**Abstract**

There is emerging evidence from different neuropsychological and empirical clinical studies that pain is now to be viewed as an experience with strong emotional and other psychological components and not merely as a sensory phenomenon. This article attempts an in-depth study of the relationship of pain & psychiatric disorders in order to make a case for modulation of pain by emotions. Further, the newer dimensions of assessment of pain and the current views of multidisciplinary management of pain syndromes relevant for concentration-liaison settings are also critically reviewed. It is argued that current trends in multidisciplinary management of pain are a welcome departure from the age old Cartesian dualistic approach to illness (German J Psychiatry 2005;8:85-93).

**Keywords:** Pain, chronic pain, depression, psychiatric disorders, pain perception

**Received:** 25.5.2005  
**Revised version:** 19.8.2005  
**Published:** 1.10.2005

**Introduction**

Pain is an extremely common problem in the general population as well as in clinical practice. The economic burden could be enormous if the medical cost, lost productivity and human suffering are all taken into consideration. Despite major advances in the physical methods of treatment large number of patients on treatment continues to report pain. Last few decades have seen a transformation in the understanding and approach to health and health problems – from the age old mind-body dualism to a holistic approach. In this emerging scenario pain remains the epitome of the ultimate psychosomatic phenomenon in terms of the newer understanding of its neurophysiologic basis and the growing empirical support for the psychologically based methods for its management.

This article has two sections. The first section attempts to examine the strength of ‘the case for modulation of pain by emotion’ from the following perspectives;

1. Interface of pain and psychiatry
2. Poor relation between the physical findings and the level of perceived pain and disability
3. Neuroanatomic basis of perception of pain
4. Imaging studies on the perception of pain
5. Current definition and nosology of pain
6. Assessment of pain
7. Chronic pain, neuropathic pain and fibromyalgia

The second section deals with the brief review of the evidences of the usefulness of psychotropic drugs (mainly antidepressants) and the psychological methods of treatment of pain.
Interface of pain and psychiatry

Two types of studies are relevant to examine the interface: (1) Pain in psychiatric disorders, and (2) psychiatric disorders in chronic pain patients. Relatively larger number of studies have addressed the latter than that are available on the former.

Pain in Psychiatric disorders

It has been reported that 59% of patients referred to a psychiatric consultation–liaison service complained of pain on initial evaluation, although only 6% of all patients were referred for this problem (quoted from King 2003). Haturvedi (1987b) identified pain in 18% of patients attending psychiatric outpatient clinic. In this study, middle-aged housewives with depression, anxiety and other neurotic symptoms reported pain. The prevalence of pain is reported to vary from 30% - 84% in depression but data on pain complaints in different subtypes of depression are not available (France et al. 1988). The prevalence of pain in a sample of 106 psychiatric inpatients was investigated in a London psychiatric hospital (Baune and Algéesh 2004). The point prevalence of pain was 50%, the 6-month prevalence 75% and 12-month prevalence 76.5%. Low back pain, headache, shoulder and neck pain were the most frequent complaints. The most frequent psychiatric diagnosis was affective disorders and neurotic stress related and somatoform disorders followed by psychotic disorders. Almost 10% of all patients had been bothered by pain for the last 3 months.

Pain is a non-criterion symptom of depression and anxiety disorders. It has been suggested that this pain is probably different from that of chronic pain patients (CPP) seen in pain centres. The pain in psychiatric patients is usually insidious in onset, located in multiple sites of cephalic and truncal regions (cf. low back and neck in CPP), transient and rarely chronic, has poor neuroanatomic correlation and nonspecific exacerbating and alleviating factors (France et al. 1988).

Psychiatric conditions in chronic pain patients

Comorbidity of pain and depression

The reported point prevalence of major depression in chronic pain population varies between 1.5% to 54.5% and the reported life time prevalence varies from 20% to 71% (Magni et al. 1990). Interestingly, dysthymia and atypical depression are associated with greater severity of pain than major depression or adjustment disorder with depressed mood (Magni-Guido et al. 1985). McWilliams et al. (2003) utilised the National Comorbidity Survey data (part II) (n=5877) to study the association between chronic pain conditions (arthritis) and common mental disorders. A positive association was found between chronic pain and mood and anxiety disorders (OR 1.92-4.27). The presence of two psychiatric disorders were associated with pain related disability but not the presence of one. A number of views have been proposed on the relationship between pain and depression.

Is there a constitutional relationship?

As depressive disorders may be more common (37.8%) (Katon et al. 1985) in the first degree relatives of people with chronic pain it has been suggested that there may be a possible environmental or genetic predisposition for developing pain (Haturvedi 1987a). Pain and depression may coexist either independently or as the result of common psychosocial or neurochemical pathway (King 2003).

Does depression precede pain?

Blumer and Heilborn (1982) suggested that emotional disturbance in pain prone individuals may give rise to pain. This type of pain could be viewed as a masked depression.

Does pain predict depression?

There are longitudinal studies to explore the causal association of pain and depression. Brown (1990) conducted a study with six waves of data collection on self reported pain and depression in 243 subjects with rheumatoid arthritis. Pain severity predicted subsequent depression in patients in the last 12 months of the study. In another study (Atkinson et al. 1991) patients with chronic low back pain (CLBP) had higher life time rates of major depression, alcohol use disorders and anxiety disorders but the first episode of major depression generally followed pain onset. Gamsa (1990, 1991) reported that pain in the pain clinic patients was associated with current levels of depression and life dissatisfaction and not with the personal history antecedents. These reports fail to support the view that pain in CPP results from pre-existing emotional disturbance.

Does pain and depression covary?

Pain affects mood as well as severity of depression and depression becomes fixed as pain continues and becomes persistent. Moreover, a positive correlation among depression, pain and pain behaviour has also been observed. So it may be concluded at the present level of evidence that “depression promotes pain and pain promotes depression” (Magni et al. 1994)

Pain comorbidity with other psychiatric disorders:

Only a limited number of studies have addressed the issue of prevalence of DSM III diagnoses other than affective disorders in CPP. The findings of the important studies are shown below.

Although several discrepancies are present across the studies, mainly for those DSM diagnoses that have questionable reliability, it is evident that a wide range of mental illness is present in CPP and that majority of CPP will have an axis I diagnosis. Furthermore, CPP may have psychopathology preceding the onset of pain (Polatin et al. 1991).
Table 1. Comorbid Psychiatric Disorders in CPP

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<tr>
<td>Somatisation disorder</td>
<td>3.9%</td>
<td>12%</td>
<td>16.2%</td>
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<tr>
<td>Conversion disorder</td>
<td>37.8%</td>
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<tr>
<td>Psychogenic pain</td>
<td>0.3%</td>
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<tr>
<td>Hypochondriasis</td>
<td>0.7%</td>
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<tr>
<td>Anxiety disorder</td>
<td>62.5%</td>
<td>7%</td>
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<td>Substance use disorder</td>
<td>14.9%</td>
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<td>Factitious disorder</td>
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<td>Personality disorders</td>
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<tr>
<td>Histrionic</td>
<td>59%</td>
<td>37%</td>
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<tr>
<td>Dependent</td>
<td>11.7%</td>
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<td>Passive Aggressive</td>
<td>14.9%</td>
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Poor relation between the physical findings and the level of perceived pain and disability

The level of perceived pain in CPP is not always associated with appropriate physical findings. It is estimated that the clear physiological aetiology cannot be identified for as many as 85% of the patients with low back pain (Deyo and Weinstein 2001). At the same time these complaints do not always warrant a psychiatric diagnosis. In a study in which MRI of the lumbar spine was performed in asymptomatic subjects, Jensen et al. (1994) found that 64% had at least one abnormal disc and 38% had two or more.

As a corollary, physicians’ prediction of functional status based on the physical examination does not match well with the CPPs’ functional status. This is due to the fact that the physicians have great difficulty in estimating anxiety, pain and activity limitation from patients’ self report and physical examination. Moreover, physical findings in CPP are not predictive of disability status, treatment outcome and return to work. It is thus likely that the mood and the pain related beliefs held by the patients are crucial factors in determining the perceived level of pain and dysfunction. However, these factors do not lend themselves to easy detection in routine medical examination.

Neuroanatomic basis of perception of pain

Pain is transmitted from the periphery to spinal cord by two types of nerve fibres- slow conducting and fast conducting. These fibres converge on the laminae of the dorsal root of the spinal cord, where it is influenced by descending tracts from the brain stem locus coeruleus and raphe nuclei. The raphe nuclei are also influenced by an inhibitory path way from the periaqueductual grey mater. The fast fibres relay at the thalamic nuclei and project to the primary and secondary sensory cortex. The slow fibres give collateral to reticular formation and relay at thalamic nuclei and project to the whole of the prefrontal cortex.

There is evidence that synaptic neurotransmitter secreted by primary afferent fibres subserving mild pain is glutamate and the neurotransmitter subserving severe pain is substance P. The latter is a polypeptide found predominantly around the free nerve ending receptors and in the spinal cord posterior horns.

The above neuroanatomical model of pain indicates that pain is not a simple sensation but is modulated by the descending influences of serotonergic and noradrenergic functions which are also involved in the regulations of mood. The prefrontal cortex that exercises executive control over all other cortical centres further influences the experience of pain.

Imaging studies on the perception of pain

Certain recent studies on imaging have supported the role of emotion and other higher mental functions in pain perception. Studies (Grachav et al. 2000, Hsieh et al. 1995a & 1995b) using PET, SPECT and fMRI have shown that experience of noxious stimuli are associated with activation of multiple areas of the brain like primary somatosensory areas, secondary somatosensory areas and anterior cingulate cortex (ACC), insula, lentiform nucleus, prefrontal cortex, inferior parietal cortex, primary and secondary motor cortex, cerebellum and brainstem. Activation of these areas represents sensory, emotional and cognitive components of the pain experience as follows:

Sensory = S1, S2
Affective = ACC and insula
Cognitive = prefrontal and parietal cortex
Inhibited motor response = lentiform nucleus and motor cortices

Mental activity training can apparently have an impact on the activation pattern to noxious stimuli. A study using fMRI (Jackson 2000, quoted from Fishbain 2003) has shown that instruction for mental imagery can modify the activation response i.e. increase the number and volume of activated regions during noxious stimuli. Similarly a study using PET has shown that hypnotic suggestion reduces the activation of ACC that is associated with the unpleasantness of perception of heat (Rainville 1997). It has been argued that this could be the neuropsychological basis of relaxation, anticipation and hypnotic response in perception of pain.

Previously experienced visceral pain (angina, labour pain) associated with strong emotion can be reproduced by stimulation of the posteroiinferior regions of thalamus that project to parietal operculum and insular cortex. Similar pain without an affective component can be elicited in patients with-
out the previous experience of such pain. Thus the thalamic stimulation probably activates a memory trace of the emotional component of pain which is the result of a previous experience. This is called a pain memory. The structure related to memory here is the insula that is now understood to play some role in preserving the memory of events related to painful stimuli (Treede et al. 2000).

**Current definition and nosology of pain**

The holistic view of pain experience is now reflected in the definition and nosology of pain. International Association for the Study of Pain (IASP) (Merskey and Bogduk 1994) defined pain as an unpleasant emotional or sensory experience associated with actual or potential tissue damage or described in terms of such damage. The operational word experience in this definition places pain outside the realm of simple sensation. Institute of Medicine committee on Pain, Disability and Chronic illness behaviour (Osterweis et al. 1987) defines experience of pain as more than a simple sensory process - a complex perception involving higher levels of CNS, emotional states and higher order mental processes.

A new pain-related category “pain disorder” has been created in DSM IV replacing the old category of somatoform pain disorder with predominantly presumed psychological underpinnings. The pain disorder is to be described with psychological factors, psychological and general medical conditions and with only general medical conditions. This approach perhaps precludes the fact that psychological and medical factors may not be differentiated in every individual case. Thus it offers a broader clinical approach to pain disorder. IASP offers a multiaxial classification of pain in which psychological factors are mentioned in axis II and V:

I – Region (head, face, mouth etc)
II – Systems (nervous system, connective tissue system etc.)
III – Temporal characteristics of pain (single episode, continuous, recurring etc.)
IV – Patients’ statement of intensity (mild, medium severe etc.)
V – Aetiology (inflammatory, neoplasm psycho physiological, psychological etc.)

**Assessment of pain**

Pain is a multidimensional experience, so there are many aspects of acute/chronic pain that the clinician and researchers may want to assess. The methods chosen will be affected by the purpose for which the assessment is made.

There are three main issues:

- To determine the suitability of a patient for treatment
- To determine the individual patient’s strength and weakness, so as to match or tailor a treatment programme effectively
- to evaluate change during treatment at follow up periods.

Following dimensions of pain are assessed from a mental health perspective:

**Pain behaviour** – this refers to all verbal and nonverbal outputs of the patient that a reasonable observer would characterize as suggesting pain (posture, facial expression, lying down, taking medication etc). Pain behaviour correlates with physical findings, perceived severity of pain, and functional impairment and scores of illness behaviour questionnaire (IBQ). Pain behaviour can be assessed in three ways. I) Direct observation – done in clinical and natural settings and is useful for knowing the environmental influences on pain so helps in functional analysis of pain. The observation is done by frequency counts or by using rating scales – like University of Alabama (UAB) Pain Behaviour Scale. (Turk and Matyas, 1992). II) Electromechanical devices – by a clock near the head or a pedometer to record the ambulatory behaviour. III) Self-evaluation – is useful as it gives responsibility to the patient but is less reliable than the objective methods, so the combined methods are better than a single method. A discrepancy between the two does not mean that pain may not be real but that there may be influences of the environmental factors on pain.

**Pain and illness behaviour** – Because pain can be influenced by such a wide variety of factors, the concept of illness behaviour has been found by some to be useful. The parameters that need to be considered in pain related illness behaviour are I) pain perception II) decision making whether to seek treatment and from whom to seek treatment III) the meaning of the pain to the patient IV) the manner in which the patient communicates about pain. V) The effect the pain has on the patient’s functioning. Among the factors that affect illness Behavior are cultural background, socioeconomic status, psychological mindedness, experiences, memory and learning.

**Pain-related beliefs** - A person’s beliefs about self efficacy and expectation from treatment are likely to alter pain response. Three psychological variables namely the tendency to catastrophize, appraisal of control and fear avoidance beliefs about pain activity were found to predict the disability in 83 CLBP patients even after adjusting for age, sex and pain intensity (Woby et al. 2004). The role of ethnicity in control appraisal, coping and adjustment to chronic pain were studied in a sample of 128 black Americans and 354 white Americans. The black patients reported lower perceived control over pain, more external pain coping strategies and a stronger belief that others should be solicitous when they experienced pain and also reported higher levels of depression and disability even after controlling for pain severity (Tan et al. 2005). However, ethnicity did not account for a significant amount in the total variance.

**Acceptance of pain**: This means that the patient should better focus on participation in valued activities and pursuit of the relevant goals and not on avoidance of the activities and control of pain. Acceptance of pain prepares the patient for self management of pain and has been shown to be associated with less pain, disability, depression and better work
status in 230 adult pain patients (Mc Cracken and Eccleston 2003).

Readiness to change: This is a construct that assesses the motivation of the patient to self-manage pain. There are four components in it – precontemplation, contemplation, action and maintenance. Glenn and Burns (2003) have shown with 65 chronic pain patients in a multidisciplinary treatment program that low precontemplation but high contemplation and action are associated with better coping strategies and improved outcome.

Meaning of pain: The social context of the meaning of pain is an important determinant of the pain response. Beecher (1956) observed that soldiers severely wounded in battle often did not complain of pain or described pain of a level far below that which would be expected. He postulated that this might have reflected the relief felt by the soldiers that they would be removed from the battlefield and their lives could no longer be endangered.

Pain and personality: A pain prone personality has been described in the literature with the characteristics of excessive guilt, pain being used to atone for guilt and repressed aggression. Personality disorders are also associated with chronic pain conditions. Neuroticism has been reported to be positively associated with passive coping and pain intensity whereas extraversion with active coping strategies (Ramirez-Maestre et al. 2004).

Chronic Pain, Neuropathic Pain and Fibromyalgia

Chronic pain may be nociceptive (i.e., resulting from injury or inflammation of somatic or visceral tissue) or neuropathic (i.e., resulting from neuronal maintenance of pains either peripherally or in the central nervous system). Three types of chronic pain are recognized: psychogenic, inflammatory, and neuropathic. The symptoms which suggest neuropathic pain include spontaneous pain, hyperalgesia and allodynia. Various mechanisms underlying neuropathic pain especially neuroplastic changes, will help us understand the patient with neuropathic pain better and also open avenues for therapeutic advances in this field (Pridmore et al. 2002). Patients with fibromyalgia also display clinical features common in neuropathic pain suggesting that there might be some overlap (Offenhaecker, Ackenheil 2005). Fibromyalgia is a chronic disorder characterized by widespread musculoskeletal pain, fatigue and multiple tender points. Research during the past decade has demonstrated similar abnormal pain processing in fibromyalgia and related chronic pain syndromes (Goldenberg et al. 2004).

Avenues for management of pain

Psychotropic drugs in chronic pain

Several placebo controlled studies have been conducted in acute and chronic pain conditions. Fairly consistently serotonergic-noradrenergic drugs have been found to be more effective than the pure serotonergic or noradrenergic drugs. However, the results are not so encouraging in pain with mixed etiology.

Four meta-analyses were performed, two on various types of chronic pain (Onghena and Houdenhove 1992, Guiraud and Chaumeil et al. 1987), one on neuropathic pain (McQuay et al. 1996), and one on alleged psychogenic pain disorder (Phillipp and Fickinger 1993). The findings are consistent in suggesting that antidepressants do have antinociceptive effects. The beneficial effects of AD for most pain condition are not related to mood. The presence of depression is not required for analgesic response. CPP may have an analgesic response to lower doses of AD than those used to treat depression.

The development of newer classes of antidepressants and second-generation antiepileptic drugs has created unprecedented opportunities for the treatment of chronic pain. These drugs modulate pain transmission by interacting with specific neurotransmitters and ion channels. Tricyclic antidepressants (e.g., amitriptyline, nortriptyline, desipramine) and certain novel antidepressants (i.e., bupropion, venlafaxine, duloxetine) are effective in the treatment of neuropathic pain. The analgesic effect of these drugs is independent of their antidepressant effect and appears strongest in agents with mixed-receptor or predominantly noradrenergic activity, rather than serotoninergic activity. First-generation antiepileptic drugs (i.e., carbamazepine, phenytoin) and second-generation antiepileptic drugs (e.g., gabapentin, pregabalin) are effective in the treatment of neuropathic pain. The efficacy of antidepressants and antiepileptic drugs in the treatment of neuropathic pain is comparable; tolerability also is comparable, but safety and side effect profiles differ. Tricyclic antidepressants are the most cost-effective agents, but second-generation antiepileptic drugs are associated with fewer safety concerns in elderly patients. Tricyclic antide-
pressants have documented (although limited) efficacy in the treatment of fibromyalgia and chronic low back pain. Recent evidence suggests that duloxetine and pregabalin have modest efficacy in patients with fibromyalgia (Maizels & Mc Carberg 2005).

Tricyclic antidepressants are thought to affect pain transmission in the spinal cord by inhibiting the reuptake of norepinephrine and serotonin, both of which influence descending pain pathways. In addition, histamine H₁-receptor affinity (associated with sedation) may be correlated with the analgesic effect of antidepressants. Amitriptyline also has an analgesic effect in patients with acute pain. Its dosage is 10 to 25 mg at bedtime; increase by 10 to 25 mg per week up to 75 to 150 mg at bedtime or a therapeutic drug level. The novel antidepressants venlafaxine and duloxetine have balanced inhibition of serotonin and norepinephrine reuptake without blockade of other neuroreceptors that are responsible for typical tricyclic side effects. Duloxetine is prescribed in the dosages of 20 to 60 mg per day taken once or twice daily in divided doses (for depression); 60 mg twice daily for fibromyalgia. The mechanism of action of bupropion is uncertain but involves blockade of dopamine uptake.

Antiepileptic drugs act at several sites that may be relevant to pain, but the precise mechanism of their analgesic effect remains unclear. These agents are thought to limit neuronal excitation and enhance inhibition. Relevant sites of action include voltage-gated ion channels (i.e., sodium and calcium channels), ligand-gated ion channels, the excitatory receptors for glutamate and N-methyl-D-aspartate, and the inhibitory receptors for GABA and glycine. Antiepileptic drugs may be categorized as first or second generation. The second-generation agents are better tolerated, cause less sedation, and have fewer CNS side effects. Gabapentin is prescribed as 100 to 300 mg at bedtime; increased by 100 mg every 3 days up to 1,800 to 3,600 mg per day taken in divided doses three times daily. Dosage of pregabalin is 150 mg at bedtime for diabetic neuropathy; and 300 mg twice daily for postherpetic neuralgia.

### Psychological methods of treatment

The multidimensional pain experience makes avenues for usefulness of psychologically based methods, for example:

- Methods targeted to the physiological system – biofeedback, hypnosis, relaxation
- Methods targeted to pain behaviour – operant conditioning (contingency management)
- Methods targeted to psychological aspects of pain – cognitive coping

**Acute pain:** The goal here is to relieve the pain. The Acute Pain Management Guideline Panel (1992) highlighted the importance of employing cognitive and behaviourally based intervention like relaxation, imagery, biofeedback as well as education and instruction in the management for postoperative and acute pain (King 2003).

**Chronic pain:** The goal of treatment is to address functioning as well as the pain. In many cases this requires refocusing the patient away from the pain. In essence patients with chronic pain must learn to have control of their lives back from pain. Following are some of the methods used for the management of chronic pain.

**Contingency management:** It has been proposed that verbal and nonverbal pain behaviour is learnt by operant conditioning with social reinforcement positively by getting higher level of attention from others and negatively by avoidance of unwanted responsibilities. The aim of contingency management is to reduce pain behaviour and improve well Behaviour and facilitate return to work. Fordyce (1976) in an inpatient programme (2-6 week) has shown the dramatic effects of this method on pain Behaviour.

**Fordyce’s work** has been criticized on three fronts that pain behaviour may not be affected by social contingencies, operant methods do not treat pain and a positive treatment outcome does not establish the operant factors as the aetiology of pain and pain behaviour. Fordyce (1989) has later emphasized the role of social contingencies and believes that pain related beliefs and avoidance behaviour of CPP is altered by operant methods.

**Cognitive coping methods:** Cognitive interventions refer to techniques that attempt to influence pain through medium of thoughts and include individuals’ intentional processes, images and self-statements. Fernandez and Turk (1992) identified 6 such techniques:

1. Pleasant imaginings (thinking of a bunch of flowers)
2. Rhythmic cognitive activity – like counting backwards from 100 in 3’s
3. External focus of attention – e.g. counting ceiling tiles.
4. Pain acknowledging – e.g. reappraising the pain objectively such as concentrating on the dullness and numbers.
5. Dramatized coping – like imaging you are playing a heroin role in a competitive setting.
6. Neutral imaginings – imagining being in neither arousing nor unpleasant situation, watching television, or attending a lecture.

The authors report a metaanalysis of 51 studies using above techniques and report that nearly 85% of the studies showed that the strategies are effective in increasing pain tolerance for experimental pain and reducing clinical pain ratings. There is perhaps more support for the usefulness of imaginary and less support for pain acknowledging.

**Multidisciplinary treatment:** There are several physical and psychological methods that are now used to treat pain. Multidisciplinary treatment approach uses any combination of these treatment methods. The indications of multidisciplinary treatment are the following:

- Chronic pain or chronic benign pain > 6months in duration
- Surgical failure (failed back surgery syndrome)
- High-level pain behaviour
- High-level functional disability
- Severe suffering
Role of the consultation-liaison psychiatrist

Convince the physicians and surgeons through active dialogue that:
- Organic and functional distinction is not always possible or desirable in every individual case
- Mental health professionals have significant role in the management of both acute and chronic pain
- A combined approach is indicated for a majority of patients
- Patients should never be referred with the suggestion that there is no physical basis for their pain and so they should seek the help of a psychiatrist.

Explain to the patient that:
- The pain and their suffering is real
- Certain methods of treatment may help them cope better
- With the pain, or get relief from the pain and regain function early
- These methods very well continue with medical and surgical methods for pain relief.

Conclusions

The age old view of pain as an unpleasant sensation has given place to the understanding that the pain is an experience comprising of nociception and perception and so all the mental processes involved in complex perception also influence the experience of pain. The dualism of body and mind is no more valid in the current approach to pain management. Effective pain management must include a multidisciplinary approach.

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