The Nose and Associated Structures: Part II

34

Nida Ilahi and Michael Perry

34.1 Nasal Congestion and Obstruction

Nasal congestion is a common problem and rarely occurs as a result of serious pathology. It arises when the mucous membranes of the nasal cavity becoming inflamed and engourged with dilated blood vessels and oedema. This may be accentuated by the nasal cycle, such that one side of the nose becomes completely obstructed, which is later relieved, alternating with the other side. Patients usually present with nasal blockage, obstruction, or a stuffy nose involving one or both sides. Nasal congestion can also interfere with hearing and speech. If obstruction is significant it can interfere with sleep, resulting in snoring and sometimes sleep apnea (although its precise role in this is still being investigated). The age of the patient is important. Newborn infants tends to breathe through the nose (termed 'obligate nasal breathing'). Nasal congestion in an infant in the first few months of life can therefore interfere with breastfeeding and occasionally result in respiratory distress. In children enlarged adenoids can cause chronic sleep apnea, which if left untreated can result in chronic hypoxia and right-sided heart failure. This usually resolves after removal of the adenoids and tonsils. Nasal congestion has many causes. These include

1. Allergies (hay fever/medication)
2. Upper respiratory tract infections ('colds and flu')
3. Rhinitis medicamentosa
4. Sinusitis
5. Pregnancy

N. Ilahi (✉)
Queen Elizabeth the Queen Mother Hospital, East Kent Hospitals University Trust, Kent, UK

M. Perry
London Northwest University Hospital, Harrow, Middlesex, UK

M. Perry (ed.), *Diseases and Injuries to the Head, Face and Neck*,
https://doi.org/10.1007/978-3-030-53099-0_34

6. Empty nose syndrome
7. Gastroesophageal reflux disease

Nasal obstruction can occur as a result of either congestion in an otherwise normal nose, or as a result of an 'anatomical' cause. In addition to the above, causes of obstruction include

1. Deviation of the nose or nasal septum (usually with a history of trauma)
2. Narrow nasal vault, or collapse of the internal nasal valve
3. Nasal polyps and tumours
4. Concha bullosa
5. Foreign bodies and infestations

It is important to remember that diseases involving the sinuses or upper teeth can also affect the nose, notably sinus infections and tumours. In some cases it may not be possible to determine whether sinus disease has extended into the nose or vice versa. However this is not important during the first stages of assessment. Diagnosis can therefore be difficult. Sometimes assessment may suggest that the nasal airway is patent, but this may differ from the patient's views. Not only is nasal obstruction a subjective sensation, difficult to quantify, but the precise cause may be difficult to diagnose. In many patients multiple causes may play a role. For example the patient could have an allergic response in a nose with polyps and pre-existing septal deviation. Treatment may therefore be complicated, requiring treatment of one or possibly all contributing factors. For this reason nasoendoscopy and CT are playing and increasing role in evaluation.

Treatment of nasal congestion and obstruction depends on the underlying cause. Decongestants may improve symptoms with inflamed and oedematous mucosa and are therefore most effective in providing symptomatic relief in conditions such as allergies or viral infections. These work in part by constricting blood vessels in the mucosa, generally applied as a nasal spray. However, topical decongestants should only be used for a maximum of 10 days. This is because on cessation a rebound congestion can occur (rhinitis medicamentosa). This then causes the patient to seek further medication which, whilst effective when used, results in a progressive worsening of the rebound phenomenon following cessation. Patients can then become completely dependent upon these drugs to provide symptom relief. Alternatively, inhalers and oral decongestants are also available. These do not have the same problems as vasoconstrictors, as they work through a different mechanism. Avoiding allergens, antihistamines and excessive use of decongestants can also provide symptom relief although this does not cure the underlaying problem. Antihistamines may be required continuously during the pollen season.

If a distressed infant is unable to breathe because of a plugged nose, urgent referral to an appropriate specialist is required. Fine nasal suction may be useful to remove the mucus. The mucus might be thick and sticky, making it difficult to expel from the nostril.

Surgical management of nasal obstruction is tailored to the underlying cause (polypectomy, septoplasty, diathermy of turbinates etc). These are discussed in the relevant sections.

34.2 Rhinitis

Rhinitis is the general term used to describe inflammation of the nasal mucosa. This is often seen in conjunction with inflammation of the paranasal sinus mucosa, resulting in the condition "rhinosiniusitis". This term is non-specific, referring to a range of disorders which result in inflammation of the 'sinonasal' mucosa. Common causes include upper respiratory tract viral infections and allergic rhinitis. Whilst most upper respiratory tract infections are self-limiting, bacterial or even fungal superinfection can sometimes develop. Progression of the acute illness to a chronic disease is also common. Bacterial and viral rhinosinusitis are difficult to differentiate on clinical grounds. Nevertheless bacterial rhinosinusitis must be identified and treated with antibiotics to prevent serious complications.

34.2.1 The Common Cold

This is a self-limiting condition caused by a virus, most commonly the Rhinovirus. However over 200 virus strains have been identified (including human coronavirus, influenza viruses, adenoviruses, human respiratory syncytial virus, enteroviruses and human parainfluenza viruses). This is why an effective vaccine is difficult to manufacture. This is spread via airborne droplet transmission and contact with contaminated objects may allow transmission to the mouth or nose. Some viruses may survive for prolonged periods in the environment (over 18 hours for rhinoviruses). The throat and sinuses are also commonly affected. Signs and symptoms are mostly due to the body's immune response to the virus and usually begin a few days following exposure. These include coughing, sore throat, runny nose, nasal congestion, sneezing, headache, muscle aches, fatigue and fever. People usually recover within 7 to 10 days, although some symptoms may persist for a few weeks. In patients with chest disease/immunosuppression pneumonia may develop. Treatment is generally supportive as no cure for the common cold exists. Nonsteroidal anti-inflammatory drugs (NSAIDs) may help with discomfort. There is some evidence to support the common belief that vitamin C and echinacea (a group of herbaceous flowering plants in the daisy family) may reduce the severity and duration of the condition. However in otherwise healthy patients it is normally treated with steam inhalations and over the counter analgesics and nasal decongestants. Antibiotics should not be prescribed. Some viruses have a seasonal pattern, depending on the virus.

34.2.2 Acute Rhinosinusitis

The sinuses are air-filled cavities within the skull. Normal ciliary function, intact mucous membranes and normal mucous production are required for sinus clearance. Many factors can disturb these mechanisms, predisposing the sinus to infection and inflammation. Acute rhinosinusitis commonly occurs when there is a failure to maintain sinus clearance. Inflammation results in the blockage of the sinus drainage pathways, especially at the ostiomeatal complex in the lateral wall of the nose,

which is particularly vulnerable to swelling and obstruction. This in turn results in mucus retention within the sinus, decreased mucociliary clearance and bacterial growth. Allergy is an important predisposing factor. This can contribute to inflammation and infection through either nasal congestion and subsequent ostial obstruction, or by its direct effects on mucosal cells. Other factors that predispose to acute rhinosinusitis include nasal polyps, deviation of the nasal septum and dental procedures (on those teeth whose roots are very close to the sinus lining). Air pollution, cigarette smoking and overuse of topical decongestants also impair ciliary function.

Acute rhinosinusitis usually begins following contraction of an upper respiratory tract infection. Some viruses, such as influenza virus, produce significant mucosal damage. Others stimulate the local production of cytokines and other inflammatory mediators. Infection results in the loss of ciliated cells and reduced mucociliary flow. Although the true incidence is unknown, it has been estimated that up to 5% of viral infections lead to sinusitis. Rhinovirus is the most common virus causing the 'common cold' and rhinosinusitis in all age groups. Symptoms can last up to 4 weeks, after which they resolve completely. Whilst the diagnosis is often relatively straightforward in most patients, with recurrent and chronic rhinosinusitis symptoms imaging is important to look for complications and to exclude fungal infection and neoplasm.

In children, viral upper respiratory tract infections together with pharyngeal colonisation with group A streptococci predispose to acute bacterial rhinosinusitis. In approximately 10% of cases, direct inoculation of bacteria into the sinus is believed to occur. Streptococcus pneumoniae and Haemophilus influenzae are the most common pathogens in adults. Staphylococcus aureus has also been reported to be a pathogen in about 10%. In children, Streptococcus pneumoniae, Haemophilus influenzae and Morraxella catarrhalis are predominant. Acute bacterial rhinosinusitis is characterised by

1. Nasal congestion and discharge
2. Purulent rhinorrheoa
3. Postnasal drip
4. Facial pain, which may be referred to the ears or teeth. There may be a feeling of pain, pressure or fullness in the nose, eyes or ears. Toothache may occur (pain referred to the upper molar teeth)
5. Headaches
6. Decreased sense of smell
7. Halitosis

Symptoms usually last for 7–14 days but should not last more than 4 weeks. Differentiation between bacterial and viral sinusitis is based mostly on the signs and symptoms, but this can be difficult. Most diagnoses are based on the history and examination. Anterior rhinoscopy and nasal endoscopy may also be undertaken to look for enlarged turbinates, pus, congested mucosa and nasal polyps. Imaging may also be indicated. Today, plain films are limited to cases where CT is not available. They are not diagnostic, but may suggest opaque sinuses and gas-fluid level.

Computed tomography (CT) is regarded by many as the "gold standard". It defines the paranasal sinus anatomy and any soft the tissue masses. It also helps plans functional endoscopic sinus surgery (FESS) in the management of chronic disease. Magnetic resonance imaging (MRI) is complementary to CT, when soft tissue masses have been identified. In such cases MRI may be required to rule out or identify fungal infection and tumours. Ultrasonography has been reported, but its role in rhinosinusitis is limited. Some specialists now recommend obtaining microbiological samples following sinus puncture, or swabs of the posterior nasal cavity and nasopharyngeal aspirates. These may be required in patients who experience symptoms for more than 7 days, or those who have purulent discharge or fever.

Many of the symptoms of rhinosinusitis can be managed using over-the-counter medications (acetaminophen or ibuprofen), decongestants (pseudoephedrine), nasal irrigation and topical steroids (prescribed with care). Nasal decongestants are commonly used to help reduce inflammation in the nasal passages. Since many decongestants are now available over-the-counter many patients self-medicate before seeing their doctor. This can be a problem as some nasal decongestants, (such as oxymetazoline), can result in rebound congestion and addiction. Allergic rhinosinusitis is often treated with antihistamines like Claritin, Zyrtec, or Allegra. Rhinosinusitis usually requires multiple courses of antibiotics and on occasion surgical procedures. Unfortunately, increasing antimicrobial resistance has made management of bacterial rhinosinusitis more difficult. As a result some infections may be difficult to completely eradicate and can persist, resulting in the development of chronic rhinosinusitis (CRS).

34.3 Chronic Rhinosinusitis (CRS)

Chronic rhinosinusitis (CRS) is a common problem, affecting about 15% of the western population. Its diagnosis requires 2 of the following symptoms to be present for at least 12 consecutive weeks

1. Anterior and/or posterior mucopurulent discharge
2. Nasal obstruction and
3. Hyposmia or anosmia

There should also be objective evidence of sinonasal inflammation (seen on endoscopy or CT). Fever is not typically present. Nasal polyposis is thought to be a subgroup of CRS.

Chronic rhinosinusitis is usually associated with some predisposing conditions such as asthma, allergy, dental disease, cystic fibrosis, polyposis and immunodeficiency. Rarer associations include Wegener's granulomatosis, Churg-Strauss syndrome and sarcoidosis. Leprosy (see below) can also infect the mucous membranes of the respiratory tract and manifest with nasal symptoms of chronic rhinitis, including nasal congestion, rhinorrhea, and intermittent epistaxis. Medications, such as aspirin, ibuprofen and beta blockers have been implicated in other cases. Chronic

rhinosinusitis is therefore best thought of as a multi-factorial disease, involving multiple host and environmental factors. Extrinsic factors include viral, bacterial, and/or fungal colonisation (with production of biofilms, super-antigens and non-IgE mediated eosinophilic inflammation), and exposure to inhaled substances (such as cigarette smoke or allergens). Intrinsic factors include anatomical variations which may affect sinus drainage and genetic abnormalities (notably cystic fibrosis and disorders of the immune system). Chronic rhinosinusitis is currently thought to be an immunologically based inflammatory disease. Two pathological mechanisms have been suggested.

1. The 'super-antigen hypothesis' This proposes that bacterial toxins within the nose stimulate massive oligoclonal expansion of T-cells resulting in eosinophil recruitment and tissue inflammation.
2. Bacterial biofilms. These are a complex organised collections of bacteria that adhere to the mucosal surface and produce an extensive extracellular coating (the glycocalyx). This serves as protection for the bacteria. When the biofilms reach a critical mass, bacteria detach and form new foci of infection and further biofilm growth. Newer non-antimicrobial therapies are currently being developed to destabilise these biofilms.

The most common symptoms in CRS are nasal congestion and obstruction. These are usually accompanied by nasal discharge (anterior or posterior), a reduction or loss in the sense of smell, severe facial pain and headaches. These symptoms can impact daily activities, including sleep. Sleep impairment is thought to be secondary to nasal congestion and obstruction which is made worse by the lower position of the patient's head plus the overnight decline of serum cortisol levels. Other symptoms include sneezing, post nasal drip, nasal itching, nocturnal or chronic cough and lacrimation. Psychological disorders (changes in mood, depression, and anxiety), fatigue and sexual dysfunction have also been reported. Patients with olfactory impairment can have problems preparing food, decreased appetite, and be unable to detect hazards such as smoke, chemicals, and gas leaks. This can severely affect some professions (chefs, fireman, plumbers, etc).

It is also important to enquire about respiratory symptoms, such as cough, wheeze, and shortness of breath. Any history of recurrent infections (skin, urinary tract or digestive tract) may indicate immunodeficiency. Connective tissue disorders (granulomatous diseases and vasculitis), medications (oral immunosuppressive agents) and allergies (exposure to smoke, animals or mould) should all be considered. A complete ear, nose, and throat examination should be undertaken, noting the mucosal appearance, any enlargement of the inferior turbinates and the presence of nasal crusting. Endoscopy will help identify active infection or obstruction to the sinus ostium. Polyps, pus, stenosis and middle turbinate anatomy should be noted and if necessary biopsies taken to rule out systemic diseases or tumours. Thin cut CT scan should also be requested to detail the bony/sinus anatomy.

Management aims to reduce mucosal oedema, improve sinus drainage, treat any associated infections and remove any predisposing causes. This often requires a

combination of topical or oral glucocorticoids, antibiotics and nasal saline irrigation. If these measures fail, the patient should be referred to an otolaryngologist for consideration of sinus surgery. Severe infections, immunosuppressed patients and children with chronic sinusitis may need to be admitted, depending on the severity of their symptoms. Current recommendations in management include

1. Confirm the diagnosis of CRS with objective evidence of sinonasal inflammation via anterior rhinoscopy, nasal endoscopy, or CT scan
2. Distinguish CRS and recurrent acute rhinosinusitis from isolated episodes of acute bacterial rhinosinusitis and other causes of sinonasal symptoms
3. Screen for and treat any underlying predisposing conditions (smoking, allergies, asthma, cystic fibrosis, immunodeficiency, ciliary dyskinesia, Gastroesophageal reflux disease)
4. Exclude nasal polyps
5. Symptoms may be relieved with topical decongestants, antihistamines, topical steroids, antibiotics, nasal saline, steam inhalations or nebulized saline spray and mucolytics (surfactants).
6. Antibiotics may occasionally be indicated. Some patients find that amoxicillin with potassium clavulanate helps. However, do not prescribe antifungal therapy in patients with CRS

Saline provides mechanical cleansing of the sinuses. It is cheap, simple and can be easily administered at home when symptoms are bad. Intranasal steroids are most effective in allergic rhinitis. They can also reduce the size of nasal polyps ("medical polypectomy"). Side-effects are usually minimal. Ideally the choice of antibiotic should be based on the culture of sinus secretions. Penicillins, cephalosporins, or macrocodes are all effective, although it is always best to follow local policy or discuss with your local microbiologists. Intranasally administered surfactants interfere with the adherence of the biofilm layer to the underlying sinus epithelium. Destabilisation of bacterial membranes and leaking of electrolytes has also been suggested. Interestingly, honey has been proposed as a promising local agent, due to its safety profile and low cost. Most decongestants are sympathomimetic agonists resulting in vasoconstriction and relief of congestion. Both topical and systemic antihistamines are effective, the former having reduced side effects.

34.3.1 Klebsiella Rhinoscleromatis

This is a gram-negative coccobacillus which is endemic to tropical and subtropical areas of Africa, America, and Asia. It causes a chronic granulomatous disease of the upper respiratory tract. The nose is almost always involved. An initial catarrhal stage presents with a purulent rhinorrhea, crusting and nasal obstruction. This is followed by a granulomatous stage, in which multiple nodules form and coalesce to form large blue-red or pale granulomas. In severe cases this can progress resulting in local destruction and nasal deformity. Broadening of the nose produces the

characteristic "Hebra nose". A final fibrotic stage results in scarring. Less commonly, the larynx and trachea can be affected. Stenosis at these sites can cause significant airway obstruction. Diagnosis is made on examination and biopsy. Treatment is with prolonged antibiotics (tetracycline, ciprofloxacin, streptomycin). Surgery may be required.

34.3.2 Mycobacterium Leprae

Leprosy is caused by a gram-positive, acid-fast bacillus. The nose is by far the most frequently involved site in the upper respiratory tract. Lesions begin as pale, nodular, plaque-like thickenings of the mucosa. These progress to ulceration, septal perforation and the development of a saddle-nose deformity. The larynx can also be involved. Diagnosis is made following biopsy. Rifampin, dapsone, and clofazimine are the most widely used drugs in treatment.

34.3.3 Allergic Rhinitis

Two types of allergic rhinitis are described—seasonal (hay fever) and perennial (allergies to pets, mould and dust mites). Both are due to hypersensitivity of the nasal mucosa, resulting in rhinorrheoa, sneezing, lacrimation and nasal congestion. Seasonal allergic rhinitis occurs at a specific time of the year when pollen counts are high. Food allergies can result in persistent symptoms. Most common food allergens include wheat, dairy, soy, corn, and eggs. Treatment generally involves avoidance of the allergen (sunglasses, avoiding going out before midday, regular showers), nasal steroid/anti-histamines sprays, systemic antihistamines and sodium cromoglicate eye drops. Oral steroids are very effective, but carry the risk of side effects if used long-term. Intranasal Ipratropium bromide may be prescribed to alleviate watery nasal discharge. Allergy testing may help identify the allergens responsible and help with avoidance.

34.3.4 Vasomotor Rhinitis

This is rhinitis which occurs in the absence of any known allergens. It is sometimes referred to as nonallergic rhinitis or nonallergic rhinopathy and is essentially a diagnosis of exclusion. The exact cause is unknown, but it can be sporadic or perennial. Symptoms are triggered by something that irritates the nose, such as:

1. A dry atmosphere
2. Air pollution
3. Alcohol
4. Certain medicines
5. Spicy foods

6. Strong odors, such as perfumes, cleaning products (especially bleach) among others

Vasomotor rhinitis is characterised by prominent symptoms of nasal obstruction, rhinorrhea, and congestion. These may vary, from a few weeks to being constant. The vasomotor effects of emotion and sexual arousal may be caused by autonomic stimulation or dysfunction. Possible compounding factors included previous nasal trauma and extraesophageal manifestations of gastroesophageal reflux disease. Management is the same way as allergic rhinitis. A step wise approach using Topical antihistamines, Topical corticosteroids, Topical cromoglycate, or their oral counterparts is frequently used.

34.3.5 Rhinitis Medicamentosa (RM)

This is rebound nasal congestion which occurs following cessation of prolonged use of decongestants. This is secondary to vasodilatation of the nasal mucosa. Chronic or repeated use of decongestants can result in atrophic rhinitis, turbinate hyperplasia, headaches, sleep disturbances, irritability and anxiety. Patients may develop nasal congestion, postnasal drip or sneezing following prolonged use of topical decongestants. Increased usage of the drug worsens the problem. In severe cases, chronic congestion results in permanent turbinate hyperplasia, which blocks nasal breathing. This may require surgery. Treatment of RM involves cessation of the offending nasal spray or oral medication, either by weaning or going "cold turkey". Saline nasal sprays may help relieve some symptoms without causing RM. Corticosteroid nasal sprays may also be required.

34.3.6 Atrophic Rhinitis

This is a chronic condition of unknown aetiology. It is commonly seen in young adults from asian / middle eastern/mediterranean countries. Formation of thick, dry crusts within the nasal cavity occurs, together with atrophy of the nasal mucosa (which is replaced by non-ciliated epithelium). This is sometimes seen following severe nasal trauma or surgery. Symptoms include a paradoxical feeling of nasal congestion, foul- smelling discharge, epistaxis, nasal obstruction, anosmia and cacosmia. Secondary infections can occur, resulting in infestations, pharyngitis and otitis media. Treatment usually involves regular nasal irrigation, antibiotics and vasodilators.

34.3.7 Rare Causes of Rhinitis

1. Some drugs have been reported to induce chronic symptoms. These include beta blockers, oral contraceptives, aspirin, NSAIDs and local decongestants

2. Systemic conditions. These may affect the ability of the sinus to clear mucus. Examples include primary mucous defect, cystic fibrosis, Young's disease, Primary clinically dyskinesis, Kartagener's syndormes, SLE, Rheumatoid arthritis, AIDS, Hypothyroidism and Pregnancy. The severity of the rhinosinusitis can vary widely.
3. Granulomatous diseases and vasculitis. Symptoms and signs may be similar to chronic rhinosinusitis and therefore diagnosis may be delayed. The list of granulomatous diseases that can affect the sinonasal tract is extensive. Sarcoidosis, Wegener's granulomatosis, Churg– Strauss syndrome are the most common. Diagnosis requires a detailed history (noting any bloody rhinorrheoa, fatigue, pulmonary symptoms, night sweating, weight loss and fever), systemic examination and biopsy of any abnormal looking mucosa (polyps, ulcers and nodules). Septal perforation, mucocele, orbital pseudotumor, or saddle-nose deformity should all be viewed with suspicion (Wegener's granulomatosis). Treatment of nasal granulomatous diseases involves sinus debridement, saline rinses and topical or systemic steroids and immunosuppressive treatment. Early referral to a rheumatology or immunology specialist is important.
4. Gastroesophageal reflux disease (GERD or GORD). This has been implicated as a contributing factor in many airway related symptoms, including dysphonia, benign vocal cord lesions, vocal laryngospasm, subglottic stenosis, asthma, CRS, post nasal drip and idiopathic cough. Direct exposure of the nasopharynx and nose to gastric acid and Helicobacter pylori has been suggested to results in local irritation and low-grade inflammation.

34.3.8 Surgical Management

Not all patients with sinus problems require surgery. However chronic sinus disease can result in permanent changes within the nose and sinuses that may require surgical intervention. Turbinate reduction (radical turbinectomy, submucous turbinectomy, submucous electrocautery, and microdebriber turbinoplasty) may be indicated if the turbinates are hypertrophic. Surgical, laser and cryotherapy directed treatments have been described. Although no single modality is regarded as the gold standard for the treatment of allergic rhinitis, the mainstay of surgical intervention targets the inferior turbinate. Septal surgery may be required if the septum is deviated. FESS (Functional Endoscopic Sinus Surgery) aims to remove any blockage of the osteomeatal complex and restore drainage of the sinuses. Nasal polyps may need to be removed. Vidian neurectomy may be indicated in the treatment of vasomotor rhinitis. This nerve is formed by post synaptic parasympathetic fibers and presynaptic sympathetic fibers. It is also known as the "nerve of pterygoid canal". The nerve exits its canal in the pterygopalatine fossa and joins the pterygopalatine ganglion. Ultimately it innervates the nasal mucosa.

34.4 Sinusitis

The clinical features and complications of the various types of sinusitis are also discussed in the chapters on the upper jaw and cheek. The ethmoid sinuses develop and expand in early childhood. They are paired and divided into multiple communicating chambers or 'air cells' (posterior and anterior). Ethmoid sinusitis rarely occurs in isolation. It often results from an extension from maxillary sinusitis. Patients complain of a deep-seated throbbing pain, deep to the bridge of the nose, between the eyes. Because the adjacent medial orbital walls are paper thin and often fenestrated, orbital cellulitis can quickly develop if this is not treated. Infection can also ascend into the frontal sinus, via the frontal sinus drainage pathways (FSDP). Chronic sinus disease can predispose to polyps, dacryocysitis and nasolacrimal duct obstruction. Clinical features include:

1. Headache
2. Facial pain
3. Sensation of dull, constant pressure over affected sinus
4. Symptoms worse on bending over, straining or lying down
5. Nasal discharge
6. Halitosis
7. Post-nasal drip

Initial investigations include CT scan, to define the extent of the infection and determine whether drainage is required. CT will also help with surgical planning (whether to limit the surgery to the anterior ethmoidal air cells or undertaken complete sphenoethmoidectomy). In the first instance medical management is commenced to reduce oedema, inflammation, alleviate pain and restore the normal mucociliary secretions. This includes

- Antibiotics
- Ephedrine nasal drops
- Menthol inhalations may reduce congestion and improve sinus drainage
- Steroid nasal drops

Some patients may need admission for intravenous antibiotics. Surgical options are considered if medical management fails, or if the patient develops rapidly progressing sinusitis, abscess formation (orbital or cerebral), or life threatening sepsis. Surgery may involve

- Sinus washout with opening of the draining channels (functional endoscopic sinus surgery)
- middle metal antrostomy plus anterior ethmoidectomy.
- Stent insertion in chronic ethmoiditis.

The main concerns with ethmoid sinusitis is the spread of sinusitis into the orbit (orbital subperiosteal abscess, and orbital cellulitis) and cavernous sinus thrombosis. These are discussed elsewhere in this book. Meningitis, thrombophlebitis of the superior sagittal sinus and cerebral abscess have also been reported. Bony involvement such as ostetits and osteomyelitis have also been documented but are rare. Chronic infections can result in mucocoele development.

34.5 Nasal Polyps

Nasal polyps are non-cancerous, fleshy swellings that arise from the nasal and sinus lining as a result of chronic inflammation. They are commonly associated with rhinosinusitis, which causes a reactive hyperplasia in the mucous membrane and polyp formation. Polyps can affect up to 4% of the population and are uncommon in children under 10 years. Childhood polyps are usually associated with conditions affecting mucociliary function such as cystic fibrosis or primary ciliary dyskinesia. In adults, asthmatics and aspirin intolerance are also commonly associated. Aspirin hypersensitivity is not a true allergic reaction but rather an alteration in prostaglandin production. Polyps tend to repeatedly recur in these groups. Diseases commonly associated with polyp formation include

1. Chronic rhinosinusitis
2. Asthma
3. Aspirin induced, asthma, or aspirin-exacerbated respiratory disease (AERD)
4. Cystic fibrosis
5. Allergic fungal sinusitis
6. Kartagener's syndrome
7. Young's syndrome
8. Eosinophilic granulomatosis with polyangiitis
9. Nasal mastocytosis

Polyps usually originate in the ethmoid sinuses and may contain inflammatory fluid. As they enlarge they become fleshy, oedematous and eventually prolapse into the nasal cavity where they may be seen in the anterior nose and confused with the turbinates. The polyp appears as a pale, mobile and insensate mass. Patients may be asymptomatic or they may experience rhinorrhoea, sneezing, sinusitis, loss of smell, post nasal drip, or nasal obstruction. Normally polyps occur bilaterally. Therefore unilateral polyps should be treated with suspicion (antrochoanal polyps or malignancy). Antrochoanal polyps present unilaterally as a large peduncualted mass in the nasopharynx. They originate from the maxillary sinus. Treatment is surgical removal. Polyps in adults are usually treated initially with topical steriods e.g. betamethasone drops. 80% of patents will usually respond well. Antihistamines may help with symptoms but do not affect the underlying disease. Oral steroids may be required for larger polyps. Antibiotics are usually not required. Surgical removal

(polypectomy) may be required if patients have severe symptoms or breathing difficulties.

Rare nasal polyps include:

1. Samter's triad associated polyp—A defect in leukotriene metabolism resulting in the combination of aspirin- accosted wheeze, late onset asthma and nasal polyps which re florid and recur frequently. Treatment included avoiding salicylates, intranasal steroids, leukotriene antagonists and then polypectomy
2. Antrochoanal polyps—these often present unilaterally as a large peduncualted mass in the nasopharynx. They originate from the maxillary antrum and appear similar to nasal polyps with a fluid filled cyst within. Treatment is by surgical removal.
3. Childhood polyps—these are usually associated with conditions affecting mucociliary function such as cystic fibrosis or primary ciliary dyskinesia. Investigations should therefore include sweat test for cystic fibrosis diagnosis, biopsy and sampling of the nasal lining to check ciliary structure and function. Treatment is with steroids or surgical removal.

34.6 Foreign Bodies

This is a common problem in children who seem to enjoy putting small objects up their nose or in their ears. The range of objects is huge, typically including rubber erasers, paper wads, pebbles, beads, marbles, beans, washers, nuts and chalk. These can be found in any portion of the nasal cavity, but are usually seen along the floor of the nose just below the inferior turbinate. Another common location is immediately anterior to the middle turbinate. In some reports, foreign bodies in the nose have been implicated as carriers of diphtheria and other infectious diseases.

Some foreign bodies are occasionally inert and may remain in the nose for years without causing symptoms. However, most will irritate the nasal lining resulting in congestion and swelling, with the possibility of pressure necrosis, ulceration, mucosal erosion, and epistaxis. Any retained secretions, decomposed foreign body, and mucosal ulceration can then result in a foul smell. Most foreign bodies are generally painless. Patients thus commonly present with unilateral foul smelling discharge, unilateral nasal obstruction and vestibulitis. Vegetable matter will absorb water and can swell and evoke an intense inflammatory reaction. Occasionally, this can be severe enough to produce toxaemia. Small batteries require urgent removal. These can result in severe an intense local tissue reaction, septal perforations, synechiae and stenosis of the nasal cavity.

If a foreign body remains unrecognised for a long time, a calciferous deposit can form over it, resulting in a rhinolith—a foetid, stony mass. Rarely, if large enough these can cause erosion of the lateral wall and floor of the nose. Loose foreign bodies in the postnasal space can accidentally be aspirated or pushed back during attempted removal and may result in acute respiratory obstruction.

Depending on the degree of cooperation of the patient and the shape of the object, removal can be relatively simple, or very difficult. It is generally best to avoid forceps, as this can push objects further into the nose. The nose is carefully inspected to identify the object if possible. Often the first attempt is most likely to be successful in young children, as repeated attempts not only cause further swelling and bleeding but can also reduce patient compliance. Multiple techniques have been described. These can be adapted depending on the circumstances.

1. Simple blowing of the nose. This may not have been tried and is a good first step. If the patient will cooperate, instructed them to take a deep breath through the mouth and then forcibly exhale through the nose. At the same time occlude the uninvolved nostril. Even if the object is not expelled, it may move towards to nostril, aiding retrieval.
2. Suction—A fine (Zoellner) suction catheter can be useful for sucking out secretions or light, flat foreign bodies. Yankuer suckers tend to be larger, making them more difficult to use in the nose.
3. Irrigation with saline may help lubrication to remove small objects
4. Wax hook/Jobson- Horne probes. These require expertise as they can push the object deeper. The hook is first passed behind the object, the tip rotated to rest just behind it and then the foreign body is gradually drawn forwards and out through the nose. Plastic objects and vegetable matter may be difficult to extract because of their tendency to break into small pieces.
5. Super-glue on the opposite end of the wooden stick of a long cotton bud is another effective method. However this requires a steady hand and cooperative patient. It is most effective in removing smooth, round objects that are often difficult to grasp.
6. 'Mummy's kiss'—With this technique the child is seated on their parents lap with the intention of getting a "big kiss'. This was first described by a New York general practitioner in the 1960s. The parent places their own lips over the child's ensuring a tight seal. The opposite patent nostril is then occluded by the parent and forceful exhalation is given into the child's mouth. This positive pressure may be enough to blow out the foreign body. Although this does carry the theoretical risk of barotrauma to airway, lungs or tympanic membranes as yet, there have been no reported cases.

If all else fails the child may need admission for general anaesthesia and removal of the foreign body. In rare cases, the only successful method of removing a nasal foreign body has been to push the object posteriorly into the pharynx. In these cases a general anaesthetic is required with endotracheal intubation and a throat pack. Whatever technique is used, once the FB has been retrieved, always remember to re-examine the nose to ensure there are no additional objects remaining.

34.6.1 Infestations

These are uncommon. Nasal myiasis is a parasitic infestation in which live larvae grow inside the nasal cavity, feeding on the tissues. It is common in warm tropical climates such as South Western United States or the Far East including India.

Infestation is primarily related to poor hygiene of the inhabitants. The most common parasite is the fly larvae, usually the Botfly. Screw worms, also known as "Texas" screw worms (which is the larval state of Cochliomya macellaria and the Cochliomyia homnivorax, "the blow fly") are serious pests to both humans and cattle. Multiple larvae can cause obstruction of the nasal passages and severe irritation and pain. The larva burrows into the living tissue and damages nasal mucosal lining. Larvae of other flies like those of aestrous, hypoderma and dermatobia can also invade the nasal cavities. Ascaris lumbricoides (a species of nematode or round intestinal worms) can also become lodged in the nose if regurgitated or coughed up.

Patients experience nasal discharge, epistaxis, a foul smell and a foreign body sensation within the nose. Sometimes the infestation is accompanied by facial oedema and unexplained fevers. Whilst rarely fatal, mortality has been reported. Associated boil-like lesions on exposed areas of the body especially the scalp, forearms and legs may also be present. These may be due to cutaneous infestation.

Although infestation is a rare condition, it is important to consider this differential diagnosis in patients who have travelled abroad or worked with animals. Myiasis is often misdiagnosed because it is rare and its symptoms are often non-specific. The Botfly is commonly found in hot and wet conditions, where livestock are kept, especially sheep, goats and cattle. Thus, consider this diagnosis in husbandry workers who present with unusual nasal symptoms. In the head and neck area the main sites of infestation are (i) nose, nasopharynx, sinuses or pharynx, (ii) in or about the eye and (iii) the ear. Investigations may include a full blood count which may reveal a leukocytosis and eosinophillia. MRI or CT may be required to establish the diagnosis, as neoplasia may be considered first.

Treatment of myiasis involves removal of the larvae directly or via nasoendoscopy. These can be 'plucked' using forceps under endoscopic assistance. The larvae are often anchored deeply to the subcutaneous tissue by hooklets and it is important to fully remove these, otherwise the will result in infection or induce a foreign body reaction. With screw worms, larvae and maggots, a weak solution of 25% chloroform can be instilled into the nasal spaces to kill the larvae. This may need to be repeated two or three times a week for several weeks until all larvae are killed. After each treatment, removal of the detached larvae is done by blowing the nose, suction, irrigation, or curettage. With Ascaris lumbricoides forceps extraction is usually required. Oral levamisole or mebendazole is also required to treat the intestinal infestation. With all infestations, antibiotics and decongestants are usually prescribed following removal. Close liaison with microbiology and tropical medicine departments is advised. As with every contaminated wound, tetanus status must be enquired upon and treated as necessary.

Myiasis varies widely in its types and effects on the patient. These depend on the species of fly and where the larvae are located. Some flies lay eggs in open wounds. The hatched larvae of other species may invade unbroken skin or enter the body through the nose or ears. The stomach and intestinal tract, exposed skin and genitourinary tract can also be infested. Nosocomial myiasis is myiasis acquired in a hospital setting. Patients with open wounds and sores can be infested if flies are present. Three main fly families cause myiasis in humans—(i) Calliphoridae (blowflies), (ii) Oestridae (botflies) and (iii) Sarcophagidae (fleshflies).

34.7 Empty Nose Syndrome

Empty nose syndrome (ENS) is an unusual clinical syndrome in which patients have clear nasal passages, yet experience a range of symptoms. It is believed to be a form of atrophic rhinitis, which is most commonly associated following excessive removal of the turbinates. Little is known about the pathognesis of empty nose syndrome, other than the anatomical changes result in alterations in the local environment and inspired airflow, thereby affecting mucosal and neurosensory mechanisms. Loss of the normal humidifying structures results in disruption of normal nasal physiology (warming and humidification). This results in chronic nasal dryness, chronic inflammation, atrophy and pain. Symptoms can occur weeks or years following turbinate surgery.

Diagnosis is often clinical, although it can be difficult to make due to the poor correlation between the patient's symptoms and objective findings. Patients often present with painful dryness of the nose. Loss of smell is also commonly described. Paradoxically patients may describe a sensation of being unable to breathe which then leads to difficulty sleeping and fatigue. Foul smells inside the nose may occur secondary to infections. On examination the nose looks abnormally spacious, lacking turbinates and the mucosa appears dry, pale or inflamed. Crusting is often present. No specific investigations exist although the 'cotton test' has been suggested—a moist piece of cotton is held in the position of the turbinate and any relief of symptoms noted. CT scans show an empty nasal space. Treatment includes mucosal humidification, irrigation the nose and use of emoillents Surgical treatment involves narrowing the over enlarged nasal cavity using various implants under the mucosa. In severe cases Young's procedure may be performed. Complete closure of the nasal cavity is undertaken with local mucocutaneous flaps. This is kept closed for 6 months. If the mucosa subsequently looks to have recovered the cavity is surgically reopened.

34.8 The Septum: Deformity and Destructive Conditions

34.8.1 Septal Perforation

The nasal septum separates the nasal cavity into the two channels. It is composed of the perpendicular plate of the ethmoid posteriorly, the vomer and maxillary crest inferiorly and the quadrangular cartilage anteriorly. The septum is an important structure in maintaining nasal projection and the midline position of the nose. Normally, the septum lies roughly central, with the nasal passages approximately symmetrical. Perforation of the septum can result in a variably sized hole or fissure. This may arise following intentional trauma (such as nasal piercings), or from unintentional injury (long-term topical drugs—intranasal steroids, chronic decongestants, metamphetamines and cocaine abuse). Perforation can occur as a complication of high energy trauma, chronic nose picking, or nasal surgery (nasal cautery, nasogastric tubes, nasal intubation, septoplasty and rhinoplasty). There have also been

reports of nasal perforation associated with chronic exposure to acid fumes, emphasising the importance of an occupational history. Rare causes include granulomatous inflammatory conditions (Wegener's, Churg-Strass, Sarcoidosis), Lupus, Lymphoma, Squamous cell carcinoma, TB and Syphilis.

Since perforations vary in size and location, clinical features can vary. In some cases the perforation is asymptomatic and discovered purely by chance. Small perforations can result in a whistling noise when the patient breathes through their nose. Larger perforations can result in crusting, epistaxis, bloody discharge, difficulty breathing and discomfort. With huge defects the nose can collapse, resulting in a "saddle nose" deformity. Generally speaking, the closer the perforation is to the nostrils, the more likely it is to cause symptoms. Perforations can be classified according to size. Site and size can be determined by CT and endoscopic evaluation.

1. Small perforations—diameter less than 5 mm
2. Medium perforations—diameter between 5 mm to 20 mm
3. Large perforations—diameter more than 20 mm (Fig. 34.1)

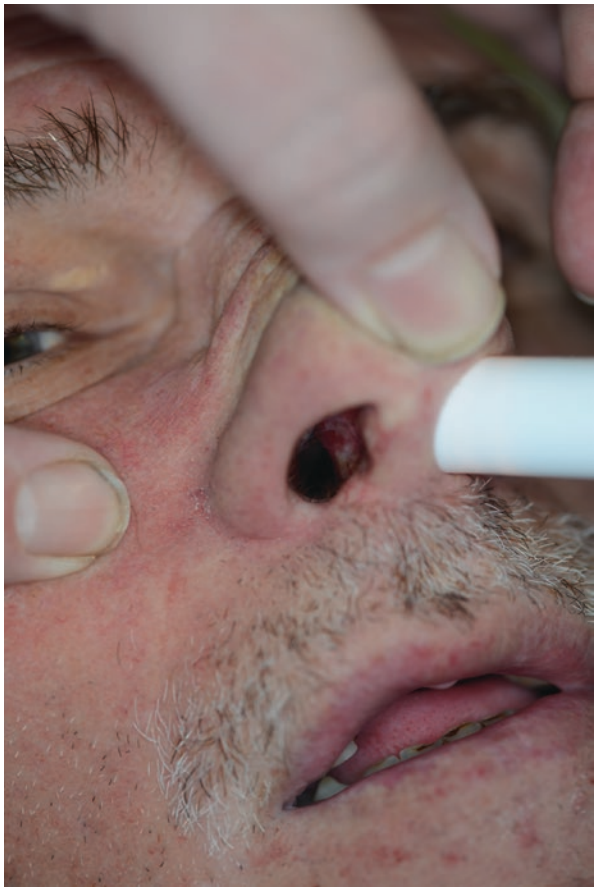

Fig. 34.1 Septal perforation

Treatment depends on the severity of symptoms and the size of the perforation. Any precipitating causes should be identified and stopped (nose picking, cocaine abuse etc). It is also important to consider more aggressive pathologies, notably the granulomatous diseases. These require specific treatments. Medical management aims to reduce symptoms, whereas surgical treatment with a prosthesis or allograft may be undertaken to repair the defect. Medical management includes nasal irrigants for crusting and humidification. Petroleum or topical antibiotic ointment may be used to control pain and dryness.

Posteriorly sited perforations and those that are asymptomatic may be managed with simple observation. With perforations that bleed or are painful, initial management includes humidification and application of protective creams to the edges of the perforation to allow healing. If successful the edges will become covered with septal mucosa. For persistent symptomatic perforations, a silicone septal button can be placed. Definitive closure may be possible in selected cases. Surgery is complicated and involves a number of different surgical approaches to the septum (sublabial, midfacial degloving, rhinoplasty). Several techniques are described in which aim to reconstruct the mucosal defect through the use of local flaps from the mucoperisoteum of the inferior turbinate. Interposition grafts may be used (such as temporalis fascia). Autologous free tissue transfer ('free flaps') have also been described, but this is quite extensive surgery.

34.8.2 Septal Deviation

Mild septal deviation is relatively common and in the absence of significant symptoms, requires no treatment. With more displaced septums the lower part of the nose may become deviated and the patient may experience breathing difficulties. In some patients deviation is a congenital anomaly (growth mismatch between the nose and the skull), but in most cases it arises following an injury (including birth trauma). Clinical features vary and include

1. Nasal deviation
2. Congestion and restricted airway. Patients may have increased awareness of nasal cycle, with intermitted obstruction of one side of the nose. Snoring may occur at night.
3. Headache and facial pain
4. Post nasal drip
5. Recurrent epistaxis
6. Recurrent sinusitis

Clinical examination usually reveals a deviated septum. This can be further evaluated endoscopically or with CT. If the patient's main concern is difficulty breathing Cottle's test should be performed to exclude internal nasal valve collapse. Acoustic rhinomanometry and computer flow manometry have been described to evaluate function problems related to the nasal valve, but these are rarely indicated in clinical practice (Fig. 34.2).

Fig. 34.2 The effects of septal collapse are analogous to camping mishaps with loss of the central tent support

If septal deviation is asymptomatic then no treatment is necessary. Otherwise, initial medical management includes a 3 month trial of intranasal steroid (to reduce nasal inflammation and improve drainage). This can take 1 to 3 weeks before any effect is noticeable. Antihistamines and decongestants may help reduce swelling and inflammation. Surgery may be indicated if obstruction is significant. Various procedures have been described, including scoring of the deformed cartilage, localised resection (submucous resection—SMR) and more aggressive reconstructions using cartilage taken from elsewhere (the pinna). At the same time other procedures may be required, such as osteotomies and repositioning of the nasal bones and diathermy or limited resection of the turbinates.

34.9 Relapsing Polychondritis

Relapsing polychondritis is a severe, painful, episodic and progressive inflammatory condition which can affect the cartilage of the nose and other cartilaginous areas of the body, including the ear, eyelids and larynx. Left untreated, the condition can cause major deformity. Systemic involvement of the respiratory tract, heart valves, peripheral joints or central nervous system, can become life-threatening.

The cause of this rare disease is unknown. However the pathogenesis appears to be autoimmune. Pathological findings include infiltrating T cells and antigen-antibody complexes against type 2 collagen. Nearly a third of patients develop systemic and cutaneous vasculitis, rheumatoid arthritis, systemic lupus erythematosus, Sjogren's syndrome, ulcerative colitis, Crohn's and Takayasu arteritis. Relapsing polycondritis has also been linked to an underlying haematological malignant process and paraneoplastic syndrome.

Patients can present widely, depending on which cartilage is involved. Involvement of the nose can result in a feeling of fullness across nasal bridge, collapse of the septum with saddle-shaped deformity, epistaxis and painful erythematous swelling. Diagnosis is often delayed due to the rarity of this condition and its unusual presentation. Blood tests may show a raised CRP or ESR but biopsy is usually required to make the diagnosis. Diagnostic criteria have been described. These include the presence of (i) bilateral auricular chondritis, (ii) non-erosive seronegative inflammatory polyarthiritis, (iii) nasal chondritis, (iv) ocular inflammation, (v) respiratory tract chondritis and (vi) audiovestibular damage.

The aim of treatment is to alleviate symptoms and preserve the cartilage. Often this is best managed with a multidisciplinary setting involving rheumatology and dermatology as well as OMFS, ENT, cardiologists, renal and neurologists, depending on the complexity of the case. Given the underlying autoimmune cause, treatment involves immunosuppressant (steroids, azathioprine, dapsone, methotrexate, cyclophosphamide and cyclosporin). There have also been case reports of successful treatment with anti-tumour necrosis factor alpha inhibitors, interleukin 1 receptor antagonist, and monoclonal antibodies. Surgery is only indicated in patients with secondary deformities of their cartilage e.g. saddle-nose deformity.

34.10 Wegener's Granulomatosis

Wegener's granulomatosis is characterised by granulomatous inflammation involving the respiratory tract and a necrotising systemic vasculitis affecting small to medium blood vessels. It can initially present as a glomerulonephritis, but the classical features are of vasculitis and granulomas involving the lungs, airway lining and the kidneys. It presents most commonly in middle age and is more common in Caucasians. In the nose it can present with crusting, epistaxis, erosions and perforation of nasal septum. Nasal ulceration and a bloody nasal discharge can occur. The nasal bridge can collapse resulting in a saddle nose deformity. Wegener's granulomatosis can also present with sinusitis, otitis media, hearing loss or oral lesions. Diagnosis can be difficult. Therefore this condition should always be considered in patients presenting with septal ulceration or perforation. Diagnosis is confirmed by detecting granulomas and vasculitic changes in a biopsy of involved tissue. There is a strong and specific association with autoantibodies directed against proteinase-3 (C-ANCA). Medications include high-dose steroids (prednisone) and immunosuppressive drugs (cyclophosphamide—Cytoxan). Recent reports also suggest that trimethoprim/sulfamethoxazole (Bactrim) can be helpful to prevent relapse.

Methotrexate has also been trialled. More recently, intravenous immunoglobulin therapy has been shown to be helpful in treating relapses of granulomatosis with polyangiitis. A relatively new treatment for granulomatosis with polyangiitis is rituximab (Rituxan).

34.11 Syphilis

Syphilis is a sexually transmitted infection caused by the spirochete Treponema palladium. It is rarely seen today. Undiagnosed it can pass through 4 stages, (i) primary, (ii) secondary, (iii) latent and (iv) tertiary. In the nose a primary chancre can present in the nasal passage, although this seems to be rare. Involvement is more common in the territory stages of the disease with gumma formation, ulceration and necrosis. This leads to stenosis and atresia of the vestibules, atrophic rhinitis, palatal and septal perforations. Secondary deformities can occur such as saddle nose deformity. Treatment in the early stages with penicillin.

34.12 Nose Bleeds (Epistaxis)

Nosebleeds are common but rarely fatal. It has been estimated that approximately 60% of people will have a nosebleed at some point in their life and only 10% of all nosebleeds are serious. Bleeding from the nose has many causes. Classically this is described as arising following minor irritation to Littles area. In these patients, control of the bleeding is relatively simple using pressure. If packing is required it is important to determine whether the bleeding is a result of trauma (notably with fractures extending into the anterior cranial fossa or orbits), or not. In traumatic circumstances packing needs to be undertaken carefully as fractures can displace further (Fig. 34.3).

Epistasis has many causes. There may be a local problem confined to one or both nasal passages, or the patient may have a generalised disorder. The history is therefore very important in determining the cause. Localised causes of epistaxis include

1. Trauma (with or without a nasal fracture)
2. Foreign bodies (including nose-picking)
3. Inflammation (e.g. upper respiratory tract infections, sinusitis, rhinitis or environmental irritants)
4. Anatomical anomalies (e.g. hereditary hemorrhagic telangiectasia, septal spurs).
5. Topical application of drugs (particularly cocaine). Nasal sprays (particularly nasal steroids)
6. Intranasal tumours (e.g. nasopharyngeal carcinoma or nasopharyngeal angiofibroma)
7. Low humidity inspired air (eg during cold winter seasons).
8. Prolonged use of nasal cannula O2 (tending to dry the nasal mucosa)

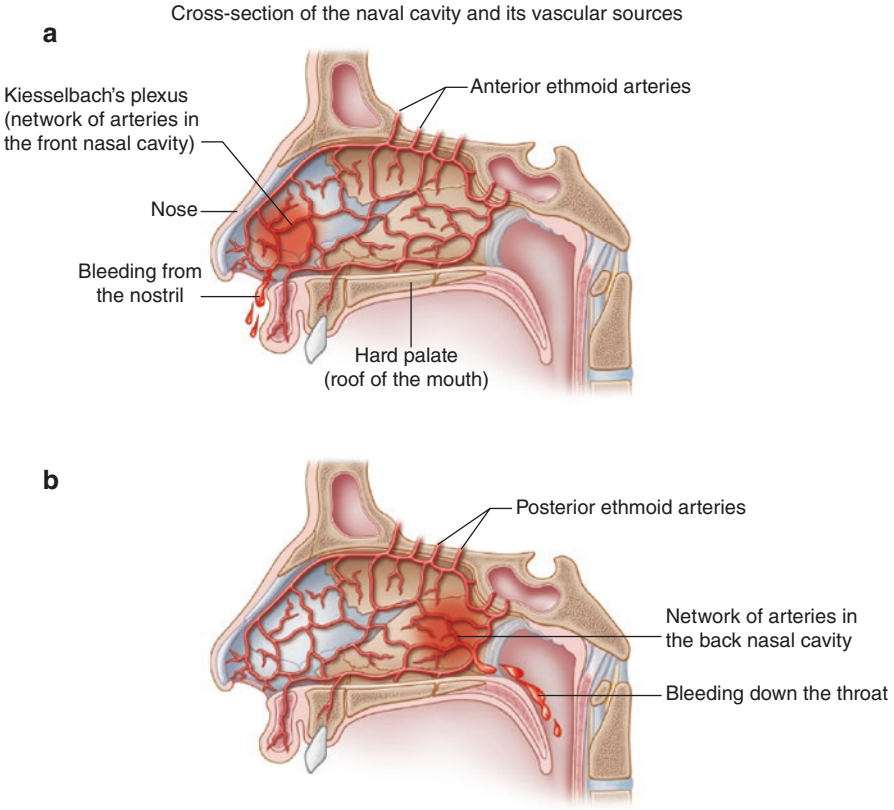

Fig. 34.3 Internal vascular anatomy and epistaxis

9. Middle ear barotrauma (aircraft or scuba diving)
10. Following surgery

A wide variety of systemic conditions, including medication can also precipitate bleeding. These include

1. Infectious diseases (e.g. common cold)
2. Hypertension
3. Alcohol (due to vasodilation)
4. Blood dycrasias
5. Connective tissue diseases
6. Drugs— Many medications can predispose to bleeding. Common drugs include aspirin, clopidogrel, warfarin, heparin and riboxaban (the effectiveness of which cannot be evaluated by haematological tests).
7. Chronic liver disease—cirrhosis causes deficiency of factor II, VII, IX, & X
8. Heart failure (due to an increase in venous pressure)

9. Haematological malignancy (such as leukemia)
10. Idiopathic thrombocytopenic purpura
11. Pregnancy (this is rare and is due to hypertension and hormonal changes)
12. Vascular disorders
13. Vitamin C and vitamin K deficiency
14. Von Willebrand's disease
15. Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)
16. Mediastinal compression by tumours (raised venous pressure)
17. Haemophilia
18. Thrombotic thrombocytopenic purpura

Spontaneous epistaxis is common in the elderly. This is because the nasal mucosa tends to become dry and friable with increasing age. Blood vessels may also be less able to constrict and the systemic blood pressure tends to be higher. With 'medical' epistaxis, packing is easier as there are no painful fractures or intranasal pathology. Nevertheless, depending on the underlaying cause packing can still present problems (as a result of partial obstruction of the airway, or failure to arrest haemorrhage if the patient has a clotting disorder). All patients should have their pulse and blood pressure checked on arrival, along with a full blood count and clotting screen if indicated by the history. Haemorrhage can sometimes be deceptive and in the elderly, with diminished cardiovascular reserve, cardiovascular compromise is common. If there are any clinical concerns patients should be admitted for observation and if necessary IV fluids.

It is important to determine where the bleeding is coming from. In the vast majority of cases, bleeding arises from Little's area on the anterior septum. Blood usually comes out of one nostril only. The blood predominantly exits the front of the nose although it can trickle into the throat. However, 5–10% of bleeds arise posteriorly, often from Woodruff's plexus, a venous plexus situated near the posterior end of the inferior turbinate. Bleeding from here can either be swallowed or exit out of both nostrils. This can continue briskly despite anterior packing. Posterior bleeds are often prolonged and can be difficult to control.

Management of significant epistaxis following trauma is discussed in the chapter on the injured patients. In the absence of significant trauma, in the first instance sit the patient forward and ask them to pinch the thin cartilaginous part of the anterior nose. This must be held for at least 5 minutes (although it can take up to 20 minutes for bleeding to stop). Patients should be encouraged patients to spit out any blood—swallowing it is a potent stimulus to vomiting. Cautery may be required with silver nitrate sticks or with needle diathermy. However be careful not to undertake extensive septal cautery. This can result in septal perforation. The use of local anaesthetic (xylocaine with adrenaline or cocaine pastes—beware overdose) can also help restrict bleeding and identify the source of the bleeding.

If bleeding continues rigid nasoendoscopy with the use of a haemostatic matrix (eg Floseal) may be necessary. If no source of bleeding can be found the cavity should be gently packed. Multiple techniques have been described including Merocel, Rapid rhino, Foley catheters, gauze impregnated Vaseline or bismuth

iodoform paraffin paste (BIPP). Since these packs are often retained for 48 hours antibiotics should be prescribed. Merocel is made of polyvinyl alcohol, which is a compressed foam polymer. This is inserted into the nose and expanded by the application of water. As the tampon swells and fills the nasal cavity, it applies pressure over the bleeding surfaces (Figs. 34.4, 34.5, and 34.6).

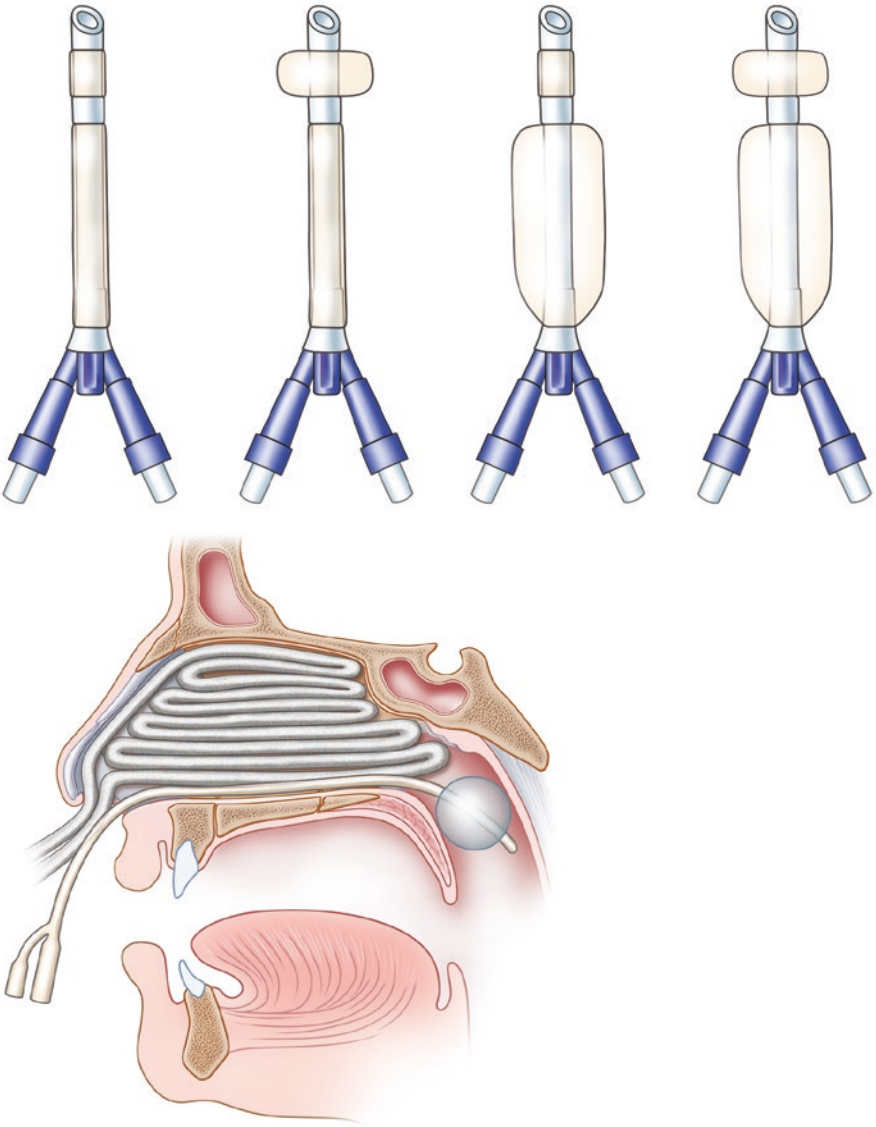

Fig. 34.4 Nasal balloons and packs

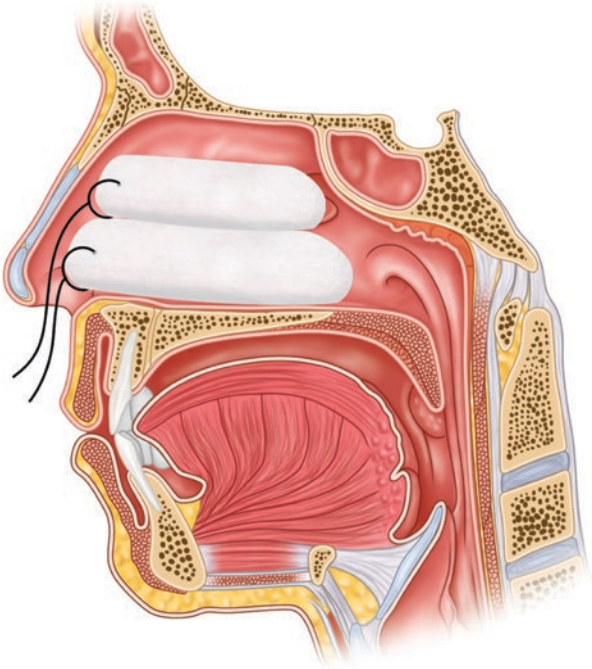

Fig. 34.5 Nasal packs (Merocel Medtronic Xomed, Jackson-ville. FL) are inserted parallel to the nasal floor to obtain useful haemostasis

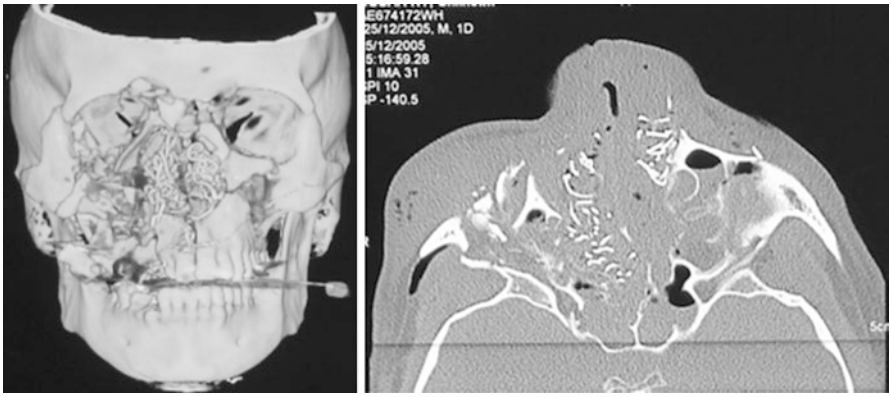

Fig. 34.6 Care with packing is required in trauma

Surgical control of epistaxis is rarely required. If so, this is now done endoscopically through the nose. Ligation of the external carotid and anterior ethmoidal artery is more of historical importance in all but exceptional cases. The anterior and posterior ethmoidal arteries pass through the orbit and can be exposed via a transorbital approach. Bleeding can also be stopped by catheter guided embolisation. Patients that require this tend to have some serious underlying conditions.

Following packing of the nose the patient should be advised

- For the next 24 hours they should avoid activities which may increase the risk of re-bleeding. These include blowing or picking the nose, heavy lifting, strenuous exercise, lying flat and drinking alcohol or hot drinks
- If the nose has been cauterised the person should avoid blowing their nose for a few hours to prevent straining of the nostril.
- If bleeding restarts and does not respond to first aid measures and the person should seek urgent medical advice.

The application of topical over-the-counter vasoconstrictive nasal sprays such as oxymetazoline or phenylephrine may be useful in the treatment of recurrent causes of epistaxis, such as allergic rhinitis. However the patient should be refers to a specialist for assessment before this is prescribed (Figs. 34.7 and 34.8).

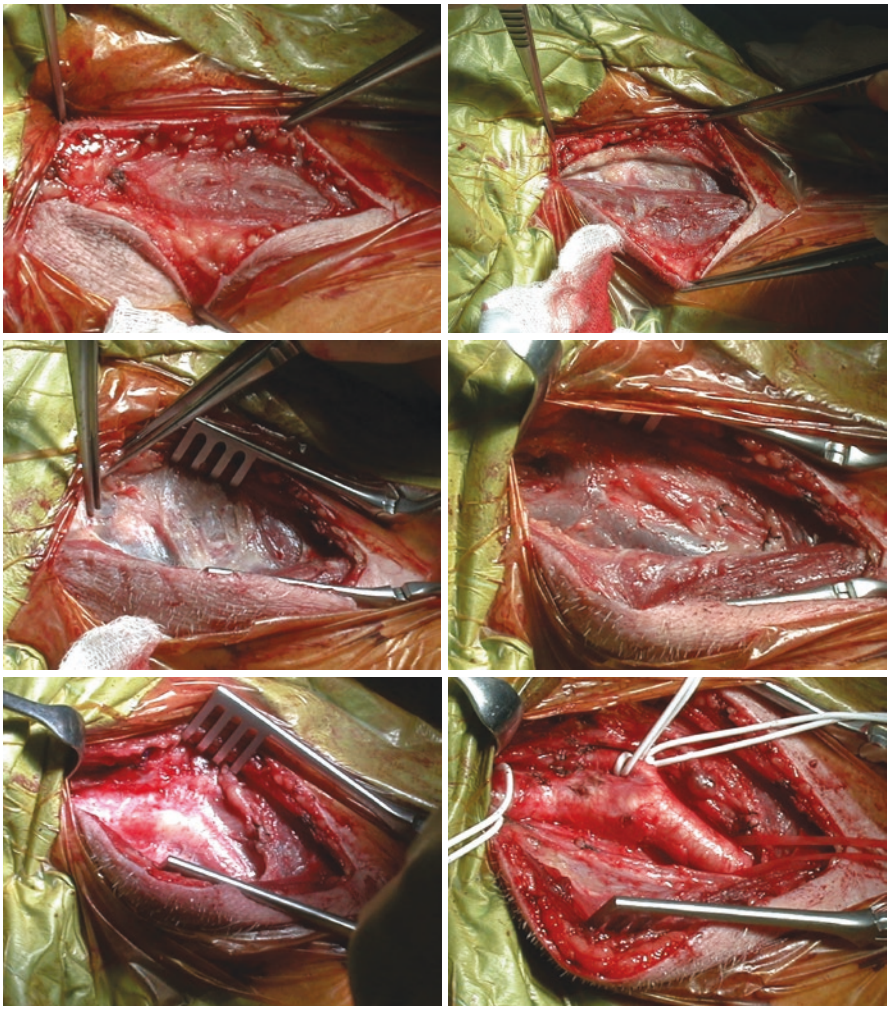

Fig. 34.7 Exposure of the external carotid artery

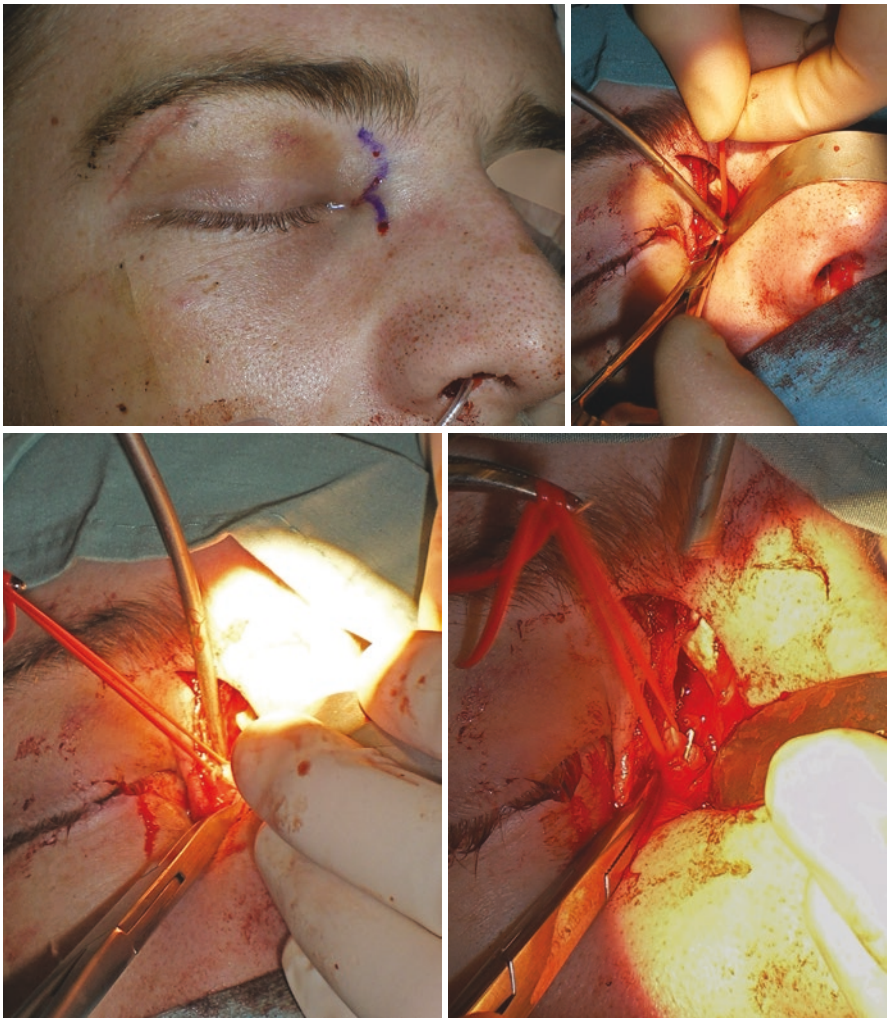

Fig. 34.8 Exposure of anterior ethmoid artery

34.13 Tumours of the Nose and Nasal Cavity

Tumours arising within the nasal cavity are uncommon. They represent around 1% of all cancers and about 3% of cancers of the upper respiratory tract. These tumours are generally placed with tumours of the sinuses in the subgroup of 'cancers of the sinonasal cavities'. Because the respiratory epithelium and adjacent bone contains diverse cellular elements, several types of malignancy can occur. These include (i) squamous cell carcinoma—SCC (70%), (ii) adenocarcinoma (10%) and (iii) adenoid cystic carcinoma (9%). These tumours comprise the vast majority of pathological subtypes, the latter two arising from minor salivary gland tissue. Rarer types include (i) malignant melanoma, (ii) olfactory neuroblastoma, (iii) malignant

fibrous histiocytoma, (iv) osteosarcoma, (v) chondrosarcoma, (vi) lymphoma, (vii) fibrosarcoma, (viii) leiomyosarcoma, (ix) angiosarcoma, (x) teratocarcinoma and (xi) metastases from elsewhere (notably renal cell carcinoma). Benign tumours include (i) inverting papilloma (schneiderian papilloma), (ii) haemangioma, (iii) benign fibrous histiocytoma, (iv) fibromatosis, (v) leiomyoma, (vi) myxoma and (vii) juvenile nasopharyngeal angiofibroma (JNA). Fibro-osseous lesions of the nasal cavity include fibrous dysplasia, ossifying fibroma and osteoma. Lymphomas (particularly in patients with HIV) and small cell neuroendocrine carcinomas often involve the anterior skull base. Rarely, intracranial tissues can also extend into the nasal cavity (encephaloceles, meningoceles and pituitary tumours). CT and MRI are therefore always essential prior to biopsy.

In most patients presentation is late. This is because the tumours do not cause problems until they have expanded significantly, or invaded through the nasal cavity. Initial symptoms are often attributed to more minor aetiologies, commonly rhinosinusitis. Early symptoms include nasal obstruction, epistaxis, pain and recurrent episodes of sinusitis. With low level or massive tumours, expansion towards the oral cavity may be associated with swelling of the gingiva or palate and loosening of the teeth. Orbital invasion can result in ocular symptoms, such as proptosis, diplopia and decreased acuity. In rare cases, upward invasion into the skull base, dura and brain may result in headaches, cerebrospinal fluid leaks and other central nervous system symptoms. Most anterior cranial base tumours arise from the sinonasal cavity and initially present with nonspecific and often subtle symptoms, including nasal obstruction, epistaxis, dysosmia, facial pain or minor visual changes.

Sinus tumours can also encroach into the nose and also present with nasal symptoms. Ethmoid tumours often present with diplopia, headache, epistaxis and unilateral nasal obstruction. Any unilateral opacification with bone destruction or remodelling on imaging requires urgent investigation to exclude or confirm a malignant lesion. Malignancy can also present as an apparent solitary "inflammatory" polyp. Therefore all polyps should be sent for histology.

CT and MRI are important in assessment, to precisely define the tumour's extent and its relationship to adjacent structures. Biopsy will also be needed to establish tumour type and aggressiveness. Full blood counts and coagulation tests should be taken as many tumours bleed easily. Scintigraphy using radiolabeled somatostatin analogs may have a role in staging in some tumours which express specific receptors. However its role in assessment has yet to be fully evaluated. Management of most cancers in now multi-discplinary, involving surgery, chemotherapy and radiotherapy. Specific treatment is generally dependant (i) histology/differentiation of the tumour, (ii) the tumour stage, (iii) the possibility of adequate surgical resection, (iv) the patient's medical status, (v) treatment risks and morbidity, (vi) reconstructive options (vii) the patient's wishes. Surgery combined with postoperative radiation therapy is the commonly undertaken for most sinonasal malignancies. These have a more favourable prognosis than sinonasal malignancies. All malignant tumours should therefore be urgently referred to a multidisciplinary setting, with input from radiologists, pathologists, surgical teams, oncologists and palliative care

teams. Squamous cell carcinoma of the nasal vestibule behaves more like cutaneous squamous cell carcinoma than sinonasal malignancies, and is staged according to the cutaneous classification system. These tumours are discussed in the chapter on the skin.

34.13.1 Juvenile Nasopharyngeal Angiofibroma (JNA)

This is a rare, benign, but locally aggressive vascular tumour involving the posterior nasal cavity, that is seen almost exclusively in prepubertal and adolescent males (age 7–19 year olds). It often presents with a epistaxis. Females may also be affected, but this is uncommon and they should therefore be sent for genetic testing if this is diagnosed. A hormonal theory for the development of these tumours has been suggested due to its occurrence in adolescent males. JNAs have been shown to have higher levels of hormone receptors and vascular endothelial growth factor (VEGF) than does normal nasal mucosa. Other theories include abnormal development of the nasopharyngeal periosteum or embryonic fibrocartilage found between the basiocciput and the basisphenoid. Paraganglionic cells of the terminal branches of the maxillary artery have also been implicated.

Histologically these tumours are composed of vascular tissue within a fibrous stroma. Their predominant blood supply is from branches of the maxillary artery, although the internal carotid artery may also provides branches. Of importance, the blood vessels within the tumour are reported to be thin-walled, lack elastic fibres and have absent or incomplete smooth muscle. This makes them fragile and at risk of rupture if traumatised (ie following biopsy).

Juvenile nasopharyngeal angiofibroma is said to commonly arise in close proximity to the posterior attachment of the middle turbinate, near the upper border of the sphenopalatine foramen. Growth below the nasopharyngeal mucosa continues until the nasal cavity is filled on one side, pushing the nasal septum over to the opposite side. The tumour may also grow toward the pterygopalatine fossa, deforming the posterior wall of the maxillary sinus. If severe cases the infratemporal fossa and sphenoid sinus can become invaded. Major intracranial complications have also been reported following erosion into the dura, orbital apex and cavernous sinus. Patients may therefore present with a variety of symptoms and signs

1. Nasal obstruction (the most common symptom)
2. Recurrent epistaxis or blood-stained discharge
3. Rhinorrhoea
4. Conductive hearing loss (Eustachian tube obstruction)
5. Recurrent otitis media
6. Anosmia
7. Visual impairment
8. Headache
9. Facial swelling

In most cases JNA presents with nasal obstruction and intermittent epistaxis. On examination the tumour may be seen as a sessile, lobulated, rubbery, pink to tan-grey mass. In some cases, the tumour can look like a polyp. Large tumours tend to be bilobed or dumbbell-shaped. Differential diagnosis includes hemangiopericytomas and antro-choanal polyps. However these lesions can bleed torrentially and it is important to be aware of this when considering biopsy. CT or MRI is usually indicated initially to assess the vascularity and extent of the mass. In some cases angiography may be required.

2 classifications of JNA have been described:

Sessions Classification

Stage IA—Tumour limited to posterior nares and/or nasopharyngeal vault
Stage IB—Tumour involving posterior nares and/or nasopharyngeal vault with involvement of at least 1 paranasal sinus
Stage IIA—Minimal lateral extension into pterygomaxillary fossa
Stage IIB—Full occupation of pterygomaxillary fossa with or without superior erosion of orbital bones
Stage IIIA—Erosion of skull base (ie, middle cranial fossa/pterygoid base); minimal intracranial extension
Stage IIIB—Extensive intracranial extension with or without extension into cavernous sinus

Fisch Classification

Stage I—Tumours limited to nasal cavity, nasopharynx with no bony destruction
Stage II—Tumours invading pterygomaxillary fossa, paranasal sinuses with bony destruction
Stage III—Tumours invading infratemporal fossa, orbit and/or parasellar region remaining lateral to cavernous sinus
Stage IV—Tumors invading cavernous sinus, optic chiasmal region, and/or pituitary fossa

Management can be complicated. Several hormone therapies have been tried for stages I and II of the disease with successful tumour shrinkage, but this has not been widely accepted. Radiotherapy has also been shown to provide good results, but is associated with complications, especially in young patients. It is therefore often reserved for extensive or intracranial disease, or recurrent cases. Surgical excision of the tumour following pre-operative embolisation is currently the preferred treatment whenever possible. A lateral rhinotomy, transpalatal, transmaxillary, or sphenoethmoidal route is used for small tumours. Endoscopic intranasal surgical resection of JNAs has also been reported in selected patients, with less blood loss and complications.

34.14 Nasopharyngeal Carcinoma

This is a common epithelial cancer involving the nasopharynx, usually around the lateral nasopharyngeal recess (fossa of Rosenmuller). Other neoplasms found here include lymphomas and sarcomas. The term 'lymphoepithelioma' is often used to describe epithelial carcinomas which contain a high infiltrate of benign lymphocytes. The aetiology of nasopharyngeal carcinoma is thought to be multifactorial, with genetic, viral, dietary, and environmental factors. It is known to occur following Epstein-Barr virus (EBV) infection and consumption of high quantities of salted fish during childhood (which contains a known carcinogen, dimethyl-nitrosamine). There also appears to be a genetic aetiology as it is more common in first degree relatives and certain racial groups. Nasopharyngeal tumours are more common in Asian, north African and middle eastern patients with peak incidences in late childhood and in 50–60 year olds. They represent about 1% of all nasal tumours in children. The World Health Organization (WHO) has classified nasopharyngeal carcinoma into 3 categories.

1. WHO-1—well–to–moderately differentiated squamous or transitional cell carcinoma with keratin production.
2. WHO-2—non-keratinizing carcinoma.
3. WHO-3—undifferentiated carcinoma, including lymphoepithelioma. These tumours consist of malignant epithelial cells with lymphocytic infiltration.

The vast majority of children are found to have WHO-3 disease. Patients can present with local symptoms including nasal regurgitation, nasal congestion, epistaxis and nasal discharge. Trismus, pain, otitis media, otalgia, hearing loss and cranial nerve palsies suggest referred symptoms or extension of the disease beyond the nasopharynx. Neuropathies occur in about 25% of patients. These occur more frequently in the relatively rare adenoid cystic carcinomas. The abducens nerve (CN VI) is frequently affected, but multiple cranial nerves may be involved—Petrosphenoidal syndrome (CN II to VI) and Villaret's syndrome (CN IX to XII and sympathetic nerves). These syndromes indicate intracranial extension (cavernous sinus) and invasion of the retropharyngeal space respectively. Trismus, dysphagia, and proptosis also indicate advanced disease. Trotter syndrome is the triad of i) unilateral conductive hearing loss due to middle ear effusion ii) trigeminal neuralgia due to perineural spread and iii) soft palate immobility. This is seen in advanced disease. Approximately 85 percent of patients will also present with cervical adenopathy and 50 percent with bilateral neck involvement. Distant metastases to the lungs, bones and liver is seen in 3 percent of patients at initial presentation, but may occur in up to 50 percent of patients during the course of the disease.

Investigations include CT and MRI of the skull base, nasopharynx and neck to determine the extent of disease. Patients should also have a chest radiograph, full blood count, urinalysis, biochemical profile and serum IgA titres to EBV capsid antigen. Liver function tests will be deranged if patient has hepatic metastases. PET scans may be required to assess for metastases. Treatment consists of surgery, chemotherapy and radiotherapy. Radiotherapy is the mainstay of treatment. With advanced disease and distant metastases, chemotherapy (cisplatin and 5-fluorouracil) is used. Neck dissection may be required if there is evidence of disease in the cervical lymph nodes. Palliation treatments include chemotherapy, radiotherapy and sometimes surgical intervention.

34.15 Squamous Cell Carcinoma

Squamous cell carcinoma (SCC) is the most common malignancy of the sinonasal tract with a peak incidence at around 60 to 70 years of age. There has been a reported association with nickel exposure, but unlike oral SCC alcohol and tobacco do not appear to predispose to sinonasal carcinomas. The tumours occur most commonly in the maxillary sinus followed by the nasal cavity, ethmoid sinus, and sphenoid sinus. Within the nasal cavity, they most commonly arises from the turbinates, followed by the nasal septum, floor, and vestibule. The lesions tend to be moderately differentiated. Surgical excision is considered the mainstay of treatment. Adjacent radiotherapy can be used depending on metastatic spread.

34.16 Adenocarcinoma

Adenocarcinomas are usually seen in men aged between 55 and 60 years. They tend to occur in the upper sinonasal region, often within the ethmoid sinuses. There is a higher incidence of adenocarcinomas in workers exposed to wood dust. Three histologic forms are common—papillary, sessile, and alveolar-mucoid. The papillary form is associated with woodworkers and tends to have a better prognosis. The sessile and alveolar-mucoid forms are more aggressive and carry a poorer prognosis.

34.17 Adenoid Cystic Carcinoma

These are cancers that arise from the minor salivary glands lining the sinonasal tract. Various types occur and adenoid cystic carcinomas accounts for almost 10 percent of sinonasal malignancies. The peak age incidence is between 40 and 60 years. Like their oral counterparts, these are locally aggressive cancers which grow slowly and recur or metastasise decades after treatment. Perineural spread is common, including the cranial nerves which provide access for spread into the skull base.

34.18 Mucosal Melanoma

These neural crest tumours constitute about 3 percent of all cancers of the sinonasal tract. The nasal cavity is more commonly affected than the paranasal sinuses. Not all lesions are pigmented, some may appear pink, resulting in diagnostic confusion. Prognosis is usually poor with local recurrence and distant metastases occurring in up to two-thirds of patients within the first year.

34.19 Olfactory Neuroblastoma (Esthesioneuroblastoma)

This is an undifferentiated neural crest tumour arising from the olfactory epithelium in the upper part of the nasal cavity. These tumours are common in both the young and older age groups. Most tumours occur unilaterally, but can become locally aggressive and grow into the opposite nasal cavity. They can also infiltrate intracranially or into the adjacent sinuses and orbit. At first they cause vague symptoms, which can result in delayed diagnosis. These include epistaxis, nasal obstruction, nasal discharge and headache. Olfactory neuroblastoma frequently invades the ethmoid sinuses, orbit, and anterior cranial fossa. Spread across the cribriform plate is the potentially most devastating complication with regards to survival. Tumours can invade the meninges at the anterior skull base and even spread to the pia without obvious destruction of the cribriform plate. The nasal septum may also be destroyed. More lateral spread can occur in to the medial wall of the maxillary sinus, the ethmoid sinuses, the pterygopalatine fossa, and the orbit. With advanced and invasive growth, later symptoms therefore include swelling, dental pain, proptosis, diplopia and increased intracranial pressure. In 10–20% of cases metastases can occur to the regional lymph nodes, lungs or bones. Tumours are usually staged according to the TNM classification

T1—Tumour involving the nasal cavity and/or paranasal sinuses (excluding sphenoid), sparing the most superior ethmoidal cells
T2—Tumour involving the nasal cavity and/or paranasal sinuses (including the sphenoid), with extension to or erosion of the cribriform plate
T3—Tumour extending into the orbit or protruding into the anterior cranial fossa, without dural invasion.
T4—Tumour involving the brain
N0—No cervical lymph node metastasis
N1—Any form of cervical lymph node metastasis
M0—No metastasis
M1—Distant metastases present

Treatment is often a combination of surgery and post-operative radiotherapy. Approximately two-thirds of patients survive 10-years. Chemotherapy is generally reserved for distant metastasis or tumours that are considered too big for surgical

resection. Some tumours run a very prolonged clinical course with relapses and recurrences over many years, while others are more aggressive and may develop rapid local, regional, and distant recurrences. Prognosis depends on the extent at presentation.

34.20 Undifferentiated and Rare Tumours

Undifferentiated tumours are highly aggressive and rapidly growing and can involve multiple sinuses and the nasal cavity. They carry a poor prognosis due to extensive local invasion. Rare tumours that have been reported include i) lymphomas, ii) sarcomas (fibrosarcoma, rhabdomyosarcoma, and osteosarcoma) and iii) minor salivary gland tumours (mucoepidermoid carcinoma).

34.20.1 Nasal Glioma

Nasal glioma (also known as nasal glial heterotopia) is a rare congenital benign lesion composed of ectopic neural tissue (glial cells), which have lost their intracranial connections. It is seen in a wide age group but typically presents at birth or in early childhood. Incomplete closure of the embryonic fontanelle between the nasal and frontal bones results in an abnormal connection between embryonic ectodermal and neuroectodermal elements. Consequently the glioma can present either as an intranasal or extranasal mass, usually in the midline of the bridge of the nose. It can also present as a polypoid swelling attached to the septum. With growth an intranasal glioma can develop into a large, firm submucosal red/bluish mass that extends inferiorly, towards the nostril.

Presenting features include nasal obstruction, cerebrospinal rhinorrhoea, meningitis, epistaxis and obstruction of the nasolacrimal duct, with epiphora. Respiratory distress can occur in infants. Ultrasound has been used to evaluate the consistency of the mass. However MRI is regarded as the gold standard to identify the site of the glioma and its extension. CT scanning may be required to exclude skull base involvement. Biopsy is usually required to confirm the diagnosis. Surgery is often curative. Extra nasal gliomas can be excised via a lateral rhinotomy, external rhinoplasty, midline nasal incision or coroanl flap, depending on the size and locations. Intranasal gliomas are now taken out using minimally invasive endoscopic surgery. If there is intracranial extension, a craniotomy will be necessary. Recurrence rates of around 10% have been reported.

34.21 Sinonasal Papilloma (Schneiderian Papilloma)

These are classified into three groups:

Septal papillomas (50%). These arise on the septum. They are exophytic and not associated with malignant degeneration.

Cylindrical cell papillomas (3%). These arise on the lateral nasal wall or in the paranasal sinuses. They are associated with malignant degeneration (10% to 15%), usually into SCC.

Inverted papilloma (47%). This is a unilateral, slow growing mass that is described as having a 'warty' texture. It can look very similar to a nasal polyp and can present with nasal obstruction. 5% of patients develop squamous cell carcinoma. Surgery is the mainstay of treatment although recurrence rates are high. Extensive or recurrent disease is treated with adjuvant radiotherapy.

34.22 Haemangioma

This is effectively a complex collection of blood vessels within the epithelial lining. They are relatively common in the head neck area, but intranasal lesions are less so. If present this can result in recurrent epistaxis and nasal obstruction. Haemangioma commonly presents in infancy, more commonly in girls. It can also be associated with PHACE syndrome (Posterior fossa brain malformations, Haemangiomas, Arterial anomalies, Coarctation of the aorta/cardiac defects and Eye abnormalities). Lesions usually proliferate in the first 6–9 months of life, and can become quite extensive. During this time they can be quite unsightly and a concern to the parents. This rapid growth phase is usually followed by a slower involution phase, although this doesn't always end with complete resolution of the lesion. Treatment may be deferred during the involutional phase. If indicated early, treatments include surgery, laser, cryotherapy and certain medications. Pulsed dye laser therapy may be indicated in the treatment of very early lesions to prevent further growth, or to remove the residual colour of a superficial involuting haemangioma. Surgery consists of debridement of the haemangioma. Only those lesions within the nose which resulted in recurrent epistaxis require treatment.

34.23 Osteoma

Osteomas are benign, slow growing bony masses, which usually arise on the surface of a bone. They can sometimes be found within the sinuses, often incidentally after a CT scan has been taken. Osteomas of the nose are rare, but can present as a small bony nodule. These are usually not painful. The exact aetiology is unknown. CT is recommended for diagnosis and surgical planning. Surgery usually involves limited excision of the bony prominence. This may only be required if the osteoma is causing obstructive symptoms. Otherwise it can be left where it is. Biopsy may be indicated.

34.24 Dermoid

Nasal dermoids are common developmental anomalies of the face. It is the most common congenital midline lesion. Most present at birth and early childhood but may become symptomatic in adulthood. It may present as a cyst, sinus or fistula and

can extend intracranially. The incidence is reported as around 1: 40000 births. Nasal dermoids are believed to be caused by incomplete obliteration of neuroectorderm in the developing frontonasal region. This often leads to a slowly growing mass, which can contain hair, teeth, fluid and skin glands. Nasal dermoids often present as a 'sac-like' cystic swelling found just above the medial canthus. There may be an opening to the skin. The cyst can extend into the frontal or ethmoid sinuses and the nasal bones. Intracranial extension is rare but they can extend through the foramen caecum or the cribriform plate and adhere to the falx cerebrae. There are a few case reports of extension onto the brain paranchyma. Dermoid cysts and their tracts can become intermittently inflamed and progress to abscess or osteomyelitis and rarely lead to meningitis or cerebral abscess.

Nasal dermoids are often diagnosed in first few years of life. Associated malformations reported include aural atresia, pinna deformities, mental retardation, hydrocephalus, pharyngeal arch sinus, cleft palate and lip deformities, hypertelorism and hemifacial microsomia. CT and MRI of the orbits are the gold standard to define the abnormality as well as any intracranial extension. Treatment is often curative. Without surgery dermoids can result in progressive enlargement leading to skeletal distortion, infection, meningitis and intracranial abscess. Early surgical intervention is therefore best so that any distortion of the developing face is kept to a minimum. The entire lesion along with any tract must be excised in order to prevent recurrence.

34.25 Nasoalveolar Cyst

A nasoalveolar cyst or Klestadt's cyst is a rare non-odontogenic, soft-tissue, developmental cyst occurring just below the nasal-alar region. It presents as an external swelling in the nasolabial fold, resulting in flaring of the nostril. The cyst is derived from epithelial cells retained in the mesenchyme following fusion of the medial and lateral nasal processes and the maxillary prominence during embryonic growth. These epithelial remnants persist and become cystic. Within the nose the cyst extends along the floor of the nose and can displace the inferior turbinate. Most cysts are usually asymptotic but can present as a painless, mobile, fluctuant, slow growing swelling in the upper lip, nose or anterior palate. It can also cause nasal obstruction. Cysts can become infected resulting in diagnostic confusion with dental abscesses. Investigations include OPG and CT. Treatment is usually surgical excision.

34.26 Vestibulitis

Nasal vestibulitis is an infection of the skin of the nasal vestibule. It is normally a minor infection at the opening of the nose. One or both nostrils can be affected. It maybe secondary to constant rhinorrhoea or viral infections such as herpes simplex and zoster. In children, foreign bodies can often be found, especially if this is accompanied by purulent discharge. Nose picking, acne and infected nasal hairs can

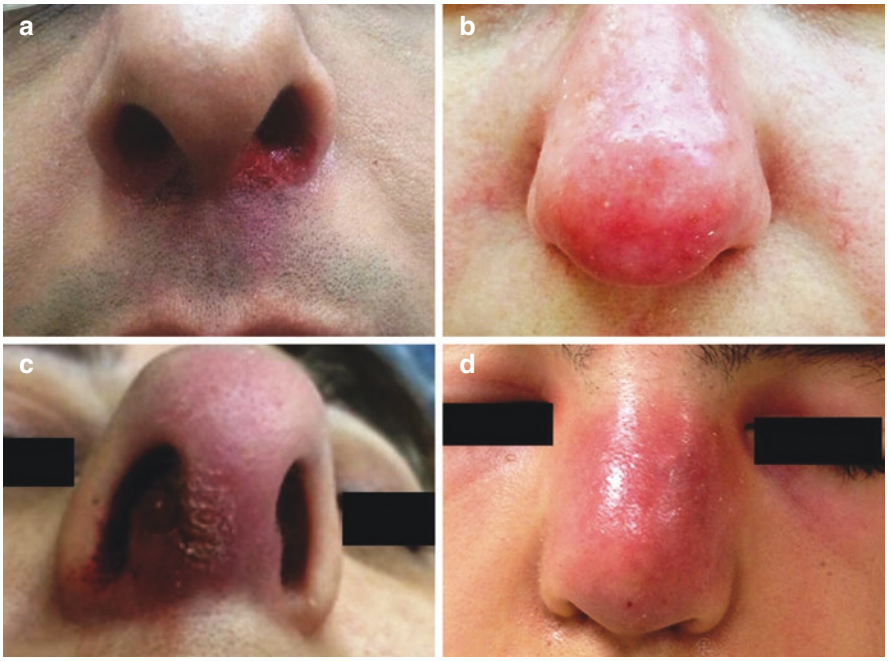

Fig. 34.9 Nasal vestibulitis. (**a**) Left nasal vestibulitis with erusting. (**b**) Nasal vestibulitis with localized cellulitis, (**c**) Mid-face cellulitis, (**d**) Vestibular abscess. Reproduced from Lipschitz et al. [15], with permission from Sage Publishing

also cause vestibulitis following introduction of bacteria and fungi into the skin. Recreational drugs such as cocaine can also lead to vestibulitis. Staphylococcus aureus is the most common bacteria, including methicillin resistant strains. Patients usually presents with crusting, rhinorhoea, epistaxis or purulent discharge. Eczema can mimic vestbulitis and should be considered in the differential diagnosis. Treatment often requires prolonged use of topical antibiotics and corticosteroid ointments over several weeks plus removal of any underlying cause. If vestbulitis does not heal biopsy is indicated. Although uncommon persistent vestibulitis can be the result of an underlying basal cell carcinoma or squamous cell carcinoma (Fig. 34.9).

34.27 Rhinophyma

Rhinophyma is an uncommon, but disfiguring condition in which there is progressive hypertrophy of the sebaceous glands and connective tissues in the skin of the nose. This leads to a large, fleshy nasal tip. The skin is thickened, leathery, highly vascular and may become grossly deformed. It can be as a manifestation of chronic acne rosacea, although, contrary to popular belief it is not related to alcoholism. Rhinophyma originates from the Greek ‘rhis’ for nose and ‘phyma’ for growth. In

advanced stages patients can develop nasal obstruction and sleep apnoea. Rhinophyma typically affects men between the ages of 45 to 60 years old. Because of the popular belief it is related to alcoholism this may lead to social stigmatisation illustrated by common synonyms of 'potato' and 'whiskey' nose. Diagnosis is usually a clinical. Investigations are often not required. Medical treatment includes topical metronidazole and tetracycline, or isotretinoin. These should be prescribed under the supervision of a skin specialist. Surgical management consists of superficial partial excision (debulking), in which 'sculpturing' of the excess tissue under local anaesthetic re-contours the shape of the nose. Specimens should be sent for histology to rule out any basal cell carcinoma or other tumour. Alternatively dermaplaning, dermabrasion, cryosurgery and laser surgery (CO2, NdYAg, Argon) maybe preferred.

34.28 Sarcoid

This is a chronic systemic disease of unknown aetiology, characterised by non-caseating granulomatous inflammation of various organs. It can affect the nose (skin, subcutaneous tissues, mucous membrane and bones), although this is uncommon. The cartilage is rarely involved and usually associated with overlying ulceration. Patients may present with nasal crusting, obstruction, post nasal discharge, recurrent sinus infections and headaches. Recurrent inflammation and healing of the nasal mucosa leads to fibrosis and nasal defects over time. Biopsy of a lesion is usually diagnostic. Treatment often involves a multidisciplinary approach depending of the organs involved. The mainstay of treatment is steroids. Other drugs that have been used as second line therapies include cytotoxic agents (e.g. Methotrexate), immunomodularors (chloroquine), monoclonal antibodies (Infliximab). The disease has a high remission rate.