ASSESSMENT OF PULMONARY AIRWAY CALIBRE

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The assessment of airway calibre is of relevance to anaesthetic practice for the following reasons. Clinical management of patients before or after anaesthesia and during intensive respiratory care often requires information about the degree of patency of airways. Following induction of general anaesthesia there may be considerable difficulty in inflating the lungs and this may be interpreted either as an increase in airways resistance or as a decrease in compliance. Clinical criteria do not enable a distinction to be made between these two entities. When measurements of resistance are made during general anaesthesia we have shown (Lehane, Jordan and Jones, 1980) that large changes occur which are undetectable clinically. Many drugs used in anaesthetic practice may influence airway calibre and methods are required for evaluating these effects in both conscious and anaesthetized patients.

PHYSIOLOGICAL BACKGROUND

In reviewing the methods available for the assessment of airway calibre it is first necessary to understand the mechanisms which determine static airway dimensions and the ways in which gas flow modifies them.

The static dimensions of the pulmonary airways, that is their dimensions when there is no gas flow, are determined by the interaction of the elastic recoil of the lungs and the elastic properties of the airways. When gas flows along the airways pressure gradients are introduced which tend to distend or compress the airways from their static dimensions.

Elastic recoil

The elastic recoil of the lungs can be demonstrated by opening the pleural cavity and noting that the lung recoils away from the parietal pleura, expelling air through the trachea as it does so. This recoil is opposed by occluding the trachea before opening the pleural cavity, the pressure in the closed airways then being equal to lung recoil pressure.

The transpulmonary pressure (\(P_{TP}\), the pressure gradient between airway and pleural space) is the force which opposes elastic recoil and is the force therefore which maintains lung volume. If there is no gas flow \(P_{TP}\) balances lung recoil pressure exactly.

The elastic recoil forces in the lungs exert traction on all structures within the lungs, pulling on airway surfaces and maintaining their patency. Lung recoil pressure varies with the volume of the lungs (fig. 1) and there are also important regional variations of recoil pressure within the lungs at any given volume. This latter effect is caused by stresses produced in the lungs which distort them from the shape they would adopt outside the thorax to the shape they must assume within the thorax (Agostoni, 1972). In consequence recoil pressure is smaller and airways narrower in the dependent zones of the lungs than in the upper zones.

Airway elasticity

The elasticity of the airways may be determined by measuring change in length and diameter of bronchial segments in an excised lung at different volumes. Hughes, Hoppin and Mead (1972), using this technique, have shown that the elastic properties of the intrapulmonary airways are similar to those of lung parenchyma. The compliance (a measure of elasticity) of the airways is close to that of the

![Diagram 1](https://example.com/diagram.png)

**FIG. 1.** Diagrammatic representation of the relationship between lung volume and airway size. The arrows represent lung recoil which is large at total lung capacity (TLC) and very small at residual volume (RV).

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whole lung over the vital capacity range, and this appears to be true both of the bronchi, which have cartilaginous support, and of the peripheral airways (Hughes, Hoppin and Mead, 1972). In consequence, the volume of the airways, containing the anatomical deadspace, is proportional to lung volume and an approximately linear relationship exists between lung recoil pressure and airway volume.

The activity of bronchial smooth muscle adds another element to the relationship between lung volume and airway dimensions. Smooth muscle contraction has the effect of stiffening the airways so that an increase in lung volume produces a smaller increase in airway dimensions and so airway calibre at each lung volume is less than it would be in the absence of bronchomotor activity.

Regional variations in recoil pressure produce regional variations in airway calibre although these have little effect on the distribution of ventilation except at small lung volumes when closure of dependent airways takes place.

Effect of gas flow

When there is no gas flow the same pressure prevails throughout the bronchial tree. The distending force on the airways is the elastic recoil pressure, and static airway dimensions are determined by the factors described above (fig. 2A). Gas flow produces a pressure gradient down the airways which depends on the rate and direction of airflow and the resistance offered by the airways. During inspiration the pressure gradient is relatively small because pleural pressure becomes more subatmospheric than it would be under static conditions and there is a slight increase in airway diameter which is greatest in the airways nearest the mouth (fig. 2B). During expiration the pressure gradient is reversed and the airways nearer the mouth become narrower than their static dimensions (fig. 2C). This is called dynamic compression of the airways and in consequence of this the expiratory resistance exceeds inspiratory resistance at the same lung volume.

![Factors affecting airway diameter](https://academic.oup.com/bja/article-abstract/54/7/751/308140/fig2)
When expiratory airflow is increased, and even with a normal flowrate in patients with pathologically narrowed airways, dynamic compression is increased. The patient may need to produce a pleural pressure which is above atmospheric in order to generate the necessary flowrate and this pressure will compress the airways downstream from the point at which intrabronchial pressure is equal to intrapleural pressure (the equal pressure point—fig. 2D). This mechanism limits the maximum expiratory flow and is said to cause "gas trapping" (Pride, 1971). It should be realized that gas is not permanently trapped by this mechanism because if expiratory effort is maintained long enough the gas will eventually be expired. When the gas flow stops this mechanism ceases, and airway dimensions assume the static dimensions appropriate to that lung volume (fig. 2A). Gas trapping by dynamic airways compression does not, therefore, account for an increase in residual volume, such as occurs in emphysema, as this volume is defined as a static lung volume (Cumming and Semple, 1980) at which there is no gas flow and no pressure gradient within the lumen of the airways.

METHODS OF ASSESSMENT OF AIRWAY CALIBRE

Airway calibre in man is usually assessed by measurements of gas flow rate under standardized conditions (that is, during forced expiration), or by measurements of both driving pressure (P) and gas flow (V) to obtain the total resistance (R) to airflow (R = P/V). In contrast, an index of regional variations in airway calibre can be derived by measurement of closing volume.

Measurements of flow during forced expiration were introduced by Tiffeneau and Pinelli in 1947, who recorded volume change v. time (fig. 3A). More recently an alternative method of analysis, the maximum expiratory flow-volume (MEFV) curve (Hyatt, Schilder and Fry, 1958; Hyatt and Black, 1973), has gained widespread use (fig. 3B).

These tests ignore the force applied by the subject, that is the driving pressure, and it was originally believed that they must, therefore, be inferior to measurements of resistance. It became apparent, however, that the results were usually reproducible because variations in driving pressure only affected the first 20–30% of the expired volume. The explanation for this lies in the mechanism shown in figure 2D. Increasing expiratory effort increases pleural pressure (P_PL) and this opposes the effects of lung recoil pressure on central airways and tends to compress them. The net result is that an increase in P_PL produces an increase in expiratory resistance and this increase is such that, once P_PL exceeds central airway pressure, no further increase in expiratory flowrate can occur. Under these conditions expiratory flowrate is largely independent of effort.

**Fig. 3.** The two methods of recording the forced expiratory manoeuvre. A: As change in expired volume v. time (spirogram) from which are obtained the forced expiratory volume in 1 s (FEV₁) and the forced vital capacity (FVC). B: As change in maximum expiratory flowrate v. change in volume (MEFV curve). Starting from total lung capacity (TLC), expiratory flow increases to a peak value (PEF) and then declines approximately linearly down to residual volume (RV). Maximum flow when 50% and 25% of the FVC (V_max 50, V_max 25) remains to be expired are read from the curve. Reproduced by kind permission of the Author and the Editor of *Br. J. Clin. Pharmacol.*, from Pride (1979)
Three factors govern the flowrate under these conditions:

Recoil pressure. The greater the recoil pressure the greater is the change in $P_{PL}$ before flow limitation occurs and thus the greater is the flowrate. In emphysema lung recoil may be greatly diminished and flow limitation may occur under normal resting expiratory flowrates, whereas in pulmonary fibrosis recoil pressure and flow are both increased.

Bronchomotor tone/bronchial elasticity. If airways are narrowed by abnormal contraction of bronchial smooth muscle, a greater driving pressure than normal is produced on expiration and $P_{PL}$ reaches values at which flow limitation occurs at lower flowrates than normal.

Mechanics of the compressed airways. In the segments of the airways in which flow limitation occurs, gas velocity is very high and this causes a reduction in pressure by the Bernoulli effect, which further collapses the airways. In consequence increasing expiratory effort may produce a reduction in flow which is referred to as negative effort dependence. The existence of negative or positive effort dependence or true effort independence under conditions of flow limitation thus depends on the shape of the airway compliance curve in the region of the equal pressure point (Jones, Fraser and Nadel, 1975). As lung volume decreases during expiration the equal pressure point tends to move upstream towards airways that have mechanical properties which produce progressively lower flowrates. Under these conditions measurements reflect dynamic airway dimensions and are heavily dependent on pressure gradients produced by air flow.

Measurements made at slower flowrates, similar to those occurring during quiet breathing, reflect airway dimensions which more closely approximate to static dimensions. Because flow limitation does not usually occur under these conditions it is necessary to determine the driving pressure and so obtain airways resistance ($R_{AW}$). $R_{AW}$ not only reflects intrapulmonary airway dimensions, but also contains a large component derived from the larynx and upper airway which is a major source of variability. Furthermore, because airflow is not truly laminar, particularly in the upper airway and larynx, resistance increases with increasing flowrate and it is therefore necessary to specify the flowrate at which resistance is measured.

Measurements are usually made to find out about the state of the airways within the lung, and in particular about the state of activity of bronchial smooth muscle. The next section reviews commonly used methods.

Peak expiratory flow rate (PEFR)

This may be measured with a simple variable orifice meter (Wright and McKerrow, 1959; Wright, 1978) and is suitable for general practice and domiciliary use even by unsupervised patients. Peak flow occurs near total lung capacity (fig. 3B) and depends not only on recoil pressure and airway elasticity but is also very dependent on the expiratory effort.

Forced expiratory volume in one second (FEV₁)

Measurements of the volume expired in 1 s, and the forced vital capacity (FVC), are commonly made, using a recording spirometer such as the "Vitalograph" (fig. 3A). Usually more than 50% of the FEV₁ is expired during the "effort-independent" phase of the forced expiration. Effort-dependence is less than with PEFR and therefore measurements more closely reflect the effects of recoil pressure and airway elasticity. Reduced chest wall or lung compliance, such as in obesity or pulmonary fibrosis, may limit FVC to the point at which FEV₁ is reduced even in the absence of airways obstruction and so FEV₁ is frequently standardized for FVC by expressing it as the ratio FEV₁/FVC.

When measurements are made before and after bronchodilator therapy it is usual to assume that any increase in FEV₁ signifies a reduction in bronchomotor tone, rather than an increase in recoil pressure. Airways obstruction reversible by bronchodilator therapy is therefore evidence of hyperreactivity of bronchial smooth muscle (that is, bronchial asthma). While FEV₁ measurement assists both in diagnosing asthma and in assessing its severity, it should be noted that reduction in FEV₁ is a poor guide to the course of anaesthesia in such patients. In one series 6.5% of symptom-free asthmatic patients developed asthmatic attacks during anaesthesia, while in those with symptoms of asthma, anaesthesia appeared to increase obstruction in 10% and reduce it in 37% (Shnider and Papper, 1961).

In patients with chronic obstructive lung disease the reduction in FEV₁ is only slightly, if at all, reversed by bronchodilator therapy. Measurements of FEV₁ do not distinguish between obstruction resulting from airway disease and obstruction caused by decreased recoil pressure, although both these may co-exist. Again, FEV₁ is a poor guide to
AIRWAY CALIBRE

the course of anaesthesia, but if it is less than 1 litre then the patient should have blood-gas analysis, and may benefit from chest physiotherapy before and after operation. Arterial carbon dioxide tension is a better guide and those patients who have an increased $P_{\text{aco}}$, are likely to require ventilatory support in the period after surgery (Milledge and Nunn, 1975).

Maximum expiratory flow–volume curve

The MEFV curve is usually obtained by measuring flow at the mouth with a pneumotachograph and integrating the flow signal electronically to obtain the change in lung volume. From the curve it is possible to measure PEFR, FVC and FEV₁ (if timing marks are displayed on the curve). The combined effects of recoil pressure and bronchomotor tone are inferred from the MEFV curve in the same way as from the FEV₁. The shape of the MEFV curve, particularly the effort-independent part of the curve, yields additional information relating to the site and nature of the obstructing lesion. Maximum flow at 50% VC ($V_{\text{max 50}}$) and 25% VC ($V_{\text{max 25}}$) serve to characterize the effort-independent part of the curve (fig. 3B).

In patients with narrowing of the peripheral airways as a result of chronic obstructive lung disease or asthma, the effort-independent part of the curve is usually concave upward (fig. 4A). A localized obstruction in the trachea, such as tracheal stenosis or thyroid compression, produces a characteristic flat-topped curve (fig. 4B) with maximum flowrate over most of the curve being highly effort-dependent. These changes in the shape of the curve reflect differences in the mechanical properties of the flow-limiting segments. The MEFV curve obtained using a helium–oxygen mixture may be compared to that obtained with air. In asthmatic patients little difference is observed as gas flow is limited in the constricted peripheral airways where flow is predominantly laminar. In patients with localized tracheal obstruction flow resistance is markedly density-dependent at higher flowrates. The characteristic shape of the curve is preserved in these patients, but PEFR is increased and this is reflected in the observation that helium–oxygen mixtures are clinically useful in relieving such localized obstruction, while they are of no benefit in asthma.

Resistance measurement during normal ventilation

Resistance may be measured by simultaneously recording gas flowrate at the mouth, volume in-
subtracting a pressure proportional to volume from the total measured pressure (fig. 5). Resistance is calculated by dividing the driving pressure by the measured flowrate (Otis, Fenn and Rahn, 1950; Mead and Whittenberger, 1953). Depending on the sites at which pressure is measured (transpulmonary or transthoracic), the calculated resistance is pulmonary ($R_L$) or respiratory resistance ($R_{RS}$) (table I). Transthoracic pressure measurements only reflect driving pressure if the lungs are passive-

### Table I. Subdivisions of resistance to airflow and their relationship to pressure sampling points. Typical resistance values are shown in parentheses (kPa litre$^{-1}$ s$^{-1}$).

<table>
<thead>
<tr>
<th>Pressure sampling point</th>
<th>Resistance obtained (with typical values, kPa litre$^{-1}$ s$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outside thorax</td>
<td>Chest wall (0.08)</td>
</tr>
<tr>
<td>Pleura</td>
<td>Lung tissue (0.01)</td>
</tr>
<tr>
<td>Alveoli</td>
<td>Lower airways (0.06)</td>
</tr>
<tr>
<td></td>
<td>Pulmonary (0.13)</td>
</tr>
<tr>
<td>Trachea</td>
<td>Airway (0.12)</td>
</tr>
<tr>
<td>Mouth</td>
<td>Upper airway (0.06)</td>
</tr>
</tbody>
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These methods are suitable for use during anaesthesia, and if the trachea can be intubated, a major source of variability, the resistance of the larynx and upper airway, can be eliminated. Interpretation of results poses problems, however, as recoil pressure can vary between successive measurements as a result of changes in lung volume associated with anaesthesia. Unless lung volume is measured, and shown not to account for the observed changes in resistance, then the changes in resistance cannot safely be ascribed to changes in bronchomotor tone.

### Airway resistance using body plethysmography

If a subject is seated inside an airtight box, and breathes to atmosphere through a port connected to the exterior of the box, then the box pressure changes proportionately to the change in the lung volume. This is because movement of the body surface during respiration displaces a volume of gas equal to the volume displaced by the lung. If the port is sealed and the subject breathes the air within the box, then it might be expected that there would be no pressure changes in the box as the change in trunk volume should exactly equal the volume inspired and expired at the mouth. During gas flow this is not quite the case as the changes of pressure in the alveoli cause compression or expansion of gas in the lungs. This causes a small change in trunk volume which is not balanced by an equal volume displaced at the mouth. For example, if the trunk volume decreases by 52 ml while 50 ml is expelled from the mouth, then 2 ml of gas is lost to the box by gas compression within the lungs. If the actual lung volume is known, then Boyles' Law can be used to

**Fig. 5.** Pressure, flow and volume recorded during a spontaneous inspiration. Compliance ($\Delta V/\Delta P$) is used to calculate $P_{el}$ which is the pressure required to overcome elastic recoil (i.e. proportional to volume change). $P_{tot}$ is the total applied pressure (i.e. $P_{el}$ plus the pressure required to overcome resistance, $\Delta P_r$). Reproduced by kind permission of the Author and Blackwell Scientific Publications Ltd, from Sykes, McNicol and Campbell (1976)
calculate alveolar pressure. Thus, in the above example, if lung volume is 2 litre and absolute pressure at the mouth is 100 kPa then:

$$\text{alveolar pressure} = \frac{100 \times 2000}{2000 - 2}$$

= 100.1 kPa

Alveolar pressure is 0.1 kPa (1 cm H$_2$O) greater than mouth pressure. If flowrate is measured at the mouth, then airways resistance can be measured (DuBois, Botelho and Comroe, 1956). In practice, box pressure changes are calibrated in terms of alveolar pressure changes, with the subject making respiratory efforts against an occluded airway. Alveolar pressure is obtained by measuring pressure at the mouth as, with the airway occluded, mouth pressure equals alveolar pressure.

Because this method requires the subject to be enclosed in a box and to make active respiratory efforts, it is unsuitable for use during anaesthesia.

**Resistance using the forced oscillation method**

An alternating airflow (typically 50 ml tidal volume at 3 Hz) is applied at the mouth using a loud-speaker or a mechanical pump connected to the breathing circuit. The oscillating flow and pressure are measured at the mouth. The points of peak inspiratory and expiratory flow produced by the oscillations are determined and the pressure difference between successive instants of peak flow is measured. Lung volume is identical at these points and the pressure difference observed is the resistive pressure across the respiratory system at that peak-to-peak flowrate (fig. 6). From these pressure and flow measurements respiratory resistance is obtained (Goldman et al., 1970; Hyatt et al., 1970). The frequency of oscillation should be sufficiently slow in relation to the time constants of the alveoli and their conducting airways so that the distribution of the oscillating airflow is reasonably uniform throughout the lungs. Frequencies up to 10 Hz are satisfactory under normal conditions in subjects with healthy lungs. With less homogenous lungs, a frequency limit of about 3 Hz is desirable if $R_{RS}$ is not to be overestimated. This method is suitable for use in anaesthetized patients, although the problem remains of distinguishing between changes resulting from change in bronchomotor tone and those caused by change in lung volume.

**Measurement of closing volume**

Measurement of closing volume provides information about regional airway calibre. To obtain the closing volume it is necessary to arrange for there to be different concentrations of a tracer gas in the upper and lower zones of the lungs. This may be achieved by inhaling a small volume of an inert tracer gas (such as argon or xenon-133) from residual volume, followed by air to total lung capacity. Because the tracer gas is introduced when dependent airways are closed, its concentration is greatest in the upper zones of the lungs. The patient then slowly exhales and the tracer concentration is measured at the mouth. When the airways in the dependent parts of the lungs begin to close again, the expired gas shows a sudden increase in tracer concentration as a greater fraction of expired gas comes from the upper zones where tracer concentration is greatest. The point at which tracer concentration in the expired air increases above the alveolar plateau signals the onset of airways closure (fig. 7). The volume expired from this point to residual volume is the closing volume of the lungs (Dollfus, Milic-Emili and Bates, 1967).

Closing volume increases with either an increase in bronchomotor tone or a decrease in recoil pressure. However, the presence of lung disease often converts the alveolar plateau into a curve on which there is no clear cut inflection in tracer concentration to enable the closing volume to be measured.

**AIRWAY FUNCTION DURING ANAESTHESIA**

Anaesthesia is usually associated with a reduction in functional residual capacity (FRC) and thus with a reduction in both recoil pressure and airway calibre.
Different anaesthetic agents may influence the extent of the reduction in lung volume and therefore have differing effects on airway calibre through mechanisms other than change in bronchomotor tone. Under these circumstances resistance measurements must either be repeated at identical lung volumes, or alternatively the way in which resistance varies with lung volume must be accurately established.

Given that gas flow within the lungs is predominantly laminar at low flowrates, it can be shown that the relationship between airways resistance and lung volume is hyperbolic (Jordan et al., 1981) and this relationship has been demonstrated in conscious subjects (Briscoe and DuBois, 1958) and in anaesthetized patients (Lehane, Jordan and Jones, 1980). The effect of a change in bronchomotor tone is a change in the shape of the hyperbolic curve (fig. 8). This change in shape may be quantitated in two closely related ways:

Specific airways conductance

From the hyperbolic relationship between resistance and volume it may be expected that the reciprocal of resistance, conductance, should be a linear function of lung volume (fig. 9). Blide, Kerr and Spicer (1964), using a body plethysmograph, and a wide-bore needle to sense tracheal pressure, showed that there was, indeed, a linear relationship between the conductance of the airways in the lung ($G_{LAW}$) and lung volume.

Change in bronchomotor tone affects the slope of this straight line relationship and this provides the basis for the standard plethysmographic method for specific conductance. $R_{AW}$ is measured as described earlier, and its reciprocal, airways conductance ($G_{AW}$) is divided by lung volume (also obtained during the measurement of $R_{AW}$ in the plethysmograph). This ratio is called specific airways conduc-
This is not an adequate model for the data for two reasons. First, some components of resistance are dependent of lung volume (lung tissue, chest wall and upper airway resistance) and these can be included in a more complete equation as the term $\rho_A$ (equation (2)). Second, all airways would close, so their resistance would be infinite, if the lungs were to be deflated to residual volume. A further term (ERV) representing this volume is also included in equation (2) thus:

$$\frac{1}{R} = s.G_{LAW} \times V_L$$  \hspace{1cm} (1)$$

$$\frac{1}{R - R_A} = s.G_{LAW} (V_L - ERV)$$  \hspace{1cm} (2)$$

This is represented graphically in figure 10. Hyperbolic regression determines the parameters ($s.G_{LAW}$, $R_A$ and ERV) of equation (2) that best fit the resistance–volume data and change in $s.G_{LAW}$ is used as an index of change in bronchomotor tone.

This approach makes it possible to distinguish between the effects of lung volume and bronchomotor tone on $R_{RS}$ in anaesthetized, mechanically ventilated patients. Figure 11 shows the effects of the addition of halothane to the inspired gas mixture in two anaesthetized patients. In both patients this produced a reduction in bronchomotor tone illustrated here by the change in shape of the resistance–volume curves. In patient 8 (fig. 11B) the resistance over the tidal volume range is even further reduced by a concomitant change in lung volume.
which is shown by the increase in ERV. The mechanism of this change is not clear, but may be a result of a reduction in central blood volume caused by peripheral vasodilatation (Lehane, Jordan and Jones, 1980). As ERV may be very small in some anaesthetized patients it can be seen that small changes in lung volume may produce gross changes in $R_{RS}$. The great variability in published data on the effects of anaesthetics on bronchomotor tone is probably a result of failure of workers to allow for the effects of change in lung volume.

**CONCLUSION**

Various methods are available for the assessment of airway calibre. For the assessment of patients before operation the simpler tests of forced expiration usually suffice. When more sensitive methods are required, usually for research purposes, then analysis of the maximal expiratory flow–volume curve or plethysmographic determination of specific conductance may be appropriate. With suitably trained subjects the forced oscillation method for $s.G_{LAW}$ is a useful alternative to the plethysmographic method. During anaesthesia and in patients requiring mechanical ventilation on the Intensive Therapy Unit, the forced oscillation method for $s.G_{LAW}$ is probably the only practical method available which

![Diagram](https://example.com/diagram.png)

**Fig. 10.** Diagram of resistance and conductance curves to show $R_A$, ERV and $s.G_{LAW}$ (see text)

![Graph](https://example.com/graph.png)

**Fig 11 A:** The effect of halothane in one patient to show a small change in ERV and a large change in respiratory resistance. The decrease in resistance over the tidal range is almost entirely a result of reduction in bronchomotor tone. **B:** A different patient illustrates another mechanism for a decrease in respiratory resistance following addition of halothane. The decrease in resistance is accounted for largely by an increase in ERV, although there is an independent reduction in bronchomotor tone.
yields data on airway calibre which can be reliably interpreted in terms of effects of bronchomotor tone and of recoil pressure.

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