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Does a successful spontaneous breathing trial accurately predict weaning outcome of mechanically ventilated chronic obstructive pulmonary disease patients in ICU?

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Chest diseases and tuberculosis

By

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List of abbreviations

APRV	Airway pressure release ventilation
APV	Adaptive pressure ventilation
ATC	Automatic tube compensation
CMV	Controlled mechanical ventilation
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
FiO_2	The fractional inspired oxygen
FIO_2	A high fraction of inspired oxygen
FRC	The functional residual capacity
IMV	Intermittent Mandatory Ventilation
KBW	Knowledge-Based Weaning
MIP	Maximal inspiratory pressure
NAVA	Neurally Adjusted Ventilatory Assistance
PAV	Proportional Assist Ventilation
PC	Pressure controlled
PCIRV	Pressure control inverse ratio ventilation
PCV	Pressure controlled ventilation
PEEP	Positive end expiratory pressure
PRVC	Pressure-regulated volume control
PTP	The pressure time product
PV	Pressure volume
RCP	Respiratory care patient
RR	Respiratory rate
SBT	Spontaneous breathing trial
SaO2	Saturation of arterial Oxygen
VAP	Ventilator-associated pneumonia
VAPS	Volume-assured pressure support
VPC	Variable pressure control
VS	Volume support
VT	Tidal volume
WOB	Work of breathing

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Introduction

COPD is a progressive disorder characterized by intermittent episodes of acute exacerbations, each of which has the potential for producing respiratory failure and a need for mechanical ventilation. The decision to intubate a patient with severe underlying COPD requires a blending of the physician's estimation of prognosis with the patient's life goals, values, and self-perceived quality of life (Heffner JE, 1998).

Also weaning or, as some physicians prefer, "liberation from mechanical ventilation" is an important issue as unnecessary delays in the withdrawal of mechanical ventilatory support increase the patient's risks for complications and increase the length of ICU stay and hospital costs, also premature withdrawal from the ventilator can also be deleterious. So this issue also should not be performed without certain outlined strategy. (Ryland et al, 2010). Reintubation has the hazards of poor prognosis, with hospital mortality exceeding 30 to 40% (Scott K and Ronald L, 1998).

To minimize the likelihood of either delayed weaning or premature extubation, a two-step diagnostic strategy is recommended: measurement of weaning predictors followed by a weaning trial. Spontaneous breathing trial has gained popularity in the past few years as the goal for most patients on mechanical ventilation is to be weaned from the ventilator. The weaning process is highly dependent on the patient's pathology, but the final common pathway to ventilator independence always includes at least one trial of spontaneous breathing. Trials of spontaneous breathing

have been shown to accurately predict the success of spontaneous breathing (Yang K and Tobin MJ, 1991) (Macintyre NR et al, 2001).

Aim of the work

This study aims to evaluate the validity of spontaneous breathing trial in predicting weaning outcome of mechanically ventilated COPD patients.

Respiratory physiology

Physiology of respiration and control of breathing

The respiratory system is a vital organ in mammals, by which oxygen is delivered to the red blood cells and concomitantly carbon dioxide is removed and expelled into the environment. Environmental air, rich in oxygen, is driven via the upper airways into the alveoli within the lungs where gas exchange takes place between alveolar air and the pulmonary circulation. The lungs are passive extensible organs and located within the thoracic cage. Movement of air into and out of the lung is performed by the respiratory muscles. The inspiratory muscles drive environmental air into the lung, while expiratory muscles remove the air from the lungs **(Ratnovsky et al., 2008).**

Respiratory muscles

The respiratory muscles are morphologically and functionally skeletal muscles. The group of inspiratory muscles includes the diaphragm, external intercostals, parasternal, sternomastoid and scalene muscles. The group of expiratory muscles includes the internal intercostal, rectus abdominis, external and internal oblique and transverse abdominis muscles. During low breathing effort (i.e. at rest), only the inspiratory muscles are active. During high breathing effort (i.e. exercise) the expiratory muscles become active as well **(Ratnovsky et al., 2008).**

1. Inspiratory muscles

The diaphragm, the main muscle of inspiration, is a thin, flat, musculotendinous structure separating the thoracic cavity from the abdominal wall. The muscle fibers of the diaphragm radiate from the central tendon to either the three lumbar vertebral bodies (i.e. crural diaphragm) or to the inner surfaces of the lower six ribs (i.e. costal diaphragm). The costal fibers of the diaphragm are two sided muscle sheet, which are driven independently. The tension within the diaphragmatic muscle fibers during contraction generates a caudal force on the central tendon that descends in order to expand the thoracic cavity along its craniocaudal axis. In addition, the costal diaphragm fibers apply a force on the lower six ribs which lifts and rotates them outward **(De Troyer, 1997).**

The external intercostals muscles are thin layers of muscle fibers that run obliquely downward and ventrally from each rib to the neighboring rib below. The lower insertion of the external intercostals muscles is more distant from the ribs axis of rotation than the upper one, and as a result, contraction of this muscle exerts a larger torque acting on the lower rib which raises of the lower rib with respect to the upper one. The net effect of the contraction of these muscles raises the rib cage **(De Troyer et al., 2005).**

The sternomastoid and the scalene muscles are accessory muscles of inspiration. The sternomastoid muscles descend from the mastoid process to the ventral surface of the manubrium sterni and the medial third of the clavicle. The scalene muscles comprise three bundles that run from the transverse processes of the lower five

cervical vertebrae to the upper surface of the first two ribs. Contraction of these muscles raises the sternum and the first two ribs and thus assist in expanding the rib cage **(Legrand et al., 2003).**

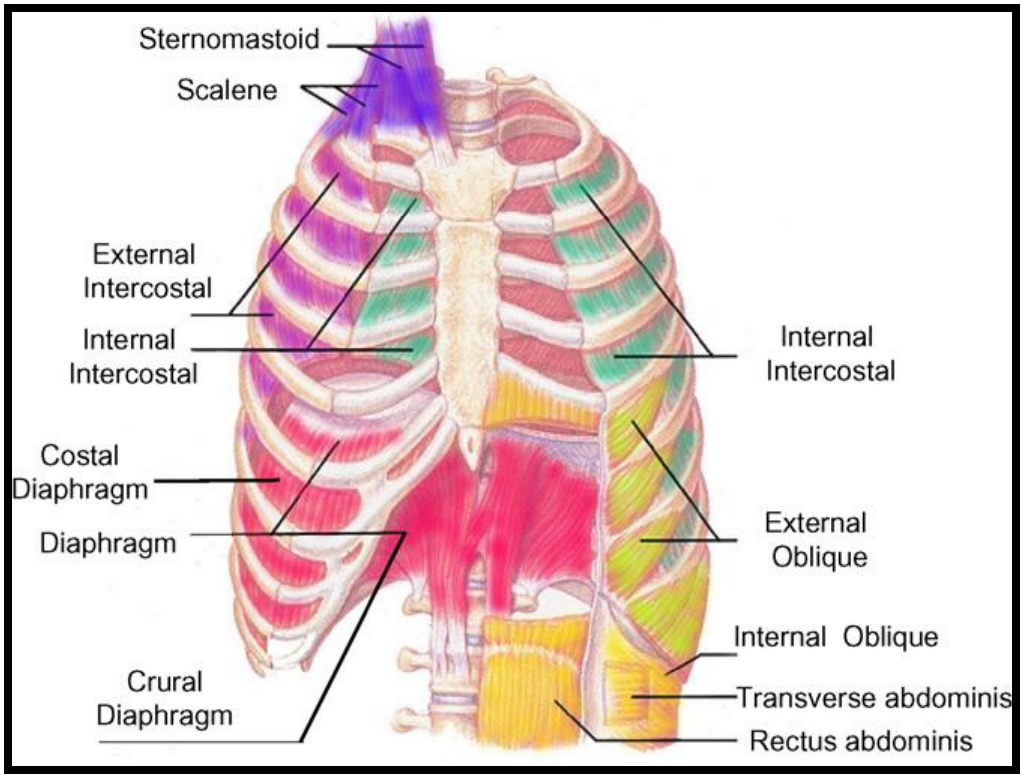

Figure (1): Schematic description of the anatomy of human respiratory muscles **(Ratnovsky et al., 2008).**

2. Expiratory muscles

The internal intercostals muscles are thin layers of muscle fibers that run obliquely downward and dorsally from each rib to the neighboring rib below. The lower insertion of these muscles is less distant from the ribs axis of rotation than the upper one, and thus, during their contraction they lower the ribs **(Wilson et al., 2001).**

The four abdominal muscle pairs forming the abdominal wall are the rectus abdominis, external oblique, internal oblique and transverse abdominis. The rectus abdominis is the most ventral one that runs caudally from the ventral aspect of the sternum and the 5^{th},

6^{th} and 7^{th} costal cartilages along the length of the abdominal wall to its insertion into the pubis **(DeTroyer, 1997).**

The external oblique is the most superficial that originates from the external surface of the lower eight ribs, well above the costal margin, and covers the lower ribs and intercostals muscles. Its fibers radiate caudally to the iliac crest and inguinal ligament and medially to the linea alba. The internal oblique lies deep to the external oblique. Its fibers arise from the inguinal ligament and iliac crest and insert into the anterolateral surface of the cartilages of the last three ribs and into the linea alba. The transverse abdominis is the deepest muscle of the lateral abdominal wall. Its fibers run circumferentially around the abdominal visceral mass from the inner surface of the lower six ribs, lumbar fascia, iliac crest and the inguinal ligament to the rectus sheath. Contraction of the abdominal muscles pulls the abdominal wall inward causing the diaphragm to move cranially into the thoracic cavity and pulls the lower ribs caudally to deflate the ribcage **(Wilson and De Troyer, 2004).**

LUNG MECHANICS:

In normal subjects, in the absence of respiratory effort, the lung will come to lie at the point of the functional residual capacity (FRC) or relaxation volume (V_{rel}). The point at which this occurs is determined by a balance between the inward elastic recoil of the lung and the equal and opposite outward recoil of the respiratory cage (mostly due to muscle tone). The intrapleural pressure (P_{pl}) at this point is −3 to −5 cm water (*Reddy R M and Guntupalli K, 2007*).

To generate a respiratory movement two factors must be overcome:

Resistance

Resistance of the airways is described as obstruction to airflow provided by the conducting airways, resulting mainly from the larger airways (down to division 6–7). This is because the cross sectional area of the upper airways is much smaller compared to the smaller airways as the smaller airways are so many in numbers. The cross sectional area expands with each division of the airway and at generation 16 it is about 300 cm2 compared to 2.5 cm2 at the trachea. This results in a decrease of both airway resistance and air flow velocity. Airway resistance to flow is present during both inspiration and expiration and the energy required to overcome it represents the actual work of breathing (WOB) (***Chandrasekaran R. , 2006***).

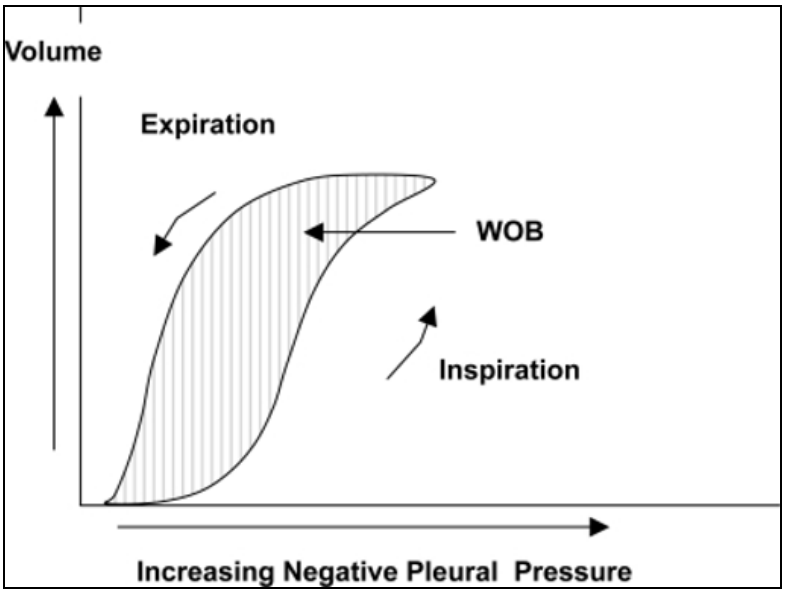

Figure (2); Pressure volume curve showing inspiratory and expiratory arms. Note that compliance is not linear ie, it varies with lung volume. Area within the inspiratory and expiratory curve represents work of breathing (WOB) (Reddy ***R M and Guntupalli K, 2007***).

Compliance

In a clinical setting this refers to the combined compliance of the lung and chest wall. It is the volume change per unit pressure change. When compliance is low, more effort is required to inflate the lungs. Compliance also varies depending on the degree of inflation, which is usually a sigmoid shaped curve in normal subjects (***Update in anesthesia.2000***).

Respiratory mechanics in COPD

The fundamental physiologic abnormality in acute exacerbation of COPD is worsening of expiratory airflow limitation and consequent dynamic hyperinflation. Dynamic hyperinflation increases the work of breathing, puts the respiratory muscles at a disadvantage ***(Orozco L, 2003***), as they have to breathe at higher functional residual capacity and can cause significant cardiac dysfunction (***Brochard, 1995***) leading to worsening hypoxemia with varying degree of hypercarbia and acidosis. The ensuing tissue acidosis further impairs ventilatory muscle function leading to ventilatory failure (***Brochard, 1995***).

The two primary patho-physiologic changes that contribute to the development of respiratory distress and acute respiratory failure in patients with obstructive lung disease are:

1. ***Increased airway resistance***:

Patients with COPD have increased expiratory airflow resistance. In COPD, the alveolar attachments that normally keep the smaller airways open via radial traction are lost. This leads to airway narrowing and collapse especially during expiration. In normal subjects, during passive

exhalation the intrapleural pressure is negative. In COPD the intrapleural pressure may be positive during exhalation due to recruitment of expiratory muscles. As exhalation occurs, the airway resistance increases further due to compression from the surrounding positive intrapleural pressure. This causes the airway segment to collapse. Soon after the collapse occurs the intraalveolar pressure is transmitted to the collapsed segment and the airway reopens because P_{alv} exceeds P_{pl} (Bernasconi 1998; West 2000).

In acute exacerbations the already narrowed airways may be further compromised by increased secretions, mucosal swelling and peribronchial inflammation. The time constant for lung emptying is therefore prolonged and end expiratory lung volume is dynamically increased. Furthermore, during an exacerbation, patients tend to adopt a rapid shallow breathing pattern which further limits the time available for lung emptying, thus promoting greater dynamic hyperinflation (DHI) in a vicious cycle. In fact, any acute increase in ventilation (such as occurs with anxiety or transient hypoxaemia) can be associated with DHI in flow limited patients (**Reddy R M and Guntupalli K, 2007**).

2. ***Dynamic hyperinflation***:

In the presence of increased expiratory airflow resistance the time available (expiratory time) to empty the inspired volume may not be sufficient. The next inspiration may start before the completion of the expiration leading to air trapping. Thus the respiratory system is unable to return to its normal relaxation volume (V_{rel}) at the end of expiration. This results in a new resting state where the FRC is greater than the V_{rel}. This condition of air trapping is otherwise called DHI. The DHI results in positive alveolar pressure at the end of expiration also referred to as auto-PEEP (positive end expiratory pressure) (***Rossi 1995; Ranieri 1996).***