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Facial pain and sinonasal surgery*

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SUMMARY

Objectives: *To examine the causes of facial pain that persists after endoscopic and other sinonasal surgery.*

Study Design & Setting: *A study of a cohort of 973 patients presenting in the outpatient clinic with symptoms of facial pain and/or rhinosinusitis. The study subgroup consisted of 75 patients with facial pain persisting after sinus surgery (endoscopic procedure n=48, other intranasal procedures n=27). The patients in the subgroup were studied with particular reference to their original presenting symptoms, endoscopic signs, and findings at CT as well as their symptoms when they presented to this unit and, importantly, these were analysed in the context of their response to treatment and follow-up after a mean of 2 years 7 months.*

Results: *Of the 75 patients who had persistent pain after surgery, 40 had no evidence of disease at endoscopy or CT at their initial presentation, whilst 35 had preoperative evidence of sinonasal disease. However, after surgery and re-evaluation, as well as neurological medical treatment, it was found that sinonasal disease was not the cause of these patients pain. The causes of their facial pain that persisted after sinonasal surgery were tension-type headache, atypical facial pain, migraine, paroxysmal hemicrania, cluster headache but the most common cause was 'midfacial segment pain', which has all the characteristics of tension-type headache but primarily affects the midface.*

Conclusions & Significance: *Some patients with facial pain are undergoing endoscopic sinus surgery in the mistaken belief that rhinosinusitis is the cause of their facial pain. All surgeons dealing with facial pain should be familiar with non-sinonasal diagnoses. A new category of facial pain, 'midfacial segment pain', is described and proposed.*

Key words: facial pain, sinusitis, tension-type headache, endoscopic sinus surgery

INTRODUCTION

Endoscopic sinus surgery (ESS) has enjoyed huge popularity over the past 10 years. Its effectiveness in the treatment of facial pain due to chronic rhinosinusitis has been described (Hoffman et al., 1989; Rice, 1989; Stammberger and Posawetz, 1990; Smith et al., 1993; Lund and Scadding, 1994; Terris et al., 1994; Harkness et al., 1997; Acquadro et al., 1997; Senior et al., 1998)

Facial pain is a common reason for referral to an otorhinolaryngology clinic. Patients' expectations are often coloured by a prior self-diagnosis of 'sinusitis'. In the medical literature, rhinological causes of facial pain include infective rhinosinusitis. Stammberger and Wolf postulated that variations in the anatomy of the nasal cavity result in mucus stasis, infection and ultimately facial pain (Stammberger and Wolf, 1988). They also stated that mucosal contact points might result in the release of the neurotransmitter peptide substance P, a recognised neurotransmitter in nociceptive fibres. Although this hypothesis was published 15 years ago there has been no in vitro or vivo work to substantiate it (Abu-Bakra and Jones, 2001a). Other authors have embraced these concepts to explain how pain might be induced by anatomical variants such as a concha bullosa (Morgenstein and Krieger, 1980; Blaugrund, 1989; Goldsmith et al., 1993; Clerico and Fieldman, 1994), or a pneumatised superior turbinate touching the septum (Clerico, 1996) whilst others have not found any evidence that contact points initiate pain (Abu-Bakra and Jones, 2001b).

Endoscopic sinus surgery has also been advocated for facial pain in the absence of endoscopic or CT evidence of sinus disease or anatomical variations. Boonchoo (1997) performed ESS on 16 patients with headache and negative sinus computed tomography (CT) scans, and reported total resolution of pain in 10 patients and partial resolution in the other 6. Cook et al. (1994) advocated ESS in patients with facial pain, which also occurred 'independently' of episodes of rhinosinusitis, with no

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CT evidence of sinus pathology. Twelve of the 18 patients who underwent surgery in their series had a significant reduction in their pain severity, yet it is very significant that the authors describe, "complete elimination of symptoms was not accomplished in any patient". They had no evidence of ostiomeatal obstruction. If the cause of their pain was due to an anatomical 'abnormality' or ostial obstruction then it might be anticipated that surgery would correct their symptoms of pain. This was not the case. Similarly Parsons et al. (1998) retrospectively described 34 patients with headaches who had contact points removed and found that whilst there was a 91% decrease in intensity and 84% decrease in frequency, 65% had persisting symptoms.

The evidence in support of these theories (contact points cause pain; opening ostia as a treatment strategy in-patients with facial pain who are CT and endoscopy negative) and of the surgical treatment of facial pain has not been substantiated by controlled studies. It is possible that the majority of the series reported in the literature describe coincidental anatomical variations in patients with facial pain. It may be that a response to surgery, which is more often partial than complete, results from the effect of the placebo effect or cognitive dissonance (Homer et al., 2000), or that surgery may have some effect in altering neuroplasticity within the brainstem sensory nuclear complex (Olesen, 1991; Bendtsen, 2000; Jensen and Olesen, 2000; Sessle, 2000).

A simple observation is that nowhere else in the body does mucosa-mucosa contact cause pain, and this undermines the hypothesis that mucosa-mucosa contact results in the release of substance P and produces pain.

Reports in the ORL-HNS literature are starting to emerge which show that even in patients with objective signs of sinonasal pathology, up to 38% have pain after sinus surgery (Becker and Cunning, 2000; Tarabichi, 2000). As well as this others have reported that patients whose primary complaint was headache or facial pain were less likely to have evidence of rhinosinusitis than those who had nasal symptoms (Rosbe and Jones, 1998). The ORL-HNS literature has been slow to assimilate the advances in the understanding of facial pain and headaches that are occurring in neurological circles (Olesen, 1991; Bendtsen, 2000; Jensen and Olesen, 2000; Sessle, 2000; Jones, 2001).

This study was initiated because of the senior author's (NSJ) dissatisfaction with his medium and long-term results of ESS for the management of facial pain, in particular as a primary symptom.

Table 1. The *primary* diagnoses in the cohort of 973 patients attending a rhinology clinic after a mean of 2 years and 2 months (note that many of those with neurological pain had coexisting allergic rhinitis - in total 697 had some evidence of nasal disease).

Diagnosis	Number	Diagnosis	Number
Allergic rhinitis	240	Barotrauma	5
Nasal polyposis	217	Paroxysmal hemicrania	3
Midfacial segment pain	107	Trigeminal neuralgia	5
Purulent rhinosinusitis	98	Churg-Strauss syndrome	21
diopathic rhinitis	67	Post-herpetic neuralgia	2
Tension-type headache	66	Wegener's granulomatosis	2
Migraine	51	Tumour of the orbit	1
Atypical facial pain	35	Skull base meningioma	1
Cluster headache	23	Thrombosed supratrochlear haemangioma	1
TMJ/Myofascial pain	22	Chronic fatigue syndrome	1
No diagnosis	14	Dental abscesses	2
Aspergillosis	8		

METHODS

This study analyses a prospectively collected database of 973 consecutive patients referred to a tertiary rhinology clinic, who fulfilled the criteria of having facial pain, headache, and/or symptoms of rhinosinusitis. The exclusion criteria were: post-nasal drip as a solitary symptom, nasal deformity, epistaxis or vestibulitis, rhinitis medicamentosa, benign or malignant tumours, valve collapse, altered olfaction as a solitary symptom, nasal granulomatous disorders without pain.

The patients' original preoperative records were obtained and their symptoms, endoscopic signs, and CT were studied as well as their presenting features to this unit and the diagnosis made here after treatment and follow-up. The mean follow-up in the subgroup was 2 years 7 months (range 22 months – 40 months, median 32 months) and in the whole cohort 2 years 2 months.

RESULTS

Of the 973 consecutive patients meeting the criteria, 564 (58%) had no pain and 409 (42%) experienced facial and/or head pain or pressure. The mean age of the cohort was 41.0 years (range 10-81 years) and 55% were female.

Of the 973 patients, 679 (70%) had evidence of sinonasal disease by rhinoscopy or endoscopy leaving 294 (30%) with no evidence of sinonasal disease. Of the 679 patients with evidence of nasal disease 308 (45%) patients also complained of pain or pressure. In the group of 308 patients with symptoms of rhinosinusitis *and* facial pain 170 had endoscopic signs of mucosal disease and 144 had CT signs consistent with infective rhinosinusitis. All 144 had evidence of purulent secretions at endoscopy and of these 38% had symptoms of pain or pressure. Of the 54 who had pain and purulent secretions 83% responded to medical or surgical treatment for sinusitis. The remaining 17% had pain from neurological causes.

Seventy-five patients had pain in spite of having surgery and on further analysis of these patients forty had no evidence of disease at their initial endoscopy or CT (although 19 of these had CT changes on presentation to this unit having had previous surgery; when their original CT was reviewed this showed

Table 2. Diagnoses in the 75 patients who had pain that persisted after sinonasal surgery after a mean follow-up of 2 years 7 months.

Diagnosis	After endoscopic sinus surgery (n=48)	After other sinonasal surgery (n=27)
Midfacial segment pain	12	10
Tension-type headache	9	8 (2 also migraine)
Atypical facial pain	9	5
Atypical facial pain/tension type headache overlap	1	0
Post surgical pain	6	0
Migraine with facial component	2	1
Paroxysmal hemicrania	2	1
Paroxysmal hemicrania/migraine overlap	2	0
TMJ dysfunction	0	1
Cluster headache	3	0
Churg-Strauss syndrome	1	0
Thrombosed haemangioma of supraorbital nerve	1	0
Post traumatic pain	0	1

Table 3. The optimum treatment to control pain in the 75 patients who had pain, which persisted, after sinonasal surgery after a mean follow-up of 2 years 7 months.

Diagnosis	Number	Successful treatment
Midfacial segment pain	22	18 Amitriptyline 1 Carbamazepine 3 nil
Tension-type headache	17	12 Amitriptyline 1 Carbamazepine (2 with coexisting migraine had that helped by triptans, one by pizotifen) 4 nil
Atypical facial pain	14	8 Amitriptyline 2 Carbamazepine 1 triptans when exacerbation 3 nil
Atypical facial pain/tension type headache overlap	1	1 nil
Post surgical pain	6	2 Amitriptyline 1 Carbamazepine 1 Phenytoin 2 nil
Migraine with facial component	3	3 Amitriptyline
Paroxysmal hemicrania	3	2 Indomethacin 1 Sodium valproate
Paroxysmal hemicrania/migraine overlap	2	2 Amitriptyline
TMJ dysfunction	1	1 Bite raising appliance
Cluster headache	3	2 Pizotifen 1 nil
Churg-Strauss syndrome	1	1 Prednisolone
Thrombosed haemangioma supratrochlea nerve	1	1 Excision
Post traumatic pain	1	1 Carbamazepine

them to have initially been clear). Their mean age was 43 years 1 month (ranged from 19-77 years) and 59% were women. Thirty-five patients had coexisting evidence of disease at endoscopy or CT preoperatively, but their pain continued post-operatively and when there was no evidence of remaining sinus disease, they then went on to respond to neurological medical treatment. These 75 patients form the study group of this report.

Symptoms

Seventy-five patients had pain that persisted after surgery. This subgroup consisted of 48 patients who had facial pain in spite of having previously had endoscopic sinus surgery (elsewhere n=30, by the senior author n=18: 11 middle meatal antrostomies, 25 anterior ethmoidectomies, 12 frontosphenoethmoidectomies). A further 27 patients, had pain that persisted after other forms of sinonasal surgery done in the past. In the latter group of 27 patients 15 had had septal surgery (2 for septal contact points), 5 inferior antrostomies, 2 had septal surgery and inferior antrostomies, 1 headlight middle meatal antrostomies, 1 Caldwell Luc procedure, 1 frontal sinus obliteration and 2 middle turbinectomies for contact points. There were 44 females and 31 males, with average age of 43.1 (range 21-77) years. Of the 75 patients who had pain that persisted after nasal surgery, facial pain was the original primary symptom in 61 patients, whilst 4 patients' main complaint was nasal obstruction, with pain being a secondary symptom. One patient had a pyocoele, 3 patient's original records were unobtainable and 6 patients had developed their pain after sinus surgery. Two of the patients who had originally had pain as a preoperative symptom said that it had become worse after ESS.

Endoscopic findings

At their original presentation of the 48 who went on to have ESS, 24 had had normal endoscopy, 10 polyposis, 2 purulent disease, 3 ostiomeatal oedema, 6 had a septal deviation with normal mucosa (2 septal contact points), and 3 patients' original records were not obtainable. In the 27 who went on to have other sinonasal surgery 11 had normal endoscopy, 1 minor polyposis, 15 a septal deviation (2 contact points) and other coexisting findings included 3 with inferior turbinate hypertrophy and 1 had profuse clear mucus and 1 had a distorted lateral wall following a Le Fort II fracture.

Computerised tomography

In the group of 75 who had pain in spite of surgery, 48 had ESS, and of these 21 had a normal CT, 12 had mucosal thickening >4mm, 2 with coexisting maxillary antral cysts, 1 had what proved to be an incidental ethmoidal mucocoele, 1 a pyocoele, 8 had minimal mucosal thickening, and 3 scans could not be obtained. Eighteen of the 27 who had other forms of sinonasal surgery had their original CT available to review, 3 other patients had already had other sinus surgery before their first CT scan was done. Of these 18 who had had a CT before surgery, 1 had >4mm mucosal thickness, 5 had minimal mucosal thickening and 1 had a maxillary retention cyst and 11 had a normal CT scan.

Endoscopic and CT findings

Of the group of 75 who had pain in spite of sinus surgery, 48 had had ESS, and of these 11 had no abnormal findings on either endoscopy or CT, a further 9 others had normal endoscopy and minimal mucosal thickening on CT, and a further 5 only had a septal deviation. In the group of 27 who had other types of sinonasal surgery, 18 of those who had had a CT. When these were reviewed 9 had no abnormal findings on endoscopy or CT and 6 had normal endoscopy and minimal mucosal thickening on CT. A further patient had a normal endoscopy and MRI. All together there were 41 patients of the 75 patients who initially had no good evidence of disease at endoscopy or CT, whilst 35 had some preoperative evidence of sinonasal changes.

Long-term follow-up

The diagnoses on which this study is based were made on the basis of their history, examination, CT, but most importantly their response to treatment after a mean follow-up of 2 years and 7 months. The causes of persisting pain in the 75 who had ESS or sinonasal surgery were primarily neurological (see table 2). The terms used to classify patients' facial pain was largely in keeping with that of that published by the Headache Classification Committee of the International Headache Society (1988). However, like other workers (Graff-Radford 2000), we have concluded that many patients cannot be fitted neatly into these categories. We believe that there many of these patients have a symptom complex that is distinctive enough to be given a name, 'midfacial segment pain' (Jones, 2001; West and Jones, 2001).

DISCUSSION

Most patients are aware that their sinuses lie behind the forehead, cheeks and either side of their nose and, understandably, label themselves as having rhinosinusitis when they have pain in this area. There is an increasing awareness amongst Otorhinolaryngologists that neurological causes are responsible for a large proportion of patients with headache or facial pain (Acquardo et al., 1997; Salman, 1999; Tarabichi, 2000; Jones, 2001; West and Jones, 2001). Rhinogenic facial pain is a common diagnosis. It is usually attributed to chronic infective rhinosinusitis although other theories include mucosal contact points as an initiating factor, vacuum headaches or septal deviation. ESS was originally advocated for the treatment of chronic or recurrent acute rhinosinusitis that is unresponsive to medical management (Kennedy, 1985; Rice, 1989; Stammberger and Posawetz, 1990; Ruoff, 1997). The increasing popularity of ESS has led to an expansion of its potential indications, for example to include 'headaches, pressure feelings, postnasal discharge, epiphora, retention cysts, tubal dysfunc-

tions, adjuvant surgery to allergy treatment', and other symptoms (Stammberger and Posawetz, 1990) ESS runs the risk of being seen as a panacea for all symptoms potentially relating to the nose and sinuses.

This report cautions against advocating ESS for facial pain as a primary symptom and in particular when there are no endoscopic or CT abnormalities to support the diagnosis. Even when there are mucosal changes on CT the quality, distribution and periodicity of the pain and the alternative diagnoses should be considered before concluding that its cause is not neurological. Several workers have demonstrated the high proportion of false positive CT scans in an asymptomatic population, hence its poor sensitivity in diagnosing rhinosinusitis (Havas et al., 1988; Lloyd, 1990; Bolger et al., 1991; Bhattachayya et al., 1997; Jones et al., 1997). The role of endoscopic sinus surgery for patients with facial pain but no additional evidence of chronic rhinosinusitis is controversial. Some authors have advocated surgery under these circumstances (Cook et al., 1994; Boonchoo, 1997) whilst other authors have reported poor results in this patient group (West and Jones, 2001). We believe that patients with facial pain who have no objective evidence of sinus disease (endoscopy negative, CT negative), and whose pain fails to respond to medical antibiotic/steroid therapy aimed at treating sinonasal disease, are very unlikely to be helped by surgery particularly in the medium and long term. A trial of neurological medical treatment should be considered before embarking on surgery.

Arriving at the correct diagnosis for the cause of facial pain can be a difficult process. There are numerous reasons why a patient suffering from headache/facial pain will present to an otolaryngologist with a presumptive diagnosis of rhinosinusitis. A sinonasal CT performed to 'rule out' an unrecognised rhinologic cause for a headache/facial pain can be expected to be abnormal in up to 30% of a normal population (Havas et al., 1988; Lloyd, 1990; Bolger et al., 1991; Bhattacharyya et al., 1997; Jones et al., 1997). Some of the symptoms of chronic rhinosinusitis are present in a large proportion of the population; e.g. up to 19% of the population have symptoms of nasal obstruction or rhinorrhoea on a regular basis. It is likely that an otolaryngologist will be an early point of referral for patients with non-rhinologic headache/facial pain, as well as patients with true chronic rhinosinusitis. The otolaryngologist is thus ideally placed to manage patients with headache/facial pain, and it is important for all surgeons to be familiar with the clinical features of common non-rhinologic headaches/facial pain.

The commonest diagnosis made in our series of patients was 'midfacial segment pain'. Midfacial segment pain is a form of tension-type headache, which has all the same qualities of tension-type headache. The only difference being that it affects the face and may involve the nasion, under the bridge of the nose, either side of the nose, the peri-orbital region, retro-orbitally or across the cheeks. Often the forehead is also affected. It is described as a dull ache, a feeling of pressure or as tightness. It can be chronic or episodic and the *skin and soft tissues* over the forehead or cheek may be sensitive to touch. These patients often take an excessive number of analgesics yet say that they confer little benefit. Ibuprofen can occasionally help to some extent in a proportion of these patients. The majority of patients with this condition respond to low dose amitriptyline, and require up to 6 weeks of 10 mg at night and occasionally 20 mg before it works. It should then be continued for 6 months before trying to stop it, but about 1 in 5 need to restart it as the pain returns. If this fails, then relief may be obtained from gabapentin, carbamazepine and occasionally sodium valproate for the same trial period and extended for 6 months before reducing it.

Next in frequency was tension-type headache. This has all the same characteristics of midfacial segment pain but it affects the forehead (Schoenen and Wang, 1997). There can be an occipital or temporal component and it can be chronic or episodic. Hyperaesthesia of the soft tissue of the forehead often occurs, giving the patient the impression they have rhinosinusitis, as they know their sinuses lie under the forehead. It most often responds to amitriptyline, but gabapentin, carbamazepine, sodium valproate or a change in lifestyle may bring successful relief of symptoms.

This term avoids the use of the term tension used in tension-type headache. It has the same characteristics as tension type headache but a different lower distribution of symmetrical facial pain. The pain usually affects the nasion, around or behind the eyes or the cheeks, and not uncommonly involves the forehead. It is described as a pressing or aching pain, similar to the feeling of constriction, pressure or tightening often described in patients with tension-type headaches. It can affect each area in isolation or in combination, but it is usually symmetrical unless there has been trauma or surgery to one side. The pain is usually persistent but it can be intermittent and is usually present on waking. It does not worsen with routine physical activity, and rarely interferes with the patient getting to sleep. To make matters more complex, the stimulus of a genuine acute sinus infection may exacerbate the symptoms, with a return to the background faceache on resolution of infection. Indeed, an episode of acute rhinosinusitis very occasionally appears to have been the initial trigger for the onset of symptoms in the first instance. It is hardly surprising that patients (and doctors) will interpret all their symptoms as being related to their sinuses. Patients often describe tenderness on lightly touching the skin of the forehead or cheeks, and there appears to be hyperesthesia of the skin and soft tissues in these areas. This is similar to the tender areas over the forehead and scalp seen with tension-type headache. It is important to note that the tenderness is felt on light touching of the skin and soft tissue, and there is no further pain on deep palpation of the underlying bone. Sufferers are often taking a

considerable number of over-the-counter analgesics, despite saying they help little if at all. In our experience the only simple analgesic of even moderate benefit is ibuprofen. The current first-line prophylactic treatment of chronic tension-type headache or midfacial segment pain is low-dose amitryptiline, given at night. Other selective serotonin reuptake inhibitors are not effective, as in tension-type headache (Bendtsen et al., 1996). It is relevant that 10mgs is insufficient to produce any analgesic effect on its own. Amitriptyline should be given for six weeks before judging its effect, and should be continued for six months if it has helped. The starting dose is 10 mg, and can be increased to 20 mg (and subsequently 50 mg) after the six weeks if pain is not only partially or not controlled. Patients need to be warned of the sedative effects even at this low dose, but they can be reassured that tolerance usually develops in the first few days. It is our practice to inform patients that amitriptyline is also used in higher doses for other conditions such as nocturnal enuresis and depression, but its effectiveness in midfacial segment pain is probably unrelated to its antidepressive properties, which would take effect much more quickly and normally require higher doses. It is often reassuring for patients to know the dose used for depression is some 7 or more times the dose used in tension-type headache. If amitriptyline fails gabapentin may be beneficial, especially when there has been a history of trauma or surgery. Following that carbamazepine or sodium valproate can help. In a proportion of patients there are migrainous features, and a triptan may help acute exacerbations. This is analogous to the overlap between tension-type headache and migraine described in the neurological literature (Raskin, 1988; Olesen, 1991; Marcus, 1992; Rasmussen et al., 1992; Silberstein, 1994; Bendtsen et al., 1996a; Bendtsen et al., 1996b; Leston et al., 1996; Schoenen and Wang, 1997). It may be that the underlying pathology is similar. The aetiology of this type of pain is uncertain but Olesen's (Olesen, 1991; Jensen and Olesen, 2000) theory that integrates the effects of myofascial afferents, the activation of peripheral nocioceptors and their convergence on the caudal nucleus of trigeminal, along with qualitative changes in the central nervous system, provides one of the best models. There is also a suggestion that there is a downregulation of central inhibition from supraspinal impulses due to psychological stress and emotional disturbances. Another factor in those who have had a peripheral injury or inflammation is that these may induce neuroplastic changes in the trigeminal brainstem sensory nuclear complex and produce central sensitisation (Sessle, 2000). All of these possibilities allow for the superadded potentiation by nociceptors on top of the peripheral/central sensitisation, which may be happening in this condition. Olesen's vascular-supraspinal-myogenic model for pain in migraine and tension-type headache is perhaps the most accepted (Olesen, 1991), and proposes that the pain is determined by the sum of nociception from cephalic arteries and pericranial myofascial tissues converging upon the same neurons, where it is integrated with supraspinal effects. Vascular input predominates in migraine, whereas myofascial nociception prevails in tension-type pain. Other mechanisms have been proposed which include sensitisation of peripheral myofascial receptors, sensitisation of second order neurons at the spinal or trigeminal level, the sensitisation of supraspinal neurons or decreased antinocioceptive activity from supraspinal structures (Bendtsen, 1996). The trigeminal caudal nucleus is the major relay nucleus for head and neck pain, and it appears supraspinal excitatory input contributes to intense neuronal activation resulting in a generalized increase in sensitivity of the nociceptive pathways, both centrally and peripherally. Midfacial segment pain may be a state of trigeminal neuronal hypersensitivity and pain facilitation. Olesen's model is tempting as it might explain much of the clinical picture of midfacial segment pain (Olesen, 1991). For example, the skin and soft-tissue hyperaesthesia that accompanies the pain may be due to the above hypersensitivity of the pain pathways. It is of interest that if surgery is mistakenly performed as a treatment for midfacial segment pain, the pain may sometimes abate temporarily, only to return after several weeks or months. It is as though the surgical stimulus alters the 'balance' of neuronal activity in the trigeminal caudal nucleus for a short time. It is possible that the placebo effect or cognitive dissonance may be responsible for the temporary improvement of her symptoms. These effects cannot explain the benefit of amitriptyline as the placebo effect normally subsides within months (Homer and Jones, 2000). We believe rhinologic surgery should be discouraged in patients with midfacial segment pain, as the pain only helps a third temporarily, in a third it makes no difference, and in third the pain is made worse.

Whilst we have described the clinical features of different clinical categories of pain we recognise that within these there are those whose pain is initiated by trauma or surgery which is likely to be neuropathic in origin, whether by a peripheral and/or central mechanism, and these patients often have a deep gnawing, burning unpleasant quality to their pain (Khan et al., 2002). There are others who have vascular features with exacerbations associated with facial flushing, the patient may feel that their cheek is puffy, they may have separate migraine but occasionally an exacerbation of their facial pain may extend or precipitate an attack of migraine. There are also those who have features of myofascial pain who have hyperaesthesia of their skin or muscles who have a poorly delineated aching sensation. These types of pain often overlap and are not distinctive enough to be diagnostic, but their study may lead us to a better understanding of the mechanisms of facial pain.

CONCLUSION

Determining whether facial pain is due to rhinosinusitis or to other causes should be the otolaryngologist's initial goal. We have presented a subgroup of 75 patients who initially complained of facial pain, of whom 48 underwent endoscopic sinus surgery and 27 other sinonasal surgery, but these patients' pain

persisted after surgery. Of particular relevance is that 41 patients had no preoperative endoscopic signs or CT changes suggestive of rhinosinusitis. Patients with facial pain without any other symptoms, no evidence of purulent rhinosinusitis at endoscopy, and a negative CT should not be considered for endoscopic sinus surgery. The diagnosis of rhinosinusitis (and therefore the indication for ESS) should never be made if the only symptom is headache/facial pain alone. There is a pressing need for training programmes to place a greater emphasis on the indications for ESS, rather than on the technical aspects of an ideal dissection. The ability of an otolaryngologist to make a correct diagnosis is of crucial importance.

The commonest cause of non-rhinologic facial pain is midfacial segment pain, a pain similar in all but location to tension-type headache. This should be considered in the differential diagnosis of all patients presenting with facial pain, especially if no objective evidence of rhinosinusitis can be found. The treatment of midfacial segment pain usually begins with amitriptyline for a minimum of 6 weeks; second-line drugs include gabapentin and carbamazepine.

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