

## STUDIES ON THE PHYSIOPATHOLOGY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN THE HORSE. VI. THE ALVEOLAR DEAD SPACE

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### ABSTRACT

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The alveolar dead space (VD<sub>alv</sub>) as a percentage of tidal volume was calculated in horses by substituting the values for partial pressure of carbon dioxide in end-expiratory gas (PE'CO<sub>2</sub>) and for partial pressure of carbon dioxide in arterial blood (PaCO<sub>2</sub>) in the equation:

$$\% \text{VD}_{alv} = \frac{\text{PaCO}_2 - \text{PE}'\text{CO}_2}{\text{PaCO}_2} \times 100$$

The mean % VD<sub>alv</sub> of 12 chronic obstructive pulmonary disease (COPD) subjects was 3 times greater than that of 22 normal subjects. Since the larger % VD<sub>alv</sub> of COPD subjects was due to an elevated PaCO<sub>2</sub>, it was considered that maldistribution of ventilation was the principal cause of their increased % VD<sub>alv</sub> compared with that of clinically normal subjects.

### INTRODUCTION

Alveolar dead space is defined as that fraction of alveolar ventilation in which gas exchange is absent or deficient (Nunn, 1969).

The major portion (66%–80%) of alveolar dead space is due to uneven distribution of blood flow, according to Severinghaus & Stupfel (1957), who also showed that the alveolar dead space may be calculated as a percentage of the tidal volume by means of the equation:

$$\% \text{VD}_{alv} = \frac{\text{PaCO}_2 - \text{PE}'\text{CO}_2}{\text{PaCO}_2} \times 100 \quad (1)$$

where % VD<sub>alv</sub> = percentage alveolar dead space  
PE'CO<sub>2</sub> = partial pressure of carbon dioxide in end-expiratory gas  
PaCO<sub>2</sub> = partial pressure of carbon dioxide in arterial blood

The validity of the above equation was subsequently confirmed by Julian, Travis, Robin & Crump (1960) and by Nunn & Hill (1960). The equation was used by Mitchell & Littlejohn (1972) to estimate alveolar dead space in adult horses and by Littlejohn & Van Heerden (1975) for similar studies in foals.

Although Weaver & Walley (1975) recorded an abnormally large alveolar dead space in an anaesthetized subject with chronic obstructive pulmonary disease (COPD), measurements of alveolar dead space in conscious horses with COPD have not so far been reported. Since emphysema causes an increase in the alveolar dead space in man (Hugh-Jones, 1958; Nunn, 1969), it was considered relevant to investigate this aspect of ventilation in horses with COPD.

The studies reported here were designed to compare the magnitude of the alveolar dead space in horses with COPD with that in clinically normal horses.

### MATERIALS AND METHODS

#### Subjects

Thirteen subjects with COPD as determined by clinical examination (Littlejohn, 1980) and 24 clinically normal horses and ponies were investigated. All subjects were unpremedicated and all were conscious and in the standing position.

#### Techniques

The PaCO<sub>2</sub> was determined by methods previously described (Littlejohn, 1981). End-expiratory gas was collected simultaneously by the technique of Littlejohn

(1969) and the PE'CO<sub>2</sub> determined with a Radiometer PCO<sub>2</sub> electrode type E5036.

The alveolar dead space was calculated by substituting the values obtained in Equation (1).

Negative values for % VD<sub>alv</sub> were obtained in 2 normal subjects and in 1 with COPD. These 3 values were attributed to technical error (Scheid & Piiper, 1980) and discarded.

### RESULTS

The mean values and standard deviations for PaCO<sub>2</sub>, PE'CO<sub>2</sub> and % VD<sub>alv</sub> in 22 normal and 12 COPD subjects are shown in Table 1.

There was a significantly larger % VD<sub>alv</sub> in the COPD horses for which a mean value of 19.98% was obtained compared with a mean of 6.1% for normal horses. Mean end-expiratory values for PCO<sub>2</sub> did not differ significantly. The increased % VD<sub>alv</sub> in COPD subjects was therefore due to an increase in their PaCO<sub>2</sub> values.

### DISCUSSION

The %VD<sub>alv</sub> of 6.1% which was obtained in clinically normal horses is somewhat larger than that obtained by Littlejohn (1969) in 14 normal resting subjects. From that study, the mean % VD<sub>alv</sub> was calculated to be 2.7%. The reason for the greater % VD<sub>alv</sub> in normal subjects in the present studies is not clear, but the increase may be associated with environmental variables present at the altitude of 1 300 m (such as ambient temperature and barometric pressure) or to minor differences in technique.

Factors influencing the magnitude of the alveolar dead space were discussed by Severinghaus & Stupfel (1957) and by Nunn (1969). These factors may be summarized as follows:

- (1) Hydrostatic failure of alveolar perfusion. The COPD subjects of the present study had a mean pulmonary arterial pressure of 50 mmHg. Thus any deficiencies of perfusion may have been due to a mechanical limitation of the capillary flow rather than to circulatory insufficiency leading to pulmonary hypotension.
- (2) Ventilation of non-vascular air-spaces. According to Donald, Renzetti, Riley & Cour-nand (1952), this is the major cause of the increased physiological dead space which is seen in human subjects with chronic lung disease.



- (3) Perfusion of non-ventilated or poorly ventilated alveoli.

In normal subjects this factor may account for 20% of the total alveolar dead space (Severinghaus & Stupfel, 1957).

- (4) Inequalities of ventilation and perfusion.

Perfusion of non-ventilated or poorly-ventilated alveoli functions as a right-to-left shunt thus elevating the  $\text{PaCO}_2$  in equation (1) (Severinghaus & Stupfel, 1957).

An excess of non-perfused alveoli thus enlarges the alveolar dead space by lowering the end-expiratory  $\text{PCO}_2$ , whereas an increase in the number of perfused alveoli which have poor or non-existent ventilation enlarges the alveolar dead space by raising the  $\text{PaCO}_2$ .

In our COPD subjects the mean  $\text{PE}'\text{CO}_2$  was not significantly different from that in the normal subjects. The mean  $\text{PaCO}_2$  value of COPD subjects was significantly higher (by 6,3 mmHg) than that of the clinically normal subjects.

The results therefore suggest that uneven distribution of ventilation was the principal cause of increased alveolar dead space in our series of COPD horses, and that consequently there was gross mismatching of ventilation to perfusion.

According to Nunn (1969) maldistribution of inspired gas in diseased lungs is usually associated with sequential rather than synchronous emptying of alveoli. Furthermore, in cases of emphysema in man, areas of the

lung with a defective ventilation are slow to empty and fill with gas. In functional terms, alveolar ventilation is reduced and alveolar dead space is increased (Nunn, 1969). Such an effect may exist in horses with COPD and it was assumed by Gillespie & Tyler (1969) that there must be an uneven distribution of ventilation in the lungs of horses with emphysema. The demonstration of an increased %  $\text{VD}_{\text{alv}}$  supports that assumption, which was based on known facts regarding chronic pulmonary disease in man. As stressed by Gillespie & Tyler (1969), the possibilities for varying combinations of ventilation and perfusion in diseased lungs are infinite.

Apart from the one observation by Weaver & Walley (1975) no comparisons between the alveolar dead space of COPD and normal horses appear to have been made. However, the findings of the present study suggest that it is a simple and informative determination which provides corroborative evidence of lung dysfunction and its effects on ventilation and perfusion.

From a correct statistical point of view the 2 sample populations of %  $\text{VD}_{\text{alv}}$  are not comparable. Applying Snedecor's 'F' test to the data gives an unacceptably large variance ratio of 5,65. This is undoubtedly due to the skew distribution of the normal values and the wide variation of the abnormal values shown in Fig. 1. Examination of the 2 histograms however leaves little doubt that the %  $\text{VD}_{\text{alv}}$  of a horse may be of clinical and functional significance.

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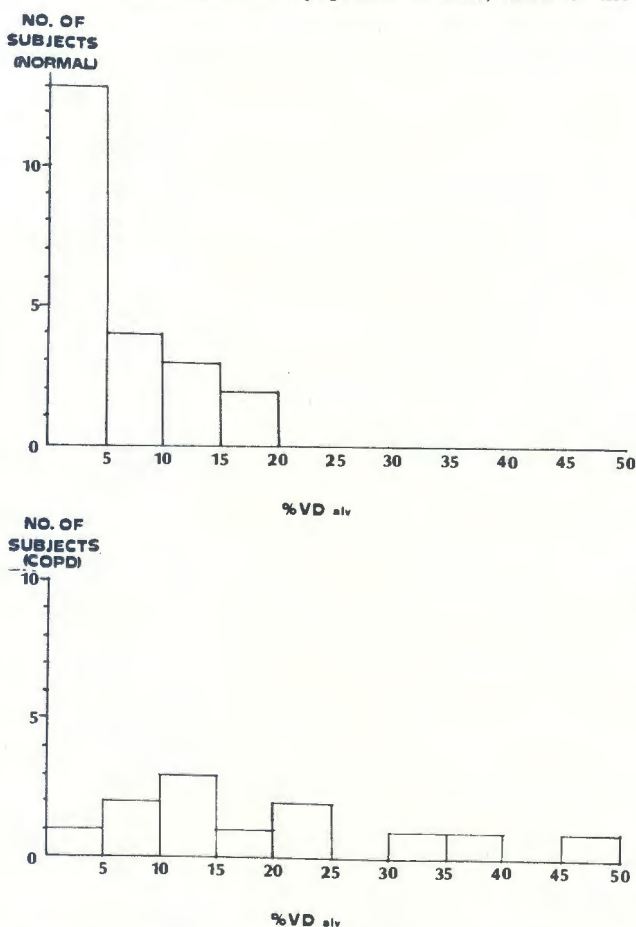


FIG. 1. Distribution of percentage alveolar dead space in 22 normal and 12 COPD subjects.