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MANAGEMENT OF FACIAL PAIN AND HEADACHE: PSYCHIATRIC APPROACH*

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Continuous or intermittent pain in all parts of the head and neck is common, affecting at least 10 per cent of the general population at some time.[1] Such pains included well known problems, tension headache, migraine, neck ache and less well recognised disorders such as facial arthromyalgia (temporomandibular joint dysfunction syndrome), atypical facial neuralgia odontalgia and oral dysaesthesia.[2] These pains differ from classical trigeminal neuralgia in that their apparent origin is from blood vessels, muscles and joints rather than sensory nerves.[3] The diagnosis requires the exclusion of physical abnormality and the demonstration of emotional problems. These disorders present clinically to a wide range of specialties so that patients maybe seen and treated by dental specialists, neurologists, ear nose and throat surgeons or psychiatrists, with little collaboration between the specialties. As medical students receive no training in dentistry and dental students none in psychiatry, medical and dental practitioners are often ill-equipped to recognise many facets of the disorders in which facial pain and headache are prominent. Emotional disturbances underlying somatic symptoms are often overlooked,[4] either because the time available at the consultation may be insufficient to reveal any emotional problem, or because the doctor or dentist is reluctant to discuss emotional problems. Early recognition and treatment of an emotional disturbance may benefit the patient by preventing the development of symptoms and the health services by avoiding the expense of unnecessary investigations and referrals to other hospital specialists.[4, 5]

Different professional specialists concerned with these patients have advocated alternative aetiological theories ranging from mechanical malfunction to emotional distress, with consequent variations in treatment. A common understanding on the classification and management of headache and facial pain associated with emotional disturbance would help to resolve these differences.

PRESENTATION AND AETIOLOGY

Facial pain. Idiopathic facial pain is usually a continuous ache with intermittent excruciating episodes localised in the non-muscular non-joint area of the face. The pain may be uni- or bilateral and may persist for months or years. Patients may also complain of facial arthromyalgia, pain and clicking in the joints, atypical odontalgia, pain in the teeth in the absence of dental pathology and oral dysaesthesia, a burning discomfort in the tongue, gingivac or lips with no abnormal haematological or oral findings. In a review of twenty cases, Engel[6] found that 'search for a cause' had led to the enthusiastic investigation of allergy, endocrine and autonomic disturbances, vasomotor factors and the sphenopalatine

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ganglion and trigeminal nerve. He considered that the pain represented serious underlying psychological disturbance. The dental profession has tried and failed to treat a physical cause by improving occlusional relationships as shown by the American Dental Association's statement that 'dentists should rid themselves of the notion of a relationship between facial arthromyalgia pain and occlusal problems' and its recommendation of conservative, reversible forms of therapy.

Tension headache. This is a steady non-pulsatile tension or ache. Patients describe a sense of 'tightness' in the temples or at the occiput, like 'tight bands' or a 'vice'. Wherever they begin the pains may radiate to the forehead, the temples, the back of the head and the neck and be uni- or bilateral. It has been assumed for many years that the pain was due to muscular contraction but Philips and Hunter[7] found a considerable overlap in the amount of electromyographic (EMG) activity during headache and non-headache periods. They suggested that factors other than muscle contraction might be associated with the presence or absence of tension headache.

Associated symptoms. In these patients headache and facial pain are not mutually exclusive, as they may recur sequentially or simultaneously. Patients may also complain, but often only if asked, of other recurrent symptoms such as chronic neck and low back pain, migraine, pruritic skin disturbances, spastic colon, dysfunctional uterine bleeding, chronic fatigue or fibromyalgia.[2] The prevalence of these symptoms is much higher than in the general population. The decision to consult a doctor or a dentist is not simply a question of the presence of symptoms.[8] It may be due to their severity or to a complex psychological and social influences. An impression of changed bodily function occurs frequently in the general population but perhaps only one-third of symptoms make an individual seek medical help. Some symptoms are more likely to lead to consultations; more people with sore throats go to their doctor than those with headache.[9]

Patients with facial pain or headache seem to have experienced a higher incidence of inadequate or unstable parents and of stresses such as marital or financial difficulties, illness in the family and bereavements. Engel[6] introduced the concept of a 'pain vulnerable person' and it may be that the combination of early childhood experience and genetic susceptibility creates such vulnerability. A history of traumatic sexual experience, physical violence and major parental upheaval before age 17 is found more frequently in patients with somatic symptoms than in symptom-free individuals. Similarly, a high percentage of emotional disorders have been reported in first degree relatives of chronic pain patients.[10] This supports the concept of a pain prone person.[6]

However, not all patients are psychiatrically disturbed. Using rigorous criteria only 33 per cent of patients were found to be depressed but 80 per cent reported a stressful event within six months of the onset of pain.[2] Stressful life events may create pain directly or make it more likely that the subject seeks help for previous discomfort.

Psychiatric symptoms appear to develop in those made vulnerable by a deprived childhood, a neurotic personality and poor social adjustment. When present, psychiatric symptoms are often mild, of brief duration and are best considered to be part of a normal reaction to distress. The close association between chronic pain, adverse events and long-standing problems emphasise the need for a thorough clinical assessment of a patient's problems and for conserva-

TABLE 1

Side effects of selected antidepressants.

	Sedative	*Anticholinergic*
Nortriptyline	low	low
Desimipramine	low	low
Doxepin	high	medium
Dothiepin	medium	medium
Imipramine	medium	medium
Protriptyline	low	high
Trimipramine	high	medium
Amitriptyline	high	high

tive management. It should be emphasised to the patient that the pain is real, not imaginary, but arising in cramped muscles and blood vessels as a response to stress.

There appears to be both a central and peripheral focus for pain. The anterior cingulate cortex, thalamus and lenticular nucleus are identified areas within the brain which are activated by pain. Patients with atypical facial pain have abnormal activation in the anterior cingulate regions of their brains and decreased blood flow in their prefrontal cortex.[11] The pain is therefore a real phenomenon with objective cerebral change.

ANTIDEPRESSANT TREATMENT

Confusion, prejudice and ignorance surround the use of anti-depressants to treat chronic facial pain. The analgesic effect is sometimes attributed, without supporting evidence, to the relief of depressed mood, apparent or masked. Other theories such as the correction of a disturbed sleep pattern, or the relaxation of tensed muscles, remain possible but unproven. Disastrous side-effects are reported with the routine prescription of antidepressant medication (Table 1). An additional problem is the confusion between the non-addictive tricyclics and the highly addictive benzodiazepines.

It has been suggested that pharmacological similarities exist between tricyclic antidepressants and centrally acting analgesics. Stress-induced excessive or irrelevant muscular activity or vascular dysfunction are useful models of a peripheral end organ dysfunction. But the most popular explanation for the action of antidepressants on pain is their effect on synaptic levels of serotonin and noradrenaline. Sternbach[12] has proposed that, once established, chronic pain depletes the level of neurotransmitters, particularly serotonin, in the area of the dorsal raphe nucleus, an area considered crucial in pain suppressor pathways. This depletion may account for the apparent ineffectiveness of analgesics in chronic pain, and the association of chronic pain with depression and hence the ability of tricyclic antidepressants to improve both symptoms. Von Knorring *et al.*[13] suggested that patients with somatoform pain disorders benefit from treatment with antidepressants with a strong effect on serotonin rather than noradrenaline re-uptake. However, tricyclics with an intact tertiary amine group (e.g. amitriptyline, clomipramine) are said to show better analgesic activity than demethylated substances such as nortriptyline and desimipramine.[14] France[15] stated that no one drug has been shown to be superior in relieving pain; drug selection is based on matching the side-effect profile of each agent with each individual patient's needs.

Results of controlled studies do not seem to show any difference between the efficacy of the more serotonergic antidepressants and the more noradrenergic. The intimate interaction between serotonergic and noradrenergic mechanisms exists and it is possible that both serotonin and noradrenaline are involved in the analgesic activity of antidepressants, although there may be differences in compliance with serotonergic drugs as they appear to be more acceptable to patients.

Tricyclic antidepressants potentate the effects of narcotic analgesics such as morphine, lending support to the suggestion that they potentate naturally occurring endorphins in blood and/or cerebrospinal fluid.[16] In addition, subnormal endorphin function has been reported in patients with chronic pain.[17] Evidence from patients with post-operative pain and from animal models of chronic pain has confirmed that the analgesic activity of antidepressants is partly related to their modulating effect on endogenously released opioid peptides.[18]

Studies of the anti-nociceptive effect of antidepressants in animals have yielded conflicting results, both in the tail flick test and in the hot plate test. The tail flick test, which is one of the most commonly used nociceptive tests, has been shown to be sensitive to changes in tail skin temperature. A negative correlation is consistently found between tail flick latency and tail skin temperature. Tricyclic antidepressants may change blood flow and thereby skin temperature of the tail. There is also recent evidence supported by the analgesic effect of antidepressants in experimental pain in man.[19] A defect in tyramine metabolism, which is said to predict response to antidepressants, has been found in depressed patients and in facial pain patients who are not depressed, suggesting a common biological abnormality between the two disorders and an explanation as to how the drugs relieve pain.[2]

The action of tricyclic antidepressants on pain appears to be independent of any antidepressant effect, and analgesia may occur at lower doses than those used for depression.[20] Furthermore, tricyclics relieve the pain in a much shorter time than that needed for relief of depressive symptoms.[21] The interaction of drug therapy and psychotherapy is complex, for example the intensity of pain has been reported to increase after psychotherapy,[21] although there appears to be a positive interaction with cognitive therapy.[22]

TREATMENT REGIME

Counselling. In our clinical practice, tricyclic antidepressants are not used in isolation. A pragmatic approach to treatment is adopted in which the diagnosis is first established by a careful history; this identifies other psychosomatic pains and seeks out stressful life events. The patient is then reassured that no serious illness is present and is counselled in terms of lifestyle. For the majority of patients particularly those seen in primary care informed reassurance is sufficient.

Drug therapy. If pain persists, tricyclic antidepressants should be taken at night in slowly increasing doses, combined with regular review at three to six weekly intervals to provide reassurance and achieve compliance. As sedation is not required in most cases, a drug with low sedative and low anticholinergic side-effects, such as nortriptyline, is recommended. Nortriptyline may be increased gradually from 10 to 30 mg then from 50 to 100 mg. Increasingly serotonin specific re-uptake inhibitors are prescribed as they are not sedative and can be taken in the morning. They do not interact with alcohol and do not cause the patient to gain weight. Fluoxetine 20 mg daily is well tolerated by most patients.

For patients with insomnia, a sedative such as dothiepin is useful, starting with a single night-time dose of 25 mg and increasing in steps up to 225 mg when necessary. In elderly patients with constipation and glaucoma, and in males with prostatic hypertrophy, the least anticholinergic drug should be prescribed. Combinations of drugs such as nortriptyline and a phenothiazine are useful when the patient's pain is accompanied by extreme anxiety or bizarre symptoms.

Changing the agenda. It is essential that the patient changes the agenda from physical concerns to discussion of psychological problems. An explanation of the process of somatisation as 'the amplification of normal bodily sensation' is often useful. The finding of a cerebral abnormality can be used as an example of the brain paying excessive attention to a part of the body.[12] Continual insistence on a physical cause and repeated investigation often cause the pain to become intractable.

Long term care. Stoicism is required on the part of the clinician, as those patients for whom medication is most essential are more resistant to this management and complain of more side-effects than others. The side-effects are often bizarre and incapacitating. In one of our studies there were more side-effects in the placebo group than with the active drug! Pain may recur on withdrawal of medication and in some patients medication has to be maintained for a year to prevent relapse. Once the patient has confidence in the clinican and the medication, and has been free from pain for two to three months, the medication can usually be reduced. The principal difficulty is convincing these patients that they require support and medication as opposed to surgery. The willingness of the patients to accept operative intervention, which may contribute to rendering their condition intractable, emerges in refractory cases. The management of intractable facial pain remains a challenge, especially where the condition appears to have been reinforced by previous, irrelevant surgery. These patients are best admitted to hospital for a supervised drug regime.

Management problems. Failure to recognise the nature of the problem occurs when an inadequate history has been taken and important medical, personal, family or social factors have not been elicited or even concealed by the patient. There may be reluctance by the patient to take medication for what has previously been 'diagnosed' as a dental problem. Furthermore, reluctance to prescribe adequate medication by a timid clinician, unfamiliar with the natural history of the condition, compounds the difficulty in managing these patients. In general medical or dental practice, the practitioner must decide whether to treat the patient or to refer to a specialist. Ideally patients should be treated in primary care but the practitioner may feel that they have insufficient time or training. If a referral is made it should be done in such a way that the patient does not feel rejected.

PROGNOSIS

Many patients respond well to the above protocol. Seventy per cent in most studies respond to an antidepressant, but as a biochemical and psychological basis may explain the chronicity of the syndrome, there is need for continued care as in migraine or trigeminal neuralgia.

INNOVATIONS IN THERAPY

Cognitive therapy, with or without medication, is being explored for chronic facial pain. There has been significant success from a combination of medication and cognitive therapy with idiopathic pelvic pain, and a recent report of moderate success in facial pain. Clinical psychologists have an established role in the management of chronic disorders manifesting somatisation; their skills need to be applied within the field of maxillofacial surgery and oral medicine.

Specific inhibitors of serotonin re-uptake appear to have fewer side effects than tricyclics, and fluoxetine are now currently being investigated for efficacy with chronic pain. There is also a possibility that the new monoamine oxidase inhibitors, such as moclobemide, which are considered to be free of tyramine interactions may have value in refractory cases.

Multidisciplinary facial pain clinics especially in dental hospitals or departments of oral and maxillofacial surgery are now mandatory. Such clinics should have a staff of an oral physician or surgeon, a liaison psychiatrist and clinical psychologist. A network of such clinics would facilitate the management of difficult cases and clinical research.

Training programmes for medical and dental undergraduates and general practitioners should be available. As the identification of patients may be carried out at both primary and secondary levels, management training programmes would provide an efficient and cost effective means of enabling continuing care to be provided in general practice, as has been shown for depression.

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