The Nose and Associated Structures: Part I

33

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33.1 Applied Anatomy and Physiology

The nose and nasal cavity consists of the nasal bones and cartilages, the nares, vestibule, septum, lateral nasal wall and roof. This is surrounded by the paranasal sinuses (frontal, maxillary, ethmoid and sphenoid) into which they drain. Diseases in one can affect the others. The lateral nasal wall contains several, highly vascular turbinates (inferior, middle, superior and occasionally, supreme) and is the site of drainage for the ostiomeatal complex and nasolacrimal duct. At the back of the nasal cavity are the ethmoid sinuses and the nasopharynx. The ethmoid sinuses are a complex bony labyrinth, directly beneath the anterior cranial fossa. These are situated between the orbits, separated from them by a paper-thin lamina papyracea (Figs. 33.1 and 33.2).

Embryologically, five separate components contribute to the development of the nose:

1. Frontonasal processes forms the bridge of the nose.
2. Two medial nasal processes form the dorsum and tip of the nose.
3. Two lateral nasal processes form the sides and alae of the nose.

In essence, the two medial processes become the septum, philtrum and premaxilla (the 'primary palate'—the small anteriormost part of the upper jaw containing the four incisors). The lateral processes form the sides of the nose. Below the nasal complex, the stomodeum (future mouth) forms. A nasobuccal membrane separates the two.

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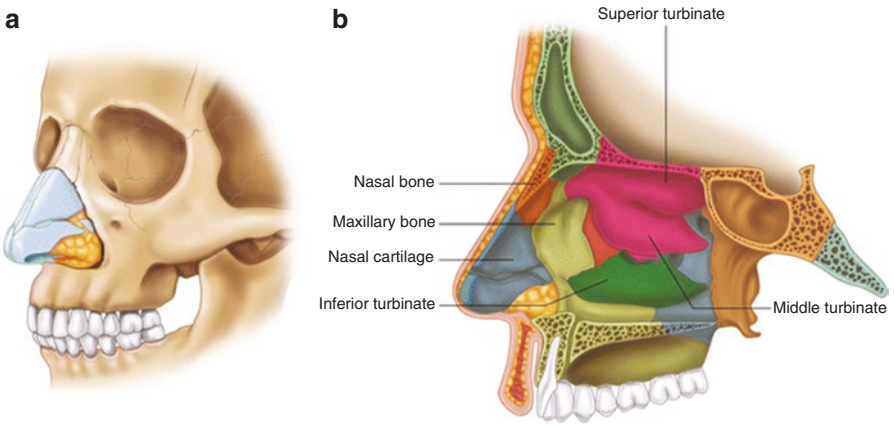

Fig. 33.1 (**a, b**) Anatomy of the nose. (**a**) External view, demonstrating nasal bones and cartilage. (**b**) Lateral wall of the nose, demonstrating the superior, middle, and inferior turbinates

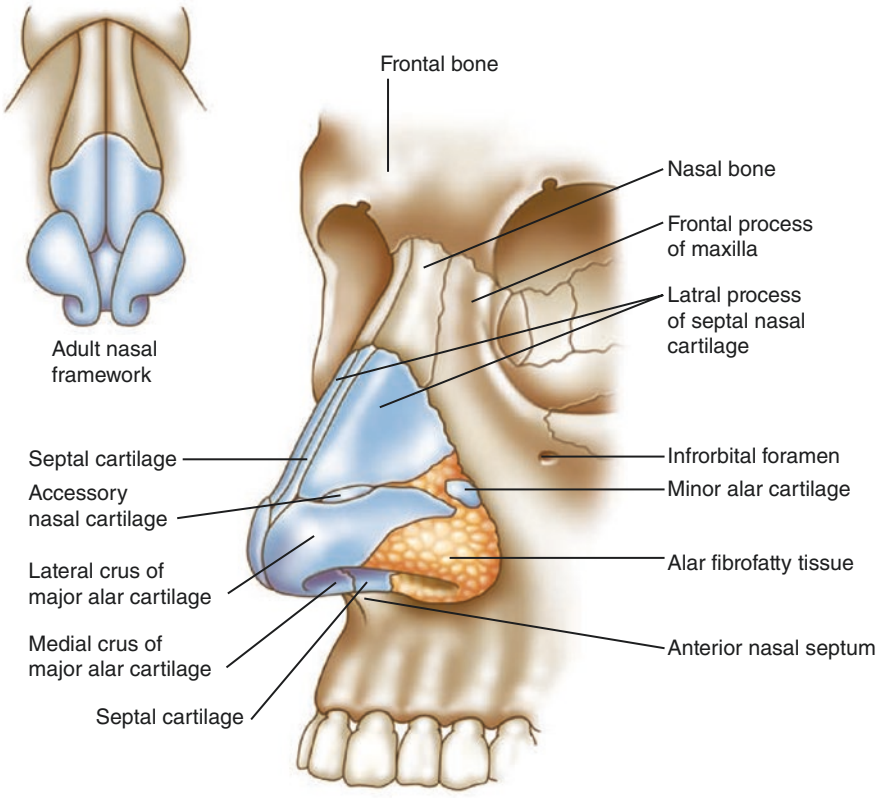

Fig. 33.2 Anatomy of the nose

Nasal placodes initially appear near the frontonasal process. These develop depressions or 'nasal pits' in the ectoderm, separating the medial and lateral nasal processes. The pits gradually move towards each other and deepen to form 'nasal sacs'. Each sac grows towards the developing forebrain to form a 'primitive nasal cavity'. The lateral nasal processes are separated from the adjacent maxillary process by the 'naso-optic furrow'. From each furrow, a solid cord of ectodermal cells develops and submerges below the skin, to later canalise and form the nasolacrimal duct. Persistence of this furrow can result in the development of an oblique facial cleft.

At the end of sixth week the nasobuccal membrane ruptures to form the 'primitive choanae'. The nasal cavity thus communicates freely with the primitive oral cavity. The conchae develop either side as curved elevations arising from the lateral wall of the nasal cavity. With the development of the 'secondary palate' and the nasal chambers, the primitive conchae come to lay at the junction of the nasal cavity and the pharynx. Around this time the ectoderm lining the roof of the nasal cavity becomes specialised to form the olfactory epithelium. This provides the origin of the olfactory nerves. The 'vomeronasal organs' of Jacobson are chemosensory structures on either side of the nasal septum. During development they appear in the 5th week and remain throughout life. In some animals they play an important part in reproduction and feeding habits. Developmental malformations of nasal cavities and nose can include:

1. Absence of a nose—nasal placodes fail to form
2. A single nostril—only one nasal placode forms
3. A bifid nose—the medial nasal processes do not merge completely. As a result the nostrils are widely separated and the nasal bridge is broad and bifid

The nose thus comes to occupy the central part of the face and as such is highly visible aesthetically. This position also makes it very susceptible to injury. The overlaying nasal skin varies in thickness significantly, both throughout its entirety and between different individuals. The nose is a highly vascular structure and therefore blood loss can sometimes be significant, even with isolated injuries. The nasal cavity lays between the orbits, separated by some of the thinnest bone in the body—the ethmoids. The anterior skull base immediately above the nose is also composed of thin bone. Therefore diseases of the nose and injuries can extend into the orbits and involve the globes. These can also potentially involve the anterior cranial fossa and its dural lining.

The skeleton of the nose is made up of bone and cartilage. This is often referred to as the 'nasal pyramid'. It is composed of the nasal bones, the frontal processes of the maxilla on either side of these and the nasal cartilages. The upper part of the nasal bones is thick, especially where they join the frontal bone. However they are thinner lower down, where they join with the upper lateral nasal cartilages. Thus nasal bone fractures are more common in the lower portion of the nasal bones. The upper lateral cartilages are connected to the undersurface of the lower end of the nasal bones. This junction (along with the nasal septum) is a key anatomical area in

nasal breathing—'internal nasal valve'. Injuries or disease here can significantly impair nasal breathing as well as affect the cosmetic appearance of the nose. The 'external' nasal valve refers to the junction of the upper lateral cartilages with the lower lateral (alar) cartilages. This is situated about 1 cm posterior to the entrance to the nose (the nares) (Fig. 33.3).

The nasal septum is a midline partition within the nasal cavity. It is composed of both bony and cartilage. It divides the cavity into two roughly symmetrical channels. The septum is composed of the perpendicular plate of the ethmoid posteriorly, the vomer and maxillary crest inferiorly and the quadrangular cartilage anteriorly. It is an important structure in maintaining nasal projection and the midline position of the nose. In a sense, this can be considered the 'tent-pole' of the nose, keeping it 'upright' (forward) and preventing it from falling to one side. Normally, the septum lies roughly in the midline, with the nasal passages symmetrically on either side. However it is common to see the nasal septum lay slightly off centre, although if it is very deviated, this is abnormal and often results in obstruction of one or both nasal passages. Obstruction to free airflow can result in poor drainage of the adjacent sinuses. This can result in a wide array of symptoms, not necessarily related directly to the nose (notably headaches, fatigue and other symptoms related to sleeping disorders or sleep apnea). Difficulty breathing and bleeding (epistaxis) are also common symptoms. By itself, a deviated septum can go unrecognised for many years without the need for surgical correction.

Internally, three pairs of conchae make up the lateral walls of the nose, the superior, middle and inferior conchae. These are lined by highly vascularised mucosa which is susceptible to bleeding. The osteomeatal complex is a surgical term used

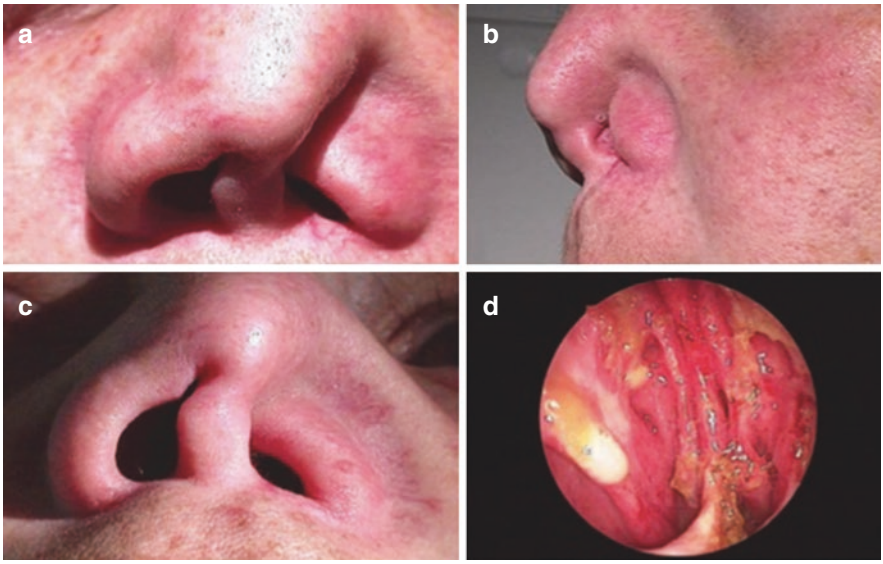

Fig. 33.3 (**a**–**d**) Alar collapse (**a**–**c**) and septal necrosis secondary to cocaine abuse

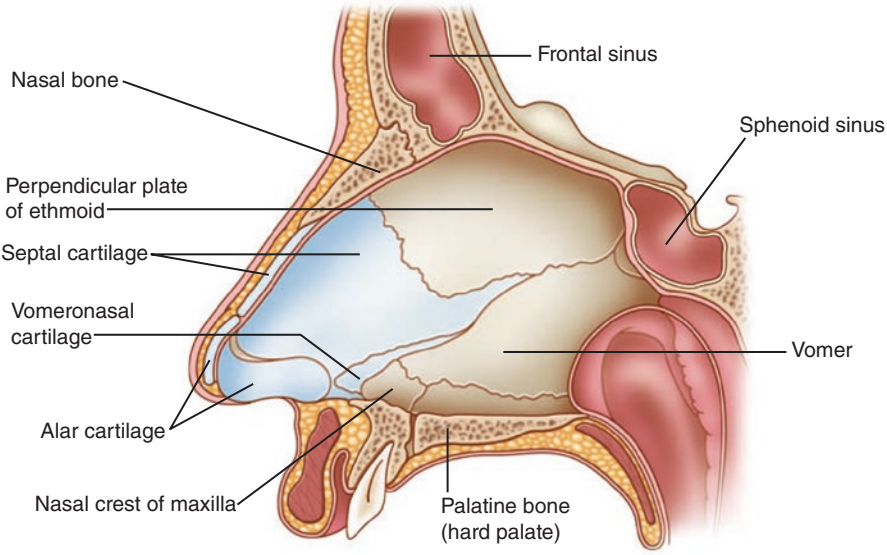

Fig. 33.4 Internal nose

for the narrow anatomical region bounded by the basal laminae, middle turbinate and orbital lamina. This space contains the ostia of the ethmoid, maxillary and frontal sinuses and is a key functional area in sinus disease. Pathology here can significantly affect sinus drainage resulting in sinusitis (Fig. 33.4).

Deep within the nose, between the orbits is the nasoethmoidal region. This is composed of very delicate bones and contains the ethmoid sinus. This region also supports the medial canthal attachments of the eyelids. This area is prone to collapse following a direct blow to the nose. The frontal sinus drains through this region and therefore nasoethmoid diseases or injuries can obstruct free drainage, resulting in frontal sinus symptoms. The nasopharynx sits behind the posterior nasal choana between the posterior edge of the hard palate, posterior pharyngeal wall and skull base. This is described later.

33.1.1 Choanal Atresia

This is a congenital condition of unknown cause in which there is closure of the posterior choanae in the nasal cavity. It is believed to occur when tissue that separates the nose and mouth during development persist after birth. Four theories have been suggested (1) persistence of the buccopharyngeal membrane, (2) abnormal persistence of mesoderm forming adhesions, (3) abnormal persistence of the nasobuccal membrane of Hochstetter and (4) ectopic neural crest cells. Atresia is rare, occurring in approximately 1 in 7000 live births and is seen more often in females

than in males. Over half of patients will have other congenital defects such as CHARGE (coloboma, heart disease, atresia of the choanae, retarded mental development, genital hypoplasia, and ear anomalies with deafness), Treacher Collins syndrome and Tessier syndrome. In newborns bilateral choanal atresia presents with airway distress is relieved by crying. Unilateral obstruction may not present until later. Treatment involves surgical dilation or transnasal resection endoscopically.

33.1.2 Congenital Nasal Pyriform Aperture Stenosis (CNAPS)

This is a rare cause of nasal airway obstruction which mimics choanal atresia but is important to differentiate as management is different. It is characterised by narrowing of the anterior nasal aperture. This may occur as a result of abnormal development of the primary palate, or bony overgrowth in the developing nasal process of the maxilla. Patients present with respiratory distress at birth, cyanosis on crying or difficult breathing during breast feeding. CT scan confirms the diagnosis when the pyriform aperture is less than 8 mm in total, or less than 3 mm on one side. Initial management includes topical decongestants, humidification and insertion of a oral airway. Surgery may be required to widen the bony channel.

33.1.3 Concha Bullosa

The middle concha plays a role in the sense of smell, air humidification and airflow regulation. Concha bullosa is also known as middle turbinate pneumatisation. It is a common anatomic variation of the middle turbinate which may be incidentally seen on paranasal CT in about one-third of cases. It can be either unilateral or bilateral and generally occurs together with a septal deviation to the contralateral side. Inferior and superior conchae bullosa have also been reported in the literature, but are quite rare. Concha bullosa is generally of little clinical importance. Most cases are asymptomatic but can occasionally present with nasal blockage and inflammatory sinus disease if very large. Treatment if required, is directed to reducing the size of the turbinate.

33.2 Blood Supply

The nose is a highly vascular structure and blood loss can be surprisingly significant if it is not controlled. The main arterial branches supplying this region include:

1. The ophthalmic artery (from the internal carotid artery). This divides into the anterior and posterior ethmoidal artery and the dorsal nasal artery.
2. Branches from the external carotid artery—the facial, infraorbital, sphenopalatine, greater palatine, superior labial, and angular arteries.

The nose therefore receives blood from both the external and internal carotid arteries. For this reason surgical control of bleeding can be very difficult. Ligation of the external carotid artery is unlikely to significantly reduce blood loss. Much of the external nose is supplied by the facial artery, which becomes the angular artery as it passes up towards the medial canthus, along the side of the nose. The dorsal region of the nose is also supplied by branches of the maxillary artery (infraorbital) and ophthalmic arteries (from the internal carotid). Internally, the lateral nasal wall is supplied posteroinferiorly by the sphenopalatine artery and by the anterior and posterior ethmoid arteries superiorly.

The nasal septum also derives its blood supply from the sphenopalatine and the anterior and posterior ethmoid arteries, with added contributions from the superior labial artery (anteriorly) and the greater palatine artery (posteriorly). Kiesselbach's plexus refers to the anastomosis of four arteries lying within 'Little's area' (the anteroinferior part of the nasal septum). These are (1) the anterior ethmoid, (2) sphenopalatine, (3) greater palatine and (4) superior labial vessels. This area is particularly prone to bleeding as a result of the drying effect of inspired air or from chronic nose picking. The veins of the nose essentially follow the arterial pattern. However they also communicate directly with the cavernous sinus. Infected emboli can therefore pass intracranially, with the risk of developing cavernous sinus thrombosis.

33.3 Nerve Supply

33.3.1 The Olfactory Nerve

What is usually referred to as the 'olfactory nerve' is more precisely called the olfactory tract and bulb. This is the first and shortest cranial nerve which develops as an unmyelinated outgrowth of the forebrain. It is one of only two nerves that do not enter the brainstem (the other being the optic nerve). It also is similar in structure to the optic nerve with a meningeal covering. The olfactory nerve is also capable of limited regeneration. Approximately 500 olfactory receptor cells are found within an olfactory epithelium, rather than connecting to a sensory ganglion (as seen in the other cranial nerves). This 'neuroepithelium' is comprised of modified nasal mucosa which lines the roof of the nasal cavity (sphenoethmoidal recess) and the adjacent upper nasal walls. The central processes from these neurons merge to form bundles (the olfactory fila), which pass through a number of small fenestrations in the cribriform plate and its attached dura, to terminate in the olfactory bulb. Collectively they become the first cranial nerve (CNI). Here they synapse with second order neurones. The olfactory nerve thus transmits neural stimuli from the olfactory epithelium of the nose to the brain. Disruption of the anterior cranial fossa by disease (notably tumours) or following significant trauma can result in irreversible damage to these fine filaments with disturbance or even loss of smell. Anosmia can also be caused by blockage of the nasal cavities, for example from nasal polyps or other growths.

Axons also connect with the limbic system (uncus and amygdala of the temporal lobe). This is why some particular smells can illicit specific memories or emotional responses and play a role in sexual arousal. Diseases involving the temporal lobe (such as epilepsy) can therefore cause olfactory hallucinations. Often these smells are unpleasant to the patient.

Olfaction has a complicated relationship with taste and the pleasures of eating and drinking involve both senses. Flavour is perceived by a combination of the senses of taste and smell and the action of the trigeminal nerve (CNV). The olfactory system recognises the odours as they pass over the olfactory epithelium. This is why an upper respiratory tract infection can temporarily result in the loss of taste. The trigeminal nerve senses texture, pain and temperature of food (for example the cooling effect of menthol or the burning sensation of spicy food).

Clinically, olfactory dysfunction can be quantitative or qualitative. It can occur bilaterally or unilaterally. Quantitative smell disorders involve complete or partial loss of olfaction (anosmia and hyposmia). With qualitative disorders (dysosmia) there is a subjective disturbance in the perception of smell. This can be classified into parosmia (an abnormal perception of an odourant) and phantosmia (the perception of an odour when there is no stimulus present). When a "phantom smell" lasts less than a few seconds, the term olfactory hallucination can be used. Cacosmia refers to an unpleasant perception of a smell as a result of nasosinusal or pharyngeal disease.

33.3.2 Sensory Innervation

The sensory innervation of the external nose comes from the ophthalmic (V1) and maxillary (V2) divisions of the trigeminal nerve. Branches from these include the infratrochlear, supratrochlear, external nasal branch of the anterior ethmoid and the infraorbital nerve. This is useful to know. By combining bilateral infraorbital nerve blocks with local infiltration around the bridge of the nose and it's base, anaesthesia of the entire external nasal surface is possible. This may be useful when treating extensive injuries, or removing skin cancers.

Internally, the upper aspect of the lateral nasal wall is supplied by the anterior and posterior ethmoid nerves. The sphenopalatine ganglion is located at the posterior end of the middle turbinate and innervates the posterior nasal cavity. This is an important ganglion involved in the sneeze reflex and some disorders such vasomotor rhinitis. The anterior and posterior ethmoid nerves and the sphenopalatine ganglion also provide sensation to most of the septum.

33.4 The Nasopharynx

The nasopharynx is a small space which connects the deep part of the nasal cavity (back of the nose) to the oropharynx. It is bounded anteriorly by the posterior nasal choanae and the nasal septum. The roof of the nasopharynx is formed by the

basisphenoid and basioccipital bones of the skull base. Its floor is formed by the superior surface of the soft palate and is therefore mobile. The nasopharynx therefore continues into the oropharynx at the level of the hard palate.

The posterior wall of the nasopharynx covers the first two cervical vertebrae, the pre-vertebral fascia and the superior pharyngeal constrictor muscles. The lateral walls lie medial to the pterygoid plates and parapharyngeal space. Here, the eustachian tubes enter the nasopharynx at Rosenmüller's fossa—a common site for cancers to occur. Because of this relationship between the nasopharynx and the eustachian tubes, disease in this area can result in symptoms related to the ears. This can sometimes resulting diagnostic confusion. Any patient with deep seated or unexplained ear symptoms should therefore have their throat examined in addition to their ears.

Branches of the external carotid artery supply the nasopharynx whilst the venous drainage is via the pharyngeal plexus, which drains into the internal jugular vein. Sensory innervation is from branches of the cranial nerves V2, IX, and X, as well as from sympathetic nerves. Some of these also supply the ear which can result also in referred pain.

Rathke's pouch cysts can develop high in the nasopharynx. These are cysts that develop from remnants of ectoderm that normally invaginate to form the anterior pituitary. Thornwaldt's cyst or bursa is a remnant of the caudal notochord. It represents persistence of an embryonic communication between the anterior tip of the notochord and the roof of the pharynx (remnant of Rathke's pouch). It can contain a jelly-like material which may become infected in later life.

33.5 Nasal Function

The nose has two major roles. It

1. Humidifies and warms air, whilst at the same time filtering out particles
2. Provides the sense of smell through the olfactory system.

The paranasal sinuses also contribute to the humidification process and diseases within the sinuses can affect the sense of smell.

33.6 Normal Commensal Flora of the Upper Respiratory Tract

Most of the upper respiratory tract (nasal and oral passages, nasopharynx, oropharynx and trachea) are colonised by normal flora. This is harmless and usually beneficial for the host. Commensal bacteria compete with pathogenic organisms for potential attachment sites and often produce toxins or acids which are bactericidal. The nose is colonised predominantly by staphylococci (Staphylococcus aureus and epidermidis) and diphtheroids. The normal nasopharynx is the chief carrier of common respiratory pathogens, including Streptococcus pneumoniae, Haemophilus

influenzae and Moraxella catarrhalis. In the oropharynx are also the alpha-hemolytic (viridans) streptococci. These bacteria help prevent invasion by pathogenic streptococci.

33.7 Sneezing

Sneezing is a protective reflex which empties the nasal cavity. It is a semi-automatic expulsion of air from the lungs through the nose and mouth, which usually occurs in response to irritation of the nasal mucosa (from dust, pollen etc.). These particles stimulate the release of histamine, which irritates the nerve cells in the mucosa and ultimately branches of the trigeminal nerve. Other stimulants include sudden exposure to bright light, a sudden fall in temperature, exposure to cold air, or viral infection. Sneezing can also be caused by paranasal sinus irritation as a result of congestion and allergies. The force of expulsion varies, but has been documented to be quite considerable. Spontaneous rupture of the pharynx is very rare but has interestingly been reported to occur as a result of "holding in a sneeze" (blocking the nostrils and mouth). Although generally harmless, sneezes spread disease through an aerosol of up to 40,000 droplets. Whilst most sneezes are normal or secondary to minor irritations of the nasal mucosa (allergies and viral infections), prolonged or intense sneezing warrants further investigation. The photic sneeze reflex (PSR, photoptarmosis), or 'ACHOO' (autosomal dominant compulsive helio-ophthalmic outbursts) is a phenomenon where sudden intense sunlight may trigger sneezing. Walking out of a dark building into bright sunlight is one example. This tendency has been reported to be an autosomal dominant trait and affects 18–35% of the population. The intensity of these sneezing sessions can increase with time.

33.8 Mucociliary Clearance and the Nasal Cycle

The nose and upper respiratory tract are a frequent site of infection and irritation because they are regularly exposed to microbes and pollutants during breathing. Mucociliary flow is an important mechanism which helps prevent irritation and infection both within the nose and the paranasal sinuses. The nose and sinuses are covered by ciliated epithelium which beats up to 1000 times a minute. This epithelium also produces mucous that traps inhaled particles and microorganisms. These are then carried out on a 'mucus blanket' which is replaced about every 10– 30 min. Mucous and fluid production within the sinuses has been estimated to be as much as 1–2 l every day.

The nasal cycle refers to a cyclical swelling of the turbinates and nasal mucosa that periodically occurs. This alternates between the two nasal passages, such that throughout a 24-h period airflow will pass preferentially through one side of the nose and then the other, alternating several times. Asymmetrical enlargement of the turbinates is normal. The nasal cycle and mucous production are regulated by the autonomic nervous system and neuropeptides from sensory neurones. Both parasympathetic and sympathetic receptors stimulate secretion. Stimulation can

therefore result in increased mucosal swelling and fluid production. Drugs that have a vasoactive effect can increase the thickness of mucosal lining and increase serous fluid production. Fluid filled sinuses may therefore be an incidental finding, especially in debilitated patients on varying medications. An gas-fluid level seen on imaging can occur in healthy patients and is not significant, unless symptomatic.

33.9 Nasal Congestion

This is discussed further later in this section. Nasal congestion is a common symptom, often seen in rhinitis (both allergic and nonallergic), rhinosinusitis and nasal polyposis. Congestion can also be caused by mechanical obstruction of the nasal passages or alteration of sensory perception. Mucosal inflammation arises as a result of the release of a wide range of active agents, such as histamine, interleukins cell adhesion molecules and tumour necrosis factor alpha. These lead to venous engorgement, increased secretions and localised swelling. Congestion can impair the sense of smell and other nasal sensations as well impairing nasal airflow. Nasal congestion is not to be confused with the nasal cycle. The latter is physiological congestion. Pathological congestion varies significantly and can have many causes including:

- The common cold/URTI
- Deviated septum
- Allergic and non-allergic rhinitis
- Rhinitis medicamentosa
- Sinusitis
- Nasal polyps
- Empty nose syndrome
- Gastro-oesophageal reflux disease
- Choanal atresia
- Concha bullosa
- Adenoid hypertrophy

Complications of nasal congestion include poor sleep, leading to daytime somnolence and occasionally obstructive sleep apnoea. Nasal congestion can also be a presenting feature of otitis media and asthma. Management therefore depends on the underlying cause. Antihistamines, decongestants and steam inhalation may also be used for symptomatic relief. Nasal congestion in an infant can interfere with breastfeeding and cause respiratory distress (infants are obligate nasal breathers).

33.9.1 Nasal Irrigation

Nasal irrigation (nasal lavage or douche), involves washing out the nasal cavity to remove excess mucus and debris from the nose and sinuses. A simple method is to pour irrigant into one nostril and let it run out through the other while the mouth is

kept open to breathe. Special "neti pots" can be used for this. Spray and nebulisers may also be used to moisten the mucous membranes. Patients with chronic sinusitis often benefit from this as an adjunct for other treatments. Nasal irrigation also alleviates symptoms in hay-fever and the common cold. Daily irrigation is undertaken using a warm salt water solution, with or without sodium bicarbonate. While ordinary tap water is often used, this is not sterile and can irritate the mucous membranes.

33.10 The 'Runny Nose': Rhinorrhoea

This term refers to a watery nasal discharge from whatever cause. In general terms, either the sinonasal mucosa produces large amounts of clear fluid, or fluid enters the cavity from elsewhere (notably CSF). Rhinorrhoea is a very common symptom and can be accompanied by sneezing, epistaxis and anosmia. It is commonly seen in allergy sufferers (hay fever) and the common cold, but can also occur during crying, exposure to cold temperatures, cocaine abuse or withdrawal from drugs such as opioids. Common and important causes include:

1. Rhinitis (all types)
2. Upper respiratory tract infections
3. Sudden exposure to cold air
4. Cocaine use/Opiate withdrawal
5. Lacrimation
6. Anterior skull base fractures (CSF rhinorrhoea)
7. Cystic fibrosis
8. Whooping cough
9. Nasal tumours
10. Cluster headaches
11. Primary ciliary dyskinesia

Management depends primarily on the cause. In most benign cases treatment is not necessary and rhinorrhoea will settle. Saline nasal sprays and vasoconstrictor sprays may occasionally be used, but prolonged use can cause rhinitis medicamentosa. Before prescribing this it is important to rule out any underlying pathology requiring specific management.

33.10.1 CSF Rhinorrhoea

This specific type of rhinorrhoea needs urgent identification. Leakage of cerebrospinal fluid through the nose occurs as a result of a communication between the nasal cavity and sub-arachnoid space. In order for this to occur there has to be a breach in four distinct tissue layers; (1) nasal mucosa, (2) skull base, (3) dura and (4) arachnoid. Since this is most commonly associated with head injuries, this is discussed in greater detail in the chapter on the head. Leakage of the cerebrospinal fluid into the

nasal (or aural) cavities may also be caused by infection, hydrocephalus, congenital malformations, tumours and following intracranial surgery. Infrequently it may occur spontaneously. Thus CSF leakage must be differentiated from allergic rhinitis or infectious rhinosinusitis.

CSF leaks place the patient at risk of meningitis. It is most commonly seen following head trauma, but can occur spontaneously or in association with intracranial hypertension. It can also occur secondary to skull base tumours and tumours arising from the nasal cavity. Connective tissue disorders have also been suggested as playing a role in some spontaneous leaks, and CSF rhinorrhoea has been reported in association with aortic aneurysms, joint hypermobility, Marfan syndrome, Ehlers-Danlos syndrome and autosomal dominant polycystic kidney disease.

Loss of CSF results in intracranial hypotension, characterised by orthostatic headaches (headaches that worsen when upright and which improve on lying down). The lack of the cushioning effects of CSF allows the brain to descend through the foramen magnum, resulting in distortion of the nervous tissue and other varied symptoms. Nausea, vomiting, dizziness and fatigue may also occur. CSF rhinorrhoea can often be successfully closed with endoscopic techniques.

33.11 Post Nasal Drip

This symptom can sometimes be both related, or confused with rhinorrhoea. It occurs when excessive mucous is produced by the sinonasal mucosa. Mucus accumulates in the postnasal space as a result of reduced mucociliary clearance. The secretions pass from the nose or paranasal sinuses into the pharynx, usually when the patient is supine. It is therefore often seen to occur at night. Post nasal drip can develop into a chronic condition following repeated episodes of colds and 'flu'. It is also recognised as one of the causes of chronic cough (along with asthma and gastroesophageal reflux disease) and is thought to be responsible for chronic cough in up to 87% of patients. Terminology can be a little confusing. In UK the term 'rhinosinusitis' is commonly used to describe "post nasal drip syndrome" and sometimes the term "upper airway cough syndrome (UACS)" is used instead of PNDS-induced cough. However it is not clear if there is any difference between these conditions. Patients often describe abnormal sensations in the nasopharynx, mucous expectoration and excess mucus in the nasopharynx and oropharynx. Intermittent cough is common.

Post nasal drip can be caused by a number of conditions which disturb the normal production of mucus from the nasal and sinus cavities. These include:

1. Allergic rhinitis
2. Enlarged adenoids
3. Sinusitis
4. Gastroesophageal reflux
5. Polyps (cystic fibrosis)
6. Mucociliary dysfunction

7. Anatomical anomalies
8. Smoking and exposure to toxins

In addition to the main symptom of cough, patients may complain of the sensation of something draining into the throat, a need to clear the throat, a tickle in the throat, nasal congestion, or nasal discharge. Hoarseness, wheeze, halitosis, constant swallowing and a chronic sore throat are also common symptoms. Treatment is both symptomatic and targeted towards the underlying cause. This includes antibiotics, decongestants, nasal irrigation, sinus massage, proton pump inhibitors (for gastro-oesophageal disease), antihistamines and surgery in selected cases.

33.12 Disturbances in Smell: Anosmia and Dysosmia

Anosmia is the complete loss of the sense of smell. This may be temporary, but in some cases, it can be permanent. This is rare. Reduction in the sense of smell (hyposmia) is more common and usually resolves. Dysosmia is a term used to describe any qualitative alteration or distortion in the perception of smell. This can be classified as either parosmia (troposmia) or phantosmia. Parosmia refers to a distortion in the perception of an odourant in which the odourant smells different from what one remembers. Phantosmia refers to the perception of an odor when there is no actual odourant present. Overall the incidence of olfactory dysfunction is unknown with reported incidences of up to 16% (hyposmia).

The causes of dysosmia are largely unknown but it is thought to be a neurological disorder. Most cases are described as idiopathic, but the main associations with parosmia are URTIs, head trauma, and nasal/paranasal sinus disease. Parosmia tends to resolve spontaneously. Overall, the most common causes of olfactory disturbances are chronic rhinosinusitis, upper respiratory tract infection and head trauma, but the list of all possible causes is extensive. Infections, tumours, foreign bodies and disorders affecting the olfactory epithelium, bulb and tract must be carefully considered. Rare causes include exposure to certain insecticides or solvents, Cushing's syndrome, Diabetes, Epilepsy, Multiple sclerosis, Olfactory esthesioneuroblastoma and amoebic meningoencephalitis. A full ENT and cranial nerve examination is therefore important.

To confirm the diagnosis, commercial available smell test kits are available. The Sniffin' Sticks test consists of felt-tip pens that are presented in front of the patient's nose. Three aspects are tested (1) the odour threshold (2) odour discrimination and (3) odour identification. These three results are added together to provide a total score. Objective assessments use an olfactory stimulus to elicit transient changes in electrographic activity. Further investigations depend on the suspected underlying cause. CT and MRI may be required to exclude inflammatory and neoplastic lesions. MRI is often the imaging modality of choice as it visualises the olfactory bulb, olfactory tract and central olfactory projection areas. Management depends largely on treatment of any underlying cause. Butyrophenones or thioridazine hydrochloride, both of which are dopamine antagonists, have been used to treat hyperosmia.

33.13 Important Considerations When Taking a History

Patients may present with a variety of symptoms related to the nose and nasal cavity. The more common symptoms include:

1. Blocked nose
2. Crusty nose
3. Epistaxis
4. Injuries
5. Irritation and itchiness
6. Loss of smell (Anosmia)
7. Offensive smell (Cacosmia)
8. Pain
9. Runny nose
10. Sneezing
11. Swelling

Many of these may be confined to one side of the nose, or they may involve both sides. Symptoms that remain confined to one side imply fixed and possibly localised pathology (tumour, trauma, ulceration, malformation etc.). Symptoms that periodically alternate between the two sides of the nose imply that both are involved, but that the underlying problem is affected by the nasal cycle. Recurrent bleeding from one side of the nose is often related to chronic trauma or vascular fragility, where as recurrent bleeding from both sides may occur as a result of a medical disorder (notably hypertension and clotting abnormalities). Symptoms of infection (swelling, redness, discharge and systemic symptoms of fevers, sweats) are important to identify. Infections arising within the nasal cavity can quickly spread to the paranasal sinuses or the overlying skin. These are potentially very serious, particularly if accompanied by a fever. Patients are at risk of significant complications including sinusitis, orbital cellulitis and cavernous sinus thrombosis. Infections involving the roof of the nose, although very uncommon, can extend upwards resulting in meningitis.

With the exception of trauma, many diseases of the nose do not present with acute pain. Rather, patients complain of symptoms of irritation or discomfort. As with elsewhere, the nature of the pain can often provide important clues as to the possible pathology. Throbbing pain implies an increased blood supply so is commonly seen in infections. Deep-seated and unrelenting pain implies local tissue destruction or pressure. Associated features are important to note, particularly loss of smell/abnormal smells, nose bleeds, blockages, nasal discharge. Whilst some pathologies may present with a single symptom (for example epistaxis related to chronic nose picking, or deranged clotting function), the presence of associated symptoms my help establish the diagnosis. Painful recurrent nosebleeds may be related to trauma, tumours, infections or other destructive pathologies. Abnormal smells may indicate the presence of infections or necrotic tissue.

Following trauma, the mechanism of injury is very important. This is discussed in more detail in the chapter on the injured patient. Whilst most nasal injuries are

self evident, it is important to consider the possibility of deeper extension of the injury to involve the nasoethmoidal region, orbits and anterior cranial fossa. Therefore ask about symptoms of other injuries, notably neck problems, CSF (clear fluid from nose), vision, headaches. With high energy impacts CT of the facial bones and skull base is usually indicated.

Finally, ask about the patient's medical, social, family and occupational history. Previous nasal surgery, medical problems and asthma may all be relevant to the diagnosis. Note any drugs that might cause nasal problems. This includes 'recreational' drugs (such as cocaine) as well as the overuse of prescribed medications. Topical vasoconstrictors should only be prescribed short term. Allergies to drugs and other irritants e.g. cats, pollen should be recorded. A sudden and prolonged change in the quality and humidity of the inspired air can have an adverse affect on the nasal mucosa. This may occur when travelling abroad. Note habits such as nose picking. A family history of asthma and bleeding disorders, smoking and occupational environmental hazards may also be relevant.

33.14 Examining the Nose and Associated Structures

Thorough examination of the nose and nasopharynx requires (1) a headlamp, (2) nasal mirror, (3) tongue depressor (to visualise the post nasal space) and (4) Thudichum's Speculum (to view the anterior-inferior nasal septum and Little's area). Note any skin lesions, lumps, lacerations, bruising, swelling and erythema, as well as the general shape of the nose. With regards to the shape and position of the nose, ask the patient whether their nose looks different from normal and if so, to define exactly how it has changed. Tilting the patient's head back allows inspection of the columella and the alar cartilages. Carefully elevate the tip of the nose with your thumb, so that the nasal cavity becomes visible. Use a pen torch or otoscope as a light source to externally illuminate the cavity. Elevating the tip of the nose will also show columellar dislocation. Inspect the nasal mucosa using a speculum, noting its colour, texture, ulceration and hydration. Also note any internal swellings such as enlarged turbinates, septal haematoma, perforation, foreign bodies and occasionally infestation (Figs. 33.5 and 33.6).

Look to see if there is any nasal discharge. If this is present note its colour, amount and see if you can identify where it is coming from. The type of discharge may give an indication of the cause:

1. Clear and watery mucous: nasal allergy or an upper respiratory tract infection
2. Yellow or green mucous: infection (viral or bacterial) or stasis
3. Mucopurulent discharge is unilateral in an adult is likely to be a sinus infection but especially in a child consider the possibility of a foreign body.
4. Thick white mucus: chronic rhinosinusitis.

Inspect the post nasal space through the mouth using the nasal mirror to look at the posterior choanae, inferior turbinates, tonsils and adenoids. Anterior rhinoscopy is essential part of the nasal examination. Ideally, either a headlight or

Fig. 33.5 Significant deformity following trauma

head mirror should be used. Anterior rhinoscopy allows direct examination of the anterior and mid-nasal structures, including most septal deviations, the turbinates and the response of tissues during quiet and forced inspiration. Alternatively an otoscope can also be useful to look inside the nose as it is readily available in most emergency departments and clinics. This has a good light source and with a wide speculum tip provides a clear view. When placing the otoscope into the nose it is important to remember to enter horizontally at first and then change the angle of the otoscope once inside the nasal cavity. In essence you are looking backwards. Swellings around the base of nose can be secondary to tumours, dental infections, tooth cysts etc. Therefore further examination of the upper teeth may be indicated.

Then gently feel the nasal bones and cartilages for tenderness and asymmetry. Nasal patency and airflow can be checked in two ways:

1. Place your thumb over the nostril not being assessed to occlude air flow. Ask the patient to breath in through their nose and note the degree of airflow. Ask them to describe adequacy of flow. Then repeat on the other nostril, noting any difference in apparent airflow.
2. Place a cold shiny surface, such as a mirror under the nose. Look for misting of the metal surface as the patient breathes out, compare the pattern corresponding to either side. Absence of misting or a disparity in the amount may suggest unequal airflow through a particular nostril.

Fig. 33.6 Internal inspection reveals a septal perforation

Reduced airflow through a particular nostril may indicate the presence of something blocking that air passage, such as a polyp, deviated nasal septum or foreign body. During inspiration look for 'alar collapse' (collapse of the lateral aspect of the lower nose as the pressure falls internally). This indicates that there is some inherent weakness within the internal nasal valve. This rarely arises as a result of disease, but can occur following injury. The function of the internal nasal valve can be further assessed using Cottle's manoeuvre. Place the thumb in the nasolabial furrow and push the cheek laterally. Then ask the patient to sniff in vigorously. A positive test is seen when the airway on that side improves with outward movement of the cheek. A more specific test is to very slightly lateralise the upper lateral cartilage with a small probe and test for reduced airway collapse on inspiration. In some cases weakness of the internal nasal valve can be treated surgically with a 'spreader' graft. Although rhinomanometry is the only quantifiable objective measure of nasal function currently available, this is not routinely used in the emergency setting.

In experience hands, nasoendoscopy may also be performed. This is normally undertaken after topical local anaesthetic has been applied. The scope is then passed.

This can be used to visualise the nasal turbinates and passed deeper into the nasal space to visualise the entire upper respiratory tract, down to the vocal cords. Following trauma to the nose, always make sure you assess and document the following:

1. Occult haemorrhage (especially in supine patients)
2. Intercanthal distance (nasoethmoid fractures).
3. CSF leaks and other signs of head injury—headaches, loss of consciousness, nausea and vomiting
4. Ocular signs—diplopia, bruising, loss of visual acuity, restricted eye movement, chemosis, proptosis
5. Septal haematoma
6. Mucosal tears

Finally, always remember to examine the Eustachian tube orifices, fossae of Rosenmüller and adenoid tissue in the postnasal space, the sphenoethmoidal recesses (above the choanae), the middle meatus and the anterior skull base. These are all intrinsically linked to the nose both anatomically and pathologically.

33.15 Investigating Symptoms and Signs

33.15.1 Laboratory Tests

A full blood count may help identify anaemia, infection and platelet deficiency. In patients with severe or recurrent epistaxis this should always be taken. A coagulation screen looking for clotting abnormalities (drug induced or pathology) is also usually required. A RAST—radio-allergo-absorbent test—maybe used to detect the presence of circulating immunoglobulins. B2 transferrin may be assayed in any persistent watery nasal discharge. This is a highly specific test for CSF which supersedes glucose and tau-protein testing. Several methods for detecting B2-transferrin have been developed (isoelectric focusing, staining with alkaline silver nitrate, radioimmunoassay, chromatofocusing and high resolution immunofixation). Nasal swabs may be required in suspected infections or if the patient requires admission (MRSA). Nasal brushings may be sent for electron microscopy to test for ciliary disease. Inflammatory markers, c-ANCA, ACE, ANA may be requested if vasculitis is suspected.

33.15.2 Clinical Tests

Allergy testing (patch testing) on the skin is normally performed by dermatologists, but is a useful test for suspected allergic rhinitis. Air flow measurements can be taken through a modified peak flow meter. Acoustic rhinometry uses sound waves to give an anatomic, not functional, description of the nasal passage. This tool is used

for research but not for routine evaluation in functional cases. Tests for anosmia are unlikely to be undertaken in the emergency situation. Ask the person to close their eyes and block one nostril with cotton wool. With an alcohol wipe a few centimetres from their nose, see if the patient can detect the odour. More sophisticated smell kits are available if more comprehensive assessment is required. Other tests include nasal nitric oxide, high speed videomicroscopy (HSVM) and transmission electron microscopy (TEM).

33.15.3 Saccharine Transit Time

Testing nasal cilial function is a highly specialised clinical test. Normal cilia pass mucus from the front to the back of the nose, where it is usually swallowed. Some diseases such as the common cold can affect cilia function leading to stasis of mucus. Primary ciliary dyskinesias are a group of conditions where the cilia fail to move in a coordinated fashion leading to mucus stasis. If primary cilial dyskinesis is suspected then a saccharine transit time can be measured. This can be done by placing a small fragment of saccharine on the anterior end of the inferior turbinate and timing how long it takes the patient to taste it. If the patient tastes the sweetness ciliary dyskinesia is unlikely.

33.15.4 Imaging

Plain films are rarely required, although plain facial views may be taken to examine the surrounding structures (e.g. zygoma, orbit, maxillary sinus fractures). In the UK and many other countries plain x-rays of the nose are no longer taken. These used to be requested for medicolegal purposes, often following alleged assaults. However this practice has now largely been abandoned. Today, if imaging is required following trauma or for some other indication, CT is usually now the first choice. This is best used to visualise the deep bony structures, such as the septum, nasa-ethmoid region an orbital walls. Indications include suspected complex trauma, including skull base fractures, investigation of CSF rhinorrhoea, suspected tumours and to evaluate the sinuses. These are discussed elsewhere in this book. MRI may also be required to evaluate similar pathologies (although generally not in trauma).