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COMPARATIVE STUDY BETWEEN SMALL TIDAL VOLUME VENTILATION WITH AND WITHOUT SUSTAINED INFLATION MANEUVER IN ARDS PATIENTS.

Thesis

Submitted to

Faculty of Medicine

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To my mother and my wife for their unlimited support, sincere efforts, and true love.

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Contents

1. Introduction	1
2. Aim of the work	57
3. Patients	58
4. Methods	59
5. Results	64
6. Discussion	99
7. Summary	115
8. Conclusions and recommendations	118
9. References	120

IMPODUCION

Introduction

Definition:

A recent consensus conference sponsored by the American Thoracic Society and the European Society of Intensive Care Medicine (ATS-ESICM) recommended that "acute lung injury" (ALI) be defined as "a syndrome of inflammation and increasing permeability that is associated with a constellation of clinical, radiologic, and physiologic abnormalities that cannot be explained by, but may coexist with, left atrial or pulmonary capillary hypertension." (1,2) Acute respiratory distress syndrome (ARDS) was simply defined as a more severe form of ALI.

ARDS is an example of a specific type of lung injury (albeit one with diverse causes), characterized by pathologic structural changes termed "diffuse alveolar damage" (DAD) that are associated with a breakdown in the barrier and gas exchange functions of the lung. The result is proteinaceous alveolar edema and severe hypoxemia--both clinical hallmarks of ARDS. (1)

Epidemiology:

No evidence has been found to suggest that ARDS is more common among patients of a certain age, gender, or race. (1) However, its exact incidence is controversial, perhaps because of the problems with definition and diagnosis. Although more precise clinical criteria have improved epidemiologic precision, estimates of incidence still vary from as low as 5 cases per 100,000 persons to as many as 71 cases per 100,000 in the United States. (3)

Morbidity and mortality associated with ALI and ARDS may be slowly declining as a result of improved supportive care and improved treatments for sepsis. (4) ARDS is strongly correlated with sepsis; as many as 40% of patients with sepsis will develop ARDS. In fact, sepsis and systemic inflammatory response syndrome (SIRS) are among the most common factors that predispose the patient to the development of ARDS. Other predisposing factors include direct lung injury (pneumonia, aspiration, inhalation) or indirect causes such as multiple trauma, pancreatitis, severe head injury, cardiopulmonary bypass, fat embolism or hypertransfusion. (5)

Diagnosis:

Patients with ARDS have severe respiratory distress (dyspnea) associated with an acute onset of diffuse chest radiographic infiltrates and hypoxemia. (16) However, so do patients with many other pulmonary syndromes. Thus a definitive diagnosis of ARDS depends on linking these signs and symptoms with the presence of DAD and increased vascular permeability. (1) Any process that can cause alveolar filling can ARDS: mimic any alveolar hemorrhage syndrome, other acute inflammatory processes such as pneumonia, as well as other causes of pulmonary edema. (1,6) Without pathology demonstrating DAD or without direct evidence of increased pulmonary vascular permeability, the diagnosis of ARDS can only be made with certainty if these other processes can' be excluded or if, in retrospect, the pulmonary infiltrates fail to clear despite treatment for these other conditions. (1)

Nevertheless, the clinical diagnosis of ARDS is still usually made by inference by using operational criteria similar to those suggested by the American Thoracic Society – European Society of Intensive Care Medicine (ATS-ESICM) consensus conference. (1)

ATS-ESICM criteria for ARDS:

- □ Identifiable cause or associated condition.
- Dyspnea (usually severe).
- Hypoxemia (usually refractory to supplemental oxygen).
- \square PaO₂ /FiO₂ < 200 mm Hg.
- □ Bilateral radiographic infiltrates (interstitial and alveolar).
- Reduced respiratory system compliance (optional).
- No evidence for cardiac factors as the principal cause of pulmonary edema.

Currently, the most popular method of quantifying "injury" in ARDS is the lung injury score (table 1) based on the degree of hypoxemia, the extent of pulmonary infiltrates on a chest radiograph, level of PEEP used, and a respiratory system compliance measurement. (1,6,7)

Table 1- COMPONENTS AND INDIVIDUAL VALUES OF THE LUNG INJURY SCORE VALUE (7)

1	1. Chest roentgenogram score	
 	No alveolar consolidation	0
!	Alveolar consolidation confined to one quadrant	1
	Alveolar consolidation confined to 2 quadrants	2
	Alveolar consolidation confined to 3 quadrants	2 3
	Alveolar consolidation in all 4 quadrants	4
	•	
	2. Hypoxemia score] '
ľ	$Pa_{O2} / FI_{O2} 300$	0
1	Pa _{O2} /FI _{O2} 225-299	1
	Pa _{O2} /FI _{O2} 175-224	2
	Pa _{O2} /FI _{O2} 100-174	2 3
	$Pa_{02} / FI_{02} < 100$	4
	2 DEED scare (when ventileted)	
	3. PEEP score (when ventilated)	. 0
	PEEP 5 cm H ₂ O	0
	PEEP 6-8 cm H ₂ O	1 2
	PEEP 9-11 cm H ₂ O	2 3
	PEEP 12-14 cm H_2 O PEEP 15 cm H_2 O	4
4		4
٦ ٦	Compliance 80 mL/cm H ₂ O	
	Compliance 60-79 mL/cm H ₂ O	0
· ·	Compliance 40-59 mL/cm H ₂ O	1 2
	Compliance 20-39 mL/cm H ₂ O	2 3
ļ	Compliance 19 mL/cm H ₂ O	3 4
The fin	al value is obtained by dividing the aggregate sum by the number of	+ 7
1	components that were used.	
1	Score	
1	No lung injury	0
-	Mild-to-moderate lung injury	0.1-2.5
1.	Severe lung injury (ARDS)	>2.5
		