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# RESPIRATORY ADJUSTMENTS TO INCREASES IN EXTERNAL DEAD SPACE

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ALTHOUGH it has been a matter of common knowledge that increases in external dead space would cause an increase in tidal and minute volumes in subjects able to increase their ventilation, the quantitative aspects of those increases, particularly with quite small dead space increments, have not been well delineated. This investigation was undertaken to determine the effects of such increases in external dead space on certain respiratory functions.

Stannard and Russ (1) utilized a total of 250 ml, as their smallest external dead space. Their subjects breathed oxygen, and end-expiratory carbon dioxide tension was determined chemically. They felt that alveolar pCO<sub>2</sub> did not change significantly under resting conditions until a total external dead space of over 500 ml, was reached. They also concluded that changes to resistance in breathing were subjectively the most noticeable element of difference between the various dead spaces and felt that particularly with the larger dead spaces the resistance factors might outweigh the rebreathing factor. Rahn, and others (2), on the other hand, felt that average alveolar CO<sub>2</sub> was not changed by increases in positive and negative pressures. Otis, Rahn and others (3-9) have done work with increased dead spaces, but for other purposes and under different conditions than the present investigation. Recent work has been done on carbon dioxide homeostasis in anesthetized patients (10).

The question of whether or not respiratory adjustments to relatively small external dead space increases prevented a rise in CO<sub>2</sub> tension or were only partially complete and the quantitative aspects of these changes were the primary reason for this investigation.

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#### Метнор

A pneumotachograph was mounted on the inspiratory side of a nonrebreathing valve. An infrared CO<sub>2</sub> analyzer was connected to the expiratory limb of the valve, and the valve was connected to the subject by means of a rubber mouthpiece. Respiratory volumes were determined by means of a dry displacement gas meter. Continuous recordings of expired CO<sub>2</sub> and respiration were made on a twin-channel recorder. The CO<sub>2</sub> analyzer was calibrated with known concentrations of carbon dioxide. Two sizes of dead space were used. The smaller, consisting of a rubber mouthpiece and the nonrebreathing valve, measured 40 ml. The larger consisted of a piece of large bore rubber tubing measuring 125 ml. and was attached between the previously mentioned mouthpiece and the nonrebreathing valve, to give a total external dead space of 165 ml. The pneumotachograph was used to

TABLE 1 Changes in End-Expiratory  $pCO_2$  and Tidal Volume

Subject	40 cc. Dead Space		125 cc. Dead Space	
	pCO <sub>2</sub> mm.Hg	TV ec.	pCO <sub>2</sub> mm.Hg	TV ec.
1	38.8*	527*	40.2*	630
2	37.8	497	40.9	572
3	38.6	567	39.8	705
4	38.8	873	41.2	1151
5	34.7	452	36.0	565
6	35.9	544	36.8	674

<sup>\*</sup> Each value is average of approximately 6 determinations.

give an evaluation of adequacy of individual respirations for measuring end-expiratory CO<sub>2</sub> levels and not for actual volume measurements.

Normal adult males were used as subjects. Determinations were made with subjects in the supine position, in a quiet, resting state. Each subject was connected to the apparatus for approximately ten to fifteen minutes before a series of readings was started. Observations were made during periods of 3 minutes, and several such periods were recorded with each subject. Dead spaces were then changed, and time was allowed after the changes until successive determinations of minute volume became stable. Insofar as possible, an attempt was made to keep the subjects from being aware of the changes in the dead space and when these changes were made. There was very little difference between respiratory resistances noted by the subjects with 40 ml. or 125 ml. added dead space. Levels of CO<sub>2</sub> were read directly from the recorder and converted to pCO<sub>2</sub> utilizing a calibration curve for CO<sub>2</sub> in air and correcting for atmospheric pressure.

TABLE 2
CHANGES IN MINUTE VOLUME
COMPARISON OF MINUTE VOLUME CHANGES AND CALCULATED INCREASED DEAD
SPACE MINUTE VOLUME

Subject	40 cc. MV1 cc.	125 ce. MV2 ce.	$MV_1 - MV_1$	125 × R.R.
ī	6405*	7684*	1279°	1542*
2	6114	7990	1876	1750
3	4908	5708	800	1014
4	7122	7801	679	829
5	6072	7339	1267	1641
6	6744	6973	229	1313

<sup>\*</sup> Each value is average of approximately 6 determinations.

#### RESULTS

Table 1 indicates the changes in tidal volume and  $pCO_z$ , averaged for each subject. Tidal volume and  $pCO_z$  both increased, but in general the tidal volume increased less than the amount of added dead space.

Table 2 expresses the changes in minute volume, again averaged for each subject. In all except one case this was quite pronounced. In addition, the increases in minute volume are compared to the increased dead space times the respiratory rate. In every case except one, the increased minute volume was less than the calculated increased dead space minute volume, which may provide an explanation for the increases in end-expiratory pCO<sub>2</sub>. However, there was a rather poor correlation between the individual minute volume discrepancy and the individual quantitative change in expired CO<sub>2</sub> tension.

Figure 1 expresses the results graphically, here averaged for all six subjects, for tidal volume, minute volume, and expiratory  $pCO_2$ , and the comparison between minute volume increase and increased dead

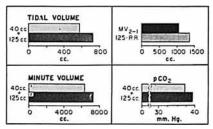


Fig. 1. Changes in tidal volume, minute volume, and minute volume increase as compared to increased dead space minute volume and end-expiratory pCO, changes, expressed graphically. (Average of 6 subjects.)

<sup>†</sup> Respiratory rate.

space minute volume. The pCO<sub>2</sub> graph has been split and extended to emphasize the approximately 5 per cent increase in end-expiratory pCO<sub>2</sub>. The changes in tidal volume, minute volume, and end-expiratory pCO2, prior to averaging them for all six subjects, were analyzed for statistical significance by the Fisher "paired t" test, and were all found to be highly significant (p less than 0.01, greater than 0.001).

#### Discussion

This investigation would appear to indicate that the small increases in external dead space found particularly in anesthesia equipment, inhalation therapy apparatus, and devices used in measuring respiratory functions are indeed of significance, even in normal subjects who are able to compensate at least partially. In the presence of disease and depression produced by a variety of drugs, where the patient is rendered incapable of compensating by himself, this increase of dead space would seem to assume even greater importance. This would apply especially in the patient with severe pulmonary disease who may already be operating at or near maximum ventilatory capacity. The relatively small decrease in dead space obtained by tracheotomy might well be of help.

#### Conclusions

Dead space increases of about 125 ml. added to a "minimum" external dead space of 40 cc. cause statistically significant changes in tidal and minute volume and end-expiratory pCO2 in unanesthetized normal subjects under quiet, resting conditions. This indicates that even in unmedicated normal subjects, the adjustments to increased dead space of this degree are not entirely complete. The factor of dead space, particularly in diagnostic and research apparatus, should be considered thoroughly since it is conceivable that less than optimum accuracy might be obtained with increases in dead space formerly considered insignificant.

#### SUMMARY

Respiratory responses of a group of normal, unanesthetized subjects to a small (125 cc.) increase in external dead space were determined, under resting conditions. A statistically significant elevation of end-expiratory pCO<sub>2</sub> was found, as well as significant increases in tidal and minute volumes. Minute volumes, however, increased less than the calculated increased dead space minute volumes.

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