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Migraine: A look down the nose



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Summary *Background:* Studies have suggested that contact between opposing mucosal surfaces in the nasal wall and cavity can be a target of the surgical treatment of migraines. Unfortunately, not enough is known about the role of nasal pathology in the pathogenesis of this condition. The co-existence of further rhinological disorders can be an impediment to defining the cause and effect of anatomical variants. The authors compared the MRI scans of migraine- and non-migraine patients (MPs and NMPs, respectively) to determine the prevalence of such mucosal contact points in order to extrapolate whether there is a significant association with migraines.

Methods: Coronal and axial MRI brain scans of 522 patients (412 migraineurs and 110 non-migraineurs) were analysed for the prevalence of anatomical variations of the nasal cavity, e.g. concha bullosa, septal deviations, mucosal swelling and contact points.

Results: The results showed no significant difference between MPs and NMPs patients for any of the parameters examined. Moreover, 87% MPs and 79% NMPs had at least one contact point. The most frequent contact point was between the middle turbinate and the septum, observed in 54% of MPs and 45% of NMPs.

Conclusions: Contact points with the nasal mucosa are highly prevalent in both MPs and NMPs. Although a contact point does not cause a migraine in the absence of the disease, the concomitant presence of migraine and contact points can trigger an attack, and therefore, it is necessary to differentiate or exclude a rhinological disorder in these patients.

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Introduction

Migraine is defined as a pain disorder with headache as its primary symptom. Although the pain is often the most prominent and easily recognizable sign, this does not account for the array of premonitory and post-dromal symptoms occurring before and after the pain. The prefixes 'pre' and 'post' are used to describe a host of diverse phenomena related to the temporal occurrence of the headache. However, these symptoms can precede, accompany or outlast the pain itself.¹

Osmophobia is a typical premonitory symptom of migraines. Patients often report intolerance to odours, i.e. that certain odours can trigger attacks and that they experience osmophobia during attacks and olfactory hypersensitivity between attacks.^{2,53} In addition, approximately 25% migraine patients (MPs) report substantial nasal congestion during attacks, which commonly occurs on the same side as the migraine pain.^{3,4} The congestion often progresses to rhinorrhoea once the attack resolves. In addition to these symptoms, patients also report a distinct nasal pain or an obviously red nose as an accompanying feature.^{5,6}

Investigations of contact point headaches date back to 1943. McAuliffe et al. stimulated the nasal mucosa with pressure, electricity and adrenaline and documented the pain perceived in assigned facial dermatomes.⁷ Although three out of the five persons tested were the authors of the publication and despite a later failure to reproduce the results, the role of the nasal mucosa in the generation of migraine pain gained considerable traction.^{8,9} In 1988, Stammberger et al. proposed a concept that the mechanical irritation of nasal mucosa causes the secretion of substance P, which mediates the pain and amplifies the stimulus.¹⁰ The link with migraine was proposed by Behin et al. who suspected intranasal contact points to act as a triggering or boosting factor.¹¹

The difficulty in interpreting these studies arises from the confounding symptoms of migraines, intranasal contact point headaches and headaches due to chronic sinusitis.¹² Migraine headaches are defined by diagnostic criteria (International Classification of Headache Disorders, ICHD-3)¹³; however, no diagnostic markers or tests are presently available. Despite the rare occurrence of chronic sinusitis, sinus headaches have a high prevalence in epidemiological statistics, suggesting that the correct application of the ICHD criteria might have shown migraine headaches as the true diagnosis in the majority of these patients.¹⁴

The nasal cavity is innervated by the olfactory nerve, the trigeminal nerve, and autonomic nerve fibres (Figure 1). The olfactory nerve plays a key role in odour recognition. The olfactory neuroepithelium is located high in the nasal vault beneath the cribriform plate and occupies merely 1 cm² on each side of the olfactory cleft. Only 10–20% of inspired air moves through this area, which is approximately 7 cm from the anterior nostrils.¹⁵ Contrary to the olfactory system, which is confined to a rather small area, the trigeminal nerve has fibres widespread throughout the nasal cavity. The superior and anterior parts of the septum and lateral nasal wall are supplied by branches of the ophthalmic division of the trigeminal nerve, while the maxillary division supplies the inferior and posterior parts. The trigeminal nerve is the

mediator of mostly unpleasant qualities such as pungent smell, stinging or pain. An example is the painful inhalation of very cold air described by MPs. These non-olfactory sensations have to be considered in conjunction with other sensations mediated by this nerve, e.g. eye irritation and painful cutaneous sensations arising from the face. Functionally, this yields at least two beneficial effects. It generates protective reflexes such as sneezing or an inspiratory stop with glottic closure, and it reduces the adaptation to stimuli of high intensity.¹⁶ Trigeminal afferents appear to be important as sentinels to the human airway. There are topographical differences within the nasal cavity, in that the mucosa in the anterior part of the nose has the highest responsiveness to chemosensory activation mediated through the anterior ethmoidal nerve.¹⁷ This corresponds to the hypersensitivity to certain odours in MPs.⁵⁴ The parasympathetic fibres synapse in the sphenopalatine ganglion and pass, together with the sympathetic fibres, through the sphenopalatine foramen into the nasal cavity to supply the arteries, veins and glands of the mucosa.¹⁸

To further understand and delineate the possible role of nasal structures in the pathogenesis of migraine headaches, we examined 412 MRI scans of MPs and 110 scans of non-migraine patients (NMPs, control group). The present study investigated whether MPs have a higher prevalence of standard variants of intranasal anatomy than NMPs.

Methods

We examined the MRI brain scans of 412 MPs and 110 patients without any known history of migraine headaches (Table 1). All MPs were referred by neurologists to a radiology clinic on the Charite University Campus in Berlin, Germany. The patients were diagnosed with migraine with or without aura and were receiving treatment from the referring neurologists at the time of the study. All scans were made between 2009 and 2014. In addition, 110 MRI brain scans of NMPs were examined as controls. The patients had no known history of rhinological or sinus disorders. All scans and epidemiological data were rendered anonymous before the examination. The 522 MRI data were available as T1 coronal and T2 axial scans (Siemens Magnetom Harmony, 1 T). The scans were examined for variations in normal nasal anatomy:

Concha bullosa

Concha bullosa was diagnosed when a turbinate was enlarged with a recognizable air-filled lumen.

Mucosal contact points

Contact points were defined as visibly non-separated areas between the turbinate and the lateral nasal wall or the turbinate and the septum.

Septal deviations

The occurrence of septal deviations and spurs was documented. The deviations were categorised as 'focal, C-

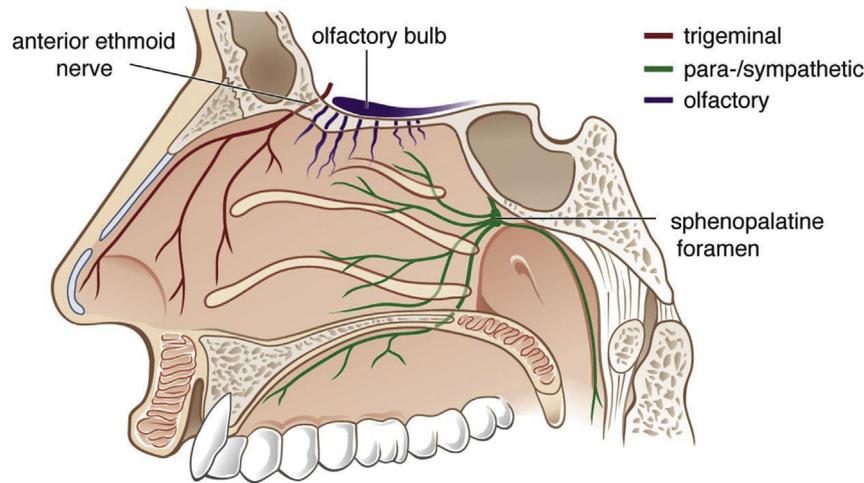


Figure 1 Innervation of the nasal cavity.

Table 1 Epidemiological data.

	Migraine group		Control group	
Patients	412		110	
Female	227	55%	57	52%
Male	185	45%	53	48%
Migraine without aura	270	66%	81	74%
Migraine with aura	142	34%	29	26%
Age, mean	40.90	SD 13.84	42.11	SD 13.71

shaped' and 'S-shaped', similar to the classification system devised by Guyuron et al.²⁰

Alterations of the sinuses

Sinuses were examined for the presence of polyps, fluid levels and mucosal swellings.

Other findings

Other anatomical findings included ethmoidal bullae, Haller cells, Onodi cells, pneumatized vomer and sinus hypoplasia.

Results

None of the scan results obtained showed any significant differences between the two groups. No significant difference was observed between the groups when the occurrence of anatomical variants, e.g. concha bullosa, septum deviation and spurs, and the functional descriptions, e.g. state of mucosal swelling and contact points were compared. There were no significant differences were found between male and female patients for any parameter.

The results of the study showed contact points between the turbinates and the septum in 82% patients of the migraine group (NMPs 80%) (Table 2). The most frequently observed contact point was between the middle turbinate and the septum, observed in 54% of the cases (NMPs 45%) (Figure 2). The second most common contact point was found between the inferior turbinate and the septum, observed in 43% MPs (NMPs 55%), whereas contact with the upper turbinate was found in only 2% (NMPs 1%). Contact points between the turbinates and the lateral nasal wall were found in 78% (NMPs 60%) of the migraine group. Of these contact points with the lateral wall, 50% (NMPs 51%) involved the middle turbinate, 46% (NMPs 48%) involved the lower turbinate and only 3% (NMPs 1%) involved the upper turbinate.

Table 2 Contact points.

		Migraine group		Control group	
		n	%	n	%
Concha bullosa	Inferior turbinate	1	0.2%	0	0%
	Middle turbinate	113	27%	19	21%
	Superior turbinate	15	4%	0	0%
Turbinate	Mucosal swelling	190	46%	43	39%
	Paradoxical twist	20	5%	1	1%
Septum	Deviation	328	80%	78	71%
	Focal	287	70%	65	59%
	c-shaped	26	6%	4	4%
	s-shaped	23	6%	9	8%
	Spur	168	41%	32	35%

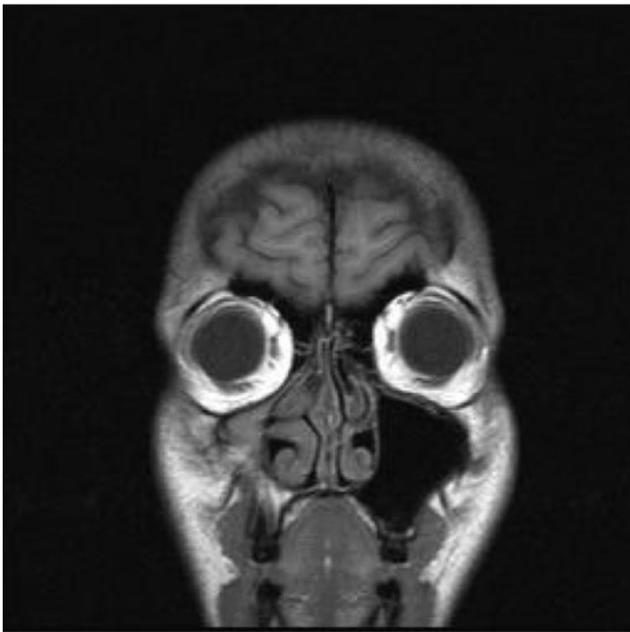


Figure 2 Concha bullosa of the left middle turbinate and hypoplastic right maxillary sinus.

In total, irrespective of the anatomical location of the contact, 87% MPs (NMPs 79%) had at least one contact point between opposing mucosal surfaces in the nasal cavity.

Concha bullosa of the middle turbinate was found in 27% MPs and 21% NMPs (Table 3). Only 4% MPs had a concha bullosa of the superior turbinate and we found only one patient with this variant in the inferior turbinate. We did not find any concha bullosa in the upper or lower turbinates of the non-migraine group.

Of patients in the migraine group, 80% had a septal deviation (NMPs 71%) (Figure 3). Out of these septal deviations, 85% in MPs vs 71% in NMPs were focal. Of the MPs, 6% had c-shaped or s-shaped deviations (4% of non-migraine patients (NMPs) had c-shaped septal deviations and 8% of non-migraine patient had s-shaped septal deviations) (Figure 4). The incidence of septal spurs was 41% in MPs vs 35% in NMPs independent of the presence of septal deviations (Figure 5). One patient had a paradoxically twisted right middle turbinate (Figure 6). Other findings of interest are displayed in Table 4.

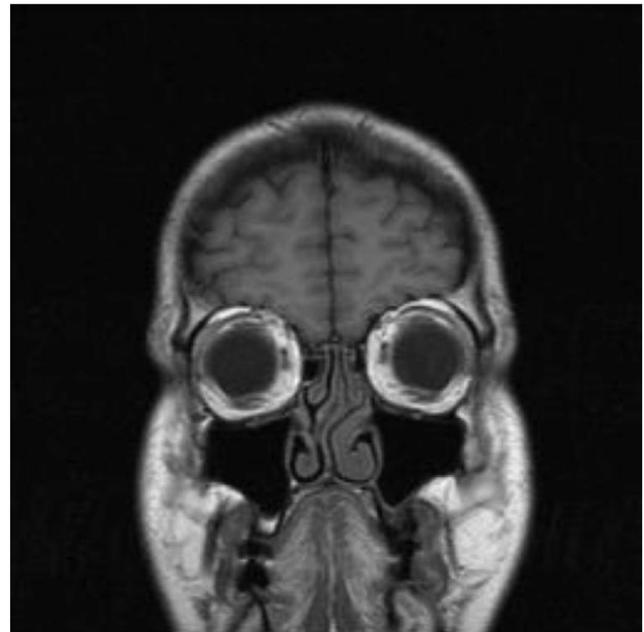


Figure 3 S-shaped septal deviation.

Discussion

Following the work of McAuliffe and Wolff described above, Bettington in 1951 reported on headaches caused by mucosal contact between the nasal septum and the middle turbinate.^{7,27} Morgenstein called the pain originating from such contact points a 'four-finger-headache' as patients were pressing their fingers on the eyelid, medial canthal area and nose.²⁸ The sensory innervation of the septum and the middle turbinate is supplied by the anterior ethmoidal nerve, which is believed to cause pain through referred signalling to other branches of the ophthalmic division of the trigeminal nerve. Goldsmith coined yet another name for the same condition: the 'middle turbinate headache syndrome'.²⁹

Reports on the surgical treatment of contact points in patients with headache date back to the mid-1960s.⁴⁰ Most of these publications appeared before any consensus on diagnostic criteria of migraine headaches existed. At that

Table 3 Concha bullosa and septal variants.

		Migraine group		Control group	
		n	%	n	%
Contact point turbinate-lateral nasal wall	Inferior turbinate	258	63%	64	58%
	Middle turbinate	279	68%	61	55%
	Superior turbinate	18	4%	5	5%
	Contact point morphology				
	focal	213	52%	41	37%
	broad-based	110	26%	25	23%
Contact point turbinate-septum	Inferior turbinate	274	67%	64	58%
	Middle turbinate	309	75%	69	63%
	Superior turbinate	13	3%	1	1%
	Contact point morphology				
	focal	216	52%	54	49%
	broad-based	124	30%	34	31%

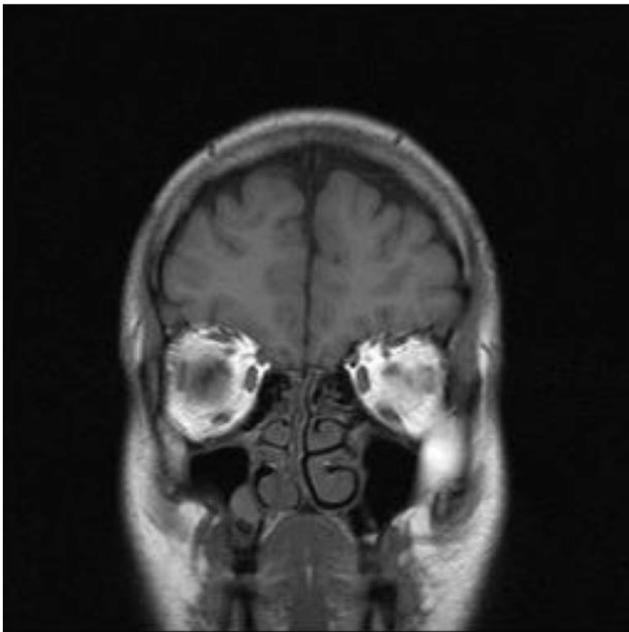


Figure 4 C-shaped septal deviation in contact with the turbinates on the right side.

time, no distinction was made between tension headache and migraine, or headaches were simply classified as migraine-like.^{30–32}

Reports on accurately diagnosed MPs showed a significant improvement or complete resolution of symptoms. In 1984, Novak reported on the influence of contact points on migraines.⁴¹ He used topical anaesthesia as a pre-surgical test to arrest developing migraine attacks. His first report on such pre-selected 42 MPs showed a significant improvement in all of them. His second study included 299 patients and showed complete remission in 78.5% and significant improvement in a further 11.5% patients.⁴² Guyuron et al. reported significantly beneficial results in 89% of 62 patients after the elimination of contact points.³⁵ In another study, a success rate of 64% (9/14) was reported in MPs.³⁶ Complete cessation of pain was reported in 43% of 21 patients in a study in which pre-operative testing with local anaesthesia and CT-scans was conducted.³⁷ A 10-year

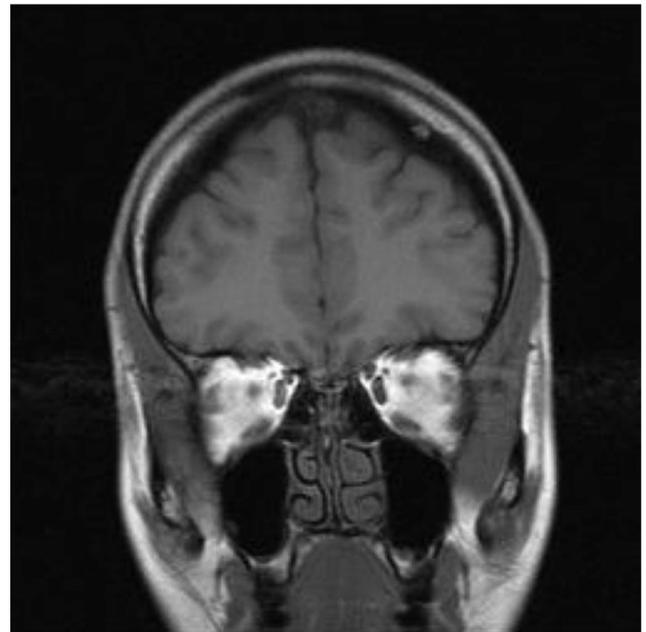


Figure 6 Paradoxically twisted right middle turbinate.

follow-up study on 15 MPs showed that 60% had achieved a significant and lasting improvement and 27% were pain-free.³⁸ Critics attributed the results entirely to placebo effects.³⁹

The discussion of conflicting theories on the role of nasal structures in the origin, or rather exacerbation of migraine headaches, requires the differentiation of entities with potentially confounding symptoms, e.g. contact point headaches and sinus headaches.

Mucosal contact point headaches are described by the ICHD-3 as a new entry to the classification, for which evidence is limited. Contact points are dealt with only in the appendix of the classification (A11.5.1) and are not considered primary or secondary headaches.²¹ The ICHD-3 knows of no association between migraine and intranasal contact points.

Sinus headache is a commonly made but is often a non-specific diagnosis and has to be differentiated from headaches attributed to rhinosinusitis. Applying appropriate

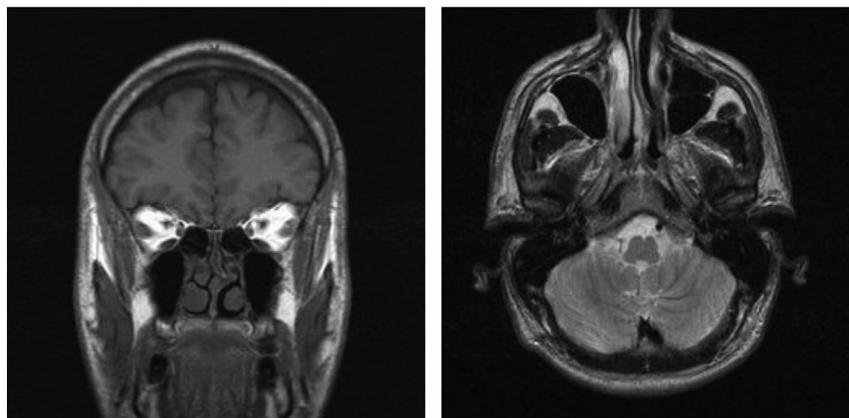


Figure 5 Septal spur in contact with the left middle turbinate.

Table 4 Other findings.

		Migraine group		Control group	
		n	%	n	%
Other findings	Ethmoidal cells	24	6%	5	5%
	Haller cells	87	21%	28	31%
	Onodi cells	0	0%	0	0%
	Pneumatized uncinuate process	14	3%	5	5%
	Pneumatized vomer	7	2%	0	0%
	Septal perforation	2	0%	1	1%
	Nasal agga cells	0	0%	0	0%
	Frontal sinus hypoplasia	6	1%	1	1%
	Maxillary sinus hypoplasia	5	1%	0	0%
	Fenestrated para-nasal sinus	5	1%	2	2%
	Concha bullosa with septum	0	0%	0	0%

diagnostic criteria, however, often shows that these patients are actually suffering from migraine without aura.²² Past attempts to define conditions that lead to migraine headaches of sinus origin showed that the definition can sometimes depend on whether the question is posed by otolaryngologists or neurologists.²³ Each discipline has produced sufficient literature either favouring the role of the sinus or refuting it.^{24,25} Overall, sinus headaches seem to be a largely over-diagnosed entity, which was corroborated by the screening of nearly 3000 self-reported or physician-diagnosed cases, of which 88% eventually fulfilled the diagnostic criteria of migraine headaches.²⁶

In the present study, 88% MPs with mucosal swelling at the time of the scan had contact points, in contrast to only 69% patients with contact points but without mucosal swelling. The highly variable prevalence of contact points depends on the state of the intranasal mucosal swelling, which in turn depends on a large number of physiological, pathological and therapeutic factors. This may explain the vast range of previous results, ranging from 4%, 30% and 55%.^{8,32,43} The matter gets even more complicated considering there is no consensus regarding the association and possible mutual influence of coexisting concha bullosa, septal deviation and rhinosinusitis.⁴⁴ A surprisingly large number of studies have either confirmed a higher coincidence of septal deviations and concha bullosa than their single occurrences or denied any such correlation.^{45–47} Our results did not find a higher associated prevalence.

The prevalence of concha bullosa is much higher in CT studies than that in cadaver dissections (34% vs 8%).⁴⁸ Our results of 27% patients with concha bullosa is comparable with the results of most other scan investigations.^{49,50}

Our findings showed septal deviations in 80% MPs and spurs in 41%. Similar results were reported previously.^{51,52} Yet again, one needs to consider whether comparable studies included mostly patients with rhinological problems or symptom-free patient groups; the latter results in prevalence ranging from 12% to 89%.^{48,52}

An overwhelming majority of 87% MPs in our study had at least one contact point. However, a similar number of 79% NMPs also showed contact points on their scans. Although contact points are highly prevalent, the diagnosis of contact point headaches is only rarely made. Thus, it seems logical to assume that although most MPs have contact points, in the majority of them, these contact points do not inevitably play a crucial role in triggering an attack as they are also present in NMPs. How can an anatomical variation as highly prevalent as nasal contact points lead to migraine headaches in only a comparatively small percentage of patients?

Triggers' are internal or external factors that can promote or elicit a migraine attack. This applies, however, only to patients with an existing migraine; these factors have no such effect in non-migraineurs. The factors in themselves are thus not capable of causing migraine as a disease, but merely triggering an attack. There is a plethora of known triggers, e.g. weather changes, certain foods, hormonal fluctuations and odours.

Most of these triggers can cause swelling of the nasal mucosa.⁴¹ This is reflected by the nasal congestion described by many MPs during an attack. Behin et al. suggested that the nasal swelling is caused by antidromic secretion of CGRP, substance P and other neuropeptides by the peripheral nerve endings of the trigeminal nerve in the nose.¹¹ The pressure build-up through this neurogenic oedema on pre-existing contact points in turn stimulates the afferent C-fibres of the trigeminal nerve. These fibres end in the neurons of the nucleus caudalis in the brain stem, which are known to be sensitised in MPs. The ensuing positive feedback loop will reinforce the migraine symptoms. The nasal pain during a migraine attack can be explained through referred signalling from the meningeal branches, which are responsible for the headache, to the nerves of the nasal mucosa, both of which are derived from the ophthalmic division of the trigeminal nerve. If the signalling is considered bidirectional, possible alterations at the meningeal end can give rise to nasal pain and vice versa, which demonstrates the potential relevance of the triggering capacity of contact points.

The contact represents a mechanical and hypoxic stimulus, which causes the secretion of substance P from nociceptive nerve fibres of the nasal mucosa leading to the formation of a neurogenic oedema.^{10,33} The swelling can reinforce the mechanical irritation only to cause the secretion of even more substance P—a viscous cycle. The topical application of intranasal substance P in the absence of contact points does not elicit pain.³⁴

A thickened nasal mucosa will enlarge and reinforce any pre-existing contact points. Next to these direct nasal mechanisms, external and internal triggers could instigate an attack by giving a so far clinically silent contact point a critical role through increased pressure secondary to the mucosal swelling. In addition, the developing nasal congestion could 'switch' any near-contact area into a full contact point and amplify the afferent signalling. These mechanisms can also depend on the MP's ictal or inter-ictal status, which would explain the contradictory interpretation of previous reports on the role of contact points in migraines.

Conclusion

Migraine headaches are an extraordinarily complex brain event. Any reductionist attempt to attribute this to one solely responsible pathophysiological component will fail to answer the plethora of open questions that remain. In assessing the role of the nasal cavity with migraines, key features should be considered such as the presence of any discomfort and pain or nasal congestion during an attack. Contact points in the nose are not the cause of migraine. They can, however, play an important role in the trigeminal afferent signalling to the brain. Contact points may thus trigger an attack and therefore should be considered in the assessment and treatment of migraines.

Conflict of interest

None.

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