INFLUENCE OF INCREASED ANATOMICAL DEAD SPACE NO FUNCTIONAL RESIDUAL CAPACITY OF LUNGS AND THE LUNG CLEARANCE INDEX

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Summary: A study of the influence of added anatomical dead space on the functional residual capacity of the lung was carried out on a total number of 40 healthy subjects of ages ranging between 21 and 50 years. The functional residual capacity was found to decrease as additional dead space was introduced in the breathing circuit. This decrease was associated with an increase in tidal volume. lung functions, as given by the lung clearance index, were found to deteriorate with each addition of dead space. There was also an increase in pulse rate in all age groups.

Key words: anatomical dead space, functional residual capacity, tidal volume, lung clearance index

INTRODUCTION

Normal ventilation is dependent, among other things, on patent airways. Any addition of airways or dead space is apt to disturb the normal respiratory mechanics. Homeostatic mechanisms, however, operate to maintain the status quo in pulmonary functions by changing the tidal volume (TV) or respiratory rate or both. By promoting compensation in one direction, such regulatory mechanisms may lead to decompensation in other directions and may produce changes in functional residual capacity (FRC) also. This parameter must be kept as close to the optimum as possible. Both an increase or decrease in this parameter are against the patients interest.

Anatomical dead space increases in a variety of situations e.g. in pressure breathing which exerts transmural pressure across the walls of airways thus increasing this space (6); under drugs producing dilatation of bronchi and bronchioles as reported by Severinghaus and Stupfel (11); breathing through tubes or masks as in divers or aviators. During sharp turns or loops, aviators are exposed to acceleration forces of high magnitude stretching the tracheobronchial tree thus increasing the dead space. Pappenheimer *et al.* (9), quoting Haldane's work, have observed that dead space increases in proportion to TV due to an increasing volume of bronchioles. Anatomical dead space also increases under hypothermia as a result of reduced vagal response, and in aged people.

Whatever may be the contributory factor, increased dead space upsets pulmonary functions. This study was therefore, undertaken to evaluate some of the respiratory parameters with different volumes of added anatomical dead space.

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MATERIALS AND METHODS

Forty apparently healthy subjects of different age groups were selected. They were clinically examined before being selected for trials. Any complaint pertaining to the respiratory system or any organic condition of lungs disqualified these individuals from being used as subjects. They were divided in 3 groups based on age as under :

Gp I	Age between 21 and 30 yrs.
Gp II	Age between 31 and 40 yrs.
Gp III	Age between 41 and 50 yrs

A total of 22 subjects belonged to Gp 1, 10 to Gp II and 8 to Gp III. Their base-line FRC was determined by the open circuit method described by Comroe (4).

Layout of the apparatus is depicted in Fig. 1. Oxygen is provided through an oxygen cylinder which is connected to the apparatus through a regulator used in aviation.

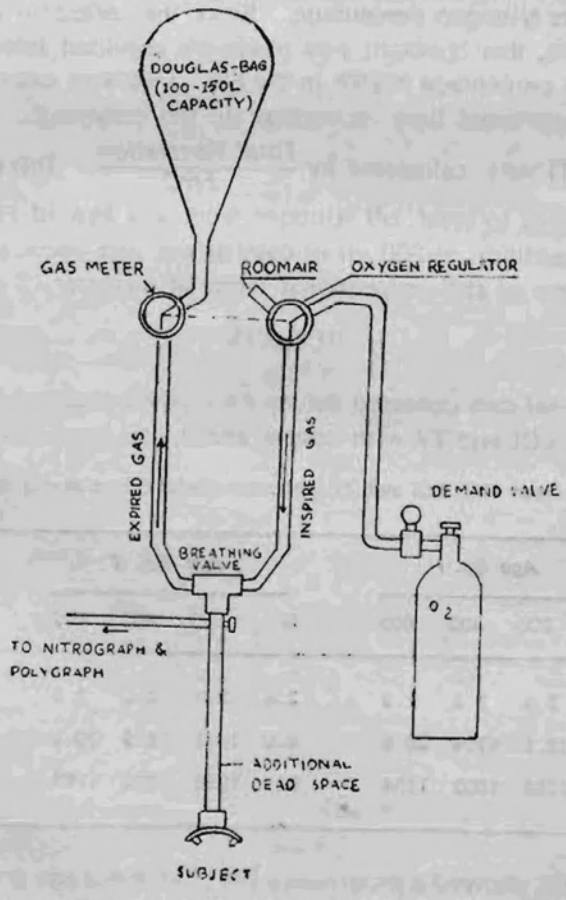

Fig. 1 : Line diagram depicting the layout of the apparatus used in he study.

This regulator can be selected to deliver atmospheric air or oxygen, as required. A gas meter was introduced in the breathing line between the subject and the Douglas bag to record the volume of air expired into the bag. A Nitrograph was connected to the breathing line through a capillary tube through which a sample of expired/inspired air was automatically collected and its Nitrogen percentage was indicated on a dial.

At the commencement of each recording, the regulator was selected to deliver atmospheric air and the subject allowed a few minutes to get accustomed to the test conditions the criteria for which was provided by the uniform deflection of nitrograph needle with each inspiration and expiration. Soon after this the regulator was selected to deliver 100% oxygen at the end of normal expiration and the expired air line was simultaneously connected to a Douglas bag which had been previously evacuated by a vacuum pump. The expired gas was collected till its Nitrogen percentage dropped to 2%. The subjected was then disconnected. Total ventilation was read on the gas meter. The entire expired air collected in the Douglas bag was mixed up thoroughly and then fed directly into the nitrograph to determine its Nitrogen percentage. Since the collection was started at the end of a normal expiration, this Nitrogen was obviously provided solely by FRC. In view of the fact that Nitrogen percentage is 79% in the FRC, FRC was calculated in each situation. Tidal volume was determined from recordings on the polygraph. Lung Clearance Index (LCI) of Becklake (1) was calculated by $\frac{Total\ Ventilation.}{FRC}$ This indicated the number of litres of oxygen required to wash off Nitrogen from one litre of FRC. These parameters were repeated after addition of 200 *ml* of dead space and again after addition of 400 *ml* and 600 *ml* in the form of stiff polyethylene tubes of diameter 17 mm.

RESULTS

The experimental data collected during the study is presented in Table I. This table gives the mean FRC, LCI and TV with normal and added dead space in the 3 groups.

TABLE I : Showing the mean FRC, LCI and TV with normal/added dead spaces of 200 *ml*, 400 *ml* and 600 *ml*.

	Age Gp. I				*Age Gp. II*				*Age Gp. III*			
	N	200	400	600	N	200	400	600	N	200	400	600
FRC(L)	4.0	3.6	3.3	2.8	3.6	3.2	3.0	2.6	4.4	3.8	3.4	3.2
LCI	8.6	13.1	17.4	28.5	9.0	10.7	18.9	30.1	9.7	12.5	18.7	32.7
TV (*ml*)	1030	1058	1203	1294	941	1028	1074	1162	966	1133	1199	1326

It is seen that FRC showed a progressive fall in all the 3 age groups as the dead space increased. It fell by about 30% of the normal as the dead space was increased by 600 *ml*.

LCI, on the other hand, showed a progressive rise with increasing dead space. The average increase in each age group for a dead space of 600 *ml* was more than 3 times the normal values. A significant observation was that while the incease was almost linear upto an additional dead space of 400 *ml*, it showed a proportionately greater increase as the dead space was increased from 400 *ml* to 600 *ml*. Third parameter in the table is TV which increased with each additional dead space almost linearly in all the 3 age groups.

The variations observed in the 3 parameters were subjected to statistical analysis. Results of this analysis are indicated in Table II and show that the fall of FRC and rise of LCI and TV are significant when additional dead space is included in the breathing line.

TABLE II : Significance of changes in FRC, LCI and TV with increasing dead space in different age groups (by the application of statistical (t' test).

Age group	*Variation in dead space*	*FRC*	*LCI*	*TV*
I	N-200	•	••	N.S.
	N-400	•	••	•
	N-600	••	••	••
II	N-200	N.S.	•	•
	N-400	N.S.	••	•
	N-600	•	••	
III	N-200	•	•	••
	N-400	•	••	••
	N-600	••	••	••

••Highly significant •Significant

DISCUSSION

Increase in baseline FRC and LCI with age is an expected response. Bouhuy (3); in his work on 80 healthy males with a negative cardiopulmonary history and normal chest X-Rays during one year preceding the study, found that increase in LCI with age was small and was due to the increased unevenness of distribution of ventilation in lungs. He attributed this unevenness to the smoking habits of subects. This view cannot be sustained by the present study because most of the subjects in that age group were non smokers. It is considered more likely that the observed changes in old age are brought about by stuctural deformation in lungs and thoracic cage which produce uneven distribution of pulmonary ventilation and/or a loss of elasticity of alveolar tissue. Fowler *et al.* (7) have observed that effects of uneven ventilation could be overcome if the total minute volume of alveolar ventilation was large in relation to the FRC. According to Parvathi (10) mean minute volume increase both in males and females if extra dead space upto 1000 *ml* was

added in the breathing line. This work suggests that the addition of dead space improves pulmonary ventilation by reducing the extent of uneven distribution of inspired air and by promoting better mixing of gases from different parts of lungs.

Decrease of FRC with added dead space can be atleast partly explained by the increase of TV which encroaches both on the inspiratory and expiratory reserve volumes (Fig.2). Reduction of FRC may also be explained by the fact that pulmonary vessels, being low pressure distensible reservoirs, accumulate a sizeable quantity of blood whenever there is any increase in pressure in the rt. ventricle. Normal inspiratory movement causes a fall of intrathoracic pressure causing an increased filling of rt. ventricle and therefore a larger flow in pulmonary circulation. Deeper breathing brought about by larger TV will enhance this effect. Comroe(5) has observed that vital capacity (VC) and total lung capacity (TLC) decrease though the residual volume remains normal when pulmonary vascular congestion increases thus implying that, among other components, expiratory reserve volume which is a component of FRC will decrease. Another factor which may contribute to a fall in FRC is the increase in elastic force at the end inspiration brought about by a larger TV since the elastic force varies directly with the volume of lung. Increased elastic force at end inspiration would increase the pressure available for expiration which again brings down the FRC.

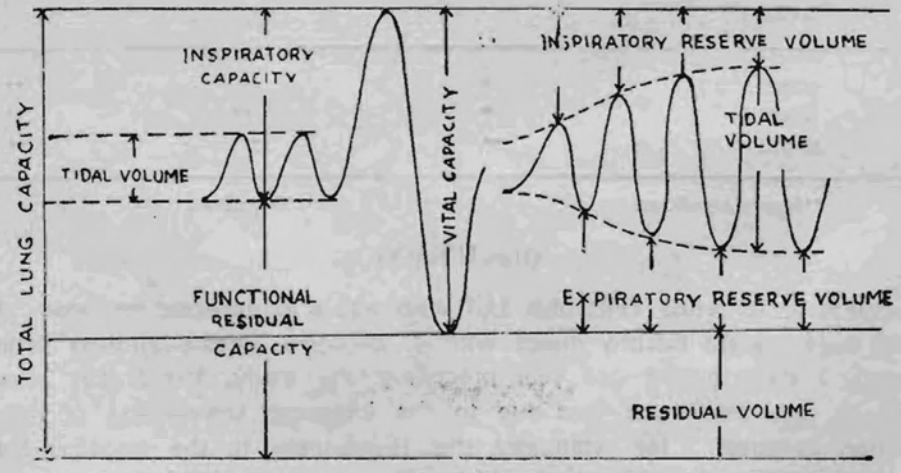

Fig. 2 : Encroachment of tidal volume into inspiratory and expiratory reserve volumes. (Reproduced from Handbook of Physiology Sect.3 : Respiration Vol. I., P. 388).

The study also shows that LCI increases with anatomical dead space. This index expresses the ventilatory efficiency as a whole and determines the degree of nonuniformity of lung ventilation as observed by Bouhuy *et al.* (2). As mentioned earlier, addition of dead

space increases uniformity of ventilation due to better mixing from different lung fractions. This should improve the LCI. According to Becklake (1), normal subjects should have LCI less than 10 which means they have to ventilate less than 10 litres to wash off Nitrogen from 1 litre of their FRC. In the present work, LCI increased with added dead space indicating deterioration which may be accounted for by the failure of TV to rise by an amount equal to the added dead space. It was seen that the increase in TV brought about by addition of dead space was much less than the actual dead space added to the respiratory circuit. This would reduce the lung efficiency and thus contribute to a deterioration in LCI. In the individual records of LCI and TV vis-a-vis added dead space, it was found that for subjects who produced a larger increase in TV as compared to the dead space showed a drop in their LCI indicating improvement. Bouhuy *et al.* (2) also have observed an increase in LCI with added space despite better distribution and an improvement in uniformity of ventilation observed by them. They have opined that this anomaly is due to a cancellation of improvement in clearance efficiency by an increased dead space. Functional efficiency of lungs therefore, falls as the dead space is increased. A disproportionate rise of LCI with increase of dead space from 400 *ml* to 600 *ml* indicates a disproportionate deterioration of lung efficiency when the dead space increases beyond 400 *ml*.

Mean TV increases with increase in dead space indicating increased alveolar ventilation which has been attributed by Parvathi (10) to increased alveolar PCO_2 brought about by added dead space. Margaria *et al.* (8) have observed that addition of dead space could be considered equivalent to addition of CO_2 to the inspired air. Suwa and Bendixen (12) have attempted to control the effect of hyperventilation by addition of a sufficient amount of dead space in the breathing circuit. They devised a mathematical approach to determine the mechanical dead space required to raise the arterial PCO_2 by a given value to counteract the effect of hyperventilation induced by artificial respiration.

An incidental finding in this study was an increase in pulse rate which can be attributed partly to an increase in lung inflation and partly to an increase in PCO_2.

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