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18.5

The upper respiratory tract

CONTENTS

18.5.1 Upper airway obstruction

James H. Hull and Matthew Hind

ESSENTIALS

The upper airway is anatomically defined by the carina inferiorly and pharynx superiorly, with pathology predominately arising from the trachea and larynx. Obstruction of the upper airway may arise because of a fixed structural pathology such as tracheal stenosis or malignancy, but can arise following loss of normal function (e.g. inappropriate vocal cord adduction).

A high index of suspicion is required to make the diagnosis because the clinical features of upper airway obstruction can mimic other respiratory conditions. Calculation of the ratio of forced expiratory volume in one second to peak expiratory flow (Empey index) may identify patients with unsuspected upper airway obstruction.

Acute upper airway obstruction is a medical emergency and usually caused by aspiration, oedema (allergic, hereditary, and acquired angio-oedema, smoke inhalation) or infection (croup, epiglottitis, quinsy, retropharyngeal abscess). Nonacute causes of upper airway obstruction include malignancy, tracheal stenosis, tracheal compression, dynamic large airway collapse, and laryngeal dysfunction.

Introduction

The upper airway can be defined as the section of the respiratory tract between the carina inferiorly and pharynx superiorly, with pathology predominantly arising from the larynx and trachea. This section of the airway should not be viewed as a simple conduit for ventilation, but serves important function including phonation and sphincteric protection of the lower airway.

Obstruction of the upper airway may arise because of a fixed structural pathology, as seen in tracheal stenosis or malignancy, or because of a loss of normal function (e.g. vocal cord dysfunction). It is remarkable how the respiratory system is able to adapt to significant degrees of upper airway obstruction without obvious consequences for respiratory function. Indeed, the airway lumen may be reduced by 70% in a healthy individual at rest with little evidence of overt clinical or physiological compromise. Beyond this point further reductions in the luminal cross-section of the airway may result in acute and catastrophic consequences for airflow and can precipitate asphyxia. This physiological reserve explains the clinical observation that patients with a developing stenosis often do not report a gradual deterioration in symptoms, but then present in acute respiratory distress.

The principal clinical manifestations of upper airway obstruction include exertional dyspnoea and 'wheeze', hence upper airway obstruction may mimic other respiratory conditions and is often treated as 'resistant' asthma or chronic obstructive pulmonary disease before the correct diagnosis is established. It is therefore vital that any clinician managing new airway symptoms considers upper airway obstruction in the initial differential diagnosis.

This chapter provides an overview of the clinical and investigation findings in upper airway obstruction and details clinical and pathological features of the most relevant conditions.

Clinical approach

History

The detection and diagnosis of upper airway obstruction often requires a high degree of clinical suspicion. This is especially true if pathology develops gradually or follows a variable time course. Indeed, while the cause may be obvious in acute disease, in a chronic and more insidious format, symptoms frequently overlap with other respiratory conditions.

Establishing the chronicity and periodicity of symptoms forms an important part of the clinical assessment; a temporally variable history suggests transient upper airway closure, whereas persistent

symptoms point to a structural aetiology. Likewise, the speed of symptom onset can be useful.

A detailed history of previous head and neck surgery and of any prior intubation or upper airway intervention is important. This should include an evaluation of prior intubation and/or tracheostomy. The development of subglottic stenoses may relate to intubation that occurred some years before.

It is also important to consider multisystem conditions that can manifest with large airway pathology such as rheumatological and vasculitic conditions, or respiratory diseases with large airway manifestations. The presence of gastro-oesophageal reflux and sino-nasal disease should be sought, and a history of medications associated with periodic upper airway obstruction (e.g. angiotensin converting enzyme inhibitors (see later) is important).

Details pertinent to laryngeal pathology include questions regarding the location of symptoms, change in voice quality, and presence of dysphagia. Importantly, several features typically taken to indicate an upper airway aetiology (e.g. a difficulty 'breathing in'), have a poor discriminatory value in separating extrathoracic causes of dyspnoea from other respiratory conditions.

Examination

Clinical examination features indicating upper airway obstruction include the presence of stridor or a fixed monophonic wheeze, prolonged inspiratory time, and abnormal thoracic movement. The 'wheeze' or whistle sound present in upper airway obstruction differs in nature from that arising from the lower airways. Specifically, wheeze or stridor arising from the upper airway is harsh and monophonic in nature, and it may be possible to identify the origin of the sound as coming from the upper chest/throat. This noise may only become apparent when ventilation is increased or may be variably present, depending on the relationship to a trigger or precipitant (e.g. exercise). It is therefore important to assess airway sounds through forced inspiratory and expiratory manoeuvres, not simply during passive respiration. Where symptoms are highly variable, it can be useful for patients to provide audio or video recording. When there is significant airway obstruction there may be evidence of abdominal paradox (i.e. abnormal inward movement of the lower thoracic cage and abdomen during inspiration).

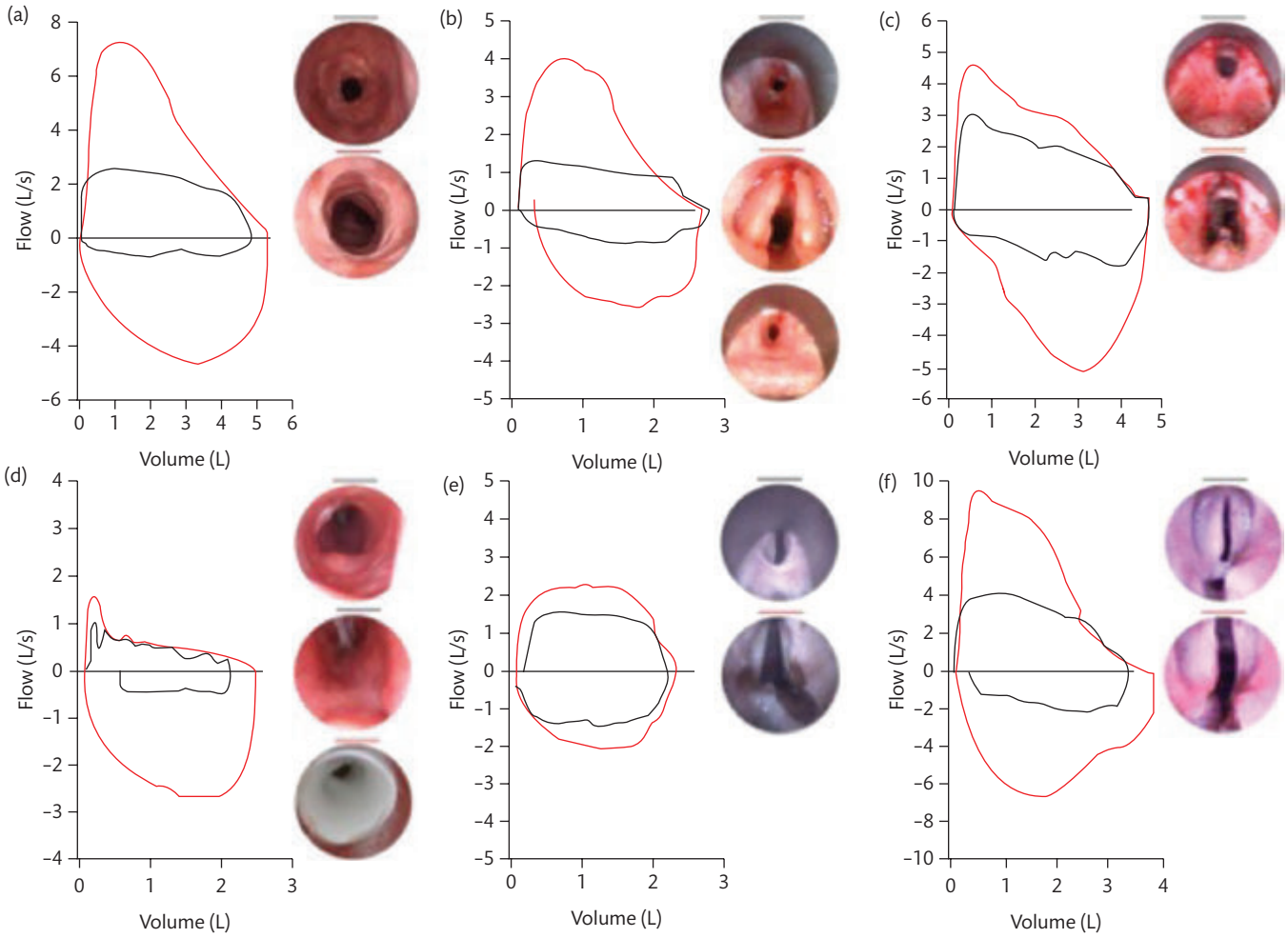

Fig. 18.5.1.1 Changes in the flow-volume loop in the setting of upper airway obstruction. (a), Idiopathic subglottic stenosis (b); including a flow-volume loop recorded at disease recurrence; granulomatosis with polyangiitis (c); relapsing polychondritis (d); and intubation-related posterior commissure stenosis (e and f).

Investigations

Lung function

Physiological measurements form a key part in the assessment of upper airway obstruction and indeed may often first highlight the presence of an abnormality. Spirometry forms the mainstay of physiological measurement. This manoeuvre requires an individual to perform a forced expiration, from total lung capacity to residual volume, and then to refill their lung immediately. This is displayed as a plot of expiratory and inspiratory flow against the volume exhaled, creating a 'flow-volume' loop. The normal appearance of the flow-volume loop is typically depicted as a 'triangle sitting on top of a semi-circle', but in many of the causes of upper airway obstruction this appearance is significantly altered—hence the flow-volume loop appearance may be considered a 'window to the airways'. In variable obstruction that occurs above the thoracic inlet (extrathoracic obstruction) only inspiratory flow phase is attenuated, whereas in fixed obstruction both the inspiratory and expiratory phases flow is impaired (Fig. 18.5.1.1).

Clinicians should note these abnormalities are not readily apparent from the standard clinic spirograph of volume versus time, or indeed in manoeuvres which do not include an inspiratory phase. It is also important to note that inspiratory manoeuvres are highly effort-dependent and thus attenuation of the inspiratory phase of the flow-volume loop, while relatively sensitive for extrathoracic obstruction, may be nonspecific.

The relationship between the peak expiratory flow (PEF) and forced expiratory volume in one second (FEV_1) can be informative. It was recognized over 40 years ago that the PEF is 'disproportionately' reduced in relation to the FEV_1 inpatients with upper airway obstruction. Such a reduction can be quantified by the 'Empey index', in which the FEV_1 (ml/sec) is divided by the PEF (litre/min),

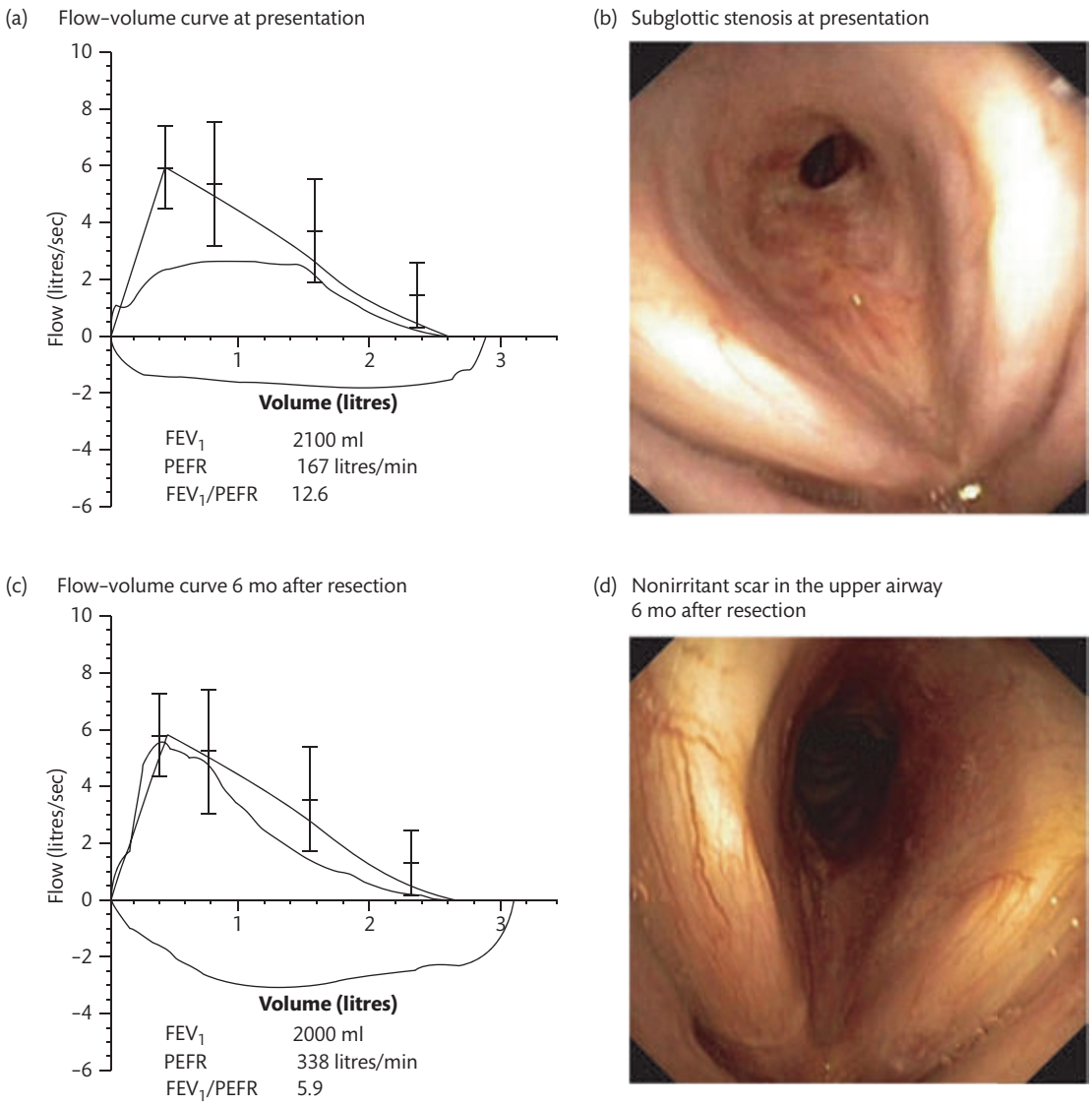

Fig. 18.5.1.2 Upper airway obstruction indicated by the change in appearance of flow-volume loop and a raised Empey index (the forced expiratory volume in 1 second (FEV_1) in millilitres divided by the peak expiratory flow rate (PEFR) in litres per minute).

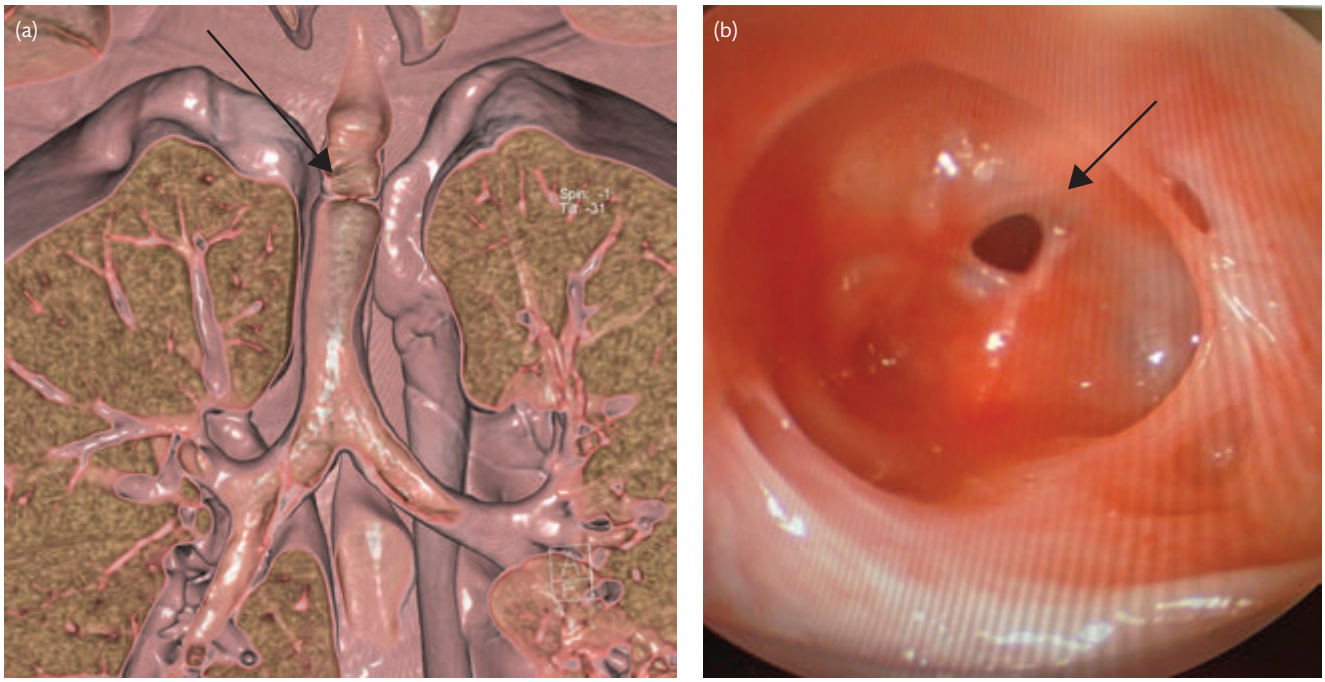

Fig. 18.5.1.3 (a) Multiplaner and volumetric CT reconstruction of the computer-generated image of the trachea. demonstrating a tight but thin tracheal web tracheal web (arrow) with a maximum diameter of just 2 mm at the site of a previous tracheostomy. (b) Rigid bronchoscopy allows clear visualization of a thin tracheal web which narrowed the diameter of the trachea to just 2 mm. Diathermy immediately improved ventilation.

and upper airway obstruction is consistently associated with a ratio typically of 10 or more (Fig. 18.5.1.2).

For further discussion of respiratory function tests, see Chapter 18.3.1.

Radiology

There are several techniques to enable rapid and focused assessment of large airway pathology.

Modern, multiplanar techniques allow complex 3D airway reconstruction and real-time assessment of laryngeal and large airway movement in the dynamic expiratory phase (Fig. 18.5.1.3). These techniques are evolving and may not be available outside specialist centres. Moreover, some CT protocols, if utilized incorrectly or if the obstruction is caused by a thin structure such as a tracheal web, may miss large airway pathology.

Direct visualization techniques

Clinic-based nasendoscopy is increasingly available and essential in visualization of the nasal cavity and larynx. The technique provides important information regarding laryngeal structure and movement and assessment of wheeze. It may reveal a cause for a patient's respiratory symptoms immediately (Fig. 18.5.1.4). However, it is also important to recognize that nasendoscopy may be entirely normal if the assessment occurs in the absence of the typical precipitant, and it may need to be repeated following exposure to a trigger. In the context of acute upper airway obstruction, direct visualization should only be carried out by clinicians with specific expertise in managing the upper airway. Subglottic causes of upper airway obstruction are not easily visualized with nasendoscopy and bronchoscopic evaluation is therefore required. Patients with nonacute suspected upper airway obstruction will initially often undergo flexible bronchoscopy, but may require a rigid procedure in order to allow safe diagnostic sampling. Bronchoscopy should include assessment of large airway movement and propensity for collapse during both tidal breathing and forced expiration (Fig. 18.5.1.5).

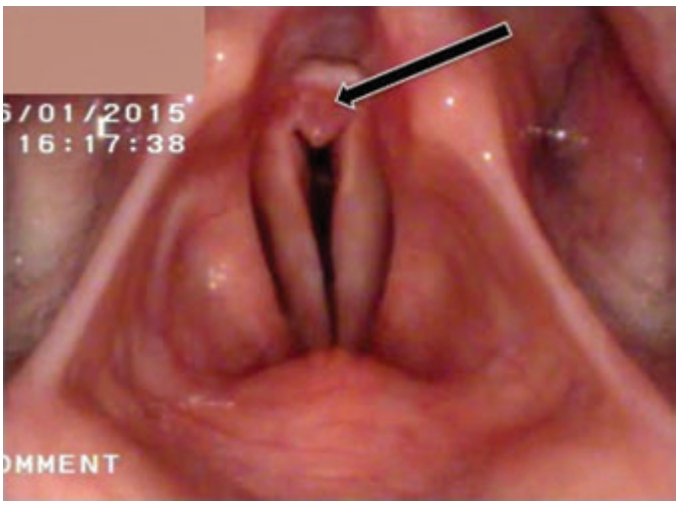

Fig. 18.5.1.4 Clinic-based nasendoscopy reveals posterior glottic stenosis, indicated by arrow, with chords maintained in fixed paramedian position. This patient was referred with treatment refractory asthma; symptoms resolved entirely following surgical intervention.

Image courtesy of Dr J Hull, Royal Brompton Hospital.

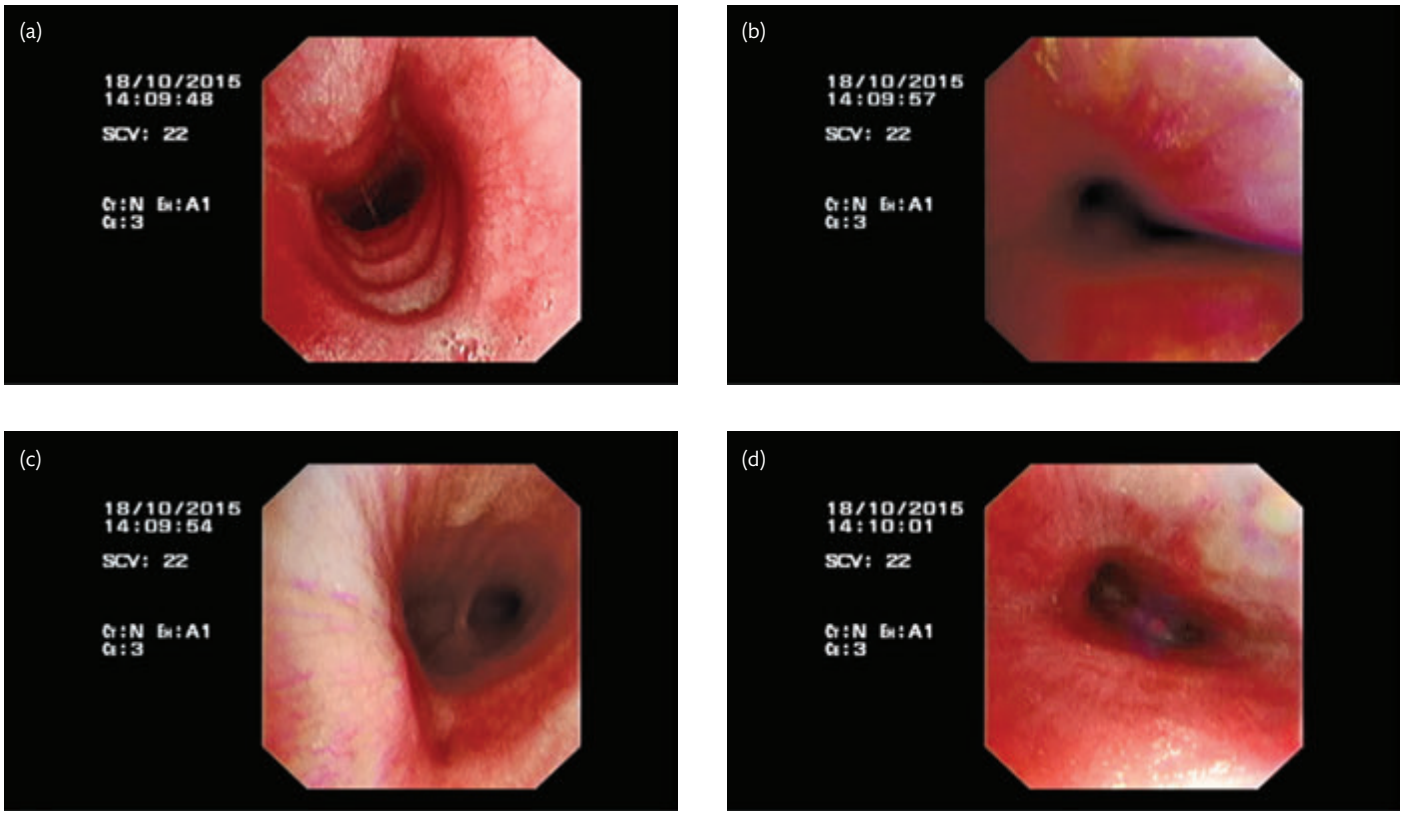

Fig. 18.5.1.5 Image stills taken during flexible bronchoscopy showing excessive dynamic airway collapse, with images taken during (a) passive respiration, and (b) forced expiration.
Figures courtesy of Dr P. Shah, Royal Brompton Hospital.

Emergency causes of upper airway obstruction

Acute airway obstruction is a life-threatening medical emergency that requires prompt treatment to avoid irreversible end-organ damage and death, which can occur within minutes following complete airway occlusion. Treatment and assessment are usually carried out simultaneously, with the objective of securing the airway. The cause is often obvious (Table 18.5.1.1). Patients who report a problem in the neck should always be taken seriously. Pharmacological intervention with steroids, epinephrine, and heliox (21% oxygen in helium) may all be useful, but clinical deterioration can be unpredictable. The patient requires close observation in an appropriate environment with facility for urgent intubation or emergency tracheostomy.

Table 18.5.1.1 Causes of upper airway obstruction

Acute	Subacute
Oedema	Tumour
Allergy	Subglottic stenosis (commonly following intubation/tracheostomy)
infections: (tonsillitis, pharyngitis, epiglottitis, croup, retropharyngeal, tonsillar, and peritonsillar abscess)	Tracheal compression (aneurism, tumour, thyroid enlargement)
Foreign body	Tracheal web
Burn inhalation injury	Tracheomalacia
	Tracheobronchomegaly
	Recurrent laryngeal nerve palsy
	Vocal cord dysfunction or inducible laryngeal obstruction (ILO)

Aspiration

In the United States, aspiration or choking on food has increased to be the fourth commonest cause of accidental death after road traffic accidents, falls, and fire. The 'cafe coronary' often involves a large, poorly chewed piece of meat which gets stuck and obstructs or partially obstructs the larynx or trachea. Incidence increases with age, comorbidities, and sedative and alcohol use. Patients usually present with immediate respiratory distress, aphonia, and cyanosis, which can lead rapidly to death. Treatment requires immediate recognition and removal of obstructing matter. The Heimlich manoeuvre, which can be done on oneself, back slaps, and abdominal thrusts are all useful. These forms an essential part of basic life support training taught by international resuscitation councils. Endoscopy, preferably using a rigid bronchoscope which allows ventilation and access for larger, grasping tools to remove the foreign body, is sometimes required. Emergency cricothryoidotomy where a small hole is made in the cricothyroid membrane can be lifesaving technique.

Infection

Infections of the upper airways rarely cause obstruction in adults but are significant causes in infants and children, particularly

streptococcal pharyngitis, tonsillitis, croup, and retropharyngeal abscesses.

Croup (viral laryngotracheobronchitis)

This is very common in children and usually caused by *Parainfluenza* 1–3 virus infection, but *Adenovirus, Respiratory syncytial virus*, and other viruses can also cause disease. The subglottic trachea is usually affected, causing tracheal narrowing which generates the steeple sign on a plain chest film. Patients usually present at night with hoarseness, a typical nocturnal barking cough, stridor, and drooling when severe. A croup score may be useful in stratification of disease. The condition usually resolves within 3–7 days. Treatment is supportive and dependent on the degree of respiratory distress. Steroids, nebulized racemic epinephrine (equal volumes of d and l-isomers of epinephrine which have prolonged action and reduced pressor effect compared with l-epinephrine alone), and heliox may be useful.

Epiglottitis

Historically more than 90% of cases were due to *Haemophilus influenzae,* which is now rare in countries where there is a comprehensive vaccination programme. It characteristically presents abruptly with drooling, dysphagia, and distress. The affected patient often adopts the tripod position with extension of the neck. Examination should be conducted extremely carefully as this can provoke complete airway occlusion. The epiglottis has a typical beefy or cherry red appearance. Management is focused on immediate securing of the airway, which should be done by an experienced paediatric anaesthetist. The prognosis is good once the airway secured. Treatment requires appropriate broad-spectrum antibiotics.

Tonsillar enlargement and peritonsillar abscess (quinsy)

Treatment is both supportive and directed toward the infection, often caused by a group B streptococcus. Quinsy often requires surgical drainage. Lemierre's disease, often secondary to a deep peritonsillar abscess caused by a *Fusobacterium necrophorum*, refers to thrombophlebitis of the internal jugular vein which can lead to sepsis and septic emboli to the lungs.

Retropharyngeal abscess

Though rare, the incidence in adults is rising and a high index of suspicion is required. It usually presents with sore throat, stridor, fever, neck stiffness, neck pain, and odynophagia. There is a high mortality due to association with carotid artery rupture, mediastinitis, epidural abscess, aspiration pneumonia, necrotizing fasciitis, and jugular venous thrombosis. Urgent ear, nose, and throat (ENT) assessment is required, with drainage and appropriate antibiotic treatment.

Oedema

In the hospital environment, extubation is commonly associated with laryngospasm and laryngeal oedema, and it has been reported that variable upper airway obstruction caused by oedema complicates up to 30% of extubations in the intensive care unit setting and is responsible for 4% of reintubations. The risk of post-extubation laryngeal oedema can be assessed by a cuff leak test where the endotracheal tube cuff is deflated and the leak determined. There are data suggesting the systemic steroids may be useful in preventing post-extubation laryngeal oedema. Early tracheostomy has also been suggested as a useful prevention strategy. Once identified, the airway must be protected with reintubation, emergency tracheostomy, or treated with medical therapy. Systemic steroids, nebulized epinephrine, and heliox are all thought to be useful. Respiratory support with noninvasive ventilation should be avoided as this may delay definitive airway stabilization.

Outside of hospital, acute oedema of the larynx or pharynx is usually due to allergy, a hereditary abnormality of the complement pathway, or occurs following inhalation of noxious gases.

Allergic oedema

Oedema affecting the face and upper airway can appear without warning. Usually, however, there is a history of atopy with a known allergy, prior history of hay fever, or the oral-allergy syndrome with pruritis of the lips, tongue, and palate after certain fruits and nuts. Whereas oral ingestion of allergens rarely progresses to IgE-mediated anaphylaxis with life-threatening upper airway obstruction (the proteins are broken down before absorption, exceptions being certain nuts, fish, and egg), insect stings can do so as they are directly injected.

Some apparent allergic reactions are not based on atopy and IgE but occur via IgG, direct activation of other inflammatory pathways, or ingestion of vasodilator substances. Treatment of allergic upper airways obstruction consists of intramuscular epinephrine (0.5 mg IM) with antihistamines and steroids (Chapter 17.3). Aerosolized epinephrine may also be useful.

Hereditary and acquired angio-oedema

Nonallergic hereditary and acquired angio-oedema are due to deficiency of plasma C1 inhibitor, a serene protease inhibitor, produced by the liver and monocytes, which regulates the first component of the complement pathway. This deficiency allows abnormal activation of the whole complement pathway, leading to activation of C1, C2, and C4 and production of vasoactive products such as bradykinin responsible for the resulting oedema. See Chapter 4.5 for further discussion.

Smoke inhalation

Smoke inhalation can cause thermal injury to the upper airways and contributes significantly to deaths due to fire. Upper airway obstruction due to heat injury and mucosal swelling usually develops within 24 h of exposure, but stenosis due to scarring can present later. A hoarse voice, stridor, severe conjunctivitis, burnt nasal hairs, and falling peak flow all suggest significant upper airway damage. Direct visualization with bronchoscopy is the best tool to determine whether there is oedema or mucosal ulceration obstructing the airways. Management usually consists of simple measures such as elevating the head of the bed and inhaling cool moist air with added oxygen. If peak flow falls, then transfer to an intensive care unit and bronchoscopy with the capability to perform an intubation, guided by direct vision, is required.

Subacute causes of upper airway obstruction

Malignancy

Cancers arising from the larynx and airway can cause airway obstruction. They are usually squamous carcinomas and are more common in smokers. Spread of a bronchial carcinoma into the trachea is probably the most common cause of upper airway obstruction seen

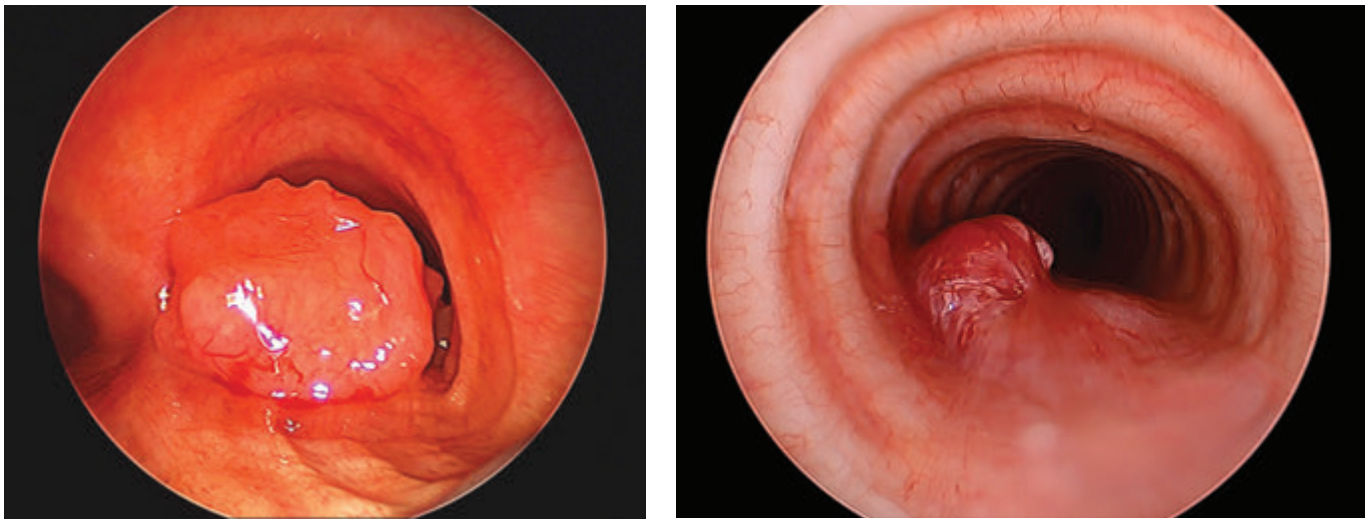

Fig. 18.5.1.6 Endotracheal malignancy viewed through rigid bronchoscope.
Figures courtesy of Mr S. Jordan, Royal Brompton Hospital.

by chest physicians. Laryngeal tumours commonly present with a change in voice and cough. Large airway tumours often present late as the chest radiograph is often normal. Assessment requires direct visualization and biopsy (Fig. 18.5.1.6).

Aside from intubation or tracheostomy (when appropriate), emergency treatment of tumours compromising the upper airway consists of dexamethasone (12 mg daily), nebulized adrenaline (10 ml of 1:10 000 up to six times daily), humidification of inspired air, and the use of heliox (21% oxygen in helium). Improvement in the airway may then be achieved by treatment of the tumour with chemotherapy or radiotherapy, but sometimes these may provoke tumour swelling, such that steroids are often prescribed first, with emergency treatments kept close to hand (heliox, adrenaline). If these therapies do not help, palliation can be achieved with the use of bronchoscopically guided laser therapy or cryotherapy, which either burn or freeze away tumour tissue with a low incidence of serious haemorrhage. However, these techniques are only of use with intraluminal tumours and cannot be applied when narrowing is due to external compression. Another approach is the use of silicone or metal endobronchial stents, some of which can be inserted either via a flexible or rigid bronchoscope. These are particularly useful when external compression is present, and can produce dramatic resolution of symptoms. It is rarely appropriate to 'debulk' a malignant tumour at thoracotomy in an attempt to improve large airway patency. Upper airway obstruction from tumour often becomes a terminal event. Adequate palliation and sedation must be given.

Some rare, nonmalignant tumours can obstruct the trachea (Fig. 18.5.1.7), and rarely granulomatous conditions such as sarcoid, Wegener's granulomatosis, and amyloid infiltration may mimic tumour.

Tracheal compression

A variety of abnormalities of adjacent structures can cause external compression of the trachea and lead to symptoms of upper airway

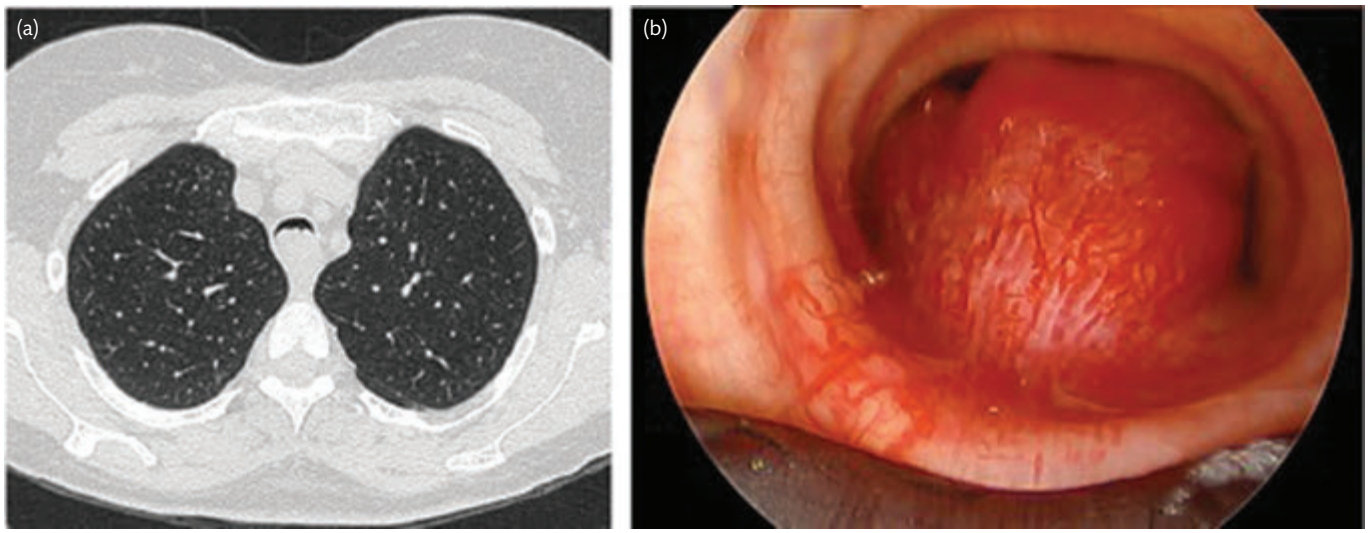

Fig. 18.5.1.7 (a) Tracheal mass virtually occluding the airway at the level of the aortic outlet on axial CT thorax. (b) Highly vascular endotracheal mass visualized at bronchoscopy.

obstruction. When present a thyroid goitre usually grows outwards, but occasionally it can wrap around the trachea and oesophagus and extend inferiorly into the superior mediastinum, causing narrowing and airway obstruction. Haemorrhage into a pre-existing cyst can cause acute worsening of symptoms. Surgery usually provides immediate relief of symptoms with the caveat that occasionally the goitre contributes to structural support of the trachea and removal can precipitate tracheomalacia.

A variety of congenital and acquired vascular abnormalities often involving the innominate artery or aorta can result in airway compromise. Mediastinal lymphadenopathy, thymoma, and fibrosis can all present with airway symptoms. Treatment for tracheal compression is dependent on the underlying aetiology, but a multidisciplinary approach is recommended with access to optimal diagnostic physiology and imaging, thoracic surgery, and/or stenting, and respiratory support with continuous positive airway pressure or non-invasive ventilation.

Tracheal stenosis

Tracheal stenosis is an important cause of iatrogenic upper airway obstruction and usually occurs at the level of the subglottis, typically caused by the cuff of an endotracheal tube or the first tracheal ring following tracheostomy. The incidence after intubation has been reported to be up to 20%, but may be less following the widespread use of low pressure cuffed tubes. CT reconstruction of the trachea may be useful, but examination with bronchoscopy and assessment by a specialist thoracic surgeon or ENT surgeon is required (Fig. 18.5.1.3).

Dynamic large airway collapse

Several conditions can be associated with an apparent collapse or excessive inward movement of the tracheal wall. Diagnostic criteria remain under discussion, but in some individuals the posterior tracheal wall may collapse to such a degree (i.e. >90%) that it almost completely occludes the large airway tract during expiration, and even during passive tidal respiration. This can have flow consequences and result in impaired exercise tolerance and cough, with difficulty clearing secretions. It is important to highlight, however, that a degree of large airway collapse occurs in healthy individuals and there can be a poor relationship between the degree of collapse visualized (e.g. on bronchoscopy or CT imaging) and impact on symptoms or lung function.

The terminology in this field is confusing and several terms have been used to describe large airway collapse (e.g. excessive dynamic airway collapse and/or expiratory central airway collapse). These conditions are probably best considered under an umbrella term of large airway collapse with symptoms, encompassing the conditions tracheobronchomalacia and excessive dynamic airway collapse. Tracheobronchomalacia is a term that should be specifically reserved to describe a pathological condition with structural weakness in the cartilaginous rings of the trachea and large airways, whereas excessive dynamic airway collapse describes invagination of the posterior membrane of the trachea leading to a 70% or more reduction in airway lumen (Fig. 18.5.1.8).

Relapsing polychondritis is a severe progressive form of tracheobronchomalacia. This is an inflammatory condition typically involving cartilage of the nose, ears, and large airways. There is often significant delay in diagnosis, with no specific diagnostic test available, although positron emission tomography scans and cartilage biopsies can be helpful. Treatment usually involves immunosuppression, with anecdotal success using novel biologic agents in addition to supportive treatment of the tracheal collapse with respiratory support, stents, and surgery. See Chapter 19.11.9 for further discussion.

Tracheomalacia and excessive dynamic airway collapse are thought to occur in a significant number of patients suffering from chronic obstructive pulmonary disease and asthma. Treatment is largely supportive and directed at coexistent conditions; optimal airway support with continuous positive airway pressure and non-invasive ventilation is often very useful.

Mounier-Kuhn syndrome or tracheobronchomegaly is a rare, abnormal dilatation of the trachea caused by atrophy of the muscular and elastic tissues of the trachea. Secondary tracheobronchomegaly can be caused by connective tissue disease. Recurrent respiratory tract infection is common. Treatment is supportive.

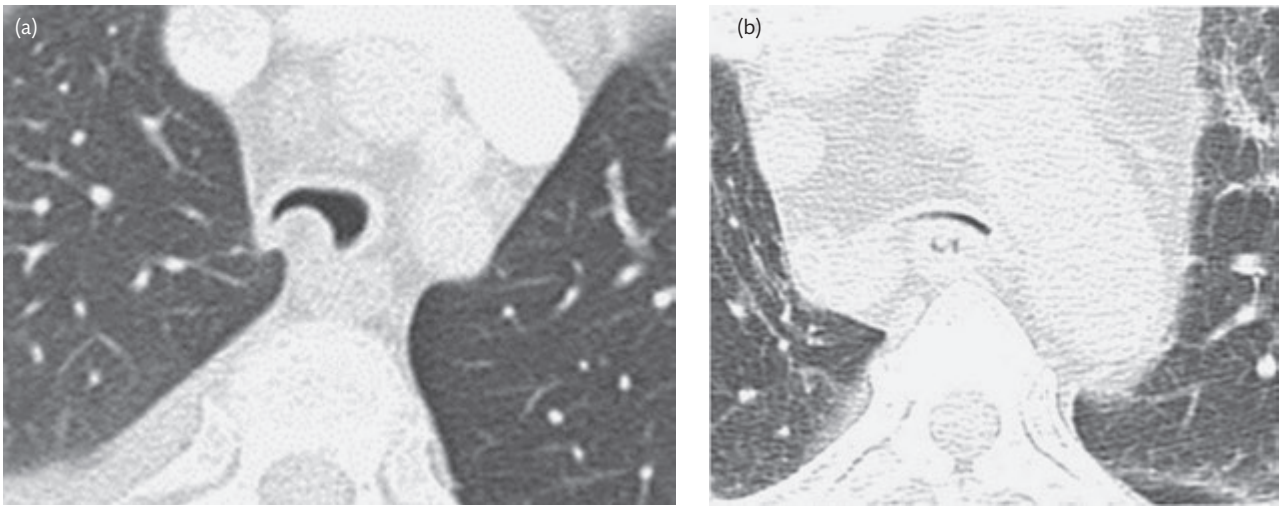

Fig. 18.5.1.8 CT images demonstrating (a) excessive dynamic airway collapse and (b) tracheobronchomalacia. See text for discussion.
Figures courtesy of Dr A Devaraj, Royal Brompton Hospital.

Table 18.5.1.2 Features distinguishing periodic occurrence of laryngeal obstruction from asthma

	Inducible laryngeal obstruction	Asthma
Onset	Rapid (within seconds)	Variable (within minutes)
Pattern	Can resolve rapidly	Variable but typically symptoms but persistent during exacerbation
Inhaled drug therapy	Largely ineffective	B_2 agonist usually effective
Breathing characteristics	Monophonic inspiratory wheeze, prolonged inspiratory phase	Polyphonic expiratory wheeze, prolonged expiratory phase
Regional limitation	Upper airways, neck	Lower airways, chest
Symptoms	Dyspnoea, wheeze, stridor, cough, throat/chest tightness, dysphonia	Dyspnoea, wheeze, cough, chest tightness
Precipitating factors	Exercise, emotional stress, cold air, strong odours/scents	Exercise, infections, cold air, allergies, stress

Laryngeal dysfunction

The anatomical position of the larynx dictates its role as the true 'gateway' to the airways, and complex reflex mechanisms have evolved to prime the larynx in a state of 'readiness for closure' (i.e. in order to protect the lower airways). Despite this, on a day-to-day basis, and in most individuals, the larynx functions autonomously many thousands of times daily, and without higher cortical response. However, in some situations the larynx may adopt a physiological state that could be considered maladaptive or 'dysfunctional' and close acutely (e.g. in the state of laryngospasm).

There is a spectrum of 'laryngeal dysfunction' disorders, which may be viewed as overlapping conditions with shared manifestations. Many patients with chronic nonproductive cough can describe features of a general 'laryngeal hypersensitivity', and at times this may extend to a clinical situation characterized by laryngeal narrowing with symptoms arising from the voice box (e.g. dysphonia or globus). However, it is important that clinicians consider and exclude structural or neurological causes of laryngeal disease before they consider the diagnosis of dysfunction or hypersensitivity.

The term *vocal cord dysfunction* has been used for over 40 years to describe the phenomenon of inappropriate vocal cord adduction, which results in distressing symptoms such as dyspnoea, wheeze, and laryngeal discomfort. Several terms have subsequently been used to describe a variety of laryngeal closure syndromes, and most recently the term *periodic occurrence of laryngeal obstruction (POLO)* has been advocated. This term is preferred because it aptly describes the temporal nature of the condition, but also acknowledges the fact that, in many cases, the obstruction may involve structures within the larynx that are distinct from the vocal cords (i.e. supraglottic adduction classically seen in exercise-induced laryngeal obstruction). The term laryngospasm is best reserved for the condition of acute, catastrophic laryngeal closure, which can result in a loss of consciousness. This typically occurs following laryngeal instrumentation.

A diagnosis of POLO may be suggested by features in the history (Table 18.5.1.2) and intermittent abnormalities on lung function testing, but ultimately a secure diagnosis is often dependent on nasendoscopy being performed when symptoms are present.

Treatment is targeted at minimizing any aggravating factors (e.g. reflux or sino-nasal disease) and use of speech and language therapy. Low doses of amitriptyline have been used to treat vocal cord dysfunction, and local botulinum toxin injection has been used successfully to treat laryngospasm.

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18.5.2 Sleep-related breathing disorders

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ESSENTIALS

Obstructive sleep apnoea and other sleep-related breathing problems significantly impair the functioning of about 0.5–1% of the population.

Obstructive sleep apnoea

Obstructive sleep apnoea in adults is usually caused by obesity and fat deposits in the neck area (typically collar size of 17 inches (43 cm) or more), when the withdrawal of postural muscle tone during sleep allows the pharyngeal dilators to be overwhelmed, leading to