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Treatment

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Manual Therapy in the 21st Centuty

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Manual therapy in its various forms has been described throughout recorded medical history. The Greek physician Hippocrates described spinal manipulation as far back as the 5th century BC. More recently, the popularity of manual therapy both with clinicians and patients has continued to grow throughout the 19th and 20th centuries in line with the spread and growth of the chiropractic, osteopathy and physiotherapy professions. Today much of the theoretical basis used to prescribe and administer manual therapy is still drawn from the original teachings and philosophies of its early pioneers.

However, while many manual therapy treatment approaches have changed little since their inception, pain science and our understanding of pain has advanced rapidly. So rapidly in fact that the last decade has seen more progress in the pain sciences than throughout the preceding history of medicine. With these advances it is widely recognised that for the first time in medical history we have started to assimilate a 'pain knowledge base' founded on good scientific evidence as opposed to the theories and quasi-empirical approaches that came before.

Responding to the emergence of this recent scientific evidence, some authors have started to review the construct of manual therapy, and its actions and efficacy (e.g. Wright 1995, Souvlis et al 2004, Zusman 2002 & 2004). Undoubtedly understanding this better will help us to decide if, or when, manual therapy is an appropriate treatment for pain. Evidence on which to base clinical decisions is welcome news for many clinicians and patients alike, particularly among the current and often endless jungle of therapies vying to 'treat and cure' those suffering with pain, disability and illness.

Manual therapy defined

For the purpose of this discussion, manual therapy will be largely considered in two of its most commonly prescribed forms, namely, joint mobilisation and joint manipulation. Direct reference to the many other forms of manual therapy such as positional release, craniosacral techniques, muscle energy etc will not be made. However, these manual techniques can easily be considered in a similar context as they are highly likely to influence the same pathways of peripheral and central processing as mobilisation and manipulation. Thus, their overall relevance via a more palatable rationale should become clearer along the way.

Maitland (1986) defined mobilisation as "passive movements performed in such a way that at all times they are within the control of the patient so that the patient can prevent the movement if they so choose". In contrast manipulation techniques have been defined as those techniques used to force a joint beyond its presumed physiologic barrier and up to its anatomical barrier (Haldeman & Hooper 1999). Thus, they are beyond the conscious control of the patient and include such techniques as chiropractic adjustments, high velocity low amplitude (HVLA), grade 5, long-lever and thrust techniques. These techniques are often accompanied by an audible clicking or cracking sound.

Mobilisation and manipulation theories

The proposed mode of action and claimed effects of joint mobilisation and manipulation ranges, in some manual therapy disciplines, from being highly specific, to others which portray them in an all-encompassing 'cure all' approach. A review of the current evidence base is helpful when considering how these proposed theoretical approaches stand alongside current scientific evidence.

Mobilisation techniques are described in terms of utilising physiological and accessory joint movements either separately or together. For the unfamiliar, physiological movements are defined as those a person can carry out actively, for example flexion, extension and abduction. Examples of accessory joint movements include the small degrees of slide, glide, spin and roll inherent within joints during normal movement. Individual accessory movements cannot be self-initiated but can be performed passively by someone else.

Maitland (1986 &1991), Edwards (1992), Mulligan (1999) and Grieve (1988) describe mobilisation techniques in terms of treating mechanically derived joint problems. Particular techniques are described as restoring the 'position and function' of the joint's intra-articular structures therefore improving joint range of motion and relieving mechanically mediated pain.

Maitland (1986) and Mulligan (1999) describe mobilisation techniques specific to manoeuvring a 'painful' torn and displaced meniscus in such joints as the knee or temporomandibular joint thus altering the position of the meniscus allowing greater pain free range of motion. In the absence of normal meniscal healing there is no evidence offered to suggest that any change or reduction in the position of menisci is reliably sustainable in the long-term and subsequent recurrence or persistent displacement are commonplace with these injuries (Norris 1993, Apley & Solomon 1994, Dandy & Edwards 1998).

The basic tenet of mobilisation techniques describes them in terms of eliminating mechanical causes of pain by restoring normal joint range of motion, therefore removing the underlying mechanical irritant and thus achieving the cessation of pain. Despite the fact that many patients present with pain that fits the diagnostic criteria described for the prescription of such techniques, treatments using this approach do not always eliminate or alleviate pain (Frost et al 2004, UK BEAM trial 2004). Non-responders to these and other types of manual therapy do often display temporary or sustainable clinical improvements in joint range of motion but may also report little or no significant improvement in their overall pain presentation (Gifford 2004). If the cause of pain in these circumstances is purely mechanical as has been postulated, then logically pain should resolve commensurate with restoration of joint range of motion. Clearly the absence of pain relief despite clinical improvements in joint range of motion highlights the multi-dimensional nature of pain and the strong indication that pain is never likely to be a pure 'mechanical' entity.

The intervertebral disc

The clinical diagnosis of prolapsed intervertebral discs (PIVD) has often been cited as a reason to attempt the manipulative reduction of prolapsed disc material (Cyriax 1974). However, the related literature does not contain any definitive or reliable evidence to support the claim that discs can be reduced by manipulation or mobilisation procedures. The improvements in diagnostic imaging over recent years has in fact resulted in accumulating evidence to the contrary, thus confirming that intervertebral discs do not revert back to any semblance of their original structure following manipulation (Cassidy & Kirkaldy-Willis 1985, Bourdillon & Day 1987, Chrisman et al 1964).

Further, Gifford (2002), has reviewed the disc healing potential, or the 'potential' of the injured disc to change, and argues that its structure is virtually inert. He concludes that all the biological processes of change within the disc are extremely slow (measured in years, not days or weeks) and that even minor tears of the outer annulus lead to disruptive and degenerative changes throughout, – not healing/recovery (importantly he urges us to remember that even a degenerate disc is still a very strong structure though).
Despite these findings and arguments, some authors have continued to produce anecdotal reports of PIVD reduction based on observational single case studies (Cox et al 1993, Hession & Donald 1993). It is true that the profile of the intervertebral disc is altered during manipulation by the torsional forces involved, but there is no evidence to suggest that these transient forces are capable of reducing a bulging annulus or sequestrated nuclear material (Matthews & Yates 1969).

There is compelling evidence that the annulus fibrosis of intervertebral discs can be injured by manipulation procedures. Farfan (1977) demonstrated annular failure with relatively small forces when applying 'manipulative like torsion' to intervertebral joints. Anatomically the annulus is vulnerable in positions where significant torque is produced. Annular failure can occur with forces as small as 113Nm, this magnitude of torque is easily attainable by manipulators, particularly in positions of axial rotation, which 'slackens' 50% of the obliquely oriented annular fibres (Bogduk 1997). In this position half of the annulus offers little circumferential support and the annulus is markedly more susceptible to injury from manipulation techniques that produce annular torsion. With this in mind it will be no surprise to learn that the majority of manipulations resulting in annular failure contain a rotational component (Grieve 1994).
The possible role of the disc presents a fascinating paradox here: that even though manipulation can relieve pain, the thing that is being manipulated may actually be injured in the process! This argument can apply to any physically forceful exercise or manual treatment procedure.

Asymmetry, mal-alignment and movement dysfunction

The concepts of asymmetry, mal-alignment and movement dysfunction are criteria that have frequently been used to form the basis of structural diagnosis and subsequent treatment by manipulation and mobilisation techniques. Many of the theoretical approaches currently in use originated from osteopathy and chiropractic, often to be adopted with little enquiry by physiotherapy practitioners (Watson & Kendall 2000).

Osteopathic and chiropractic approaches were founded on the concept of somatic dysfunction, (DiGiovanna & Schiowitz 1991, Greenman 1996, Lee 1999, Nathan 1999) this is considered to be due to an impaired or altered function of the bodily framework (somatic system). Osteopathy considers this impaired function to be due to changes in skeletal, arthroidal and myofascial structures including their related vascular, lymphatic and neural elements.

In osteopathic literature the mnemonic ART is often used to describe the three classical diagnostic criteria deemed to be present with so called 'somatic dysfunction. "A" represents asymmetry, "R" range of motion and "T" tissue texture abnormality. Manipulation and mobilisation techniques are thus prescribed on the basis of observable or palpable asymmetry and considered alongside 'abnormal' range of motion (ROM) that is said to be detected by motion tests.

The clinical finding of 'structural asymmetry' is fundamental to this approach, which advocates an evaluation of the differences in position of anatomical landmarks. However, although many patients present with asymmetry this can be due to multivariate factors including, congenital and developmental anomalies as well as ageing and disease states (Bogduk 1997). Congenital and developmental anomalies can include the failure of one or more parts of a vertebra to develop (agenesis), failed vertebral union affecting the secondary ossification centres of vertebrae and also changes in the number or identity of vertebrae resulting in observations of lumbarisation, sacralisation or bony union elsewhere in the spine or pelvis. Other anatomical features such as cervical ribs and the adaptive process through which the clavicle on an individuals' dominant side develops naturally to be shorter and thicker than the opposite (non-dominant) clavicle, can all add to the 'appearance' of asymmetry (Lewis 2004). Many observable states of asymmetry such as torticollis, scoliosis and 'pelvic shift' occur temporarily during periods of healing and recovery.

It is arguable that asymmetry is commonplace, possibly even normal, and in the presence of some growth and disease states, a very adaptive process. This perspective thus applies in the presence of congenital or developmental anomalies as well as through disease states, ageing and also during healing and normal skeletal development. Asymmetry for many individuals must be considered to be 'normal for them' and therefore of questionable clinical relevance in many cases (Robson 2003). After all, asymmetry often persists after symptom resolution. These very important factors are fundamentally overlooked by this clinical approach (Freburger & Riddle 1999).

In addition to 'asymmetry' the 'ART' approach utilises motion tests said to detect 'movement dysfunction'. The motion tests described in much of the literature are predominantly for the spinal and pelvic regions (DiGiovanna & Schiowitz 1991, Greenman 1996, Lee & Walsh 1996, Lee 1999).
It is claimed that the detection of abnormal spinal motion is possible by assessing 'perceived differences' in the coupled movements at spinal joints. Coupled movements are based on the principle of Newton's second law, which states that the motion of an object is directly proportional to the applied force and occurs in the direction of the straight line along which the force acts (Davis et al 1986). Clinically, segmental spinal motion testing is said to be possible by palpating over the transverse processes for perceived differences in motion at the underlying zygapophysial joints during spinal movements such as flexion and extension.

Primary movements of spinal joints do create small 'coupled' movements in other planes during bending and twisting, which minimises resistance to the primary movement. However, in-vitro measurements of coupled movements demonstrate that they are small, inconsistent and change with posture (Panjabi 1989, Cholewicki et al 1996). This is not consistent with claims made in much of the related literature or by proponents of this approach, who purport that coupled movements are a predictable and identifiable phenomenon that can be used to formulate diagnoses and plan treatment (DiGiovanna & Schiowitz 1991, Greenman 1996, Lee 1999).

Bogduk (1997) found that, 'there are average patterns of coupled movements, but not all individuals exhibit the same degree of coupling at any segment or necessarily in the same direction as average; nor do all normal individuals necessarily exhibit the average direction of coupling at every segment'.

In summary, motion tests based on spinal coupling have been shown to be both unreliable and invalid methods of assessing spinal function. Despite these findings some authors have continued to advocate this approach. For example, in her book 'The Pelvic Girdle,' Lee (1999) supports her decision to use this system by stating that 'even if the biomechanics of the lumbosacral junction were confirmed and conclusive, the potential for altered biomechanics to exist is high, rendering 'perceptive clinical observation of a patient as the most direct way to assess spine motion clinically, despite its lack of objectivity'.

Motion tests have also been described as part of the diagnostic procedure used to inculpate 'pelvic and sacro-iliac joint (SIJ) related pain and dysfunction. However, the very question of dysfunction and mechanically related pain at the SIJ's is in itself contentious. In the related literature the incidence of pelvic joint pain has been postulated to be as high as 96% down to less than 1% of those people presenting with lumbo-pelvic pain (Pulisetti

& Ebraheim 1999, Ressel & Rudy 2004). Anaesthetic joint blocks have often been described as the 'gold standard' method of implicating the SIJ as the primary pain source (Maigne et al 1995, Schwarzer et al 1995, Slipman et al 1998, Kokmeyer et al 2002). However, it is generally recognised that without image guidance, injection into the SIJ is not only technically very difficult but also unreliable (Pulisetti & Ebraheim 1999).

It is now evident that pain and tenderness/hypersensitivity to physical testing located in the region of the SIJ's may well be 'referred' rather than originating in the local structures. Referred pain and tenderness follow changes that occur in the spinal cord and higher centres of the central nervous system (CNS). Thus, second-order neurons in the dorsal horn of the spinal cord become sensitised by noxious afferent stimuli arriving from the periphery (for example from injured or pathological tissues and nerves in the lumbar spine) (see chapter 20). This afferent 'barrage' of sensory nerve activity may then lead to changes in the 'characteristics' of second-order nerve cells so that they become 'excited' by non-noxious stimuli arriving from neural networks that relate to other tissues. Hence, normal inputs from networks relating to the normal SIJ tissues may be processed as pain and in so doing not only make the area painful, but also, hypersensitive to touch and to physical tests. This normal tissue hypersensitivity phenomenon is termed secondary hyperalgesia in the pain science literature (Wall 1991, Fortin et al 1999, see also chapter 20). The clinical finding of SIJ area pain and tenderness has the potential to be a false positive as far as damage to the tissues of the SIJ are concerned. The finding may well be a simple manifestation of 'central sensitivity' rather than a true reflection of actual physical damage or pathology.

Some further consideration of central sensitisation and secondary hyperalgesia can be helpful when examining the mechanisms by which anaesthetic injection of the SIJ's might sometimes achieve pain relief. Anaesthetising first-order neurons in the region of the SIJ's can essentially 'shut off', or significantly reduce, sensory input to related second-order neurons in the dorsal horn. This reduced 'normal' input, in some cases, appears to be enough to bring about rapid change in central activity and as a result greatly reduce central sensitisation and thus the perception of pain (Melzack 1994). Interestingly and unsurprisingly, Pulisetti & Ebraheim (1999), found that the hypoalgesic effects of SIJ anaesthetic blocks wore off in 2-14 days in 90% of patients, which questions the long-term value of this technique in therapy.

The clinical reasoning process used here is no different to that used in other clinical scenarios such as calf tenderness secondary to primary lumbar L5/S1 nerve root derived pain (sciatica). Most clinicians would probably feel comfortable reasoning that the calf tenderness is 'referred' from the L5/S1 nerve root and dorsal horn sensitisation. Accordingly the calf would

not be considered to be the primary source of pain. It seems that in the case of SIJ pain this clinical reasoning process is often ignored in favour of inculpating the SIJ as a primary source of mechanically related pain. Yet in the absence of inflammatory pathologies there is no sound evidence available to suggest that the SIJ's are a common site of mechanical dysfunction and/or pain.
The range of movement at the SIJ's is notably very small, even in those diagnosed with hypermobility. Studies of SIJ movement occurring around the transverse, longitudinal and antero-posterior axes found that the mean ROM was 1-2 degrees, with no individual ranges greater than 4 degrees (Sturesson et al 1998, Jacob & Kissling 1995). Studies of the commonly used pelvic and SIJ clinical motion tests have concluded that they are both an unreliable and invalid method of assessing and diagnosing dysfunction and / or pain related to these joints (Potter & Rothstein 1985, Dreyfuss et al 1996, Harrison et al 1997, Slipman et al 1998, Freburger & Riddle 1999, Levangie 1999, O'Haire & Gibbons 2000, van der Wurff et al 2000, 2000a, Riddle & Freburger 2002).

The poor reliability and validity of this approach is aptly demonstrated by the results of a study by Toussaint et al (1999) who carried out a series of standard manual SIJ dysfunction tests on 480 male construction workers and found that although 92.1% had tests deemed to be positive for dysfunction, yet these workers were completely asymptomatic. In summary, there is as yet no sound evidence to suggest that clinical approaches using asymmetry and joint motion testing to formulate diagnoses and treatment are either scientifically valid or reliable.

Joint cavitation phenomena

Joint manipulation is often accompanied by an audible 'popping' sound that has been said to result from cavitation within the joint. Greenman (1996) described the appearance of negative shadows on radiographs taken immediately after manipulative cavitation had occurred. These 'shadows' were said to be due to changes in the density of nitrogen gas resulting from manipulation, which it has been suggested, causes synovial fluid to change from a 'liquid to a gaseous state'. However, recent improvements in diagnostic imaging including computerised tomography (CT) have not identified any post-manipulative evidence of gas in the joint space or of any increase in the width of zygapophysial joint spaces (another claimed effect of manipulating these joints). These findings were the same whether or not traction was applied to the joint during manipulation (Cascioli et al 2003).

Meniscoid and capsular entrapment

Various theories surrounding entrapment of the articular capsule or meniscoid within zygapophysial joints have been proposed as a cause of spinal pain and dysfunction (Bogduk 1997). High resolution CT and MRI imaging has never identified meniscoid or capsular entrapment and both their presence and amenability to treatment via mobilisation or manipulation is entirely speculative.

'Subluxation' theory and evidence

Chiropractic philosophies of assessment and treatment were founded over 100 years ago based on the belief that joint subluxation or 'positional faults' occur, particularly at spinal joints, and that this 'abnormality' interferes with the body's health. Despite significant progress within orthodox medical sciences, chiropractic subluxation theory has changed little since Mr D.D. Palmer, a nineteenth century grocer and 'magnetic healer', claimed to have 'discovered' chiropractic. Chiropractic literature cites subluxation as a major contributory cause of many human conditions as diverse as pain, autism, epilepsy, hypertension, Bell's palsy and mental illness (Jamison 1987, Sandteur & Adams 1987,Goff 1988, Pistolese 2001, Alcantra et al 2003, Ressel & Rudy 2004). The list of diseases and ailments chiropractors consider to be related to subluxations is both extensive and extraordinary. The plethora of conditions claimed to be amenable to chiropractic treatment is almost endless. For example, Chiropractic literature includes such topics as a role for chiropractic therapy in influencing the body's immune system in people with acquired immunodeficiency syndrome (AIDS) (Lucido 1988) as well as in Parkinson's disease (Elster 2000 & 2004), multiple sclerosis (Elster 2004) and infertility (Rosen 2003, Shelley 2003).

One example of chiropractic education literature aimed at encouraging the general public to seek chiropractic treatment proclaims that, "subluxation is interference in your body that can lead to decay, dysfunction, and in some cases death" (www.discoversubluxation.com). This sinister and very worrying sounding statement is accompanied by the 'encouraging' message that regular chiropractic treatment to correct spinal subluxations can prevent this from happening! However, again, and perhaps reassuringly, there is no worthy evidence whatsoever to support chiropractic claims that visceral, immune or mental disease states emerge as a result of subluxation induced compression of autonomic nerves in the intervertebral foramen of spinal joints.

Rigorous literature searches of diagnostic scanning and radiological studies have failed to identify the heralded phenomenon of 'subluxation'. Interestingly and encouragingly, some chiropractic authors (Keating 1988, Keating et al 2005, Seaman 2004) have voiced their professional concern regarding the total absence of evidence surrounding the existence of subluxations, suggesting the need for more research and a move away from the existing chiropractic paradigm. Due to an absence of radiographic evidence Dalseth (1976) stated that 'the popularly used chiropractic x-ray diagnosis of positional faults or 'subluxations' is illusory'.

As this notably 'theosophical' chiropractic approach was founded on the proposed existence of subluxations, the subsequent lack of any reasonable evidence for 'subluxations' naturally nullifies all of the self-generated claims that have been made regarding the merits of chiropractic theory as it currently stands. It is not unreasonable to point out that a treatment cannot be prescribed to 'cure' something (subluxation) that does not exist.

A brief history of pain science

Since the 16th century specificity model of pain was proposed by Rene Descartes, the 'Cartesian' model has dominated the medical treatment of pain until recent times. Several 'pattern theories' followed Descartes model before publication of the now classic paper in 1965 by Melzack and Wall (1965) who described the 'gate control theory'. This was the first model to describe in any detail the involvement of descending pathways from the brain to spinal cord and their proposed role in modulating pain. To this day the gate control model has endured and expanded to include contemporary scientific knowledge. Our present understanding of pain offers a number of important factors in relation to physical medicine and manual therapy. As the preceding review of traditional manual therapy techniques indicates, many earlier theories have been expounded and nullified by a growing understanding of pain science. Much of the guesswork has been replaced by an expanding knowledge of the mechanisms specific to manual therapy that are capable of modulating pain.

It is known that the overall experience of pain involves the interplay of receptors, connecting neural pathways and processing within the central nervous system (CNS). There is no single un-modifiable, 'one-way only', 'Cartesian like' pathway in the nervous system responsible for pain. Pain is a highly complex amalgamation of 'in-parallel processing' of multiple sensory, cognitive and affective/emotional elements, all of which contribute to our pain experiences all the time, whether acute or chronic (Gifford 1998, 1998a). The fact that pain pathways are not 'hard wired', but modifiable, emphasises the importance and true holism of the biopsychosocial factors involved in everybody's experience of pain. Contrary to the way that many

theoretical paradigms of pain-related musculoskeletal dysfunction shackled us firmly to the peripheral bodily tissues; current pain science directs us centrally to give our long overdue attention to the functioning of the brain, the nervous system and its inherent plasticity.

Despite limited and varying degrees of clinical success, manual therapy has become the preferred mode of treatment for many practitioners (UK BEAM trial team 2004). Evidence shows that some manual therapy techniques including mobilisation and manipulation can have effective pain relieving qualities. However, there is no evidence to suggest that pain relief resulting from manual therapy occurs via the traditional mechanically based theories discussed. As it has already been argued, if musculoskeletal pain problems were purely of a 'mechanical' nature, surely we would expect more consistent outcomes from every patient-manual therapy interaction? 'You-have-normal-range-of-movement-therefore-you-have-no-pain'just doesn't always follow. The very lack of consistency begs us to provide more credible explanations. At the same time, however, we need to have a reserved respect for the likelihood that when an individual has some movement impairment, homeostatic monitoring mechanisms may well be continually striving to promote 'mechanical' well-being and harmony. For example, by producing a pain response component whose only fulfilment is the restoration of normal movement, mechanical well-being and adequate intrinsic support. With perspectives like this though, there is a need for a wider appreciation of an individual's conscious and unconscious information monitoring and processing and the plethora of mechanisms that must gather to produce a response, – the very stuff that goes to make up the biopsychosocial approach argued for here.

It is recommended that if a more effective use of manual therapy is to be achieved and promoted there is an urgent need to incorporate much more of the current available pain science evidence into most manual therapists' clinical reasoning. This may well require an unpalatable and possibly radical shift of philosophy for a great many practitioners. The aim is to focus the inclusion of manual treatments more appropriately, and at the same time offer a sound and mature scientific framework that is acceptable and understood by the wider medical community and the general public alike, who, in the opinion here, have been mislead for far too long.

Pain related neurobiology

(for greater detail of pain neurobiology see chapters 18 and 20 this volume)

It is beyond the scope of this discussion to include an in-depth review of the neurobiology involved in the production of pain or the mechanisms involved in pain facilitation and inhibition; however a basic understanding of this is fundamental to maximising available therapeutic interventions. There is now compelling evidence to demonstrate how both transient and potentially permanent pain related changes take place within the CNS following tissue injury. This has provided considerable insight into the neurobiological basis of pain.

Central sensitisation

It is known that *first order* nociceptive specific (NS) neurones (nociceptors), not only undergo changes in their peripheral terminals following tissue injury, but also at their central terminals within the dorsal horn of the spinal cord. The process is called 'central sensitisation' by pain scientists (Wall 1991, Fields & Basbaum 1999, Woolf & Slater 2006). Further, synapsing *second order* 'NS' cells, that lie in the dorsal horn and project to the brain, also become increasingly more sensitised to incoming sensory impulse traffic (Woolf & Slater 2006). As already discussed in the sacro-iliac joint section earlier - once sensitised, modest or even normally sub-threshold stimuli from previously dormant collateral synapses that relate to input from quite normal structures, become capable of eliciting a response and hence producing pain. Thus sensory input from normal structures can be processed in terms of pain. These changes in the behaviour of NS first and second –order cells result from complex chemical and anatomical changes of the cell (see chapter 20). It is now well known that the second order NS cell's characteristics 'plastically' change to become more akin to those of so called 'wide dynamic range' (WDR) neurones (Wall 1991). WDR neurones, whose cell bodies are situated in the dorsal horn region of the spinal cord, normally respond to a 'wide' range of stimuli. Hence responses can occur from mechanical, chemical and thermal peripheral stimuli that range from non-noxious at one end of the spectrum to noxious at the other (Craig & Dostrovsky 1999). If any of these types of stimuli from the periphery reach the 'sensitised WDR like' central NS neurones the resulting output that surges upwards to the brain can be massively 'exaggerated' or amplified. Thus, minor input from the periphery via primary afferents subserving damaged tissue (primary hyperalgesia & mechanical allodynia), as well as input from surrounding and distant *undamaged* tissues (secondary hyperalgesia) can cause an explosive central reaction that may be processed in some situations as quite marked and severe pain. What is even more remarkable is the observation that sensitised dorsal horn cells are capable of producing spontaneous 'ectopic' output in the absence of any primary

afferent input from the tissues at all (Barker et al 2003) – hence the potential for pain that is felt in tissues actually deriving from within the CNS (Gifford 1998a). The fascinatingly close parallel this has with the mechanisms thought to underlie phantom limb pain have been discussed at length by Gifford (1998a).

It is of great clinical significance that the sensitivity of the dorsal horn of the spinal cord is strongly influenced by descending pathways from the brain; and that these can act to both facilitate and inhibit dorsal horn activity (Wright 1995, Price & Bushnell 2004, see also chapter 20). Furthermore, the brainstem nuclei from which the descending pathways originate are extensively connected to, and influenced by many cortical and subcortical regions (Price & Bushnell 2004). As well as processing 'nociception', these higher brain regions are involved with processing psychological information such as thoughts, beliefs, emotions, attention, motivation and pain context. Collectively these psychological factors exert immense influence over descending pathways and the overall experience of pain (Zusman 2002, Price & Bushnell 2004). It is becoming evident that the way in which an individual psychologically responds to pain derived from nociceptive activity is likely to have a neurobiological impact throughout the whole of the nervous system involved. Powerfully, this is saying that psychological factors, like focus of attention, anxiety and fear, cause actual physical and neurophysiological changes in the nervous system that can lead to long term pain states (Gifford 2006). Hence, *chemical* changes, like the quality, amount and type of neurotransmitters, *physiological* changes for example in impulse processing and transmission; and actual *anatomical* changes in nerve cell architecture like axon growth and the formation of new synapses (Woolf & Salter 2006).

Increased focus on pain that links to pain anxiety and pain concern is likely to dampen the effectiveness of the descending inhibitory control system but at the same time facilitate the antagonistic descending excitatory control system. Thus concerned attention to pain may turn the 'pain-off' cells off and the 'pain-on' cells on (see discussion below and Fields et al 2006). The result of this is increased nociceptive activity and increased pain, - a very important ingredient that leads to long-term central hyperexcitability and the resulting chronic maladaptive pain. The mechanism by which this occurs is called 'Long-term-potentiation', a term that has been taken from the field of memory biology because of its remarkable similarity to the neurobiological processes observed in pain (e.g. Rose 1992, Pocket 1995, Ji et al 2003, Woolf & Salter 2006). It seems that pain gets established in the central nervous system in a way that is very similar to that occurring in memory acquisition (Gifford 1998a, 2006 and chapters 18, 20 of this volume).

Long-term potentiation

Long lasting nociceptive afferent stimulation of dorsal horn neurones can evoke long lasting 'central sensitisation' with the potential to lay down 'pain memories' (e.g. Gifford 1998a, Sandkhuler 2000). As discussed above, this involves actual chemical and anatomical changes of these cells, which are mediated by activation of inherent cellular protein kinases. Protein kinases are 'messenger molecules' found in neurones that stimulate gene transcription factors causing specific genes to 'switch-on' and express in favour of increased production of proteins that are used to form more receptor sites and new synapses on the post-synaptic cell membrane (see Chapter 20 this volume for detail). More receptor sites essentially act to 'strengthen' synaptic connectivity and hence, it's efficiency. This process is known as long-term potentiation or LTP (see LeDoux 2002, Zusman 2004). Thus, if LTP occurs at multiple sites it causes the formation of a new 'pain-memory pathway', it leaves an 'imprint' or 'memory-trace' in the CNS that has the potential to be easily reactivated or even maintained permanently in an active state. Thus, once pain 'gets into the system' it may be very hard to get rid of – consider how difficult it is to forget something and then you can appreciate how difficult it may be to get rid of a 'pain' imprint! It is very difficult to take your mind off an unpleasant memory, and if you do manage it, it doesn't take much to rekindle it again - just like those who suffer ongoing maladaptive chronic pain (see Gifford 1998a).

Cortical reorganisation

There is compelling evidence that the primary sensory and motor areas of the cortex are continuously changeable or 'plastic' throughout life (chapter 18 this volume). The more we use or do something – the greater the activity and the greater the density of neurones involved. Conversely, the less something occurs, the more its representational pathway tends to diminish (see chapter 189). Activity in cortical zones representing our various body regions is not only altered by injury but also by behaviourally relevant stimulation and training. Utilising this knowledge as part of a therapeutic approach when treating notoriously difficult conditions such as complex regional pain syndrome and phantom limb pain has produced some extremely impressive results (Flor et al 1997,McCabe et al 2003, Flor 2002, Moseley 2004). Some authors have described cortical changes related to pain in terms of the size and delineation of cortical somatic zones involving shrinkage and expansion as well as 'smudging' of these regions. However, the involvement and degree to which cortical reorganisation is responsible for causing pain is still uncertain. (discussed at length in chapter 18)

As discussed, the therapeutic application of this new knowledge requires a fundamental shift away from traditional clinical theories. Zusman (2004) referred to the importance of this when he discussed how achieving extinction (new synaptic learning) was about logically "teaching the nervous system (not muscles!) to instruct muscles on how to negotiate everyday movements without (undue) pain provocation" and thus enable the laying down of a 'new' adaptive motor 'memory' (extinction).

Flor (2004) has demonstrated how psychological processes, such as operant conditioning, classical conditioning and selective attention, influence cortical reorganisation and pain memory. These findings suggest a number of directions amenable to therapeutic interventions and these will be discussed later.

In summary, it is now known that neuroplastic changes take place at all levels within the central nervous system following injury. These changes occur in the regions of the spinal cord, brainstem, thalamus and cortex and they all play a vital role in the development and maintenance of pain (Price & Bushnell 2004). Psychological processes are capable of enhancing or diminishing the experience of pain and as we have seen, can strongly influence neuroplastic changes. It is becoming more and more apparent that despite the commonality of the neurobiological and psychological mechanisms involved in the development and maintenance of pain, each persons pain experience is likely to be made up of different quota's of each of these 'products'. In fact the content of these products will also be subject to change along with each individual's state of health and life experiences. It is hardly surprising that the formulaic 'recipe type' approaches of manual therapy and physical medicine have so far merely scratched the surface of a much deeper problem (Waddell 2003, Frost et al 2004, UK BEAM trial team 2004).

Pain relief following mobilisation and manipulation

Gate control

Good evidence exists to demonstrate how manual therapy can effectively stimulate central mechanisms resulting in *hypoalgesia*. One component of this involves activation of the spinal component of the gate control mechanism within the dorsal horn of the spinal cord. Rapid pain reduction via spinal gating can occur if large low-threshold A-beta fires are stimulated manually (or otherwise) in the periphery. A-beta fibres synapsing at dorsal horn cells have an inhibitory effect on the nociceptive output of these cells essentially reducing the flow of nociceptive output through the spinal gate

(Wall 1999a). One problem that has to be reconciled is that the process of central sensitisation described earlier results in A-beta fibre input being processed as pain, hence 'proprioceptively' and 'light-touch' exacerbated pain. Manual therapy therefore has the ability to aggravate pain when these underlying conditions occur.

There are two further important considerations with regards manual therapy and its relationship to pain-gate 'closing' potential.

Firstly: Loss of A-beta neurones may be an important consideration because they are notoriously susceptible to injury with subsequent dysfunction, degeneration and death. While it needs closer scrutiny, it is likely that anyone who has suffered a significant nerve root pain, e.g. sciatica or brachialgia, will have some kind of A-beta fibre impairment. Loss of A-beta fibres from the periphery means the loss of a normal, peripherally based, inhibitory mechanism at the pain gate. Hence, a clear tissue-based reason for the lack of effect/aggravating potential, of manual therapy input in many patients.

Secondly, the death of spinal cord inter-segmental neurones that are characteristically inhibitory (gate-closing) in nature has also been observed to occur in animal models of nerve-injury related pain (see Woolf & Salter 2006). Here, high levels of afferent nociceptive traffic following nerve injury leads to massive increases of excitatory amino acid release in the dorsal horn. Via 'excitotoxic' effects this massive over-load of chemicals can lead to the death of vulnerable intersegmental inhibitory interneurones – and hence to a loss of effectiveness of normal gate-control. Just from reasoning at this level it is hardly surprising that there are such a wide range of responses to pain treatments whose effects mostly derive from tissue based inputs.

Descending pathways

Further to the inhibition of nociceptive flow at the dorsal horn via input derived from the tissues, is the influence that the descending pathways from the brain are likely to have on the 'sensitivity' and 'plasticity' of the spinal cord processing mechanisms (Wright 1995, Fields & Basbaum 1999, Souvlis et al 2004, Zusman 2002, 2004,). A number of brain regions and descending pathways responsible for the hypoalgesia associated with manual therapy have been identified (Casey 1999, Fields et al 2006).

The periaqueductal gray (PAG) area is an important modulator of nociception. Activation of PAG nuclei and their related pathways via afferent stimulation is thought to be capable of activating descending inhibitory systems (Skyba et al 2003). 'Maitland grade 3 type' joint

mobilisations are an example of 'afferent stimulation'. Other pain relevant inhibitory nuclei and pathways that are thought to be stimulated by the physical effects of manual therapy include those relating to the nucleus cuneiformis, locus coeruleus (LC), nucleus reticularis gigantocellularis (NGC), nucleus reticularis dorsalis (NRD) and rostral ventromedial medulla (RVM)(see chapter 20). The RVM is significant since it is a major source of brainstem axons projecting to the dorsal horn and is capable of both inhibiting and facilitating nociception via two classes of intrinsic neuron involved in 'switching' nociception on or off. Research indicates that the descending pathways identified utilise noradrenaline and serotonin (5HT) as their neurotransmitters, however the sensitivity of the RVM is also strongly influenced by both endogenous and exogenous opiates (endorphins & enkephalins) and is therefore also modulated by other non-mechanical stimuli (Wright 1995, Fields & Basbaum 1999, Souvlis et al 2004, Fields et al 2006).

Diffuse, or 'distant, noxious inhibitory control (DNIC)

Contemporary as well as ancient pain treatments use pain to relieve pain. Throughout history therapeutic counterirritation produced by such modalities as heat, cold, chemical irritants and intense mechanical stimuli have been used and documented. Contemporary physical therapy is not in short supply here either, Geoff Maitland has taught students for many years to: 'find what hurts and hurt it'. The technique usually involves the therapist driving their thumbs into the specific pain sensitive tissues with extreme vigour. A major aim of physical examination taught to most Physiotherapy students is to find the 'physical source' of the pain and focus treatment on the area. Many practitioners use manipulations and mobilisations that do hurt patients and of course can (but not always!) relieve symptoms for a while. Acupuncture, acupressure and transcutaneous electrical nerve stimulation has also been observed to produce varying levels of counterirritation and subsequent pain relief in human as well as animal experiments (see Melzack 1994 for an excellent review). Upon discovery of the PAG – RVM – dorsal horn modulatory pathway it was proposed that intense and noxious stimulation actually activated the descending inhibitory control systems and thus reduced nociceptive transmission to the brain from the level of the spinal cord. This effect appears to be mediated by the enkephalin group of endogenous opiates, which are known to be released at spinal and supraspinal levels (Fields et al 2006). In puzzling contrast to this it is also known, as one would expect, that cutaneous noxious stimulation can activate the pain 'on cells' and inhibit the pain 'off cells' at the RVM – hence pain now actually enhancing pain. Here, noxious stimulation in one part of the body *facilitates* nociception and pain in other parts of the body – think of the hypersensitivity of many chronic pain patients, – even minor discomfort

produced by quite modest manual therapy away from the pain areas can produce marked exacerbations of their pain state. In other patients noxious stimulation using 'getting at the pain' techniques or vigorously and painfully manipulating well away from the area of pain can pleasingly activate quite significant analgesic effects. Most of us are familiar with the experience of stubbing a toe leading to the realisation that some pain elsewhere has been relieved.

DNIC is thought to occur via a 'surround' inhibition that heightens the contrast between the area being stimulated and the surrounding region. This would in effect increase the perceived intensity of pain at the area of stimulus but would also result in a net analgesic effect in the regions outside of the stimulated zone. It seems that the brain is well organised to focus all its attention on the item of most immediate concern, and put on-hold, or even completely re-evaluate, less important or less pressing issues for later consideration.

In summary, noxious stimulation from modalities including manual therapy activate multiple CNS networks. Some of these networks facilitate nociception and some inhibit it. Whether an individual's response to noxious stimuli is increased or reduced pain depends on factors like the location of stimulus, its duration, the circumstances in which it is delivered and the context and meaning of the noxious stimulus for that person. What they are thinking, feeling and focusing on during the treatment process may be a very important consideration (Thacker & Gifford 2002, Gifford 2006). We should also never forget to consider a vast spectrum of individual differences in sensitivity – one person's pleasure is another's pain. Unfortunately, just because one individual gets benefit from a painful intervention does not mean a similar problem for another individual will receive the same benefit from it! Further, a given individual's 'sensitivity-setting' may show a considerable *normal* fluctuation, day to day, hour to hour, minute by minute. Logically, all this means that for *any* physical treatment to have a positive effect there has to be a serious consideration of the patient's 'processing' or 'appraisal' of the situation – hence, 'top-down – before bottom-up' (Gifford 2006) – meaning work on tissues cannot begin (or is unlikely to succeed) until the brain is 'set-up' to accept it. It seems highly likely that whatever we do with patients must make sense to them if it is going to stand any reasonable chance of helping (Gifford 2006).

Habituation

There is compelling evidence that graded exposure to mechanical stimuli such as mobilisation techniques can reduce and sometimes abolish pain through the process of habituation. Habituation occurs as calcium ion channels on the presynaptic terminal of a nerve become less efficient

following repeated stimulation and opening, this results in a marked reduction of the available calcium essential for the release of synaptic neurotransmitters. The subsequent decline in volume of neurotransmitter decreases the postsynaptic nerve potential and reduces the nerves ability to reach the threshold necessary for impulse generation. Essentially, habituation can be considered to be a decline of a conditioned response following repeated exposure to the conditioned stimulus (Jastreboff & Hazell 2004). Most therapists will have observed how pain can gradually subside or even change location during treatments using manual techniques or repeated exercise. This mechanism has been proposed by Gifford (2002b) to, at least partially, explain the changing pain response that can be achieved using the repeated movements advocated by the 'McKenzie approach'. Gifford (2004a, 2006) calls it the 'pain-boredom' effect or 'pain-boredom-acquisition processing', when explaining it to patients! For it to be successful one important 'top-down' ingredient is that the patient must feel comfortable and confident that repeatedly bringing on a pain is not harming.

Extinction

As already discussed, under certain conditions the CNS develops 'pain memories' through the process of LTP. New learning, involving the acquisition of 'painless memories', can bring about 'extinction' of an ongoing and maladaptive pain memory (Zusman 2004). Extinction involves the formation of a new memory via synaptic learning and LTP. In order for this new memory to be of any therapeutic value it needs to supersede the former pain memory in the ongoing 'competitive struggle' for perception dominance within the CNS. Therefore the stronger and more practiced the mode of learning the greater likelihood there is for extinction and clinical success. However, new memory acquisition / LTP does not eliminate an older memory and therefore old pain memories are still capable of 'breaking through' periodically or permanently into perception. This will be recognisable to clinicians as symptom 'flare ups' and the relapsing and remitting nature of many peoples pain.

Facilitation and inhibition of motor activity

A number of studies have reported manual therapy as having both inhibitory (Wright 1995, Zusman 1992) and facilitatory (Keller & Colloca 2000, Colloca & Keller 2001, Dishman et al 2002) effects on the motor system. However, some of these studies lack scientific validity, insofar as the experimental models used were based on hypothetical treatment approaches and clinical theories. As a comment and in contrast, descending, cortico-spinal 'motor' pathways, are known to send inhibitory and

excitatory branches to the sensory and nociception processing regions of the outer layers of the dorsal horn (reviewed in Galea & Darian-Smith 1995). Hence an evolutionary elegant pathway whereby pain can be dramatically stopped in order to allow movement to proceed. Limping away in pain is of no survival value when a lion is chasing you! No wonder function-with-purpose has always been such a good painkiller.

Sympathetic nervous system (SNS) responses

Similarly, some studies performed on SNS responses to manual therapy have also lacked construct validity (Clinton & McCarthy 1993). However, Souvlis et al (2001) and Vicenzino et al (1999) have demonstrated an excitatory response of the SNS to grade 3 type mobilisation techniques, these include sudomotor (increased body temperature) and cutaneous vasomotor (blood vessel dilation) responses.

Placebo

As with many therapeutic interventions it is inevitable that varying degrees of placebo responses to manual therapy techniques will be observed. The Oxford dictionary (1996) defines the 'placebo effect' as a 'beneficial (or adverse) effect produced by a placebo and not due to any property of the placebo itself'. It is recognised that placebo is a complex interaction of biological, psychological and sociological processes (as already discussed), and there are a number of factors surrounding placebo that are pertinent to manual therapy.

- Foremost, it is a myth that 33% of patients receiving placebo treatment for painful conditions will respond to the treatment with at least 50% reduction of their pain. Placebo responses have been shown to vary between almost 0 – 100% depending on the circumstances of the study (Wall 1999, Roche 2002).
- Circumstances that can increase placebo response include the level of confidence and belief practitioners display and have in the treatment techniques they use and the perception of this by their patient's.
- The more 'powerful' a treatment appears to be, the greater the placebo response is to it.
- Placebo does not merely affect the psychological aspects of pain; it can initiate real physiological effects including analgesia and decreased tissue inflammation.

(The placebo response is discussed at length in Topical Issues in Pain Vol 4: Gifford 2002a)

Integrating evidence into practice

Current scientific evidence offers insight into a number of potentially appropriate treatment modalities for pain, including manual therapy. In order to incorporate this growing body of evidence into clinical practice a perpetually changing and adaptive approach to clinical reasoning is fundamental if maximum therapeutic benefit is the goal. Many of the current 'hands on' and 'hands off' techniques can be integrated with current evidence and utilised in treatment. However, as discussed, much of the past theoretical basis surrounding 'hands on' techniques is at best scientifically very shaky and most likely invalid, hence a somewhat urgent need for new paradigms and shifts in thinking. As each person's pain experience involves dynamic elements of biological, psychological and social factors, it is important that each of these elements will need to be entered into the clinical reasoning equation. It is not trite to suggest that paying mere 'lip service' to this process of reasoning is likely to result in poor outcomes.

As the basis of this discussion involves a review of manual therapy a detailed analysis of biopsychosocial assessment will not be made. However, current evidence indicates that a good working knowledge of red and yellow flags is essential and readers are referred to works by Roberts (2000), Watson (2000), Watson & Kendall (2000) and Gifford et al (2006).

Clinical guidelines (CSAG 1994, Waddell 2003) that are derived from much relevant research (e.g. Linton 1996, 1998, 2004) suggest that it is essential to carry out a comprehensive 'low-tech' physical examination of the involved neuromusculoskeletal system. This can be used to not only screen for red flags but also to provide information regarding the patient's neurological and musculoskeletal function, including the presence or absence of such factors as mechanical allodynia and/or hyperalgesia. These findings provide an overview of the patient's neuromusculoskeletal 'sensitivity' state. As psychosocial factors are likely to be critical in modulating levels of 'sensitivity' we advocate carrying out a full or modified psychosocial assessment during initial examination of all patients. This not only 'flags' those at risk of chronicity but also provides information to assist the clinical reasoning process and planning of treatment. Although the 1994 CSAG guidelines for back pain suggest adding a psychosocial assessment at 6-weeks for 'non-responders', recent evidence indicates that the neuroplastic changes associated with chronicity are in place within hours of tissue injury, it is therefore proposed that early detection of known associated psychosocial risk factors is vital when planning both initial and ongoing treatment.

Although 'yellow flags' were not specifically intended for planning manual therapy treatment, they can be of enormous benefit when assessing all patients with pain. An appreciation of each patient's attitudes, beliefs and behaviours offers insight towards their individual levels of distress and coping. To demonstrate this it is worth considering some typical psychosocial assessment findings of a patient with low back pain. These findings might include reports of high pain intensity, a belief that pain is beyond their control, fear that pain is harmful and must be abolished before returning to activity as well as fear avoidance behaviours including the cessation of exercise and withdrawal from some activities of daily living (see clinical example chapter 1 this volume). If it is found, following assessment of a patient, that the sum of their yellow flag findings indicates high levels of 'sensitivity' and distress, it might be important to question the appropriateness and wisdom of administering a strong manipulative thrust technique to the patient. Clinical reasoning utilising the biopsychosocial model and approach would suggest that such a technique could well result in 'flaring up' this patients symptoms. The possible detrimental consequences of using manipulative treatment in these circumstances may appear blatantly obvious, but if no enquiry regarding yellow flags was made how else could this have been reasoned and predicted? If we now contrast this with the 'recipe like' reasoning implicit within the traditional manual therapy philosophies discussed earlier, this same patient and clinical circumstances could easily have resulted in a decision to prescribe and administer strong manipulation.

Clinically, it is important to view the physical findings for this 'sensitised' type of patient from a perspective that sees the observations of movement and testing as being a reflection of their overall 'bio-psycho-social status'. For example loss of range of forward bending could be interpreted in one or all of the following ways:

1. A clear physical impairment (for example ankylosed lumbar spine and tight hamstrings) = **'Bio'** (the range is the same whether awake and alert or under deep sedation of an anaesthetic.)
2. The amount of pain produced and the response to it = **bio-psycho**
3. A reflection of the patient's loss of confidence, fear of re-injury and fear of exacerbating the pain **(bio-psycho-social)**.

Clinical reasoning using the biopsychosocial paradigm indicates that it might be more appropriate to begin patient management by enquiring and explaining about pain and thus initially attempting to reduce levels of fear and distress (Butler & Moseley 2003, Gifford 2006, Gifford et al 2006). It might also be reasoned that management could be commenced with a 'hands off' approach such as graded exposure, which aims to help patients challenge fears associated with factors like movement-related pain or injury by gradually encouraging the restoration of 'thoughtless fearless' functional

movement (Gifford 2003, 2006). For the patient described, a 'hands on' technique like gentle soft tissue massage might be a more effective and appropriate treatment to start the 'desensitising' and confidence building process.

From the patient example, what seems to be imperative is the need for both an appreciation and acknowledgement of the patient's overall state combined with an understanding of the biopsychosocial factors that underpin it. This creates a much healthier multidimensional forum for sound clinical reasoning. Clinical reasoning that has its underpinnings firmly anchored in good science and sound clinical management is free from the constraints imposed by the mostly one-dimensional traditional treatment approaches that are widely taught and promoted.

Considering the evidence discussed earlier regarding the efficacy of manipulation and mobilisation, it hopefully becomes clearer how many current techniques can be utilised to assist with pain relief. The available evidence also strongly emphasises that manual therapy and other passive therapies are only one part of a much bigger biopsychosocial picture. Used judiciously manual techniques can sometimes offer a useful window of opportunity to assist patients with management and understanding of their pain but they are rarely likely to be the sole answer in the long-term.

A new paradigm for manual therapy

Available evidence now outlines a number of pain relevant neurophysiological mechanisms that are amenable to manual therapy. For example, mobilisation and manipulation techniques like those described by Maitland (1986 & 1991) involving passive types of physiological and accessory joint movements can be used to stimulate inhibition of pain processing in the dorsal horn, as well as 'triggering' descending inhibitory pathways acting at the level of the spinal cord.

However, it is hard to see how the rather transient nature of a manipulation technique could produce significant levels of habituation. The oscillatory or sustained nature of many mobilisation techniques may well lend themselves towards achieving clinically effective states of habituation if the context in which they are performed is subtly altered. For example, as LTP has qualities of both specificity and associativity, any new synaptic learning (LTP) would need to result in some useful functional improvement for the patient if it is going to have any clinically meaningful value. New synaptic learning would be of little use to a patient if it were associated with lying face down on a therapists couch while mobilisations were performed on their lumbar spine. However, if the patient can see and feel that it is

possible to bend forward with no pain and hence start to 'learn' pain-free movement, then this could have enormous functional significance. Experimenting with various mobilisation techniques while asking the patient to move can produce better and less painful movement. Some familiar examples of the sorts of techniques that can be used include 'so called' sustained natural apophyseal glides (SNAGS) and mobilisation with movement (MWM) (Mulligan 1999). Used in the right context, for example with lots of explanation combined with therapist assistance and reassurance, these types of techniques can sometimes help patients challenge feared or restricted movements.

Additionally it has been demonstrated that 'psychological products' such as classical and operant conditioning and selective attention and verbal cueing (Flor 2002a) are all capable of modulating neuroplasticity, including that of the cortical regions. This suggests the need for incorporation of a number of adjunct techniques while performing manual treatments. Thus, various forms of verbal encouragement, positive reinforcement and positive use of selective attention can be incorporated. Selective attention is fascinating, since simply asking the patient to focus their attention on a neutral or non-threatening stimulus such as pain free joint mobilisations can reduce or stop the supply of some of the 'psychological fuel' necessary for pain perception. However, in order to ensure that the patient is not being encouraged to concentrate on a painful or threatening stimulus (thus reinforcing their pain) it can be helpful to make basic enquiries during the administration of mobilisation techniques. This might include questions such as, 'does that feel okay'? 'Would you like me to use more or less pressure'? Or statements like; 'It's important that you feel comfortable with what I'm doing'. 'If it is causing pain I want it to be a type of pain that you feel is doing it good if at all possible'. 'This is much better if it's nice pain rather than nasty pain'.

As LTP requires regular periods of 'training' to achieve and maintain new synaptic learning, the prescription of exercises specific to restoring part or all of 'missing' functional movements is fundamental to achieving successful rehabilitation. It has been demonstrated that cortical reorganisation requires behaviourally relevant training and does not occur with passive stimulation (Jenkins et al 1990). Therefore isolated passive treatment in the form of mobilisation or manipulation is unlikely to be helpful in cases of chronic pain, regardless of the dosage of passive treatment administered. This emphasises the vital importance of an all-encompassing biopsychosocial approach, including exercises and training that achieve functional rehabilitation specific to each patients requirements and in particular their desired activities of daily living. Recent evidence has helped to outline both the indications and limitations of manual therapy.

The safety of mobilisation and manipulation techniques

As far as we are aware there is no evidence to suggest that serious tissue based injuries occur with 'reasonable' grades of mobilisation techniques. On the other hand, there is little doubt that even modest mobilisation techniques administered to patients with significant peripheral neurogenic or maladaptive central sensitivity are easily capable of 'stirring up' pain and associated symptoms. Knowledge of maladaptive pain mechanisms and the potential they have to cause significant flare-ups *without* tissue damage must surely help therapists and patients to have a better and more confident understanding of the situation. As we all know, it is never pleasant to stir up a patient's pain, especially with the belief that some ghastly damage has occurred.

The literature does contain numerous studies detailing incidents and accidents resulting from spinal and pelvic joint manipulation. Stevinson et al (2001), Grieve (1994), Dupeyron et al (2003) and Assendelft et al (1996) identified vertebrobasilar artery (VBA) injuries, intervertebral disc prolapse / sequestration and cauda equina syndrome as the most common accidents following manipulation. Other documented manipulation related accidents include death, stroke, nerve root compression, paraplegia, vertebral fractures and embolisms. It is pertinent to note that there are as yet no designated pre-manipulative screening protocols capable of eradicating these manipulative accidents (Haldeman et al 2002).

The average age of those patient's suffering VBA related strokes following cervical spine manipulation is 38-years old, this is far lower than that of national 'stroke averages'. This is particularly worrying in light of the otherwise 'rare' occurrence of this type of stroke in young people. There are conflicting reports in the related literature surrounding the incidence of manipulative accidents. Risk factors have been quoted to be between 1 in 20,000 to 1 in 4,000,000 manipulations performed. Until recently it has been difficult to ascertain a realistic incidence of manipulative accidents. This has largely been due to the significant under reporting of these events. However, Dupeyron et al (2003) studied the incidence of manipulative accidents occurring to patients who went on to present to 133 physicians including neurologists, neurosurgeons, and rheumatologists over a 2-year period. They found that the incidence of VBA accidents alone was 30 times higher than that in published series. Potentially, this indicates that the risk of serious manipulative accidents could be as high as 1 in every 666 manipulations performed.

In view of the incidence of 'side effects' associated with spinal and pelvic joint manipulation, it is probably fair to assume that if these techniques were subject to the same clinical trials and scrutiny as new drugs, their

licensing would be rejected on the basis of their inherent risk of injury. Furthermore, there is no evidence whatsoever to suggest that manipulation techniques have any superior clinical effect above and beyond alternative non-injurious mobilisation techniques. In view of the available evidence regarding benefit and risks, it is our belief that there are no circumstances in which it would appear appropriate to recommend the use of manipulation in clinical practice.

A willingness to change?

Jones (1995) stated that 'the principal fault behind many of the colossal misdirection's throughout scientific history has been the blind acceptance of what is written or professed as the truth at the time'. Historically, Cartesian reasoning and a preoccupation with various 'guru' driven theories have exerted a considerable and prevailing directional influence over the 'physical therapies'. However, even when elements of the 'truth' begin to emerge good clinical reasoning can still prove difficult if we don't know what clinical approach to adopt or which professionals to believe.

Currently the dissemination of 'best evidence' appears to be somewhat haphazard and while being met with enthusiasm in some quarters is feared and even reviled in others. The sheer volume of intra and inter-professional debate surrounding the emerging evidence base over recent years has highlighted this situation. Much of the criticism directed at evidence-based practice by some clinicians has contained the accusation that it removes a therapist's 'flair' and individual freedom to practice those techniques they feel are instinctively right for their patients. Most realistic clinicians probably agree that while clinical practice does need to directly reflect pain science it should not be so pedantic as to confine practitioners to only what is known. However, professionalism can never be equated to a 'green light' enabling unquestioned administration of any technique that is currently 'in Vogue'. Neither should it provide an open door for proponents of manual therapy (or other) techniques to attach 'evidence based' labels to their clinical approaches regardless of reliability and validity. Clinical management based on the growing biopsychosocial evidence allows practitioners to adapt and change their clinical reasoning and treatments to suit each patient's individual circumstances and thus provides a platform for the clinical practice of true 'therapist flair'.

Successful integration of scientific evidence into clinical practice not only requires a paradigm capable of incorporating perpetual change; it also requires honesty on the part of each clinician involved and genuine professional altruism. As this involves some very human qualities, the process is always open to unconscious and conscious misdirection during pursuit of personal, financial, professional or political gains.

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