EDITORIAL

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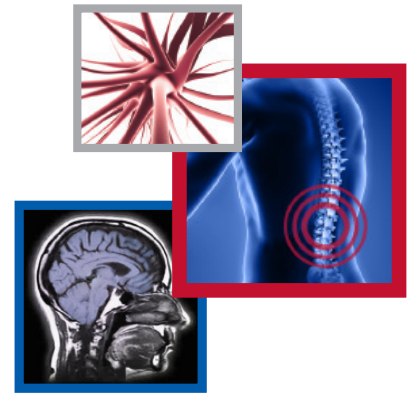

Teaching people about pain: why do we keep beating around the bush?

G Lorimer Moseley*

"We tend to endorse the complexity of the brain and its fundamental role in what we experience. Unless, of course, we are talking about pain."

A frank approach to interpersonal communication brings with it some challenges, but having to dig oneself out of a hole, created by strategically avoiding the truth, is not one of them. This frank approach is well suited to science – the scientific process requires us to pursue and report the truth, the whole truth and nothing but the truth. We do not tend to avoid aspects of the truth because we think that they are too hard for people to understand; for example, we do not avoid the amazing truth that the brain constructs visual experience from the information available to it, and instead suggest that the eyes themselves actually create visual experience and send the visual experience to the brain to be registered; we do not suggest that the ears capture words and bird-calls and racing cars and send those sounds to the brain to be registered. We tend to endorse the complexity of the brain and its fundamental role in what we experience. Unless, of course, we are talking about pain.

Some 25 years ago, Patrick Wall, as frank a communicator as any, lamented the trend towards beating around the bush when it comes to pain: "The labeling of nociceptors as pain fibers was not an admirable simplification, but an unfortunate trivialization under the guise of simplification" [1]. Of course, equating pain to activity in nociceptors is seductive – nociception and pain seem so tightly coupled. However, are nociception and pain really so tightly coupled? This issue was actually settled a couple of decades ago – there is not an isomorphic relationship between pain and nociception, nor between pain and tissue damage [2]. A very large amount of research has explored the multifactorial nature of pain (see [3] for a clinic-friendly review). Modulators broadly fit into one of three categories: prioritization, meaning and transmission/processing. Prioritization depends on the survival value of a nociceptive stimulus. Observational data abound; for example, the extensive work with military and civilian injuries – the soldier feels little pain until he is safe behind lines [4]. Experimental data have corroborated this – noxious stimuli do not hurt in cases of extreme air hunger [5] – and the pain threshold is higher after a bout of startlingly loud noises [6].

The second category – meaning – is, in my view, the most important to those of us

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working in the clinical pain sciences. Meaning is a very potent modulator of the relationship between nociception and pain. Indeed, one might argue that meaning is the critical determinant of pain, because if a nociceptive input is not evaluated by the brain as reflecting a threat to body tissue, pain would clearly be an erroneous output, serve no survival function and offer no evolutionary advantage. Again, anecdotal data abound – most strikingly, there are religious or cultural practices that involve severe noxious input, but no pain is reportedly experienced – and experimental data have corroborated this: a very cold noxious stimulus hurts more if there are explicit [7] or implicit [8] cues that provide credible information to suggest that the very cold stimulus is actually very hot, which would be more dangerous to body tissue.

The third category – transmission/processing – refers to both the well-established state-dependent functioning of nociceptive pathways and real-time modulation of transmission and processing by, for example, expectation [9]. Real-time modulation can involve neurally, neurochemically or humorally driven alteration of the response profile of neurones within the nociceptive neuraxis [10].

"...'explaining pain' ... as a therapeutic strategy leads to rapid changes in pain-related beliefs and attitudes and increased pain threshold during movement."

So, the mislabeling of nociceptors as pain fibers was indeed a trivialization, but was it really that unfortunate? I contend that one need only look at the huge burden of chronic pain to uphold a resounding 'yes'. Chronic pain is terribly costly to our societies – approximately US$1500 per person per year in the USA [101] – and to the individual sufferers of pain, who often descend into a spiral of increasing economic, social and personal disadvantage. The evidence that tissue pathology does not explain chronic pain is overwhelming (e.g., in back pain [11], neck pain [12] and knee osteoarthritis [13]). Yet we continue to avoid the truth that tissue damage, nociception and pain are distinct. I would go so far as to suggest that even the use of these erroneous terms – pain receptors, pain fibers and pain pathways – leaves the patient with chronic pain feeling illegitimate and betrayed, and leaves the rehabilitation team lacking credibility when they look beyond the tissues for a way to change pain.

Generally speaking, however, are these issues usually more than just semantics? Can one argue that it might be unhelpful to simplify things when we are talking about chronic pain, but it is fine when we are talking about acute pain? This suggestion requires us to adopt a different understanding of biology once someone 'goes chronic'. This is problematic, not least because those in chronic pain first had acute pain, and to change our story once they are chronic might give the distinct impression that we are clutching at straws. Indeed, by the time people progress from acute to chronic pain, our previous avoidance of the truth – our unfortunate trivialization – has dug a very big hole from which it is difficult to climb out.

All is not lost, however. There is an emerging body of literature that suggests that we can change the way people understand their pain. We can reconceptualize pain in a way that makes clear the distinction between tissue damage, nociception and pain. The bulk of the work in this area is guided by a model that suggests three phases of intervention:

- Provide evidence against the current (and inaccurate) conceptualization;
- Provide evidence for a new (and accurate) conceptualization;
- Test, confirm and finally embed this new conceptualization, such that it can guide behavior.

Each phase has its challenges. For example, the first phase needs to avoid being retarded by the cognitive defenses we all possess in order to guard our own views. That is, key conceptual challenges need to be 'snuck in under the radar', as it were. We also need to exploit methods to make our interventions memorable – to maximize the likelihood that they will 'stick'. For this, we need to engage emotional systems and use multiple media styles. Our group has recently tested the utility of using metaphors to induce a conceptual shift in the understanding of pain [14]. Metaphors can be described as understanding and experiencing one kind of thing in terms of another and are thought to provoke contemplation and increase the potential for reorganization of previous meanings. In short, simply giving people a book of short stories that are used as metaphors for key concepts in pain biology [15] led to measurable shifts in the knowledge of pain biology and in pain-related catastrophizing [14].

Much of the research into reconceptualization of pain has focused on the second phase of the above list – the provision of evidence for a new conceptualization. These experiments and randomized controlled trials show that 'explaining

pain' (see [3] for coverage of material) as a therapeutic strategy leads to rapid changes in pain-related beliefs and attitudes [16,17] and increased pain threshold during movement [18,19]. When integrated into a behavioral or functional upgrading approach, explaining pain is associated with better pain- and function-related gains than upgrading alone [19–22], and when intensive cognitive–behavioral pain management is preceded by explaining pain, the long-term outcomes seem substantially better [23].

Of course, the loftier goal here is to reconceptualize pain before people have chronic pain (i.e., when they have acute pain or, better still, before they have any pain at all). This will clearly require a team effort. I argue that we can start by truly taking notice of Patrick Wall's advice from 25 years ago and stop calling nociceptors 'pain receptors', nociceptive pathways 'pain pathways' and noxious stimuli 'pain stimuli'. These are erroneous terms. That is, let us not fool ourselves that the mislabeling of nociceptors as 'pain fibers' is an elegant simplification – we need only sit with a patient in chronic pain to see that this mislabeling is indeed a most unfortunate trivialization.

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