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Feature article 1:

Acceptable paradigms 1 – The centralisation and peripheralisation phenomenon under scrutiny.

By Mick Thacker

Those of us who have their backgrounds in manual therapy have been exposed to numerous working models of how our interventions work and how we can explain to patients what is going on. Whilst this provides many advantages there are associated problems with respect to accuracy. The need to address these issues has been highlighted in the modern arena of evidence based practice and clinical reasoning frameworks. As a profession we have resisted the need to confront these topics and to alter our hypotheses and paradigms to those that have firmer support from the current literature. I believe that this is no longer acceptable. We have a duty to attempt to integrate research findings into practice and to look for the best explanation possible to explain our clinical observations.

One of the most widely accepted theories in musculoskeletal physiotherapy is the McKenzie Disc Model (or Derangement syndrome). I believe from my McKenzie training, reading, discussion and research that it is still suggested that:

1) Centralisation is a positive clinical symptom behaviour and
2) Conversely peripheralisation is a negative positive clinical symptom behaviour.

Proponents of this approach have taken this concept further and suggested that the symptom behaviour of peripheralisation is related to negative alterations in the mechanical behaviour of the disc whilst centralisation is evidence for positive mechanical effects, McKenzie wrote, "the pain of derangement occurs as a consequence of a change in disc shape with related malalignment of the mobile segment and its associated abnormal stresses".
(Mckenzie 1994 , 2003)

The correlate to this thinking is that repeated movements alter the disc's shape and this thus produces pain via peripherally generated nociceptive mechanisms or if the disc impinges on neural tissue that peripheral neurogenic pain may be generated. Whilst offering a simple explanation for the generation of pain the truth is rather more complex.

Pain is never truly mechanical it is always neurological, the process of transduction is the major function of primary afferent neurons and we know that the relationship between afferent activity and provoking stimulus is complex and modulated by many factors (Wall 1996). Modern thinking warns against simplified/crude explanations of pain behaviour as the relationship between pain behaviour and movement is at best weak and at worst intangible.

Many therapists have been educated to believe that we fully understand how mechanical forces result in pain, this is unfortunately not the case, many of our preconceptions have no support or are refuted by the literature (Wall 1999, Zusman 1998). One basic problem is that

many therapists tend to extrapolate from work that looks at mechanisms involving cutaneously generated pain. Recent advances have demonstrated that pain arising from stimulation of different tissues/structures has many different and unique (to that tissue) mechanisms (Woolf 1994). The transduction of mechanical stimuli into afferent neuronal firing is still poorly understood. (See Tanner et al 1997 for a detailed review). Most of the studies detailing mechanically produced pain highlight the requirement of pre-sensitisation of the mechanically sensitive afferent by exposure to inflammatory mediators (Kushlich et al 1991, Nygaard et al 1997, Siddell & Cousins M.J. 1997, Aoki et al 2002)

But does this fact have any relevance to the disc model proposed by McKenzie? Is it important for us?

I believe so, many recent (and not so recent) studies report that damaged discs release a myriad of pro inflammatory chemicals including and perhaps most importantly growth factors, cytokines and matrix metallo-proteinases (Hasue 1993, Kawakami et al 1994a&b, Olmarker et al 1997, Kikuchi et al 1998, Olmarker & Larsson 1998, Aoki et al 2002, DeLeo & Winkelstein 2002). Their release has been repeatedly linked to the generation of pain and mechanosensitivity following disc injuries (Kushlich et al 1991, Hasue 1993, Kawakami et al 1994a&b, Olmarker et al 1997, Olmarker & Rydevik 1997, Olmarker & Larsson 1998, Aoki et al 2002, DeLeo & Winkelstein 2002). An interesting feature of this phenomenon is that contained and un-contained disc lesion actually produce a different repertoire of chemicals (Nygaard et al 1997).

The question here is how can this be incorporated into the disc model of Mckenzie?

Some may say easily and be tempted to see this as support for the work of people like Donelson et al (1997) who have demonstrated that contained and non contained disc show different responses to repeated movements. The problem here is that such simple reasoning would rely on these chemicals having effects limited to local tissues/structures. The truth is that they do not (McMahon et al 1993, Hopkins&Rothwell 1995, Hanai et al 1996, Kawakami et al 1994a&b, Cavanagh et al 1997, Olmarker et al 1997, Kikuchi et al 1998, Olmarker & Larsson 1998, Colburn et al 1999, DeLeo & Winkelstein 2002).

Several of these chemicals are known to cause central changes that are linked to the production and maintenance of pain. The main effect of these chemicals is to produce alterations in the sensitivity of the dorsal horn (McMahon et al 1993, Woolf 1994). (See also Gifford (1998) for detail re the processes involved)

There are several clinical manifestations associated with central sensitisation, but the key one in terms of this discussion is the capacity for changes/spreading of receptive fields.

Receptive fields are basically the areas of tissue that a single or group of sensory nerves look after. Charts of dermatomes and peripheral cutaneous nerve supplies are examples of theoretical large-scale receptive fields. Smaller fields exist for every afferent fibre (PNS), projection (Dorsal Horn) and receiving (CNS) neuron; and fields are not restricted to skin. Unfortunately most people believe these are anatomically fixed entities. The fact is that they are not, they represent a dynamic "snapshot" of the function of the sensory processing and scrutinising systems of the nervous system. That is any change in the perception of afferent input, or even the suggestion of input will alter the size and sensitivity of the receptive field. Put more plainly thoughts and feelings can change the size, spread and sensitivity of

receptive fields i.e. alter what many people believe are fixed entities. These changes can be measured in terms of changes that occur in thousandths, hundreds and tenths of seconds as well as minutes, hours, days and months and are easily manipulated by movement (physical) and suggestion (psychological).

Insert figure here

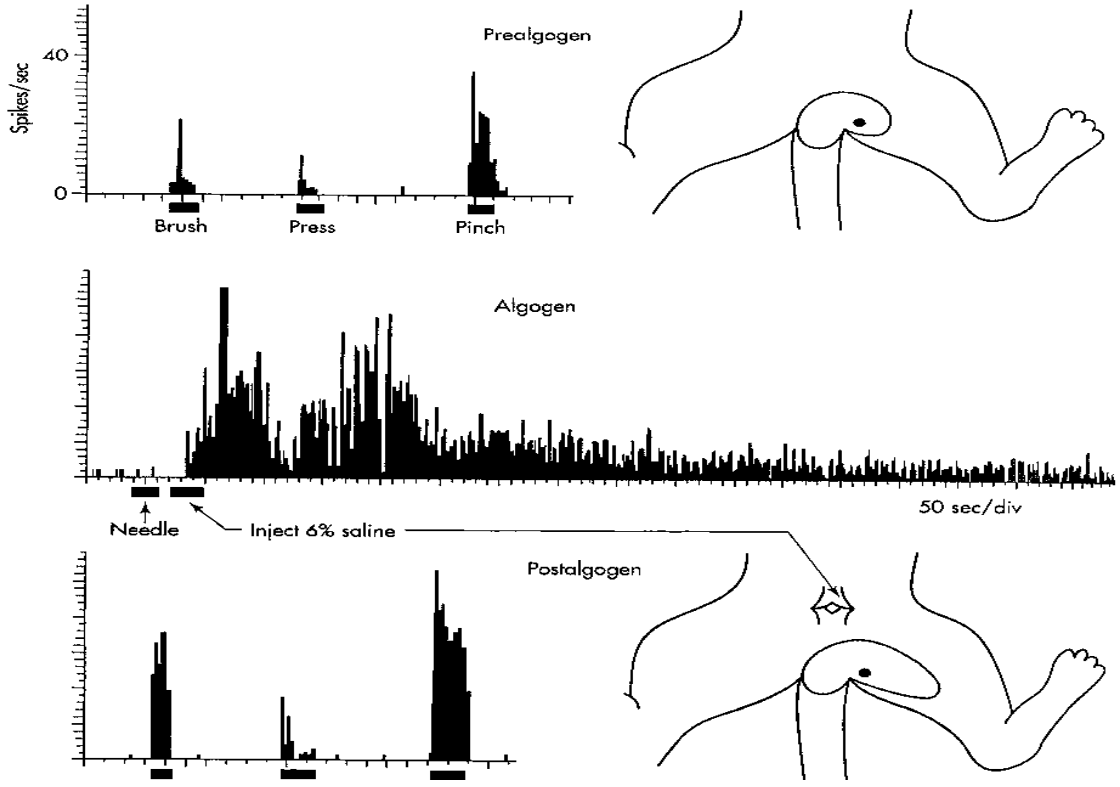

Figure 1
Modified from Gillette 1995

Figure 1. demonstrates this effect and I believe shows a clear example of peripheralisation. There are several points to note

1) The receptive field in the top diagram (resembling a "Pac man") in the upper diagram changes its shape and extends down (to now resemble a "Clanger") into the leg following a nocicpetive event.

2) The nociceptive event (in this experiment) driving this process is the result of hypertonic saline infused into the facet joints - nothing at all to do with the disc!

3) Following sensitisation previously non-painful stimuli are now pain promoting (brush).

The importance of these points is

Peripheralisation involves as alteration in CNS processing and has to be understood in these terms. As stated the above alteration in the sensitivity dependent receptive field expansion was produced by an experimental paradigm that did not involve the disc, an essential finding in terms of this account. Kellgren (1938 & 1939) and Hockaday & Witty (1967) demonstrated years ago that nearly all the structures of the lumbar spine (and in particular those posterior to the facet joints) are able to produce buttock, thigh and leg pain in many cases that extended into the foot.

To state the obvious; if a patient presents with pain that is made worse by, for example, flexion and as a result the pain extends either into or further down the limb then even the most uninformed of us could suggest either that this movement be carefully limited or that the opposite movement may help. Sticking with a purely mechanical (narrow) view we could suggest that stress is being removed from an irritated tissue rather then the disc changing shape.

Any structure that is sited posterior to the zygoapophysal joint will be tensioned by forward flexion. Thus any 'posterior' tissues that are damaged or exposed to algogenic substances will produce a nociceptive input to the CNS and have the potential to result in an alteration in peripheral receptive fields similar to those seen in the above experiment. Further, any normal afferent barrage that inputs into a sensitised nervous system has the potential to produce similar alterations in symptom behaviour.

I believe that this sort of information seriously undermines the suggestion that alterations in disc shape/mechanics are responsible for the behaviour of symptoms in the so-called derangement syndrome.

Pain is modulated by both tonic (on going) and phasic (recruited during painful experiances) endogenous mechanisms. These include both physical and psychological stimuli.

Responses to movements perceived as helpful (including those erroneously seen by therapists as reducing the derangement) may be exerting their effect by altering the balance between the afferent (pain producing) and the modulatory (pain relieving) systems.

I believe that this perspective has received little attention because it is not something the organised bodies of the McKenzie institute or McKenzie himself has encouraged. McKenzie wrote, "Our treatment methods must be directed at improving the pathologic condition rather

than modulating pain" (McKenzie 1995). I am not sure that the pathologic condition he refers to has actually been proven as the source of the symptoms that his approach attempts to fix!

The basic message I have attempted to convey is that the behaviour of pain is an expression of the nervous system and is dependent on the balance between afferent input/central processing and modulation of pain. It is important to remind the reader that both sides of this equation are as susceptible to manipulation via the mind as from physical stimuli such as exercise.

So far in this account I have accepted the basic premise that centralisation represents a positive clinical phenomenon. I believe however that this may not be as clear cut as originally thought/proposed. Practitioners often tell patients that even if the pain in the spine is worse then as long as the peripheral pain is reducing/gone then they can consider that as a postive outcome of treatment.

Marshall Devor has suggested (Devor 1991 & 1999) that local (either central or unilateral) back pain may be the result of dorsal primary ramus entrapment. Recollection of the anatomic course of these nerves would suggest they are vulnerable to compression during extension like maneuvers. Could it be possible that the increased back pain noted and which is often difficult to relieve is in fact, a neuropathic phenomenon?

In the last edition of PPA News Louis Gifford suggested that extension is also potentially dangerous as it may contribute to a stenotic like syndrome. I believe that these two pieces of information are important as they promote a new aspect to our reasoning that involves the assessment of risk during our interventions. We must all be prepared to undertake a cost (i.e. danger to the patient) benefit (i.e. helpful to the patient) assessment. It would be a shame if we continued to promote approaches that involve potential risks on the basis of a perceived benefit that is based on non-rational and out dated reasoning.

Conclusion

The aim of this brief account was to challenge some of the widely held beliefs about a well established model of pathology in musculoskeletal medicine. Rather then an attack on the Mckenzie system, this account attempts to highlight a common problem in the wider world of physiotherapy. That is the continued (long term) acceptance of a theory despite contradictory/alternative evidence from modern science.

This is a problem not limited to physiotherapy, Pat Wall once said:

“ it is nearly impossible to replace a well known simple scheme with a more complex one.”

I believe that this applies directly to the case of the Mckenzie disc model and the subsequent pain behaviours it is thought to produce.

Rather then being destructive this account is motivated by a need for all of us to assess our practice in the light of current knowledge. I believe that it demonstrates the limitations of thinking in one dimension (i.e. biomechanical) and calls for broadening of approach to encompass modern neurophysiology and pain science.

It also raises the question of the need to make cost-benefit analysis to assess risk in patient management.

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