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Prevalence of nasal mucosal contact points in patients with facial pain compared with patients without facial pain

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Abstract

A cohort of 973 consecutive attendants at a rhinology clinic was studied prospectively and divided into patients without facial pain (n = 566, 58 per cent) and patients with facial pain (n = 407, 42 per cent). The prevalence of nasal mucosal contact points was the same in both groups, being four per cent in patients with nasal contact points without facial pain and four per cent in patients with facial pain. A contact point is defined as when contact remains after topical decongestion. Of the 18 patients with facial pain, nine had a spur contacting the lateral nasal wall and nine had a middle turbinate contacting the septum. These 18 patients were followed up for a mean of two years and two months. In the light of their treatment and response the following diagnoses were made: five had tension-type headache, six had midfacial segment pain, one had migraine, two had cluster headache and four had purulent nasal disease. Of the four with unilateral symptoms, two had a contact point on the contralateral side. Eleven of these 18 patients responded to medical treatment for tension-type headache or midfacial segment pain, migraine and cluster headache, three patients were better after surgery for coexistng purulent nasal disease and one patient had a spur removed surgically and remained better at 2 years follow-up, whereas three patients were no better after the same procedure. The results demonstrate that the prevalence of nasal contact points in patients with facial pain is the same as in those within pain. Surgery undertaken to remove mucosal contact points for facial pain is usually unnecessary as the aetiology of this facial pain appears to be a more central processes.

Key words: Nasal mucosa; Facial Pain; Substance P

Introduction

The relationship, if any, between facial pain and nasal mucosal pressure points is uncertain.[1] A causal relationship between nasal contact points and facial pain is repeatedly quoted in the ENT literature.[2–6] A mucosal contact or pressure point is when, after decongestion, two structures in the nasal cavity are pressing at each other with a consequent blanching of the nasal mucosa.[7] The ENT literature describes the middle turbinate contacting the nasal septum or the lateral nasal wall,[7–9] the inferior turbinate contacting the septum,[10] the ethmoid bulla contacting the middle turbinate,[11] a nasal spur contacting the lateral nasal wall,[12] or the superior turbinate as a cause for referred facial pain.[13]

Many ENT workers have described small series and case reports of patients with both nasal mucosal contact and facial pain.[4,6,9–13] The question is whether these represent a coincidental finding or not. Over the years many pseudonyms have been used to describe the various forms of facial pain and amongst these were middle turbinate headache,[7] nasal spur headache,[13] four-finger headache[9] and nasal contact headache.[4]

This study investigates the relationship between nasal mucosal contact points and facial pain. Does a causal relationship exist? Answers to this question will lead to a better understanding of the relationship between the nose and headache or facial pain.

Materials and methods

A total of 973 consecutive rhinology clinic attendants were recruited in this observational study according to the criteria shown in Table I. Patients were excluded according to the criteria shown in Table II. Data were collected prospectively. A full, structured history was obtained from each patient. All patients had outpatient rigid nasoendoscopy as part of their examination. The cohort of 973 patients was retro-

TABLE I
INCLUSION CRITERIA

1.	Facial pain
2.	Headache
3.	Symptoms of rhinosinusitis

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TABLE II
EXCLUSION CRITERIA

1. Catarrh or postnasal drip as only symptom
2. Nasal deformity without rhinosinusitis
3. Septal deviation without rhinosinusitis
4. Nosebleeds
5. Rhinitis medicamentosa
6. Benign or malignant tumours
7. Valve collapse
8. Olfactory dysfunction without rhinosinusitis
9. Granulomatous disorders
10. Vestibulitis

spectively divided into patients with facial pain and patients without facial pain. The prevalence of mucosal blanching contact points which remained after topical decongestion with either cocaine six per cent or cophenylcaine, confirmed on rigid nasoendoscopy, was noted. The prevalence of these contact points was compared in patients with and without facial pain. A diagnosis was made on the basis of treatment and follow-up after a mean of two years and two months.

Results

The cohort of 973 consecutive attendants in our rhinology clinic was divided into patients without facial pain (n = 566, 58 per cent) and patients with facial pain (n = 407, 42 per cent) (Figure 1).

The prevalence of nasal mucosal contact points was the same in both groups, 22 patients with nasal contact points without facial pain (four per cent) and 18 patients with facial pain (four per cent).

Nine of the 18 patients with both facial pain and contact points had a spur contacting the lateral nasal wall and the other nine had a middle turbinate contacting the septum (Table III). Of the four patients with unilateral symptoms, two had contact points on the contralateral side. All these 18 patients were offered treatment and were followed up. Eleven responded to medical treatment for tension-type headache, midfacial segment pain, migraine or cluster headache. Three had coexisting purulent disease and were better after surgery for sinusitis.

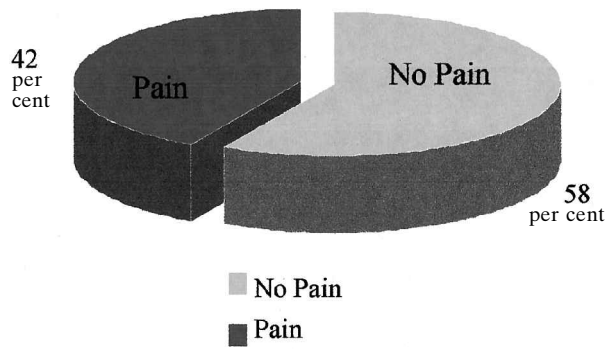

FIG. 1
Symmptoms of pain (n = 793)

There was no clear response in any of these patients to the application of cophenylcaine or cocaine as opposed to sterile water.

Some patients found some relief with both topical analgesic and placebo, and some with neither. None had relief with a topical analgesic but none with placebo. Four patients underwent surgery for mucosal contact points and had their septal spur or middle turbinate removed surgically. All were better for three to 12 months after surgery. In three of these patients, their pain returned and they were no better at follow-up, although one patient remained asymptomatic at two years.

Discussion

This study investigated the hypothesis that nasal mucosal contact points cause headache or facial pain. For this hypothesis to stand scrutiny, the prevalence of nasal mucosal contact points should be higher in the population of patients with facial pain than in that those without pain. Our results found that the prevalence of contact points was the same in both populations. Therefore, the presence of facial pain and nasal mucosal contact point is likely to represent a coincidental rather than a causal relationship.

The majority of patients with both facial pain and mucosal contact points responded to neurologically based medical treatment. Furthermore, whilst all the patients who had surgery for their contact points

TABLE III
PATIENTS WITH BOTH CONTACT POINTS AND FACIAL PAIN (N = 18)

Diagnosis (number)	Contact point (CP)	Surgery	Outcome
Tension-type headache (5)	Septal spur to later wall (3) Middle turbinate to septum (2)	No surgery for CP	All better with medical treatment, e.g. low dose amitriptyline
Midfacial segment pain (6) (a form of tension-type headache involving the midface and 60% have involvement of the forehead)	Septal spur to later wall (2) Middle turbinate to septum (4)	One had a spur removed, one MT removed	One better with surgery for spur, rest better with medical treatment, e.g. low dose amitriptyline
Migraine (2)	Middle turbinate to septum (2), one on the contralateral side	No surgery for CP	All better with medical treatment, e.g. triptans or pizotifen
Cluster headache (2)	Septal spur to lateral wall (2), one on the contralateral side	Both had spurs removed	No better with surgery for CP, responded to medical treatment, e.g. triptans or pizotifen
Coexisting purulent nasal disease (3)	Septal spur to lateral wall (2) Middle turbinate to septum (1)	No surgery for CP	All better after surgery for sinusitis

CP = contact point

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were better initially, only one remained symptom-free at 24 months. It is possible that these patients responded temporarily because of a placebo effect, cognitive dissonance or an alteration in the sensory pathway or connection with the caudal nucleus of the trigeminal. This emphasizes that the contact point itself, and that surgery to correct it, is unlikely to be helpful in the medium- and long-term and that their facial pain is caused by processes other than the incidental finding of a mucosal contact point.

Much of the proposed evidence for this relationship is based on the work by McAuliffe in 1942. McAuliffe[3] reported experimental work with five healthy subjects and 10 patients with neurological lesions (cranial nerves sectioned for intractable pain relief). He demonstrated that stimulation of various areas of the nasal cavity caused referred facial pain that was felt in specific cutaneous distributions of the trigeminal nerve. However, McAuliffe's findings have not been reproduced since. The concept of nasal contact facial pain has been propagated over the years. In 1948, Wolff, McAuliffe's co-author, described contact headaches as being due to pressure points within the nose.[6] He said that referred facial pain may occur due to contact between a turbinate or other regions of the nasal cavity. He postulated that shrinking or anaesthetizing the affected turbinate could relieve the pain. In 1954, Williams described nasal-contact headache.[4] He felt it was due to contact between the turbinate and the septum and suggested resection of the turbinate. In 1978, Cottle also attributed a cause of unilateral facial pain to middle turbinate compression (Cottle MH, Scientific exhibit at the American Academy of Otolaryngology Meeting, Las Vegas, Nevada, September 1978).

In 1986, Greenfield theorized that trigeminal nerve afferent fibres from nasal mucosa enter into the cortex together with the afferent fibres of the cutaneous divisions of the nerve, thus suggesting an explanation for the sensation of facial pain due to nasal stimulation (Greenfield H, Scientific exhibit at the American Academy of Otolaryngology Meeting, San Antonio, Texas, 1986). He further theorized that regions of contact points in the nasal cavity may be associated with local reflex engorgement and the release of vasoactive amines which may be involved in either causing pain or lowering the threshold of pain. In 1988, Stammberger and Wolf[5] theorized that stimulation of nasal mucosal receptors causes substance P liberation via both a central impulse and a peripheral local impulse. Local substance P causes vasodilation and hypersecretion, while the liberation of substance P in the central nervous system causes referred pain via unmyelinated C fibres to the cortex. Although substance P has been shown to be localized in sensory C-fibres in the human nasal mucosa,[14,15] there is no evidence that it is produced locally by mucosal contact points. Nowhere else in the body does a mucosa–mucosa contact point produce pain.

Many workers have described small series and case reports of patient with both nasal mucosal contact and facial pain.[4,6,9–13,16,17] These, in the light of our study, probably represent coincidental findings of nasal mucosal contact points in patients with facial pain. There is no evidence of a causal relationship.

Morgenstein in 1980 described 19 patients with headache which he said was due to the middle turbinate touching the septum.[9] They underwent middle turbinate resection and 15 were better after surgery, two had partial relief and two were no better. However, their follow-up was limited. In 1984, Gerbe sited 20 patients with recurrent unilateral headaches, nasal spurs and no sinus disease who had surgery on their contact points.[20] Nineteen were followed up post-operatively. Thirteen had complete relief, while the remaining six had only partial relief after a medium follow-up period of 18 months. Hoover reported in 1987 on a mixed group of 80 patients with headache due to various rhinological causes[16] and 39 attributed to allergy-induced headaches, with 51 per cent having an occipital component which is characteristic of tension-type headache. The headache of 33 patients was attributed to an impacted septum or turbinate and 23 of these had surgery. All patients had relief post surgery but again follow-up was limited. In 1989, Blaugrund reported a case of a patient with an impacted middle turbinate who presented with facial pain that was relieved by excision of the middle turbinate and antrostomy.[17] Brown presented a retrospective study of 74 patients with headache.[18] These were selected from 570 patients who had 1170 operations. He picked two patients with a septal/lateral wall contact who were better six months after surgery.

Goldsmith, in 1993, presented eight patients described as having nasal-contact facial pain.[7] Two were better after medical treatment for rhinosinusitis. Six had surgery for their contact points. Five were asymptomatic post-operatively, while one patient continued to have occasional headaches at three months. The average follow-up was 10.8 months, ranging from two to 24 months. In 1994, Chow described 18 patients with a 'rhinologic headache'[19] attributed to a variety of causes including 12 patients with septal spurs, three had retention cysts, three with mucosal contact points and one patient had a dehiscent infraorbital nerve. Palpation of the contact points in these patients induced the same type of headache and the application of local anaesthesia relieved it. Seventeen of the 18 patients underwent surgery. Eight patients were cured of their headache or facial pain, six had a significant improvement of their symptoms while three patients were not any better after surgery although there was no mention of the length of post-operative follow-up. Clerio presented a series of three patients in 1996 with common migraine whose superior turbinate touched the septum.[13] Cocaine lessened or relieved their headache and resection helped. Follow-up ranged from six to 14 months.

TABLE IV
THEORIES ON THE AETIOLOGY OF TENSION-TYPE HEADACHE

- Convergence of somatovisceral afferents on the caudal nucleus V
- Nocioceptive activation and sensitization of neurons due to input from non-nociceptive stimuli
- Ephaptic transmission from sensorineural afferents to nocioceptive fibres
- A reduction in the supraspinal inhibitory control

Reviewing the ENT literature shows that whilst surgery advocated for removal of contact points helps some patients in the short term, none of these series demonstrated complete relief for all their patients which questions the aetiology of their pain. Furthermore, most of these studies failed to follow-up their patients post-operatively for an adequate length of time. The mean post-treatment follow-up in this study is 26 months.

None of these series and case reports considered 'neurological' medical treatments available for the commonest causes of facial pain and headache, like tension-type headache, midfacial segment pain (an extension of tension-type headache that affects the midface with 60 per cent also involving the forehead), migraine and cluster headache, in the management of their patients. These should be considered in the differential diagnosis of facial pain. It is therefore essential to obtain a structured history from the patient with facial pain and not ending up treating the investigation rather than the individual and inflicting unnecessary surgery on patients. This is echoed by some authors who considered different forms of neuropathic pain early in chronic facial pain with minimal sinus disease, especially in unusual pain presentations that may include heightened sensitivity, unusual pain patterns, emotional exacerbation, and persistence of pain with resolved or subclinical sinus disease.[20] Theories by Olesen[21] on the aetiology of tension-type headache may apply to the face (Table IV). Such theories explain the complexity of the clinical picture and overlap.

Conclusions

This study adds to the body of opinion which questions the role of mucosal contact points in the aetiology of facial pain. This study shows that the prevalence of nasal contact points in patients with facial pain is the same as in those without pain. Surgery undertaken to remove mucosal contact points for facial pain is usually unnecessary as the aetiology of this facial pain is likely to be related to other more central pathological processes.

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