Hyperalgesia is observed in postoperative patients as primary hyperalgesia at the site of the surgical wound, and as secondary hyperalgesia in the healthy tissue surrounding the surgical wound (Johnson, 2009).

The intensity of the noxious stimuli and the chemical mediators are responsible for initiating an action potential in the nociceptor. The action potential is then converted to a nerve impulse, which is felt as pain.

Transmission
The nerve (pain) impulse is transmitted along the A delta and C fibres from the PNS to the dorsal horn of the spinal cord, where the A delta and C fibres terminate (Fig 1).

The nerve impulse transmits information about the noxious stimuli, including:
- Intensity of pain: for example sharp or severe, which is associated with the A delta fibre; and dull and aching, associated with the C fibre;
- Location: for example the incision site, intravenous cannula site or wound drain site.

Various neurotransmitters, such as glutamate and substance P, are released in the spinal cord in response to noxious stimuli. These neurotransmitters enable the transmission of the nerve impulse across the spinal cord and along the ascending spinal pain pathways to the brain stem (ANZCA, 2010).

Perception
Perception is the process whereby pain becomes a conscious sensation.

The brain does not have a single centre associated with pain. There is a complex interplay between many centres, which depends on their activation by pre, intra and postoperative neuronal, psychological, environmental and social influences.

The level of this activation and interplay of the brain centres depends on patients’ individual pre and postoperative circumstances, including:
- Fear of postoperative pain;
- Previous pain experience;
- Emergency or elective surgery;
- Pre-operative anxiety;
- Gender;
- Culture;
- Age.

This interplay of information between the different centres of the brain is termed the “pain matrix” and is not fully understood. The overall evaluation of this information will result in the patient’s own perception of their POP.

An evaluation of patients’ pre and postoperative psychological and environmental influences should therefore be carried out.

Modulation
The transmission of the nerve impulse associated with noxious stimuli can be inhibited or facilitated via the peripheral A beta fibre (sensory cutaneous) stimulation and/or the descending modulatory pain pathway (DMPP) from the brain to the spinal cord. This is referred to as modulation (Fig 1).

Transcutaneous electrical nerve stimulation (TENS), massage, acupuncture and heat/cold are examples of non-noxious A beta fibre stimulation. If A beta fibre stimulation is strong enough, nerve impulses will inhibit or partially inhibit the A delta and C fibre nerve impulse from travelling across the spinal cord. Consequently the perception of POP is inhibited or partially inhibited. There is limited evidence to support TENS, massage, heat/cold for effective POP management (ANZCA, 2010).

Persistent POP may cause changes in the A beta fibres. This includes a change in the balance of mediatory and inhibitor chemicals at the receptor and fibre sites, altering the response to non-painful stimuli, which results in non-painful stimuli being transmitted to the CNS as noxious information. This may result in the patient perceiving the non-noxious stimuli to be painful, and is called allodynia.

<table>
<thead>
<tr>
<th>TABLE 3. RISK FACTORS ASSOCIATED WITH CHRONIC POST SURGICAL PAIN</th>
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<tbody>
<tr>
<td><strong>Preoperative factors</strong></td>
</tr>
<tr>
<td>Pain, moderate to severe, lasting more than one month; repeat surgery; psychogenic vulnerability; preoperative anxiety; female gender; younger age (adults); workers’ compensation (patients involved in a compensation case regarding the cause of the injury/pain); genetic predisposition; inefficient diffuse noxious inhibitory control (the body’s natural pain control mechanism system is no longer effective)</td>
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<tr>
<td><strong>Intraoperative factors</strong></td>
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<tr>
<td>Surgical approach with risk of nerve damage</td>
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<td><strong>Postoperative factors</strong></td>
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<tr>
<td>Pain (acute, moderate severe); radiation therapy to area; neurotoxic chemotherapy; depression; psychological vulnerability; neuroticism; anxiety</td>
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<tr>
<td><strong>Sources</strong></td>
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<tr>
<td>ANZCA (2010); Kehlet et al (2006); Macrae (2008)</td>
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