CARDIOPULMONARY RESUSCITATION

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PREFACE

The purpose of this textbook is to present advances in cardiopulmonary resuscitation. From recent investigations has come a new level of understanding of the principles of resuscitation.

The text has been designed to present the information to its readers in a format which identifies the current standards of care as well as the recent advances. The total product should be a clinically useful, cohesive text which aids all health practitioners delivering emergency cardiac care.

In the first chapter, an overview of the problem of sudden death and emergency cardiac care is given. New concepts in ventilation are presented in Chapter 2. This is a comprehensive chapter that considers the physiology and management of respiratory failure associated with cardiac arrest. Chapter 3 presents an exciting prospectus on perfusion during CPR. Included in the chapter are current modalities employed to augment flow as well as detailed information regarding "cough CPR." The issue of brain resuscitation is addressed in Chapter 4. The pharmacology of resuscitation is considered with emphasis placed on the commonly used drugs in Chapter 5. A useful set of guidelines is developed for the clinician. An in-depth discussion of the most recent advances on defibrillation is addressed next. Current recommendations are delineated as well as the scientific basis for the dose levels. Prehospital aspects of resuscitation represent a significant addition to the text. Protocols for paramedics as well as the base station physician are presented. Next follows a discussion of pediatric resuscitation, an important component in the continuum of emergency cardiac care. The medicolegal aspects of resuscitation are presented in Chapter 9. Specific case studies are related with recent trends in legal decisions emphasized. Finally, an increasingly more common entity is discussed—the traumatic cardiac arrest.

Throughout the text, emphasis has been placed on new developments in the care of the cardiac arrest victim. In many aspects of resuscitation, the approach and certain techniques are undergoing modification in light of new knowledge. A major effort has been made in selecting topics written by the leading clinician-investigators to provide the reader with an authoritative volume on resuscitation.

This publication should be useful to a wide audience of professionals in medicine, including emergency physicians, generalists, cardiovascular specialists, academic investigators, paramedic personnel and critical care nurses.

Ann L. Harwood, M.D.

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I am deeply grateful to those contributing authors who gave of their time, energies and expertise.

Special recognition is due those investigators who have been courageous in challenging the time-honored concepts of resuscitation and allowed new scientific knowledge to emerge.

A.L.H.

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Chapter 1

INTRODUCTION

COSTAS T. LAMBREW, M.D.

Cardiac arrest is the clinical picture of unresponsiveness, apnea, and absence of a pulse, reflecting cessation of circulation.

There are multiple causes of cardiac arrest, including electric shock, asphyxiation, trauma to the chest, drowning, drug overdose, or therapeutic interventions which increase vagal tone or myocardial irritability. However, the major cause of sudden death is ischemic heart disease. Atherosclerotic coronary disease causes myocardial infarction in nearly one million people each year; in 1977, 638,427 of these patients died, nearly 60% of them outside the hospital, most within 2 hours after the onset of symptoms.[1] Sudden death, without premonitory symptoms, accounts for nearly 25% of all deaths from ischemic heart disease. Community mortality from sudden death reached epidemic proportions by the mid-20th century.

The problem of sudden death is not new. Pliney, in his *Historia Naturalis*, describes the sudden death of Roman citizens. The first sudden death to be related to cardiovascular disease at autopsy is well described in the notebooks of Leonardo DaVinci (1452–1519).[2] However, a practical approach to cardiac arrest did not become available until 1960, when Kouwenhoven and his co-workers[3] discovered the efficacy of closed chest compression in causing circulation of blood from the arrested heart. Coupled with the earlier description of mouth-to-mouth ventilation, modern cardiopulmonary resuscitation (CPR) was born and implemented.[4,5] CPR became the keystone of the approach to in-hospital cardiac arrest. Promptly applied CPR complemented by defibrillation and stabilization of rhythm through use of drugs resulted in significant salvage of hospitalized patients with acute myocardial infarction who developed cardiac arrest in the coronary care unit. Monitoring the hospitalized patient at risk for dysrhythmias and aggressive antidysrhythmic therapy to prevent progression to cardiac arrest further contributed to the impact of the coronary care unit.[6] However, community mortality continued its inexorable rise in the early 1960s. It became clear that since most ischemic cardiac deaths occurred out of the hospital, resuscitation would have to be extended to these patients in the field. Pantridge, in Belfast, was the first to implement the mobile coronary care unit concept, documenting the high incidence of life-threatening dysrhythmias in the first hours after the onset of symptoms of acute myocardial infarction and demonstrating the effectiveness of the unit in resuscitating these patients from ventricular fibrillation.[7]

Subsequent experience has demonstrated that critical determinants of survival include the intervals of onset of cardiac arrest to initiation of CPR,

and onset to availability of advanced life support interventions. Therefore, large segments of the lay public have been taught CPR. Lay person CPR, applied within 4 min, even before arrival of rapidly responsive advanced life-support units, has almost doubled survival, if complemented within approximately 8 min by advanced cardiac life support.[8,9] Ventricular fibrillation is the most common electrical mechanism responsible for cardiac arrest in this population; prompt defibrillation is that advanced life-support intervention which will have the greatest impact on survival of patients who have been maintained by CPR in the first 4 min.

Yet, even under the best of circumstances, the impact on survival is limited. Prompt identification and rapid response is mandatory. The widespread use of CPR has prompted renewed investigative interest in the mechanics of blood flow with external chest compression. These investigations have raised questions about the basic mechanism of flow and suggested variations in technique which would optimize flow.[10] Protection of the ischemic brain and resuscitation directed specifically to prevention of neurological deficit have been pioneered by Safar and his colleagues and add yet another dimension to the approach to these patients.[11] The impact of the level of defibrillator output on success or failure of defibrillation has been extensively discussed and appropriately investigated.[12] The empiric use of drugs has been questioned. Secondary prevention in the patient at risk of cardiac arrest is under extensive study. Identification of the symptomless patient at risk of sudden death is not yet possible with any consistency. Efforts at primary prevention must be continued in the form of risk factor intervention.

These questions and problems have resulted in a renaissance of interest in resuscitation and new issues: medical, legal, and ethical. It reflects a rapidly changing state of the art based both on past experience and current investigation. These issues will be addressed in subsequent chapters.

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Chapter 2

VENTILATION DURING CARDIOPULMONARY RESUSCITATION

WILLIAM H. MONTGOMERY, M.D.

The ultimate objectives of providing ventilation during cardiopulmonary resuscitation are to (1) relieve hypoxia, anoxia or hypoxemia; (2) protect the airway from obstruction by foreign material, either extrinsic or intrinsic; and (3) to assist in the maintenance of normal acid-base balance. Satisfying these three objectives, although admittedly sometimes not easy to accomplish, will many times result in a successful resuscitation effort.

PULMONARY PHYSIOLOGY

Pulmonary physiology which includes both pulmonary function and gas exchange in the lungs can be subdivided into four major areas including ventilation, distribution of ventilated gases, diffusion of gases, and perfusion of blood.

Ventilation

Ventilation is the process whereby air is brought into and out of the lungs, by rhythmic contraction of the diaphragm and intercostal muscles. The term ventilation should not be confused with respiration. Respiration includes not only ventilation but also all processes involved in gas exchange and utilization at the cellular level in the body tissues. There are three centers in the brain which control ventilation. These are the upper pons, the lower pons, and the medulla. These cannot be separated in clinical medicine and are lumped together as the "respiratory center." This center will determine the depth and rate of ventilation after receiving input from various stimuli. The most important stimuli are hypercarbia, hypoxemia, and acidosis. Increased carbon dioxide (CO_2) in the blood by way of its effect on the pH in the brain is the most potent stimulus that the respiratory center receives. Small changes in the arterial carbon dioxide tension (P_aCO_2) do not act directly on the respiratory center but CO_2 diffuses freely into the cerebrospinal fluid (CSF) to affect CSF pH.[75]

Hydrogen ions are released by the following reaction:

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

Because CSF has no proteins and is poorly buffered its pH changes rapidly with change in the CSF CO_2. The respiratory center reacts to a decreased P_aCO_2 by decreasing the depth and frequency of ventilation and to an increase in P_aCO_2 by increasing rate and depth of ventilation.

Hypoxemia is a weaker stimulus to ventilation than hypercarbia. If hypoxemia induces an increase in ventilation the resultant hypocarbia will interfere with the hypoxic drive. Until the inspired oxygen concentration (F_iO_2) reaches 15–16% the respiratory center will remain unresponsive. Hypoxemia may indirectly stimulate the respiratory center via the chemoreceptors located in the carotid bodies. Although the response to hypoxemia is not strong, the victim does not adapt to a continuously low F_iO_2 and the hypoxia drive remains indefinitely or until death occurs. In patients with chronic obstructive pulmonary disease, hypoxemia may be the only stimulus for respiration. If the respiratory center is depressed by drugs or chronic hypercapnea, the hypoxemia stimulus may be reduced in these patients.

Acidosis without any change in P_aCO_2 will increase both the respiratory rate and volumes secondary to stimulation of the respiratory center as well as peripheral receptors. As stated above, most respiratory changes caused by CO_2 are related to its effect on CSF pH.

A number of terms are necessary to be familiar with in order to understand the ventilation cycle. There are four lung volumes and five lung capacities. Tidal volume (V_T) is the volume of air moved in and out of the lung during normal resting ventilation. Inspiratory reserve volume (IRV) is the maximal volume of air that can be inspired from the end-inspiratory position. Expiratory reserve volume (ERV) is the maximal volume of gas that can be expired from the end-expiratory level. Residual volume (RV) is the volume of gas remaining in the lung at the end of a maximal expiration. The capacities contain at least two or more primary volumes and are as follows. Total lung capacity is the amount of air contained in the lung at the end of a maximal inspiration (RV + forced vital capacity (FVC)). Vital capacity (V_c) is the maximal volume of gas that can be expelled from the lungs by forceful effort following a maximal inspiration (V_c = IRV + V_T + ERV). Inspiratory capacity (IC) is the maximal volume of gas that can be inspired from the resting expiratory level (V_T + IRV). The functional residual capacity (FRC) is the volume of gas remaining in the lungs at the resting expiratory level (ERV + RV). The forced vital capacity (FVC) is the volume of air that the patient can exhale after a maximal inspiration.

The relationship between lung volume and capacities (total lung capacity, TLC) can be expressed as follows:

$$TLC = RV + V_C$$

$$V_C = IRV + V_T + ERV$$

$$TLC = FRC + IC$$

$$FRC = ERV + RV$$

$$IC = IRV + V_T$$

An example of the normal lung volumes in a young healthy patient would be as follows:

$$TLC = RV + V_C$$

$$TLC = RV + (IRV + V_T + ERV)$$

$$5.6\ L = 1.2\ L + (2.1\ L + 0.5\ L + 1.8\ L)$$

Lung capacities in this same patient would be as follows:

$$V_C = IRV + V_T + ERV$$

$$V_C = (2.1\ L + 0.5\ L + 1.8\ L)$$

$$V_C = 4.4$$

$$IC = IRV + V_T \qquad FRC = ERV + RV$$

$$IC = 2.1 + 0.5\ L \qquad FRC = 1.8\ L + 1.2\ L$$

$$IC = 2.6\ L \qquad FRC = 3.0\ L$$

Tidal volume V_T in a normal male averages 400–500 cc; 150–200 cc of this never reaches alveoli that are perfused and this part of the tidal volume is called dead space (V_D). Under normal conditions the ratio of dead space to tidal volume (V_D/V_T) in healthy persons is 0.3–0.4. To determine how much air is available for each blood-gas exchange (alveolar ventilation, V_A), subtract $V_D - V_T$. Alveolar ventilation per minute (V_A) can then be calculated as follows:

(1) $V_A = V_T - V_D$

(2) $V_A = V_A \times f$ where f = respiratory frequency per minute.

Because V_D remains fairly constant, different tidal volumes can produce significant changes in V_A, even when the total minute ventilation is constant,

(1) Assume V_D 150 cc, V_T 500, f = 16,

$$V_A = V_T - V_D \qquad V_A = V_A \times f$$

(2) Assume V_D = 150 cc, V_T − 200 cc, f = 40

$$V_A = V_T - V_D \qquad V_A = V_A \times f$$

$$V_A = 50 \qquad 2\ L/min = 50 \times 40$$

(3) Assume V_D = 150, V_T = 1000, f = 8

$$V_A = V_T - V_D \qquad V_A = V_A \times f$$

$$V_A = 850\ cc \qquad 6.8\ L/min = 850 \times 8.$$

At higher tidal volumes the V_D/V_T ratio decreases and the alveolar ventilation is much more efficient.

There are two types of dead space, anatomic and pathologic or alveolar dead space. Anatomic dead space refers to the air conducting pathways and is roughly equal to the patient's weight in pounds. Pathologic or alveolar dead space is the volume of alveoli that are ventilated but not perfused. An example of alveolar deadspace would be pulmonary embolus where the alveolus is ventilated but not perfused.

Increased alveolar dead space causes a rise in arterial P_aCO_2 and tends to lower the expired and alveolar P_aCO_2. The relationship between the gases and the dead space is relatively constant and can be used to calculate the amount of dead space present. One needs to measure the P_aCO_2 and the patients expired CO_2 ($P_{\bar{e}}CO_2$) collected in a Douglas bag. In normal situations the $P_aCO_2 = 40$ mm Hg, the average $P_{\bar{e}}CO_2 = 28$ mm Hg and therefore the ratio of physiologic dead space to tidal volume is about 0.3; i.e.:

$$V_D/V_T = (P_aCO_2 - P_{\bar{e}}CO_2/P_aCO_2 = \text{Bohr equation}$$
$$= (40 - 28)/40$$
$$= 12/40$$
$$= 0.3.$$

This assumes that the alveolar CO_2 is equal to the arterial CO_2.

If alveolar dead space increases much, then alveolar CO_2 (P_ACO_2) will be lower than the arterial CO_2 (P_aCO_2); the average expired CO_2 ($P_{\bar{e}}CO_2$) will also be lower than normal; i.e.:

$$V_T = 500,\ P_aCO_2 = 40 \text{ mm Hg},\ P_ACO_2$$
$$= 30 \text{ mm Hg},\ P_{\bar{e}}CO_2 = 24 \text{ mm Hg}$$
$$V_D \text{ anatomic} = ((P_aCO_2 - P_{\bar{e}}CO_2)/P_aCO_2) \times 500$$
$$= ((30 - 24)/30) \times 500$$
$$= 100 \text{ ml}$$
$$V_D \text{ total} = ((P_aCO_2 - P_{\bar{e}}CO_2)/P_aCO_2) \times 500$$
$$= ((40 - 24)/40) \times 500$$
$$= 200 \text{ ml}.$$

Thus: alveolar dead space would be $200 - 100 = 100$ ml.

The P_aCO_2 is an accurate indication of the quality of alveolar ventilation. With a normal alveolar ventilation of 4–5 L/min, the P_aCO_2 will be about 40 mm Hg. If alveolar ventilation falls to 2 L/min the P_aCO_2 will rise to 80 mm Hg and if alveolar ventilation is increased to 8–9 L/min, the P_aCO_2 will fall to 20–25 mm Hg.

Distribution

The distribution of ventilated gas in the lungs is determined largely by local changes in the transpulmonary or distending pressures. Other factors that may alter air distribution include airway closure, loss of surfactant, decreased elasticity of portions of the lung, partial or complete obstruction of bronchi and increased lung water.

Diffusion

The movement of gases back and forth between plasma and red blood cells is accomplished largely by the process of diffusion. Because CO_2

diffuses so easily, hypercarbia due to impaired diffusion alone is very unlikely. Oxygen is much less diffusible and impaired diffusion may be a significant cause of hypoxemia. The most frequent cause of impaired or decreased diffusion in the lung is an increased distance between the alveolar and the red cell membranes caused by increased interstitial fluid, as may occur in the adult respiratory distress syndrome or congestive heart failure. Even mild changes in capillary permeability may cause an increase in the amount of interstitial fluid and a drop in P_aO_2.

Perfusion

The total volume of blood in the pulmonary circuit is about 500–750 ml in an average adult or about 10–15% of the total blood volume. In patients who are extremely vasoconstricted peripherally, the central blood volume is increased whereas the reverse is true in peripheral vasodilation. Of the blood in the pulmonary circuit, approximately 60–65% is in the pulmonary veins and about 35–40% is in the pulmonary arteries and capillaries. Pulmonary arteries are very compliant and blood flow through the pulmonary vascular bed can often increase 2–4 times with no significant increase in pressure. Blood flow through the lungs is almost equal to cardiac output. This can be estimated by using the Fick principle which correlates the oxygen in the lungs or oxygen consumption by the tissues with cardiac output and the arteriovenous oxygen difference.

Cardiac output (CO) = O_2 consumption/arteriovenous O_2 differences

$$\begin{aligned} CO &= (250 \text{ ml/min})/(5 \text{ vol\%} \times 10) \\ &= 250/10 \\ &= 5 \text{ L/min.} \end{aligned}$$

The average oxygen hemoglobin saturation (SaO_2) in the pulmonary artery is 70–75% and in the systemic arteries it is 95–98%. Blood from the superior vena cava ordinarily has an oxygen content similar to that of mixed venous blood in the pulmonary artery.

Under normal circumstances the entire lung has an average alveolar ventilation of about 4 L/min and a perfusion ($\dot{Q}$) of approximately 5 L/min so that the average $\dot{V}/\dot{Q}$ for the entire lung is about 0.8. Perfusion in the lung is largely determined by gravity. Thus a large fraction of the pulmonary circulation goes to the dependent areas of the lungs. In the upright person there is an increased $\dot{V}/\dot{Q}$ ratio at the apices. The $\dot{V}/\dot{Q}$ ratio is high in areas of the lung where there are increased deadspace areas; ie. areas ventilated but perfused poorly or not at all. The $\dot{V}/\dot{Q}$ is low if there is increased shunting: ie. areas perfused but poorly ventilated.

Air at sea level has a barometric pressure of 760 mm Hg and is made up of 20.93% oxygen and 0.04% carbon dioxide and nitrogen making up the balance. The partial pressures of O_2 and CO_2 at sea level are 159 and 0.3 mm Hg, respectively. When inhaled air is warmed and saturated with water which has the effect of reducing the total partial pressure of the inhaled air by 47 mm Hg to about 713 mm Hg, the inspired oxygen pressure in the

trachea drops to 713 or 149 mm Hg. As water saturated warm air reaches the alveoli, the partial pressure of O_2 is reduced to 104 mm Hg. Oxygen then diffuses through the alveolar capillary membrane into the plasma and into the red blood cells. Carbon dioxide diffuses from the blood into the alveoli. The mixed venous blood brought to the pulmonary capillaries has an average PO_2 of about 40–45 mm Hg and a PCO_2 of 45–50 mm Hg. The alveolar gas tensions that result from the mixing of inspired and venous gases will usually average about 105–110 mm Hg for the PO_2 and 35–45 mm Hg for the PCO_2.

For each milliliter of oxygen that leaves the alveolus, 0.8–1 ml of CO_2 enters it. This relationship is called the respiratory quotient (RQ) which refers to the ratio of the volume of excreted CO_2 to the volume of absorbed O_2. The alveolar oxygen tension P_AO_2 can be calculated by the following formula because alveolar CO_2 (P_ACO_2) is generally equal to arterial CO_2 (P_aCO_2). $P_AO_2 = P_iO_2 - P_ACO_2$ (1.2).

Carbon dioxide diffuses so rapidly that the P_aCO_2 usually provides an excellent index of the adequacy of alveolar ventilation and reflects the ventilation of perfused alveoli. If the P_aCO_2 is greater than normal, one can assume that the ventilation is inadequate and/or the alveoli being ventilated are not adequately perfused, i.e., there is increased dead space. Inadequate ventilation in critically ill or injured patients usually indicates the presence of advanced respiratory acidosis to compensate for a primary metabolic alkalosis.

An elevated P_aCO_2 in the presence of metabolic alkalosis may occur in a patient with normal lungs. However, an elevated P_aCO_2 in a patient with metabolic acidosis generally indicates severe pulmonary insufficiency.

When P_aCO_2 and pH changes are evaluated it is important to remember that the pH is affected by both the bicarbonate and the P_aCO_2, which can change 100–200 times faster then the bicarbonate. Using this information one can often gain some impression of the acuteness of various respiratory changes by noting the effects of the P_aCO_2 on the pH. For each 1 mm Hg acute rise or fall in the P_aCO_2, the pH decreases or increases by 0.01. This assumes that the plasma bicarbonate levels remain relatively constant, as they often will for up to 1 or 2 hours after an acute P_aCO_2 change.

If a patient with a pH of 7.40, P_aCO_2 of 40 mm Hg, and plasma bicarbonate of 24 mEq/L were suddenly to begin hyperventilating and reduce his P_aCO_2 to 30 mm Hg within a few minutes, the plasma bicarbonate would change only minimally and the pH would rise to about 7.50. On the other hand, if the patient's P_aCO_2 change were present for more than a few hours, the plasma bicarbonate would have a chance to compensate somewhat, and the pH change from normal would be less than expected on the basis of the P_aCO_2 alteration alone. For example, if the P_aCO_2 is 60 mm Hg and the pH is 7.35, one can generally assume that the high P_aCO_2 has been present for some time and that the plasma bicarbonate has risen somewhat to partially compensate for the respiratory acidosis.

The arterial PO_2 in normal, healthy young adults under ideal conditions is considered to be about 95–100 mm Hg. The P_aO_2 is extremely important because it not only reflects the functional capabilities of the lungs but also determines the rate at which oxygen enters the tissue cells.