Significance of Respiratory Dead Space Alterations

Since 1905, when Haldane and Priestley first estimated the size of the respiratory dead space in man from expired and alveolar CO₂ concentrations, many modifications of their CO₂ technique have been proposed. It was not, however, until Fowler’s studies in 1948 that a relatively simple and reproducible technique was devised for measuring anatomic dead space. With this new method, interest was stimulated in studying the changes in respiratory dead space under various physiological and clinical situations.

Dead space is simply the volume of gas in the respiratory tract which does not participate in the gas exchange between pulmonary capillary blood. The volume, known as physiologic dead space, is subdivided into anatomic and alveolar dead spaces. The anatomic dead space is represented by the transporting or conducting airways, only: nose, mouth, pharynx, larynx, trachea, and bronchial tree down to the level of the air sacs and alveoli which participate in gas exchange. The alveolar dead space is that volume contained in purportedly inadequately functioning alveoli. These are the alveoli that are adequately ventilated but inadequately perfused, or adequately perfused but over ventilated. The alveolar dead space may be virtually non-existent in healthy subjects, or it may be greater than the anatomic dead space as in some emphysematous patients.

Anatomic dead space is usually measured by the nitrogen washout technique in which the degree of dilution of alveolar nitrogen is measured following a single breath of oxygen. It may also be measured by a comparison of mean expired and alveolar CO₂ concentrations. The degree to which alveolar CO₂ is diluted by dead space gas is reflected in the expired CO₂ according to the Bohr equation. Physiological dead space is similarly determined, but the comparison is made between mean expired and arterial CO₂ concentrations. Alveolar dead space is the difference between the physiologic and anatomic spaces and is never measured independently.

The physiological dead space is a dynamic volume, normally influenced by many factors. Some of these have been identified through in vitro studies, others by in vivo studies, in animals and man. Those with the capacity to alter dead space significantly include tidal volume, respiratory rate, pattern of breathing, lung volume, pulmonary blood flow, bodily position, alveolar P₉0₂, and alterations in bronchomotor tone. With the exception of atropine, the effect of drugs used in anesthesia on dead space has been poorly studied. (See reviews of Rossier and Buhmann, Dripps and Severinghaus, and Widdicombe.) Since all general anesthetics exert marked effects on circulation and respiration, large changes in dead space should and do result from general anesthesia.

In this issue of the Journal, Askrog, Pender, and Eckenhoff describe a well documented increase in dead space occurring during operation performed under deliberate hypotension. The significance of this change is difficult to interpret. In their study a hypotensive agent and simultaneous head-up tilt increased both
anatomical and alveolar dead spaces. This change was incompletely reversed by a return to the supine position. The mean maximum increase in physiological dead space was 62 ml. A change of this magnitude, however, has been observed in unanesthetized normotensive subjects changed from the supine to the erect position, suggesting that it is a normal physiological response. Despite this, the remarkable correlation between increase in dead space and degree of hypotension strongly suggests that decreased pulmonary blood flow was primarily responsible for the increase in dead space. In spite of adequate alveolar ventilation, relatively poor perfusion of the alveoli led to the large increase in physiological dead space.

To compensate for increased dead space from this cause, alveolar capillary perfusion must be increased by increasing cardiac output, or the oxygen tension of the inspired gas must be increased. Increasing the overall pulmonary ventilation alone will not improve oxygenation. Indeed, increased ventilation, if associated with an increased mean airway pressure, may further decrease perfusion and increase dead space. In contrast, increases in anatomic dead space may be adequately compensated for by increased ventilation. In this case, increasing minute volume of ventilation by increasing the amount of inspired gas presented to the alveoli will maintain oxygenation and CO₂ elimination.

It is apparent from this discussion that the role of dead space changes in the adequacy of respiration in the anesthetized patient is not clearly delineated. It is also apparent that work of the calibrator presented in this issue by Askrog and his colleagues provides important data which contribute to the eventual understanding of the dynamics of the dead space.

Stanley C. Allison
William I. Kracke
Arthur S. Keats, M. D.
Division of Anesthesiology
Baylor University College of Medicine
Houston, Texas

References

Phantom Limb

Nerve block anesthesia, apart from its usefulness as an anesthetic technique, provides the anesthesiologist with a fascinating opportunity to study the physiologic effects of nerve sections at first hand. Thus significant contributions to knowledge of the anatomical pathways involved and also of the physiological function of nerve fibers of different sizes and peripheral sensory receptors have already been made and will continue to be made if one is prepared to carefully examine the patient before and after block. That complex experimental techniques are not necessarily required is well demonstrated in the article, "Phantom Sensations During Spinal Anesthesia," which appears elsewhere in this issue.

The authors have convincingly demonstrated that sensory information, in this instance position sense of an extremity, is retained proximal to the site of block, presumably at the spinal cord level, and persists at least for the duration of the block. This neu-