SECOND EDITION

NURSING
Interventions for the Critically Ill



Shuva Das Gupta

JAYPEE

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CHAPTER 4

Essential Interventions for the Critically Ill Adults

Objectives

After completing this chapter the learner should be able to:

- Define airway management.
- Identify patients requiring CPR.
- Enumerate the steps of CPR and conduct CPR.
- Make intelligent assessment of critically ill patients and initiate appropriate nursing interventions for them.
- Maintain and monitor airway of patients including those on assisted ventilation.
- Identify life-threatening events and make appropriate interventions.
- Identify signs and symptoms of shock and initiate appropriate interventions.
- Take appropriate precautions to prevent complications in critically ill patients.
- Describe the nursing responsibilities for patients with unconsciousness, cardiovascular involvement, multiple organ failure and polytrauma.
- Interpret Glasgow Coma Scale scores.

Introduction

This chapter deals with specific interventions undertaken for the patients admitted in a critical condition to the ICUs and CCU. This is an ever-expanding vast arena that takes into account the latest developments of the advancing medical technology. Needless to add, it is not possible to cover the entire gamut of these specific interventions. This chapter is, therefore, confined to those essential interventions about which a critical care nurse

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must have a thorough knowledge like airway management, nursing management of patients on assisted ventilation, nursing responsibilities for the critically ill with cardiovascular involvement, and nursing interventions for unconscious patients, for patients with shock and patients with multiple organ dysfunction syndrome [MODS].

This will be a heavy reading all through; it is therefore, advised that the learners relate these discussions with the patients they are nursing or have nursed. Then alone they would benefit from these discussions. It is but essential for them to constantly bear in mind the Indian view of the cycle of life and death and the ultimate goal of life and that service to these patients is service to God. This attitude together with a prayerful nature will enable the ICU/CCU nurses to cope with their responsibilities with efficiency and calm.

There are six sections—viz—sections on

- Airway management
- Common cardiovascular problems in ICU
- Unconscious patients
- Shock
- MOSF and polytrauma
- Nursing responsibilities.

The discussion begins with airway management. A review of relevant anatomy and physiology of the respiratory system is given below (Ref. B10301). For better understanding of airway management.

Section A: Respiration

Respiratory System

- Primary function is to obtain oxygen for use by body's cells and eliminate carbon dioxide that cells produce.
- Includes respiratory airways leading into (and out of) lungs plus the lungs themselves.
- Pathway of air: Nasal cavities (or oral cavity) > pharynx > trachea > primary bronchi (right and left) > secondary bronchi > tertiary bronchi > bronchioles > alveoli (site of gas exchange) (Fig. 4.1, Plate 1). Refer to Appendix 3 for pulmonary volumes.

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The exchange of gases (O_2 and CO_2) between the alveoli and the blood occurs by simple diffusion: O_2 diffusing from the alveoli into the blood and CO_2 from the blood into the alveoli. Diffusion requires a concentration gradient. So, the concentration (or pressure) of O_2 in the alveoli must be kept at a higher level than in the blood and the concentration (or pressure) of CO_2 in the alveoli must be kept at a lower level than in the blood. We do this, of course, by breathing—continuously bringing fresh air (with lots of O_2 and little CO_2) into the lungs and the alveoli.

Breathing is an active process—requiring the contraction of skeletal muscles. The primary muscles of respiration include the external intercostal muscles (located between the ribs) and the diaphragm (a sheet of muscle located between the thoracic and abdominal cavities).

The external intercostals plus the diaphragm contract to bring about inspiration:

- **Contraction of external intercostal muscles** > elevation of ribs and sternum > increased front-to-back dimension of thoracic cavity > lower air pressure in lungs > air moves into lungs.
- **Contraction of diaphragm** > diaphragm moves downward > increases vertical dimension of thoracic cavity > lower air pressure in lungs > air moves into lungs (Fig. 4.2, Plate 1).

To exhale:

- **Relaxation of external intercostal muscles and diaphragm** > return of diaphragm, ribs and sternum of resting position > restores thoracic cavity to preinspiratory volume > increases pressure in lungs > air is exhaled.

Intra-alveolar Pressure during Inspiration and Expiration

As the external intercostals and diaphragm contract, the lungs expand. The expansion of the lungs causes the pressure in the lungs (and alveoli) to become slightly negative relative to atmospheric pressure. As a result, air moves from an area of higher pressure (the air) to an area of lower pressure (our lungs and alveoli). During expiration, the respiration muscles relax and lung volume decreases. This causes pressure in the lungs (and alveoli) to become slight positive relative to atmospheric pressure. As a result, air leaves the lungs (Fig. 4.3).

The walls of alveoli are coated with a thin film of water and this creates a potential problem. Water molecules, including those on

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the alveoli walls, are more attracted to each other than to air, and this attraction creates a force called surface tension. This surface tension increases as water molecules come closer together, which is what happens when we exhale and our alveoli become smaller (like air leaving a balloon). Potentially, surface tension could cause alveoli to collapse and, in addition, would make it more difficult to 're-expand' the alveoli (when you inhaled). Both of these would represent serious problems, if alveoli collapsed they'd contain no air and no oxygen to diffuse into the blood and, if 'reexpansion' was more difficult, inhalation would be very very difficult if not impossible. Fortunately, our alveoli do not collapse and inhalation is relatively easy because the lungs produce a substance called surfactant that reduces surface tension (Figs 4.4. and 4.5, Plate 2).

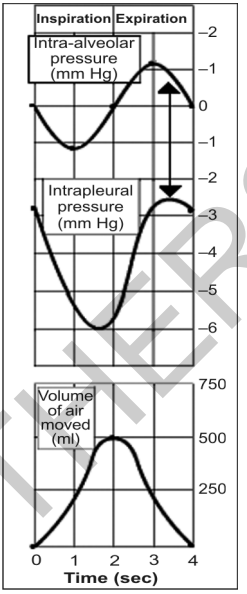

Fig. 4.3: Intra-alveolar pressure

Role of Pulmonary Surfactant

Surfactant decreases surface tension which:

- Increases pulmonary compliance (reducing the effort needed to expand the lungs)
- Reduces tendency for alveoli to collapse.

Exchange of gases

External Respiration

- Exchange of O_2 and CO_2 between external environment and the cells of the body.
- Efficient because alveoli and capillaries have very thin walls and are very abundant (the lungs have about 300 million alveoli with a total surface area of about 75 square meters).

Internal Respiration

- Intracellular use of O_2 to make ATP.
- Occurs by simple diffusion along partial pressure gradients.

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What is Partial Pressure?

- It's the individual pressure exerted independently by a particular gas within a mixture of gasses. The air we breathe is a mixture of gasses: primarily nitrogen, oxygen and carbon dioxide. So, the air you blow into a balloon creates pressure that causes the balloon to expand (and this pressure is generated as all the molecules of nitrogen, oxygen and carbon dioxide move about and collide with the walls of the balloon). However, the total pressure generated by the air is due in part to nitrogen, in part to oxygen, and in part to carbon dioxide. That part of the total pressure generated by oxygen is the 'partial pressure' of oxygen, while that generated by carbon dioxide is the 'partial pressure' of carbon dioxide. A gas's partial pressure, therefore, is a measure of how much of that gas is present (e.g. in the blood or alveoli).
- The partial pressure exerted by each gas in a mixture equals the total pressure times the fractional composition of the gas in the mixture. So, given that total atmospheric pressure (at sea level) is about 760 mm Hg and, further, that air is about 21% oxygen, then the partial pressure of oxygen in the air is 0.21 times 760 mm Hg or 160 mm Hg.

Partial Pressures of O_2 and CO_2 in the Body (Normal, Resting Conditions)

- Alveoli
  - PO_2 = 100 mm Hg
  - PCO_2 = 40 mm Hg
- Alveolar capillaries
  - Entering the alveolar capillaries
  - PO_2 = 40 mm Hg (relatively low because this blood has just returned from the systemic circulation and has lost much of its oxygen).
  - PCO_2 = 45 mm Hg (relatively high because the blood returning from the systemic circulation has picked up carbon dioxide).

While in the alveolar capillaries, the diffusion of gasses occurs: Oxygen diffuses from the alveoli into the blood and carbon dioxide from the blood into the alveoli.

- Leaving the alveolar capillaries
  - PO_2 = 100 mm Hg
  - PCO_2 = 40 mm Hg

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Blood leaving the alveolar capillaries returns to the left atrium and is pumped by the left ventricle into the systemic circulation. This blood travels through arterioles and into the systemic, or body, capillaries. As blood travels through arteries and arterioles, no gas exchange occurs.

- Entering the systemic capillaries
  - PO_2 = 100 mm Hg
  - PCO_2 = 40 mm Hg
- Body cells (resting conditions)
  - PO_2 = 40 mm Hg
  - PCO_2 = 45 mm Hg

Because of the differences in partial pressure of oxygen and carbon dioxide in the systemic capillaries and the body cells, oxygen diffuses from the blood and into the cells, while carbon dioxide diffuses from the cells into the blood.

- Leaving the systemic capillaries
  - PO_2 = 40 mm Hg
  - PCO_2 = 45 mm Hg

Blood leaving the systemic capillaries returns to the heart (right atrium) via venules and veins (and no gas exchange occurs while blood is in venules and veins). This blood is then pumped to the lungs (and the alveolar capillaries) by the right ventricle. Figures 4.6a and b, Plate 3 show the partial pressures and diffusion at the respiratory membrane and in he body tissues respectively. Refer to Figures 4.7 and 4.8, Plate 4.

How are Oxygen and Carbon Dioxide Transported in the Blood?

Oxygen is carried in blood:

1. Bound to hemoglobin (98.5% of all oxygen in the blood)
2. Dissolved in the plasma (1.5%).

Because almost all oxygen in the blood is transported by hemoglobin, the relationship between the concentration (partial pressure) of oxygen and hemoglobin saturation (the % of hemoglobin molecules carrying oxygen) is an important one.

Hemoglobin Saturation

- Extent to which the hemoglobin in blood is combined with O_2.
- Depends on PO_2 of the blood.

The relationship between oxygen levels and hemoglobin saturation is indicated by the oxygen-hemoglobin dissociation (saturation) curve (Fig. 4.9, Plate 5). You can see that at high partial

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pressures of O_2 (above about 40 mm Hg), hemoglobin saturation remains rather high (typically about 75%–80%). This rather flat section of the oxygen-hemoglobin dissociation curve is called the 'plateau'.

Recall that 40 mm Hg is the typical partial pressure of oxygen in the cells of the body. Examination of the oxygen-hemoglobin dissociation curve reveals that, under resting conditions, only about 20%–25% of hemoglobin molecules give up oxygen in the systemic capillaries. This is significant (in other words, the 'plateau' is significant) because it means that you have a substantial reserve of oxygen. In other words, if you become more active, and your cells need more oxygen, the blood (hemoglobin molecules) has lots of oxygen to provide.

When you do become more active, partial pressures of oxygen in your (active) cells may drop well below 40 mm Hg. A look at the oxygen-hemoglobin dissociation curve reveals that as oxygen levels decline, hemoglobin saturation also declines and declines precipitously. This means that the blood (hemoglobin) 'unloads' lots of oxygen to active cells—cells that, of course, need more oxygen.

Factors that Affect the Oxygen-Hemoglobin Dissociation Curve

The oxygen-hemoglobin dissociation curve 'shifts' under certain conditions. These factors can cause such a shift:

- Lower pH
- Increased temperature
- More 2, 3-diphosphoglycerate
- Increased levels of CO_2.

These factors change when tissues become more active. For example, when a skeletal muscle starts contracting, the cells in that muscle use more oxygen, make more ATP, and produce more waste products (CO_2). Making more ATP means releasing more heat; so the temperature in active tissues increases. More CO_2 translates into a lower pH. That is so because this reaction occurs when CO_2 is released.

$$CO_2 + H_2O \longrightarrow H_2CO_3 \longrightarrow HCO_3 + H^+$$

And more hydrogen ions = a lower (more acidic) pH. So, in active tissues, there are higher levels of CO_2, a lower pH, and higher temperatures. In addition, at lower PO_2 levels, red blood cells increase production of a substance called 2, 3- diphosphoglycerate. These changing conditions (more CO_2, lower pH, higher temperature, and more 2, 3-diphospho-glycerate) in active tis-

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sues cause an alteration in the structure of hemoglobin, which, in turn, causes hemoglobin to give up its oxygen. In other words, in active tissues, more hemoglobin molecules give up their oxygen. Another way of saying this is that the oxygen-hemoglobin dissociation curve 'shifts to the right'. This means that at a given partial pressure of oxygen, the percent saturation for hemoglobin will be lower. So, a 'shift to the right' in the oxygen-hemoglobin dissociation curve (Fig. 4.9, Plate 5) means that more oxygen is being released by hemoglobin—just what's needed by the cells in an active tissue!

Carbon dioxide—Transported from the body cells back to the lungs as:

1. Bicarbonate (HCO_3)—60%
   - Formed when CO_2 (released by cells making ATP) combines with H_2O (due to the enzyme in red blood cells called carbonic anhydrase) as shown in Figure 4.10, Plate 5.
2. Carbaminohemoglobin—30%
   - Formed when CO_2 combines with hemoglobin (hemoglobin molecules that have given up their oxygen)
3. Dissolved in the plasma—10%
   See Figure 4.10, Plate 5.

Points to Remember

- O_2 tension in tissue and of body is less than 40 mm Hg.
- The oxyhemoglobin (HbO_2) in RBC is exposed to low O_2 tension. (Hence HbO_2 breaks and O_2 enters the body tissues while hemoglobin remains in the blood).
- CO_2 tension in tissue end is very high, hence CO_2 from tissue cells (body) enters venous blood.
- Lastly CO_2 comes out through the lungs during expiration.

Control of Respiration

Your respiratory rate changes. When active, for example, your respiratory rate goes up; when less active, or sleeping, the rate goes down. Also, even though the respiratory muscles are voluntary, you can't consciously control them when you're sleeping. So, how is respiratory rate altered and how is respiration controlled when you're not consciously thinking about respiration?

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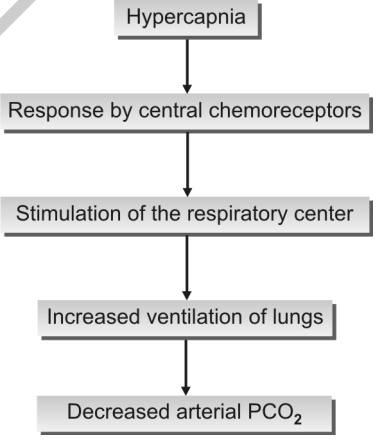
Factors Involved in Increasing Respiratory Rate

- Chemoreceptors—Located in aorta and carotid arteries (peripheral chemoreceptors) and in the medulla (central chemoreceptors).
- Chemoreceptors (stimulated more by increased CO_2 levels than by decreased O_2 levels) > stimulate rhythmicity area > result = increased rate of respiration.

The respiratory center: Normally control of respiration is involuntary. During speaking and singing activities voluntary control is exerted. Groups of nerve cells situated in the brainstem, medulla oblongata and the pons control the rate and depth of respiration. There is a complex interrelationship between these groups of cells. The medulla oblongata has inspiratory neurons and expiratory neurons. These neurons in the medulla are influenced by the pneumotaxic and apneustic neurons situated in the pons. Through the phrenic nerve and the intercostal nerve the motor impulses leaving the respiratory center pass to the diaphragm and intercostal muscles respectively.

Chemoreceptors: These are situated centrally—referred to as *central chemoreceptors and peripherally*—referred to as *peripheral chemorecptors*. Changes in partial pressures of O_2 and CO_2 in the blood and CSF cause responses in the chemoreceptors.

- *Central chemoreceptors*: These are located on the surface of the medulla oblongata and are bathed in CSF. Even in mild hypercapnia (rise in arterial PCO_2), chemoreceptors start acting as shown below:



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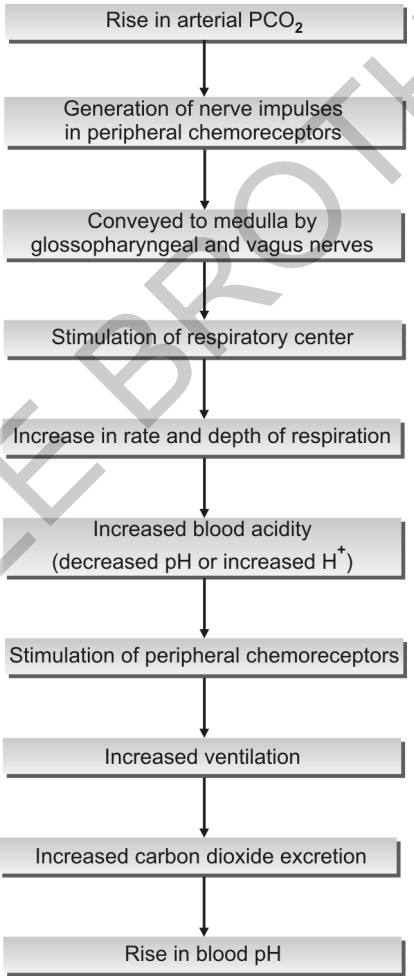
The central chemoreceptors' sensitivity to raised arterial PCO_2 or hypercapnia plays the most important role in the maintenance of homeostasis of blood gases in health. A slight decrease in PO_2 has the same effect but mild; But substantial hypoxemia (lower PO_2) produces and depressing effect.

Peripheral Chemoreceptors

These are located in the arch of the aorta and the carotid bodies. They act in the following manner:



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The rhythmicity center of the medulla:

- Controls automatic breathing.
- Consists of interacting neurons that fire either during inspiration (I neurons) or expiration (E neurons).
- I neurones—Stimulate neurons that innervate respiratory muscles (to bring about inspiration).
- E neurons—Inhibit I neurons (to 'shut down' the I neurons and bring about expiration).

Apneustic center (located in the pons)—Stimulate I neurons (to promote inspiration).

Pneumotaxic center (also located in the pons)—Inhibits apneustic center and inhibits inspiration.

Other factors having an effect on respiration are drugs such as sedatives, alcohol, sleep, speech, singing, emotional displays like fear and crying.

Temperature: In fever respiration increases. In hypothermia respiration decreases. It is important to remember, homeostasis of blood gases may be obtained by effective control of respiration in a widerange of physiological, pathological, and environmental conditions. Yoga therapy has therefore, gained a momentum in the therapeutic regimen of variety of ailments.

Important among these ailments are *respiratory acidosis* and *respiratory alkalosis*, both of which are 'primary pathophysiologic states'. In respiratory acidosis, there is a decrease in alveolar ventilation (Carbon dioxide excretion) in relation to carbon dioxide production. In respiratory alkalosis, on the other hand, there is increase in alveolar ventialation relative to carbon dioxide production.

Causes of *respiratory* acidosis and *respiratory alkalosis* given by George L. Ackerman and Jose AL Arruda, are listed in Table 4.1.

The physiological variables that control respiration may also be recalled here. These are: *Elasticity, compliance, and airflow resistance*.

Elasticity: Ability of the lungs to comeback to their normal shape after each breath. Its loss causes forced expiration and increases inspiratory effort.

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Disorder/Condition	Respiratory Acidosis Mechanism	Effect
Trauma Coma Anesthesia Tumor	Depression of the respiratory center in the brainstem	Decreased alveolar ventilation
Trauma Poliomyelitis	Spinal cord, respiratory muscles and chest wall affected resulting in loss of expansion of chest wall	Decreased alveolar ventilation
Airway obstruction by foreign bodies obstructive pulmonary disease severe pulmonary edema	Airway and lungs are affected resulting in decreased ventilation or diffusion	Decreased alveolar ventilation
Disorder/Condition	**Respiratory Alkalosis Mechanism**	**Effect**
Emotion acetyl salicylic acid organic disease progesterone	Direct stimulation of the respiratory center in the brainstem	Increased alveolar ventilation
Hypoxemia	Reflex stimulation of the respiratory center by the stimulation of the peripheral chemoreceptors	Increased alveolar ventilation
Localized pulmonary disease	Reflex stimulation of the respiratory center by the intrathoracic receptors	Increased alveolar ventilation

Table 4.1: Causes of respiratory acidosis and respiratory alkalosis

Compliance: Relates to the effort needed to inflate the alveoli; if the compliance is low, the effort required will be greater than normal. If elasticity is reduced or when pulmonary surfactants (phospholipids controlling the surface tension of air-liquid emulsion present in the lungs) are not sufficient.

Airflow resistance: Bronchoconstriction causes increased airflow resistance, which requires increased respiratory effort for inflation of the alveoli.

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NURSING
Interventions for the Critically Ill

Salient Features

- A ready reference for the nurse teachers and practitioners dealing with critical care.
- Two new chapters have been added in this edition.
- Provides latest recommendations of the AHA based on the ongoing research findings and biotechnological trends.
- Enriched with numerous tables, charts and photographs.



Shuva Das Gupta RN RM RPHN MN MA(USA) is an eminent personality in nursing education in India. She is the Founder Principal of the West Bengal Government College of Nursing, Kolkata, set up in 1974. She has taken up various advisory and consulting assignments in India and Nepal since her retirement.

Professor Das Gupta has served in various senior technical capacities with WHO, UNICEF and ODA during her outstanding career. Her wide administrative, teaching and community health nursing experience together with her incisive insight as a practicing nurse in various government hospitals of Kolkata merit special mention. She has received several awards and distinctions including President's National Award of 1982. Quite a few publications on delivery of maternal and child health services, nursing management and critical care nursing have been coauthored and authored by her.

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