Clinical commentary

Acute low cervical nerve root conditions: symptom presentations and pathobiological reasoning

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SUMMARY. Acute low cervical nerve root conditions may be easily misdiagnosed. The perspective presented is that their symptom presentation is not as straightforward as the classic descriptions of brachialgia would have us believe. This clinical commentary presents a series of observations and reasoning models that are relevant to patient symptom presentations believed to be of cervical nerve root origin. Clinicians are urged to consider low cervical nerve root assessment in the light of our current understanding of neural sensitivity, pain science, nerve root biomechanics and the presence and effect of degenerative changes. This particularly relates to thoughts about cervical movements and postures being able to bring forces to bear on nerve roots via compressive as well as elongation forces. © 2001 Harcourt Publishers Ltd.

INTRODUCTION

Nerve root disorders are often difficult to evaluate and may be misdiagnosed or go unrecognised. The early part of this clinical commentary urges practitioners to consider much of the recent work on the pathophysiological mechanisms relating to peripheral nerve related pain. The second part presents a descriptive analysis of the common features of patient pain presentations which are believed to have their origins in low cervical nerve roots. It is intended that clinicians may find the material useful during history taking and the clinical reasoning of aches and pains in the neck/shoulder and upper limb regions. Much of the material presented can be used in an appropriately modified form to help educate patients with acute and subacute cervical nerve root problems.

The clinical descriptions relate to acute nerve root disorders – here defined as the first 6–12 weeks. It is accepted that the material is speculative and can be challenged, being based on carefully recorded patient report and clinical observation and interpretation. There is plenty of scope for the observations described to be more robustly recorded and tested in an unbiased setting.

THE SPECTRUM OF CLINICAL PRESENTATIONS

It is proposed that many conditions exist on a clinical continuum, or spectrum, that range from the full-blown and blatantly obvious to the more obscure and harder to detect. It is as if these obscure and often minor problems have one or two components of familiarity about them but do not have the full compliment of features necessary to make a confident clinical diagnosis. Maitland (1986) used the term ‘subclinical presentation’. Three examples that represent the extremes of the nerve root clinical spectrum follow:

1. Diagnosis – ‘trapped nerve’
   ‘I’ve got pain from my neck down the arm, it’s constant agony, there’s numbness and pins and needles in the thumb and first finger, I think I’ve trapped a nerve in my neck’ (even the patient has a reasonable concept of the problem). When examined, neck movements clearly influence
symptoms and loss of reflex and segmentally related muscle strength are easy to detect.

2. Diagnosis – ‘Sprained shoulder muscles’
A 43 year old patient presents with a 10-day history of vague right shoulder aching pain following a series of vigorous tennis matches over several days. He has normal range of neck movement and some shoulder movements produce inconsistent sharp pains radiating from the shoulder down into biceps. For the patient, the focus is understandably on the shoulder and reinforced when the clinician finds positive pain responses to tests that mechanically load shoulder tissues.

On closer questioning it is revealed that the patient has had vague (‘of no consequence’) feelings of heaviness in the arm that last about 30 sec when rising in the morning and brief sharp pains around the medial border of scapula that he has noticed on and off for the last 3–4 months. Vague, but rare, arm pins and needles are also noted. Closer physical examination reveals a markedly reduced triceps reflex and exacerbation of the shoulder ache when the neck is rotated to the right and gentle right side flexion added and sustained for 10–15 seconds. By 15 seconds arm paraesthesia becomes evident. Sustained gentle pressure over the ipsilateral C6 nerve root anteriorly on the medial aspect of the transverse process of C6 increases the shoulder ache and reproduces fleeting sharp scapula pains similar to those mentioned.

3. Other Diagnoses
- ‘supraspinatus tendinitis’ for shoulder pain with positive static muscle tests,
- ‘sprained rib muscle’ for medial scapula pain
- ‘carpal tunnel syndrome’ if hand pins and needles only
- ‘epicondylitis’ if pain in the forearm

Closer scrutiny often reveals an atypical presentation for the given diagnosis. For example a carpal tunnel diagnosis where the patient reports paraesthesia in the whole arm as well as the hand.

At this ‘low’ end of spectrum it is not uncommon for patients to report symptom distributions and behaviours that show no detectable clinical evidence of loss of conduction but which are similar to other cases that show such losses. Example 2 above with a less dominant pain state and without any paraesthesia or loss of reflex would be a good example. Thus, the only way a component of nerve root involvement can be suspected is via a balanced analysis of physical tests that rely on symptom response and the fact that similar pain distributions and pain behaviours are often associated with detectable conduction losses in other patients. Suspicion of nerve root culpability is further strengthened when the patient suffers later episodes with similar pains that do reveal conduction abnormalities. It is not uncommon for conditions like those listed above to develop into a classic nerve root problem.

NERVE INJURY AND SENSITIVITY

Normal nerve roots are generally considered to be mechanically insensitive with the exception of the dorsal root ganglion area (Howe et al. 1977; Kuslich et al. 1991). However, nerve fibre axons (Devor & Seltzer 1999) and nerve fibre terminals within peripheral nerve sheaths (Bove & Light 1997) may upregulate their sensitivity in response to changes in their local environment. This may be a result of direct mechanical insults, changes in circulatory perfusion or the inward diffusion of irritative inflammatory chemicals from damaged or disordered adjacent soft tissues like the disc and facet joints (Olmarker et al. 1993, Byrd et al. 1995; Clatworthy et al. 1995; Tracey & Walker 1995).

It is feasible for nerve roots to be physically injured by extreme spinal movements, or by less extreme movements in the presence of degenerative changes that compromise the normal foraminal or spinal canal dimensions. Hence, disc protrusions and extrusions, vertebral approximation, osteophytes, facet enlargements, synovial cysts, and enlarged osteoligamentous structures like the uncovertebral joints, may all play a part in increasing the vulnerability of nerve roots to adverse postural or movement related forces. From studies of the lumbar spine it is clear that the likelihood of roots being mechanically compromised by normal end range movements increases with increasing degenerative change (Penning & Wilmink 1981; Penning 1992). In two neck cadaver studies, extension and ipsilateral rotation were found to be the most root compromising movements (Yoo et al. 1992; Farmer & Wisneski 1993, Byrod et al. 1995; Clatworthy et al. 1995; Tracey & Walker 1995).

Clinicians are urged to consider injuring forces in relation to movements that tend to compress or elongate nerve roots.

Nerve roots may become sensitised by changes in circulatory perfusion. Anything that diminishes space in the foramen is likely to alter normal circulatory pressure gradients and hence the normal circulatory flow through the nerve root. Space occupying material may be transient, e.g. oedema or extruded disc material (Maigne & Deligne 1994), or more permanent, e.g. osteophytes. Significant sensitising of the nerve may be achieved via compression of foraminal venousplexi, which as a result produces a back pressure and circulatory stasis within the nerve (e.g. Olmarker et al. 1989). Ongoing circulatory stasis may lead to ischaemia and the potential for intraneural oedema, inflammation and fibrosis (reviewed in Butler 1991; Gifford 1997a) (see Fig. 1).
The potential for circulatory distress to cause nerve fibre injury, degeneration and upregulation of sensitivity is evident. This knowledge highlights the likely detrimental effects of prolonged immobility, especially in postures that compress or elongate the nerve roots. It also draws attention to the fact that direct physical compression of a nerve root is not necessary for it to be injured and alter its sensitivity state. Thus, discs do not necessarily have to directly pinch or compress nerve tissue to produce pathological changes or sensitivity changes to the nerve.

Recent revelations about the capacity of axons to become pathophysiologically sensitised and active self-generators of impulses, goes a long way to help explain the way in which pain behaves clinically. Injured or degenerate/regenerating axons within nerve trunks or nerve roots can become sites demonstrating enhanced sensitivity as well as sources of ongoing and self-sustaining barrages of impulses that have the potential to cause long lasting and high intensity pains (Wall & Devor 1983, Devor & Seltzer 1999). These zones of abnormal impulse generation on axons are referred to as ectopic impulse generating sites (Devor 1996, Devor & Seltzer 1999), since impulses are normally generated at nerve fibre terminals.

Clinically, it may be worth analysing sensitivity states in the following ways (see Gifford 1997a & b):

- Increased mechanosensitivity. Here pressure and/or stretch on a nerve produces immediate symptoms (Devor & Seltzer 1999). Clinically, this means a more or less instantaneous increase in symptoms when nerves are either elongated – think of movements 'away from side of pain and components of neural tension/neurodynamic testing; or, when nerves are compressed – think of manoeuvres that tend to compress nerve, like cervical extension and movements towards the painful side. Note however that ectopic impulse generating sites may not always react in parallel with changes in the force applied to them (Devor & Seltzer 1999). Clinically, this may translate into sudden bursts of pain which instantly go, even if the pressure of the test is maintained or a test movement repeated, or symptoms continuing on after the stimulus is removed.

- Increased ischaemosensitivity (Gifford 1997b). Here, reasoning suggests that sustained compression or stretch of a nerve will compromise neural circulatory perfusion leading to ischaemia. Ischaemia, for example, via a lowering in local pH

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**Fig. 1—Mechanisms of root injury that can lead to symptoms.**
(Issberner et al. 1996; Steen et al. 1996), may then give rise to a steadily increasing barrage of impulses from an ectopic impulse generating site, or sensitised region of nerve. A common example is the progressive increase in distal paraesthesia provoked by holding the elbow in a flexed position, or crossing the legs for a long time. A nerve demonstrating clinical ischaemosensitivity would develop symptoms over time, but far more quickly and intensely than normal. Phalens’ test for carpal tunnel syndrome is a classic example where the onset of symptoms in the wrist flexion position may take only a few seconds to come on and then steadily builds up (see Durkan 1991). The same type of response is obtained in many acute nerve root disorders when test positions are sustained (see example 2 above).

- Increased adrenosensitivity. Sensitised nociceptors, dorsal horn cells and ectopic impulse generating sites on axons have all been shown to be capable of becoming sensitive to adrenaline and noradrenaline (Koltzenburg 1996; Devor & Seltzer 1999). This is thought to be the principle pathophysiological mechanism that underpins sympathetically maintained pain precipitated by nerve injury (overviewed in Janig & Stanton-Hicks 1996; Gifford 1997b; Gifford & Butler 1997; Gifford 1998c; Devor & Seltzer 1999; Scadding 1999).

  Clinically this link of adrenaline-like substances to increases in neural impulse generation helps turn our attention towards the potential influences of anxiety and stress, via a peripheral mechanism, on the behaviour of pain.

As noted above, ectopic generating sites can, of their own accord, generate ongoing or intermittent barrages of impulses spontaneously – when they are termed ‘ectopic pacemaker sites’. Clinically this may represent ongoing pain which waxes and wanes for no apparent reason or pain that mysteriously appears and disappears. Both are very common scenarios for acute nerve root pains and a source of much concern for the mystified patient.

Ectopic impulse generating sites are also known to be capable of becoming sensitive to many chemical mediators that includes inflammatory chemicals (Devor & Seltzer 1994; 1996; 1999). An injured nerve root that contains nerve fibres which have developed ectopic impulse generating capability may well be surrounded by inflammatory chemicals derived from nearby tissues that have been injured or are pathological. The inflammatogenic potential of nuclear fluid that has leaked out of the disc via radial fissures is a useful example (e.g. McCarron et al. 1987; Olmarker et al. 1995; Saal 1995). Inflammatory chemicals that derive from adjacent pathological or injured soft tissues may not only play a part in giving rise to ectopic impulse generators but may also be involved in stimulating and maintaining their activity. A major consideration is that pain behaviour is not necessarily mechanically patterned and the cause of the pain may not always be the result of direct mechanical insult.

In summary, this section highlights the need for clinicians to integrate known neural pathophysiology into reasoning in order to better explain the variable nature of peripheral nerve related pain behaviour. Relying solely on mechanical explanations to explain pain and its behaviour are likely to be inadequate. Inflammatory/metabolic sensitivity, ischaemosensitivity, adrenosensitivity plus central and higher centre processing also need to be considered.

**SYMPTOM PRESENTATION**

1. **Distribution of symptoms**

Even in a classic nerve root disorder, symptoms are usually distributed in a vague way – not in neat dermatoma patterns and packages that most literature on nerve root pain would have us believe. The patient usually indicates the area of symptoms in a general and imprecise way. The very nature of the pain is often one of being hard for the patient to accurately localise. It is pleasing for clinical reality that non-dermatomal and widespread distributions of symptoms have now been quantified by direct root stimulation in patients with cervical radicular pain (Slipman et al. 1998).

  In acute low cervical root irritation, common areas of distribution are: the lower neck spreading laterally towards the point of the shoulder; the medial border of the scapula; the whole of the scapula; the anterior neck to the upper pectoral area; down the back or front of the arm (the patient often grips the triceps/biceps to indicate the pain area); the lateral or medial forearm and into the hand. Symptoms are often more intense at particular sites and these are not necessarily proximal. For example, deep forearm ache that is particularly intense over the lateral elbow. It is not uncommon for some patients to use the term tennis elbow to help describe their symptoms. Another common area of intense pain is located deeply along the medial border of the scapula, or over the upper scapula area.

  Importantly, the area of symptom distribution is very variable, can be very patchy and is often hard to localise to a particular nerve root distribution (e.g. Fig. 2). When a patient describes their symptoms, it is important to be content with what is described and worry less about a textbook dermatome or precise root level that one might wish the pain to fit. A review of Slipman et al. (1998) confirms this.

  Other areas of symptoms that further challenge dermatomal pattern thinking include:

- Patients with C6 or C7 nerve root deficit having pain in the axilla area frequently radiating down...
onto the lateral chest wall and medial upper arm.
Many patients report symptoms spreading anteriorly into the pectoral/breast area
- Axillary pain with acute low cervical nerve roots seems very common
- Symptoms may also be reported in the anterior neck, clavicular and pectoral area.

At the lower end of the spectrum, symptoms may be found in any one of the above areas – alone, or in combination, and may have no symptoms at all anywhere near the neck (Fig. 2). Frequent descriptions include: deep intermittent aching in the biceps or triceps region and occasional sharp shooting pains in the lateral forearm; an annoying localised burning pain or itch in a small area along the medial scapula border that keeps the subject awake at night accompanied by a disturbing heaviness and tiredness of the ipsilateral arm during the day.

Symptoms of paraesthesia may fit more consistently into dermatomal patterns, though frequently do not (Fig. 2). Thus patients may report paraesthesia of the whole arm in vague patterns, or from the elbow to the hand with difficulty in describing the boundaries of its distribution. Alternatively, paraesthesia may be described in a single fingertip or one or two fingers that are typical of the relevant dermatome. Caution is strongly advised when allotting a specific root-level based on paraesthesia or anaesthesia location alone. For example, it seems common to find that a patient will complain of numbness/pins and needles in the thumb (classically, C6) yet be found to have a weak triceps and/or diminished triceps reflex (C7).

The important point is that symptoms rarely fit into neat dermatome distributions and are frequently in isolated areas well away from the nerve root and neck region.

2. Symptom quality
Symptom quality and behaviour may be a key defining feature of pain that has neurogenic origins. Well known authorities on the spine insist that nerve root pain is sharp and shooting in quality and well localized (Bogduk 1997; Waddell 1998). Time and again, this does not seem to fit with the everyday clinical reality observed. Even classic ‘objectively proven’ severe acute cervical and lumbar nerve root presentations invariably describe their pain as an unremitting intense tooth-ache like pain. The pain can be ghastly, unrelenting and extremely wearing, even for the most stoical and uncomplaining of people. Patients are desperate for help. Most patients would do anything, take anything or have anything done to them to get pain relief.

Sharp shooting pains do occur, but seem to be more common in elderly patients. Symptoms may be ‘sharp shooting’ or more commonly ‘knife in’ or
‘gripping’ pains, anywhere from the neck across the shoulders to down the arm. Sharp shooting pain may manifest on its own without any ache. Like aching, the shooting pain can be ghastly, the patient clutching the area of pain and strongly wincing for many seconds until the ‘after’ pain gradually subsides. It often occurs for no apparent physical reason, hence shooting pains at rest, whatever the position, or occurring one moment with a neck movement, the next with an arm movement. Patients are understandably worried, even frightened by these intense and mysterious pains. It is very easy to see why patients often view their condition in terms of serious pathology.

Symptoms are occasionally more bizarre. For example, feelings of crawling, trickling and sometimes extremely uncomfortable ‘gripping’ sensations. A very common description of the arm is that it feels ‘heavy’, ‘leader’ or ‘tired and useless’ a lot of the time. ‘The kind of feeling you get when you continuously overwork a muscle’ is a common description. At the less intense lower end of the spectrum words like ‘annoying’, ‘distracting’ and ‘irritating’ are used.

While these descriptive terms are not difficult to fathom, what is, is the difficulty the patient often has in tying the symptoms to particular things that they have done. An appreciation of ‘ectopic impulse generating sites’ capacity to develop sudden, spontaneous or ongoing activity or be influenced by non-mechanical stimuli, helps in the understanding and explanation of many of these types of phenomenon.

3. Symptom behaviour

Time: 24 hour behaviour

Acute nerve root symptoms may be constant and unrelenting 24 hours a day, with the result that the patient gets very little useful rest or sleep. Many patients report pain far worse at night and yet that they can manage during the day when on the move or distracted by their daily tasks. Pain at night is viewed as a marker of serious pathology and this must always be considered (CSAG 1994; RCGP 1996; Roberts 2000).

Less severe nerve root presentations show huge variability over 24 hours having no particular time related pattern from one day to the next. However, many do report having wakeful nights. Night symptoms are a common feature of many peripheral neurogenic disorders, recall the persistent night symptoms associated with carpal tunnel syndrome.

As nerve root syndromes recover they can often ease up significantly for a day or two, with the patient greatly relieved, but then return with former severity, adding much to the patient’s concern. It is worth warning the patient of this likelihood so that they know what to expect. Improvement often involves lurching from good periods to bad, but as time goes on the flare-up times get shorter and less intense.

Symptom behaviour related to posture and movements

Patients with intense acute nerve root pains rarely find consistent positions of relief and if they do it is only for a brief period of time. A key feature is that the patient becomes physically restless with the pain and greatly appreciates an understanding of this problem. ‘The doctor told me to take paracetamol and lie down and rest for 10 days’... ‘The last physiotherapist insisted I sat up straight and kept my neck in perfect posture. I’m sorry I just can’t keep it up, at first its better for a short time then I have to move and get relief by bending my neck forward’ – are frequent comments from patients.

Acute low cervical nerve root disorders may find short term relief in the following ways:

- Arm overhead – relief usually lasts for short periods. Patients may be forced into this position frequently if they have to sit for long periods – for example, driving the car with one hand on the wheel while the affected arm is raised with the hand resting on the head. This has been called the ‘shoulder abduction relief sign’ (Davidson et al. 1981; Beatty et al. 1987; Fast et al. 1989) and has been shown to significantly reduce intraforaminal pressures on the C5, C6 and C7 nerve roots in fresh cadavers (Farmer & Wisneski 1994). The mechanisms these authors proposed for this are: First, the shoulder abduction may cause the intervertebral foramen to enlarge therefore reducing pressure on the sensitised nerve root (If you do this your head tends to flex forward slightly).

Second, the abducted position reduces the tension on the nerve root. While this can be challenged by concepts of neurodynamics that use arm abduction to add neural tension, it should be appreciated that normal arm abduction allows the scapula to elevate and rotate towards the spine. Hence, the coracoid process may move several centimetres closer to the neck thus allowing considerable slack into the brachial plexus area and nerve roots (Davidson et al. 1981). All standard upper limb tension tests (ULTTs) either prevent the scapular elevation occurring or add in scapular depression before the arm abduction component is added (e.g. Butler 1991; Elvey 1994; Elvey & Hall 1997).

From this one would expect the patient to gain relief from sustained shoulder shrugging or sitting with plenty of pillow support along the forearm so that the whole arm/scapular unit is raised into elevation. Surprisingly, this does not always occur suggesting that either scapular rotation is needed as
Postures and movements into flexion and away from the side of pain. In a similar way to lumbar nerve root conditions, many patients with classic acute cervical nerve root disorders adopt postures that flex slightly and deviate away from the side of pain. Moving towards the side of pain or into extension is often very provocative if the nerve is in an extremely mechanosensitive state. This clinical finding fits with knowledge that the intervertebral foramina of the low cervical roots enlarge in flexion and movements away from the side of pain and diminish in extension or movements towards the symptomatic side (Ehni et al. 1990; Yoo et al. 1992). Thus, movements that decrease the size of the foramen tend to compress and increase pressure on the roots, and movements that increase the foramen size tend to decrease pressure on the root (see Farmer & Wisneski 1994). Interestingly, Farmer and Wisneski (1994) noted unpredictable results in their pressure change observations in cervical flexion. For example, C5 and C7 roots demonstrated modest increases in pressure in flexion and C6 showed modest decreases in pressure. It appears that the actual pressure exerted on the nerve root is not wholly dependent on changes in foraminal sizes in flexion positions. Proximal and distal tethering effects by variable intraspinal and extraspinal ligamentous structures that may tether the roots (Moses & Carman 1996); movements of dura relative to the often angulated course of nerve roots within the spinal canal (Nathan & Feuerstein 1970); postural effects on circulatory supply, and the position of the shoulder complex and arm are the sorts of issues that need to be considered too. Whatever the biomechanical findings from these few cadaver studies, in the clinic it is very common to find patients getting relief by adopting varying angles of, and sometimes extreme, neck flexion. It may be that there is far more significant reduction of root compression in flexion in the presence of local swelling, or a disc bulge, herniation or frank protrusion.

Many patients find that the only position that they can get comfortable enough to get to sleep in is by lying supine and having 2 or 3 pillows wedged behind the neck to maintain end of range flexion. While this goes against many principles of physiotherapy management it can be seen as a very adaptive ‘nerve saving/pain relieving’ position to adopt during the early very acute phase of the disorder. Rapid progression to restore normal posture and range may exacerbate or maintain pain (and possibly increase neural damage/sensitivity too) for longer than it otherwise would if left to ‘natural’, well ingrained reflex antalgic postures that have survived the test of time. Ubiquitous antalgic postures seem to have biological wisdom that should really dictate a graduated recovery of range and posture approach to management.

Postures and movements towards the side of pain. It seems that there are a small percentage of patients with cervical nerve root disorders who obtain relief by adopting postures deviating towards the side of pain. Although this appears incompatible with thoughts of foraminal compression it may be that some relief in root tension is achieved. In keeping with this is the clinical finding that the majority of those patients who prefer deviation towards the side of pain tend to have very clear-cut responses to neurodynamic testing. In these cases, the neural tissue appears to be more sensitive to stretch/elongation than to compression. Arm postures that can be associated include scapular elevation and the arm held into the side with the elbow flexed across the abdomen.

Patients with less obvious or more minor root problems often have great difficulty in identifying any clear relieving postures or movements. Consideration of factors mentioned earlier relating to the activity of ectopic impulse-generating sites and the ischaemo-sensitivity model for delayed onset symptoms may help make sense of the presentation and direct the type of examination testing. In desperation patients may find quite odd positions for relief. Examples from the authors patient population include: on all fours dangling the head down; a yoga shoulder stand; extreme cervical extension in prone and wedging the head in side flexion away.

If all else fails for the patient, the most common methods of relief come down to protective use of the part where the pain is felt, something which may draw clinical attention away from proximal often non-symptomatic areas and towards the hurting tissues as the primary source of the pain mechanism. It is important to bear in mind that mechanical effects on nerve roots can be altered by arm postures. Thus a patient may well find several arm postures that provide a degree of relief.

Even in some classic nerve root conditions, patients may not associate dominant distal symptoms with neck postures and movements. It is quite common to find patients having quite normal and free neck movements but considerable distal ‘radicular’ pain. It often takes a good examination and explanation to convince the patient that the primary site of pain generation is proximal and related to enhanced neural sensitivity where the nerves enter and leave the vertebrae of the neck.
The following postures and movements are frequently found to exacerbate the symptoms of acute cervical nerve root disorders:

- Looking up and extending the neck, sustained postures involving extension, repeated cervical retraction exercises, and movements towards the painful side.

One of the most consistent aspects of acute low cervical root syndromes is the production and provocation of symptoms with cervical extension movements, exercises and postures. This clearly fits with the decrease in foraminal dimensions reported earlier. The presence of extruded or bulging disc tissue, or any space occupying tissue or material will obviously enhance this effect. Clinicians need to bear in mind the age of the patient, the likely degenerate encroachment and the actual mobility of the low cervical segments. Loss of range of low cervical joints may account for a disproportionately low level of symptom provocation with standard neck movements in the elderly ‘stiff-necked’ patient – it may be that the motion segments just do not move enough to have any immediate physical impact on the sensitised nerve roots they contain.

Patients often report provocation of symptoms when performing activities like shaving, lying supine in bed with a single pillow, lying prone with the head in full ipsilateral rotation, swimming with the head up and hair washing in neck extension under a shower. Analysis often reveals significant and often sustained low cervical extension and that minor alterations of posture quickly or slowly change symptoms.

All positions and activities that patients volunteer as being provocative are worth analysing with thoughts of neural compression or neural elongation effects. For instance, patients often report being able to lie comfortably on one side but not on the other. Analysis of the number of pillows used combined with thoughts about compression/elongation can be useful. Thus a right-sided nerve root problem that is mainly provoked by root compression postures/movements tends to be more comfortable lying on the right side if the pillows are sufficient to side flex the head to the left. When the patient turns onto their left side, with the same number of pillows, the neck side-flexes towards the right, compresses the roots and hence tends to exacerbate the symptoms. Patients often find it odd that they are more comfortable lying on the painful side rather than off it. This type of ‘mechanically-based’ thinking can help explain the apparent paradox.

Clinicians are urged to be aware that nerves may respond in a delayed manner to insult or injury (Devor 1994; Devor et al. 1994; Devor & Seltzer 1999) and that this may well be from examination or therapeutic movements. Relying on pain response to mechanical testing like repeated movements may reduce or change the location of symptoms at the time but cause significant flare-up or worsening in the hours or days that follow. Personal experience of this delayed flare-up is so frequent that any repeated movements are prescribed with considerable caution. Movements that tend to compress nerve roots, like repeated extension or side flexion/rotation/side gliding towards the painful side appear to be most provocative.

- Movements away from painful side

This response fits with the cluster of patients who present with symptoms relieved by postures towards the painful side and who have positive neural tension/neurodynamic tests, i.e. ‘neural elongation’ root sensitivity.

- Flexing the neck

While many patients report relief in flexion, if the movement is taken towards the limit of range, symptoms may be further provoked. End-range flexion occasionally provokes distal/arm symptoms but more commonly produces local neck and yoke-area related discomfort. A small proportion of presentations have all symptoms severely brought on in flexion – these patients are usually extremely limited in most cervical movements.

- Arm movements

Patients with arm pain of root origin frequently report difficulties using the arm. Closer scrutiny usually reveals a very variable stimulus response relationship. Again, analysis should bear in mind neck posture and the effects of arm/shoulder/scapula posture on neural tension/elongation. Increased pain as a result of carrying shopping can be interpreted in terms of increased neural tension on the hyperalgesic roots. Positive pain responses to distal joint and muscle testing may reflect a secondary hyperalgesic state rather than any localised primary lesion (see below but also see Hasue 1993).

OTHER FACTORS TO CONSIDER

Clinicians should be far more aware that barrages of impulses arising from ectopic generating sites on peripheral nerves or as a result of nociceptor activity following tissue injury will cause changes in the central nervous system processing of normal sensory input from normal tissues (reviewed in Zusman 1992; Gifford 1997b; Johnson 1997; Gifford 1998a; Wright 1999). Barrages from ectopic impulse generating sites on sensory axons and cell bodies in peripheral nerves can be particularly unpleasant. Central nervous system changes result in the phenomenon of secondary hyperalgesia whereby normal inputs from normal
tissues get processed in the central nervous system in terms of pain, rather than innocuous sensations. The clinical significance of this is that many tissues that produce pain when physically tested by manual techniques using physiological movements, static muscle tests or palpatory pressures may be relatively normal. Thus, the central consequences of tissue/peripheral nerve injury may easily result in ‘false’ positive findings in examined peripheral muscles, nerves, joints, skin and any other soft tissues in areas segmentally related to the nerve, and in extreme/severe cases, tissues well beyond normal segmental limits. Physically testing or pressing on a particular structure and reproducing the pain the patient complains of does not therefore mean that the definitive source of the problem has been found (see Gifford 1997b; Gifford 1998a). All any ‘positive’ test response does is reflect the sensitivity state of the tissue examined and/or the sensitivity of the relevant processing pathways in the central nervous system. A sensitive tissue may or may not be significantly pathological.

For example, a 34 year old patient complained of having a heavy tired right arm with a low grade and fairly continuous forearm aching sensation that was starting to disturb his sleep. His major concern was a knife like pain well localised about one third of the way down the right medial scapula border. The problem was about 2 weeks old and described as worsening and becoming worrying. He reported having had 2 sessions of manipulative treatment for the scapular pain. This involved ‘firm’ neck and thorax ‘cracking’ but to little effect other than a feeling of freedom of movement for several hours. The arm problem worsened about 3–4 days after his last manipulation session. On examination the medial scapula area felt thickened and tight compared to the other side and modestly firm palpatory pressures exacerbated the arm pain within 15 seconds or so. Was this the source of the problem? It clearly was to the patient, but there were further striking findings to be taken into account. The patient had no triceps reflex on the affected side and the triceps muscle was markedly weaker to the astonishment of the patient. Neck movements were normal except end range low cervical extension which brought on a vicious ‘bite’ of neck pain. Anterior palpation of the neck over the ipsilateral transverse process of C7 also reproduced the scapula pain. This involved ‘firm’ neck and thorax ‘cracking’ but to little effect other than a feeling of freedom of movement for several hours. Upper limb tension tests were unrevealing.

Taking into account results like this adjusts the focus of attention towards low cervical nerve dysfunction and altered central sensitivity and relegates the medial scapula tenderness more to a secondary hyperalgesia status with the possibility of secondary changes (swelling and thickening) influenced via efferent neurogenic signaling (overviewed in: Hasue 1993). In fact, in this patient, further palpatory investigation revealed widespread and significant tenderness, or ‘pain related impairments’ over the lateral epicondyle of the elbow, the radial nerve in the radial groove and the belly of the supraspinatus muscle where medial border of scapular pain could again be reproduced. Skin light-touch comparisons with the non-symptomatic forearm revealed skin hypersensitivity too. Overpressure to glenohumeral flexion and elbow extension produced markedly more discomfort when compared to similar contralateral overpressures. A cursory examination is likely to miss significant findings that might otherwise add to the possible nerve root origins of the problem and the appreciation of altered processing factors within the central nervous system. There are great dangers, or ‘reasoning errors’, in assuming that just because a tissue palpated or tested reproduces a patient’s pain it has to be the ‘source’ of the problem. Pain distribution and pain response to testing are often very misleading and always difficult to interpret, even in acute conditions like these.

Finally, it is worthwhile making an observation with regards recovery and natural history. In the early days some nerve root disorders seem unrelenting. As a clinician one feels impotent and as a patient one feels desperate. Typically, the worst period is the first one to two weeks but may be as long as four or five weeks. Thereafter, and if well managed the condition usually gradually subsides with more and more good periods and the gradual restoration of normal activity. For patients, the ‘good news’ is that the symptoms do settle and most gradually settle, the ‘bad news’ is that it can take three months or longer. Some become chronic pain sufferers, but this possibility is reduced if they are managed well in the early stages (Waddell 1998; Watson 2000; Kendall & Watson 2000; Watson & Kendall 2000; Linton 1999).

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