Medical management of facial pain

HC Romer

This article describes some of the means used to manage facial pain once serious pathology or any treatable cause has been excluded. It includes pharmacological, physical and psychological therapies.

Some facial pain can be managed by surgical or pharmacological treatment of the cause. However, signs of serious conditions, such as cranial tumours, must be excluded before the medical management of the pain itself starts.

PHYSIOLOGY OF PAIN

The physiology described below covers facial pain originating from the neck. If one considers the brainstem to be analogous to the dorsal horn, then the same explanation can be used for pain in the distribution of cranial nerves, accepting that, in fact, the physiology in these cases is more complex and less well understood.

Ascending pathways

Aδ and C nerve fibres transmit pain from the periphery to the spinal cord. They are the smallest fibres in the myelinated and unmyelinated groups. The myelinated Aδ fibres receive input from up to twenty mechanoreceptors per fibre and transmit the signal relatively fast (6–30 m/sec). They adapt rapidly to a constant stimulation, so the signal fades quickly. Conversely, each C fibre has one bare nerve ending acting as a polymodal nociceptor. Pain signals in C fibres travel at less than 2 m/sec to the spinal cord and adaptation to a constant pain is slow.

Pain fibres terminate in Rexed's laminae in the dorsal root horn of the spinal cord. A series of interneurones between lamina 2 (the substantia gelatinosa) and lamina 5 allow alteration of the signal before it reaches the spinal tracts.

Most pain fibres decussate to ascend in the contralateral spinothalamic tract. Connections exist with the thalamus, reticular formation, frontal lobe, hypothalamus, limbic system and sensory cortex (*Figure 1*).

Modulation of the pain pathway and descending pathways

Researchers continue to find more pathways and neurotransmitters involved in the modulation of the ascending pain signal. Of clinical significance here is that the descending tracts relying on serotonin (5HT) and noradrenaline as their neurotransmitters decrease ascending pain signals.

The descending tracts are thought to interact with the ascending pain pathway at the level of the dorsal root horn. It is also at this level that the modulations explained by Melzack and Wall's gate control theory of pain occur. Sensory input from the periphery, carried by Aβ fibres, can modulate and sometimes block the pain signal travelling through the network of interneurones ('close the gate') (*Figure 2*). Activation of N-methyl-D-aspartate (NMDA) receptors in this region leads to 'wind up' with recruitment of more interneurones and amplification of the pain signal.

More peripheral modulation of the pain pathway tends to increase pain. Antidromic flow in C fibres and efferent sympathetic flow cause release of substance P and noradrenaline respectively. Both of these, together with local inflammatory mediators such as bradykinin and prostaglandins, sensitize peripheral receptors.

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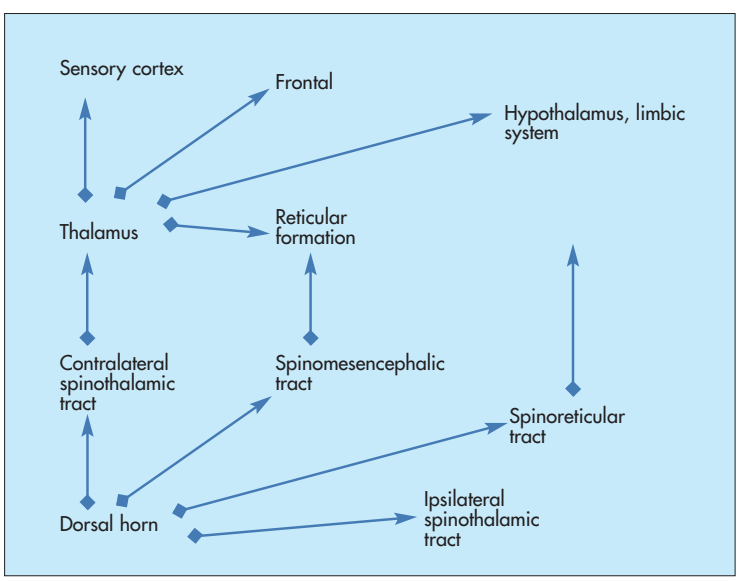


Figure 1. Ascending pain pathways.

PHARMACOLOGICAL MANAGEMENT OF CHRONIC PAIN

The World Health Organization's analgesic ladder for mild, moderate and severe pain in cancer can be used in chronic pain. Most patients will already have discarded simple analgesics. However, these are worth reviewing to check that they have been tried and also to ensure that non-steroidal anti-inflammatory drugs (NSAIDs) have not been overused, which can lead to medication misuse chronic daily headache.

Non-steroidal anti-inflammatory drugs

NSAIDs act in the periphery by decreasing inflammatory mediators and are also thought to act centrally via receptors in the spinal cord. The cyclooxygenase-2 (Cox-2) inhibitors, rofecoxib and celecoxib, are recent additions to the UK market. Designed to interact minimally with the 'housekeeping' Cox-1 prostaglandin receptor, they aim to have fewer side effects.

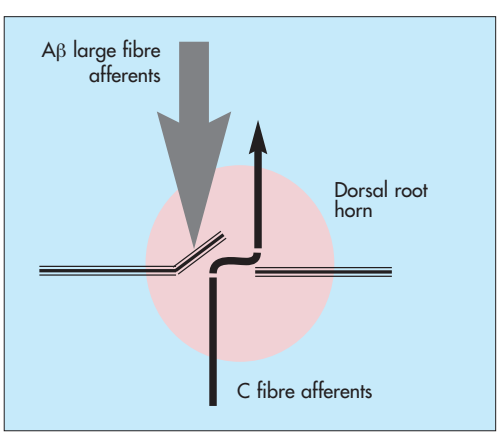
Indomethacin is especially worth mentioning as it is a particularly effective in the short-lived but frequent chronic paroxysmal headache (Goadsby, 1999).

Opiates

The use of opiates in chronic facial pain is controversial. Against their use are fears of addiction together with long-term failure as a result of habituation, ineffectiveness and intolerable side effects. Research into their success in neuropathic pain has mixed results, but a trial of treatment may be worthwhile.

The oral opiates include codeine, dihydrocodeine and tramadol as well as morphine, oxycodone and methadone for severe pain. Slow release forms and fentanyl patches are available once the dose has stabilized. In order to avoid increasing doses, drug rotation should be employed. The patient alternates between two or three different drugs over the course of a year, thereby taking 'drug holidays' from each substance. The risk of addiction or abuse is between 0–24% but lower in those with no previous psychiatric or abuse history (Dellemijn, 1999).

Figure 2. Diagrammatic representation of the gate control theory of pain.



Antidepressant drugs

This group of drugs includes amitriptyline, nortriptyline, doxepin, dothiepin and imipramine. Gabapentin, a structural γ-aminobutyric acid (GABA) analogue, works in a similar way to traditional antidepressants. Nortriptyline and gabapentin have fewer side effects (Beydoun, 1999; Kanazi et al, 2000) than amitriptyline, and it has been suggested that they should be used first out of this group.

Prescribed in lower doses than in depression, the antidepressants block the reuptake of biogenic amines, serotonin and noradrenaline in the brainstem, thereby increasing the effect of the descending modulatory spinal tracts. They also potentiate endogenous and exogenous opioids. They do not, at the dosage used in pain management, act as antidepressants, and this may need to be explained to the patient.

Antidepressants are particularly useful in pains of a burning nature, tension (Cerbo et al, 1998) and cluster headaches as well as in allodynia. They should be started at a low dose and increased gradually as benefits and side-effects dictate. Unwanted effects include thirst, constipation and dysphoria. Taken at night, drowsiness is an advantage. Other benefits may take 2–3 weeks to be noticeable.

Selective serotonin-reuptake inhibitors

This newer class of antidepressants includes fluoxetine, paroxetine and citalopram. They are used in the same way as older antidepressants but avoid the noradrenergic side effects of these drugs.

Sumatriptan is a cranioselective serotonin agonist of particular use in migraine. It has not shown clinical benefit in atypical facial pain.

Ion channel blockers

Anticonvulsant drugs: Carbamazepine, clonazepam, phenytoin and sodium valproate block sodium channels in active nerves, thereby decreasing the spontaneous impulses that can occur in damaged C fibres, which patients describe as 'stabbing' or 'sharp' pain (such as in trigeminal neuralgia) or an unpleasant 'pins and needles' sensation. Carbamazepine is the drug of first choice for the treatment of trigeminal neuralgia.

Others: Lignocaine infusions can relieve neuropathic pain but, not being available orally, are not clinically useful in chronic pain. Topical lignocaine has been successful in treating postherpetic neuralgia (Comer and Lamb, 2000). Oral mexiletine has had mixed results.

Lamotrigine stabilizes a subtype of sodium channels and suppresses neuronal release of glutamate. It can be used alone, or in combination with carbamazepine, in the treatment of neuralgia (Sindrup and Jensen, 1999).

NMDA antagonists

Ketamine is a non-competitive NMDA receptor antagonist which is thought to minimize the wind-up phenomenon. Intravenous ketamine has had good results in the treatment of neuropathic pain, and results of trials on the oral form are awaited. Oral dextromethorphan has a lower receptor affinity and is usually unsuccessful in treating facial pain.

The synthetic opiates, methadone and tramadol, block NMDA receptors in addition to their effects on opiate receptors.

Sedatives and antispasmodics

Diazepam can be of use in chronic orofacial muscle pain (Singer and Dionne, 1997). This may be as a result of its antispasmodic or anxiolytic effects.

Baclofen, a GABA receptor agonist, usually used as an antispasmodic, has been shown to be useful in the treatment of central pain neuralgias (arising after brain injury), cluster headaches (Hering-Hanit and Gadoth, 2000) and chronic daily headache from medication misuse.

Miscellaneous drugs

Capsaicin: The alkaloid capsaicin, applied as a cream to the hypersensitive area, causes release of substance P from sensory nerve terminals and also has effects in the spinal cord, possibly as a result of absorption or neuronal transport. Used regularly, this results in a depletion of substance P and a decrease in paraesthesia, dysaesthesia and allodynia (Lincoff et al, 1998). As the initial treatments are painful, it may be prescribed with EMLA cream for the first few days and needs to be used regularly.

Oxygen and ergotamine: Thought to cause transient cerebral vasoconstriction, oxygen can abort a cluster headache as can inhaled (nasal spray) or subcutaneous ergotamine (A Leach, personal communication, 2000).

Lithium carbonate: Lithium may be used in the prophylactic treatment for chronic cluster headaches but plasma levels must be monitored.

PHYSICAL MANAGEMENT OF CHRONIC PAIN

Transcutaneous electrical nerve stimulation

Using an electric current which is passed between two electrodes attached to the skin, transcutaneous electrical nerve stimulation (TENS) stimulates Aβ fibres and therefore modulates the pain sensation at the level of the dorsal root horn. With experimentation, the settings can be adjusted so that all sensation of pain is lost while the TENS machine is used and sometimes for some hours afterwards.

Drawbacks include difficulty in attaching the electrodes and skin reactions to the electrode glue. TENS should be tried for a variety of conditions, however, it is of particular use in myofascial pain deriving from neck and strap muscles.

Physiotherapy

Physiotherapy aims to restore normal muscle function, and it is of most benefit in myofascial pain where trigger points in the neck muscles cause radiation to distant areas (*Figure 3*). Stretching, mobilization, cooling sprays or ice are used with local anaesthetic injections and acupuncture. Where bruxism contributes to continued muscle malfunction, dental splints may be use.

Acupuncture

Acupuncture is used in its traditional form and in trigger point treatment. It has been successfully used in postherpetic neuralgia and atypical facial pain. Studies have shown that some of the benefits gained from acupuncture may be the result of patient motivation and placebo effect (McMillan et al, 1997).

Nerve blockade

Bupivacaine can be used to block the greater occipital and supraorbital nerves and cervical facet joints to treat cervicogenic headaches.

In order to abort a cluster headache, patients can be taught to perform a sphenopalatine block

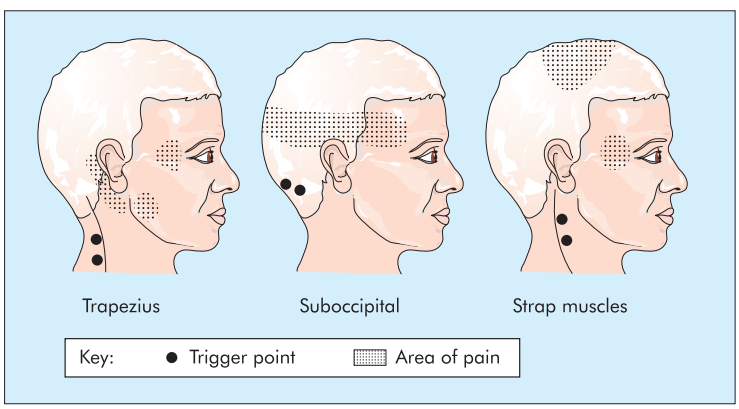


Figure 3. Trigger points and pain radiation.

by positioning two cotton buds soaked in local anaesthetic inside the nose.

Botulinum toxin

Botulinum injection into the masseter muscle can relieve spasm in temporomandibular dysfunction but should be carried out by someone experienced in this technique.

PSYCHOLOGICAL MANAGEMENT OF CHRONIC PAIN

The psychological state, coping mechanisms and personality of the patient will affect the perception of pain. Pain, in turn, will affect the patient's mental wellbeing. Ignoring this codependence will limit the success of pain management.

Clinical and latent depression

Psychiatric treatment may be necessary for the few patients for whom severe clinical depression, an anxiety state or a personality disorder is the underlying cause of pain.

Most patients will benefit from psychological support, ranging from a sympathetic consultation with a doctor who acknowledges their pain to active psychotherapy (Haythornthwaite and Benrud-Larson, 2000).

Cognitive-behavioural therapy

Chronic pain has been compared to a phobia, where the patient learns that a particular activity or movement causes pain. Over time, this can build up to the point where even thinking of the activity brings on the pain. Cognitive-behavioural therapy, together with biofeedback, seeks to support the patient while they change their behaviour, learn to pace themselves and complete tasks by alternate means.

Education

A deeper understanding of the mechanism of pain and the idea that 'hurt' does not automatically equal 'harm' can benefit most patients. Exploration of the emotional and social impact of pain and an insight into pain behaviour increase patients' understanding of the condition and decrease fear and pain.

Relaxation and hypnosis

Relaxation can be used as prophylactic pain management in stress avoidance and can be combined with physical and psychological therapies. Hypnosis can be of help in pain that is resistant to other treatments (Simon and Lewis, 2000).

OTHER ALTERNATIVE THERAPIES

These include homeopathy, Reike, aromatherapy and reflexology. The success of each therapy is very individual and may owe much to patient motivation and the effect of a compassionate, interested practitioner. Some patients receive remarkable benefit from these other alternative therapies, but the scientific basis of these therapies has yet to be elucidated.

Osteopathy and chiropractic use treatments similar to manipulative physiotherapy.

CONCLUSION

Serious, treatable causes of pain must be excluded. Pain treatment relies on the integration of pharmacological, physical and psychological support. Pain management programmes seek to provide this and increase the patient's self-reliance so that they can retake control of their management and future. HM

Conflict of interest: none.

KEY POINTS

- Exclude serious or treatable causes of pain.
- Start with analgesics such as non-steroidal anti-inflammatory drugs, opiates and others on the World Health Organization's analgesic ladder.
- Antidepressants, ion channel blockers and other drugs act on the nerves and receptors that transmit pain signals from the periphery to the brain.
- Physical treatments include transcutaneous nerve stimulation, physiotherapy and nerve blocks.
- Do not forget the psychological component of pain and its treatment.
- Effective pain management includes components of all three types of therapy — pharmacological, physical and psychological.

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