The British Pain Society

Pain News
2015, Vol 13(1) 24–28

SAGE

Reflections upon the pain experience – part 1: symptom validity and robustness of the pain diagnosis

Dr Rajesh Munglani *Consultant in Pain Medicine, Cambridge*

The great art of life is sensation, to feel that we exist, even in pain.

Lord Byron

Certainty generally is illusion, and repose is not the destiny of man

Oliver Wendell Holmes, Associate Justice of the Supreme Court of the United States from 1902 to 1932

A day in the life …

A not infrequent scenario for me is to see somebody in the pain clinic, who, having failed conservative therapy, turns up to find out if 'anything more can be done' in the pain clinic.

It sometimes feels more like an Undertaker's than a place of hope. By the time patients come to me, patients often also have a fairly firm view of what is wrong, as well as being more despairing.

I am told in no uncertain terms by the patient that the *real* reason for their sciatica is that the sacroiliac joint or facet joint 'keeps slipping out', and it is only through the skilled ministration's of a particular osteopath or chiropractor (usually) that the patient has been as mobile as they have been until now.

Additional diagnoses I am presented with include restricted cranial suture movement causing inhibition or blockage of cerebrospinal fluid (CSF) flow and dental mal-alignment, both causing chronic widespread pain or fibromyalgia.

Very smugly, I point out that there is little movement in the sacroiliac joint and that a dislocated facet joint would be intensely painful and sometimes extremely difficult to treat without operative intervention. As for dental mal-alignment, I remain to be convinced that it causes anything more than headaches in some.

Restricted cranial suture movement? I usually use *that* comment to come to a rapid decision that the belief structure of that patient is such that the sort of medicine I practice is unlikely to be of help.

How do we diagnose pain?

However, these encounters caused me to think about how certain we are about *any* diagnosis in a field where we have to rely primarily and fundamentally upon the testimony (if available) of the patient.

Unfortunately, we have made things difficult for ourselves, or more precisely Harold Merskey did in 1964 when he decided to define pain in terms of tissue damage in the well-known definition which was subsequently adopted by the International Association for the Study of Pain (IASP):

> *an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*. (Harold Merskey, *Pain 1969*: 6; 250)

This means, unwittingly, at times, all of us who work in the healing arts are likely at some point, if at all possible, to try and justify someone's experience of pain in terms of actual bodily pathology.

On this basis, therefore, we continue to seek out consensus or agreement when examining patients who clearly are suffering and distressed by the magnitude of their symptoms which they (assuming that they can) call 'Pain'.

Going back to the chiropractors, an interesting study showed that if you took five chiropractors and asked to examine the same patients with low-back pain, the kappa value for agreement about manipulating part of the lumbar spine was 0.47 (where a kappa value of 1 means complete agreement, 0 means chance agreement and –1 would mean precisely no agreement at all). This means, very roughly, that it was more likely than not that they *would agree only* less than 50% of the time, beyond chance, which segment to manipulate.[1]

One could say that I am being a little harsh, and in fact, it was almost evens chance that they might agree on a painful lumbosacral spine. However, the same study suggested that agreement that if it was thought to be either the L4/5 segment specifically or the sacroiliac joint, there was only a slight agreement of approximately 0.09 or thereabouts. At the L5-S1 level, the correlation was slightly better at 0.25, and of course, this is a common level for

spinal problems. This does not inspire one to confidence, especially if one is spending money and time going for such treatment.

But do doctors fare any better? In one study, they performed *worse* than physiotherapists when comparing inter-examiner reliability for low-back pain, but to be fair, the doctors had not worked as long together as the physiotherapists in the study.[2]

But there is an even more fundamental problem: what happens when the sign that you are looking for, say, a tender trigger point, also seems to be prevalent in the more or less asymptomatic general population?

In one study, physicians examining patients in pain reported that the prevalence of trigger points of active myofascial trigger points was 46% ± 27.4%.[3] In contrast, Simons[4] (of Travell and Simons Trigger Point Manual fame) noted that the prevalence of trigger points among fit healthy and young Air Force personnel was 54% in women and 45% in men.

What about something as simple as neuropathic pain? Some of the most eminent researchers in the field recently concluded,

> *We still lack gold standard of diagnosing neuropathic pain, i.e., there are no clinically feasible means, in the clinic or laboratory, to differentiate neuropathy with pain from a neuropathy without pain …*[5]

A well-respected reviewer of the epidemiology of neuropathic pain also commented as follows:

> *It was surprising that some articles did not provide a working definition for neuropathic pain as a starting point*[6]

before finally concluding in that same article that the incidence of this poorly defined entity of neuropathic pain was 7% to 10% in the general population.

Looking again at inter-examiner reliability for neuropathic pain reveals that agreement was associated with a kappa value of 0.8 which is good compared to consensus about pain in general which is about 0.5 in most studies; however, more importantly, clinicians *could not agree* on the severity of the neuropathic pain in an individual patient with a kappa value of only 0.3.[7] This has profound implications for which patients we treat and whom we do not, as we certainly cannot treat all 7% to 10% of the population who have neuropathic pain. Should we be exposing patients with only modest neuropathic pain problems to potentially very aggressive treatments, which may have significant morbidity mortality, for example, insertion of spinal cord stimulation or microvascular decompression. Even long-term administration of co-analgesics carries its own risks.

Diagnosis in complex regional pain syndrome and leaving patients in a diagnostic wilderness?

Dr Andreas Goebel, who is currently one of the world leading researchers in complex regional pain syndrome (CRPS), when asked about why he wanted to study this particular pain condition stated that it was because 'one can see something'. I understood what he meant, that is, to diagnose CRPS, one has to have, as well as pain, a set of signs as well of symptoms which include changes in sweating swelling temperature, skin texture, nail and hair growth and so on. Many of us will be very familiar with some of the more gross examples.

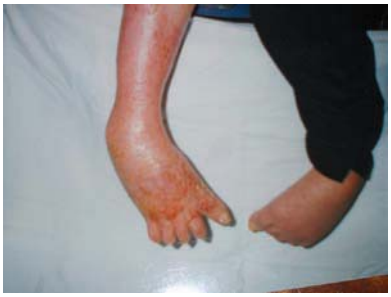
The problem is that many other conditions can give rise to a very similar clinical picture to CRPS including chronic infection, chronic arthritis, connective tissue disorders, erythromelalgia, compartment syndrome, crush injuries and even variants of neuropathic pain.

Goebel has significantly contributed to our understanding of CRPS as an autoimmune-like condition which may in many cases be triggered by a seemingly minor trauma (or occasionally no trauma at all) leading to development of a picture which, in many cases, is not too dissimilar to other autoimmune conditions, that is, soft tissue/arthritic process of inflammation. Because, the antibodies which seem to be activated in CRPS are not present all the time, it is the chance association of trauma with the transient presence of antibodies which seems to cause the onset of CRPS, which is why many other traumas, in the same patient, at other times may not give rise to the condition.[8–13]

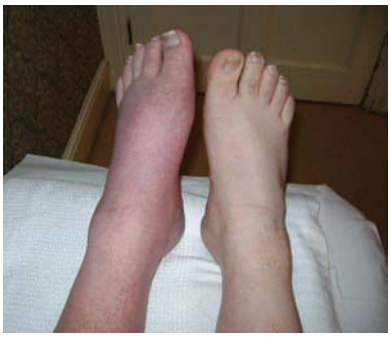
It is likely that within the next few years we shall have a biomarker, a gold standard for CRPS, possibly based around an activated bone protein or possibly the antibody in question, laying to rest much argument about how to define this disease process.[14]

However, at the present time, in the absence of any validated biomarkers, we rely upon clinical criteria, most recently redefined by Harden and his colleagues, which are known as the 'Budapest' criteria and have now been adopted by most clinicians and researchers in the field as the best way of diagnosing CRPS. These criteria are much more strict than the previous criteria of the IASP and Veldman to diagnose CRPS.[15] Unfortunately, despite their best intentions, the presence of such varied criteria has caused considerable uncertainty both for clinicians and patients.

It is important to emphasise again at this point that currently in the diagnosis of CRPS, we are relying on collections of clinical signs and symptoms, none of which are really specific and which can easily be due to other diseases because we still do not have a gold standard for a diagnosis of CRPS. With this inherent weakness in mind, a study in 2007 looked at clinicians' ability to diagnose CRPS using three sets of diagnostic criteria (the IASP, Bruehl et al. and Veldman et al.) based on patient reports



This patient probably has CRPS …



But what about this patient?

and physicians' assessments of signs and symptoms in 372 outpatients suspected of having CRPS. They found agreement between CRPS I diagnosis among the three sets was poor (kappa range: 0.29–0.42), leading to positive CRPS I diagnoses according to Veldman et al.'s criteria in 59% of patients. Using the less strict IASP criteria, a consensus diagnosis of CRPS was achieved in 72% of patients, and using the strictest Bruehl et al. criteria (which then formed the basis of the Budapest criteria), consensus diagnosis of CRPS was achieved in just 35% of patients.

In another study, the final diagnosis of CRPS showed poor clinician agreement with a kappa value of only 0.2. However, the application of Bruehl's (Budapest) criteria resulted in an increase in agreement between clinicians achieving a kappa of 0.38, but then *frequency* of CRPS diagnosis decreased from 73% to 43% in comparison with physician's own diagnosis. Thus, again stricter CRPS criteria mean more certain agreement between physicians and probably a more certain diagnosis, but importantly, this was achieved in fewer patients.[16]

Leaving patients without a diagnosis

Currently, the Budapest criteria of signs and symptoms are considered the clinical 'gold standard' for the diagnosis of CRPS since we do not have a specific 'biomarker gold standard'.

Thus, the increasing diagnostic certainty achieved by strict application of the Budapest criteria unfortunately has meant that many patients with pain associated with odd features of swelling temperature changes and so on have been left in a 'diagnostic wilderness' as they no longer achieve these stringent standards set for a diagnosis of CRPS.

Is this merely an academic point or is it actually something more profoundly troubling? Many patients need the 'dignity of a diagnosis' (a remark attributed to Bogduk in 1994) to validate their pain experience and many have now lost this. This causes acute distress to many patients who believe they do suffer with significant CRPS pain who now rightly feel that they are 'no longer believed'. In such a situation, medico-legally, a lack of a CRPS diagnosis may have very significant adverse financial consequences for a patient/claimant.

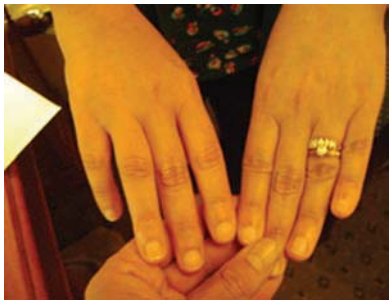
Unfortunately, it is not just disgruntled patients who we have to deal with, there is now a political and racial dimension that too needs to be addressed. It is now being recognised that the Budapest criteria were based around an overwhelmingly White Anglo-Saxon population. It does seem that even when the diagnosis of CRPS is likely to be certain, other races such as the Japanese may present with CRPS in different ways and by using the Budapest criteria they may 'miss out' on a diagnosis of CRPS.[17] The response of the Budapest group to this observation was to reject the suggestion that racially specific CRPS diagnoses should be set up as they were worried about the uncertainty it would cause clinicians![18] This obviously raises the question about what the role of the clinician is. Whom do we serve? If the tests that we all decide upon are actually shown not to be helpful and leave patients highly distressed and uncertain, then whose uncertainty should we tolerate? Ours, as clinicians? Or that of our patients?

On a more positive note, a recent paper showed that the use of thermal imaging to 'visualise' temperature changes in CRPS does seem to significantly improve consensus diagnosis in CRPS and may be particularly helpful in the medico-legal setting in my experience as seen in the example that follows.

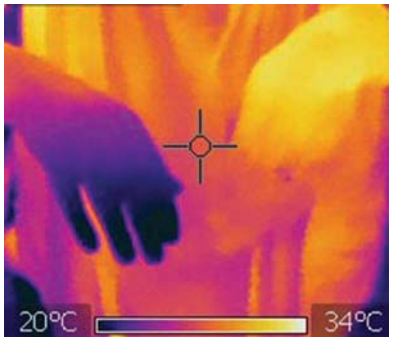
Criticism of the IASP definition

Again one wonders, therefore, whether there is any meaningful correlation between the presence and absence of *physical* symptoms and the presence of pain. We come back to question the validity underlying the implicit message of the IASP that 'pain represents actual tissue damage or is described in terms of such'.

Understandably, the definition of pain adopted by the IASP has come under



This patient complained of severe pain every time she used the dominant right hand; it swelled. It certainly felt different, but the photograph itself only shows possibly slight swelling on the affected side and one could wonder what the problem is. Really the photograph is not that convincing. Because she was no longer able to work as a highly paid professional as she previously did, the claim was for a very large amount of money.



Thermographic imaging, in this case, revealed that the temperature of the two hands were in fact very different, and this convinced the judge to settle considerably in her favour.

increasing criticism.[19] Wright argues that fundamentally of course no part of this statement can be assumed.

Wright goes on to ask the question 'what meaningful statements can be made about subjective (*pain*) experience?' and he continues,

> *The IASP's solution is to qualify an imprecise characterisation of pain's phenomenal qualities through an association with tissue damage … and an ability to recognise pain sensation.*

Wright argues further that another fundamental weakness of the IASP definition is that one actually has to report pain. What about neonates, those suffering from dementia and others who do not have the ability to communicate? On a positive note, he recognises that the role of the IASP was to assist clinicians examining patients in attributing pain to them – if that is what they felt was true – using terminology that would be recognised and accepted by other clinicians in the field, allowing treatment of the patient and pain, if possible.

Thus, objective physical external correlates of the 'internal pain experience' continue to be sought actively by many of us, not only early on but also years on into the pain experience of an individual.

Pain: a sensation versus a feeling and a clue to what is going on here

There has been an argument raging, in a meaningful sense, probably for the last 2,500 years about *whether pain is a sensation* (like touch is) or *pain is more of a feeling or emotion*.[20]

It is important to understand the difference. If we say pain is a sensation, then that means it is very specific and, for example, is completely separate from the sensation of say itch or the perception of cold or heat.

The second main theory is that pain is primarily a feeling or an emotion. Aristotle (384–322 BC) considered the heart to be the seat of feelings and understood the cognisance of pain to be the most important factor. He therefore argued that pain was an emotion. Not all the Greek philosophers agreed with him; however, his view prevailed at the time.

Galen (AD 130–201), a leading physician and Surgeon General of Alexandria, used experimental studies and disagreed with Aristotle. While Galen recognised that the brain was the seat of feeling, he placed the pain completely in the sphere of a sensation, that is, a distinct sensation that we distinguish, for example, from touch, temperature or itch, as indicated above. Avicenna (AD 980–1037), a renowned Muslim philosopher and physician, also recognised that pain can disassociate from touch or temperature and again proposed the pain to be an independent sensation.

Recognition that there are specific anatomical pathways for pain indicating pain must be a specific sensation just like touch is.

Very little progress was then made on this argument until the last 200 years when the exact anatomical pathways, that is, the somatosensory pathways, for pain have been characterised.

It is recognised that there can be, within these spinal cord and brain pathways, both amplification and indeed diminution of the specific pain sensations or interpretation of non-painful stimuli as painful. This modulation of the signal is the area where many pain consultants work. We spend our lives trying to turn down the 'amplifiers' within the spinal cord or brain when the system seems to go hay wire and not control the level of symptoms adequately. The concept of hyperalgesia (i.e. an ordinary painful stimulation being amplified up to something more severe) or allodynia (i.e. a non-painful stimulus being interpreted as a painful stimulus) comes from these latter studies over the last 200 years.

There is evidence that as pain becomes more chronic, the neural circuitry becomes more centralised in the brain and focuses on the feeling/emotional areas.

The emotional aspect cannot be forgotten because now that we have functional magnetic resonance imaging (MRI), we recognise that with time, pain shifts from the initial specific somatosensory circuitry (i.e. a very distinct anatomical pathway associated with pain) to the more emotional circuitry.[21]

That is, as pain becomes more chronic, it changes from a 'sensation' perhaps associated with more peripheral inputs to a 'feeling or an emotion', which is much more centrally driven.

Therefore, having spent the last 200 years outlining the specific peripheral somatosensory pathways of pain, we now are back to the original conversation whether pain continues to be a sensation based around somatosensory circuits or whether it is now more of a feeling or emotion based in the brain; the 2,500-year-old discussion continues and will be continued also in the next linked article.

Declaration of conflict of interests

This essay is loosely based on an invited talk that was given at a 9 Gough Square Seminar at the Law Society, Chancery Lane, on 11 September 2014.

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