Physiology of Fixed Airway Obstruction

Ross K. Morgan and Ruaidhri Keane

Contents

Abstract

Upper airway obstruction frequently presents to the interventional bronchoscopist, and common causes include malignant disease of the trachea and postintubation airway stenosis. The physiological changes that occur with upper airway obstruction are a consequence of increasing turbulence of airflow at the site of stenosis and a consequent increase in airway resistance which leads to an increased work of breathing, hypoventilation, and exercise limitation. Upper airway obstruction is associated with distinct changes on pulmonary function testing. Careful inspection of the inspiratory and expiratory limbs of a well-performed flow-volume loop demonstrates three identifiable patterns of variable intrathoracic, variable extrathoracic and fixed airway obstruction in which there is flow limitation throughout the respiratory cycle. A number of quantitative criteria obtained from spirometry have also been found to have value in recognition of upper airway obstruction, in particular in differentiating this from chronic obstructive conditions such as COPD (chronic obstructive pulmonary disease) and asthma which affect the smaller airways but may coexist. Studies have found that patterns on lung function tests suggestive of upper airway obstruction can be found in as many as 7% of

R. K. Morgan (✉) · R. Keane
Beaumont Hospital, Dublin, Ireland
e-mail: rossmorgan@beaumont.ie; keaner5@tcd.ie

L. Yarmus et al. (eds.), *Principles and Practice of Interventional Pulmonology*,
https://doi.org/10.1007/978-3-031-49583-0_6-1

consecutive tests. While abnormalities on spirometry and flow-volume loops are often the first indicator of airway obstruction and are cheap and widely available, they lack sufficient sensitivity for diagnosis and when suspected airway inspection by bronchoscopy and airway imaging, usually by computerized tomography, are required.

Keywords

Chronic obstructive pulmonary disease · Airway obstruction · Airway resistance · Tracheal stenosis · Small airway disease

1 Introduction

This chapter will focus on fixed upper or central airway obstruction which may present to the interventional bronchoscopist for assessment and management. It is important to recognize this as it is commonly misdiagnosed and the physiological changes that occur can be detectable on pulmonary function testing. Fixed *airflow* obstruction is a term used to describe the consequence of airway remodeling in the face of unchecked inflammation in asthma or the irreversible small airway dysfunction seen in chronic obstructive pulmonary disease and will not be discussed here.

The causes of fixed upper airway obstruction are numerous and are listed in Table 2. This chapter will describe the physiological changes that occur, demonstrating the effects on pulmonary function testing that may be useful in diagnosis and provide a number of illustrative cases. The normal airway anatomy and dynamics of airflow and airway resistance will first be described as a good working knowledge of these is required before considering the altered physiology of central airway obstruction.

2 Airway Anatomy

The upper airway is made up of three sections: the anatomic spaces of the nose, mouth, and pharynx, the larynx, and the trachea. The larynx extends from the root of the tongue to the trachea and consists of three parts: the supraglottis, which includes the epiglottis and the false vocal cords; the glottis, which includes the vocal cords and surrounding structures; and lastly the subglottis, a 1.5–2-cm segment below the cords that is completely surrounded by the cricoid cartilage and below which the airway becomes the trachea.

The trachea can be divided into two segments by the thoracic inlet; the shorter cervical trachea extends for the first 2–4 cm from just below the cricoid cartilage to the thoracic inlet. The intrathoracic trachea then continues for a further 7–9 cm until dividing into the right and left main stem bronchi at the main carina. The wall of the trachea contains up to 22 horseshoe-shaped cartilaginous rings that line the anterior and lateral walls and give the tracheal its shape. A membranous component which does not contain any cartilage completes the posterior wall.

The length and diameter of the trachea is roughly proportional to the height of the individual. In the adult male, the trachea is approximately 10–13 cm long with external diameters of 2.3 cm coronally and 1.8 cm sagittally. In women, corresponding tracheal dimensions are 2.0 and 1.4 cm. There are approximately 2 C-shaped rings per cm. The trachea has inherent flexibility and some elasticity; this significantly reduces with age as calcification of the rings occurs. In addition, in the elderly as the vertebral height is reduced and kyphosis occurs, the trachea may take on a more horizontal course which can result in more likelihood of extrinsic compression. The cross-sectional horseshoe shape of the trachea can also be markedly altered in the elderly, particularly in those with COPD in whom the distal trachea becomes flattened from side to side, taking on a so-called sabre-sheath shape.

2.1 Trachea: Anatomic Relationships

When considering ways in which the trachea can get compressed or obstructed, it is worth reviewing the anatomic relationships of the trachea along its length. Throughout its course, the trachea lies in close contact with the esophagus which runs slightly to the left and behind the airway. Anteriorly, the thyroid isthmus crosses over the superior part of the trachea at the level of the second and third tracheal rings. At the tracheal midpoint, the brachiocephalic artery crosses over from the aortic arch to the right side. On the left lateral side, the aortic arch makes an impression on the left lower tracheal wall. The tracheal bifurcation is at the level of the fifth thoracic vertebra. Mediastinal lymph nodes lie adjacent to the airway wall in paratracheal, pretracheal, and subcarinal locations.

2.2 Lower Airway Anatomy

Below the carina, the airways divide approximately 22–25 times before terminating in alveolar sacs, the functional unit of ventilation. The first 16–18 divisions make up the conducting airway passages of the human adult lung, and the remaining six or seven divisions are considered the respiratory zone composed of respiratory bronchioles from which alveoli bud from the walls. Finally, the airways terminate in the alveolar ducts which are lined with alveoli.

3 Physiology of Breathing

During inspiration, the thoracic cavity volume increases mainly due to diaphragmatic contraction and flattening and, to a lesser extent, through the action of the external intercostal muscles which raise the ribs, drawing air into the lung and down to the terminal bronchioles. This part of inspiration occurs with large velocity, and the pressure required to move gas along is very small as the airways are uniquely designed to conduct air to the terminal bronchioles. For instance, a flow rate of 1 l/s can be achieved with a pressure drop along the airway of less than 2 cm water. Thus, with the normal swings in intrathoracic pressure during resting ventilation of 3 or 4 cm water (change in intrapleural pressure from +2 cm to −2 cm water), air can move with considerable velocity down to the respiratory zones. Once air arrives there, the cross-sectional area of the acini, or functional respiratory units, is so large (50–100 m^2) and the distances so short that there is a rapid fall in velocity of inhaled air and rapid diffusion within the respiratory units occurs.

Accessory muscles of inspiration include the scalene muscles which elevate the first two ribs as well as the sternomastoids which can elevate the sternum to some extent. Associated small muscles that play a minor role include the alae nasi, which cause flaring of the nostrils, and some of the extensor neck muscles, all of which try to increase the diameter of the upper airway passages.

The compliance of the lung reflects the change in volume for unit pressure, and in health, the lungs are naturally very distensible with a compliance of about 200 l/cm water. Lung compliance increases in diseases such as COPD where there is reduction in lung elasticity due to tissue destruction and emphysema. Reduced compliance is associated with conditions that make the lungs stiff, such as pulmonary oedema and pulmonary fibrosis.

4 Airway Resistance and Flow

Resistance can be defined by the mathematical formula:

$$R = \Delta P / V$$

in which R is resistance, ΔP is the pressure difference or driving pressure, and V is the flow. It is expressed in $cmH_2O/L/s$. Resistance to airflow into the lungs as they expand is contributed to by intrinsic pulmonary resistance (e.g., elasticity) of the lungs and chest wall but mainly is accounted for by airway resistance, which makes up about 80% of the total resistance. As discussed above, the cross-sectional area of the lower airways is so great, and the branch points so numerous that resistance is almost negligible at the lower levels. Thus, most of the overall airway resistance comes from the upper airways from the level of the trachea to about the seventh-generation bronchi. Within these larger airway tubes, resistance to flow is inversely related to the radius of the tube to the fourth power. Therefore, in the presence of fixed obstruction that narrows the airway radius by half, for example, a tracheal obstruction that reduces the lumen to 1 cm, a 16-fold increase in airway resistance results.

Air obeys the principles of fluid dynamics as it moves along the airways from higher- to lower-pressure areas. Flow can occur in three different patterns within the airways: laminar, turbulent, and transitional with eddy formation at branch points or when encountering irregularities along the airway surface (Fig. 1). Laminar flow is streamlined with air in the center of the tube moving fastest, and increasing driving pressures will increase flow proportionately. With turbulent flow, a higher pressure is required to maintain the flow rate, and the relationship between driving pressure and flow is nonlinear (Fig. 1d). In normal conditions in most of the bronchial tree, the airflow is transitional. Whether flow will be laminar or turbulent also depends to a large extent on the Reynolds number (Re). The Reynolds number (Re) is the ratio of inertial forces to viscous forces, and it is used to predict flow patterns. It is defined by a mathematical formula that describes the relationship between the density and viscosity of a gas:

$$\mathrm{Re} = \rho \cdot v \cdot d / u$$

where ρ is the density, d is diameter, ν is velocity, and u is viscosity. Laminar flow occurs at low Reynolds numbers, while turbulent flow occurs at high Reynolds numbers. Low-density gases like helium tend to produce less turbulence. This underlines the utility of heliox, a mixture of 60–80% helium and 20–40% oxygen, in patients with central airway obstruction and respiratory distress. Helium has a density one-third that of nitrogen. By reducing the Reynolds number, heliox decreases the tendency for turbulent flow to develop and thereby results in an increase in flow for the same driving pressure, reducing dyspnea and work of breathing. This may buy some time in initial management of central airway obstruction in the acute situation.

5 Measurement of Airway Obstruction: Pulmonary Function

Airflow is typically measured in the pulmonary function laboratory using the spirometer and through the generation of flow-volume loops. A flow-volume loop is created when the patient inhales deeply to a total lung capacity (TLC) and then forcefully exhales until the lungs have been emptied to the residual volume (RV) followed by rapidly inhalation again to reach the TLC. A typical normal loop is shown in

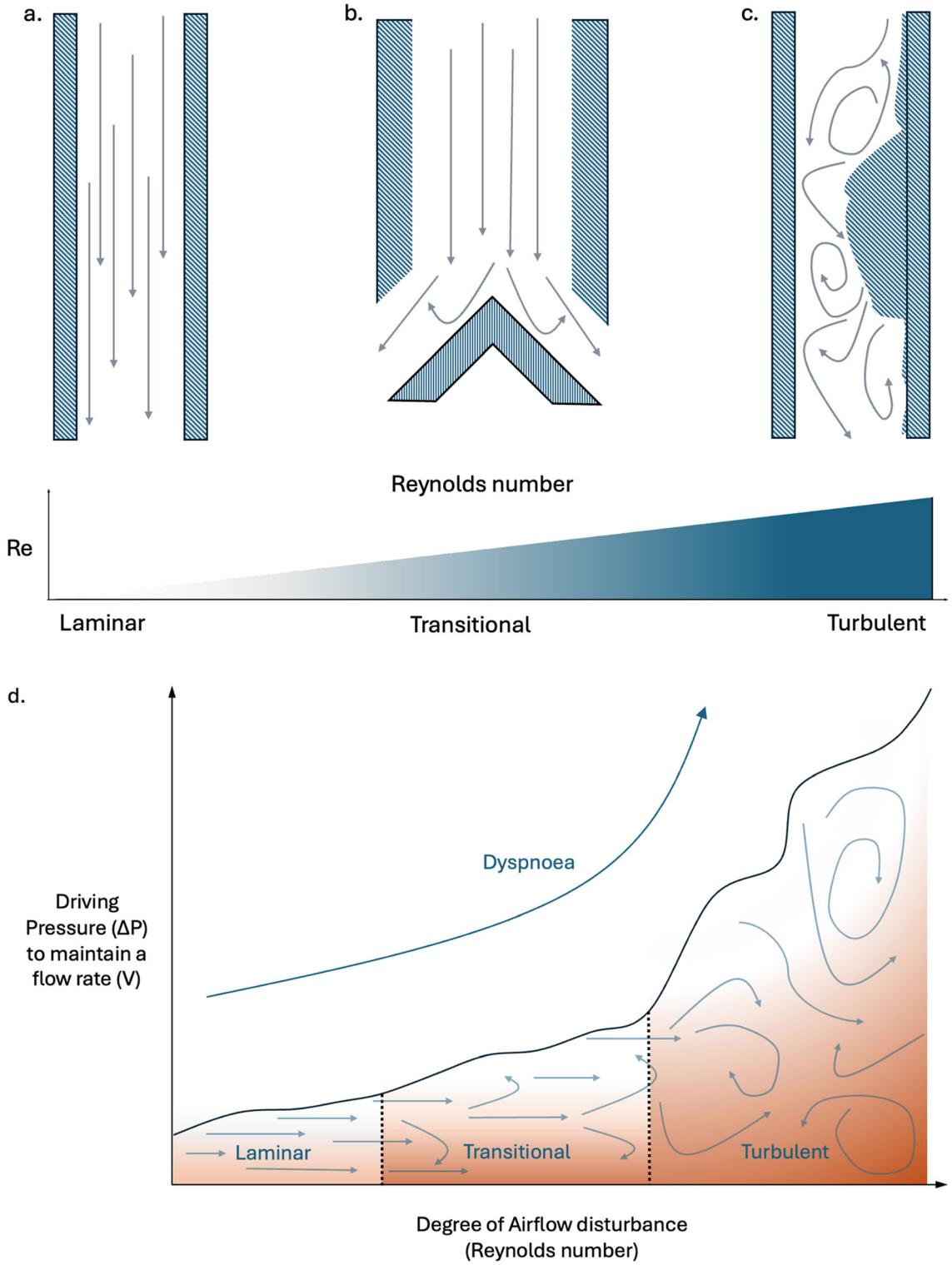

Fig. 1 Airway airflow, (**a**) laminar flow in normal airway, (**b**) transitional flow at airway division, (**c**) turbulent flow at airway abnormality, accompanying graphical representation of airflow disturbance with increasing Reynolds number. (**d**) Graphical illustration demonstrating that an increasing driving pressure (ΔP) is required to maintain a flow rate (V) in the context of increasing airflow disturbance or Reynolds number. The relationship between driving pressure (ΔP) and turbulent flow is nonlinear. This increased driving pressure (ΔP) can be perceived as dyspnea

Fig. 2a. The upper portion is the expiratory limb and has been well studied and found to contain a wealth of information on airflow obstruction, in particular for small airway disease in COPD. The inspiratory portion gets less attention but as discussed below can be particularly useful in observation of large airway obstruction. It should be noted however that forced inspiratory maneuvers in particular are effort dependent and that reaching acceptability and repeatability criteria such as they exist for the inspiratory limb of the loop can be difficult. Additionally, most of the studies on the use of flow-volume loops in airway obstruction which are outlined below are observational and limited to small numbers of patients.

5.1 Flow-Volume Loops

The seminal work on the use of flow-volume loops in airway obstruction was published by Miller and Hyatt in the late 1960s [8]. They proposed a classification based on pressure changes around the site of obstruction and identified three patterns from the contours of the flow-volume loop: (1) variable intrathoracic, (2) variable extrathoracic, and (3) fixed obstruction. For these studies, they simulated airway obstruction by having normal subject's breath through tubes of increasing resistance (smaller diameter) and found that flow rates plateaued during inspiration and expiration and that plateaus were reached at lower flow rates as the resistances were increased Fig. 3.

During normal respiration, there are changes in transmural pressure along the tracheal length that impact on the caliber of the large airways. The extrathoracic airway is surrounded by positive atmospheric pressure, tending to collapse the airway during inspiration when tracheal pressure is negative. At the same time, the intrathoracic airway is surrounded by pleural pressure which becomes more negative during inspiration helping to keep these airways open [1]. Thus, obstruction within the extrathoracic airway will impact on flow to a greater extent during inspiration (Fig. 2b, variable extrathoracic obstruction), whereas obstruction within the intrathoracic airway will affect flow more during expiration when the transmural airway pressure increases and these airways are tending to collapse (Fig. 2c, variable intrathoracic obstruction). If the obstruction is stiff or circumferential, these dynamic changes do not impact on the flow as it is limited throughout the cycle, and this can be seen as plateauing of both the inspiratory and expiratory limbs of the flow-volume loop (Fig. 2d, fixed obstruction). Where there is unilateral main bronchus obstruction, the maximum inspiratory flow tends to be higher at the beginning than toward the end of the forced inspiration because of a delay in gas filling.

Since these original observations by Miller and Hyatt, a number of other criteria based on visual inspection of the flow-volume loop have been proposed as useful although non-specific for the detection of upper airway obstruction [8].

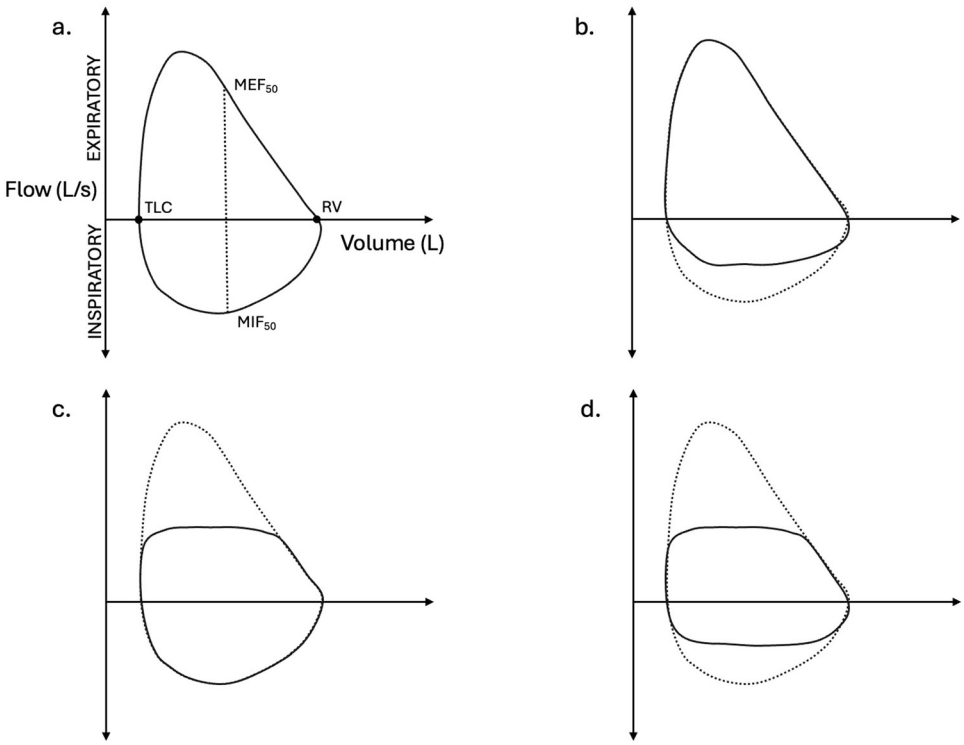

Fig. 2 Flow-volume loops. (**a**) normal, (**b**) variable extrathoracic obstruction, (**c**) variable intrathoracic obstruction, (**d**) fixed airflow obstruction

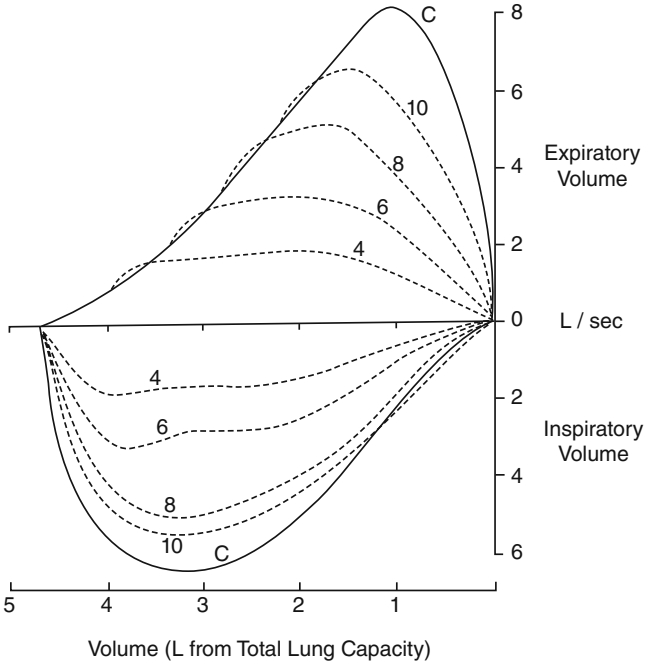

Fig. 3 Flow-volume loops obtained from breathing through progressively smaller orifices

Table 1 Proposed criteria for distinguishing *upper airway obstruction* on pulmonary function tests

Visual criteria	
Presence of a plateau	Miller and Hyatt [7]
Biphasic shape	
Presence of oscillations	
Quantitative criteria	
Increased PEFR:PIFR	Nairn and McNeill [10]
Increased MEF50 to MIP50	Jordanoglou and Pride [5]
FIF 50% ≤ 100 L/min	Rotman [14]
FEF 50%/FIF 50% ≥ 1	Rotman [14]
FIF25-75/FEF25-75 < 1	Owens and Murphy [11]
FEV1/PEFR ≥ 10 ml/l/min—the peak flow is proportionately more reduced by upper airway obstruction than is the FEV1	Empey [3] and Rotman [14]
FEV1/FEV0.5 ≥ 1.5—the FEV0.5 is proportionately more reduced by upper airway obstruction than the FEV1	Yernault [17] and Rotman [14]
DLCO single breath unchanged	Sackner [15]

MIP50 maximal inspiratory flow at 50% of the vital capacity, *FIF50%* forced inspiratory flow at 50% of the vital capacity, *FEF50%* forced expiratory flow at 50% of the vital capacity, *PEFR* peak expiratory flow rate measured in liters per minute, *FEV1* forced expiratory volume in 1 s measured in milliliters, *FEV0.5* forced expiratory volume in ½ s measured in milliliters

These include biphasic shape and oscillations in the expiratory or inspiratory curves (Table 1).

Table 2 Causes of fixed large airway obstruction

Nonmalignant	**Tumors**
Extrinsic	**Benign tracheal tumors**
Goiter	*Hemangiomas*
Lymphadenopathy	*Granular cell tumors*
Vascular compression	*Papillomatosis*
Postintubation stenosis	*Lipoma*
Idiopathic	*Pleomorphic adenoma*
Infection	*Chondroma*
Tuberculosis	*Neurofibroma*
Histoplasmosis	**Malignant**
Mucormycosis	*Bronchogenic carcinoma*
Nocardia	*Adenoid cystic carcinoma*
Granulation tissue, for example, anastomotic	*Mucoepidermoid carcinoma*
Inflammatory/infiltrative	*Angiosarcoma*
Amyloid	*Carcinoid tumors*
Sarcoidosis	*Kaposi's sarcoma*
Relapsing polychondritis	**Metastatic disease to airway**
Wegener's	*Lung*
Foreign bodies	*Renal*
Iatrogenic	*Breast*
Tracheobronchopathia osteoplastica	*Thyroid*
	Colon
	Melanoma

5.2 Spirometry

Central airway obstruction primarily affects airflow by worsening airway resistance. Consequently, spirometry, which is a simple plot of expiratory volume against time, might be expected to be useful in detection. This was first proposed by Jordanoglou and Pride in 1968, and their observations were extended by Empey in 1972. While the FEV1 alone is an insensitive marker, a number of ratios of flow and other quantitative criteria have been investigated over the years and have been found useful in discriminating patients with airway obstruction proximal to the level of the carina. These quantitative criteria are outlined in Table 1.

The essential finding from these studies is that the cardinal feature of upper airway obstruction is a reduction in flow at large lung volumes, whether inspiratory or expiratory. This is a consequence of the fact that at large lung volumes, flow is effort-dependent, whereas at low lung volumes, the intrathoracic airways are compressed, and flow becomes dependent on lung elastic recoil which is effort independent. This becomes useful in the discrimination of small airway obstruction (COPD) from upper airway obstruction.

It is important to be aware when interpreting the spirometry and flow-volume loops that patient effort has a major impact on all these ratios, and so it is crucial that the patient

effort is as near maximal as possible, particularly during inspiration. Poor inspiratory effort is common and when isolated rarely indicates upper airway obstruction, and the technician should confirm the quality of the test and effort on the report.

5.3 Total Lung Volumes/Diffusion Capacity

In the absence of coexisting peripheral airway obstruction, asthma, or other pathology, the overall lung volumes as measured by nitrogen washout or plethysmography should be normal in fixed airway obstruction as is the single breath diffusion capacity of carbon monoxide. However, these tests are often difficult for the patient with central airway obstruction to perform.

5.4 Emerging Techniques

Impulse oscillometry (IOS) is an emerging diagnostic method for assessing airway obstruction. It is based on the principle of the *forced oscillation technique*, which describes the impact of a superimposed small amplitude oscillation on the air pressure-flow relationship at different frequencies. It quantifies airway impedance by assessing two main parameters: resistance (R) and reactance (X). Unlike traditional spirometry, IOS does not require forced expiratory maneuvers. IOS is performed during tidal breathing, and this may be more suitable for those with symptomatic airway obstruction. Recent small studies, case series, and case reports have provided a signal for its use in distinguishing between fixed and variable central airway obstructions. A recent study from Japan demonstrated that resistance (R) remains nearly constant across different frequencies in fixed central airway obstruction [4]. This was significant when compared to the marked frequency dependency of resistance they observed in variable central airway obstruction, where R5 was greater than R20, similar to COPD. Interestingly, this study also reported that all IOS measurements significantly improved postinterventional bronchoscopy for central airway obstruction, and this correlated well with a positive symptomatic response.

6 Effect of Exercise and Posture

In central airway obstruction, airflow turbulence and airway resistance increase with higher respiratory rates. The maximal voluntary ventilation (MVV) maneuver can therefore bring out unsuspected airway obstruction because as the respiratory rate rises, there is a fall in exercise capacity due to hypoventilation associated with the climbing airway resistance, and this leads to a large fall in observed MVV [6]. A ratio of maximal voluntary ventilation to forced expiratory volume in 1 s of less than 25 is usually observed. Similar changes are seen with exercise, and thus, patients with fixed obstruction will often first complain of dyspnea or stridor on exertion.

Alterations in the contour of the flow-volume loop can also occur with changes in posture, with the decreases in normal values seen in recumbency in normal subjects exacerbated in the setting of upper airway obstruction. Obtaining flow-volume loops in lying and standing positions may therefore increase the sensitivity of this method in detecting an upper airway lesion.

7 Fixed Airway Obstruction and COPD

Detection of concomitant upper airway lesions in patients with COPD may pose a particular challenge to the clinician as symptoms of progressive airway obstruction such as dyspnea, and wheeze may be common to COPD and can be missed [13]. The expiratory limb of the flow-volume loop takes on a characteristic coved-out appearance in COPD due to loss of small airways and elastic recoil. As a consequence, the absence of a classic plateau in expiratory portion of the flow-volume loop does not rule out the presence of coexisting upper airway obstruction.

The FEV1 to FEV0.5 ratio has been proposed as a useful index for separation of the functional abnormalities seen in COPD from those of upper airway obstruction. When the FEV0.5 is less than 60% of the FEV1, a diagnosis of central airway obstruction is suggested. A further nonspecific method of discrimination can be observed with inhalation of heliox. Heliox improves peak flow in the setting of turbulent airflow as seen in airway obstruction but has no impact on the obstruction seen in COPD, which is a small airway disease in which flow is predominantly laminar.

The various indices from the flow-volume loop and spirogram that have been found useful in distinguishing upper airway obstruction and COPD are given in Table 1.

8 Interpretation of PFTS

While the changes in visual and quantitative findings on spirometry or flow-volume loop delineated in Table 1 are well described, most of the observations and original studies on which they are based were carried out in normal subjects or in patients breathing through a mouthpiece where airflow obstruction was simulated. This should be borne in mind as the diagnostic performance of the criteria outlined in detecting central airway obstruction is uncertain, and in fact to date, there is a dearth of published data evaluating this. A

recent paper from Modrykamien and colleagues from the Cleveland Clinic evaluating performance of these criteria in consecutive patients undergoing pulmonary function suggested a presence of central airway obstruction of 7.5% [9]. However, the criteria performed poorly alone, and while there was an increased sensitivity when an aggregate performance scale was used, overall sensitivity of these physiological tests was less than 70%.

While this suggests better criteria to help predict the presence of upper airway obstruction may be established, for now, it underlines how essential further evaluation with airway inspection by bronchoscopy and imaging is in the assessment of suspected central airway obstruction.

9 Clinical

9.1 Presentation

Chronic upper airway obstruction may progress insidiously and as a result goes unrecognized and often misdiagnosed, often masquerading for years as asthma or chronic obstructive pulmonary disease. Figures 4, 5, 6, and 7 provide case examples with classic flow-volume loops showing fixed airway obstruction. With anatomically fixed obstruction, wheezing and dyspnea are typically unresponsive to bronchodilators, and failure of a patient to improve with these treatments should prompt further evaluation.

Dyspnea is the usual presenting symptom and may be worse on lying flat as well as with exercise. Typically, exertional dyspnea occurs when the airway luminal diameter is reduced to about 8 mm. In patients with a laryngeal level of obstruction, dysphonia may also be a feature. Resting dyspnea does not usually result until the airway diameter falls to 5 mm, at which point on examination stridor is the cardinal feature. Stridor can be defined as an abnormal, high-pitched sound which results from turbulent airflow through a partially obstructed upper airway. It is heard best when the airflow is maximal, that is, with a deep breath and at the level where the airway lumen decreases, for example, over the neck. The tone of stridor varies, and it can be variously described as harsh, musical, or breathy. It should be easy clinically to differentiate from stertor, which is heavy snoring-type inspiratory sound typically generated at the level of the naso- or oropharynx and not associated with airway disease.

Worsening stridor can be observed when minute ventilation increases, such as during exercise. Because the airway lumen can reduce so dramatically before symptoms intervene, over half of patients with fixed airway obstruction will present with respiratory distress. Frequently, this is precipitated by an upper respiratory tract infection which causes increase work of breathing and further airway lumen compromise.

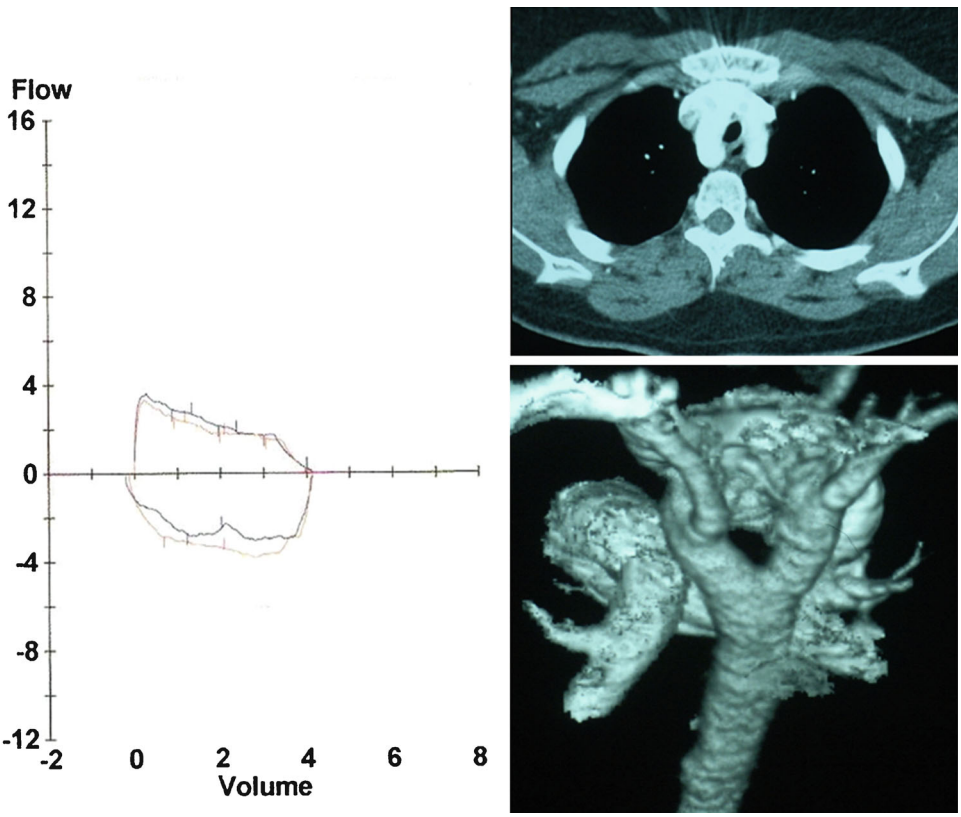

Fig. 4 A 16-year-old girl presented with a 6-year history of wheeze. The flow-volume loop shows characteristic plateau in inspiratory and expiratory limbs, suggesting fixed airway obstruction. A thoracic CT angiogram was performed and shows a vascular ring with dual aortic arch and ascending aorta bifurcation anterior to the mid-trachea causing compression. (Images courtesy of Dr. Sanjay Chotirmall)

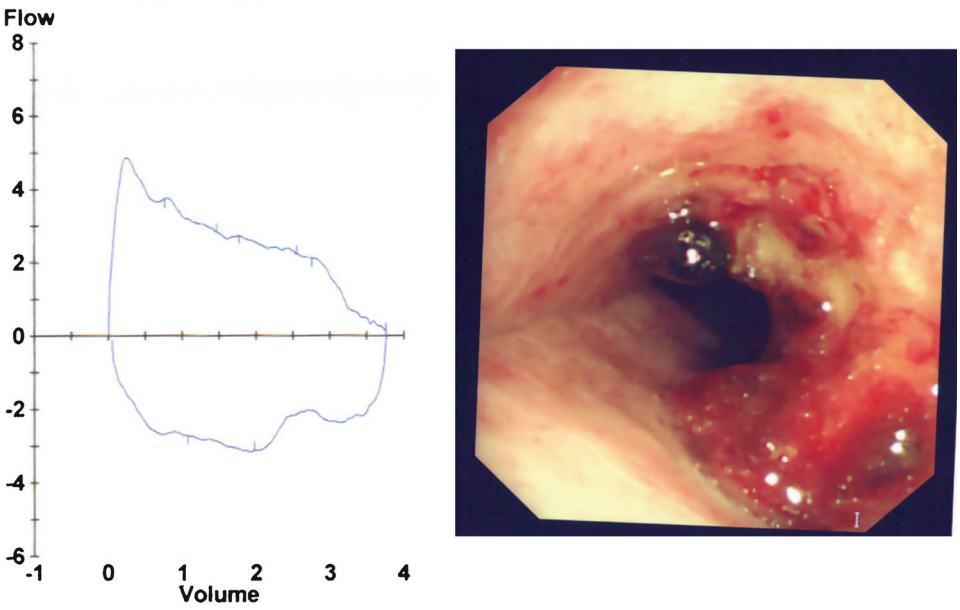

Fig. 5 A 65-year-old man with a history of locally advanced esophageal cancer presented with exertional dyspnea and stridor. Flow-volume loop and bronchoscopic view of his trachea are shown. There is fixed airway obstruction with tumor arising from the anterior and right tracheal wall. Biopsies confirmed metastatic esophageal cancer, and the patient underwent airway debridement and stenting followed by radiotherapy

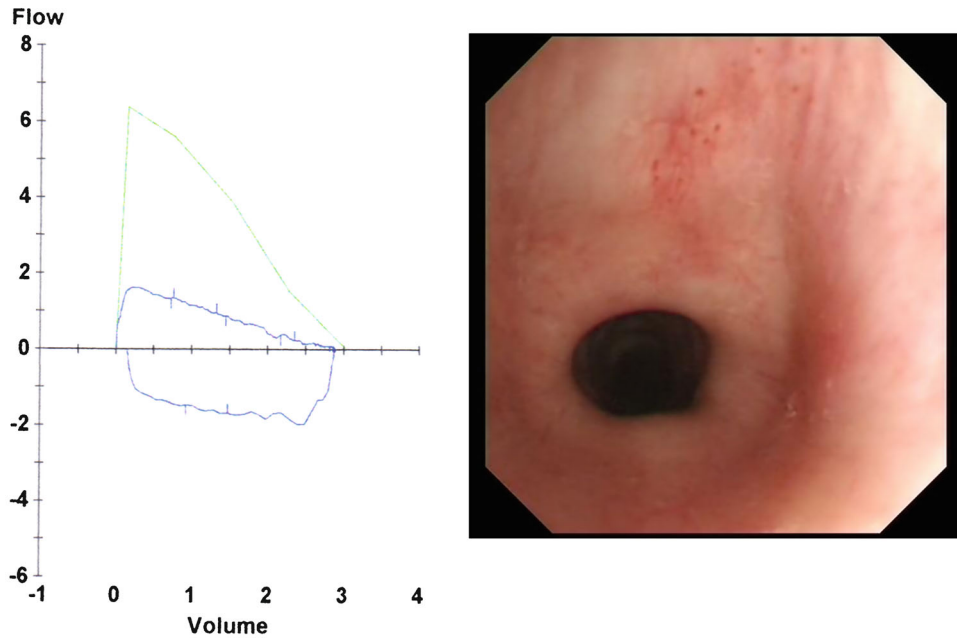

Fig. 6 A 53-year-old woman with a history of relapsing polychondritis presents with exertional dyspnea and stridor. The flow-volume loop demonstrates classical fixed airway obstruction, and bronchoscopic view shows a trapped first tracheal ring

9.2 Etiology

There are a multitude of causes of fixed upper airway obstruction, and these are listed in Table 2. Outside of malignant airway disease, a common and growing cause of fixed airway obstruction is previous intubation and tracheostomy [2]. The incidence of all types of injury to the larynx after endotracheal intubation in the past ranged from 60% to 94%, and significant tracheal stenosis occurred in up to one in five cases in the past [12]. This has thankfully been reduced in the last decade with the use of low-pressure, high-volume cuffs and the increase in use of tracheostomy where endotracheal intubation for longer than 2 weeks is required. Factors that have been found to increase the risk of laryngeal injury include laryngeal trauma during extubation, large endotracheal tube calibere, oral intubation, severe respiratory failure, diabetes, and female gender. The incidence of tracheal stenosis following tracheotomy varies, and it can occur at the site of the stoma, cuff, or tip. Severe stenosis requiring surgical intervention probably occurs in less than 5% of patients.

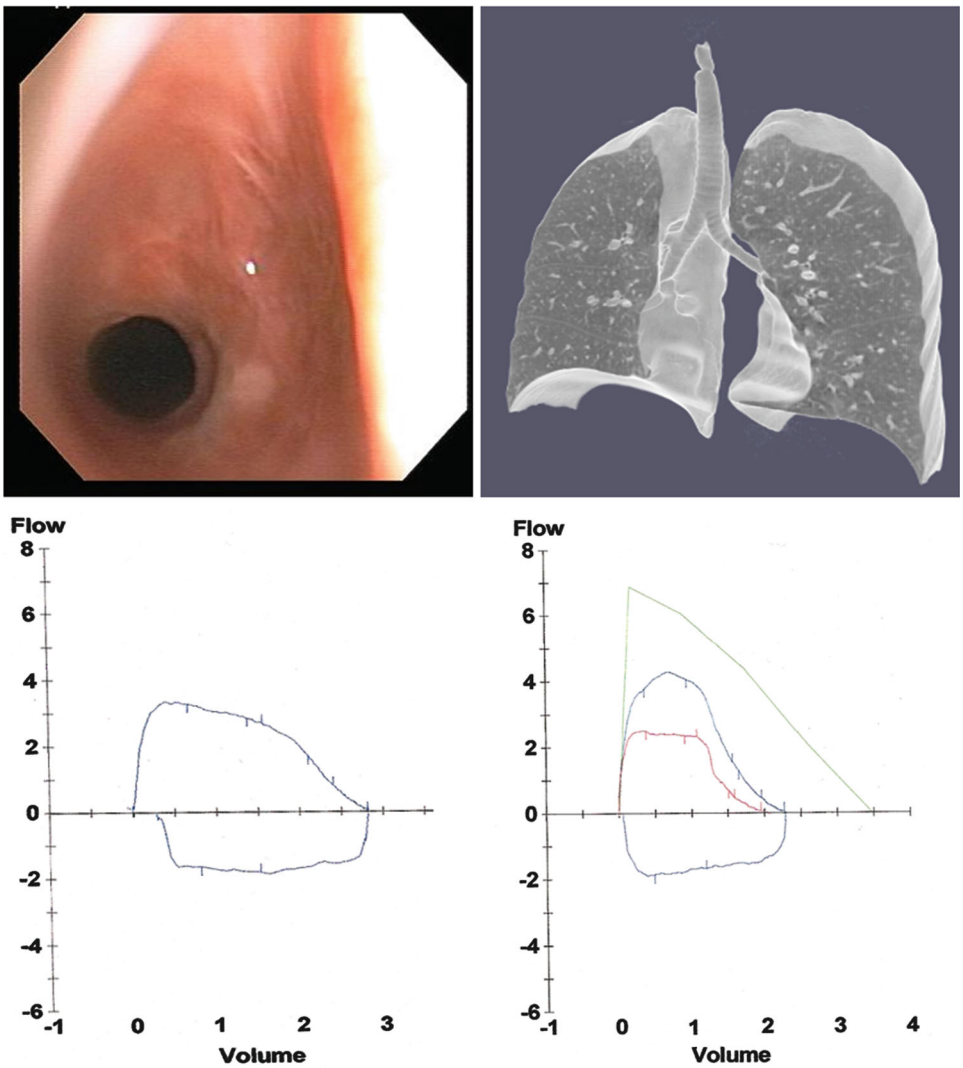

Fig. 7 A 31-year-old woman presents with idiopathic subglottic stricture. The upper airway bronchoscopic image and CT reconstruction above shows short segment of circumferential tracheal stenosis. On the bottom, the flow-volume loops before (left) and 6 weeks after bronchoscopic dilatation showing improvement in expiratory limb of the curve

9.3 Diagnosis

A complete history and physical is essential in the evaluation of the patient with suspected fixed airway obstruction, and lung function studies, as has been highlighted in this chapter, can be extremely useful. However, endoscopy with either rigid or flexible bronchoscope and additional imaging studies with computerized tomography (CT) of the neck, trachea, and thorax are usually required to further define the cause. Other reported useful imaging adjuncts in assessment of level and severity of obstruction include spiral CT, virtual bronchoscopy with multiplanar reformatting, morphometric bronchoscopy, and magnetic resonance imaging (MRI). MRI is the preferred modality in evaluating paratracheal masses in patients who have allergy to iodinated contrast material used for CT scans and, because it does not involve ionizing radiation, may also be useful for evaluating paratracheal abnormalities in children. Endobronchial ultrasound (EBUS), in particular the radial probe device, has a particular role in that it has been found to be more sensitive than CT in distinguishing tissue invasion from external compression of the airway. The 20-MHz frequency of this probe providing a resolution of less than 1 mm and the high level of structural detail of the airway wall that is provided can be instructive in treatment planning.

10 Conclusion

Fixed airway obstruction can be defined physiologically as obstruction that persists throughout the respiratory cycle. It should be suspected in any patient with upper airway symptoms of wheeze, stridor, or exertional dyspnea in particular where these symptoms have been unresponsive to conventional therapy or where a history of tracheal intubation exists. Symptoms result from turbulent airflow around the site of

obstruction causing increased airway resistance. Pulmonary function testing, especially visual inspection of the flow-volume loop, remains an effective way of detecting upper airway obstruction [16]. Plateauing or flattening of both the inspiratory and expiratory limbs during flow-volume measurement is the hallmark of fixed obstruction. Where only one limb is flattened, this suggests that the obstruction is variable in nature. Upper airway obstruction should not be confused with airflow obstruction that occurs in chronic obstructive pulmonary disease or asthma, conditions which affect smaller airways and can be discriminated from upper airway obstruction by careful examination of the flow-volume loop where characteristic patterns can be seen. Where upper airway obstruction and obstructive airway disease coexist, a number of quantitative criteria have been proposed based on pulmonary function to discriminate.

While the flow-volume loop and spirometry are useful in detecting fixed airway obstruction and are cheap, readily available and generally easy to perform, testing is volitional in nature, and it is therefore important that the technician records the patient effort, in particular during the inspiratory cycle which is often performed poorly. Overall, while very informative, tests of pulmonary function lack sufficient sensitivity for upper airway obstruction, and where suspected clinically, further assessment by bronchoscopy and airway imaging is necessary.

Competing Interest Declaration The author(s) has no competing interests to declare that are relevant to the content of this manuscript.

References

1. Acres JC, Kryger MH. Clinical significance of pulmonary function tests: upper airway obstruction. Chest. 1981;80(2):207–11.
2. Elliott CG, Rasmusson BY, Crapo RO. Upper airway obstruction following adult respiratory distress syndrome. An analysis of 30 survivors. Chest. 1988;94(3):526–30.
3. Empey DW. Assessment of upper airways obstruction. Br Med J. 1972;3(5825):503–5.
4. Handa H, Huang J, Murgu SD, et al. Assessment of central airway obstruction using impulse oscillometry before and after interventional bronchoscopy. Respir Care. 2014;59(2):231–40.
5. Jordanoglou J, Pride NB. A comparison of maximum inspiratory and expiratory flow in health and in lung disease. Thorax. 1968;23(1):38–45.
6. Melissant CF, Lammers JW, Demedts M. Relationship between external resistances, lung function changes and maximal exercise capacity. Eur Respir J. 1998;11(6):1369–75.
7. Miller RD, Hyatt RE. Obstructing lesions of the larynx and trachea: clinical and physiologic characteristics. Mayo Clin Proc. 1969;44(3):145–61.
8. Miller RD, Hyatt RE. Evaluation of obstructing lesions of the trachea and larynx by flow-volume loops. Am Rev Respir Dis. 1973;108(3):475–81.
9. Modrykamien AM, Gudavalli R, McCarthy K, Liu X, Stoller JK. Detection of upper airway obstruction with spirometry results and the flow-volume loop: a comparison of quantitative and visual inspection criteria. Respir Care. 2009;54(4):474–9.
10. Nairn JR, McNeill RS. Adaptation of the Wright peak flow meter to measure inspiratory flow. Br Med J. 1963;1(5341):1321–3.
11. Owens GR, Murphy DM. Spirometric diagnosis of upper airway obstruction. Arch Intern Med. 1983;143(7):1331–4.
12. Perry M, Santos Ali, Afrassiabi Ernest A, Weymuller. Risk factors associated with prolonged intubation and laryngeal injury otolaryngology–head and neck surgery. 1994;111(4):453–459. https://doi.org/10.1177/019459989411100411.
13. Robertson DR, Swinburn CR, Stone TN, Gibson GJ. Effects of an external resistance on maximum flow in chronic obstructive lung disease: implications for recognition of coincident upper airway obstruction. Thorax. 1989;44(6):461–8.
14. Rotman HH, Liss HP, Weg JG. Diagnosis of upper airway obstruction by pulmonary function testing. Chest. 1975;68(6):796–9.
15. Sackner MA. Physiologic features of upper airway obstruction. Chest. 1972;62(4):414–7.
16. Sterner JB, Morris MJ, Sill JM, Hayes JA. Inspiratory flow-volume curve evaluation for detecting upper airway disease. Respir Care. 2009;54(4):461–6.
17. Yernault JC, Englert M, Sergysels R, De CA. Upper airway stenosis: a physiologic study. Am Rev Respir Dis. 1973;108(4):996–1000.