that stretched the nerves of the brachial plexus (Quintner 1989). Elvey could have just as easily suggested that this manoeuvre placed the glenohumeral capsule in a certain stretched position, arguing that nerves supplying the capsule are stretched in a specific way leading to arm paresthesiae. As Ferrari et al. (2002) have pointed out, we would then today be using the glenohumeral capsular test of Elvey as a basis for these same symptoms. The way Elvey designed the test by only viewing what the manoeuvre did to the brachial plexus and not other tissues and to then say this manoeuvre, when it exacerbates a symptom indicates a brachial plexus lesion is of course tautological. Again, the clinical specificity of the so-called brachial plexus test of Elvey or other similar manoeuvres has never been studied with regards to how often the sign appears in people with clear evidence of brachial lesions vs other diagnoses. In an area, filled with controversy, such as whiplash, we need more objective and carefully controlled studies before labelling patients with various disorders and pathology.

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References
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