PHYSIOLOGICAL CONSIDERATION OF RESPIRATORY DISEASE

A Symposium

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RESPIRATORY AND PULMONARY PHYSIOLOGY

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NOTES ON PHYSIOLOGICAL BASIS IN CLINICAL PEDIATRICS SITUATIONS WHICH ALTER PULMONARY VENTILATION

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CYSTIC DISEASE AND EMPHYSEMA OF LUNGS, CONGENITAL AND ACQUIRED

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POLIOMYELITIS AS IT AFFECTS RESPIRATION

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A. Physiologic Factors Concerned in the Regulation of Respiration

Physiologists now agree that there is a medullary respiratory center which has intrinsic rhythmicity. Nevertheless this center can be influenced profoundly by many chemical and nervous factors. One of the most important of these is carbon dioxide. Under ordinary conditions the medullary center is exquisitely sensitive to changes in carbon dioxide pressure. When the respiratory center is depressed (by deep anesthesia, large doses of morphine or barbiturates, trauma, cerebral edema, increased intracranial pressure, severe anoxia or by high concentrations of carbon dioxide itself) it is no longer responsive to carbon dioxide though it may still permit reflex activity and continuation of respiration.

Anoxemia may also stimulate respiration; this occurs through reflexes originating in chemoreceptors of the carotid and aortic bodies. It appears certain that these chemoreceptors are functioning in the normal full-term newborn though they may not be functioning or functioning properly in prematures. When these chemoreceptors are in operation, anoxia will stimulate respiration and oxygen therapy will abolish such hyperventilation. When the chemoreceptors are not in action, one would expect no reflex effects from either oxygen or anoxia; oxygen therapy, however, might relieve cerebral ischemia and permit respiration to improve.

B. Physiologic Methods for Evaluating Respiratory and Pulmonary Function

The function of the lungs is primarily to oxygenate the venous blood and to remove excess carbon dioxide from it. To accomplish this, there must be normal respiratory volumes, normal lung volumes and aerating surface, even distribution of the inspired gas to the alveoli, unimpaired diffusion across the alveolar capillary membrane, and uniform distribution of pulmonary capillary blood flow to the functioning alveoli. Tests were described which permit the measurement of these factors in patients with pulmonary disorders. It is recognized that data obtained from these tests often does not permit the physician to determine why a patient is dyspneic. Newer tests measuring the total work of breathing and the work of the various component parts of breathing should shed new light upon the problem of dyspnea.

NOTES ON PHYSIOLOGICAL BASIS IN CLINICAL PEDIATRICS SITUATIONS WHICH ALTER PULMONARY VENTILATION

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A. Clinical Recognition of Abnormal Respiration

There is no simple or direct way by physiologic laboratory technics to determine alveolar ventilation, which is the measure of efficiency of absorption of oxygen or secretion of CO₂. Alveolar ventilation is a complex resulting from many factors, some of which can be measured, but the sum of all the studies cannot give a direct answer. Studies of CO₂ content and pH with estimation of alveolar pCO₂ are the best laboratory aids at present available. The oxygen saturation hemoglobin is not as reliable a guide for the detection of deficiencies of alveolar ventilation.

Observation of a patient by a skilled clinician can go a long way towards evaluating alveolar ventilation. The recognition and evaluation of dyspnea is best accomplished by simple but informed observation. One studies in an orderly fashion and with some attempt at quantitative evaluation (a) rate, (b) rhythm, (c) depth and (d) the shape of the respiratory curve. The last is the hardest to evaluate. The rate, efficiency of the shape, the depth of respiration modified by its regularity or irregularity, results in a more or less efficient pulmonary ventilation.

It is important to estimate the effectiveness of respiratory effort and to realize that an effective respiration resulting in normal alveolar ventilation may be made at the cost of great effort, which eventually may fail. One, therefore, cannot be satisfied only with a physiologic estimation of the results of respiratory effort, but must also evaluate the effort that is being used to produce this respiration.

The technic of peroral auscultation is of great aid in evaluating the depth of respiration as well as any interference by partial respiratory obstruction. Listening with a stethoscope over the nose and mouth of a patient can enable one to get a fairly accurate idea of the "curve" of respiration, and so its efficiency. A simple diagram is used for illustration.
b. Cause of abnormal pulmonary ventilation

I. Primary disturbance of the nervous control of respiration caused by CNS pathology.
   a. Meningitis
   b. Encephalitis
   c. Poisoning
   d. Anoxia (particularly in the newborn)

Any of these conditions can cause a primary disturbance of the respiratory center. In the case of meningitis and encephalitis, usually manifested by hyperpnea, though not necessarily, any gross irregularity of breathing may result. The first symptom of meningitis or encephalitis in an infant may be an hyperpnea or a grossly irregular respiration. Anoxia usually does not result in hyperpnea, that is, severe anoxia, but quickly depresses respiratory effort or makes it irregular. The first effect of anoxia, however, may result in hyperventilation. Irregularities of breathing are more common than either a slow depressed respiration or an hyperpnea.

II. Caused by disturbances in acid base equilibrium.
   a. Vomiting
   b. Diarrhea and dehydration with secondary kidney defect
   c. Primary kidney defect
   d. Poisoning

We are familiar with hyperpnea as a compensating mechanism to prevent a great change in pH resulting from the accumulation of some foreign acid element in the blood serum, such as in ketosis, which specifically results from starvation or vomiting in infants. As common is the accumulation of chloride and organic acid due to renal dysfunction, itself the result of dehydration from diarrhea. In these circumstances CO₂ is excreted to make room for other but abnormal acid radicals.

Poisoning, such as with ammonium chloride, can result in a direct CNS stimulation of respiration due to a true acidosis.

III. Primary pulmonary pathology

Here we have to consider obstructive lesions in the respiratory tract, asthma, bronchitis, capillary bronchitis, or the reflex disturbance of respiration which we get in a case of lobar pneumonia, where we have rapid shallow respiration, not due to important encroachment on the pulmonary space but due to vagal reflexes. Accumulation of CO₂ may result.

IV. Defects in circulation directly interfering with ventilation.

V. Neuromuscular defects such as congenital defects with hypotonia or amyotonia, or such paralysis as poliomyelitis with respiratory muscle involvement. These lead to direct respiratory failure with CO₂ acidosis as well as anoxia.

CYSTIC DISEASE AND EMPHYSEMA OF LUNGS, CONGENITAL AND ACQUIRED

WILLIS J. POTTS, M.D.

In the otherwise normal infant there are 2 primary reasons for inadequate respiration: obstruction in the air passages and inadequate absorption area. The former is the field of the endoscopist. A few conditions illustrative of the latter will be discussed.

Mediastinal emphysema is caused by too vigorous artificial respiration in the newborn, coughing, choking, asthma, etc. If an alveolus breaks in the lung mediastinal emphysema may follow. If the alveolus breaks through the pleura pneumothorax results and requires emergency treatment.

Lobar emphysema in infants may be due to obstruction in a bronchus or it may develop without demonstrable cause. If no obstruction in the bronchus exists and if the offending emphysematous lobe causes respiratory distress it must be removed.

Congenital cysts of the lung are fairly common in early infancy and produce severe dyspnea and cyanosis. Emergency relief is often necessary. Surgical removal of the offending lobe yields excellent results.

Acquired cysts of the lung in infancy usually occur in connection with staphylococci pneumonia. The cysts vary greatly in size and therefore may or may not produce symptoms. The treatment is definitely conservative.

These conditions will be illustrated with lantern slides.
Although its paralytic effect on the respiratory motor unit is probably the most common respiratory defect due to poliomyelitis, the disease frequently and seriously affects many other aspects of the respiratory system. Disturbances of reflex and secretory activities in the pharynx produce troublesome respiratory complications. Medullary involvement may critically affect both the nervous and chemical aspects of the control centers. Closely related involvement of circulatory centers in the medulla affect respiration indirectly, particularly because of the close interrelationships of pulmonary circulation and lung function. Circulatory along with other possible factors operating in the lungs lead to pulmonary edema and atelectasis. The latter respiratory complications of poliomyelitis interfere seriously with oxygen supply to the body, whereas deficiency of ventilation due to muscle paralysis or other factors leads primarily to carbon dioxide retention and respiratory acidosis.

Measures designed to correct respiratory dysfunction must be considered as to whether they specifically correct a defect and whether they may at the same time introduce harm or hazard. Respiratory obstruction urgently requires correction by postural drainage, aspiration and sometimes tracheotomy; a too-small tracheotomy tube constitutes respiratory obstruction and the airway is sometimes further compromised by insertion of a catheter supplying oxygen. Ventilatory deficiency is primarily a problem in carbon dioxide removal and must be treated by increasing ventilation; oxygen administered without attention to ventilation may make respiratory acidosis worse. Several methods are available for increasing pulmonary ventilation; the body respirator is rightly the most widely used and safest. The physiologic effects of the respirator are the same as those of pressure breathing given by a mask over the face. Combinations of the 2 methods of applying pressure have been used for nursing convenience and for increasing ventilation when pulmonary edema or high airway resistance increase the amount of pressure necessary to produce an adequate tidal volume. The best guide to the adequacy of pulmonary ventilation is measurement of alveolar carbon dioxide or arterial pH and carbon dioxide. If ventilation is satisfactory and hypoxia remains, oxygen should be added to the inspired air. However, hypoxia may be present on a circulatory basis in the presence of an adequate or even excessive ventilation; in this event, the circulatory depression may be due to excessive negative pressure in the tank and may be corrected by addition of a positive expiratory phase, with corresponding decrease of the negativity during inspiration.

Other aids to respiration suitable for special needs are rocking beds, chest respirators, and apparatus for electrically stimulating the phrenic nerve.
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