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FOCUS ON: PAIN MEDICINE APPLICATIONS

Novel trends in pain management – Neuromodulation

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SUMMARY

Neuromodulation is one of the most exciting developments in pain management. Spinal cord stimulation, peripheral nerve stimulation and intrathecal drug delivery systems are used increasingly to provide pain relief and improve the quality of lives of patients in whom conventional medical management has failed to provide satisfactory results. Though initial costs may be high, these techniques have proven to be cost effective in the long term. A good understanding of the principles and techniques involved in neuromodulation and their benefits and limitations is essential to achieve best results.

Neuromodulation is one of the most exciting developments in pain management. It involves the alteration of action of central, peripheral and autonomic nervous systems using electrical stimulation or intrathecally applied pharmacological agents. Besides improving the quality of lives of patients suffering from certain types of chronic pain, neuromodulation also finds application in treatment of spasticity, movement disorders, epilepsy, intractable angina, peripheral vascular disease and certain psychiatric disorders. An overview of various techniques used in neuromodulation is given in Table 1. In this article, we will focus mainly on spinal cord stimulation, peripheral nerve stimulation and intrathecal drug delivery systems. Other techniques are more relevant to neurosurgeons and are beyond the scope of this article.

1. Spinal cord stimulation

Spinal cord stimulation (SCS) or dorsal column stimulation (DCS) has been part of the pain clinician's armamentarium for the past four decades. However, it is only in the last decade that firm evidence of its benefits in the management of chronic pain has emerged. Spinal cord stimulation (SCS) evolved from the gate control theory of pain proposed by Melzack and Wall in 1965.[1] The gate control theory states that activity in large diameter cutaneous afferents (Aβ fibres), which generally carry non-painful touch sensations, inhibits activity in small diameter afferents (Aδ and C) which generally transmit painful sensations. Electrical stimulation of Aβ fibres in the dorsal columns or dorsal roots results in reduction in the perception of pain. In 1967, electrical stimulation of the dorsal white column was introduced by Shealy and colleagues to treat chronic pain.[2]

Electrical stimulation is achieved by epidural or subdural electrodes controlled either via an external radiofrequency transmitter or a completely implantable, battery driven, externally programmed pulse generator. In early years, subdural electrodes were implanted surgically and this required laminectomy or partial laminotomy. These plate electrodes were sutured to the dura and this occasionally led to CSF leakage. Subdural placement of electrodes also caused local fibrosis. To avoid these complications, epidural electrodes are now used. With improvements in technology, percutaneous electrodes have been introduced which can be implanted via a modified Tuohy epidural needle, without the need for laminotomy.[3] Early systems used monopolar electrodes; later, bipolar, quadripolar and octapolar electrodes became available (Figs. 1 and 2). Electrical pulses are generated through external radiofrequency transmitters or pulse generators implanted subcutaneously, usually in the anterior or lateral abdominal wall. A period of trial stimulation is usually employed to assess the potential response to a permanent system. The trial generally consists of insertion of a transcutaneous epidural electrode under local anaesthetic and this is connected to an external current generator. The patient is asked to indicate the position of paraesthesias on his/her body. The electrode is then moved in the epidural space until the painful zone is covered with painless paraesthesias. The use of trial stimulation prior to definitive implantation is widespread but not universal. The trial period may vary between a few minutes in some centers to 4 weeks in others. During this period, both patient and physician can assess the success of the therapy and arrive at the most appropriate level of stimulation by altering the generator settings depending on the activities of daily living of the patient, degree of pain and any discomfort due to

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Table 1

Neuromodulation techniques
Spinal cord stimulation
Peripheral nerve stimulation
Intrathecal drug delivery systems
Deep brain stimulation
Motor cortex stimulation
Cranial nerve stimulation
Functional electrical stimulation

stimulation. Experience has shown that a trial will demonstrate that appropriate paraesthesias can be achieved in a particular patient, will indicate the optimal placement and to some extent, 'educate' the patient about the effects of permanent implantation. It will not, however, reliably predict long-term outcome of the therapy.[4]

The mechanism by which SCS works is not clearly known. Various mechanisms have been postulated to explain how SCS brings about pain relief in different chronic pain conditions. The gate control theory still forms the basis of SCS. Stimulation of large fibres transmitting non-painful sensations is thought to close the gate to transmission of nociceptive signals through smaller diameter afferent fibres. This theory does not, however, entirely explain the mechanism of action of SCS because all types of pain are not equally suppressed. A conduction block in spinothalamic fibres is proposed by some authors,[5] whereas some others think that supraspinal mechanisms involving spinobulbar, spinocortical and spinothalamic connections may be implicated.[6] In neuropathic pain states, SCS may inhibit hyperexcitability of the wide dynamic range (WDR) cells in the dorsal horn. Increased GABA release as a result of SCS is thought to inhibit release of excitatory amino acids like aspartate and glutamate in the dorsal horn which account for phenomena such as allodynia and hyperalgesia.[7] The effects of SCS on ischaemic pain may be due to inhibition of sympathetic activity and antidromic vasodilatation mediated by release of calcitonin gene related peptide (CGRP).[7]

1.1. Indications FOR SCS (Table 2)

1.1.1. Angina pectoris

Over the past 10 years, SCS has become a promising therapeutic option in patients with intractable angina, especially those in whom conventional treatments have failed. In 1987, Murphy and Giles reported the use of SCS for angina pectoris.[8] The mechanism of action of SCS includes reduction in pain perception, reduction in myocardial oxygen consumption, reduced sympathetic outflow and antidromic vasodilatation.[9,10] There is also evidence for a direct anti-ischaemic effect on the myocardium and improvement in exercise tolerance and electrocardiographic and echocardiographic changes.[11,12] Up to 80% of patients with severe coronary artery disease that is unresponsive to drug treatment or revascularization procedures benefit from it. Studies suggest that the efficacy of spinal cord stimulation in angina is similar to that of coronary artery bypass surgery.[13] Therefore SCS may be a therapeutic option in high risk patients who are unsuitable for surgery. A long-term follow-up study has shown beneficial effects in patients with intractable angina pectoris including pain relief, more control over anginal attacks and improvement in quality of life.[14] In SCS for intractable angina, the stimulating electrode is generally placed in the upper thoracic posterior epidural space to the left of the midline. This provides paraesthesia in the area corresponding to angina pain.

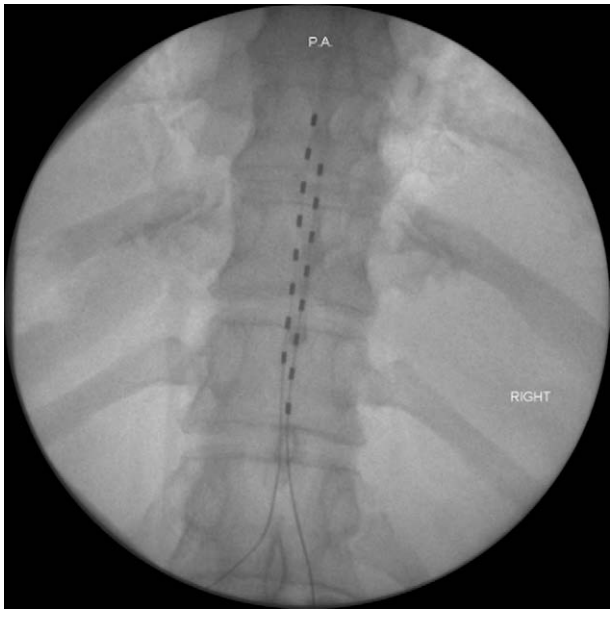

Fig. 1. Dual octapolar electrodes placed for treatment of FBSS pain.

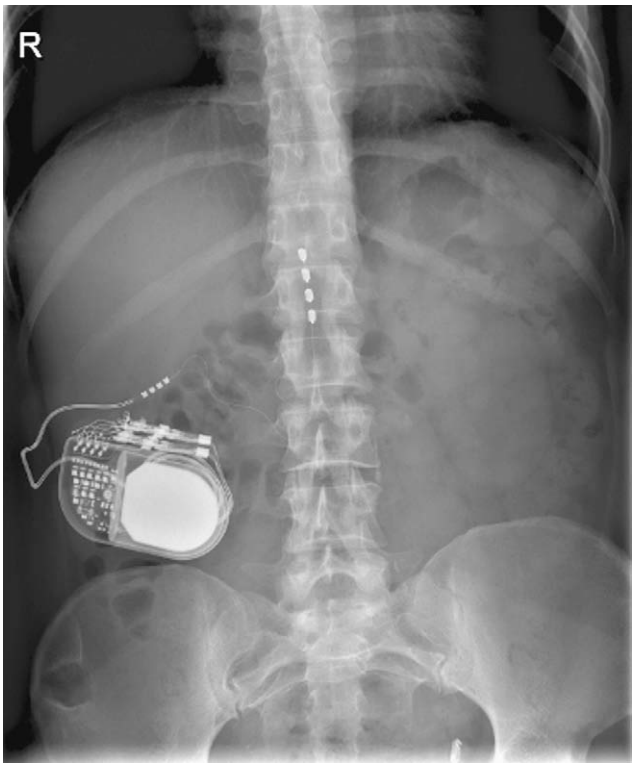

Fig. 2. Surgical paddle electrode and implanted pulse generator for CRPS type II of lower limb.

1.1.2. Peripheral vascular disease

SCS is increasingly used in the treatment of peripheral vascular disease especially in Europe. It is particularly effective in conditions

Table 2

Common indications for SCS
Failed back surgery syndrome (FBSS)
Refractory angina pectoris
Peripheral vascular disease
Complex regional pain syndrome (CRPS)
Neuropathic pain secondary to peripheral nerve damage
Post-herpetic/post-thoracotomy neuralgia
Post-amputation phantom limb pain