



Acute low cervical nerve root conditions: Symptoms, symptom behaviour and physical screening

This article briefly reviews some theoretical and clinical issues relating to both the blatantly obvious and the easily missed low cervical nerve root presentations. One of the great difficulties rests in knowing whether what we see and describe clinically, actually represents what we believe it to be pathologically. This author freely acknowledges that there are many clinical observations, speculations and assumptions presented that are open to alternative interpretation. These are my clinical observations and interpretations.

It is proposed that, like many conditions, nerve root problems exist on a clinical continuum, or spectrum, that range from the full-blown and blatantly obvious to the more obscure and far harder to detect presentations. It is as if these obscure and often minor problems one encounters 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. Cervical nerve root disorders provide an excellent example of this spectrum concept. Three examples follow:-

1. Medical diagnosis given - '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. Medical diagnosis given - 'Sprained shoulder muscles'. A 43 year old patient presents with a 10 day history of vague shoulder aching pain following a series of vigorous tennis matches over several days. He has normal full 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.

One week and three treatments to the shoulder later, the patient is much the same or worse. The clinician reviews the situation and finds that the patient has in fact had

vague ('of no consequence') feelings of heaviness in the arm that last about 30 seconds when rising in the morning and a short lasting sharp pain around the medial border of scapula that he has noticed on and off for the last 3-4 months. He hadn't volunteered these when interviewed the first time because he hadn't even thought about them or been concerned about them. Closer physical examination revealed markedly reduced triceps reflex and exacerbation of the shoulder ache when the neck was rotated towards the side of pain, gentle side flexion added and sustained gently for 15-20 seconds, by 30 seconds the hand paraesthesia became evident. Sustained anterior palpation of the neck over the ipsilateral C6 nerve root on the medial aspect of the transverse process of C6 increased the shoulder ache and reproduced fleeting sharp scapula pains similar to those mentioned.

3. Medical diagnosis given - e.g.s supraspinatus tendinitis for shoulder pain with positive static muscle test, sprained rib muscle for medial scapula pain, carpal tunnel syndrome if hand pins and needles only, epicondylitis if pain in forearm etc...

Finally, at this lower end of the spectrum, it is not uncommon to have patients that report symptom distributions and behaviours that show no detectable clinical evidence of loss of conduction but are similar to other cases that do 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 we can assume nerve root involvement is via a balanced analysis of physical tests that rely on pain 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. In other words the condition has moved further into the pathophysiological spectrum.

A note on sensitivity

Normal nerve roots are generally considered to be mechanically insensitive with the exception of the dorsal root ganglion area (e.g. Howe *et al*, 1977; Kuslich *et al*, 1991). Axons of nerve fibres and nerve fibre terminals within

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nerve roots 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 adjacent soft tissues like the disc (for overview, see Gifford, 1997a) (see figure 1). Thus, individual nerve fibres within nerve roots may become mechanically sensitive as a result of local inflammatory mediators perfusing into the roots (e.g. Byrod *et al*, 1995; Olmarker *et al*, 1993) and/or as a result of adverse physical forces:- Nerve roots may 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. Thus, the likelihood of roots being mechanically compromised by normal end range movements increases with increasing degenerative change (e.g. Penning, 1992; Penning and Wilmink, 1981).

Any space occupying material 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 - for example oedema or extruded disc material (Maigne, 1994), or more permanent, for example osteophytes. A significant injuring mechanism is thought to

be via compression of foraminal venous plexi, which as a result produce 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 figure 1). The potential for this to cause nerve fibre injury, degeneration and upregulation of sensitivity is self evident. This knowledge highlights the likely detrimental effects of prolonged immobility, especially in postures that tend to compress the nerve roots (see below). 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. For example, discs do not necessarily have to directly pinch or compress nerve tissue to produce pathological changes in the nerve.

Injured or degenerate axons within nerve roots can become sites demonstrating enhanced sensitivity as well as sources of ongoing and self sustaining barrages of impulses that result in continuous pains (reviewed in Gifford, 1997b). Zones of abnormal impulse generation on axons are referred to as *ectopic* impulse generating sites (Devor, 1994; Devor, 1996), since impulses are normally generated at nerve fibre terminals and then travel along axons. Sensitivity may be of several kinds:

- **Mechanosensitivity.** Here pressure and/or stretch on a nerve produces markedly increased firing and hence pain (Devor, 1994)

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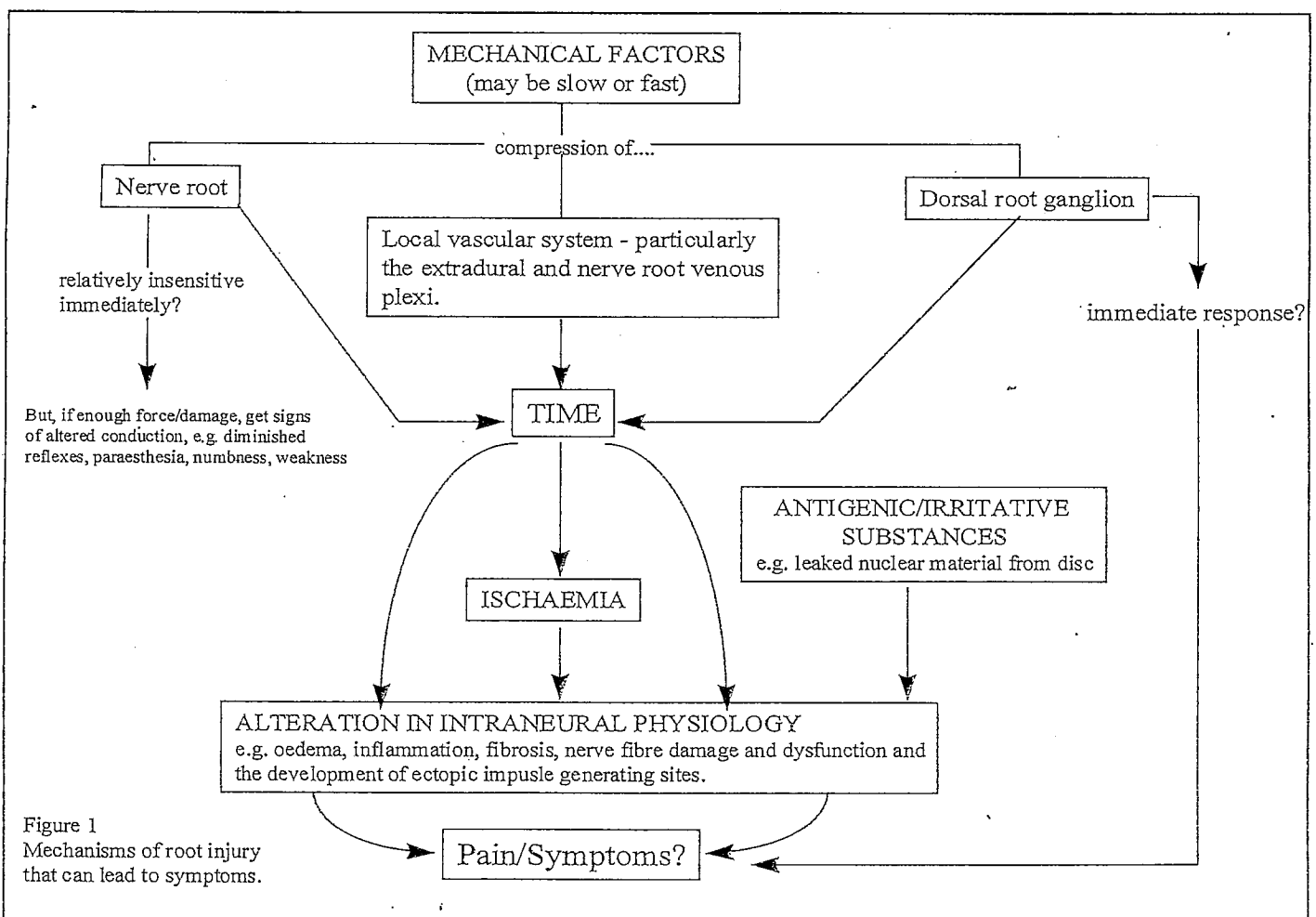


Figure 1: Mechanisms of root injury that can lead to symptoms.

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Clinically this means a more or less instantaneous increase in symptoms when nerves are either *elongated*:- think movements 'away' from side of pain and components of neural tension/neurodynamic testing. Or, when nerves are *compressed*:- think of manoeuvres that tend to 'pinch' nerve, like cervical extension and movements towards the painful side. Ectopic impulse generating sites may not always react in parallel with changes in the force applied to them (Devor, 1994). Clinically, this may translate into sudden bursts of pain which instantly go, even if the pressure of the test is maintained, and symptoms continuing on after the stimulus is removed.

- **Ischaemosensitivity** (this is speculative: see Gifford, 1997a). Here, sustained compression or stretch on a nerve compromises neural circulatory perfusion and results in steadily increasing barrages of impulses from the ectopic impulse generating site. If you think about it, this happens normally with some nerves - holding the elbow in a flexed position for a long time gives rise to distal paraesthesia in some people. Some of us are clearly more sensitive to this than others which amply illustrates the great variability of response likely to occur between individuals - whether there is pathology or not. Clinically patients with nerves demonstrating pathological ischaemosensitivity develop symptoms over time but far more quickly than normals do, symptoms are far more intense and includes pain as well as dysaesthetic symptoms. 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 steadily builds up. The same type of response is obtained in many acute nerve root disorders when test positions are sustained (see below).

- **Adrenosensitivity.** Ectopic impulse generating sites have been shown to be capable of becoming sensitive to adrenaline and noradrenaline (Devor, 1994; Koltzenburg, 1996). This is thought to be the principle pathophysiological mechanism that underpins sympathetically maintained pain precipitated by nerve injury (overviewed in Janig and Stanton-Hicks, 1996, but see also Gifford, 1997b; Gifford, 1998; and Gifford and Butler, 1997). 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 on pain perception via mechanisms based in peripheral tissues.

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 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. 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 inflammatorogenic 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 give rise to ectopic impulse generators but may also play a part in maintaining their activity.

Symptoms:

1. Distribution of symptoms

Even in a classic nerve root disorder, symptoms are usually distributed in a vague way - not in neat dermatome patterns and packages that most of the literature on nerve root pain would have us believe. The patient often indicates the area of symptoms in a general and imprecise way.

Key areas are: the lower neck spreading laterally towards the point of the shoulder; the medial border of scapula; the whole of the scapula; 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 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.

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. Thus, when a patient describes their symptoms, we should be content with what is described and worry less about a textbook dermatome that we would wish the pain to fit into.

Symptoms of paraesthesia do tend to fit more consistently into dermatomal patterns, especially distally in the hand. This is probably because paraesthesia is felt in the skin and the skin has an excellent representation in the sensory cortex. In my clinical experience it is fairly 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).

Other areas of symptoms that further challenge dermatomal pattern thinking include: C6 or C7 nerve roots having vague pain in the axilla area frequently radiating down onto the lateral chest wall and even spreading anteriorly into the pectoral area. Axillary pain with cervical nerve roots is 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 alone or in combination in any one of these areas and there may be no symptoms anywhere near the neck. Examples are deep intermittent aching in the biceps or triceps region and occasional sharp shooting pains in the lateral forearm. It may be an annoying localised burning pain or itch in a small area along the medial scapula border that keeps them awake at night accompanied by an annoying heaviness and tiredness of the ipsilateral arm during the day.

2. Symptom quality

Symptom quality and behaviour may be a key defining feature (Figure 1) of pain that has neurogenic origins. Classic nasty nerve root presentations often describe their pain as a

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horrendous tooth-ache like pain in the arm. The pain can be ghastly, unrelenting and extremely wearing, even for the most stoical and uncomplaining of people. Patients are desperate for help and it is very tempting for clinicians to do desperate things to help them out of their misery.

Some patients complain of sharp shooting pains or 'knife in' pains, anywhere from the neck across the shoulders to down the arm. Sharp shooting pain may manifest on its own without any ache. Again the shooting pain can be ghastly, the patient clutching the area of pain and strongly wincing for many seconds as the 'after' pain gradually subsides. Shooting pain without any accompanying ache seems more common in elderly patients and often occurs for no apparent physical reason - hence shooting pains occurring at rest, whatever the position, or occurring one moment with a neck movement the next with an arm movement. Patients understandably are often very worried, even frightened by these ghastly and mysterious pains. It is very easy to see why patients often view their condition in terms of serious pathology.

Paraesthesia and numbness have already been mentioned. They are usually fairly well localised.

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', 'leaden' 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 lower end of the clinical spectrum similar words (ache, sharp, knife, heavy, dull, tired) are used to describe the symptoms but the intensity and frequency is far less. Words like 'annoying', 'distracting' and 'irritating' are used. While this is 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 and spontaneous activity helps in the understanding of these types of phenomenon.

3. Symptom behaviour

Time-24 hour behaviour

Acute nasty nerve root symptoms may be constant and unrelenting 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.

Patients with less severe nerve roots show huge variability over 24 hours having no particular time related pattern from one day to the next. However, many do report having troublesome times during the night too. Night symptoms are a common feature of many peripheral neurogenic disorders, recall the persistent night problems that patients with carpal tunnel syndrome report.

As nerve root syndromes recover they can often ease up significantly for a few days, with the patient greatly relieved,

but then return with former severity, adding much to the patients concern. It is worth warning the patient of this likelihood so that they know what to expect. Improvement often involves lurching from several good days to several bad, but as time goes on the flare-up times get shorter and shorter and less intense.

Symptom behaviour related to posture and movements

Patients suffering nasty acute nerve root pains rarely find consistent positions of relief and if they do they are only comforting for a brief period of time. A key feature is that the patient become *restless* in their quest for relief and greatly appreciate an understanding of this problem. 'The doctor told me to take paracetamol and lie down and rest for 10 days'; 'The last physio 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 while then I have to move and get relief by bending my neck forward'.

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

- **Arm overhead** - relief lasts for *short periods* in most cases and patients are forced into this position frequently if they have to sit for long - e.g. driving the car - one hand on wheel while the affected side arm is raised with the hand resting on the head. This has been called the 'shoulder abduction relief sign' (Beatty *et al*, 1987; Davidson *et al*, 1981; 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 and 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. Second, the abducted position reduces the tension on the nerve root. While this seems to fly in the face of thoughts about classic neural tension manoeuvres - 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 (see Butler, 1991).

From this one would expect that the patient would gain similar relief from sustained shoulder shrugging or sitting with plenty of pillow support along the forearm so that the whole arm/scapular unit was raised into elevation. Surprisingly, this does not always occur suggesting that either scapular rotation is needed as well, or, relief by reducing root tension or pressure is only one possible explanation behind the 'shoulder abduction relief' position.

- **Postures and movements into flexion and away from the side of pain.**

Patients with classic nasty 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 (immediate pain with movement). This clinical finding fits with knowledge that the intervertebral

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foramen 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 and 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; movements of dura relative to the often angulated course of nerve roots within the spinal canal (Nathan and 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 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.

• Extreme cervical flexion in lying

Some 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 very acute phase of the disorder. Hurrying to restore normal posture may often maintain pain (and possibly increase neural damage too) for longer than it otherwise would if left to 'natural' well ingrained reflex antalgic postures that have survived the test of time.

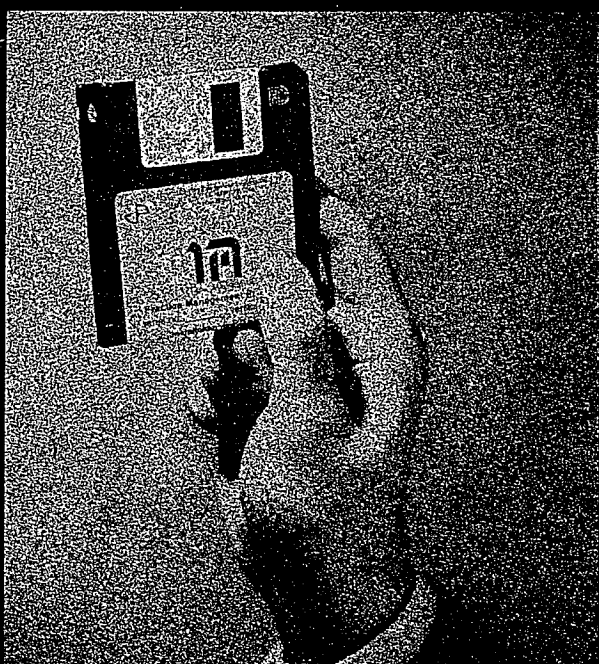
• Postures and movements towards the side of pain.

It seems that there are a small percentage of cervical nerve root disorders that get 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 a very clear cut and positive response to ULTTs. In these cases, the neural tissue appears to be more sensitive to stretch than compression.

Patients with less obvious or more minor root problems often have great difficulty in identifying any clear relieving postures or movements. The level of symptoms simply may not demand it or there may be insufficient or inconsistent mechanosensitivity for the patient to make sense of any particular movement or posture that may be provocative. Delay in symptom provocation - as suggested by ischaemosensitivity, further

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ONE STEP AHEAD



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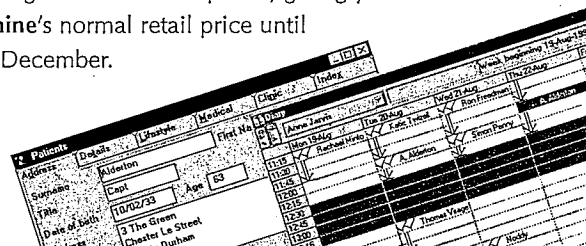
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detaches the perceived connection of a symptom increase from a provocative movement or posture.

Occasionally, odd arm positions that may include arm elevation, cervical postures in flexion, deviation away or towards, and rarely, cervical extension are reported. This type of information from patients with odd presentations can provide useful hints for the inclusion of an investigative focus on the mechanosensitivity of the low cervical nerve roots.

If all else fails, the most common methods of relief come down to protective use of the part where the pain is felt, something which may draw our clinical attention away from proximal non symptomatic areas and towards the painful tissues as the primary source of the problem. 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 postures involving the unaffected arm.

Even in some classic nerve root conditions, patients may not associate dominant peripheral symptoms with neck postures and movements. Frequently patients have quite normal and free neck movements but considerable nerve root pain. It often takes a good examination and explanation to convince the patient that the primary source is proximal and related to enhanced neural sensitivity where the nerves enter and leave 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, 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 pathology, will obviously enhance this effect. 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. Sitting, especially slumped postures with forward head positions that promote low cervical extension, commonly aggravates the symptoms. 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 (see below). 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

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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 thinking can help explain the apparent paradox.

- **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 and most of the tests discussed below are found to be positive too.

- **Arm movements**

Patients with arm pain of root origins 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).

4. Other factors to consider:

Up to date clinicians should be aware that barrages of impulses arising from ectopic generating sites on peripheral nerve 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 (e.g. see Gifford, 1997b; Gifford and Butler, 1997; Johnson, 1997). Barrages from ectopic impulse generating sites are particularly spiteful. These 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 therapy techniques using physiological movements, static muscle tests or palpatory pressures may be relatively normal.

Thus, nerve root 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

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problem has been found (see Gifford, 1997b). All any 'positive' test response does, is reflect the sensitivity state of the tissue examined - it may or may not be significantly pathological (see next section).

For example a 34 year old patient complained of having a heavy tired 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 medial scapula border on the same side. The problem was about 2 weeks old and described as worsening and becoming worrying. He had 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 4-5 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 extension which brought on a vicious 'bite' of pain in the medial border of scapula. The patient was unaware of this finding until testing. Anterior palpation of the neck over the ipsilateral transverse process of C7 also reproduced the scapula pain and increased the forearm discomfort. ULTTs were unrevealing.

Taking into account results like this adjusts the focus of attention towards nerve dysfunction at the C7 root level 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 signalling and secretions (for good overview see Hasue, 1993). In fact, further palpatory investigation of this patient revealed widespread and significant tenderness 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 more cursory examination, or one that is wholly biased to a single tissue approach is likely to miss significant findings that add to the likely nerve root origins of the problem and the appreciation of altered processing factors within the central nervous system. There are great reasoning errors in assuming that just because a tissue palpated or tested reproduces a patients pain it has to be the 'source' of the problem. Pain distribution and pain response to testing are often very misleading, even in acute conditions like these.

Physical screening:

There are not many formerly recognised tests for cervical nerve root dysfunction and I am only aware of three that have been subjected to reliability and validity testing (see, Viikari-Juntura et al, 1989). These tests are Spurling's test, axial manual traction and the shoulder abduction test. Spurling's test - neck

extension, rotation towards add compression (Spurling and Scoville, 1944), is pain provocative, while the other two are held to be positive when pain is relieved. These researchers attempted to include the brachial plexus tension test but unfortunately discarded it from validity analysis because of poor inter-examiner reliability (see Viikari-Juntura, 1987). When compared to the simplicity of the SLR, the ULTTs may be far too demanding of manual dexterity to be usefully repeatable for untrained researchers. The fact that intra and extra examiner reliability has been repeatedly shown for these tests means that researchers either need better training, or the use of a simplified version of the ULTT (see below)!

The three tests above were shown to be highly *specific* for cervical nerve root disorders (Viikari-Juntura et al, 1989). If a test demonstrates high *specificity*, a 'positive' response will always 'rule in' the diagnosis. However, although the three tests showed high *specificity*, they unfortunately showed low *sensitivity*. When a test demonstrates high *sensitivity* the diagnosis can be confidently ruled *out* if the test is *negative*. Thus, because the three tests have a low sensitivity value, when they are not positive on a given presentation it does not necessarily mean that the nerve root is normal. Many abnormal and highly sensitive nerve roots do not have a positive Spurling's test and do not show a lessening of symptoms with the arm abduction or traction tests. On the other hand, if the tests are positive the clinician should have increased confidence that the nerve root is a highly likely source. Note that these tests were deemed positive in relation to changes in 'radicular symptoms in the forearm-hand' (Viikari-Juntura et al, 1989). The clinician should appreciate that Spurling's test, for example, physically challenges a great many tissues in and around the neck. Thus producing local pain with this test may have more to do with local soft-tissue sensitivity rather than with nerve root pathology/sensitivity. Thus, the key confidence enhancer, is alteration of distal symptoms.

This research based approach serves as a useful challenge to our clinical reasoning skills which are so often clouded by bias to a favourite theory or test protocol. The rational and open minded clinician wisely sees other possibilities during testing, especially when tests rely on symptom responses and physical strain on multiple tissues. For example: if an SLR reproduces a patients back and leg pain it is faulty reasoning to say that a nerve root *must* be demonstrating enhanced sensitivity as the SLR influences many other structures that could well be partly or wholly culpable too. It is also faulty reasoning to say that if the SLR and slump test did not reproduce this person's back and leg pain it cannot be a sensitised nerve root. It could still be related to nerve root but due to anatomical anomalies, root tethering, lack of range in some joints, protective spasm by hamstrings etc. the mechanical effects of the SLR may not reach the sensitive segment of the nerve root. In addition, injured nerve may be sensitised but not necessarily mechanically sensitised, or not mechanically sensitised to elongation/stretching forces. The irrational dominance of one particular test (like a positive SLR or ULTT or palpation reproducing symptoms) in deciding whether a particular condition is present or not means that many patients are often denied adequate investigation and management options, or are subject to unnecessary invasive procedures.

Reliance on pain responses to physical testing to give unequivocal tissue based diagnostic power is not really tenable. Positive pain responses to physical tests merely builds-up an 'enhanced sensitivity' picture of various tissues considered to be influenced by the test procedure. Clinicians are urged to consider that pathobiological mechanisms responsible for pain are extremely complex and go far beyond the tissues examined physically (Gifford, 1998b; Gifford, 1997b; Gifford and Butler, 1997). The safest conclusion of any test is therefore that the tissue(s) under consideration are demonstrating an enhanced sensitivity state (hyperalgesia/allodynia) and may or may not be 'pathological'. Even if there is pathology in the tissues this may still have little bearing on the outcome of the condition or the response to treatment.

The next section presents a series of tests and a conceptual approach to testing whose formal purpose is to gather evidence that can be scrutinised and weighted before a diagnostic hypothesis is made with regard to a sensitised low cervical nerve root. Many of these principles can be applied to lumbar and thoracic nerve root disorders too.

Nerve root testing rationale

- When performing any physical movement or test procedure it is simple to think: Is this test *compressing* the root or *elongating* the root? The recent wave of enthusiasm for neural tension tests has rather sidelined thoughts of tests that tend to compress neural tissue. Combinations of compression and elongation are of course possible. For example, the median nerve in the carpal tunnel will be elongated and compressed when the hand is taken into wrist and finger extension. Performing an ULTT with the patient's neck in extension and ipsilateral rotation can also be viewed as having elongatory and compressive components.

The subjective examination should provide clues as to whether a root problem has a bias towards provocation by neural compression, or provocation via neural tensioning. For example if movements, activities and postures that provoke pain are analysed as being more towards the painful side or into extension, expect more of a 'compression' sensitivity type.

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- Note the timing of the pain response to a test. Is it immediate? If so then it can be assumed that the tissue(s) under test are demonstrating enhanced mechanosensitivity. If symptoms take a while to appear and slowly build up and spread, the pain response may relate more to an ischaemosensitivity. Cervical nerve root syndromes are often slowly increased by sustained ipsilateral rotation. The longer the position is held the more intense the pain becomes and the more it tends to spread distally and involve all areas. Sometimes the pain is described as slowly 'oozing' down the arm in tandem with slowly increasing symptoms of paraesthesia. A problem with this simple division between ischaemosensitivity and mechanosensitivity is that symptoms often come on immediately and build up and up anyway, even after the test is completed. Ectopic impulse generating sites may only require a brief mechanical stimulus to initiate a cascade of ongoing impulses that can take many hours to settle. (This is discussed further in: Gifford, 1997a).

An issue of particular importance is the potential of end range tests, many manual therapy techniques or therapeutic exercises to harm or severely irritate a nerve without any clear or major pain response at the time. Mechanosensitive peripheral neurogenic problems of all types are frequently much worse after physical examinations and treatments - even though they appear to respond well at the time in terms of pain reduction and phenomenon like pain 'centralisation'. Devor (1994) highlights the important laboratory finding that nociceptive C fibres may only develop significant spontaneous ectopic activity days or even weeks after the injury process. Thus, injury now, may mean worsening symptoms in several days time. Relying on instantaneous pain response for treatment efficacy needs to be done with caution.

Physical screening and analysis:

For descriptive purposes physical screening analysis has been divided into two phases. The experienced clinician can skilfully shift back and forth between the two phases and selectively focus on particular aspects at any given time.

Phase 1:

Neurological examination essential (see below).

Movement overview:

Begin by briefly observing posture, gross movement quality and ranges. Look for tentative movements, non-detailed symptom responses and major or minor abnormalities in range that may be focused in on later.

- All cervical movements
- All glenohumeral movements
- All scapula movements
- Forearm and hand movements
- Other movements, for example provocative or relieving postures and movements that the patient has already mentioned.

Phase 2:

Specific physiological tests:

- For all tests below, if appropriate: add overpressure gently, if no response - sustain up to 15 - 20 seconds. Sustaining up to

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45 seconds has been advocated for peripheral neuropathy (see Durkan, 1991).

- For test responses to more strongly add weight to a nerve root 'focus' they should particularly bring on distal symptoms, or symptoms in tissues not physically influenced by the testing.
- The direction taken for closer scrutiny depends on prior knowledge of provocative movements - i.e. from the movement overview above and from features of significance in the subjective examination. For simplicity the condition may be divided into neural compression type or neural elongation type.
- The order of the tests given is the rough order in which they may be performed. The key to the approach is maximal information with least force and least potential provocation to the sensitised nerve root. Obviously it may not be necessary to perform all tests.

Nerve root demonstrating 'compression' sensitivity: One or several of these tests may alter symptoms:

- **Patient Standing:**
 - Shoulder abduction test, often done first and classically relieves symptoms.
 - The active ULTT (may or may not be provocative with 'compression' type). This looks at relevant components. For example the effects of active scapula elevation and depression with elbow extension or flexion. In a more precise way, face the standing patient, and give the patient the following instructions while you also demonstrate the components: 'Keep your arm by your side, keep your elbow braced comfortably straight, hands and fingers straight, now reach downwards by letting your shoulder drop, extend your wrist and fingers back, now keep all that on and slowly take your arm out to the side....' It may only be necessary to go to very early stages of the test which should be performed very slowly and carefully. Neck side flexion can be added in at any of the stages.

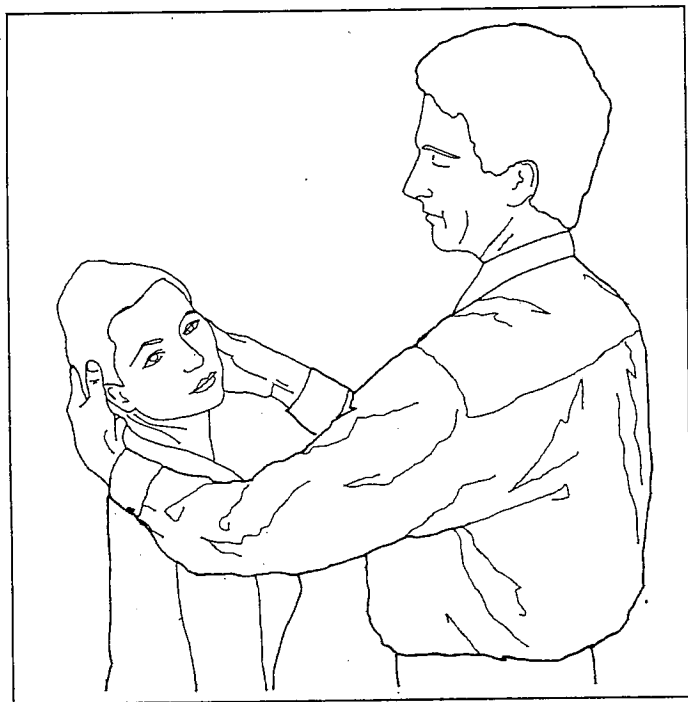


Figure 2: Examination of cervical spine: Right rotation plus right side flexion tends to compress the right low cervical roots in the intervertebral foramen. It will also elongate the roots on the left side. (Reproduced, with permission, from Butler and Gifford 1998 *The Dynamic Nervous System*. NOI Press, Adelaide.)

It is my opinion that in many circumstances the patient, if taught well, can be far more accurate in establishing and maintaining joint positions for complex tests like this than can be achieved by purely passive testing.

It is always best to perform the test on the non-symptomatic side first - it provides practice and it can also be used as an instant comparison by the patient. I often say to the patient something like: 'The next test tests the nerves in your neck and arm for their sensitivity to being stretched slightly. It normally produces some sensations of stretching or pulling in the arm and occasionally the hand. Before doing it on your bad arm I am going to get you to perform the test movements on your good arm so that we can find out what your normal response is to compare to the bad side in a moment.... If you get any sensations let me know what they are and where and remember them so as to compare when we look at the bad side'.

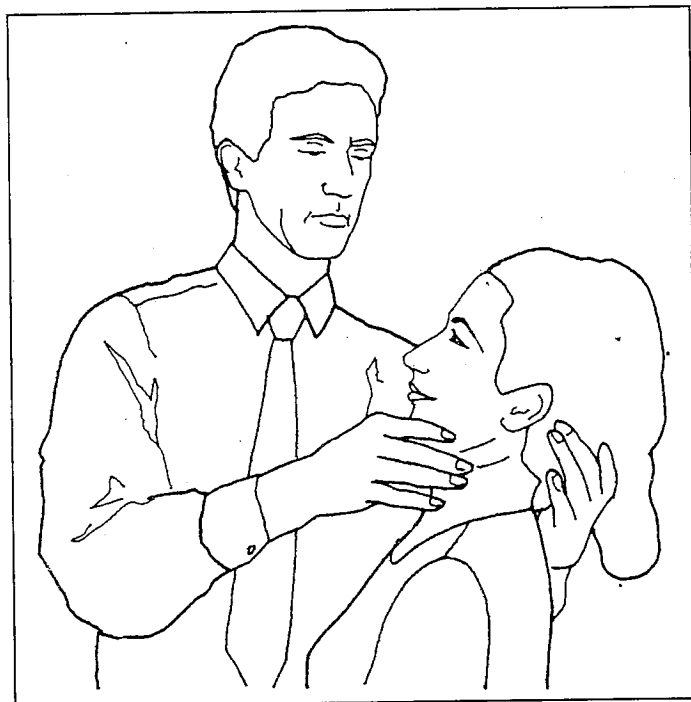


Figure 3: Retraction plus low cervical extension - bilaterally narrows the low cervical intervertebral foramen and can be very provocative for low cervical nerve roots that have become mechanically sensitised. (Reproduced, with permission, from Butler and Gifford, 1998, *The Dynamic Nervous System*. NOI Press, Adelaide.)

In the roots demonstrating dominant 'compression' sensitivity there may only be minor differences between sides for this test or any of the ULTTs. This is rather like a sciatic problem having relatively normal range and sensitivity of SLR and slump test, yet symptoms easily provoked by movements and postures involving extension.

- **Patient Sitting**
- Rotation towards.
- Side flexion towards (if necessary making sure that the lower cervical spine is moved)
- Rotation towards plus side flexion towards (figure 2).
- Extension. (Extension plus rotation or side flexion towards - rarely used)
- Retraction.

- Retraction plus low cervical extension (figure 3).
- Other movements and combinations of in detail if necessary (in good clinical reasoning it is necessary to investigate in terms of pathological sources as well as in terms of the extent of the sensitivity - clinicians need to know all the tissues and movements that are sensitised in order to appreciate the extent of the impairment and to be able to plot its response to management/treatment inputs over time).

• Patient supine:

- Cervical traction (classically relieving) often done when patient first lies down - see below.
- An appropriate ULTT. In compression type nerve roots ULTTs surprisingly show only a moderately enhanced sensitivity response when compared to the contralateral side. Always check it and check the effects of sustaining it just like the other tests. Note the 'active' neurodynamic test described above.
- Anterior palpation over the exiting nerve root.

In my experience the above tests are all that are necessary. However, if you are still suspicious and wish to examine more fully the test progression can be taken further:-

• Sitting:

- Cervical compression in neutral.
- In rotation towards, plus side flexion towards add cervical compression. Or, Extension plus rotation towards add compression - Spurling's test. Both are similar to the low cervical quadrant position described by Maitland (1986) but with the addition of compression.

The addition of compression in this test should be performed with great caution and in my experience is unnecessary and

certainly not worth risking in the great majority of acute presentations. It is not at all comfortable for normal necks and if there is any degree of degeneration/underlying sensitivity even the gentle application of this test may actually precipitate a nerve root condition, or strongly aggravate and extend the time to resolution of a relatively minor one.

• Lying:

- Many combined tests can be engineered: e.g. neural tension tests with neural compression. For example, patient supine in side flexion towards, add ULTT. Unilateral antero-posterior palpation in side flexion towards. Experience dictates a very careful approach to prone palpation and the prone position, in acute and reactive, or potentially reactive, cervical nerve roots. The extension it induces may actually provoke radicular symptoms in the arm with neurological deficit.

Usually relieving tests:

Both the following tests have high specificity (see above) and are far less threatening to the nerve root than Spurling's test.

- Manual cervical traction in supine. I suggest great care with this test in very acute, highly reactive and early cervical nerve root disorders. Cervical traction, even very gently applied may be relieving at first but intensely provocative a short while afterwards. Care also with assuming that just because a few moments of manual traction are dramatically relieving, that traction treatment will be helpful. While it often is useful, there are many who are made significantly worse by it - especially if the pull is into extension. Again, relief of distal 'radicular' symptoms by traction provides the strength to this test.

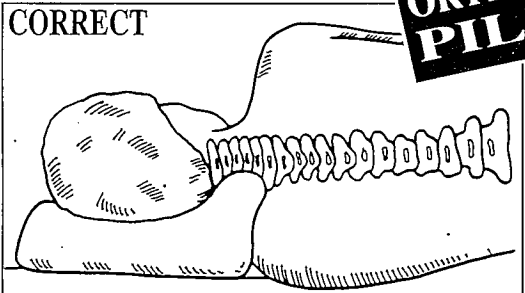
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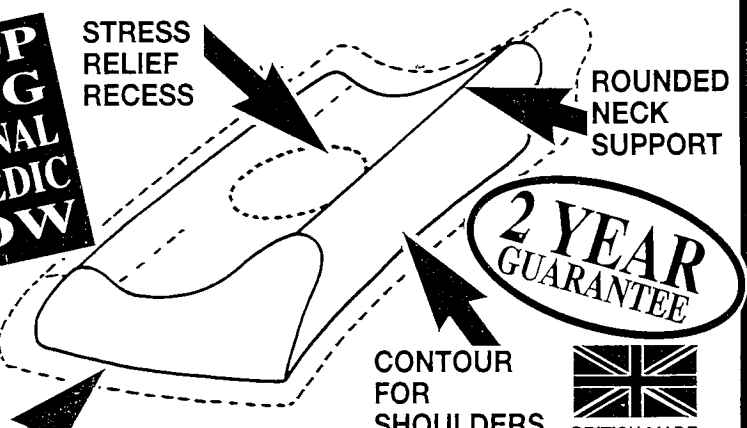


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- **Shoulder abduction test** - the patient simply places the hand of his affected arm on top of his head. It is often useful to explore the effect of isolated shoulder elevation and depression on symptoms (see 'active ULTT' above). For instance, if pain is relieved by arm overhead one would be interested to see if active scapula elevation and depression influence symptoms too. A major focus of the first encounter with the severe nerve root disorder is to find as many options for relief as possible - especially with regards to help with sleeping.

Nerve root demonstrating 'elongation' sensitivity: One or several of these tests may alter symptoms:

The examining sequence can be much as that above but with emphasis on seeking responses that elongate the root tissues, i.e. movements away rather than towards. The following tests are of particular interest:

- Shoulder abduction test
- Active ULTT
- Cervical movements away.
- Combining movements can often be helpful. For example, rotation away plus side flexion away, or scapular depression with arm by side plus rotation or side flexion away.

- **Neural tension tests.** There is generally no great need to opt for any particular one of the four standard ULTT tests as they do not specifically tension any particular low cervical nerve root with precision. Thus, even a clear-cut C8 nerve root distribution into the medial side of the hand which tends to make clinicians think 'ulnar nerve therefore ULTT3' can still be easily provoked by standard ULTT's biased to the median or radial nerves. A simple and quick passive way of examining the sensitivity of the low cervical nerve roots that might be called the 'upper limb SLR' is: (described for right sided problem) patient supine, neck with or without a pillow (best with to avoid extension); stand to the right of the patient and face patient, slide your right hand under their right scapula and gently bring the scapula into depression; the left hand all the while holds the extended arm at the elbow; now passively abduct the arm - if necessary the patient adds active wrist and finger extension to further sensitise the test. This last component is the equivalent of ankle dorsiflexion in the SLR. The test can easily be repeated after placing the neck passively in side flexion away (towards the left with the right sided problem). This is how I have taught all my local GP's who are interested to perform the test. I know it sounds patronising, but does take physiotherapists many hours of practice to become proficient at the ULTTs - they are complex and difficult tests to perform well. The simpler tests become, the more likely they are to show widespread validity.

A few notes on neurological testing:

- A neurological examination at every visit is essential.
- There are many therapists who would argue that reflex testing/neurological testing is unnecessary when symptoms are proximal only. I would strongly recommend that reflexes and muscle power are routinely performed in all presentations when first seen. For example, loss of triceps or biceps power and/or reflex in a patient presenting with two days of local medial border of scapula pain and a referring diagnosis of local muscle sprain. It is not uncommon to find normal biceps or triceps reflexes but marked loss of power, if it is feasible, therefore, always try to test both.

Lack of reflexes is a common finding in the older patient, especially reflexes relating to roots that exit from segments notorious for degenerative changes. An important point too is that pathological changes do not have to hurt. Histological studies of humans at autopsy show that 50 percent of those who have no record of ever complaining of symptoms have connective tissue changes and nerve fibre changes in neural tissue, particularly in vulnerable places (Neary *et al*, 1975).

If the patient is in considerable pain and/or anxious about overstraining causing further injury, static testing for muscle power may be impossible to adequately examine. Pain/anxiety can dominate the presentation to such an extent that conclusions with regard to muscle power may be quite difficult. Check for scapula 'winging' which may occur with C6 or C7 nerve roots (serratus anterior is innervated by the long thoracic nerve whose derivations are mainly C6,7 roots). Check for muscle atrophy, for example, observe the suprascapular and infrascapular (supraspinatus and infraspinatus), and deltoid areas for C5-6; the posterior arm (triceps) for C7; the thenar eminence for C8 and between the thumb and index finger for T1 (first dorsal interosseous) (Ellenberg *et al*, 1994). Bear in mind that obvious muscle wasting in an acute nerve root pain of only several days, may not relate to the current episode (although it sometimes does). The wasting may be the result of a well established and ongoing 'silent' neuropathy that has only just become symptomatic, or may be an indication of significant root compression or quite serious pathology. Take into consideration the age of the patient, the degree of likely degeneration (stenosis) and whether they have had any previous neck injuries or nerve root problems that could account for the wasting.

While pain distribution and pain behaviour give good clues as to the type of pain mechanism operating (Gifford, 1997b) they rarely give accurate clues as to the exact level of root involvement. Since there is apparently far less overlap in myotomes than dermatomes, muscle weakness, if it is present, rather than sensory change or loss, is likely to be the most reliable guide we have as to the segmental origin of a root disorder. Interpretation of muscle testing findings must of course take into account the patient's willingness to produce a full and firm contraction.

A final comment:

The recent CSAG report for low back pain (CSAG, 1994 p54-55) presents a diagnostic triage that divides back related problems up into three: 'simple backache', 'nerve root pain' and 'possible serious pathology'. It recommends a different therapeutic approach for each. This is commendable, but further scrutiny reveals what I believe to be quite an inadequate and simplistic presentation of the clinical features of the lumbar nerve root condition. My prime concern is that it represents only the classic end of the clinical spectrum and if adhered to, many very common atypical presentations will be incorrectly categorised. This can mean inappropriate management and much misunderstanding for the patient whose condition is often very resistant or worsened by standard treatment approaches. It seems highly likely that similarly inadequate diagnostic 'features' will be applied to cervical root disorders whose presentations, I believe, can be even more difficult to fathom than those relating to many lumbar nerve roots.

(Continued on page 19)

In Touch, Winter 1998 No. 85

Access to Health Records Act 1990

Can a solicitor demand a copy of a self employed private practitioner's notes under the above act?

This area has long been a problematical one for members. There is an increasing trend for solicitors to ask for notes to be sent to orthopaedic surgeons in order that the surgeon in question can compile a medical report. This practice has grown up as a matter of custom and goodwill without any sound legal basis. The purpose of the physiotherapist's notes is to facilitate the care, treatment and support of the patient and from a practical view they are an integral part of care and are a tool available to the practitioner.

There is no doubt that the notes remain the property of the physiotherapist concerned who is not bound to automatically comply with any request for them offering a report instead, produced for the appropriate fee. Indeed this avenue may well be preferred once the solicitor understands that the physiotherapist may not feel able to "support" any report produced by a third party from an interpretation of the physiotherapist's clinical records.

To return to the initial question, OCPPP's solicitors advise that they would argue that member's records fall under the following sections of the act as detailed below and as such access is able to be demanded.

Physiotherapists are specifically referred to in the Act as "health professionals"

Section 1 "Health Record" and related expressions.

- (1) In this Act "health record" means a record which-
(b) has been made by or on behalf of a health professional

in connection with the care of that individual.

(2) In this Act "holder" in relation to a health record, means- (C) in any other case, the health professional by whom or on whose behalf the record is held.

(3) In this Act "patient", in relation to a health record, means the individual in connection with whose care the record has been made.

Our solicitors do not believe that a member would be able to argue that rights of access may be partially excluded. Furthermore, there is a fee that can be charged but not one exceeding the maximum prescribed under section 21 of the Data Protection Act 1984. (£10 Jan 1998) A fee can be charged for copying the record or extract but it must not exceed the cost of actual copying together with the cost of postage.

As stated above, once a solicitor understands that the physiotherapist may not feel able to "support" any report produced by a third party from an interpretation of the physiotherapist's clinical records, they may well prefer the safety provided by the production of a report by the physiotherapist.

The medico-legal aspects of physiotherapy is of growing concern and OCPPP are working closely with the CSP and the Medico Legal special interest group to formulate and provide guidance for members.

Your attention is also drawn to the CSP Information Paper PA2 "Access to Health Records". This is available directly from the CSP or OCPPP.

Please send an A4 stamped addressed envelope with your request.

(Continued from page 16)

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