Understanding the experience and physiology of pain

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Summary
This article, the third in a series of articles written by pain nurse specialists, explains the underlying physiology of pain. Nociceptive pain describes the normal physiological process relating to tissue damage, and neuropathic pain occurs when there is damage to or dysfunction of the nervous system. These two types of pain are analysed using a case study to provide insight into the multidimensional nature and unique experience of pain.

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PAIN IS A PERSONAL, emotional and subjective experience. Each painful event is unique in terms of underlying physiology, cognitive and emotional response, and social, cultural and financial contexts. This article uses a case study to explore the physiology of pain and illustrates the factors that make pain an individual experience even if people have undergone the same procedure or have a similar injury. An understanding of the physiology of pain is essential. However, it is important to note that the interplay of numerous factors make pain a unique experience that is complex in nature. Appreciating this complexity helps nurses to address the patient’s needs and promote effective pain management.

Case study
Charles is a 42-year-old man who has recently undergone an amputation of his hand following a road traffic accident. He works as a courier and was answering his mobile phone when he drove into the path of an oncoming vehicle at a road junction. He is a married father of three children and his wife works part-time on night shifts at a local residential home.

Physiology of pain
Pain has traditionally been categorised into three main types:
- Acute pain that lasts for less than three months and subsides with healing.
- Persistent or chronic pain that lasts beyond the normal period of healing or has an unidentifiable cause.
- Cancer pain associated with a malignancy.

Pain may be considered as being either nociceptive, a term that describes the normal physiological process relating to tissue damage, or neuropathic, which relates to pain caused by damage to, or dysfunction of, the nervous system. As with many conditions, the article’s case study will have an element of both nociceptive and neuropathic pain, and each will be explored.

Nociceptive pain
Charles has experienced tissue damage associated with his injuries and subsequent surgery. Nociception has a protective function that alerts the patient to the damage and pain. It is a continual reminder of the need to protect the damaged area until healing is complete.
The term nociception describes the process beginning with a painful stimulus or tissue damage, transmission of electrical nerve impulses to the spinal cord and brain, conscious awareness of pain, and alteration of pain signals (McCaффery and Pasero 1999). These stages are summarised as (McCaффery and Pasero 1999):

- Transduction.
- Transmission.
- Perception.
- Modulation.

**Transduction.** A noxious stimulus is converted to an electrical signal in the nerve cells through the process of transduction. Cells may be damaged by extremes of temperature, laceration, compression, shearing, inflammation, chemicals, localised oedema, dehydration and/or infection. This damage causes the cells to release a number of chemicals, including prostaglandins, histamine, bradykinin and serotonin, which initiate wound healing and sensitise nerve endings. Free nerve endings (nociceptors) are distributed throughout the body and pick up chemical, mechanical and thermal information and convert it into an electrical signal to be transmitted to the spinal cord. Local anaesthetics such as lidocaine work at this local level of pain transmission and prevent the signal, or action potential, from being formed.

Damaged cells in Charles’s wound stump will have sensitised the nociceptors and perhaps activated silent nociceptors that are usually dormant until inflammation occurs. This contributes to worsening of the pain some time after the injury as the inflammatory process becomes established. Innocuous stimuli, such as touch and movement, become painful (peripheral sensitisation). Sensitivity of the wound site, known as primary hyperalgesia, occurs as well as of the surrounding tissues, referred to as secondary hyperalgesia.

**Transmission.** During the transmission stage, information about the electrical signal is relayed along two types of peripheral nerve fibre (A-delta and C fibres) (Table 1) to the dorsal horn in the spinal cord. A-delta fibres are covered in a fatty myelin sheath that insulates the fibres, ensuring that the signals travel quickly. These nerves are responsible for the localised, sharp, stabbing pain that initially happens with injuries, alerting the person to the damage. C fibres are unmyelinated so the signals travel slower and create the general, dull, throbbing pain that continues after an injury to try to promote rest and prevent further damage (Melzack and Wall 2008). C fibres can alter the rate of firing of the signals depending on the intensity of the painful stimulus (Urch 2007).

A-delta and C fibres are found in different concentrations around the body. For example, the skin and muscle have a greater proportion of A-delta fibres than visceral organs, where C fibres dominate (Melzack and Wall 2008). Abdominal pain is therefore significantly different and more generalised than pain arising from a skin laceration. Given the extent of Charles’s injury and surgery, both types of nerve fibre will be involved in transmission to the spinal cord (Figure 1).

Neurotransmitters help impulses travel between nerves, and the electrical signal travels up the spinothalamic and spinoreticular tracts in the spinal cord. These tracts travel through the brainstem and end in the thalamus of the brain.

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**TABLE 1**

<table>
<thead>
<tr>
<th>Type</th>
<th>Responds to</th>
<th>Diameter</th>
<th>Speed</th>
<th>Myelination</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Touch, pressure</td>
<td>5-15μm</td>
<td>Fast: 30-100m/sec</td>
<td>Myelinated</td>
</tr>
<tr>
<td>Delta</td>
<td>Pain: mechanical and thermal stimuli</td>
<td>1-5μm</td>
<td>Fast: 6-30m/sec</td>
<td>Unmyelinated</td>
</tr>
<tr>
<td>C</td>
<td>Pain: mechanical, thermal and chemical stimuli</td>
<td>0.25-1.5μm</td>
<td>Slow: 1.0-2.5m/sec</td>
<td>Unmyelinated</td>
</tr>
</tbody>
</table>

(Adapted from Melzack and Wall 2008)

**FIGURE 1**

*Nociceptive pain*
The thalamus processes sensory and motor information acting as a relay station to various regions in the brain (Melzack and Wall 2008). The autonomic (stress) response to noxious pain is created in the brainstem, increasing the physiological response (Table 2); and these effects can increase the risk of complications in people with comorbidities or in the acute phase of their recovery. As with any physiological measurement, a number of factors influence these parameters. This means that by themselves, vital signs are unreliable indicators of pain. This stress response is also significantly reduced in persistent pain conditions.

**Perception** This stage is characterised by conscious awareness of pain. The somatosensory cortex helps to locate and interpret pain. Arousal, motivation and emotions are controlled by the reticular activating and limbic systems (Melzack and Wall 2008). The person is made aware of pain, responds emotionally and is motivated to take action. Research has provided clues about the role of memory in pain. Studies by Taddio et al (1997, 2002) compared young infants exposed to procedures such as repeated heel pricks and circumcision without local anaesthetic. Following up these children at their first immunisation, those who experienced painful events four to six months previously had a greater physiological and behavioural reaction to the injection compared with control groups. This suggests that these children were sensitised to pain because of their early experiences. It is likely that experiences of pain throughout life are a key part of one’s physiological and emotional memory, contributing to the uniqueness of any pain experience.

**Modulation** The human body modulates pain experiences as the final stage of nociception, and a number of mechanisms decrease or increase pain impulses. The body produces endogenous opioids (endorphins, enkephalins and dynorphins) that inhibit noxious stimuli – variations in opioid production may be one factor that explains why people undergoing the same procedure experience different levels of pain.

Descending pathways from the brain extend to the dorsal horn in the spinal cord (Figure 1). They can act as a control mechanism, similar to a volume control, for the pain signals, amplifying or reducing the messages reaching the brain. Attention, distraction and sensory input can dampen the signals and perception of pain, particularly in mild to moderate pain. Pain experienced at night may actually be worse because of the lack of sensory input. This, along with the action of endogenous opioids and adrenaline, explains why a sportsperson, for example, may not notice an injury until after a competition. Children, in particular, are adept at using play as a distraction technique when in pain. Adults may also experience a reduction in pain through socialising, reading, listening to music and watching television. The technique of guided imagery takes advantage of this process by helping people to visualise an alternative place or journey to distract them from the pain, induce relaxation and boost endogenous opioids. The mechanism of inhibiting or amplifying pain signals sent from the spinal cord to the brain was a major component of the gate control theory of pain proposed by Melzack and Wall (2008).

A-beta fibres are large fibres that carry information about non-noxious stimuli such as touch (Table 1). These nerves transmit impulses faster than A-delta or C fibres and stimulating them can reduce or prevent pain signals from being transmitted to the brain. This is the mechanism behind ‘rubbing it better’ for mild pain and transcutaneous electrical nerve stimulation (TENS). A TENS machine is a small electrical device that attaches to the skin via electrodes. Low-intensity electrical impulses stimulate the A-beta fibres and, after a short period of time, TENS may also encourage endogenous opioid production (Nnoaham and Kumbang 2008).

Some factors, including fear, anxiety and depression, increase pain and amplify pain signals. In Charles’s situation, his accident will have had a major effect on his role and body image. It may result in the loss of his job and income, a police investigation and claims from other injured parties. These fears and anxieties will have a negative effect on his pain. A key part of Charles’s recovery and rehabilitation will

**TABLE 2**

<table>
<thead>
<tr>
<th>Multidimensional effects of pain</th>
<th>Cognitive, emotional and social consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiological effects (acute pain only)</td>
<td>Cognitive, emotional and social consequences</td>
</tr>
<tr>
<td>Increased heart rate, but decreased myocardial blood flow.</td>
<td>Urged to obtain relief.</td>
</tr>
<tr>
<td>Increased blood pressure.</td>
<td>Reduced cognitive function.</td>
</tr>
<tr>
<td>Increased respiration rate, but decreased depth of inhalation.</td>
<td>Anxiety and fear.</td>
</tr>
<tr>
<td>Elevated blood sugar levels.</td>
<td>Irritability and/or aggression.</td>
</tr>
<tr>
<td>Decreased gut motility.</td>
<td>Depression.</td>
</tr>
<tr>
<td>Nausea and vomiting.</td>
<td>Withdrawal.</td>
</tr>
<tr>
<td>Increased sodium and water retention.</td>
<td>Reduced appetite.</td>
</tr>
<tr>
<td>Muscle spasms and ‘splinting’ of wound.</td>
<td>Sleep deprivation.</td>
</tr>
<tr>
<td></td>
<td>Reduced mobility.</td>
</tr>
<tr>
<td></td>
<td>Suicidal thoughts or loss of will to live.</td>
</tr>
<tr>
<td></td>
<td>Change in family or social roles.</td>
</tr>
<tr>
<td></td>
<td>Effect on school or employment.</td>
</tr>
</tbody>
</table>
therefore be to support him and his family in coping with these issues and ultimately reduce his pain.

The physiology of the human body is altered in the presence of pain. The intensity and frequency of input from the peripheral nervous system influences the central nervous system to make chemical and anatomical changes. Spinal neurones become more sensitive with a lower threshold for activation and they increase their receptive field, picking up signals from a wider range of peripheral nerves. This process is referred to as central sensitisation (Melzack and Wall 2008).

The normal physiology of nociceptive pain begins to explain the individuality of pain and how it is a multidimensional experience involving physiological, cognitive, emotional, social and cultural factors. More importantly, it becomes possible to see how these factors are related and influence each other, making it difficult to separate the physiology of pain from the effect it has on a person.

Neuropathic pain

As a result of his amputation, Charles will not only experience nociceptive pain, but also neuropathic pain. Neuropathic pain has no protective function and is described as pain arising as a result of nerve lesions, damage or dysfunction of the nervous system (Urch and Dickenson 2008). This can occur in the peripheral and/or central nervous system.

Charles described a typical pattern of neuropathic pain sensations, including stabbing, shooting and burning. Spontaneous pain occurs, often accompanied by tingling, pins and needles, and numbness. Allodynia – induced pain from non-painful stimuli such as a light touch – may also occur (Johnson 2009). The pathophysiology of neuropathic pain is complex and is not understood fully, but a number of mechanisms are thought to be involved (Box 1).

Central sensitisation that develops as part of nociception should disappear as healing occurs, but in some cases it can continue and become permanent. This sensitisation can be prevented through effective pain management during acute or postoperative recovery. Research continues to explore the reasons why, in some people, this process does not resolve on healing and they continue to experience neuropathic pain.

The plasticity of the nervous system means that a number of other changes occur in the spinal cord. The increased receptive field for peripheral nerve input may continue, interneurones can die and there may be an increase in neurotransmitter release. Where there is a loss of input from the peripheral nervous system through nerve damage or avulsion – where the nerve is torn away from its origin – interneurones in the spinal cord may spontaneously transmit signals to the brain.

In addition, the descending inhibitory mechanism that attempts to modulate pain may be affected in people with neuropathic pain. This means that endogenous opioid production and the pathways that decrease pain may not be effective. Peripheral nerves may also undergo changes as a result of nerve damage. Increased excitability and spontaneous firing of neurones may occur and they alter after injury. During attempts at repair and regeneration, sprouting and enlarged nerve tissue forms a structure called a neuroma. Neuromas have the ability to produce random signals along the nerve pathway (Johnson 2009).

With the combination of changes to the peripheral and central nervous system, it is possible to see how there can be pain in the absence of injury, or after an injury has healed, and why there are such variations in the type and intensity of neuropathic pain. It is also possible to understand why the pain experience described by individuals can be complex and vary greatly.

Phantom limb pain

Charles may experience phantom sensations and phantom limb pain. This may occur both
in traumatic and surgical amputations. Nikolajsen and Jenson (2005) referred to the experience of people who have undergone amputation as a ‘phantom complex’ and highlighted the importance of distinguishing between the following:

1. Phantom pain – painful sensations felt from the missing limb.
2. Phantom sensation – other sensations felt from the missing limb.
3. Stump pain – pain directly from the amputation stump.

Each of these related elements should be considered when planning a multimodal approach to pain management. In the early stage of his recovery, Charles’s experience will have both nociceptive and neuropathic elements as a result of the surgical wound and nerve damage. The mechanism for the development of phantom limb pain is not fully understood, but theories relate to the peripheral (peripheral sensitisation occurs and damaged nerves form neuromas), spinal (central sensitisation) and central theories. Central theories relate to the reorganisation of the somatosensory cortex, suggesting that the area of the brain previously responsible for the limb is taken over by an adjacent area. This may explain why brushing a cheek can produce phantom sensations in the arm, but may also contribute to the pain sensation (Richardson 2008). The neuromatrix theory relates to the abnormal reorganisation of the matrix of neurones responsible for that area following amputation. These mechanisms are explored in more detail later in this series of articles, and research continues to pursue the understanding, prevention and treatment of phantom limb pain.

Cancer pain

The physiology of cancer pain is a mixture of nociceptive and neuropathic pain. Pain can also arise because of changes in the peripheral neurones or nerve compression as a result of a growing tumour; it may also have an inflammatory element (Urch and Dickenson 2008). The variations in the type, location and degree of nociceptive or neuropathic pain explain the individual nature of cancer-related pain. Many cancer treatments, such as surgery, chemotherapy and radiotherapy, can also cause pain that requires effective management.

Conclusion

Understanding the multidimensional nature and physiology of pain provides an insight into the unique experience of pain. This complexity demonstrates the need for a comprehensive assessment, empathic support of the individual and a multimodal approach to pain management involving pharmacological and non-pharmacological treatments. This series of articles explores these approaches and the nursing care of people with specific pain management needs NS.

Acknowledgement

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References