

Explanation of terminology and concepts in PAIN & PERSISTENT PAIN

Based primarily on available research, persistent, discordant +/- amplified pain may be best explained via diligent consideration of:

- pain neuroscience, neuroplasticity processes and differentiation of nociception, pain and sensitisation,
- the mind/body effects of placebo and nocebo responses.
- comparison of the biomodel and biopsychosocial models, and

This conceptualisation is elaborated as follows:

- **NOCICEPTION** is defined as activity in specialised peripheral sensory neurons known as nociceptors (C and Aδ fibres) which alert to potentially damaging stimuli by detecting changes in **temperature and pressure and injury-related chemicals**, and transduce these stimuli into long-ranging electrical signals that are relayed to higher brain centres. This is the **input** mechanism and is not actually pain itself.
- **PAIN** is a complex constellation of unpleasant sensory, emotional and cognitive experiences provoked by real or perceived tissue damage and manifested consciously by certain autonomic, psychological, and behavioural reactions. **It is a multiple system, emergent and protective output response.** Pain is essentially produced as a protective response based on the degree of danger perceived and is therefore highly influenced by any 'fear', considerations of threat to person or lifestyle, and suboptimal motivational environments.
- **Nociception is not sufficient or necessary for pain, nor is it proportionate.**

- **To put it most simply, pain is always generated by the brain as a conscious, protective output experience. It is not an 'input' to the brain. It will be reliably generated when the brain's perception of 'danger' is greater than the perception of 'safety'. DANGER = nociceptive input when present, perception of harm, damage, injury, informed structural faults and 'fragility', threats to person, security or entitlements, confusion, inconsistent information, contextual factors, poor support, past experiences and memories, being told 'there is nothing wrong with you' when you are in pain etc, etc. SAFETY = perspective, knowledge, reassurance, confidence, consistent information and PAIN LITERACY etc.**

- **Discordant, wider or persistent pain can be well explained by central (+/- peripheral) nervous system neuroplasticity processes +/- overt sensitisation** amplifying and perpetuating pain responses, often imprecisely, and this manifestation of the system's protective mechanism, once entrenched, is difficult to 'switch off'. In my opinion based on a very large number of case reviews and my clinical and personal experience, this explains poor responsiveness to management (including often even high dose opiate analgesia when used) and is a spectrum condition likely commonly present in many varying degrees at all times rather than an 'all or nothing' and time-based phenomenon. I acknowledge the limitations of personal perspectives and that current consensus definitions are paradoxically more linear.
- **Neuropathic pain** can refer to similar processes related to actual direct nerve damage or compression when evident; this term is also often applied confusingly to sensitised pain when no objective nerve damage is evident. Neuropathic pain is associated with more overt symptomatology (eg: 'burning', 'numbness' etc) and sometimes objective clinical signs. It seems to have greater potential for amplification and persistence which can still be disproportionate to the degree of demonstrable injury. **Interestingly overt mechanical nerve compression including canal stenosis can still be seen not uncommonly in asymptomatic and minimally symptomatic situations.**
- The whole process is also influenced by individual genetic, inflammatory and **biopsychosocial** factors; the latter seeming to provide greatest reliable prognostic value for outcomes and is **intrinsically suboptimal in situations involving perceived third party fault and/or compensation.**
- **An important aspect of the biopsychosocial model is that It is most reliably the 'context' of the injury and the management pathway that is predictive of outcomes and not the injury severity or degree of 'pathology' itself.**

- *There is extraordinarily little if any robust evidence correlating severity of investigation visualised structural / degenerative or constitutional changes and levels of pain and pain-related disability. I am unaware of a degenerative structural change that cannot be observed in asymptomatic or minimally symptomatic individuals. In fact the vast majority of degenerative 'disease' is in fact asymptomatic and well adapted.*
- *There is similarly no robust evidence, to my knowledge, that correlates progression of these changes to the clinical ability to predict future outcomes robustly, yet comments predicting outcomes are often made to patients based on these visualised changes.*
- ***A major flaw in the bio-model paradigm is the absence of a linear or temporal relationship between discovered changes and pain / pain related disability.***
- *Communication of the perceived 'significance' of visualised constitutional investigation findings is being increasingly recognised as having intrinsic **iatrogenic** (medical management causing adverse outcomes) potential via a likely '**nocebo**' construct.*
- *A definition of the nocebo is an inert substance that creates harmful effects in a patient. The nocebo effect results from a patient's expectations and perceptions of how the substance will affect him or her. Though they originate from psychological sources, **both placebo and nocebo effects are now known to be psychological and physiological.** The same is seen with negative 'fear-inducing' information commonly communicated in a biomodel based approach focussed on visualised scan changes.*
- *The aim of various interventions, particularly surgical interventions, is restricted to altering nociceptive and demonstrably neuropathic contribution to pain. These interventions have their own varying nociceptive and neuropathic contribution potential in addition to standard risks, which is a significant problem when pain is mainly due to discordance and neuroplasticity +/- overt sensitisation processes.*
- *Placebo responses are only relatively recently being researched in relation to surgical interventions with interesting outcomes Eg: vertebroplasty and knee arthroscopy. Surgery is a relatively powerful placebo construct due to the 'end-point' nature of this intervention, patient's perceptions based on expert advice given of something needing to be 'fixed', and subsequent compliant and focussed rehabilitation. Unfortunately, surgery does not qualify as a 'harmless' placebo and therefore consideration needs to be given to the fundamental 'first, do no harm' principle.*
- ***The available results of spinal fusion surgery in the compensable cohort reveal statistical results that are easily perceived as being less than what would be anticipated from a surgical placebo construct. Relevant sham controlled trials have not as yet been performed.***
- *The evidence in support of spinal fusion and disc replacement surgery is based largely on patient reported outcomes; to my knowledge so far these are via insufficiently independent 'lower level' studies, and problematic when compared with actual observed epidemiological outcomes well assessed by, for example, subsequent analgesia / management / implantable pain therapy requirements. There is very little support for such spinal surgery in systematic reviews.*
- *The compensation system process is very often viewed and blamed as a confounder to successful surgical intervention outcomes; in my opinion **this firmly supports** the viewpoint that it is the centrally processed protective pain output response which is variably, and sometimes minimally, related to structural factors that is the main problem. Otherwise the expert structural / nociception focussed intervention would produce an optimal and reliable result regardless of the context and the timing of implementation once the original anticipated injury 'healing' time has passed.*
- ***Unfortunately and paradoxically despite advances in investigation and intervention technology, and therapeutic options targeting nociception and pain in recent decades, chronic / persistent musculoskeletal pain and pain-related disability outcomes have increased significantly during the same period across age groups.***
