PAIN - BASIC CONSIDERATIONS

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Introduction

Pain is a sensory and emotional experience. The emotional component is variable from person to person and in the same person from time to time. Management of pain has to take this fact into consideration. The patient must be believed about the pain. It is the physician's duty to relieve suffering. In addition, unrelieved pain can cause physical damage too. For one thing, it would worsen the pain experience by muscle spasm, peripheral and central sensitization and recruitment and by muscle spasm. Unrelieved acute pain can cause chronic pain, and long standing pain can cause anatomical and even genetic changes in the nervous system.

Pain can be classified in several ways, but the most relevant in terms of therapeutic application is into nociceptive and neuropathic. In addition to such identification of the type of pain, it is also necessary to quantitate pain. Several scoring systems are available like the numerical scale, but it needs to be remembered that the patient is the only person who can quantitate his pain.

The objective of this paper is to enable the reader to understand some basic facts about pain that have importance in actual management of people with pain. It is not a comprehensive review of pain mechanisms. Much of the known basic science on the subject, which may have future implications on management, is not included here.

What is pain?

The International Association for Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."

It is to be noted that pain is not just a physical sensation. It is also an emotional experience. It varies from person to person and in the same person from time to time. There may be a strong emotional component contributing to the pain experience; but that does not mean that the suffering is less important. A simple no-nonsense definition of pain is “Pain is what the patient says, hurts.” The emphasis is on the patient's experience.

Let us take the practical experience of a patient waiting for surgery. If he has not been given adequate explanations about what to expect; but has been rudely snatched away from the relatives and wheeled into a strange frightening environment with bright lights, gleaming frightening contraptions and weird masked creatures, he will be anxious and apprehensive. An attempted spinal anaesthesia will cause severe pain in this person because he will be unable to position himself properly and because his muscle tone is high. Thus the electrical impulse generated by the stimulation of the nociceptors will be more in this person than in a calm person. In addition, for any given amount of neural stimulation, his emotional experience of pain will be exaggerated too. Fear, anxiety and sleeplessness are all factors which worsen the pain experience. A few words of reassurance to the patient (“I am going to stay with you all through the operation and look after you; I shall make sure that you are pain-free”) can easily reduce the pain experience. In the context of long-standing pain, the emotional component becomes even more important.

Box 1: Points of clinical application

1. Emotional reasons can worsen the pain experience; and that is not the patient's fault. It is only a reason for exploring for the emotional problem and for providing adequate analgesia.

2. Only the patient (and no one else) can assess his pain properly. A dictum that arises out of the latter definition is, "believe the patient about the pain".

3. Any one attempting to take up pain medicine needs to learn the fundamentals of psychosocial support and principles of communication.

What is Chronic Pain?

Chronic pain is defined as pain which persists a month beyond the usual course of an acute disease or a reasonable time for an injury to heal, or is associated with a chronic pathological process which causes continuous pain, or pain which recurs at intervals for months or years.
Mechanism of pain sensation

Fig. 1: Diagrammatic representation of the pain pathway from the nociceptor to the dorsal horn of the spinal cord (N= Nociceptor. DRG= Dorsal root ganglion. DH= Dorsal horn of the spinal cord).

Fig. 2: Recruitment: peripheral and central.

Mechanism of pain sensation

Fig. 1 shows an oversimplified pain pathway from the periphery up to the dorsal horn of the spinal cord. The peripheral bare nerve ending subserving pain is called the nociceptor. Mechanical, thermal, electrical or chemical stimulation of the nociceptor causes an electrical impulse to be generated and transmitted up the peripheral nerve through A-delta and C fibres to the dorsal horn of the spinal cord. In the dorsal horn cells, the impulses get modified before onward transmission to the thalamus and the cerebral cortex where the pain is appreciated.

Pain must be treated. Why?
1. **Relieving suffering**: First and foremost, pain is suffering and it is our duty to relieve suffering. This applies whether pain is acute or chronic, but understandably, the longer the suffering the more the necessity.
2. **Muscle spasm**: Pain causes reflex muscle spasm. This has two negative implications:
   a. It can cause respiratory embarrassment. Pain in upper abdominal surgery is a classical case in point. Muscle spasm can cause regional hypoventilation and contribute to post-operative respiratory problems. In a multiple rib fracture, pain relief may be the single factor that can avoid the need for artificial ventilation.
   b. The muscle spasm itself can be a major cause of pain. Even short term muscle spasm can cause severe pain – for example, backache – but when long-lasting, it may result in development of myofascial trigger points. These areas of sustained contraction then act as new foci of pain.
3. **Untreated pain will keep getting worse**. This is true for surgical trauma too, but understandably, is even more relevant in chronic pain. There are several neurophysiological reasons for this:
   a. **Recruitment of nociceptors**: Silent or ‘sleepy’ nociceptors are those that do not respond to noxious stimuli normally; but are activated in the inflamed tissues. Once they are recruited, the same degree of peripheral stimulus generates more number of electrical impulses, thereby resulting in worsening of pain (fig 2).
   b. **Central recruitment**: With persisting pain, adjacent spinal segments (or adjacent supraspinal areas) get recruited, so that pain gradually spreads to larger areas (fig. 2). This could be the reason for the occasional patient with cancer pain or some chronic pain to complain of pain from “head to foot”.
   c. **Sensitisation of nociceptors**: The nociceptors get sensitized with time so that response threshold is lowered. Chemicals involved in the inflammatory process mediate this peripheral sensitization. Other agents implicated are purines, cytokines, leukotrienes, nerve growth factor and various neuro-peptides. Prostaglandins are believed to have a major role in this process of sensitization and this accounts for the reduction in pain obtained with the use of Non-steroidal anti-inflammatory agents (NSAIDs).
   d. **Central sensitization (‘wind-up” phenomenon”)**: The dorsal horn cells get sensitized. This is compared to a wound-up spring, hence the common term, “wind-up” phenomenon. When the painful stimulus persists, the same peripheral input produces progressively increasing electrical response from the dorsal horn cell. It manifests as:
      e. An increase in the receptive field for sensitised dorsal horn neurons.
      f. An increase in the duration of response.
      g. A reduction in response threshold.
   N-Methyl D Aspartate (NMDA) is believed to be the most important neurotransmitter involved in the “wind-up” mechanism.
4. **Neuroanatomical re-organisation**: This seems to accompany central sensitization often. In the face of continued barrage of pain impulses, the integrity of the dorsal horn of the spinal cord gets disturbed so
that neurons subserving input from A-beta fibres form functional connections with neurons in lamina-2 subserving input from A-delta and C fibres. Thus, stimulation of A-beta fibres, which normally causes sensations only of touch and pressure, in this case causes pain. This is called **allodynia**. (Such abnormal sensations can occur also in peripheral nerve lesions.)

5. **Anatomical and genetic changes:** It has been clearly proved now that persistent unrelieved pain can cause anatomical changes in the nervous system, as well as genetic changes in the dorsal horn cell.

**Box 2**

1. Pain must be treated early. Unrelieved pain causes worsens pain both in extent and severity.
2. In long-standing pain associated with central sensitization, NMDA antagonism with ketamine would have a role to play.
3. Once anatomical changes in the nervous system have occurred in chronic pain, total relief may be unlikely.

**Classification of Pain**

The peripheral nerve ending (nociceptor) transmits the pain impulse to the dorsal horn of the spinal cord, where it gets modified before onward transmission to the brain. Any pain caused primarily by stimulation of the nociceptor can be said to be **nociceptive** pain. If pain is **not** caused by a stimulus applied to the nociceptor, but is caused by impulse generation within the pathway proximal to the nociceptor (this could be in the nerve, the spinal cord or the brain), it is called **neuropathic** pain (fig. 3).

This does not mean that all nociceptive pains are similar in presentation or management. For example pain arising out of smooth muscle spasm can be very different from the pain of skeletal muscle spasm.

**Neural injury** pain can be said to involve anatomical abnormality in peripheral nerves, in pain receptors or in the central pain pathway. The following three features help to diagnosis a neuropathic pain.

1. The nature of the pain may be shooting, stabbing, pricking, aching or burning.
2. It has a neural or dermatomal distribution.
3. It is often associated with abnormal sensation in the area of pain. This can take the form of hypo- or hyperesthesia. Unfortunately it often takes the form of *dysaesthesia*, that is, an unpleasant abnormal sensation whether spontaneous or evoked. Allodynia and hyperalgesia are examples. Allodynia has already been described. Hyperalgesia is increased response to a stimulus, particularly a repetitive stimulus, as well as an increased threshold (an exaggerated response with an increase in pain threshold).

**Nerve compression** pain occurs when there is extrinsic pain on the neural structure, as for example with a nerve root compression in prolapsed inter-vertebral disc or with collapsed vertebrae from metastatic lesions.

Neuropathic pain can also be sub-classified into peripheral and central, depending on site of origin of abnormal impulse. The relevance is that central pains often behave differently from peripheral neuropathic pains, particularly in their response to drugs. Central neuropathic pains are commonest in injury to the CNS – for e.g. Spinal cord injuries, stroke etc. It must be remembered that pain originally of peripheral nerve origin, can become centrally established – by somehow altering the CNS. Once this has happened, a peripheral nerve block or neurolysis may not successfully remove the pain.

**Box 3: Points of clinical application**

1. Depending on site of origin of pain, different types of pain may warrant different modalities of management. It is necessary to understand the type of pain, to decide on management.
2. Neuropathic pain is often burning, aching, stabbing or pricking, is of neural or dermatomal distribution and is usually associated with abnormal sensation in the area of pain.

**Evaluation of pain:** A proper evaluation of pain is essential for proper treatment. Patients often have more than one type of pain. Some of them may be unrelated to, or only indirectly related to the basic disease. The different pains, for e.g. muscular, neuropathic etc. would need different modalities of treatment.
The following points are worth remembering

i) Pain being a somato-psychic experience, the patient is the only person who can assess the intensity of his pain.
ii) Muscular pains often can be more severe than the original pain.
iii) Treatment itself may cause new pains – e.g. opioids inducing constipation.

The body chart

The body chart (fig. 4) is a useful tool in evaluation of pain. The different sites of pain should be marked at the time of evaluation along with the nature of each, so that it is available at the time of review.

The following facts must be recorded in the chart about each pain. (i) Duration of the pain. (ii) Intensity of the pain. (iii) How does movement affect the pain? (iv) Is the pain superficial or deep? (v) What is the nature of the pain? (Stabbing, pricking, burning, aching etc.) (vi) How does the pain affect sleep?

Quantitating pain

To assess success of treatment, some form of quantitation of pain is necessary. Many pain scoring systems are available.

a) Categorical: A four or five point scale could grade the pain as none, mild, moderate, severe and excruciating etc. Probably this scale is not sensitive enough, but it has the advantage of simplicity.
b) Numerical scale: This is a 11 point scale where “0” means no pain and “10” is the worst imaginable pain.
c) Visual Analogue Scale (VAS): A 100 mm scale with no pain at one end and worst imaginable pain at the other, is commonly used. There are no graduations on the side that the patient sees; the 100 graduations are only visible to the person who makes a record of the patient’s evaluation.
d) McGill Pain Questionnaire (MPQ): MPQ attempts to assess the quality of pain too; on sensory, affective, cognitive and miscellaneous domains.
e) A child or an illiterate person could use a set of faces (happy or sad) to indicate the severity of his pain.

Evaluation of outcome in chronic pain

Most patients with chronic pain get some degree of response to any new modality of treatment that is introduced. This could be partly due to placebo effect and partly due to reduction of the emotional element of pain. So it is to be remembered that success of a treatment regime can be decided on only if the positive response lasts long enough – at least for three weeks. And the evaluation is best made in terms of quality of life too, not just with a pain score.

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References