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Case Report

Augmentation of phantom limb pain by normal visceral function

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Following major nerve injury or amputation, a number of changes occur both peripherally and centrally within the nervous system due to the deafferentation process. The occurrence of phantom-type sensations are extremely common after this type of injury. These sensations are painful in many cases, especially if there was pain immediately before deafferentation. Two cases are described which are unusual, since in addition to pain within the distribution of deafferentation, there is visceral involvement.

Case 1 A twenty-three year old male involved in a motor cycle accident sustained major trauma to his left buttock, including deep lacerations, a fracture of his left greater trochanter and shredding of the sciatic nerve. He described the pain in his leg as a shooting, stinging and stretching sensation especially around his left foot. He also had pain in his left buttock while sitting, with reduced sensation of his lower leg and the posterior aspect of his thigh. Defaecation and micturition intensified the pain and he had difficulty sleeping.

On examination foot drop was present, together with, muscle wasting of lower left leg and foot, in keeping with an S2-S3 lesion. He was treated with co-dydramol, amitriptyline. 50mg nocte and ibuprofen. Later carbamazepine 800mg per day together with transcutaneous nerve stimulation was also used. It was only possible to achieve minimal relief of his pain with the above therapy and gradually each of these therapies were withdrawn.

Case 2 A forty-five year old male was injured in an explosion. His injuries required amputation of some of his right upper-limb digits together with a left above knee amputation. He also had vascular and bony injuries to his right leg. He was first seen by the pain team twenty days after the incident. His symptoms were sleep disturbance and pain in both legs, including a phantom left leg pain. His initial treatment was with celecoxib 200mg for the soft tissue injury and slow release oxycodone 20mg bd for the multiple aching pains. An escalating dose of an anticonvulsant active at the calcium channel site, (gabapentin to1800mg per day) was added for the phantom pain. Nine days after starting the above treatment, he had no aching pain so oxycodone was reduced to 10 bd. The phantom limb pain and sleep disturbance were less troublesome so gabapentin was continued at the previous dose.

Twelve days later his left phantom pain became much more intense than before, but only on micturition. On further questioning it became clear that the exacerbation of his phantom limb pain occurred only after his urinary catheter was removed. The dose of gabapentin was increased to 2400mg per day and an anticonvulsant active at the gamma-amino-butyric acid (GABA) receptor site, was added (baclofen 10mg qds). Since the pain on micturition did not improve on this combination of anticonvulsants after three days, his baclofen was discontinued and an anticonvulsant active at the sodium channel (carbamazepine in a rapidly escalating dose) was added to the gabapentin.

Three weeks later there was no improvement in his micturition induced phantom limb and on discharge for rehabilitation his medications were gabapentin 1800mg per day together with carbamazepine retard 400mg bd on a declining dose, with a view to using gabapentin alone since it controlled the phantom limb pain at rest and he could now tolerate the pain associated with micturition.

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DISCUSSION

Phantom limb pain occurs among most amputees and is difficult to treat[1]. Similar sensations and problems occur following nerve trunk injury. In the early part of the last century it was thought that phantom limb pain was a psychiatric problem due to unresolved grief for the missing limb. In the last few decades, phantom limb pain has been recognised as a distinct pain syndrome, in which a sensation is perceived in the denervated or missing limb. Both peripheral and central factors cause phantom limb pain[2].

Stress magnifies the pain as in most chronic pain states. Phantom limb pain is frequently described as a burning, cramping, shocking or shooting sensation. Peripheral factors include muscle spasm in the residual limb and this can lead to the tight, cramping sensations often experienced. Phantom "exercises" can be carried out to alter the muscle tension in the residual limb and this has been found to reduce this quality of pain. Surface blood flow around the stump is often altered in association with the nerve injury, due to the cross-coupling of sensory with sympathetic fibres, resulting in a burning sensation. These burning sensations often respond to interventions that increase blood flow to the residual limb, for example, beta blockers by reducing the sympathetic activity within the vasculature of the limb. Ectopic discharges from the neuroma within the stump, together with those within the dorsal root ganglion and the dorsal horn of the spinal cord are respondible for the intermittent sharp, shooting pains. These sensations are due to central factors such as reorganisation of the amputation zone within the somatosensory cortex where a sensory memory map exists[3]. Indeed it has been shown that suppression of afferent input from the amputation stump by brachial plexus anaesthesia eliminates both cortical reorganisation and phantom limb pain in approximately 50% of subjects[4]. In some amputees, cortical reorganisation and phantom limb pain are maintained by a peripheral input, whereas in others intra-cortical changes are more important.

In 1988 Bach *et al* carried out a controlled study of lumbar epidural blocks prior to amputation[5] and concluded that pre-emptive analgesia resulted in less phantom pain but recent studies have been equivocal about the value of this technique. Loss of a limb or major nerve injury pain management revolve around the use of N-methyl D-aspartate (NMDA) antagonists and GABA agonists, as these are directed at modifying the progression of spinal sensitization and cortical reorganization respectively. In the second of the two cases described, the patient experienced phantom limb pain which was successfully treated with gabapentin. This drug is an alpha-2-delta calcium channel blocker which has a therapeutic range similar to that used in the management of epilepsy.

The further development of micturition related phantom pain can be explained by the physiology of sympathetic-coupling to somatic nerves. Nerve laceration triggers a massive sprouting of sympathetic fibres at the injury site and increased activity within the dorsal root ganglia. This is associated with an upregulation of alpha-adrenergic receptors in the primary afferent neurons. These receptors mediate the excitatory effects of postganglionic sympathetic efferent[6]. It has been shown that there is considerable cross-talk within neuromas, between efferent sympathetic nerves and afferent nociceptors[4]. Therefore any stimuli in the body which increases sympathetic tone will also increase the likelihood of phantom limb pain or nerve injury pain. The use of alpha-adrenergic antagonists can be effective in managing this aspect of the pain.

With an understanding of the factors involved in phantom limb pain treatment can be more effectively orientated towards altering these unpleasant perceptions:

- Educating patients about phantom sensations, to alleviate stress.
- Anticonvulsants to reduce spontaneous neuronal firing of deafferent nerves
- Alpha-adrenergic antagonists to reduce sensory-sympathetic coupling
- Beta blockers to improve circulation to the stump

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