Management of acute pain

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Abstract

Acute pain is an important fear for most patients and influences their recovery and overall experience. Poorly treated, it could lead to undesirable effects and patient dissatisfaction. Hence, it is important to understand, assess and treat acute pain effectively. Pain is regarded as the fifth vital sign and should be addressed as important as other vital parameters. Management of pain involves team work, including acute pain services, especially in dealing with complex problems. Management of pain ideally starts at the pre-assessment visit or from first presentation to the clinician. It is important to anticipate and treat acute pain effectively, which may prevent the development of chronic pain syndromes. Patients should be given information about analgesic options, the risk:benefit ratio of the treatment options at the earliest opportunity and ideally have an individualized management plan.

Keywords Acute pain; acute pain management; acute pain service; adjuvant analgesics; non-opioid analgesics; opioid analgesics; postoperative pain

Introduction

'By any reasonable code, freedom from pain should be a basic human right, limited only by our knowledge to achieve it'.

Ronald Melzack

It is the basic duty of all healthcare professionals to relieve pain, and the most important indication for treating pain after surgery is humanitarian. Pain is defined by the International Association for the Study of Pain (IASP) as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'.[1] Since pain is highly subjective, it may also be described as being 'what the patient says it is'. Pain is an individual, multifactorial experience influenced, among other things, by culture, previous pain experience, belief, mood and ability to cope. Pain may be an indicator of tissue damage but may also be experienced in the absence of an identifiable cause. The degree of disability experienced in relation to the experience of pain varies; similarly there is individual variation in response to methods to alleviate pain.[1]

Pain may be classified according to its presumed aetiology. *Nociceptive pain* is due to the stimulation of nociceptors by noxious stimuli, whereas *neuropathic pain* is the result of dysfunction of the nervous system. Although these may coexist as mixed pain, the acute post-surgical pain pattern is regarded as nociceptive.

Pain may also be classified into *somatic* and *visceral pain*, for example pain from cholelithiasis, renal calculi, etc. It is important to understand the type of pain to direct appropriate treatment and to understand the need for traditional and non-traditional analgesia.

An alternative classification is based on duration. Acute pain is defined as 'pain of recent onset and probable limited duration. It usually has an identifiable temporal and causal relationship to injury or disease'. The point at which acute pain becomes chronic has been suggested at about 12 weeks or when the pain is no longer thought to be due to the initial insult. Chronic pain commonly persists beyond the time of healing of an injury and frequently there may not be any clearly identifiable cause.[1] It is increasingly recognized that acute and chronic pain may represent a continuum rather than distinct entities.[2,3]

Physiology of pain

The ability of the somatosensory system to detect noxious and potentially tissue-damaging stimuli is an important protective mechanism that involves multiple interacting peripheral and central mechanisms. The neural processes underlying the encoding and processing of noxious stimuli are defined as 'nociception'. The detection of noxious stimuli requires activation of peripheral sensory organs (nociceptors) and transduction into action potentials for conduction to the central nervous system. Nociceptors are stimulated by chemical, thermal or mechanical damage and trigger the nociceptive impulses. Nociceptive primary afferents are widely distributed throughout the body (skin, muscle, joints, viscera, meninges) and comprise both lightly myelinated A-delta fibres (diameter 2–5 μm) and slow-conducting unmyelinated C-fibres (diameter <2 μm). These fibres enter the dorsal horn of the spinal cord and synapse at different sites (Aδ at Rexed laminae II and V; C at Rexed laminae II). The substantia gelatinosa (lamina II) integrates these inputs and second-order neurons form the ascending spinothalamic and spinoreticular pathways on the contralateral side (Figure 1). The larger Aβ fibres conducting 'touch' and descending pathways stimulate inhibitory interneurons within the substantia gelatinosa and inhibit C fibre nociceptive inputs. This is the basis of the gate theory of pain. Pain may be modified by altering the neural pathway from its origin at the nociceptor to its interpretation within the central nervous system by various agents.

Psychological factors that influence the experience of pain include the processes of attention, other cognitive processes (e.g. memory/learning, thought processing, beliefs, mood), behavioural responses, and interactions with the person's environment.

Adverse physiological and psychological aspects of pain

Acute pain is one of the activators of the complex neurohumoral and immune response to injury. Both peripheral and central injury responses have a major influence on acute pain mechanisms. Thus acute pain and injury of various types are inevitably

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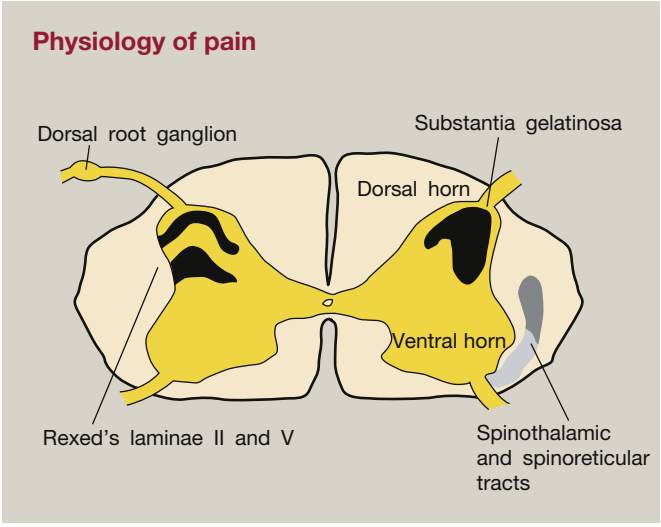

Figure 1

interrelated and if severe and prolonged, the injury response becomes counterproductive and can have adverse effects on outcome.[1]

Clinically significant injury responses can lead to a range of physiological effects that may lead to adverse clinical effects (Table 1). Patients at greatest risk of adverse outcomes from unrelieved acute pain include very young or elderly patients, those with concurrent medical illnesses and those undergoing major surgery.[1]

Sustained acute nociceptive input, as occurs after surgery, trauma or burns, can also have a major influence on psychological function, which may in turn alter pain perception. Failure to relieve acute pain may result in increasing anxiety, inability to sleep, demoralization, a feeling of helplessness, loss of control, inability to think and interact with others—in the most extreme situations, where patients can no longer communicate, effectively they have lost their autonomy.[4]

Effect of postoperative analgesia on surgical outcome

By reducing the unwanted effects of surgery on the pulmonary and cardiac systems and by reducing ileus, the stress response and thromboembolic complications, effective multimodal analgesia may improve patient morbidity. However, these benefits are only achieved when dynamic pain relief is targeted to allow early mobilization and other interventions are facilitated such as early feeding with analgesia forming only part of a multidisciplinary approach.[5]

Pain assessment

The assessment and measurement of pain are fundamental to the process of assisting in the diagnosis of the cause of a patient's pain, selecting an appropriate analgesic therapy and evaluating then modifying that therapy according to response. Pain should be assessed within a biopsychosocial model that recognizes that physiological, psychological and environmental factors influence the overall pain experience.

The assessment of acute pain should include a thorough general medical history and physical examination, a specific 'pain history' and an evaluation of associated functional impairment along with any side effects of treatment. In acute pain management, assessment must be undertaken at appropriately frequent intervals. It is useful to draw the distinction between the different types of pain because the likely duration of the pain and the response to analgesic strategies may vary.

Pain history and contents

SOCRATES is a useful mnemonic acronym commonly used by healthcare professionals to evaluate pain (Box 1).

The definition of pain underlies the complexity of its measurement. It is difficult to objectively measure the intensity of pain an individual is suffering from. This is usually assessed by individual patient's own report. These self-reporting measures may be influenced by mood, medication and sleep disturbance. Sometimes associated factors such as hyperalgesia (e.g. mechanical withdrawal threshold), the stress response (e.g. plasma

Effects and consequences of pain

System	Effects	Consequences	Clinical issues
Cardiovascular	Increased sympathetic activity	Increased heart rate, inotropy, and blood pressure resulting in increased myocardial oxygen demand	Risk of cardiac ischaemia, particularly with pre-existing cardiac disease
Gastrointestinal	Increased sympathetic activity	Reduced gastrointestinal motility	Ileus
Respiratory	Ineffective cough and reduction in functional residual capacity	Inability to cough effectively and a reduction in functional residual capacity	Atelectasis, ventilation-perfusion mismatch and hypoxaemia
Metabolic	Activation of sympathetic and hormonal systems (catecholamines, cortisol, endorphins, etc.)	Inflammation, hyperglycaemia, protein catabolism, increased free fatty acid levels (lipolysis)	Delayed wound healing, reduced immune function
			Diminished muscle strength
		Changes in water and electrolyte flux	Poor wound healing
Other	Reduction in patient movement	Hypercoagulable state	Thromboembolism
	Suppression of cellular and humoural immune function	Reductions in natural killer cell function, proliferative responses	Possible effects on tumour growth/ metastasis

Table 1