The Olive Sands Memorial Lecture 1993

Examining and Treating Signs of Neural Tension

This lecture is given annually in memory of Olive Sands who was not only a Founder Member and Founder Secretary of the OCPPP but was a tireless worker on behalf of the profession as a whole.

Introduction

I would like to start by making a few comments and sharing a few thoughts.

1. We are specifically looking at upper limb presentations and the relationship of nervous system abnormalities to them.

To a great extent the system and skills of neural tension testing in the upper limb has evolved from one branch of medicine: **Physiotherapy**, and in particular manual therapists who have Maitland backgrounds. It seems that frustration with a pure 'signs and symptom' approach has led to greater questioning of underlying causes of the things we see.... What is the underlying pathophysiology and pathomechanics of a condition?

It is the sifting through of the scientific literature in order to find explanations for our clinical findings which to me is the only way forward in our branch of this profession. 'Techniques', 'schools' and 'approaches' have perhaps been done to death.

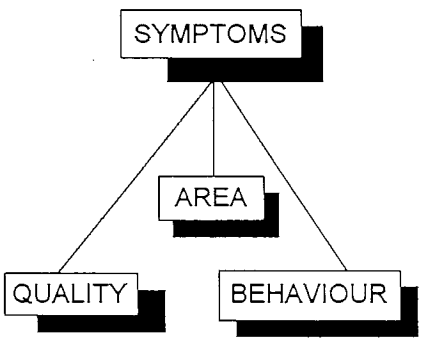
It should mean that we become far better at prognosis, at understanding the time courses of the diseases and injuries we see and that we are able to accurately predict the effectiveness of management and treatment. I believe that only a very small percentage of patients require pure manual techniques and that the term manipulative therapist badly expresses the true nature of specialists who treat 'out patient' type problems.

2. The courses currently being run by a few of us interested in nerves seem to appeal to a diverse group of physiotherapists all of whom happily fit it into their concepts. This includes the neurological speciality. The key thing is that what we teach is based on work written up in the literature and helps explain many of the common and not so common conditions seen.

3. The human body has only a limited number of responses to injury and disease. It stands to reason that easy patterns of symptom presentations should be apparent in every patient; however apparently awkward or bizarre.

4. If we all understood and recognised underlying abnormal processes there would be a more uniform approach to treatment and management.

Symptoms can be evaluated in three basic ways:



It is difficult to discuss each in total isolation as they are so intimately related. Some overlap in each section has to be excused.

Quality of Symptoms

Quality can be considered in terms of Neuropathic Pain/Symptoms and Nerve End Pain/Symptoms: see fig. 1.

Nerve End Pain/Symptoms

This is a big topic, but specifically primary afferent nociceptor nerve ends (PANs), or terminals, in the periphery, respond to noxious non-damaging or noxious damaging stimuli and signal pain to the higher centres. There are only three categories of stimuli which activate this impulse transmission: via Chemical, Temperature or Mechanical means and no other: see fig. 2.

If the noxious stimulus is non damaging, the pain literature refers to this as 'physiological pain'. Its role is to inform of potential danger.

'Pathological pain', by contrast, arises following tissue or nerve damage: see fig. 3.

In its acute form it is associated with tissue damage and inflammation. In this instance the pain is useful in that it helps protect tissues while the process of recovery proceeds.

In its chronic form it is commonly associated with nerve damage, ie. neuropathic pain. But please note ... chronic inflammation and inflammatory disease, and, what I call 'tissue repair pain' where inflammation has subsided, repair processes are in progress and tissue strengthening is taking

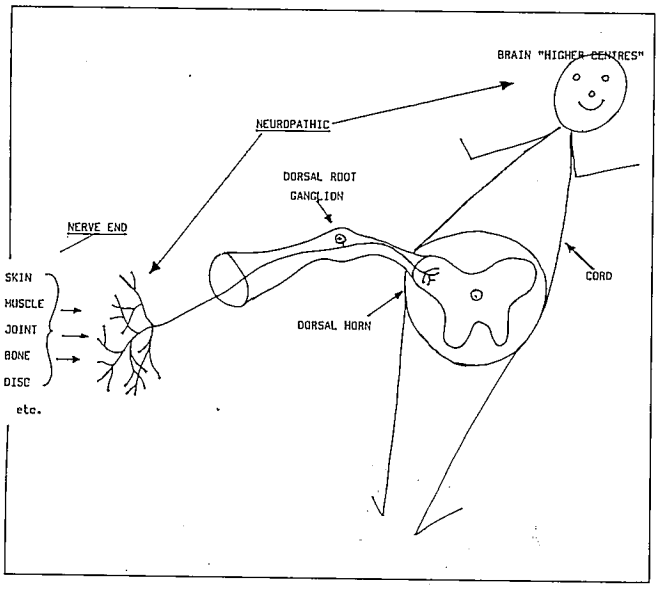


Fig: 1.

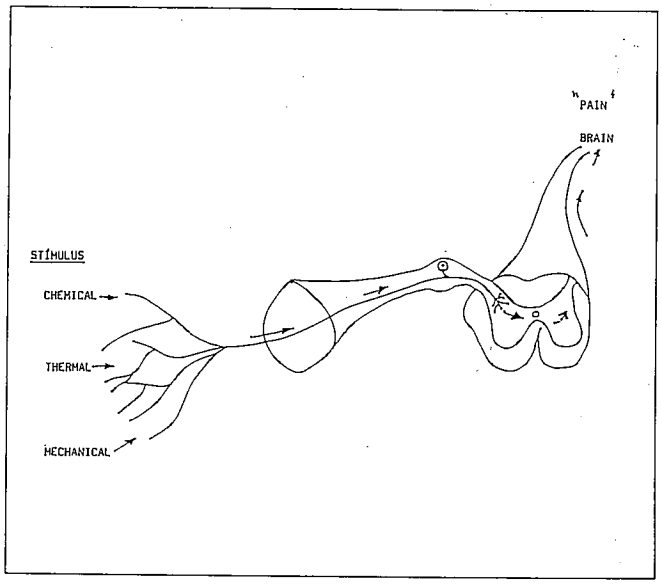


Fig: 2.

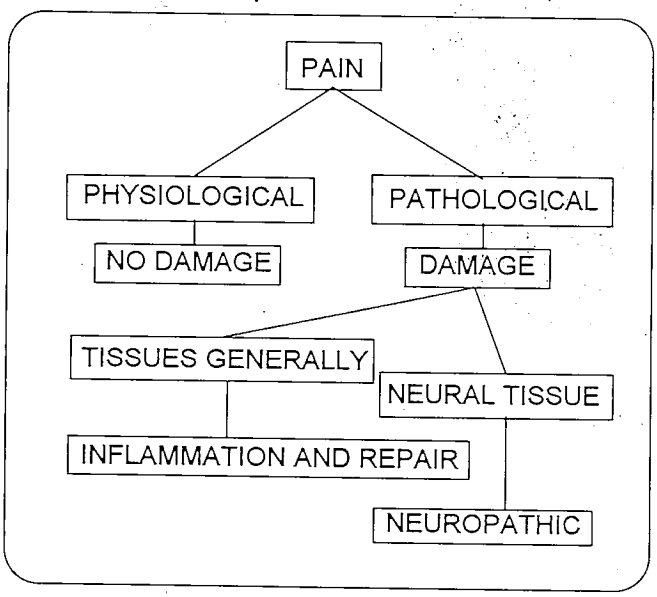
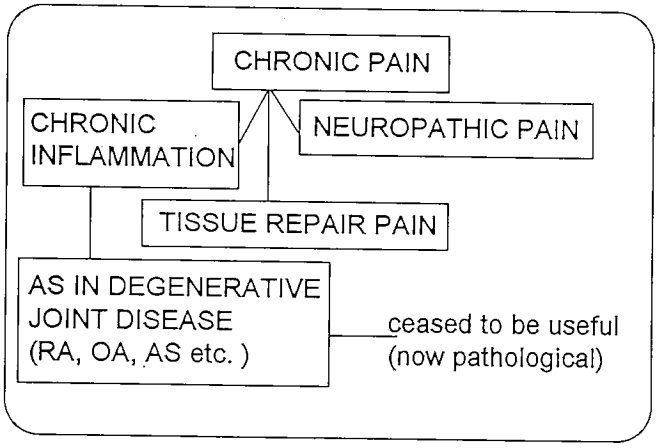


Fig: 3.

The common features of inflammatory and repair pains will not be discussed here, but all clinicians should be able to recognise the quality, behaviour and history of inflammatory pain (this is another big topic). They should also be able to differentiate whether the inflammatory pain/nature is 'useful', and therefore should be respected when examining and treating, or 'pathological' which may require a different approach.

Inflammatory pain is a 'nerve end pain' and thus originates from nerve terminals. Here, pain may be caused by chemical activation and is perhaps the ongoing background ache that is present in most acute and subacute problems. The typical sharp, stabbing or knife like pain produced on movement in these cases is most likely a mechanical stimulation of sensitised primary afferents in the inflamed area (primary hyperalgesia) and/or immediately around it (secondary hyperalgesia): see fig: 5.

place ... these tissues still need protection and require informative pain. Research has shown that collagenous tissues (ligaments and tendons for example) take up to one year to gain full strength (Nordin & Frankel 1980): see fig: 4.



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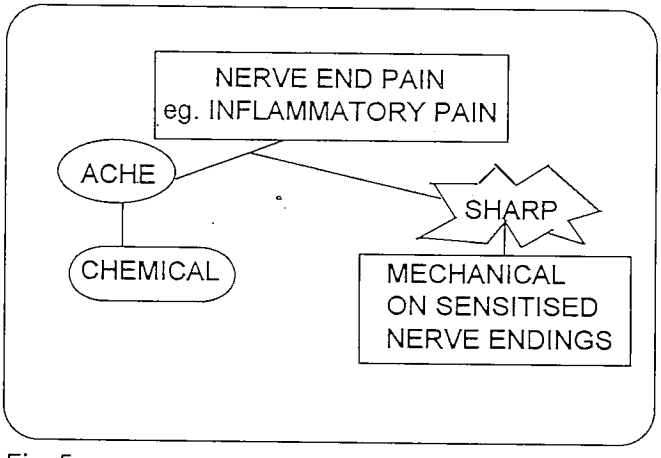

Fig: 5.

Remember, tissue tenderness to palpation may be simple secondary hyperalgesia which is not an indication that the tissues are necessarily culpable, merely that they are in an area adjacent to the primary source of pain. When tissues are in an acute hyperalgesic state it makes it almost impossible to

accurately pin-point by manual differential diagnosis a specific tissue as culpable. This is perhaps the 'all tests are positive' scenario. The end result is heaps of false positives and the most likely tissue responsible is gleaned from the history and subjective assessment generally.

Neuropathic pain by definition arises from the nervous system: see fig: 6.

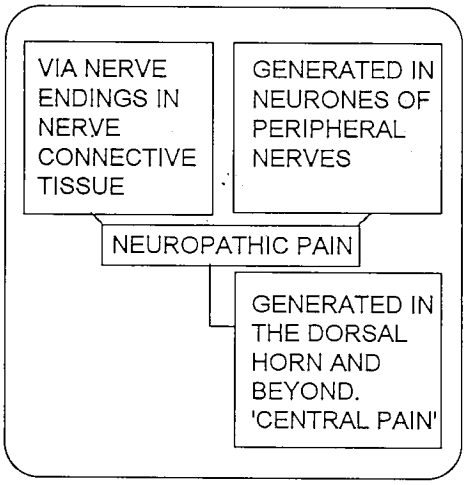

Fig: 6.

It should be realised however that the nervous system itself has its own connective tissue and that these, like tendons and ligaments, have their own nerve supply. We should therefore consider one component of neuropathic pain as being perhaps 'nerve end' type pain (Asbury and Fields used the term 'nerve trunk pain' - Asbury & Fields 1984).

The peripheral nerves have an intrinsic nerve supply called the nervi-nervorum (Hromada 1963). Consider for a moment a situation where the epineurium has become irritated. As in any collagenous type tissue one would expect dull aching, which may be continuous or intermittent depending on the severity of the inflammation; sharp pain on movement, particularly with movements of the surrounding interfacing tissue which can bring forces to bear (eg. contraction of supinator muscle over the posterior interosseous branch of the radial nerve) or with movements of the nervous system itself, ie. tension tests and movements which stress the epineurium. One would also expect the pain quality to be more familiar to the patient. David Butler, with the agreement of Sir Sydney Sunderland (world authority on nerve injury and repair) has suggested that lines of pain, particularly those along nerve trunks, indicates nerve connective tissue pain in its referral.

Within the spinal canal much of the connective tissues of the neuraxis are innervated by the sinu-vertebral nerve. The quality of this pain will be discussed below.

Pain generated from abnormalities in nerve conducting tissues is considered to be truly **neuropathic**: see fig: 6. It is the recognition of this component of pain which is commonly missed and perhaps the major reason for misguided management in many stubborn disorders we commonly see.

This is a vast topic but I will give two examples of how the nervous system can 'go wrong' following injury and the likely clinical scenario.

1. In the first case, injuring, or pathological processes that are enough to cause neurone injury or cell death in peripheral nerve can set up abnormal neural processing mechanisms that give rise to *false* sensations, the *distortion* of sensation (paraesthesias?) and unnecessary? *pain*. Injuries that actually cause Wallerian degeneration are far more common than we are normally led to think. You may think of common disorders such as carpal tunnel, brachial neuralgia or nerve root syndromes, thoracic outlet syndromes, even some chronic tennis elbows which have perhaps minor 'neural' symptoms, as well as the entrapment neuropathies that occur at the anatomically and biomechanically vulnerable sites along the nerve bed (eg. ulnar nerve in Guyon's Canal or in the Cubital Tunnel; median nerve in the proximal forearm; radial nerve in the Arcade of Frohse or any cutaneous nerve where it pierces fascia).

Experiments on animals have shown that injured axons, at the point of growth, or where there are local patches of demyelination along the axon, and often the dorsal root ganglion (DRG) cells as well, become sources of abnormal, ectopic discharge: see fig: 7. The brain interprets the abnormal impulses as originating in the tissues originally served by the intact nerve. One explanation is that substances produced in the DRG and normally transported to the nerve terminals for normal nerve activity actually get dammed up at the injury site in abnormal concentrations, which leads to these areas becoming hyperexcitable.

These microscopic little swellings may be massed within a small region of nerve, ie. a nerve end neuroma or microneuroma, or, they may be disseminated throughout a nerve or its target tissues. In the literature these areas of nerve hyperexcitability are termed 'ectopic neural pacemaker sites or nodules' and are highly responsive to mild mechanical input. As far as we are concerned the nerve becomes far more sensitive to palpation and gentle tapping, (Tinels Test has been shown to be highly successful in isolating the sites of nerve injury, Saal et al 1988) and, of course, to neural tension tests.

Additionally these sites are highly sensitive to ischemia and anoxia, inflammatory mediators and cold. Chemicals released by sympathetic efferents are also irritative, see fig: 8.

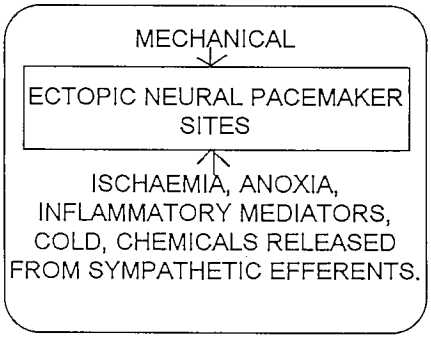

Fig: 8.

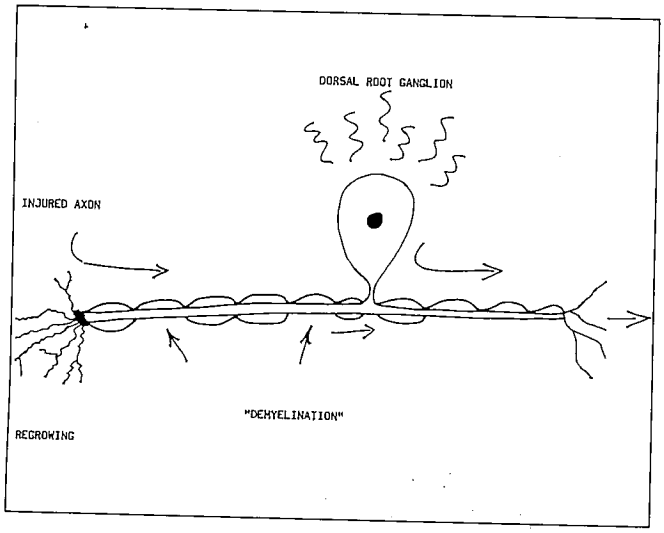

Fig: 7.

Once an ectopic pacemaker has been provoked, by for example a brief mechanical stimulus, it can continue firing for seconds, minutes or even hours, often with massive bursts.

Can you envisage the patient? see fig: 9.

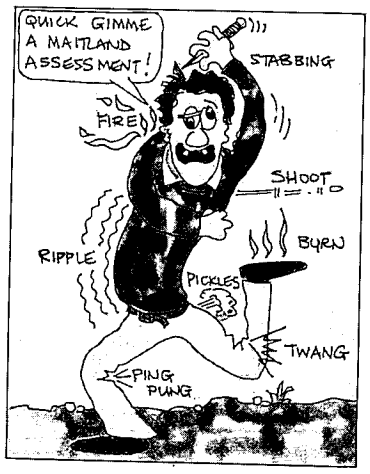

Fig: 9.

Yes the one who normally gets labelled as a complete nut case .... Chronic pain, comes and goes for no apparent reason, pain is often horrendous, builds up and lasts for a long time with only minor provocation; has a bizarre quality to it (shoots, shocks, twangs, ripples, strings pulling, pickled, on fire, burning, stabbing etc.); is often exacerbated by changes in weather especially cold, or changes in mood/emotion/ stress (effect of sympathetic discharge?); has odd distribution, or non dermatomal distributions of paraesthesia and anaesthesia, may have skin hypersensitivity and generally is classified as a highly 'irritable' disorder by Maitland physiotherapy assessment. Gentle grade II p/as 3 times per week for 6 weeks can achieve very little here and is probably costing the Health Service and the private patient dearly? That is rather provocative I know, but aren't these the patients we are so often afraid of? and lets be honest, do not fully understand. Why do some patients end up in this sort of mess after only relatively minor injury while others suffer ghastly injury and recover without any chronic repercussions? One interesting avenue of recent research indicates that there may be an inherited factor, perhaps even a single recessive gene, that predisposes the individual to the development of neuropathic pain (Devor & Raber 1990).

2. I would now like to consider aspects of so called 'central' neuropathic pain: see fig: 6. It is now known that high intensity afferent barrage (via primary afferent nociceptors) can cause long lasting changes in the neural chemistry and circuitry of the dorsal horn: see fig: 10. Examples here might be whiplash, crush injury or following an operation. A common example in the pain literature is amputation. It is also conceivable that *any* continuous barrage of pain from nerve ends may have similar effects. Chronic inflammatory pain in Rheumatoid Arthritis or even the pain associated with the postural stresses of keyboarders are possible examples. The end result of this is that, due to these neuro-chemical changes in the dorsal horn, pain can be generated spontaneously in the dorsal horn. Further, normally non painful input from the periphery, via for instance A Beta fibres (eg. information about joint movement), can trigger dorsal horn cells to relay impulse trains to higher centres that are again interpreted as pain. Can you see that again the brain may interpret problems as seemingly awful when in fact the reality of the problem is not in simple tissue pathology but in the generation and supply of abnormal information *from within the nervous system.*

The features and quality of the symptoms are similar to those discussed above with the exception that *nerve palpation* and *neural tension tests* may show little or no abnormality. Perhaps the key **quality** of neurogenic pain is that it is often described as 'burning' and there may be spontaneous 'shoots' or 'stabs' as well as the more familiar pain qualities like aching and throbbing. Quality of pain needs to be considered alongside its behaviour. Inflammatory pain generally behaves in a far more relational way to posture and movement.

It is pertinent to emphasise here that most chronic problems we see have components of all the pathological pain processes mentioned, ie. centrally and peripherally generated neuropathic pain as well as nerve end pain, such as the inflammatory or repair pain discussed. Hence the only moderate success of narrow approaches to these problems. We should all be united by the recent advances in the understanding of pain neurophysiology. We need to understand and be able to recognise underlying abnormal pathological processes far better.

The desire to precisely identify specific tissues at fault while laudable is often impossible.

Most chronic pain patients require skilled manual treatment, skilled use of appropriate pain management modalities and skilled use of exercise,

and here it must be problem specific as well as improving general fitness and physical and mental tolerance to the problem. The successes of various 'school for bravery' type approaches in this country, the States and in Australia are well documented.

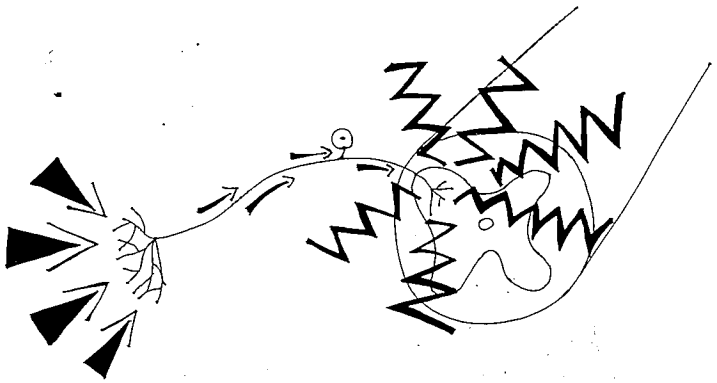

Fig: 10.

Area of Symptoms

So many of us are obsessed with getting the area of pain precisely. It is often necessary, but it is often frustratingly difficult for the patient, his pain is just not of a precise nature. Simply watch how a patient uses his hands to indicate an area of pain. If it is a precise area pain he uses the index finger to indicate the spot or area, if it is vague he vaguely waves his hand around or may even simply say 'its in my hand and shoulder' rather than indicate at all. Please don't get cross with him he is only relating the problems his nervous system has with interpreting the nature of his problem. If a patient has a precise pain, he will surely have his sleeve rolled up and be showing you the spot in the first 30 seconds of the assessment.

I would now like to cover some aspects of 'areas' of pain that we commonly see and briefly discuss the underlying mechanisms that *may* be responsible.

Axoplasmic transport is the specialised to and fro movement of essential cellular materials and molecules between the neurone cell body and all aspects of the neurone including the dendritic terminals (which may be several feet in a sciatic nerve neurone).

Normal axoplasmic flow is essential to the maintenance of a healthy neurone, its terminals and the tissues it innervates.

It is now becoming apparent from recent literature that minor compression of a nerve, or simple depletion of circulation to a nerve will cause the slowing and even halting of this flow. For instance it is known that pressures less than those measured in mild carpal tunnel syndrome easily stop the flow and that recovery of flow does not take place immediately. Dahlin & McLean (1986) showed that 2 hours compression with a pressure of 400mm Hg took **7 days to recover.**

These findings have strong implications as far as prognosis and the outcome of treatment are concerned. For instance, a patient who has a chronic upper limb nerve root problem (6 months plus, and who has sensory loss, paraesthesia, reflex diminution, muscle weakness, as well as pain etc.), may require daily home traction for several months before any change in symptoms is reported. The theory behind the approach is perhaps simple regular release of pressure on the nerve allowing some of the surviving affected neurones to recover a better axoplasmic flow. Can you see that the instantaneous assessment of signs and symptoms will show little to positively guide treatment. TIME is perhaps the big factor.

Minor nerve compressions are going on all the time .... you are all sitting, probably uncomfortably, on your sciatic nerves, firstly making them ischaemic and therefore painful, and secondly causing a slowing of axoplasmic flow and thereby contributing to the insidious decline in health of the nerve and its target tissues!!

Intermittent slowing of flow is a part of daily life, but can you see the detrimental effect of prolonged day in day out sitting as in taxi and lorry drivers? More about posture and the upper limb shortly ... we have wandered from the theme of 'area' somewhat.

You are all probably familiar with the patient who comes with a carpal tunnel syndrome and has neck problems at the same time, or who has developed carpal tunnel syndrome after the start of a neck pain. This phenomenon has been frequently described in the literature and is termed the 'Double Crush'. This refers to the fact (see Mackinnon 1992 for excellent summary) that a minor nerve compression in one part of a nerve makes it more susceptible to minor compressions further along its length. The mechanism is probably via the detrimental effects of slowed axoplasmic flow on nerve health. The literature uses the term the 'sick' neurone. One envisages the situation where a patient has minor spondylotic changes, one day does some sort of neck stressing movement and precipitates a local pain. The outcome of this is perhaps a slight increased pressure on the already slightly compromised lower cervical nerve roots (inflammatory swelling?) which leads to a modest axoplasmic flow sufficient to allow a long standing tight flexor retinaculum in the wrist to set up a symptomatic reaction in the nerve here ....

The terms 'reversed double crush' and 'multiple crush' are also used in the literature. We must think of this mechanism when the patient relates a story whereby symptoms start in one area and then jump or spread unexplainably to areas proximal or distal. Do not just consider movements of symptoms in the limbs, so often, for instance whiplash patients describe symptoms starting locally in the neck, then spreading to the arm as well as the mid thoracic region, the lumbar region and even consistent areas in the lower limb such as the back of the knee. The patient often

indicates the area as a 'clump' of pain with his whole hand around the area.

Do not be frightened to ask about all areas of pain even if a patient has only been sent for treatment to his tennis elbow! Chronic problems such as this often have patterns consistent with the double/multiple crush mechanism. If you don't ask, you miss the pattern!

I mentioned the sinu vertebral nerve's role in nerve end pain with respect to the innervated connective tissues within the spinal canal. Cyriax once said that if the symptom area does not make sense with respect to classic dermatome/segmental referral zones, then suspect the dura. From my reading, one sinu vertebral nerve can wander up and down the spinal canal up to eight segments. It is not surprising that any structure in its innervation field which is irritated demonstrates an implausible referral of pain/ symptoms. At this stage of knowledge we should go beyond merely suspecting the dura, but suspecting all the structures that this nerve innervates.

Behaviour of Symptoms

I hope you will now agree that in order to fully understand the behaviour of symptoms we must have prior knowledge of the capricious behaviour of neuropathic pain.

This section really loads the weighting to a few aspects of pain that is of the 'nerve end' type discussed above.

It must be emphasised however that some neuropathic pains that have their origins in pathology of neurones in the periphery, where hypersensitive ectopic neural pacemaker sites exist, may be highly responsive to any movement, pressure or tension that is brought to bear. These patients stand out a mile and it is extremely easy to provoke their pain and cause it to spread, especially with tension tests. The key thing here is great care.

So called 'mechanical' pain is easy to identify, ? pain on ? pain off with movement. It often likes movement and hence gets less with it, at least there and then in your treatment cubicle. It does provoke it later quite often though? I think you will agree, at least in some cases.

Moderately inflamed tissues can behave in this 'mechanical' way. Just think of a mouth ulcer, it doesn't hurt all the time unless its really nasty, but it does require some sort of mechanical force to hurt in most instances. Mechanically irritate it too much and it gives you stick later. It may be an unfair analogy but it fits clinically.

Nerve end pain requires mechanical thinking. As far as the nervous system is concerned we should think in terms of two things when considering movements that the patient tells us that aggravate the pain.

1. What is happening to the tissues around the nerve?

A patient has had pain down his arm for 7 weeks, he has tingling in the thumb and the first finger, he can't sleep because of the pain in his shoulder blade and it all hurts when he looks up or turns to the side of pain. It is all relieved when he sticks his arm up in the air or flexes his neck.

This fellow *probably* has a compromised intervertebral foramen and he is telling you that movements which make its dimensions smaller squash the highly irritable and inflamed tissues in the confined space. It is highly likely that he has no limitation, or only very slight limitation, of neural tension tests and that symptoms are not provoked in the way one would expect when compared with the lower limb equivalent (acute sciatica with limited SLR). You may find this lack of neural tension component hard to accept, but cervical nerve roots are highly protected against longitudinal forces via their ligamentous attachments in the gutters of the transverse processes. It is I agree, hard to explain.

The same thinking process must be applied in the periphery too. The pain on the front of my wrist gets far worse when I bend my elbow - work that one out!

2. What is happening to the tension, movement and length of the most likely nerve/ plexus involved?

I hope this audience is aware that for instance, the median nerve in the upper arm can move anything up to 2cms. with movements of the arm, and that all nerves must have biomechanics in order to adapt to changing body movement.

The answer to the above question is obvious to you folk who have already been revising your nerve anatomy and playing around with the tension tests in an enlightened way. Consider the movements and tensioning of the major nerves as a whole and you see the end positions of many provoking movements as end range nerve too.

What about this. A patient complains that pins and needles over the back of her thumb and a low grade annoying ache are heightened when she tight-reins her new horse: see fig: 11. Consider the area of pain, innervation field of the superficial radial nerve, the course of the nerve, which moves when the wrist ulnar deviates, and you have big clues as to the best way to test the problem and the likely tissue responsible.

The influence of static posture or positioning on the nervous system is particularly interesting. The most important consideration is perhaps the effect of circulation on a nerve. Constant positions produce

constant pressures and constantly deprive tissues of vital circulation. The nervous system requires 20% of the available oxygen in the circulating blood yet consists only 2% of body mass. Circulation is required for impulse transmission and to power axoplasmic transport mechanisms. Short term loss of circulation to a nerve causes slowing of these processes. Longer term loss such as that which may arise from prolonged compression (as in carpal tunnel syndrome), may lead to the nerve, or discrete sections of nerve, becoming oedematous, swollen and eventually fibrotic with neurone cell degeneration and death. This process may only affect one or two fascicles and leave many normal ones. It is worth commenting here that a nerve like this may have normal electrophysiological tests even though a 'neuropathy' is patently present. Nerve conduction tests are not 'fascicle specific'. I think this situation is very common with the types of disorders physiotherapists encounter.

Fig: 11.

Can you see that a nerve that has become fibrotic will be less elastic and therefore less adaptable to longitudinal stress, the tension test will be limited and the nerve more vulnerable to injury further along its course. This is perhaps another mechanism which could account for symptoms jumping from one area to another.

Quite astounding changes in pressure occur in nerves with changes of position. For instance, the pressure within the ulnar nerve in the cubital tunnel at the elbow doubles when the limb is moved from a position of extension to one of elbow flexion with wrist extension. If this elbow and wrist position is maintained and then glenohumeral elevation added the pressure has been shown to increase on average nearly 7 fold (Pechan & Julius 1975). The prolonged static postures of keyboards, computer operators and taxi and lorry drivers cannot be nerve healthy, let alone anything else. The functional analysis of patients persistent postures with a background and knowledge of some basic peripheral nerve anatomy may help greatly in explaining their symptom picture.

WE should also consider the effect of longitudinal stress on the vascular supply to nerve. Firstly, it is known that if a nerve is elongated by as little as 8% its blood supply will be severely diminished and by 15% elongation the supply stops. Pressure within a nerve is increased by tension and elongation. Consider the elongation of the median nerve. From a position of arm flexion and wrist flexion to a position of full arm stretch with wrist and fingers extended the nerve bed of the median nerve gets 20% longer. The median nerve has to adapt to this by initially unfolding and then progressively greater elongation. The end range position of ULTT1 is just this position and symptoms are easily provoked in normal arms, the reason most likely being the sudden anoxia precipitated by the force of elongation cutting off the blood supply.

We must go back to the slumped position of the keyboard operator. Considerable longitudinal forces on the brachial plexus are caused by the scapular depression, the poor old ulnar nerve takes the brunt of the elongation as well as focal increases in pressure at the elbow and the wrist. The moral of this story is make sure you have a good range of all ULTTs before embarking on a career in an office and probably more importantly always be restless rather than static. Static postures, no matter how good, always put some adverse pressure or force on some structure.

The pain and dysesthesias of neuropathies are classically worse at night. One only has to recall the carpal tunnel syndrome patient who's paraesthesia wakens him and causes violent shaking of the arm to provide relief: see fig: 12. The nightly drop in blood pressure may provide one answer in that less pressure means less force driving required blood through already constricted/tethered or fibrotic nerve. It is apparently far commoner for neuropathies to occur in hypotensives.

Fig: 12

Before insisting on plenty of bed time aerobic exercise for your patients it is well worth spending time to analyse night time arm/body position/posture for reasons already mentioned. For instance, it is not uncommon to sleep on the side with the wrists in full flexion. The pressure in the carpal tunnel is dramatically increased when the wrist is taken from neutral to full flexion or extension. This is the basis for 'Phalen's' (flexion) and 'reverse Phalen's' test for median nerve compression in the carpal tunnel syndrome.

I would like to finish off in a similar way to when I started, by sharing a few thoughts, this time on treatment.

1. Before we can begin to manage/treat with any confidence we have to understand the nature of the underlying disorder. By this I mean the chemistry or the pathophysiology as well as the pathomechanics. We have to know more about common patterns of injury and disease and be able to pick them up better. We must understand the qualities and behaviour of symptoms that are neuropathic as they account for a large percentage of chronic problems. I think if a patient with vague arm pain went to 10 different practitioners he would more than likely get 10 different diagnoses. This is not meant to be an insult to us as it applies to the medical profession as a whole and other fringe and mainstream alternatives too. It is a major problem of orthopaedics, neurology and the pain speciality. I would like to go further and say that diagnoses like 'cervical spondylosis' or 'rotational subluxation' of C3 only indicate the state of the bony tissues on X-ray and say nothing about the pathophysiological processes responsible for symptoms. The X-ray certainly gives clues about weak spinal tissues and likely success in achieving gains in range, but nothing about the underlying cause of the pain. It may be simply mechanical or show a variety of states from the extreme of central neuropathic pathology to mere nerve end pain due to local inflammatory processes. One may be relatively easy to help, another extremely difficult.

2. In conditions where neural tension tests are positive, direct nervous system mobilising may be highly inappropriate. The upper limb tension tests produce symptoms that are difficult to analyse in normals, let alone in patients. A cautious reminder therefore is to be careful of false positives and the danger that hinges on diagnoses labelling one particular tissue or system at fault. Most problems have multiple components that need addressing. The biggest ones may be lifestyle, fitness and lack of movement in daily life.

3. Nervous system mobilising produces vast changes in pressures within the nerves addressed and therefore responses that are the result of changes in circulation must be considered. The dispersion of intraneural oedema is one example.

4. We must be affecting systems at the cellular/ sub-cellular or even chemical levels. It is known that axoplasm decreases its viscocity with movement. It is conceivable that restoring normal neural movement enhances the flow of axoplasm and thereby restores health to the neurone and the target tissues. It may take several weeks or months to produce changes with little objective evidence other than slow changes in symptom patterns to guide us. If we are to develop the field of manual therapy further we must look more closely at the response over time without the constraint of producing instantaneous objective changes based purely on ranges of movement.

5. You are probably wondering how on earth to approach the bizarre neuropathic presentation? Remember, there are descending inhibitory pathways that can modify the ongoing abnormal impulse traffic arriving at, or being generated in, the dorsal horn. We must in the first instance find physical ways into a presentation, ie. via the nerve ends and nerve trunks, and secondly, via the complexities of the cortex using whatever means. Physiotherapists are in the unique position to use the physical to help the mental. It may be the most powerful manipulating tool we have?

6. You may perhaps be upset by this final point but I feel that the greatest problem our profession has now is the lack of willing and able clinical teachers to filter the information and skills to a greater number of knowledge thirsty therapists.

This final point is also a plea for greater agreement through science, and the merging of the many different and often antagonistic 'schools of manipulation' and 'approaches' through this.

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Suggested further reading

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