Assessment of Progressive Diaphragmatic Atrophy in Pediatric Acute Respiratory Failure

Thesis

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By

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List of Abbreviations

Abb.	Full term
ARDS	*Acute respiratory distress syndrome*
CBC	*Complete blood picture*
CBG	*Capillary blood gases*
CNS	*Central nervous system*
CRP	*C- reactive protein*
Fio2	*Fractionated inspired oxygen*
ICU	*The intensive care unit*
MAP	*Mean airway pressure*
MV	*Mechanical ventilation*
NMB	*Neuromuscular blockade*
PEEP	*Positive end expiratory pressure*
PICUs	*Pediatric intensive care units*
PIP	*Peak inspiratory pressure*
R.R	*Respiratory rate*
Tdiexp	*Diaphragmatic thickness at end expiration*
Tdi-insp	*Diaphragmatic thickness at end expiration*
TF	*Thickening fraction*
Ti	*Inspiratory time*
US	*Ultrasound*
VIDD	*Ventilation-induced diaphragm dysfunction*

INTRODUCTION

Mechanical ventilation (MV) has been used throughout history as a basic tool in the treatment of patients with respiratory failure, and as a means of improving prognosis. Almost 40% of mechanically ventilated patients have difficulties during MV weaning, due to multiple factors.

A delay in weaning can prolong the stay in the intensive care unit (ICU) and lead to a poorer prognosis and increase in mortality compared to patients with no weaning problems ***(Hermans et al., 2015).***

In the last years, attention has focused on the study of ventilation-induced diaphragm dysfunction (VIDD) as one of the complications associated with MV. This term refers to diaphragm dysfunction that occurs soon after initiating MV ***(Jaber et al., 2011).***

VIDD worsens prognosis and is associated with extubation failure, which in turn prolongs MV and increases the risk of mortality ***(Supinski and Ann Callahan, 2013).***

Diaphragm dysfunction in the mechanically ventilated patient is a disorder that is still undefined and underdiagnosed. It appears early after the initiation of MV and is associated with risk factors such as sepsis and multiorgan failure. It affects a high percentage of patients and leads to extubation failure.

The need for early diagnosis with an easily available, non-invasive technique explains the growing use of ultrasonography in the evaluation and follow-up of diaphragm function and inspiratory effort ***(Baldwin et al., 2011).***

Several studies using ultrasonography have shown good accuracy and reproducibility for evaluating diaphragm function in critical patients ***(Vivier et al., 2012).***

Diaphragm thickness (as a sign of atrophy), shortening fraction, and diaphragm mobility studies (as a sign of diaphragm activity) are the parameters most commonly evaluated by ultrasonography.

AIM OF THE WORK

- To assess Diaphragm Atrophy in mechanically ventilated pediatric patients.
- To assess the effect and the impact of Diaphragmatic Atrophy in weaning of mechanically ventilated patients and their fate.

Chapter 1

ACUTE RESPIRATORY FAILURE IN CHILDREN

Introduction:

Paediatric respiratory emergencies are among the most common reasons for hospital admission and result in a significant number of deaths, particularly in children under 1 year of age.

Acute respiratory infections account for about 20% of all deaths in children under the age of 5 years worldwide ***(Mathers et al., 2009).***

Acute respiratory failure is mostly the result of progressive or acute and sudden deterioration of respiratory and circulatory function during the course of various diseases.

Definition:

Respiratory failure can be defined as the inability to provide O2 along with removal of CO2 at a rate that matches the body's metabolic demand.

Gas exchange and the resultant blood gas tensions are dependent on four processes:

1) Transport of O2 to the alveolus.

2) Diffusion of O2 across the alveolar–capillary membrane.

3) Transfer of O2 from the lungs to the organs (depends on cardiac output and hemoglobin concentration).

4) Removal of CO2 from the blood into the alveolus with subsequent exhalation.

Although respiratory failure may be defined simply in terms of blood gas abnormalities:

1. Partial pressure of oxygen in the blood (PaO2) < 60 mmHg.
2. Partial pressure of carbon dioxide in the blood (PaCO2) > 55 mmHg.
3. Saturation level of oxygen in hemoglobin (SaO2) < 90%).

Etiology:

Respiratory failure can evolve from intrinsic lung disease, airway disease or inadequate respiratory effort.

Disorders involving primarily the respiratory tract:

- **Upper airway obstruction**: (e.g., croup, foreign body aspiration, epiglottitis, tonsillar hypertrophy)
- **Lower airway obstruction:** (e.g., bronchiolitis, status asthmaticus, BPD)

- **Lung disease** (e.g., pneumonia, ARDS, pulmonary edema, near-drowning)

Mechanical impairment of ventilation:

- Neuromuscular disorders/myopathies/infant botulism/ Guillain-Barre' syndrome
- Chest wall trauma and malformations, severe congenital scoliosis
- Large pleural effusion, pneumothorax

Failure of the central nervous system to control ventilation:

- Status epileptics, infection of the central nervous system, intoxication, trauma, apnea of prematurity

Failure to meet oxygen needs of the tissue:

- Hypervolemia, septic shock
- Cardiac insufficiency
- Metabolic disorders, intoxication

The physiology behind respiratory failure in children why are infants more vulnerable?

The considerable differences in respiratory physiology between infants and adults explain why infants and young children have a higher susceptibility to more severe and speedier manifestations of respiratory diseases and why respiratory failure is a common problem in neonatal and pediatric intensive care units (PICUs).

The appreciation of the peculiarities of pediatric respiratory physiology is essential for the correct assessment of any ill child ***(Hammer and Eber, 2005).***

Metabolism:

The basal metabolic rate is about 2–3 times higher in infants than in adults, this means that infants have less metabolic reserve if O2 consumption needs to be increased during critical illnesses.

Control of breathing:

A considerable amount of maturation of the control of breathing occurs in the last few weeks of gestation and in the first few days of life, which explains the high prevalence of apnea in infants born prematurely.

The responses to hypercapnia or hypoxia are decreased and of variable sensitivity, which renders the young infant