Movement of any flow (\( \dot{V} \)) through a pipe requires a driving pressure to overcome frictional resistance. The magnitude of flow depends on the difference in pressures (\( \Delta P \)) across the pipe and the resistance (\( R \)) offered by the pipe itself [1]:

\[
\dot{V} = \frac{\Delta P}{R}
\]  

(1)

Flow resistance is proportional to the length (\( l \)) of the pipe and varies inversely with the fourth and fifth power of the radius (\( r \)) for laminar and turbulent flow, respectively, as described by the Poiselle's law:

\[
R = \frac{\eta \cdot l \cdot 8}{\pi r^4}
\]  

(2)

where \( \eta \) represents the viscosity of the gas and \( 8/\pi \) is a constant.

It is generally accepted that, in normal subjects, airway resistance is linear during quiet breathing and can be expressed as a single number, according to Eq. 1 in general, about 2-4 cm H\textsubscript{2}O sl\textsuperscript{-1}. However, airway resistance (Raw) is only 1 component of total respiratory system's resistance (Rrs), which also includes tissue resistance of the lung (RTL), thus giving total pulmonary resistance (RL), and chest wall resistance (RW) (discussed below) [2]. The resistive characteristics of the respiratory system were first described by Rohrer with the equation:

\[
Pre_s = K_1 \dot{V} + K_2 \dot{V}^2
\]  

(3)

where \( K_1 \) and \( K_2 \) are constants. Another equation has also been used:

\[
Pre_s = a \dot{V}^b
\]  

(3a)

where \( Pre_s \) is the resistive pressure drop, \( a \) is the value of \( Pre_s \) at \( \dot{V} \) of 1 l/s and \( b \) is a dimensionless index describing the shape of the pressure-flow relationship—concavity and convexity to the \( \dot{V} \) axis are represented by \( b<1 \) and \( b>1 \), respectively. When both components of Eq. 3 are divided by \( \dot{V} \) (see also Eq. 1), the result is:

\[
R = K_1 + K_2 \dot{V}
\]  

(4)

This is a first degree equation, where \( K_1 \) represents the value of resistance.
extrapolated to zero and $K_2$ represents the slope of the increase in $R$ with increasing flow ($\dot{V}$). Eq. 4 is the basis for one of the tenets of respiratory mechanics, namely that, at a given lung volume resistance should increase with increasing flow. Another basic tenet is that at a given flow, resistance should decrease with increasing lung volume because of a decrease in airway resistance due to bronchial dilatation. Assuming that the flow-dependent pressure losses within the thorax are also reduced because the linear velocity of thoracic tissues decreases with increasing lung volume, total respiratory system resistance ($R_{rs}$) should decrease with increasing volume, thus Eq. 4 becomes:

$$R_{rs} = R_t + K_1 + K_2 \dot{V}$$  \hspace{1cm} (5)

where $R_t$ represents thoracic tissue resistance. Equation 5 assumes that the thoracic tissues exhibit ohmic (Newtonian) behaviour. However, a recent series of studies on respiratory mechanics have shown that this is not the case. In fact, $R_t$ measured during constant flow inflation from relaxed functional residual capacity (FRC) is not constant but increases with the duration of inspiration ($T_i$) and decreases progressively with increasing flow at fixed lung volume. As a consequence, $R_{rs}$ initially decreases with increasing flow as a result of the stress relaxation until at high flow the term $K_2$ becomes predominant.

### Endotracheal tubes

In ventilator-dependent patients, a significant component of the total flow resistance is provided by endotracheal tubes, which have highly curvilinear flow-resistive properties [3]. In normal subjects, flow is laminar during tidal ventilation and becomes turbulent only with increasing ventilatory demands. As a result, Eq. 1 has been commonly used when assessing normal breathing. By contrast, in intubated patients, Eqs. 3 and 3a have been used to describe their pressure-flow characteristics. The flow resistance offered by the endotracheal tubes increases markedly with increasing flow and varies with the size of the tube.

Flow resistance is increased in all patients with acute respiratory failure (ARF) due to airway diseases, such as asthma or an exacerbation of COPD. Abnormal flow resistance has been also found in some patients with acute respiratory distress syndrome (ARDS) and pulmonary oedema, particularly in the early stages [4]. Various explanations have been offered for this finding including: 1) inflammatory fluid and cellular debris in the bronchial lumen; 2) reflex bronchoconstriction; 3) decreased FRC; and 4) reduction in the number of ventilating airways. Some authors have reported essentially normal values of airway resistance in ARDS patients, although total respiratory system resistance may be increased. Bronchodilator-induced smooth muscle relaxation decreases flow resistance in ventilator-dependent patients with ARDS [5]. This suggests that increased tone of bronchial smooth muscle also plays a role. Total flow resistance includes the resistance of the endotracheal tube, which can be greater in vivo than in vitro due to secretions in the lumen, or kinking and...