The role of equal pressure points in understanding pulmonary diseases

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The respiratory system is composed of a conducting part and a respiratory part. The conducting airways, i.e., the trachea, bronchi, and bronchioles, differ in architecture. Both trachea and bronchi are surrounded by rings of hyaline cartilage for support, whereas the bronchiolar wall does not contain any cartilage and therefore tends to collapse in response to changes in intrapleural or airway pressure. As a result of airflow resistance, pressure that is generated in the alveoli drops along the airways during expiration (1, 3). This is also known as friction loss. When airflow pressure has dropped to a level where it equals intrapleural pressure during forced expiration, an equal pressure point (EPP) is reached (4). At the EPP, airways that are not supported by cartilage will collapse (Fig. 1). In healthy lungs, the EPP will be reached in cartilaginous airways as a result of sufficient alveolar driving pressure and only a gradual drop in pressure due to minimal airway resistance (4). In the case of (severe) airway obstruction, resistance to airflow will be much greater, and the pressure drop will be much steeper. The EPP will move upstream toward the alveoli and will be reached in the thin-walled bronchioles, causing airway collapse and the typical depression in the flow-volume curve (2, 3).

Although the clinical importance of EPPs, especially in obstructive airway diseases, is unquestionable, relatively little effort has been put into a comprehensive description of the dynamics involved in this phenomenon. This is unfortunate, because correctly applying complex physics in a clinical context can be a difficult task. Therefore, this report aimed to provide a straightforward mathematical approach to the concept of EPPs in the airways by combining important principles of airflow dynamics in a conceivable way. This results in an equation (Eq. 1) that can be used to get a better understanding of how airflow and airway properties alter the tendency of airways to collapse:

\[ L = \frac{Tr^2}{4\eta R_v} \]  

where \( L \) is length, \( T \) is wall tension, \( r \) is the cross-sectional radius of a segment, \( \eta \) is viscosity, \( R \) is the alveolar radius, and \( v \) is velocity.

The derivation of Eq. 1 will be discussed stepwise in this article. Table 1 shows a glossary of the parameters.

Mathematical approach. Alveolar pressure (\( P_{alv} \)) is the result of recoil pressure (\( P_{rec} \)), created by expansion of the alveoli, and intrapleural pressure (\( P_{pl} \)) and produces the driving force for airflow through the respiratory system (2–4). According to Pascal’s law, pressure will be dispersed equally in all directions throughout the enclosed intrapleural cavity. Therefore, \( P_{pl} \) exerted on the alveoli will equal \( P_{pl} \) exerted on the airways. Assuming that the alveoli are perfect spheres, Laplace’s law (Eq. 2) can be used to describe \( P_{rec} \) in terms of \( R \) and \( T \), which is the result of lung tissue elasticity and surface tension (6):

\[ P_{rec} = \frac{2T}{R} \]  

The equation for \( P_{alv} \) (Eq. 3) can now be written as follows:

\[ P_{alv} = P_{pl} + P_{rec} = P_{pl} + \frac{2T}{R} \]  

As air flows during expiration, pressure inside the airways will drop as a result of friction loss. The relationship between the pressure drop (\( \Delta P \)), resistance to airflow (\( R_{AW} \)), and airflow (\( \Phi \)) is given by Poiseuille’s law (Eq. 4), which can be applied to the laminar flow of Newtonian fluids (4, 6). Airflow becomes more turbulent as flow velocity and airway diameter increase. Therefore, larger airways are much more prone to turbulent flow than bronchioles (5). To apply Poiseuille’s law, laminar flow is assumed in the smallest airways in this model, although even in bronchioles airflow will not be entirely laminar during forced expiration. \( R_{AW} \) depends on \( \eta \), the length of the airway segment through which air flows (\( L \)), and the cross-sectional radius of this segment (\( r \)):

\[ \Delta P = R_{AW} \Phi = \frac{8\eta L}{\pi r^4} \Phi \]  

The continuity equation for \( \Phi \) (Eq. 5) essentially states that what goes in at one end of the airway segment must come out at the other end. The volume of air (\( \Delta V \)) that passes a certain point per unit of time (\( \Delta t \)), i.e., the flow, will not change.

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This result (Eq. 6) can be combined with Poiseuille’s law (Eq. 4) as follows:

\[
\Phi = \frac{\Delta V}{\Delta t} = A \frac{\Delta r}{\Delta t} = A v = \pi r^2 v = \text{constant} \quad (5)
\]

This result (Eq. 6) can be combined with Poiseuille’s law (Eq. 4) as follows:

\[
\Delta P = \frac{8 \eta L}{\pi r^4 \pi r^2 v} = \frac{8 \eta L v}{r^2} \quad (6)
\]

As mentioned above, an EPP will arise where airway pressure has dropped to a level where it equals \( P_{pl} \) (4). Airway pressure at distance \( L \) from the alveoli is defined as \( P_{alv} \) minus \( \Delta P \) over \( L \), as follows:

\[
P_{alv} - \Delta P = P_{pl} + \frac{2T}{R} - \Delta P = P_{pl} \quad (7)
\]

Because \( P_{pl} \) on the left side of the equation is the same as on the right side of the equation (Pascal’s law), the EPP will occur where \( P_{rec} \) equals friction loss:

\[
\frac{2T}{R} = \Delta P = \frac{8 \eta L v}{r^2} \quad (8)
\]

Rearranging Eq. 8 produces the relationship between the distance from the alveolus to the EPP (\( L \)) and the factors that influence it:

\[
L = \frac{Tr^2}{4\eta R v}
\]

Discussion and conclusions. This report provides a comprehensible mathematical approach to the dynamics of a pulmonary EPP. The equation that has been derived in this article (Eq. 1) shows that as the value of \( L \) decreases, the EPP moves away from the cartilaginous airways toward the alveoli, causing an obstruction to airflow as soon as the EPP moves to a collapsible region of the airway. This can be illustrated by the altered airflow dynamics in a number of pulmonary diseases (3, 4). In asthma and chronic obstructive pulmonary disease, for instance, airway inflammation reduces \( r \) (increasing \( R_{AW} \)) and therefore reduces the distance from the alveolus to the EPP. The same effect on \( L \) can be seen in emphysema, as a result of the decrease in \( T \) and an increase in \( r \), due to the disintegration of alveolar septa. Conversely, pursed lip breathing (which is commonly used instinctively by persons suffering from chronic obstructive pulmonary disease) decreases \( v \), increasing the airway pressure, which moves the EPP downstream toward the cartilaginous airways, preventing collapse.

Taken together, it is important to note that it is not the aim of this equation to calculate EPP positions exactly, and it should not be used for this purpose. Rather, the derived equation should be considered a means for students or clinicians to get a better understanding of how (changes in) relevant parameters influence the tendency of airways to collapse in pulmonary disease.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: P.V. conception and design of research; P.V. analyzed data; P.V. and H.v.H. interpreted results of experiments; P.V. prepared figures; P.V. and H.v.H. drafted manuscript; P.V. and H.v.H. approved final version of manuscript; H.v.H. edited and revised manuscript.

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