What is Pain? I: Terms, Definitions, Classification and Basic Concepts

DR. ERIC J. VISSER, MBBS FANZCA FFPMANZCA
Pain medicine specialist and anaesthetist at Fremantle Hospital, Joondalup Health Campus and Mercy Medical Centre, Western Australia

DR STEPHANIE DAVIES, MBBS FANZCA FFPMANZCA
Pain medicine specialist, anaesthetist and Head of Pain Medicine Unit, Fremantle Hospital and Bethesda Hospital, Western Australia

“Pain is a more terrible lord of mankind than even death himself” (Albert Schweitzer)

INTRODUCTION
Pain management is ‘core-business’ for the anaesthetist. Indeed, anaesthesia developed from the humanitarian desire to control pain during surgery by (pharmacologically) altering consciousness, initially with chloroform, nitrous oxide or ether and prior to the 19th century with opioids, alcohol and even asphyxiation. Involvement of anaesthetists in the management of acute post-surgical and post-trauma pain, labour pain, chronic and cancer pain soon followed.

But what is this phenomenon called pain and how is it different to (and often confused with) nociception, which is defined as the “neural processes of encoding and processing a noxious (tissue-injuring) stimuli”? 1

ORIGINS AND MEANINGS OF THE WORD ‘PAIN’
“What's in a name?” (William Shakespeare)
Exploring the origins of the English word ‘pain’, provides insights into its meaning and conceptualization in Western and other civilizations.

The word ‘pain’ was probably used for the first time in the Middle Ages and is a derivation of old French ‘peine’ and the Latin ‘poena’ (as in ‘subpoena’) meaning ‘punishment’ or ‘penalty’ and the earlier Greek root ‘poine’ with essentially the same meaning. ‘Poneros’ is Greek for ‘evil’ or ‘grievous’.

‘Poena’ was the spirit of punishment in Roman mythology and the servant of Invidia (Latin) or Nemesis, the Greek goddess of divine retribution. This etymology promotes the concept of pain as an evil, punitive experience, judgment or personal nemesis, perhaps reflecting the religious (‘wrath of God’) and cultural overtones of Europe in the Middle Ages. ‘Algós’ is Greek for pain and is again linked to sorrow or punishment; ‘odyne’ (Greek) is also used to describe pain but means ‘to eat or consume’ and ‘nocere’ (Latin) means to injure, damage or harm.

The Latin word ‘dolor’ with derivatives still used in modern languages such as French and Spanish, means ‘hurt’ or ‘ache’ which is more descriptive of the sensory experience, although there is still linkage to ‘emotional’ words such as ‘sadness’, ‘suffering’ or ‘anguish’. In some Asian languages such as Japanese or Bahasa, the word for pain is used interchangeably with ‘disease’, ‘illness’ or ‘hurt’ without reference to punishment or suffering.

The concept of pain as an ‘evil punishment’ expressed in many languages, cultures and epochs, suggests that it is more than simply an unpleasant sensation or ‘hurting’; it is a negative emotional experience linked to ‘suffering’ with social, spiritual and philosophical dimensions.

DEFINITION AND CONCEPTS OF PAIN.
A sub-committee of the International Association for the Study of Pain (IASP) Task Force on Taxonomy headed by Professor Harold Merskey, ‘crafted’ the most commonly used definition of pain in 1979. 2 A recent update of IASP pain terminology was remarkable in that, after due consideration and debate, it was decided not to modify the original definition at all after 30 years, despite major advances in pain-related fields as diverse as neuro-science and philosophy. 1 However, this document is still subject to revision after a period of consultation.

PAIN
Pain is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” 2,3

Breaking-down the components of this seemingly simple line of text is useful in gaining an understanding of the concepts of pain.

“Pain is an unpleasant sensory and emotional experience.” Pain has to be unpleasant, however similar unpleasant sensations such as dysesthesiae, itch or cold are not pain. Curiously, some patients with cortical injuries (such as after a stroke) clearly report ‘pain’ (as understood from their past experiences) but do not experience it as ‘unpleasant’. This is pain asymbolia and causes a dilemma for the IASP definition. Thanks to the Marquis de Sade, ‘pleasure from pain’ (sadomasochism or algolagnia) further complicates the definition!

“Pain is...a sensory and emotional experience” (and not just a sensory experience). This statement is of critical importance in conceptualising pain. Pain is more than perception, ‘sensory processing’ or ‘nociception’. To stress this point, consider that pain isn’t even one of the five primary senses. Pain not only has ‘sensory-discriminative’, but also ‘emotional-affective’, ‘cognitive-evaluative’, ‘motivational’ and perhaps even spiritual dimensions. These ‘higher dimensions’ of pain are important in the expression of ‘pain language’.4
“Pain is ...associated with actual or potential tissue damage.” Personal experience and neurobiology demonstrates that pain is usually associated with tissue damage in the body. However by including the word ‘potential’, the definition avoids the obligation of ‘tying’ pain to tissue damage.3 This is a revolutionary change from the time-honoured Cartesian concept of pain as a (real-time) ‘alarm system’ for injury.

Pain in the context of “potential tissue damage”, reflects situations where damage has not actually occurred but may occur (so called ‘tissue threat’) for example, pressing hard on your thumbnail or briefly touching a hot plate, or perhaps in situations where pain is reported by persons who simply ‘perceive’ that their tissues are ‘under threat’. In some cases this is conceptualised (rightly or wrongly) as ‘psychogenic’ or somatoform pain.

A person can clearly experience pain in the absence of tissue damage with ‘phantom pain’ (where there’s no tissue at all) being the classic example. ‘Phantom phenomena’ clearly demonstrate that ‘experiences’ such as pain, touch and even our sense of ‘self’ (this is ‘my’ limb) can be ‘generated’ in the absence of real-time sensory inputs (such as nociception) from the physical body. The phenomenon of allodynia (pain due to a stimulus [touch] that is not normally painful) is another example of pain in the absence of tissue damage.

The definition of pain continues...or described in terms of such damage.” Pain is a totally subjective experience of the sufferer’s ‘internal world’ of the self, which is expressed to others in the ‘outside world’ (doctors, family or even insurance case managers) using the ‘language of tissue damage’ (pain narrative), either actual or threatened. For example, “my injured disc is causing pain in my back” or, “my muscle pains get worse when I am stressed and anxious; someone on the internet said it was fibromyalgia.” “Individuals learn the application of the word (pain) through their experiences related to injury in early life.”3

An important note appended to the IASP definition of pain states that, “in the absence of tissue damage or any likely patho-physiological cause...if they regard their experience as pain and if they report it in the same way as pain caused by tissue damage, it should be accepted as pain.”

In other words, pain is always what the sufferer says it is. There is no way that ‘we’ as external observers can really ‘know’ otherwise. “There is usually no way to distinguish their (the sufferer’s) (pain) experience from that due to tissue damage, if we take the subjective report” (which we have to).3

Such license further ‘unites’ pain from the obligation of tissue damage. However it opens-up a potential dilemma with concepts such as ‘psychogenic’ or somatoform pain disorders. Is this ‘real’ pain according to the IASP definition? The answer is yes, given the sufferer experiences and reports they are in pain (there is ‘perceived’ tissue threat and they express themselves in ‘terms of such [tissue] damage’). However the validity of the definition (“...or described in terms of such damage”) clearly fails in factitious disorder or malingering, where the subject feigns pain (this may be considered ‘acting’) when there is no actual or even potential tissue damage.

The IASP definition further explains that, “...(pain) is always a psychological state...pain in the absence of tissue damage or any likely patho-physiological cause...usually happens for psychological reasons.” Curiously, having just made the great leap forward of ‘untying’ pain from tissue damage (in the body) this statement simply serves to re-define pain as a problem of the mind instead (psychological ‘damage’).

“...or described in terms of such damage.” A criticism and potential limitation of the IASP definition of pain is reliance on verbal reporting by the sufferer. This obviously ‘excludes’ non-verbal humans (eg infants, dementia) and animals. However, the definition does not technically preclude non-verbal humans or animals from experiencing the unpleasant sensory and emotional experience of pain.

Verbal reports may be seen as an ‘efferent’ response to the internal (pain) experience. However other efferent responses, in particular pain behaviours (grimacing, groaning, rubbing an injured arm or running away) are not addressed in the IASP definition and yet in clinical practice and in everyday life, are keystones for identifying persons in pain, especially those who are non-verbal or non-lingual; persons who simply can’t ‘speak the (pain) language’. There may be a place for changing the definition of pain slightly from ‘described’ to ‘expressed’ (in terms of such damage) to encompass pain behaviours.

Despite limitations, the IASP definition of pain remains essentially valid, widely applicable and clinically useful. Importantly, it unites pain from obligatory tissue injury and in so doing has ethical merit by promoting ‘belief’ of the sufferer’s pain reports and alleviating the stigma of skepticism.5

Table 1. Summary of pain concepts, based on the IASP definition

- Pain is a sensory and emotional experience.
- Pain is an entirely subjective experience of the ‘self’.
- Pain is not the same as nociception (see below).
- Pain does not require the presence of tissue damage.
- Pain is expressed by the sufferer in the ‘language’ (terms) of tissue damage.
- The definition relies on verbal reports of pain.
- The definition refers to pain in humans but not in other species.
- The definition was designed as an explanatory clinical tool and not to define mechanisms, models or pathological concepts of pain.
Table 2. Criticisms & questions surrounding the IASP definition & concepts of pain

- Pain is conceptualized as either a problem of the body (tissue damage) or the mind (psychological ‘damage’).
- The definition may not apply to humans (eg neonates) or animals incapable of self report.
- The definition does not address pain behaviours.
- The definition does not address physiological (reflex, neuro-endocrine, autonomic) or psychological (affective, cognitive) phenomena associated with pain.
- The definition does not specifically address neuropathic pain, where there is nerve (but not tissue) damage.
- The definition does not address mechanisms or disease-based models of pain.
- The definition does not address the philosophical, spiritual, societal-cultural and ethical aspects of pain.
- The definition does not address the meaning or purpose of pain and links to ‘suffering’.

NOCICEPTION, HYPERALGESIA AND ALLODYNIA

Nociception

Nociception is defined as “the neural processes of encoding and processing noxious stimuli.”

A noxious stimulus is “an actual or potential tissue-damaging event”, usually in the form of physical (mechanical, thermal, electromagnetic) or chemical energy. It is interesting to note that not all noxious stimuli (eg X-rays) cause tissue damage and even if they do (for example, a slow growing liver or brain tumour) they don’t always activate nociceptors and cause pain.

A nociceptor is, “a sensory receptor that is capable of transducing and encoding noxious stimuli.” In other words, nociceptors transform the ‘energy of tissue damage’ (mechanical, thermal or chemical) into electrical energy for neural transmission, just like the rods and cones of the eye convert the electromagnetic energy of light into electrical impulses.

Nociceptive ‘traffic’ ascends from the tissues via nociceptive neurons, the dorsal horn and various spinal cord tracts to the brainstem, midbrain, thalamus and various cortical regions and is modulated by descending inhibitory and facilitatory pathways. Technically speaking there are no ‘pain’ pathways but rather nociceptive pathways for transmission. In other words, the spinothalamic tract does not actually transmit ‘pain’.

Neuro-physiological processes that ‘amplify’ nociception produce sensitization, which may be defined as “increased nociceptive output for a given input.”

When these processes occur in central nervous system (CNS) (mainly in the dorsal horn) it is called central sensitization which is characterized by increased (nociceptive) responsiveness, decreased threshold for activation (allodynia: see below), increased spontaneous activity (‘ectopy’) and an expanded receptive field.

Central Sensitization

Central Sensitization is defined as, “increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input.”

Clinically, central sensitization can only be inferred by the presence of hyperalgesia or allodynia.

Hyperalgesia is a psychophysical term defined simply as “increased pain sensitivity” (a painful stimulus feels more painful than ‘usual’).

Allodynia, which used to be defined as, “pain due to a stimulus which does not normally provoke pain”, is now defined specifically as, “pain in response to a non-nociceptive stimulus.” The only stimulus which doesn’t stimulate nociceptors (with certainty) is tangentially brushing the skin (with a camel hair brush or tissue). This only activates A-beta (touch) fibres that should not normally initiate nociception, except where central sensitization has occurred (where A-beta touch fibres have gained ‘access’ to the nociceptive system in the dorsal horn).

When touch feels painful (like having a hot shower with sunburn), this is evidence that central sensitization has developed and is always associated with ‘pathological’ pain states. In other words, allodynia is the clinical sign for central sensitization.

Hyperpathia is defined as a “painful syndrome characterized by an abnormally painful reaction to a stimulus, especially a repetitive stimulus (such ‘poking’ a painful region repetitively with a toothpick, at 3 Hz for 30 seconds) as well as an increased threshold.” It may occur with allodynia, hyperesthesia, hyperalgesia, or dysesthesia and reflects the phenomenon of ‘wind-up’.

Wind-up is a specific experimental and clinical paradigm which demonstrates increased pain sensitivity with repetitive stimulation, usually over seconds-to-minutes; an amplifier effect. Wind-up is not the same as central sensitization and the terms should not be used interchangeably.

Long-term potentiation is a nociceptive ‘memory’ or ‘capacitor’ effect (persisting output from nociceptive neurons in the CNS, in the absence of an afferent input) and is similar to the processes of laying down memory in the hippocampus.

Processes of descending neuromodulation that inhibit or ‘dampen down’ (ascending) nociceptive traffic are collectively termed Diffuse Noxious Inhibitory Control (DNIC).
NOCICEPTION IS NOT THE SAME AS PAIN
John Connor: Does it hurt when you get shot?

Nociception was defined for the first time in the 2008 IASP revision and reflects the enormous expansion of knowledge in basic neurosciences over the past 30 years, including functional brain imaging.

Explanatory notes accompanying the definition clearly highlights that pain and nociception are not the same thing; “pain is a subjective phenomenon whereas nociception is the object of sensory physiology.”

Nociception (due to tissue damage) is the sensory process that most commonly (but no exclusively) ‘triggers’ the multidimensional and conscious experience of pain (the classical ‘pain-as-an-alarm’ paradigm). However pain can clearly occur in the absence of nociception (tissue damage) (eg. phantom pain or allodynia) and nociception can occur without ‘triggering’ pain (nociception in tissues during surgery under local anaesthesia or whilst unconscious during general anaesthesia).

Pain is an absolute function of consciousness whereas nociception is not. There is no ‘pain centre’ in the brain and strictly speaking, there are no ‘pain pathways’. Pain does not cause changes in the nervous system, although various processes such as cortical changes on fMRI are associated with pain.

To find a sensory metaphor, nociception is the comparable to the process of sound energy being converted into nerve impulses in the inner ear, which are transmitted to the auditory cortex. Hearing is the conscious experience of these auditory stimuli and pain is more like ‘music’, a complex sensory and emotional experience. Like pain, you can experience music in the absence of sensory (auditory) inputs (like a tune playing in your head).

CLASSIFICATION AND TAXONOMY OF PAIN
Functional classification
Physiological pain: ‘Adaptive pain’ with a clearly protective (alarm) function, usually ‘acute’ and short-lived.
Pathological pain: ‘Maladaptive pain’ with no beneficial role, usually (but not always) persistent or chronic, associated with hyperalgesia and often neuropathic in aetiology.

Aetiological, patho-physiological & anatomical classification

Figure 1. A classification of pain

Nociceptive pain: Is “pain due to activation of nociceptors” in cutaneous, somatic or visceral structures and is the ‘tissue injury pain’ of the classical, physiological alarm system and is therefore usually ‘adaptive’.

Neuropathic pain: Is “pain arising as a direct consequence of a lesion or disease affecting the somatosensory nervous system”, either in the periphery (eg painful diabetic neuropathy) or in the CNS (central pain) (eg post-stroke, MS or spinal cord injury). The definition was modified in 2008 to remove the term ‘dysfunction’ (of the nervous system) which was thought to be too broad and non-specific. Disorders such as fibromyalgia, with evidence of dysfunction in certain nervous system processes, were sometimes classified as neuropathic pain. Neuropathic pain is usually maladaptive, although one may consider that acute radicular leg pain due to a lumbar disc protrusion might force an individual to rest and therefore help to limit further ‘damage’.

Dysfunctional pain: Although not listed in the taxonomy, this term was suggested to classify pain that is neither nociceptive nor neuropathic in aetiology, with fibromyalgia as an example. Other terms including ‘idiopathic’ (unexplained) pain and perhaps (somatoform) pain disorders may fall under this category. The term alloplastic pain has been proposed as an alternative (Dr S Davies, 2009: personal communication. For details, see the chapter, ‘What is pain? Part II’, in this publication).

Cancer Pain: Is pain associated with a neoplastic process or its treatment (eg radiotherapy) which pathologically-speaking, may be nociceptive and/or neuropathic in nature.

Cutaneous Pain: Is pain associated with activation of nociceptors of the skin. Cutaneous pain is ‘sharp’, fast, well-localized and transmitted via (in evolutionary terms) neo-nociceptive pathways (eg spinothalamic tract) to the cortex. It is a fast, reactive system that responds to external (environmental) tissue threat and is of great survival benefit.
Visceral pain: *Is pain associated with activation of nociceptors (kidney stones) or neuropathy (porphyria) in visceral organs.* Visceral pain is usually poorly defined and localized (referred), often ‘dull’, ‘aching’ and diffuse and associated with considerable autonomic and emotional activation.

Somatic pain: *Is pain associated with activation of nociceptors in muscle, tendon, ligament, bone or ‘lining tissues’ such as the peritoneum.* The qualities of somatic (eg musculoskeletal) pain seem to share features of both cutaneous and visceral pain, which might reflect embryology (mesoderm) or function, in evolutionary terms.

**TEMPORAL CLASSIFICATION**

Acute pain: There is no IASP definition for acute pain, which has been defined as, “pain of recent onset and probable limited duration; it usually has an identifiable temporal and causal relationship to injury or disease.”

Chronic (persistent) pain: Although quite remarkably, there is no IASP definition of ‘chronic pain’, it is commonly defined as, “pain lasting greater than 3 or 6 months duration”, or “pain that persists past the normal time of (tissue) healing.” The latter definition does not reflect situations such as chronic inflammatory arthropathy (rheumatoid arthritis), neuropathic pain or hyperalgesia.

Temporal definitions of pain are relatively artificial, with acute pain commonly considered as ‘adaptive’ or ‘physiological’ and associated with a proximate cause, and chronic pain as ‘maladaptive’ often without a clear perpetuating pathology. There is considerable overlap between these terms and they likely exist on a temporal and patho-physiological continuum.

**DISEASE-BASED CLASSIFICATIONS**

ICD 10 classifies pain purely as a symptom of various diseases states in organ systems. Where pain is not referable to an organ system, region or disease, it is defined as ‘pain not elsewhere classified’ which in turn may be acute, chronic, intractable or ‘pain not otherwise unspecified’.

The IASP has a coded 5 axis taxonomy for describing chronic pain disorders, based on body region, organ system, temporal characteristics, intensity and aetiology. Pain concepts will be discussed further in ‘What is pain? Part II’ located in the next chapter of this publication.

**REFERENCES**