Respiratory emergencies in ICU

Essay

Submitted for the partial fulfillment of the master degree in critical care medicine

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2010

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List of abbreviations

AARC	American Association for Respiratory Care
ALI	Acute lung injury
APRV	Airway pressure release ventilation
ARDS	acute respiratory distress syndrome
As	surface area
BAL	bronchoalveolar lavage
BNP	B-type natriuretic protein
BTS	British Thoracic Society
CBF	cerebral blood flow
C_{DYN}	Dynamic compliance
CMV	conventional mechanical ventilation
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
C_{ST}	Static compliance
CT	computed tomography
C_{TL}	Compliance
D	Diffusion
DLO_2	The diffusing capacity for oxygen
DTI	Difficult tracheal intubation
DVT	Deep vein thrombosis
ECLS	extracorporeal life support
EELV	end-expiratory lung volume
EIT	electrical bioimpedance tomography
$El_{,lung}$	elastance of the lung
El_{cw}	elastance of the chest wall
ERV	The expiratory reserve volume
ETT	endotracheal tube
EVLW	extravascular lung water
FiO_2	inspired fraction of oxygen
FOI	Fiberoptic intubation
FRC	functional residual capacity
FRC	The functional residual capacity
HFOV	High-frequency oscillatory ventilation
HIT	Heparin-induced thrombocytopenia
IAP	intra-abdominal pressure
IC	The inspiratory capacity
ICP	intracranial pressure
ICU	intensive care unit
IL	Interleukin
LIP	lower inflection point
LMA	The laryngeal mask airway

LMWH	Low molecular weight heparin
LV	Liquid ventilation
MMPs	matrix metalloproteinases
MW	molecular weight
NIV	Noninvasive ventilation
P/V	pressure/volume
P	Partial pressure difference of the gas
$P_{0.1}$	occlusion pressure
$PaCO_2$	arterial partial pressure of carbon dioxide
PaO_2	arterial partial pressure of oxygen
P_{aw}	airway pressure
PCO_2	partial pressure of carbon dioxide
PE	Pulmonary embolism
P_ECO_2	partial pressure of carbon dioxide in mixed expired gas
PEEP	positive end-expiratory pressure
$P_{El,lung}$	transpulmonary pressure
P_{es}	esophageal pressure
$PetCO_2$	end-tidal partial pressure of carbon dioxide
PFC	Perfluorochemicals
P_{flex}	inflection point
PH	Acid base
Ppa	Pressure of pulmonary artery
P_{pl}	pleural pressure
R_{aw}	Resistance
rt-PA	Recombinant tissue plasminogen activator
RV	The residual volume
RV	right ventricle
SABAs	Short acting b2-agonists
SBT	spontaneous breathing trial
SvO_2	mixed venous oxygen saturation
T	membrane thickness
TLC	The total lung capacity
TNF	tumor necrosis factor
TV	The tidal volume
UFH	Unfractionated heparin
UIP	upper inflection point
V_A/Q	ventilation perfusion ratio
VC	The vital capacity
Vd_{alv}	alveolar dead space
Vd_{aw}	airway dead space
Vd_{phys}	physiologic dead space

VILI	ventilator-induced lung injury
vs	versus
V_T	tidal volume
WOB	work of breathing

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Introduction

Emergency medical admissions constitute a substantial proportion of the workload of the respiratory and cardiology wards, and of the emergency departments. Mortality among these patients is significant and may be determined by the quality of care provided. According to the risk stratification of the patients, the mortality rate may vary from 1% to 30% **(Smith et al., 2009).**

Interestingly enough, among the numerous variables associated with ICU mortality, two of the most powerful independent predictors are respiratory rate and oxygen saturation, suggesting that the respiratory system is very often involved either as a primary trigger of the emergency or as a secondary target of another organ's acute dysfunction **(Smith et al., 2009).**

Most pulmonologists and intensivists, when asked about what they considered to be a respiratory emergency, are likely to suggest the occurrence of acute respiratory failure, either hypoxic or hypercapnia **(Alan et al., 2006).**

The main causes of acute respiratory problems are considered to be an exacerbation of chronic obstructive pulmonary disease or a restrictive disease, pulmonary infections, acute respiratory distress syndrome and cardiogenic pulmonary oedema **(Alan et al., 2006).**

However, in our daily practice, we have to face other important, although less frequent, respiratory emergencies, such as haemoptysis, ingestion of foreign bodies, pneumothorax, drowning and inhalation injury **(Alan et al., 2006).**

On the other hand, respiratory emergencies in patient admitted to ICU can be secondary to other organ dysfunction such as neuromuscular disorders and iatrogenic causes **(Peruzzi et al., 1997).**

Evaluation of patient admitted to ICU with respiratory problem is often a challenge, since the differential diagnosis is a broad. So making correct diagnosis by understanding the significance of the tests of pulmonary function and the pulmonary imaging studies are essential **(Nava et al., 2005).**

Respiratory emergencies support encompasses a lot of prophylactic, therapeutic and diagnostic interventions. The application of correct modality at appreciate time will often result in good outcome with minimal risks and avoid the use of more invasive or expensive supportive measures **(Alan et al., 2006).**

Aim of work

The aim of this work is to discuss different causes, clinical pictures, investigations and managements of respiratory emergencies in ICU

ANATOMY AND PHYSIOLOGY OF THE RESPIRATORY SYSTEM

Anatomy of the Respiratory System:

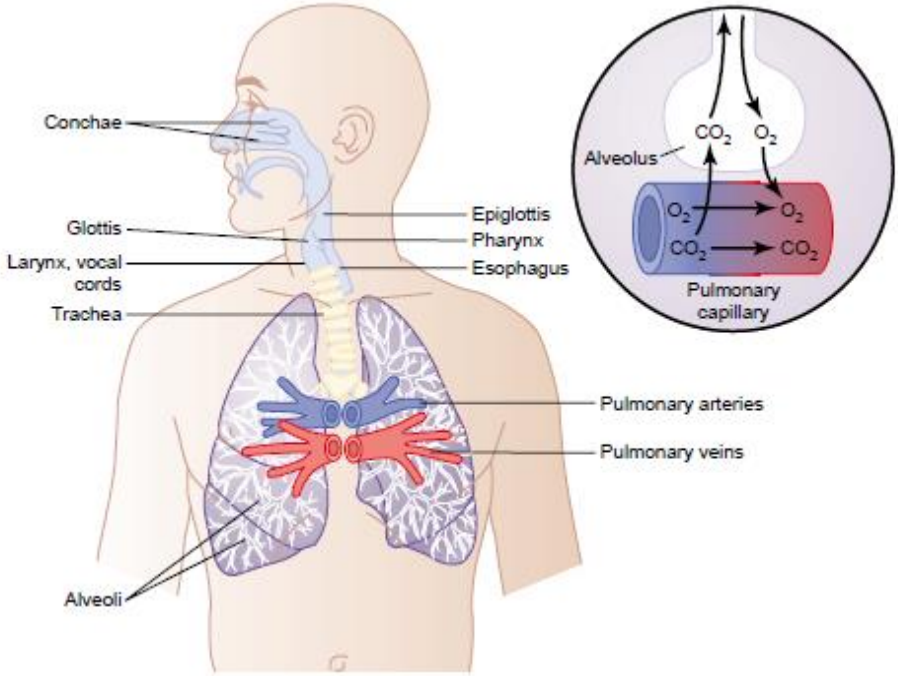


Figure (1-1): Respiratory passages **(Guyton and Hall, 2006).**

I. Upper airway:

Air travels from the nasal passages to the pharynx, and then into the larynx. The larynx lies at the level of upper cervical vertebrae, C4-6, and its main structural components are the thyroid, cricoid and arytenoid cartilages. The thyroid and cricoid cartilages are linked anteriorly by the cricothyroid membrane, through which access to the airway can be gained in an emergency **(Roberts, 2000).**

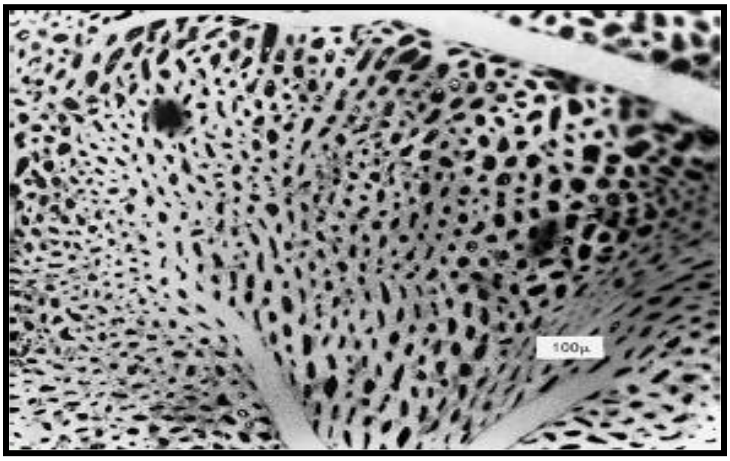
The vocal cords are thin white borders on the lower pair of mucosal folds lining the pharynx; the cords are drawn apart during inspiration and relax toward midline during expiration. During swallowing the epiglottis flaps down to direct swallowed materials into the esophagus, thus guarding the opening of the larynx **(Roberts, 2000).**

II. Lower airway:

Approximately the first 16 divisions of the tracheobronchial tree take no direct part in gas exchange and are thus designated the conducting zone; it's volume is approximately 150ml and is known as the anatomic dead space. The trachea extends from below the cricoid cartilages to the carina**.** Most of the tracheal circumference is made up of a series of C shaped cartilages; the posterior aspect is made of smooth muscle and lies adjacent to the esophagus. Excessive pressure on this smooth muscle by the cuff of an artificial airway can lead to erosion and tracheoesophageal fistula. When the trachea bifurcates, the right main bronchus is less sharply angled from the trachea than the left, making aspirated material more likely to enter the right lung **(Corrin, 2000).**

After penetrating the lung, the main stem bronchi divide into lobar bronchi which then bifurcate and trifurcate into segmental bronchioles or terminal bronchioles that supply the lung segments on the left and right. The bronchioles lack cartilages and are made of connective tissue that contains elastic fibers and limited smooth muscles and are held open by radial traction from the elastic recoil forces of the lung tissue. With the lack of supporting cartilage, these airways are susceptible to bronchospasm **(Des Jardins, 2002).**

The terminal respiratory unit, or acinus, is that portion of the lung arising from a single terminal bronchiole. The acinus is the primary gas exchanging unit of the lung, consisting of the respiratory bronchiole, alveolar ducts, alveolar sacs, and the alveoli. Gas exchange occurs efficiently at the alveolar-capillary membrane. The cellular make up of the alveolus makes it an efficient gas exchanger **(Guyton and Hall, 2006).**



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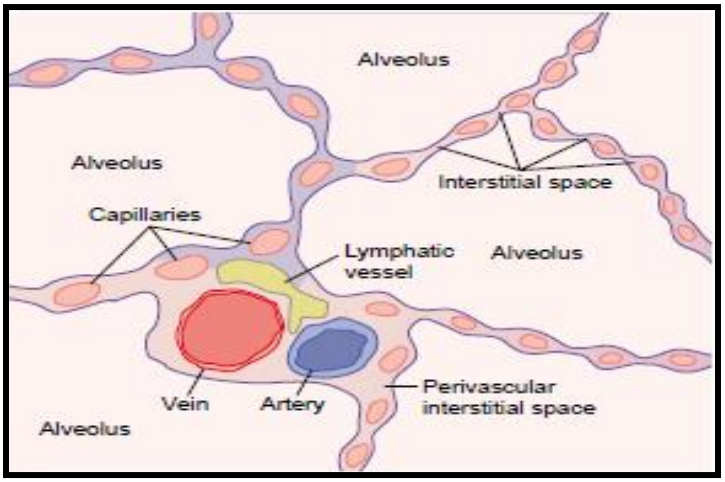

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Figure (1-2): A, Surface view of capillaries in an alveolar wall. B, Cross-sectional view of alveolar walls and their vascular supply **(Guyton and Hall, 2006).**

Type I alveolar cells are structured to promote gas exchange and prevent fluid transudation into the alveolus. Type II cells produce surfactant, a lipoprotein that reduces the surface tension within the alveolus and so prevents the alveoli and bronchioles from collapsing specially during expiration, which makes it easier to expand the lung (increase compliance) and so reduces the work associated with breathing. Surfactant loss leads to atelectasis, impaired gas exchange and increase work of breathing **(Ochs and Weibel, 2008).**

III. Vascular supply:

The lungs have a double blood supply, the pulmonary circulation for gas exchange with the alveoli and the bronchial circulation to supply the parenchyma of the lung itself **(Pierce, 2007).**

i. The bronchial circulation

The primary function of the bronchial circulation is to nourish the walls of the conducting airways and surrounding tissues by distributing blood to the supporting structures of the lungs. Under normal conditions, the bronchial circulation does not supply blood to the terminal respiratory units (respiratory bronchioles, alveolar ducts, and alveoli); they receive their blood from the pulmonary circulation **(Pierce, 2007).**

Bronchial arterial pressure is approximately the same as aortic pressure, and bronchial vascular resistance is much higher than resistance in