PHYSIOLOGICAL DISTURBANCES DUE TO OPENING AND OPERATING WITHIN THE CHEST*

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TODAY, intrapleural thoracic surgery consists of three main types: (1) surgery of the lungs; (2) surgery of the heart and the great vessels; (3) surgery of the upper gastrointestinal tract via the transthoracic approach.

As the main organs of the thorax are the heart and lungs and their associated blood vessels, let us consider briefly their function in the *closed* chest before attempting to determine how this physiology is disturbed by *opening* and *operating* within the chest.

Since the heart and lungs function as a unit, it is difficult to consider them independently. The main function of this cardiopulmonary unit is to furnish a continuous supply of oxygen and nourishment to all cells of the body and to remove the carbon dioxide and metabolic end products. Thus an accurate knowledge of the partial pressure of both oxygen (arterial p O₂) and carbon dioxide (arterial p CO₂) in the arterial blood is essential to determine the cardiopulmonary function (1).

Normal values for arterial p O₂ range from 71.5 to 100.1 mm Hg.

Values below 70 mm Hg are pathological.

This evidence of oxygenation is much more valuable than an evaluation of the oxygen saturation of the haemoglobin.

Normal values for arterial p CO₂ =

35.0 ± 1.44 mm Hg.

or

46.8 ± 0.73 volumes per cent

To obtain efficient gas exchange in the lungs the following four main factors must be adequate:

I. *Ventilation*, designated as V, or more specifically alveolar ventilation

II. *Distribution*, i.e., gas mixing in the lungs

III. *Diffusion*

IV. *Circulation*

Alveolar ventilation (V) is not only the first and most important single factor, but is the variable that is most readily influenced by the anaesthetist and the surgeon

FIVE CONCEPTS IN CARDIOPULMONARY PHYSIOLOGY (2)

1. The lungs are said to be passive structures in so far as air movement is concerned. Under natural conditions air movement is entirely a result of and directly proportional to the changes in the volume of the thorax caused by movement of

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the ribs and diaphragm. On the other hand, Lucas (3) has drawn our attention more particularly to the dynamics of the bronchial tree, suggesting that the latter is not a passive structure but one which expands and elongates during inspiration and shortens during expiration. The wall of the whole bronchial tree, with the exception of the alveoli, contains an intricate pattern of smooth muscle and elastic tissue. Ciliary action has been described well and stressed rightly by Negus (4, 5) but few anaesthetists give its significance any thought. Tremble (6) has beautifully illustrated with moving pictures "the waving fields of grain" covered by "a blanket of mucus" (4).

Although anaesthetists have a time-honoured maxim "Keep the Airway Clear," their attention is too often concentrated on the *upper* respiratory tract, and a *lower* respiratory obstruction is neglected.

Nosworthy (7) has again reminded us that "it is upon the patency of the bronchiolar lumen and the quiescence of the bronchial reflexes that smooth anaesthesia largely depends." This site is of particular significance as we emphasize the importance of alveolar ventilation.

Incidentally, asthma is considered (8) to be a derangement caused by uneven ventilation of the lungs. The narrowing of the bronchial lumen may vary greatly in different parts of the lung but the major cause of occlusion is viscid secretions rather than spasm of bronchial muscle.

Herxheimer (9) has given attention to the activity and the level of the diaphragm. Emphysema, even in milder degree, is associated with a low but active diaphragm, whereas the obese patient with a large amount of abdominal fat has a relatively high diaphragm that moves poorly.

Another factor influencing the respiratory apparatus is the so-called compliance of the lung, i.e., the inherent ability to distend and collapse.

In addition, the lungs are capable of considerable over-distension before showing signs of impaired function. However, over-distension is a relative matter, for the simple change from the recumbent to the erect position in a normal person actually doubles the size of the lungs, i.e., there is 100 per cent over-distension (10).

2. The lungs and thorax act as the "pump" which brings air to the pulmonary capillary bed for gaseous exchange. Normally there is a tremendous pulmonary reserve, whereas cardiac output can only be increased four or five times.

3. The lung *per se* is an enormous capillary bed, suspended in air, which can transmit a widely variable volume of blood with relatively slight changes in pulmonary artery pressure (2). As the vascular resistance is low, the pulmonary circulation time is extremely short. It will only take about four seconds for blood from the right ventricle to reach the left auricle via the pulmonary system (1).

4. The body is possessed of an adaptive mechanism whereby circulation is shunted from a collapsed or diseased area to a more normal area. Poorly or unventilated but still distended portions of lung have a diminished pulmonary capillary blood flow.

5. The remarkable degree to which the pulmonary capillary bed can be enlarged by increase of cross-section area of the capillaries and perhaps by opening of previously nonperfused capillaries is demonstrated by the fact that, when both

lungs are approximately normal, ligation of a pulmonary artery to one lung causes only transitory elevation of the pulmonary artery pressure (2).

PHYSIOLOGICAL DISTURBANCES

Whereas our knowledge of human cardiopulmonary physiology with the closed chest is developing and changing very considerably, actual studies during thoracic surgery with the open pneumothorax are few. Fourteen years ago, Nosworthy (11) clearly depicted in a classical manner, with linear drawings, the problems arising when the chest is opened. Later Crafoord (12), Organe (13) and others also described this complex situation

When a patient, with pathology in the thorax, is anaesthetized, placed in some specific position, and then has the left or right side of his chest *opened widely* for some intrathoracic surgical procedure, various physiological disturbances may appear. It is therefore a specific challenge to the anaesthetist and the surgeon to foresee and prevent all such disturbances.

Efficient alveolar ventilation, in the fullest sense of the phrase, should be the watchword of every anaesthetist and if it were constantly heeded, almost all of the following undesirable disturbances could be prevented:

Physiological Disturbances

I Disturbances of Respiratory Dynamics

1. Lung collapse and shift of the mediastinum
2. Paradoxical respiration
3. Mediastinal flap or flutter

II. Disturbances due to Lack of Oxygen

III. Disturbances due to Excess Carbon Dioxide

IV. Disturbances of Cardio-Circulatory Dynamics

1. Cardiac output
2. Cardiac arrhythmias
3. Cardiac arrest

V. Disturbances due to Posture

VI. Disturbances due to Reflexes

VII. Disturbances due to Loss of Body Heat

I. *Disturbances of Respiratory Dynamics* (14)

1. *Lung collapse and shift of the mediastinum*

When the pleural cavity is widely opened the previous negative pressure is converted into that of the surrounding atmosphere. The pulmonary elastic tissue tends to collapse the exposed lung. Also the mediastinum moves towards the opposite side, tending to compress the other lung. This latter effect is more marked in the lateral than in the prone position (15). The situation may be aggravated by gravity causing some overload in the pulmonary vascular bed of the lower lung.

Bronchspirometry studies with the patient in the lateral position and the chest closed show that the lower lung has the greater respiratory exchange. However, when the upper side of the chest is open, the tidal volume to the lower

lung is reduced resulting in inadequate ventilation (15). Spontaneous respiration with the open chest is far too turbulent for cardiac surgery and the associated hypoxia and reflex shock from mediastinal flap would be fatal in many of these poor-risk cases (16).

2. *Paradoxical respiration*

This to-and-fro movement of air from one lung to the other makes the movements of the lung on the open side paradoxical, since it collapses with inspiration and expands with expiration.

During such procedures as thoracoplasty, paradoxical respiration may also occur when the stability of the chest wall is lost—even if the pleura remains intact.

3. *Mediastinal flap or flutter*

A further handicap to efficient ventilation is a to-and-fro movement of the whole mediastinum. Oxygen lack and excess carbon dioxide stimulate the respiratory centre to produce exaggerated respiratory efforts. Such efforts in the presence of a mobile mediastinum result in a jerky flap or flutter of the mediastinum. It greatly embarrasses the circulation, resulting in a marked fall in blood pressure and rise in pulse rate, thereby is completed the vicious circle of anoxia.

II. *Disturbances due to Lack of Oxygen*

The cells in the vital centres of the medulla of the central nervous system and in the "specific tissue," as well as the ventricular muscle tissue of the heart, are those most vitally in need of a *continuously* adequate supply of oxygen.

III. *Disturbances due to Excess Carbon Dioxide*

Oxygen lack and excess carbon dioxide must be closely linked in all our thinking because the effects of each are very closely related and often overlap.

Gibbon (1950) and Etsten (1953) have concluded that the open pneumothorax and not the particular posture (whether lateral or prone) causes inadequate alveolar ventilation and consequently excessive retention of carbon dioxide.

All grades of respiratory acidosis during intrathoracic surgery have been reported (17).

IV. *Disturbances of Cardio-Circulatory Dynamics*

1. *Cardiac output*

(a) Inefficient spontaneous breathing as well as some types of pressure breathing may cause impairment of cardiac output (16, 18, 19).

(b) Cardiac handling and surgery may produce a low cardiac output with a fall in peripheral resistance, which responds to vasopressor drugs (20).

(c) Vasopressor drugs *per se* may lower the cardiac output (21).

(d) Inadequate blood replacement or excessive replacement may interfere with an adequate output of the heart.

Inadequate coronary blood flow may sometimes be directly improved by intra-aortic transfusion of blood.

2. *Cardiac arrhythmias*

During cardiac surgery, direct stimulation of the heart may be the cause of cardiac arrhythmias. These are most readily corrected by having the surgeon desist from constant handling of the heart (22).

Johnstone (23) has reported that retention of carbon dioxide is the cause of the ventricular arrhythmias associated with cyclopropane or pentothal anaesthesia. Such arrhythmias can occur in spite of good oxygenation.

3. *Cardiac arrest*

(a) *Cardiac standstill* may be defined as a very sudden failure of the heart to pump blood. It is many times more common than ventricular fibrillation (24). Reid thinks that there is a "trigger reflex mechanism" which eventually precipitates the condition but that there are various underlying causes. Of the latter, excess carbon dioxide is seriously incriminated, as well as lack of oxygen. In over 1500 cases of cardiac arrest, none were apparently adequately atropinized. We should consider cardiac arrest as an avoidable situation rather than one to be dramatized by spectacular therapy.

(b) *Ventricular fibrillation* It is generally conceded that the prelude to ventricular tachycardia and fibrillation consists in the development of multiple ventricular extrasystoles.

Ventricular fibrillation is described (26) as an unco-ordinated type of contraction which produces no useful beats. There are typical "rippling" movements of the ventricular muscle which give the impression to the hand of a "bag of worms" (M Thorek, Chicago).

The phenomenon of post hypercapnoeic ventricular fibrillation is a striking one. The relationship between potassium and hypercapnia is a fascinating study (27). Sealy *et al.* suggested that the rise in potassium levels after the cessation of hypercapnia *may* be due to a sudden release of epinephrine.

V. *Disturbances due to Posture* (15, 17)

1. Ventilatory
2. Problem of Secretions
3. Cardiac Function

VI. *Disturbances due to Reflexes*

Nosworthy (7) has emphasized the relative value of the stimulus and the threshold of irritability of the respiratory tract, indicating that the sensitivity increases at its lower levels—e.g. bronchial reflexes may remain acutely sensitive.

When a patient "reacts," the expiratory muscular effort against a spastic glottis or bronchospasm impedes the blood flow to the left auricle. This causes an immediate fall in cardiac output and a severe drop in systolic blood pressure. The pressure in the pulmonary artery and right ventricle increases, resulting in a rise in venous pressure, which makes distended veins and cyanosis in the "cape region" very noticeable to the anaesthetist.

(The significance of the cough reflex must never be forgotten)

Many other reflexes are associated with vagal stimulation but their real significance in anaesthesia has not yet been proven.

VII. *Disturbances due to Loss of Body Heat* (11)

Even when all loss of blood, fluids, and electrolytes is made good, loss of body heat is a deciding factor in how much can be done inside the chest. Loss of heat is considerable when the chest wall is widely opened and the time factor is important.

RÉSUMÉ

Les organes principaux du thorax, le cœur et les poumons fonctionnent en synergie et il est difficile de les considérer indépendamment. Leur fonction en cavité thoracique close est mieux connue de jour en jour et doit être mieux comprise avant qu'il soit possible de déterminer ce qui en perturbe la physiologie quand on ouvre et opère dans le thorax.

La fonction principale de cet ensemble cardio-pulmonaire est de fournir un apport constant d'oxygène et d'éléments nutritifs à toutes les cellules de l'organisme et aussi d'évacuer le dioxyde de carbone et les déchets métaboliques.

Un échange gazeux efficace dans le poumon requiert la fonction efficace de quatre principaux facteurs:

- I. La ventilation, désignée par V; ou plus précisément la ventilation alvéolaire
- II. La distribution, $\dot{V}_A$, le mélange des gas dans le poumon
- III. La diffusion
- IV. La circulation

La ventilation alvéolaire (V) n'est pas seulement le premier et le plus important facteur mais aussi celui qui est le plus facilement modifié par l'anesthésiste et le chirurgien.

Les études actuelles au cours d'interventions thoraciques avec pneumothorax ouvert sont peu nombreuses. Il y a quatorze ans, Noseworthy décrivit clairement d'une façon classique avec tracés linéaires, les problèmes que pose l'ouverture du thorax.

Une ventilation alvéolaire efficace dans tout le sens de ce qu'elle implique devrait être le but de tout anesthésiste. Si elle est maintenue de façon constante, presque toutes les perturbations nuisibles énumérées plus bas pourront être évitées.

Perturbations physiologiques

- I. Perturbations de la dynamique respiratoire
 1. Collapsus pulmonaire et déplacement du médiastin
 2. Respiration paradoxale
 3. Flutter médiastinal
- II. Perturbations dues au manque d'oxygène
- III. Perturbations dues à un excès de dioxyde de carbone
- IV. Perturbations de la dynamique cardio-vasculaire
 1. Débit cardiaque
 2. Arythmie cardiaque
 3. Arrêt cardiaque
- V. Perturbations dues à la position du patient
- VI. Perturbations dues aux reflexes
- VII. Perturbations dues à des pertes de chaleur corporelle

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