Headaches

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Facial Pain

Facial pain or secondary headache can be caused by different disorders within the head and neck region. These may range from trauma, inflammatory disorders and tumours. The main focus of this chapter will be to discuss facial pain that is attributed to the nose/paranasal sinuses (previously known as sinugenic facial pain) and non-sinugenic facial pain-related disorders (see Table 14.1). Non-sinugenic facial pain is caused by disorders of the head and neck region, other than nose/paranasal sinus disorders and primary headaches [1]. These include several regions in the head and neck region such as temporomandibular joint and cranial nerves. There is an overlap of shared symptoms and aggravating factors between migraine and rhinosinusitis, and therefore patients with migraine may end up being referred to an otolaryngologist [2].

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Table 14.1 The differences between sinugenic and non-sinugenic pain [2]

Features	Sinugenic pain	Non-sinugenic pain
Pain severity	Mild to moderate	Moderate to severe
Pain quality	Facial pressure and nasal congestion	Throbbing and excruciating pain
Duration	72 h or more	Less than 72 h
Site	Depends on the sinus that is involved, mostly unilateral	Maybe be generalised or localised. There is a poor correlation between location of pain and sinus anatomy
Aggravating factors	Changes in atmospheric pressure (e.g. diving, skiing and flying)	It depends on the site of inflammation. Chewing, exercise, light touch and certain food (chocolate and cheese), bright lights
Associates signs and symptoms	Anterior and post nasal drip, nasal congestion, obstruction, hyposmia and anosmia	Photophobia, phonophobia, with or without aura, nausea or vomiting.
ENT examination	Nasal congestion and thick purulent rhinorrhoea	Anterior and posterior rhinoscopy normal
Rhinoscopy	Anterior and post nasal drip Oedema of nasal cavity with or without nasal polyps	Anterior and posterior rhinoscopy normal
Paranasal CT-scan	Opacification of paranasal sinuses and occlusion of osteomeatal complex	Normal or minimal findings on paranasal CT scan

Primary Headache Disorders

Primary headache disorders are functional illnesses that are not caused by anatomic, inflammatory, infectious, or physiological abnormalities. Approximately 98% of patients who present with a headache for medical evaluation will have a type of primary headache. The two major primary headache categories are migraine and tension-type headache [3].

Migraine

Migraine is an inherited disorder of the brain in which the brain is hypersensitive to the changes in the environment as well as changes that occur within the body. Changes in sleep, stress level, activity level, hormones and any traumas or other medical conditions that the body is experiencing can be common triggers – triggers are usually partial and additive.

Many epidemiological studies have documented its high prevalence and socio-economic and personal impacts. In the Global Burden of Disease Study, migraine is the second leading cause of years lived with disability (YLDs) and accounts for more disability than all other neurologic disordered combined [4].

The diagnosis is based on clinical criteria established by the International Classification of Headache Disorders, third Edition (ICHD-3): Migraine is a recurrent headache disorder manifesting in attacks lasting 4–72 h. Typical characteristics

of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia [5]. Migraine is a functional disorder and is a disabling primary headache disorder. Brain imaging is usually unrevealing, but nonspecific white matter lesions can be seen in the subcortical or periventricular white matter regions demonstrated on MRI of the head, reported in 12–48% of migraine patients compared with 2–11% of control subjects [6].

Migraine has two major types: migraine with and without aura. Migraine without aura is a clinical syndrome characterized by headache with specific features and associated symptoms as discussed above. Migraine with aura is primarily characterized by the transient focal neurological symptoms (visual, sensory, motor, retinal and brainstem) that usually precede or accompany the headache [5].

The understanding of the pathogenesis of migraine remains incomplete: it involves the trigeminal nerve and its axonal projections to the intracranial vessels. Nociceptive signals from the trigeminovascular system are related to areas of the brain that are responsible for the perception of pain [7]. There are signalling molecules involved in a migraine attack, such as calcitonin gene-related peptides (CGRP), which are potent vasodilators that are widely distributed in the trigeminovascular system [7] (Fig. 14.1).

CGRP ligands and receptors are widely distributed in the trigeminovascular system.

The physiological basis of the aura phase of migraine is hypothesized to be related to cortical spreading depression, a self-propagating wave of depolarization across the cerebral cortex that disrupts ionic gradients and is followed by cerebral hypoperfusion [9]. Hemodynamic changes accompanying cortical spreading depression have been documented on neuroimaging in patients who have migraine with aura, and not in patients who have migraine without aura [10].

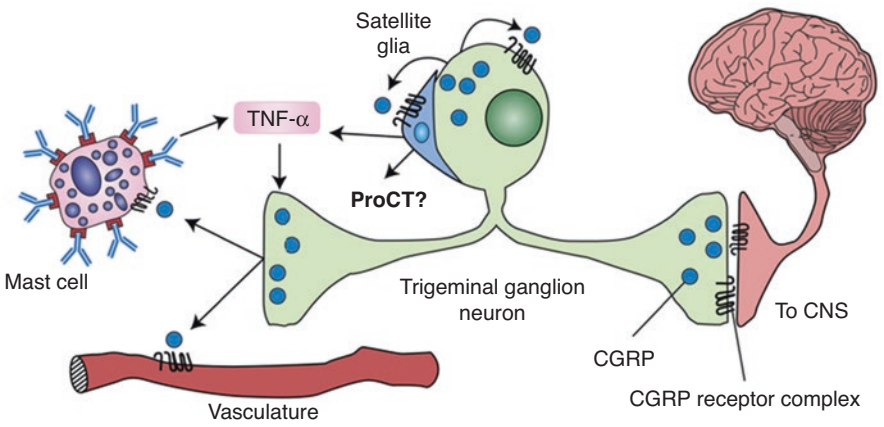

Fig. 14.1 CGRP action at peripheral receptors [8]

Misdiagnosis – 'Sinus' Headache

It is worth mentioning since migraine is a functional disorder and imaging is often unrevealing, misdiagnosis is common. A very large population-based study, entitled American Migraine Study II [11], demonstrated that many people who were diagnosed with migraine thought they had 'sinus' headache. Significantly, there were almost 20,000 study participants – only about 50% who were diagnosed with migraine knew they had migraine before the study. The most common misdiagnosis was 'sinus' headache.

True 'sinus' headache, more properly called rhinosinusitis, is a rare condition and is secondary to a viral or bacterial sinus infection characterized by thick, discoloured nasal discharge, alteration of smell, facial pain and/or fever. Symptoms should resolve within 7 days after the remission of viral symptoms or after successful treatment with antibiotics [12].

In a study of 3000 patients with 'sinus' headache, 88% of the participants were found to have migraine headache and not 'sinus headache' upon evaluation. Strict criteria from the ICHD-3 were used to tell the difference between the two headache types. In addition to their common symptoms of nasal and sinus congestion and facial pain and pressure, migraine patients had nausea, photophobia, moderate to severe headache, pulsing/throbbing pain and/or headache worsened by activity. In this study, almost 3000 patients with the complaint of 'sinus' headache were taking excessive over-the-counter and prescription decongestants, analgesics, antihistamines and nasal sprays without good relief. The lack of response supports the pain is related to migraine and not rhinosinusitis [13].

Facial Pain Secondary to Rhinosinusitis

Sinugenic facial pain is an uncommon cause of isolated facial pain. Incorrect diagnosis may lead to unnecessary medical costs [14]. Acute rhinosinusitis (ARS) or acute-on-chronic rhinosinusitis can be attributed to the cause of facial pain or headache. Chronic rhinosinusitis (CRS) alone typically does not cause significant facial pain unless there is an acute exacerbation. The site of facial pain depends on which paranasal sinus is involved. Patients commonly present with unilateral facial pain, fever and nasal symptoms (see Table 14.2). Toothache and unilateral facial pain are usually attributed to maxillary sinus infection. Frontal sinusitis may present with fever and facial pain around the eye and the supraorbital ridge. Recurrent acute sinusitis is often not sinusitis, and acute CT scans performed at the time of concern have demonstrated an absence of sinus disease [15].

The International Consensus Allergy and Rhinosinusitis (ICAR): rhinosinusitis 2021 has formulated recommendations for the management of rhinosinusitis in adults (see Table 14.3). The primary goal of performing endoscopic sinus surgery (ESS) is to optimize sinus drainage and allow for better topical therapy delivery. A long-term study of over 5 years has shown the resolution of facial pain post ESS in 47% of 51 cases [16].

Table 14.2 Diagnosis of ARS and CRS [17]

Rhinosinusitis	Duration	Symptoms	Nasal endoscopy	CT changes
ARS	Sudden onset Less than 12 weeks	Inflammation of the nose and paranasal sinuses characterised by two or more symptoms, one of which should be either nasal blockage/obstruction/ congestion or nasal discharge (anterior/posterior nasal drip): ± facial pain/ pressure ± reduction or loss of sense of smell and either	Nasal polyps and/ or mucopurulent discharge primarily from middle meatus and/or oedema/mucosal obstruction primarily in middle meatus and/or	Mucosal changes within the osteomeatal complex and/ or sinuses
CRS	More than 12 weeks			

Table 14.3 Recommendations from international consensus allergy and rhinology: rhinosinusitis 2021

	ARS	ABRS
Duration	<7 days	ARS > 7 days
INCS Fluticasone 100 μg Mometasone 200 μg Budesonide 400 μg	Recommended	Recommended
Saline irrigation	Not recommended	Recommended
Antibiotics	Not recommended	Recommended
Decongestants	Not recommended	Not recommended
Antihistamine	Not recommended	Not recommended

Medical Therapy

Corticosteroid Therapy in ARS

Corticosteroids have anti-inflammatory properties and therefore help to reduce intranasal mucosal inflammation which restores mucociliary clearance (MCC). Intranasal corticosteroid (INC) therapy has been recommended to be used as monotherapy within 7 days of ARS [18]. Although several studies [19–21] have looked at the use of oral corticosteroids in ARS, there is no conclusive evidence to support the use of oral corticosteroids in ARS. A Cochrane review meta-analysis of 1193 patients did not provide evidence to support the use of oral corticosteroids in ARS [22].

Antibiotics in ARS

Literature has shown that a watchful waiting approach may be beneficial in patients with ARS. It is recommended that antibiotics may be considered if there is no improvement after 7 days or if the patient develops worsening signs and symptoms. The choice of antibiotics is amoxicillin and clavulanate at a high dose of 4 g per day.

A systematic review has shown improvement of 88–97% in patients with acute bacterial rhinosinusitis (ABRS) in penicillin-resistant pneumococcal and beta-lactamase positive infection [23]. A Cochrane review has shown that the most common side effects such as gastrointestinal upset have been implicated in patient discontinuing antibiotic therapy. Moreover, these side effects were not debilitating, patients could still continue with their respective activities of daily living. Options after failing amoxicillin ± clavulanate or for penicillin allergy include trimethoprim-sulfamethoxazole, doxycycline or a fluoroquinolone [24].

Saline Irrigation in ARS

The use of saline irrigation as an adjunct to antibiotics has been recommended for patients with ABRS. Use of large volume (250 ml) saline nasal irrigation is a preferred method over low volume (10 ml) saline irrigation [24, 25]. Inanli et al. [26] assessed the effects of different concentrations of saline on MCC. Three groups of subjects were 10 ml 0.9% saline group, 10 ml 3% saline group and group without topical treatment. The resultant MCC time was compared amongst the groups and showed no difference. However, Gerlardy et al. showed the benefits of using 250 ml saline irrigation over 10 ml saline with improvement in rhinorrhea and postnasal drip [27].

Decongestants in ARS and ABRS

Decongestants help to reduce nasal congestion and restores patency of the sinuses. The risk of using decongestants poses a risk of the patient developing rhinitis medicamentosa if not monitored. The use of decongestants may help to reduce nasal congestion in ABRS [24].

Antihistamine in ARS and ABRS.

Braun et al. [28] in an RCT have shown an improvement in the use of loratadine in patients with allergic rhinitis. However European position paper on rhinosinusitis and nasal polyps (EPOS) 2020 guidelines and ICAR rhinitis 2021 have shown that there is no support for use of antihistamine in ARS/ABRS [24, 29].

Migraine Treatment

The foundation of migraine management is lifestyle modification. Specific strategies to create a regular and predictable schedule and environment that can be helpful for migraine. It is imperative that the patient keep a regular sleep schedule and meal schedule. Studies show routine aerobic exercise can decrease migraine frequency, severity and disability, ranging from 10% to 50% improvement [30–33].

In terms of pharmacological options, it can be divided into abortive and preventive treatments.

The most widely used abortive medications for migraine are nonsteroidal anti-inflammatory drugs (NSAIDs), which are low-cost, over-the-counter analgesic agents. Effectiveness has been best documented for acetylsalicylic acid, ibuprofen, and diclofenac with a success rate of around 20% for achieving pain freedom in 2 h [34–36] (Fig. 14.2). NSAIDs have anti-inflammatory effects via depression of prostanoid biosynthesis by inhibiting the COX enzymes and are able to prevent neurogenic inflammation [37].

Triptans are considered second-line medications. Triptans bind to 5-hydroxytryptamine (5-HT serotonin) receptors in the brain. Increasing the effects of serotonin mediates pain and mood. The downstream effect results in a reduction of CGRP. Currently, there are seven oral triptans (almotriptan, eletriptan, frovatriptan, naratriptan, rizatriptan, sumatriptan and zolmitriptan) available for clinical use. Standard dose triptans relieve headaches within 2 h in 42–76% of patients, and 2-h sustained freedom from pain was achieved for 18–50% of patients. Standard dose triptans provided sustained headache relief at 24 h in 29–50% of patients, and sustained freedom from pain in 18–33% of patients [39]. An example of treatment response with sumatriptan is illustrated in Fig. 14.3.

In practice, providers might use subcutaneous sumatriptan preparations for a patient who requires rapid onset of action. For patients with nausea and vomiting, rizatriptan or zolmitriptan are good alternatives. Rizatriptan, almotriptan and eletriptan have the advantage of the fastest onset. Almotriptan, frovatriptan and naratriptan have the advantage of having a favourable side effect profile. Frovatriptan and naratriptan offer advantages if the patient has problems with recurrence and if the headaches have a slow onset. Both might also have advantages in prevention and/or migraine with a prodrome [40]. Refer to Table 14.4 for the pharmacokinetics of the triptans.

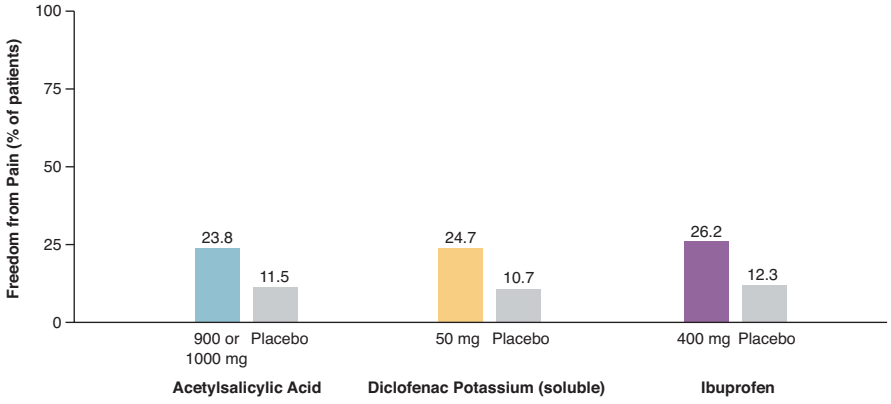

Fig. 14.2 Freedom from migraine pain (% of patients) from acetylsalicylic acid, diclofenac potassium (soluble) and ibuprofen [38]: all three analgesics demonstrated pain freedom in greater than 20% of patients in each respective studies [34]

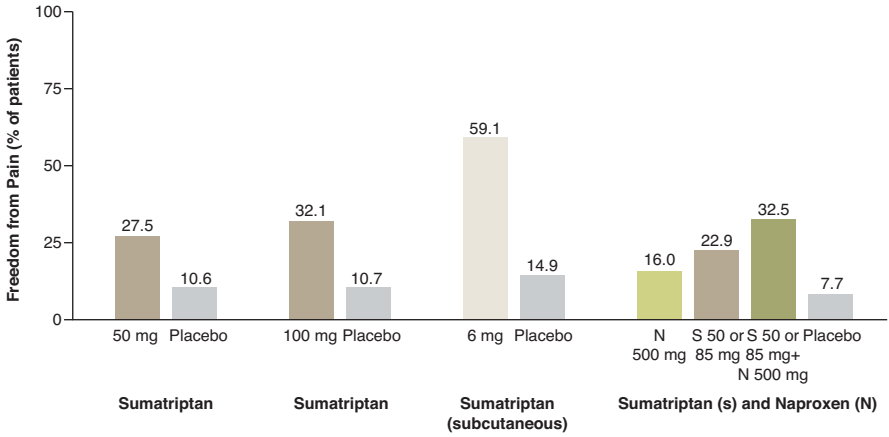

Fig. 14.3 Freedom from migraine pain (% of patients) from sumatriptan [38]

Table 14.4 Pharmacokinetics of the triptans [41]

Drug	Route	Onset	T_{max} (h)	$T_{1/2}$ (h)	Bioavailability
Sumatriptan	SQ	10–15 min	0.17	2	97%
	Intranasal	15–20 min	1.5	2	17%
	Oral	30–90 min	1.5	2	15%
Naratriptan	Oral	1–3 h	2	5.5	70%
Rizatriptan	Oral	0.5–2 h	1	2	45%
Zolmitriptan	Oral	1 h	1.5	2–3 h	40%
Almotriptan	Oral	1–3 h	2.5	3	70%
Frovatriptan	Oral	2–4 h	3	26 h	20–30%

NSAID-triptan combinations, dihydroergotamine, non-opioid combination analgesics (acetaminophen, ASA and caffeine) and several anti-emetics (metoclopramide, domperidone and prochlorperazine) are additional evidence-based options for migraine treatment. Opioid-containing combination analgesics may be helpful in specific patients but should not be used routinely [42]. Consensus guidelines advise against the use of opioids and barbiturates in the treatment of migraine, because of adverse effects and the risk of dependency and medication overuse headache [42].

New abortive migraine treatments are becoming available: small-molecule CGRP receptor antagonists, called gepants and the 5-HT1F receptor agonists, called ditans. The ditans act specifically on the 5 HT1F receptor as opposed to the traditional triptans which act on 5HT 1B and 1D receptors, hence the ditans can be administered in patients with comorbid or history of cardiovascular diseases [43].

Long-term management, such as preventive treatment may be required. The aim is to reduce the frequency, duration and/or severity of migraine attacks. The most widely used drug classes are antihypertensive agents (e.g. beta-blockers and

candesartan), antidepressant agents (e.g. amitriptyline) and anticonvulsant agents (e.g. topiramate and sodium valproate) [44]. For chronic migraine (greater than 15 headache days a month for more than 3 months, at least 8 migraine days and 5 migraine attacks) [5], the evidence-based effectiveness of topiramate and onabotulinumtoxinA (Botox) has been documented [45, 46]. New mechanism-based preventive therapies have recently been introduced, targeting the CGRP ligands or receptors. These include four injectable monoclonal antibodies (eptinezumab, erenumab, fremanezumab, and galcanezumab), which all have documented effectiveness in randomized trials for the preventive treatment of episodic and chronic migraine [47–53].

Tension-Type Headache

Tension-type headache is the most common type of primary headache disorder, with a lifetime prevalence in the general population ranging in different studies between 30% and 78% and it has a high socio-economic impact [4].

Tension-type headaches usually last from 30 min to 7 days. It is often a bilateral pain described as 'a band around the head' or vice-like. The pain is generally mild to moderate and is not worse with routine physical activity, which means that most people with tension-type headache continue their normal daily activities despite having their headache. A tension-type headache is not accompanied by nausea or vomiting. It may be accompanied by photophobia or phonophobia, but not both [5, 54].

The infrequent episodic tension-type headache usually has very little impact on the individual and, in most instances, requires no attention from the medical profession in contrast to frequent episodic tension-type headache which can be associated with disability [55]. Chronic tension-type headache (greater than 15 headache days a month for more than 3 months) is associated with decreased quality of life and high disability [56].

The exact mechanisms of tension-type headache are not known. Peripheral pain mechanisms are most likely to play a role in infrequent episodic tension-type headache and frequent episodic tension-type headache [46], whereas central pain mechanisms play an important role in chronic tension-type headache similarly in chronic migraine [56]. Increased pericranial tenderness can be seen in patients with any type of tension-type headache [57, 58].

Treatment.

Simple analgesics, such as NSAIDs or aspirin, are treatment options for infrequent episodic tension-type headaches. The use of combination therapies containing either butalbital or opioids for the treatment of tension-type headache is generally not recommended because of the risk of tolerance, dependency, toxicity, and the development of medication overuse headache [59]. If tension-type headaches are frequent, long-lasting, or associated with a significant amount of disability, then

preventive treatment is recommended. Amitriptyline has shown to be effective in both episodic and chronic tension-type headaches [55, 60, 61] and non-pharmacological treatments, such as biofeedback, relaxation, cognitive-behavioural therapy, acupuncture, massage therapy and/or physical therapy have shown to be beneficial in the management of tension-type headaches [62–67].

Diagnostic Dilemma

The diagnostic difficulty most often encountered among the primary headache disorders is in discriminating between tension-type headache and mild forms of migraine. Stricter diagnostic criteria have been suggested for tension-type headache in hope of excluding probable migraine that phenotypically resembles tension-type headache [5, 68]. There is a debate that tension-type headache and migraine are on the same disease spectrum and not completely distinct [69, 70].

Facial Pain

Functional facial pain can involve the trigeminal nerve, which is responsible for pain and sensation of the face through three main branches, ophthalmic: maxillary, and mandibular nerves. Trigeminal neuralgia (TN) is one of the most common causes of facial pain [71]. It is reported that 150,000 people are diagnosed with TN every year and it is most common in people over the age of 50 with a preponderance for females [72].

TN is a disorder characterized by recurrent unilateral, brief electric shock-like pains, abrupt in onset, affecting one or more divisions of the trigeminal nerve and triggered by innocuous stimuli [5, 73]. It may develop without an apparent cause (idiopathic) or as a result of a secondary cause such as demyelination, space-occupying lesion, infection, inflammation, neoplasm, etc. [74]. Classical TN is one of the most common types of TN suggestive of a vascular loop is in proximity to the trigeminal nerve identified on imaging [75].

Idiopathic TN is managed symptomatically. Anticonvulsant medications such as carbamazepine and oxcarbazepine are first-line treatments [76]. Other medications include gabapentin, baclofen, amitriptyline, nortriptyline, pregabalin, phenytoin, valproic acid, clonazepam, lamotrigine and topiramate [77].

If medications are ineffective in treating TN, several surgical procedures may help control the pain including microvascular decompression for classical TN [78] or lesioning procedures for idiopathic TN (percutaneous radiofrequency rhizotomy, percutaneous balloon compression, percutaneous glycerol rhizotomy and stereotactic radiosurgery) [77, 79–82]. If a secondary cause is identified, addressing the underlying cause is the treatment of choice.

Other cranial nerves and branches of the cranial nerves can be affected in a similar fashion as TN, including the glossopharyngeal nerve and nervus intermedius of the facial nerve [83, 84]. Treatment is similar to TN depending on the aetiology [84, 85]. The diagnostic criteria for these conditions can be found in Sect. 13 of the ICHD-3 [5].

Other Headaches and Red Flags.

Other primary headache disorders to consider include the trigeminal autonomic cephalalgias (TACs), primary cough headache, primary exercise headache and primary headache associated with sexual activity.

TACs are characterized by unilateral head pain associated with ipsilateral cranial autonomic features such as lacrimation, conjunctival injection and rhinorrhea [5]. A brain MRI is required to exclude intracranial pathologies, such as cavernous sinus and pituitary lesions [86]. Examples of TACs include cluster headache, paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA) and hemicrania continua [87]. The diagnostic criteria of these TACs are based on the frequency and duration of the headaches [88]. Acute treatments for cluster headache include triptans and oxygen [89, 90]. Treatments of prevention for cluster headache include verapamil [91], lithium [92] and galcanezumab [93]. Treatment for paroxysmal hemicrania and hemicrania continua respond completely to therapeutic doses of indomethacin [94]. SUNCT and SUNA may be responsive to lamotrigine or intravenous lidocaine [95].

Primary cough headache is a headache precipitated by coughing or other Valsalva (straining) manoeuvre and headache last between 1 s and 2 h [96]. Brain imaging is required to rule out a space-occupying lesion, especially in the posterior fossa [97]. Primary exercise headache is a headache precipitated by any form of exercise and the headache lasts for less than 48 h [98]. Primary headache associated with sexual activity is a headache precipitated by sexual activity. It often lasts from 1 min to 24 h with severe intensity and up to 72 h with mild intensity [99]. Brain imaging with vessel imaging is required to rule out vascular aetiology in both primary exercise headache and primary headache associated with sexual activity [100]. Indomethacin has been used to manage these primary headache disorders with some effectiveness [101].

Other rare primary disorders not discussed here are listed in the ICHD-3, including primary stabbing headache, nummular headache, hypnic headache and new daily persistent headache [5].

Contact Point Headache (Sluder's Neuralgia)

This is facial pain due to a contact point between two structures within nasal cavity, such as a nasal septal deviation or spur, a concha bullosa or medialized/hypertrophied middle turbinate. This phenomenon is also referred to as Sluder's neuralgia or

sphenopalatine ganglion neuralgia [2, 102]. The International Headache Society has recognized it as a diagnosis with the following criteria: (A) Intermittent pain in the periorbital, temporozygomatic or medial canthal region, (B) Endoscopy and/or CT evidence of contact points without acute sinusitis, (C) Evidence that the pain can be attributed to mucosal contact based on worsening with dependent positioning or on abolition of pain with local anaesthetic and (D) Pain resolves in 7 days following surgical removal of contact point [5].

The first-line treatment is a trial of nasal decongestants or local anaesthetic which should provide temporary relief of the symptoms. Surgical intervention maybe offered in the form of septoplasty or excision of concha bullosa. However, this is debatable, especially in cases where the mucosal contact point does not result in nasal obstruction [1]. Most evidence for surgical therapy is level IV, with 11–67% being pain-free after the procedure. A systematic review found there is insufficient evidence to support the removal of contact points in the management of facial pain and suggest an initial 6-week trial of amitriptyline [103].

Facial Pain and the Temporomandibular Joint

Facial pain can be directly related to pain arising from the temporomandibular joint (TMJ). Patients present with a history of facial pain that is caused by movement of the jaw, and chewing. On examination, application of pressure to the TMJ exacerbates the pain. Medical treatment involves the use of anti-inflammatories, analgesia, local warm compression to the TMJ or bite appliances. Second-line treatment may involve botox injections or be surgical and a referral to a dentist or oromaxillofacial surgeon is advised. The resolution of facial pain improves as the underlying TMJ disorder resolves [5].

Red Flags

Red flags can be elicited from a detailed headache history and exam. A commonly used published mnemonic is SNOOP (Table 14.5) [104]. If these features are identified, a secondary cause should be investigated. With regards to the choice of imaging, MRI is preferred over CT scan for most cases due to increased sensitivity, particularly for lesions in the posterior fossa, neoplasms, cervicomedullary lesions, pituitary lesions, intracranial hypertension/hypotension and vascular disease [6]. However in the acute setting, such as the emergency department, a CT scan could be performed first, especially for ruling out a gross lesion and haemorrhage including subarachnoid haemorrhage [6, 105].

Table 14.5 SNOOP mnemonics [104, 106]

	Stands for	Example	Differential diagnosis
S	Systemic symptoms	Fever, weight loss and fatigue	Infection (meningitis and encephalitis), giant cell arteritis, metastases and leptomeningeal carcinomatous
	Secondary risk factors	Malignancy, immunosuppression and HIV	
N	Neurologic symptoms/ signs	Focal neurologic deficits, altered consciousness and confusion	Mass lesion, stroke and hydrocephalus
O	Onset	Thunderclap and abrupt	Most common include: Subarachnoid haemorrhage, reversible cerebral vasoconstriction syndrome, pituitary apoplexy, cerebral venous sinus thrombosis and vasculitis
O	Older (especially >50 years)	New onset and progressive headache	Mass lesion and giant cell arteritis
P	Positional	Change lying versus sitting	Intracranial hypotension
P	Prior/ progressive	Different in quality from baseline	Mass lesion
P	Papilledema	Visual obscurations	Idiopathic intracranial hypertension
P	Precipitated by	Valsalva, coughing and sneezing	Posterior fossa lesion

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