**Editorial Reply following responses - by Louis Gifford: published in PPA News 15 June 2003.**

# Memes, dreams and dualism … the flexion-extension debate and beyond.

I would like to thank everyone who has written in regarding my last editorial. I have clearly, and not surprisingly, upset the Mckenzie teaching faculty and their responses and counter arguments are reproduced in full here [see pages….].

It is important to state that a great deal of what has been said and put on paper by those who have responded is hugely valuable. Time needs to be taken here because in the undercurrents and eddies that lie between the tension created by the main thrust of the arguments, there is a great deal of agreement and commonsense. Note for example Peter Ward's letter, in particular the last paragraph [page ], and a great deal of Julie Shepherd's editorial [page ]. Things are clearly changing and those who are involved are aware of the need for change.

There are two sides to me on the debate I raised in the editorial: One that was (and is still) passionate, as well as somewhat confrontational (and I apologise for that). The other, probably much more useful, is better reasoned and more considerate.

## Firstly: The passionate -

One that goes, great, good, I believe it was needed, a bit of shock and awe - especially if it starts debate, gets some alternative hypotheses and reasoning, widens existing hypotheses, and sets some reasonable challenges down on paper that could lead to the productive evolution of ideas that are good for us professionally.
We can't all agree and it's normal healthy science to battle, bitch and barter. Go to any scientific meeting or conference to witness this.
Since the editorial I've had plenty egging me on to 'have a go at 'Maitland', have a go at 'muscle imbalance' have a go at 'visceral mobilisation', have a go at 'cranio-sacral'….. all, notably, in my view, are treatments that seem to be dominated by a focus on pain and pain relief and minor physical impairments at the expense of functional confidence and a bigger picture. But, shouldn't we be asking, in the narrow sense of pain relief results…, why a treatment might work for one 'patient – therapist' and not for another? Julie Shepherd, the editor of the Mckenzie Institute Newsletter has queried this discrepancy too [page ]. How might it be explained? What appears rational and works for you may not for the next person. Why is that? Doesn't this very observation beg us to look for alternative explanations for the underlying mechanisms of treatment for pain?

If we all want to unite (I'm assuming we do, but might be dreaming here) we have to look for common ground and be prepared to consider alternative explanations for our observations. Then we can move on. I believe that the more we avoid (or reject) discussing pain processing and modulating and the more the current models for explanations of mechanism *and treatment success* remain doggedly tissue bound the longer the divides will continue and uncomfortable pressures remain. I try and argue a little more of my case again below, a great deal more could be added. Mick Thacker's 'Acceptable paradigms 1 – The centralisation and

peripheralisation phenomenon'[page…..], adds his useful and supportive perspective to this challenging debate.

A major problem in accepting pain mechanisms and modulation as a core factor to explain why treatments work (and don't work) for some and not others is probably because it is undermining and uncomfortable and requires massive shifts to be made. David Butler is the only well known international figure I know of who has comfortably got up on stage and admitted that much of what he taught was based on false, challengeable and probably quite wrong assumptions and he openly changed (Butler 2000).
There is a still a huge general need for 'shift', and change is always going to be frustratingly slow. Reasoning pain in all its dimensions is one thing, (it's tricky, but exciting!), adding and considering *function* is the other.
In reflecting on what I have witnessed since I trained (1981) is the observation that a great many therapists devote most of their treatment time to constantly focusing on and asking about, listening to and trying to change pain (note some of the dialogues transcribed in Kerstin Lüdtke's article [page…]. We all basically go on courses until we find the one system that fits our beliefs, works best for us as well as integrating bits of some others occasionally (see Peter Ward's letter page… for example). Pain treatment and pain 'attention' is often at the expense of/dominant over, function and often ends in a passive approach (creating reliance) at the expense of an active one (creating independence).
Function can be two things:

- …something to muck the pain improvement up (best be careful/avoid therefore),

Or, astonishingly, working in almost exactly opposite way when therapist-with-patient adopt a *less tissue fearful* stance and *reactivate* using the best cognitive behavioural principles (for prevention of chronicity, I admire Linton's work and impressive results: see (Linton 1996, Linton & Bradley 1996, Roland 1996, Linton 1998, Linton 1999) …. is…

- …probably the single most important and useful pain inhibitor there is.

So, why did I target the Mckenzie system? The main reason is because it is in every physiotherapists head who has been awake in their training or working for the last 20 years and its simple straightforward basic rules are easy to pick up and quick to pass on. Yes, like a virus, or in Richard Dawkins' language, a 'meme' (Dawkins 2003). The 'Mckenzie' principles are in all of us all over the world and they have become 'accepted truths'.
It is pleasing to hear that things are trying to change within the Mckenzie faculty (see the letters). A good deal of what Robin Mckenzie taught has been and still is very useful – he was an innovator and in the early 1980's provided a hugely welcome relief in a world of passive therapy madness that still lingers on today. Mckenzie offered self management and patient empowerment, but there are two things that greatly trouble me and I feel are here to stay for a long time yet:

**1.** The 'must' rules/viruses/memes that promote/imply that in the vast majority of presentations, extension is safe and flexion as dangerous. Sorry, but the 'must do' rules are there, printed in the manuals, the self-help books on the shelves of bookstores and the clinicians text and they have been taught and been spreading into the everyday fabric of physiotherapy for many years. I look forward to seeing the changes made in the new text and the current work books and hope that there have been significant changes.
I have pondered whether or not I should do this, but examples are helpful to see what I'm trying to get at (I'll be slammed again I expect). There are many, here are a few that concern

me from a 3-4 year old level A Mckenzie manual (stay calm, this is nothing to do with being up-to-date, it's about information *taken as truth* and strongly embedded in the current physiotherapy culture and consciousness):

- Page 18 – Derangement two …. 'Central or symmetrical pain across L4/5. With or without buttock and/or thigh pain. With deformity of lateral shift. Major postero-lateral disc disturbance. Progression of derangement two or three – may be converted into derangement six….
- Reduction of derangement day one (page 21): Commence with lying prone, lying prone in extension, extension in lying, extension in lying +SAG. Instruct to maintain lordosis at all times, must sit with lordosis and use lumbar roll. May benefit from supportive night roll.
- Page 41 – general instructions when in acute low back pain….**Sitting –'**when in acute pain you should sit as little as possible, and then only for short periods. At all times you must sit with a lordosis. Therefore you must place a supportive roll in the small of the back'….etc. For the whole page is advice to avoid moving out of lordosis at all costs and to avoid a whole variety of activities…e.g. 'when in acute pain you should avoid activities which require bending forwards or stooping, as you will be forced to lose the lordosis….' 'When in acute pain you should avoid lifting all together…'

This is teaching physiotherapists and hence patients fear avoidance, totally unreasonable fear avoidance, just like, or very similar to, my 'mischievous' criticisms of Peter Ward's case histories in my previous editorial which I highlighted to serve to demonstrate the ways in which we subtly, though not necessarily intentionally, produce fear of movements. If we are not aware then we cannot change. Some may not wish to change. Fair enough! A key issue is the subtleness with which beliefs and attributions can be transferred and picked up – argued again later in this piece..

I know of no other system within recent physiotherapy management of back pain that has been so dogmatic or so prescriptive with diagnosis and advice. To me this is no different from Dr's advising rest or putting a plank of wood under the mattress for all back pains. Once 'red flags' have been triaged, we need to be peddling memes/viruses of normality not pathology and structure based fear.

**2.** The reliance on wholly structure based models, particularly the disc derangement model and the trust and belief given to the 'hear and now' pain response when physical testing. The fear of the disc damaging further and damaging a nerve by flexing 'meme' is, I believe, at the heart of the advice sighted above.

## Secondly: The reasoned and considered: -

My other side is slower and gentler, but probably bound to be better in the long run. One aspect of the cognitive-behavioural strategy is to be non-confrontational, to confront clearly results in a great deal of time spent on defence of the position held by the confronted, rather than on shifting (see May et al letter)! Cognitive-behavioural approaches to obstacles and barriers try to provide challenging and alternative material that allows the individual to *consider* a sound balance of evidence and hopefully then make better informed decisions based on this. What is required is being open to listen to all sides of an argument, to new hypotheses and then decide – rather than being rigid. In recent years I have always started my lecture programmes with a simple comment – 'this stuff is my interpretation of what I have read, have seen with patients and how it makes sense to me… it is not set in stone, it is challengeable, it needs challenging, its good to challenge, and what appears rational and

reasonable now may be shown to be quite wrong in the future. Take what you like and leave what you don't like.'

We are working in times of great change and sometimes uncertainty. Think that we all are actually witnessing, even being a party to, the dramatic dismantling of the centuries old medical model and playing a part in the reconstruction of another model whose ramifications are likely to endure for a very long time to come.
Patients and the pain in our culture have never seemed so complex. The ways in which new research material is dealt with by clinicians can sometimes create a great deal of discomfort and vulnerability. The outcome, I believe, is likely to be far better than anything that we have been taught so far. Struggle, yes, uncertain, yes, exciting and challenging, yes!

We are living in times of alternative and better hypotheses and as a consequence much of the material and advice that we give needs revisiting and challenging. There are two pieces in this Newsletter that are of great importance here because they give more examples of unnecessary fear-avoidance. Firstly, Vicki Harding has written a fascinating account about RA and knee pain in relation to traditional Japanese settings where kneeling is commonplace [page ], and secondly, with Anne Daykin [page ] our new scientific officer [page ], has highlighted the need for the formation of a 'health information' group within the PPA. They site information given for osteoporosis.
For those of you who work in outpatients departments, take a look around your waiting room and department and scan the posters and information provided for patients and assess the quality of information given. A great deal of it that I see on my travels promotes the broader 'structural fear' meme I am trying to highlight here. For example see the 'Adam Rouilly' 'Spine disorders' chart!

I would like to make a few more 'response' points:

**1. My perspective on extension** has been criticised by May et al**. [page ]**
As per my editorial in PPA News 14. I stick to my points wholeheartedly. To reiterate, it's a normal movement that should be no more scary than any other and gradually recovered just like flexion should be and, I would argue, alongside/at the same time as other movements - hence my 'twisted ankle approach' to back pain = all movements from the start – some are going to be harder and some easier to get back and do clearly, but start with them all, do whatever can be done, and move on at a rate the patient and their problem/pain can best manage (and play down/avoid pain talk as much as possible!).
There are plenty of considerations though and I don't think they should be ignored. For example, the effect of spinal movement on nerve roots and normal and sensitised nervous tissue is long overdue for review and much wider inclusion in our clinical reasoning. Its very modest inclusion in Adams, Bogduk, Burton and Dolan's otherwise brilliant book 'The Biomechanics of Back Pain' (see page 166-7) – reviewed by Sue Mickleburgh [page….] is an example here.

In brief, and simplistically - flexion pulls/elongates nerve roots and extension tends to compress them. Nerve root pain can be very hard to stop once it has been started and it often starts hours or days after being injured (Devor & Seltzer 1999). As argued in my editorial, I think repeated extension (or any other repeated movement), into pain, to end range and beyond in acute or chronic pain is unwise/risky action.

My proposals and all the 'supporting' references in relation to cervical nerve root pain, its presentation, behaviour and assessment, including discussions of nerve root compression tests and protocols are presented in:
Gifford L S 2001 Acute low cervical nerve root conditions - symptom presentations and pathobiological reasoning. Manual Therapy 6(2): 106-115

A similar article relating to the lumbar nerve roots and root compression testing is in preparation. I have already illustrated and written about the mechanics and effects of lumbar movement on nerve roots in relation to flexion and extension in:
Gifford L S 1997 Neurodynamics. In: Pitt-Brooke (ed) Rehabilitation of Movement: Theoretical bases of clinical practice Saunders, London 159-195

From my experience teaching courses on nerve root pain and its management, I also believe that there is a great need for better inclusion in therapist education in relation to the following:

- The annulus bulges backwards in extension – effectively narrowing the radicular canal and foramen areas. The superior facet moves forward towards the bulging annulus further compromising the area. The greater the degenerative changes the more marked the effect. Flaval ligament and retro-dural fat-pad compression is also involved here (see Penning 1992). In some patients with degenerative changes, dents can be seen in nerve roots that have been created by the annulus and facet joint.
- A disc bulge, herniation, prolapse or protrusion produces a space occupying mass and therefore has a potential stenotic impact on the foramen, radicular and/or spinal canal. Some may be transient….see next point.
- The natural history, clearance and recovery of extruded disc material (e.g. (Saal & Saal 1989, Saal et al 1990, Saal 1996).
- Herniations, bulges prolapses, protrusions are likely to have biomechanical impacts on nervous tissue, both in extension and in flexion.
- The excitability of nerves following nerve injury and.. as discussed and referenced in the Gifford 2001 article above. David Butler's recent book (Butler 2000) is another resource here too.
- Relevant to misunderstanding by May et al letter [page ] and most others here - clarification of peripheral neurogenic pain and radiculopathy. A great many nerve root pains do not necessarily have signs of abnormal nerve function/loss of competency. Pain can have its origins in peripheral neurogenic mechanisms without loss of gross and clinically obvious nerve competency (ie. Radiculopathy).
- Effects of inflammation, oedema and ischaemia on nerve tissue within the radicular and intervertebral foramen. This adds a more physiological/chemical dimension to reasoning pain from a tissue perspective. Research findings into the effects of movement on the chemical and fluid environment in the nerve root radicular canal and exit region would be of great interest.

**2. The importance of clinical observations and open reasoning:**

May et al strongly criticised me for being un-referenced and anecdotal.

The piece I wrote was an editorial – in it I openly admitted it was my opinion/my observations (e.g. Geoff and the effects of scepticism), I asked questions, made some suggestions and invited/wanted comments. I put my case, I admitted I might be alone and I apologise if my tone was offensive, I was passionate.

My experience, knowledge and scepticism makes me want to come up with some better explanations.
My unease here is with the suggestion that clinical observation and hypothesis making is a lowly item that's worthy of insult. Clinical observations often differ from research observations. The answers that researchers get are often dependent on the style and form of the research questions used and the way researchers listen and record.
My hero, Charles Darwin said:
*'False facts are highly injurious to the progress of science for they often endure long; but false hypotheses do little harm, as everyone takes a salutary pleasure in proving the falseness; and when this is done, one path toward error is closed and the road to truth is often at the same time opened'..* (In Ramachandran & Blakeslee 1998 pxvi).

In my editorial, 'Geoff' was just one clinical observation that made me think differently and made me question what I had been told should happen – just like Robin Mckenzie's 'Mr Smith' gave him an idea, and just like centralisation stopping working for me when I had 'lost faith made me sceptical.
Clinical observation is what has lead to some of the greatest discoveries in science and medicine (e.g. see Le Fanu 1999). Clinical observations often lead to the generation of productive research questions – and some daring research has often lead, usually with great resistance and difficulty, to the smashing of long held beliefs imparted by those in respected positions– read Lewis Wolpert's 'The Unnatural Nature of Science'(Wolpert 1992) and 'The Hungry Gene: The Science of Fat and the Future of Thin' (Shell 2002) for examples and discussions.
My humble clinical observations continuously make me think that there must be many more and somewhat better explanations than those on offer and that have been offered.
What I was taught and told about musculoskeletal pain presentations, and the descriptions I still read in textbooks compared to what I see and listen to every day – seem to be a sad travesty of what it is really like for the patient. Am I being unreasonable to assert that medicine and physiotherapy, are guilty of continuously squeezing a patients pain experience into the anatomy and biomechanics that we feel comfortable with? .. and not really listening?
The tone and style of the May et al letter convinces me that I am right to continue to speak out. There is great cannon power in 'proof' and mass referencing. My advice is be wary, be sceptical, read carefully and be aware that there is often a lot of evidence from other perspectives that hasn't been screened and put forward or even considered [see Mick Thacker article page ]. It's not difficult to amass references that support your observations and beliefs, as I have a little here and in the articles cited too.
In a letter to his 10 year old daughter, the evolutionary biologist Richard Dawkins wrote (Dawkins 2003). : … 'I want to move on from evidence, which is a good reason for believing something, and warn you against three bad reasons for believing anything. They are called 'tradition', 'authority' and 'revelation' . It is a powerful and useful letter and well worth reading.

**3. Mind-body separation …. 'Bamboozling pain' ….. 'if it works do it' ….. … 'it's mechanical pain' …. 'the disc is the best explanation'…..'we avoid leading questions'…..**

The mind-body separation problem with pain still seems to be misunderstood – for example: - 'its either tissue or its psychosocial'… 'if it's acute it's tissue, if it's chronic it's psychosocial', … 'it's tissue or it's central….', 'its mechanical pain' etc.

A tissue gets injured and its nociceptors fire. The injured tissue goads its nociceptors to selfishly scream the message 'me, me, me look after me' (could easily be the disc, or a nerve, or both, or any other structure)….but the nervous system may have other ideas:-
If we look at the research on skin where individual neurons can be recorded, the more threatening the force/chemical or temperature used on it the more nociceptors tend to fire(e.g. Raja et al 1999). The increased barrage of impulses arrive at the first synapses in the dorsal horn of the spinal cord and, like it or not, get modulated – whether acute, sub acute, chronic or 'mechanical'. Modulation involves gating and gating allows or prevents the pain message from proceeding further to consciousness. Nociception needn't hurt, might hurt a little or could hurt a great deal for a very long time. It depends on how the nervous system deals with it, whether or not it decides to give it a chance. Gating occurs in all pain states and is powerfully dependent on rapid descending modulatory currents from the brain (brain here includes the mind and conscious 'you' with all your thoughts and attributions). Some currents enhance pain (causing hyperalgesia) and some dampen pain producing analgesia (Melzack & Wall 1965). There are dedicated pathways relating to analgesia, anti-analgesia and hyperalgesia (Maier et al 1992, Fields & Basbaum 1999). What we are thinking, doing, having done to us, what we are involved in, what we believe about the therapy and the therapist, our past experience of therapy, what we have heard about the therapist from a neighbour …. all affect the form of the descending modulatory currents and the ultimate impact of peripherally derived nociception. This means that two people with exactly the same injury and the same amount of nociception may experience completely different pain responses – in any setting including the therapeutic one ((Melzack & Wall 1996, Wall 1999).
ACUTE SO CALLED 'MECHANICAL PAIN' IS NOT IMMUNE TO MODULATION FROM YOU AND YOUR BRAIN/MIND!
The way the therapist sets up the treatment session – the atmosphere, the explanation, the empathy, the talk the reassurance, the threat, the fear, the anxiety, the subtle suggestions, the enthusiasm, the type of movement selected, the firmness or gentleness of the input etc. etc. – all act together to modulate what is coming in from the periphery. This is bamboozling at its best (i.e. helpful and pain relieving) or at its worst (for example distorting and dirtying attempts at a pure research investigation of a pain therapy!).
In 1990 Donnelson et al managed to get a remarkable 85 - 90% of patients with acute (pain less than 4 weeks) and subacute (pain for 4-12 weeks) pains to 'centralize'. In 1997 Donnelson's group managed to centralize 50% in a chronic pain group.
Bamboozling is not a criticism, it's making excellent use of modulation/gate control. Thus, good 'pain' therapists are good at bamboozling pain in the direction they want it to go – they are usually enthusiastic believers and confident and comfortable in what they do. The requirements of the patient are that they have to be bamboozleable! This stance is supported by the findings that psychosocial factors are the strongest predictors of treatment outcome. Be a believer, be optimistic and your pain is more likely to do what it is told. Promote it! Tell the patient about it!
'Wrong' gating/modulating/bamboozling is how I would like to propose why centralisation and repeated movements don't work for me and why the explanation has to go beyond a naïve tissue-only based explanation involving fluid movement and disc mechanics. If it was down to disc mechanics my skill at Mckenzie/MDT and centralizing pain that I had should not have disappeared, and it seems logical to suggest that MDT skills shouldn't need changing and evolving either – why would discs be any different in their responses in 2003 compared to 1997, 1990 and 1982? Last point on the disc – its virtually a dead structure – how can such quick changes in pain location and intensity be explained by a structure whose metabolic turn-over rate is at least 500 days and which has virtually no healing capacity – why does it bother to hurt? How on earth can it get better so rapidly in a treatment session or

a few treatment sessions?(see Gifford 2002 for further discussion and references). Perhaps one of the best things about discs is that when material gets extruded there is an excellent clearance mechanism that takes anything from 3 months on to do its work (see some of the Saal references).

There are two important caveats associated with modulation, gating and psychophysiological effects:

- One this is that the effect of provenly *active/specific* treatments can be significantly dulled, nullified or even reversed by nocebo effects and conditioning. The way you are on the day influences your outcomes! Yes, you are right, my belief is part of my problem. But, think of the implications for those undergoing surgery (or repeated movement with me) … you better believe… and hope that your surgeon and his team are on form and optimistic too! (See the adrenaline example on page 67 of Topical issues in pain 4 to see what I mean (Noon 2002)
- The other is that central effects via gating modulation and other psychophysiological pathways are likely/do reach the periphery. The notion of gating within the tissues has already been reported (Reichling & Gold 1996), the effects of stress on hormones that control immune function and inflammation is well known (e.g. Sternberg & Gold 1997) and animal studies are showing that changes in central processing can have powerful peripheral effects. For example, if morphine is injected into brain stem areas known to dampen sympathetic outflow activity there is a significant reduction in experimentally induced arthritis in rats (Levine et al 1986, Coderre et al 1991).

The term '**Mechanical pain'** seems to infer that the tissues must be the cause of the pain and that pure tissue treatments are all that are necessary. I think we need to divide and expand our thinking – knowing a pathology, treating and correcting it is fine. Fine for curable diseases but terribly hit and miss for musculoskeletal type pain. Pain science says that if nociception results in pain with movement or mechanical force and the sensitisation of the movement - it is, and has resulted from a mechano – chemo – electrical phenomenon. I think the term mechanically *patterned* nociception/pain should be used – and with care too because even straight forward acute pain with good clear mechanical patterning is subject to abrupt change and inconsistencies (see last editorial and (Gifford 2001). Normal acute pain can hurt with one movement one minute, or in one position a test can be positive, seconds later or in a different situation or position it can be negative – this just cannot be ignored. (Sure different forces come into play and this needs explaining to patients)…. One minute a patient limps, in another environment or circumstance the patient walks normally. A patient can't bend standing but sitting can tie their shoes with ease… they can bend backwards standing up, but lying prone for 5 minutes and they can hardly get off the couch. They are comfortable walking for 10 minutes, get relief sitting but then have to move after 10 minutes … one moment extension type postures relieve the next flexion – these so called 'preferences' change all the bloody time!! An upper limb tension test produces exquisite pain early in range, a similar *active* movement produces fluid pain-free movement…. and so forth. Listen and watch, observe the discrepancies - please! Pain, *especially mechanically patterned* pain is processed differently all the time – it's normal, natural and in an evolutionary context, necessary, it's aided our survival and as therapists we probably need to make better use of it (see Gifford 2002 for full discussion).
Mechanical inclusions in reasoning *obviously must have a place* but the influence of fear and anxiety on physical findings need consideration too. Paul Watson's conclusions from his research on palpation findings need publicity (Watson 2002). For example, he states that in low back pain and in fibromyalgia patients, pain reported in relation to pressure or palpation

is influenced more by the patient's fear of being hurt or injured than by the current pain state. Cant we relate this to repeated movement testing, or any other test…? As I said, pain is slippery!

Some thoughts now about - 'We always use open ended questions and avoid leading questions and statements'…. follows on here. Is this a good idea therapeutically? Well, it depends what you are after, but, my answer might be no, not if you believe in the effectiveness of modulation and persuasion for pain as suggested here! The best bamboozlers adapt their language, touch and communicate to suit the patient, and ultimately the skill they offer is persuasive towards what is desirable. Witness therapists like Brian Mulligan at work – a great bamboozler! And I have my suspicions about Ron Donelson and colleagues too (this is not an insult unless you maintain a wholly tissue orientated perspective!).
Whatever you try an do with your questioning style, being unbiased is notoriously difficult, if not impossible. Your intentions and your beliefs are subtly conveyed to the patient, come what may. In chapter 2 of Topical Issues in Pain 4 – Nigel Lawes (Lawes 2002) gives some wonderful examples of beliefs of clinicians about the trial treatment effect, in single and double blind research protocols, being subtly transferred to the patient subjects and influencing outcomes (see particularly from the last paragraph bottom of page 49 and all page 50/51 to see my point). If you can appreciate this, then you have to appreciate that being unbiased in the clinical situation is unachievable and almost a silly objective.
Lawes' brilliant writing on the placebo, along with all the other chapters in the section have some really important messages for physiotherapy and for issues like those under scrutiny here.

As Nigel Lawes points out, **every treatment** (whether it has proven efficacy or not) has at least three components:

1. The specific biological effect of the treatment
2. The effects mediated by the patients thoughts about the treatment
3. Other non-specific effects such as spontaneous remission.

A wise stance, if you go along with this notion of "the therapist as gate controller/pain modulator/bamboozler" is that you can just as easily make someone worse as make them better. If you don't feel comfortable doing a particular technique – for example a grade V manipulation or a repeated movement examination to assess pain centralisation, as I don't, … don't do it, and don't feel guilty that you don't achieve the results that others do with whatever it is you have seen or learnt! Just because a guru can do it dosen't mean it will work for you. If you have never done it before and wish to have a go – try to use a graded exposure approach yourself – in other words gently and slowly at first if at all possible. If this isn't allowed or possible (e.g. grade V! and as far as I can see, repeated movement testing for centralisation effects too) – it may be best to not try, or not be surprised if you don't do too well. However, our natural bias towards seeing all our successes and ignoring non-successes and failures (Sutherland 1992), helps our optimism grow – so keeping practicing can be helpful! If it doesn't work for you, don't worry about it. Just because something works for therapist X doesn't mean it will work for therapist Y. Just as things must fit and be comfortable for the patient, so they must for the therapist.

So how would you feel if one day our Government guidelines for the management of acute back pain insist/advise you use grade V manipulations for low back pain management? Or more likely – the use of the Mckenzie/ MDT approach as is happening in Denmark and

?France? This could mean your funding or insurance financing be withdrawn if you don't comply. I resent the threat of this potential pressure being foisted on me. It shouldn't happen if the issues like those discussed here are taken into account and taught, but it will if explanations and underlying philosophies remain entrenched in hard tissue based mechanistic thinking. The plea is for multidimensional explanations and an understanding of pain treatments in the light of pain mechanisms and individual effects mediated via thoughts and beliefs as well as a plethora of effects generated by chemical and physical effects from within the tissues.

It is my opinion that a balanced appraisal and more widespread teaching of the full implications of pain mechanisms and an emphasis on the goal of the best possible 'fearless' function are what should be at the heart of all physiotherapy training. The dogged and widespread determination of powerfully sponsored and power wielding quasi-logical old-school orthopaedic and tissue based reasoning within medicine and physiotherapy means that I am not holding my hopes very high at the moment.

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