PHILIPS

RESPIRONICS

BiPAP A40 EFL

Basics of EFL in COPD

Topic: what is expiratory flow limitation (EFL)?

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The basics of breathing mechanics

The respiratory system is comprised of the upper airway (nasal passage, mouth and oropharynx) and the lower airway (trachea, primary bronchi and subdivisions of the primary bronchi leading to bronchioles and alveoli). In order for gas exchange to occur, air needs to move from the upper airway to the lower airway; this occurs by contracting the respiratory muscles, including the diaphragm and intercostal muscles, which results in changes in pressure that must occur for gas to flow in and out of the lungs.

The diaphragm is the principal muscle of inspiration (although other muscles, such as parasternal, external intercostal, scalene and sternocleidomastoid muscles, also contribute to inspiration).

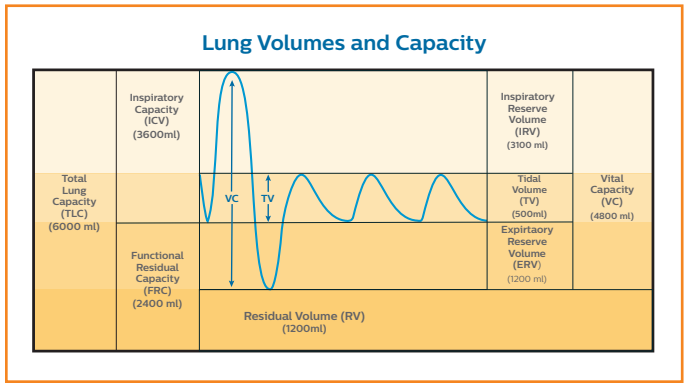


Figure 1

During contraction, the muscle fibers of the diaphragm shorten, the lower ribs elevate and the diaphragm flattens, descends and displaces abdominal contents downwards. This movement causes pleural pressure to fall and lung volumes to increase and for air to move into the lower respiratory tract.

During inspiration and expiration, lung volumes change through contraction and relaxation of the muscles of respiration, with the ability of the lung volumes to alter dependent upon the distensibility or compliance of the lung (Figure 1).

Compliance of the respiratory system is calculated by plotting the lung volume against the distending pressure in a pressure-volume curve (Figure 2); i.e. compliance is a measure of the change in volume (V) over the change in pressure (P), thus compliance = $\Delta V/\Delta P$. The compliance (C_{RS}) of the respiratory system is calculated from the summation of the compliance of the lung (C_L) and the compliance of the chest wall (C_{CW}) ($C_{RS}=C_L+C_{CW}$).

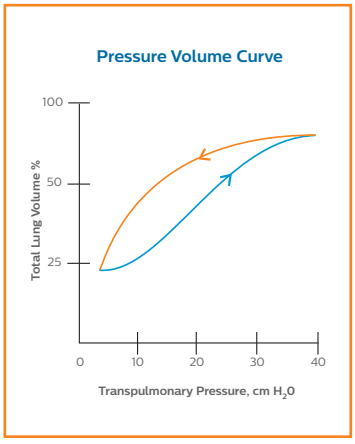
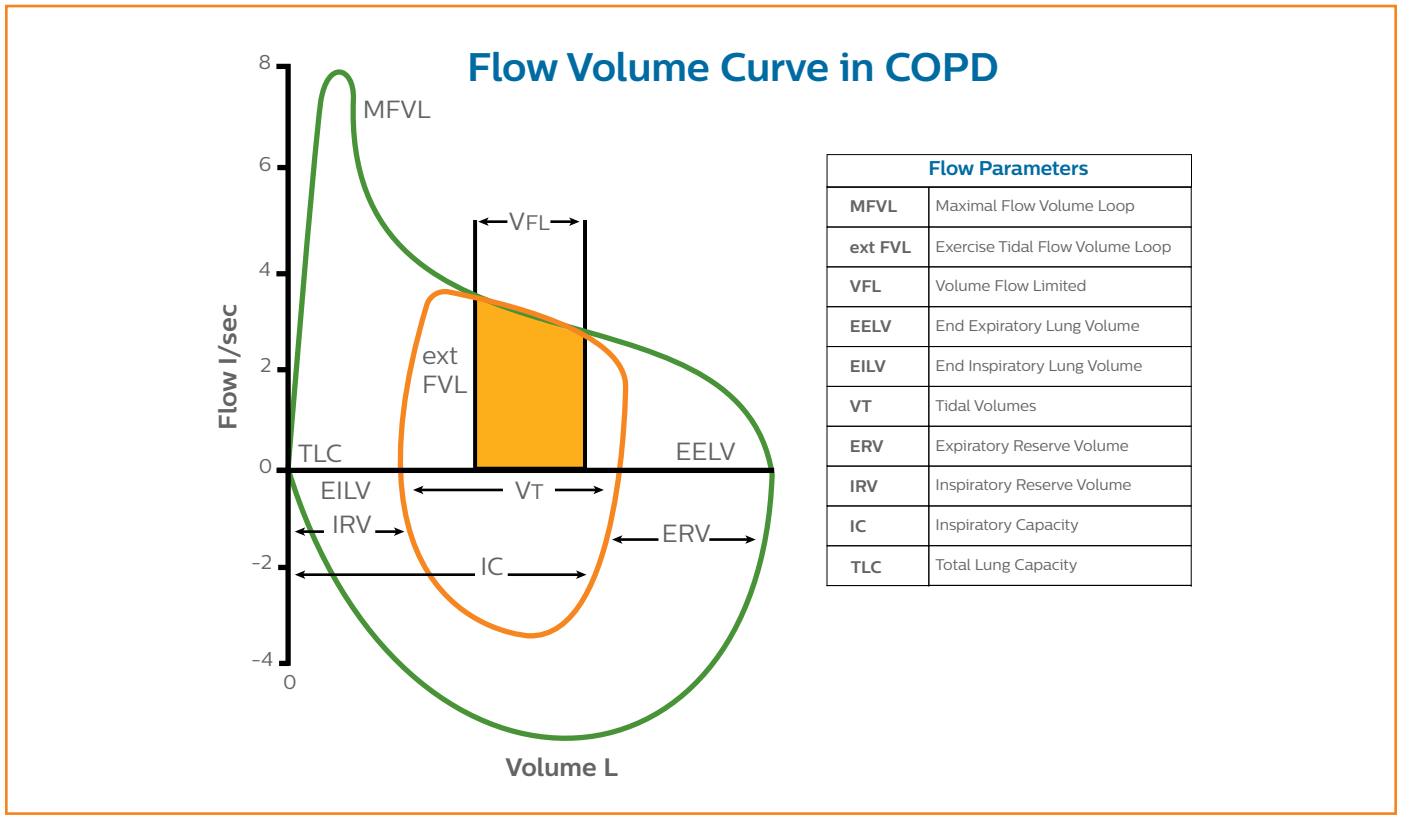


Figure 2

Lung volumes are affected in COPD

Normal total lung capacity (TLC) in a healthy adult male is around 6 L; this is comprised of tidal volume (VT) (0.5 L), expiratory reserve volume (ERV) (1.2 L), residual volume (RV) (1.2 L) and inspiratory reserve volume (IRV) (3.1 L) (Figure 1).

In COPD, lung volumes are changed with an increase in RV and a reduction in inspiratory capacity (IC). Additionally, compliance is reduced as patients breathe on the non-linear part of the pressure volume curve (Figures 3, 4), resulting in an increase in end expiratory lung volumes (EELV). As end expiratory lung volumes increase, expiratory flow limitation (EFL) occurs.



Flow Parameters	
MFVL	Maximal Flow Volume Loop
ext FVL	Exercise Tidal Flow Volume Loop
VFL	Volume Flow Limited
EELV	End Expiratory Lung Volume
EILV	End Inspiratory Lung Volume
VT	Tidal Volumes
ERV	Expiratory Reserve Volume
IRV	Inspiratory Reserve Volume
IC	Inspiratory Capacity
TLC	Total Lung Capacity

Figure 3

These changes in physiology can result in ventilation-perfusion (V/Q) mismatch (ventilation [V] is the air that reaches the alveoli; perfusion [P] is the blood that reaches the alveoli via the capillaries), progressive respiratory failure and a deterioration in symptoms.

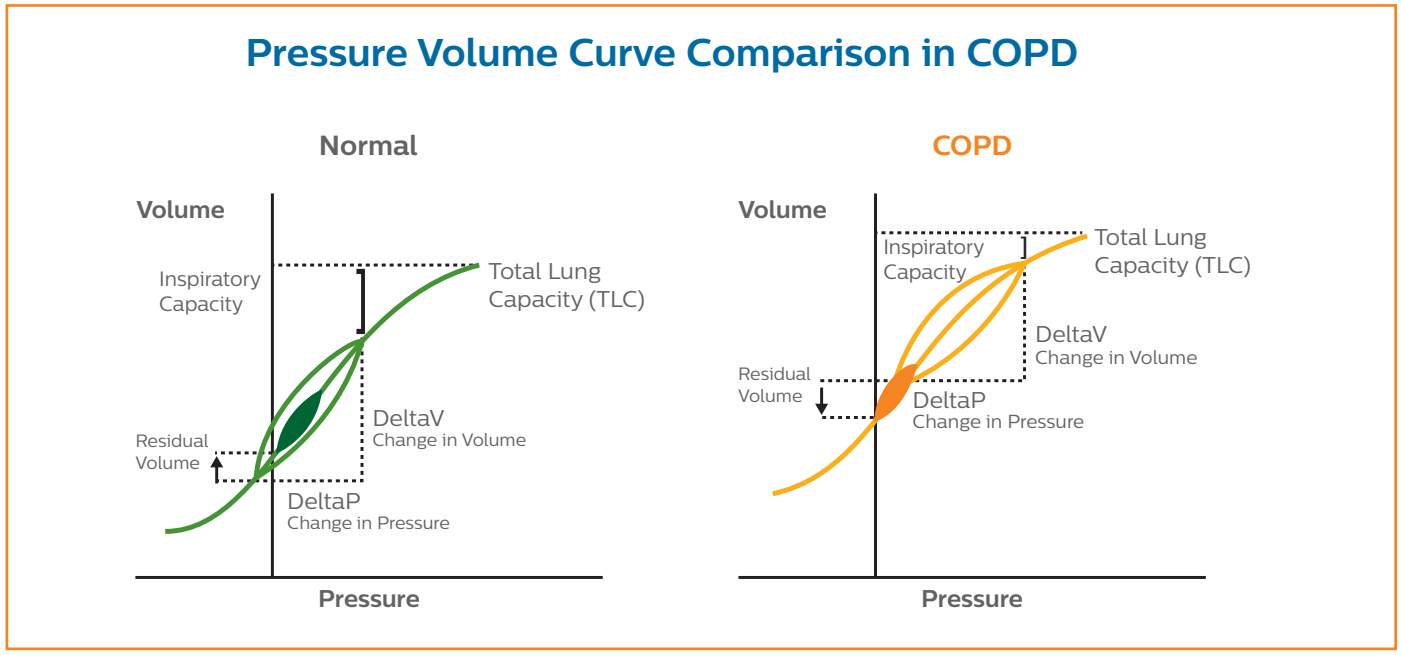


Figure 4

Review of COPD and symptoms

Chronic obstructive pulmonary disease (COPD) is defined by irreversible airflow limitation that is usually progressive and caused by exposure to noxious agents; in the United Kingdom, it is most commonly caused by tobacco smoking. However, worldwide, the most common causative agent is exposure to biomass fuel, such as wood, crops/plant matter, manure and garbage.[1]

Patients with COPD can experience symptoms of breathlessness, cough, wheeze and chronic sputum production, with a reduction of quality of life and exercise capacity.[2] Patients with COPD often experience recurrent flares or acute exacerbations of their COPD, which can result in hospital admission, progression in their disease process and death.

In the U.S., the estimated prevalence of COPD is 5.1%; it imposes a huge economic burden, with data demonstrating that over one year, COPD caused 1.5 million emergency department admissions, 726,000 hospitalisations and 119,000 deaths[3] with direct costs of $29.5 billion USD and indirect costs of $20.4 billion USD.[4]

What is Tidal EFL and why does it occur?

During normal physiology, the lung volumes at end expiration are close to that of the relaxation volume (the volume at which inward elastic recoil equals that of outward elastic recoil), thus there is no air left within the peripheral airways at the end of expiration. When compliance is changed (e.g. loss of elastic recoil in emphysema), end expiratory lung volumes are lower than that of relaxation volume, EFL and early airway closure occurs and leads to gas trapping. Thus, EFL at rest is the inability to increase expiratory flow from the airways despite an increase in expiratory muscle effort (i.e. increasing pleural pressure). The only way in which expiratory flow can be increased is by increasing operating lung volumes (i.e. moving towards end expiratory lung volumes) (Figure 4).

EFL can occur due to several reasons:

1. With increasing age, closing volume and capacity decrease, resulting in closure of airways above end expiratory lung volume
2. In those with chronic obstructive pulmonary disease, reduced compliance and peripheral airway collapsibility (due to destruction of the alveoli) result in EFL
3. Breathing at lower lung volumes, such as in obesity, contributes to EFL

What is hyperinflation and work of breathing?

Most commonly, COPD occurs as a result of damage to the airways from noxious particles, including cigarette smoke and biomass fuel exposure. This results in damage and progressive changes to the respiratory tract or remodeling to the small and large airways and to the blood supply to the airways.

The hallmark sign of COPD is fixed airflow obstruction due to the loss of elastic recoil in the airways, increase in secretions, increased smooth muscle tone and hypertrophy of the submucosal glands. Due to this obstruction and the inability to fully empty the lungs during expiration secondary to EFL, hyperinflation ensues.

Hyperinflation can be either static or dynamic. Static hyperinflation occurs during tidal breathing and is due to the relaxation volume being higher than that of end expiratory lung volumes, due to loss of elastic recoil.

Dynamic hyperinflation often occurs during periods of higher respiratory rates, e.g. during exercise or exacerbations of COPD, where a patient's respiratory rate is increased. During these periods, as the respiratory rate is increased and the expiratory phase of the respiratory cycle is shortened, the patient is unable to fully empty the lungs at the end of expiration, leading to further gas trapping and V/Q mismatch.

This hyperinflation imposes a threshold and elastic load, which increases the work of breathing (i.e. the energy required to breathe in and out). It also ultimately leads to worsening breathlessness, exercise limitation and eventually respiratory failure. Given that exercise limitation in COPD is an important predictor of health-related quality of life, it would be important to try to target interventions to reduce hyperinflation and improve exercise capacity.[5]

The increased work of breathing is due to the extra effort required to initiate inspiration as a result of the air trapping due to EFL leading to hyperinflation and increased (more positive) intrathoracic pressure (Figure 5). Thus, extra inspiratory effort or additional work of breathing is required to overcome the positive intrathoracic pressure or intrinsic peep before inspiratory flow can be initiated. Additionally, hyperinflation results in downward displacement of the diaphragm into a flatter position, with resultant shortening of the muscle fibers. These shortened fibers find it more difficult to generate the inspiratory pressures required to generate inspiratory flow. Thus, this mechanical disadvantage of the diaphragm results in increased work of breathing to perform its function.

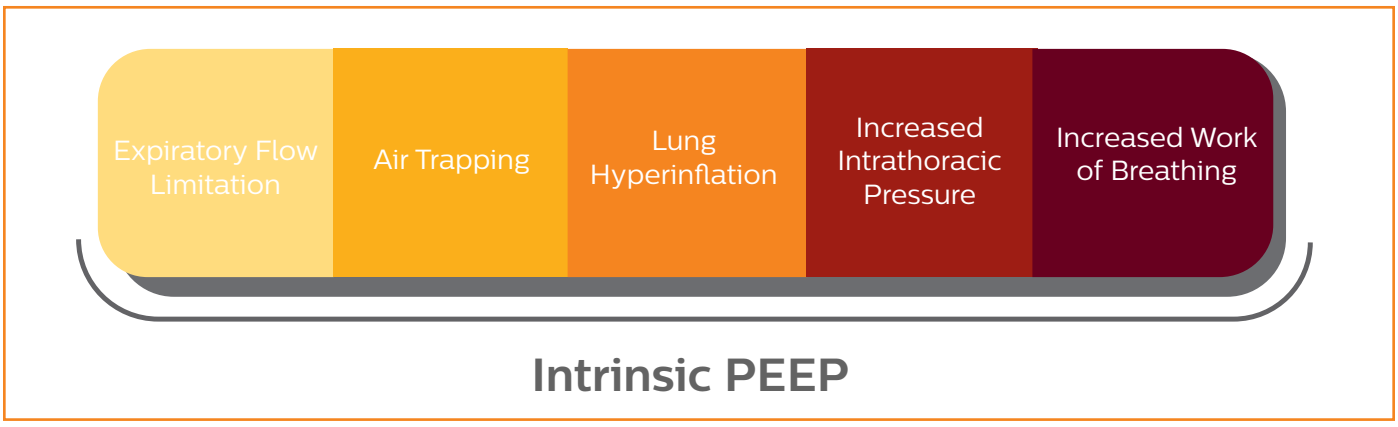


Figure 5

What are the misunderstandings about flow limitation?

Patients with COPD have fixed lower airway obstruction and changes in their lung volumes, which will be contributed to by EFL. EFL can occur in both the central and peripheral airways. While flow limitation in the central airways can contribute to airways resistance, it does not lead to the physiological changes that peripheral airway EFL causes. It has been suggested, however, that flow limitation can contribute to excessive dynamic airway collapse of central airways or the occurrence of choke points (Figure 6). Additionally, depending on the method by which EFL is measured, in patients with obesity and obstructive sleep apnea, EFL may indicate upper airway closure rather than closure of the small airways. The early airway closure caused by peripheral EFL results in air trapping, dynamic hyperinflation, V/Q mismatching and increased work of breathing. Thus, it is important to distinguish between these types of flow limitation.

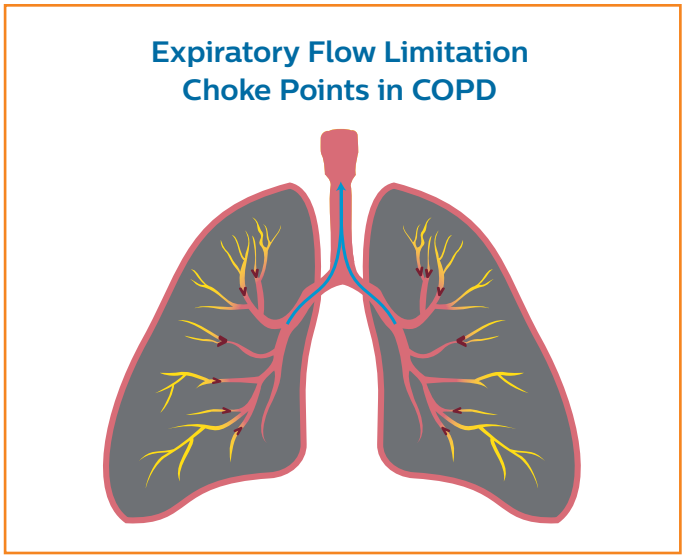


Figure 6

Why should the medical community care about treating EFL?

EFL may contribute to the development of hypercapnic respiratory failure and is involved in the complex interplay of hyperinflation, increased work of breathing, breathlessness and exercise limitation. When an acute exacerbation occurs, a patient's respiratory rate will increase in response to an increased ventilatory demand. As a result, the lungs cannot fully empty by the end of each breath, which leads to an increase in end expiratory lung volumes (gas trapping), leading to dynamic hyperinflation. This results in ventilation/perfusion mismatching, contributing to potential hypercapnia. Dynamic hyperinflation also imposes a threshold load upon the system due to the development of increased intrinsic positive end expiratory pressure, necessitating an increased effort to overcome the positive pressure to allow inspiratory flow to occur. When the load placed upon the respiratory system outweighs the capacity, despite an increase in neural respiratory drive and hypercapnic respiratory failure can ensue. Hence, treating EFL may improve physiology in patients with COPD, thus improving symptoms and exercise capacity.

Recent work by Suh, et al., (in preparation for publication) has demonstrated that utilising technology to target EFL in a noninvasive ventilator (NIV) reduces neural respiratory drive[6] (is a marker of the load: capacity balance of the respiratory system) and transdiaphragmatic pressure (a marker of diaphragmatic effort/strength).[6] While recent technology has been able to auto-titrate the expiratory positive airway pressure (EPAP) during NIV use, this is aimed at opening the upper airways and will not necessarily eliminate EFL. NIV that utilises technology to abolish EFL is speculative, but has many potential clinical applications. NIV that automatically manages EFL may improve hypercapnia, reducing work of breathing, reducing breathlessness and improving exercise limitation but, as mentioned, further data is required.

Reducing work of breathing and a faster improvement in hypercapnia during an acute exacerbation of COPD or could be important from both a quality of life and a health economic perspective with the potential to reduce bed days and costs of exacerbations.

In patients with chronic respiratory failure requiring NIV at home, the BiPAP A40 EFL NIV would abolish EFL and may result in reducing hyperinflation, improving lung volumes and putting the diaphragm at a more mechanically advantageous position, thus reducing the patient's work of breathing both on and off the ventilator.

Simple Glossary

Chronic Obstructive Pulmonary Disease (COPD) – is an umbrella term used to describe progressive lung diseases including emphysema, chronic bronchitis and refractory (non-reversible) asthma. This disease is characterised by increasing breathlessness.

Compliance – elasticity of peripheral lung or a measure of the lung's ability to stretch and expand (distensibility of elastic tissue). In clinical practice, it is separated into two different measurements, static compliance and dynamic compliance.

End expiratory lung volume – is the same as functional residual capacity (FRC) or the lung volume at the end of a normal expiration at ambient pressure, when the muscles of expiration are completely relaxed. At FRC, the tendency of the lungs to collapse is exactly balanced by the tendency of the chest wall to expand.

Exacerbation – an acute change in a patient's baseline dyspnea, cough or sputum that is beyond normal variability and that is sufficient to warrant a change in therapy.

Hypercapnia – also known as hypercarbia and CO_2 retention, is a condition of abnormally elevated carbon dioxide (CO_2) levels in the blood. Carbon dioxide is a gaseous product of the body's metabolism and is normally expelled through the lungs.

Intrathoracic pressure – also called intrapleural pressure refers to the pressure within the pleural cavity. Normally, the pressure within the pleural cavity is slightly less than the atmospheric pressure, in what is known as negative pressure.

Neural respiratory drive – neural input from the brain to the breathing muscles to maintain adequate ventilation.

Pleural pressure – is the pressure surrounding the lung, within the pleural space. During quiet breathing, the pleural pressure is negative; that is, it is below atmospheric pressure. The pleura is a thin membrane which envelopes the lungs and lines the walls of the thoracic cavity.

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