

# Artificial Increase of Dead Space for the Improvement of Post-Operative Ventilation — A Review

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A reduction in post-operative pulmonary ventilation is almost invariably present amongst patients undergoing upper abdominal surgery<sup>(1, 2)</sup>. It is very common amongst those having lower abdominal surgery and very rare following orthopaedic operations<sup>(3)</sup>.

Nichols and Howell (1970)<sup>(4)</sup> analysing their results conclude that there is no significant difference in ventilatory capacity changes between patients receiving the classical pre- and post-operative physiotherapy and between those patients who did not receive this treatment. However, they excluded from this trial patients with pre-existing acute or chronic obstructive lung-disease because, for this type of patient, there is general agreement about the therapeutic value of classical physiotherapeutic procedures. From their trial it is evident that a post-operative decrease of pulmonary ventilation is not correlated with an obstructive pattern of respiration. It is, however, clearly correlated with a restrictive pattern of ventilation i.e. shallow breathing, as a result of diaphragmatic inhibition. Further evidence to their conclusions is promulgated by the work of Finer<sup>(5)</sup> who studied the variability in expiratory efforts before and after cholecystectomy. This author stresses the importance of encouragement — "the elicitation of the latent encouragement potential," in order to overcome diaphragmatic inhibition and to improve pulmonary ventilation. However, he also stresses the clinical value of post-operative pain relief, which he effects by different and mutually compared kinds of nerve blocks (intercostal, epidural and splanchnic). Controlled measurements of vital capacity (V.C.), forced expiratory volume (F.E.V.) and peak expiratory flow showed that the best results were obtained by a combination of encouragement and intercostal block. A somatic and visceral pain block combination did not appear to be superior to a somatic pain block alone. The author's opinion is that the relief of somatic pain by analgesic therapy, following cholecystectomy, is the more important factor in the improvement of post-operative expiratory efforts.

Diaphragmatic inhibition causes a consequent reduction of lung volume. This reduced lung volume is a contributory cause of segmental and massive lung collapse, of raised diaphragm and of pleural effusion. In the early stages of their development these complications frequently cannot yet be diagnosed by clinical symptoms and have to be detected by radiological changes.

It is thus clear that, in order to prevent the clinical complications of reduced lung volume, diaphragmatic inhibition must at all costs be overcome. Because of the inadequacy of classical physiotherapeutic measures for the improvement of post-operative pulmonary ventilation, a new therapeutic approach is needed, by which at will an increase of ventilation, especially after abdominal surgery, can be obtained.

The next problem posed is the method of choice, because different methods have been proposed and evaluated. A recent comparison has been made by

Jones (1968)<sup>(6)</sup> in a clinical study of the rather small number of 15 patients who had recently undergone thoracotomy or laparotomy operations. He compared five commonly employed methods for enhancing ventilation, using them sequentially in each patient for two to five days post-operatively and estimating their effectiveness towards the increase of the  $V_T$ . All these methods were applied without the administration of narcotics for four hours previous to the study. In six of the patients involved pre-operative as well as post-operative studies were done.

From this study it is apparent, as it is from the above literature, that following abdominal or thoracic surgery there is a diminished ventilatory ability, the post-operative breathing values averaging 60% of the pre-operative values.

The comparison between the five different clinical methods for enhancing ventilation had the following results:

1. Rebreathing with the Adler rebreather (1967)<sup>(4)</sup> was relatively ineffective in increasing depth of respiration.
2. I.P.P.B. (Intermittent Positive Pressure Breathing) with the conventionally employed inspiratory pressure of 15 cm H<sub>2</sub>O and the inhalation of 5% CO<sub>2</sub> were methods of intermediate value. They were, however, infrequently equal or superior to voluntary efforts in producing significant increases in  $V_T$ .
3. Voluntary deep breathing was frequently quite effective. Increases in  $V_T$  of 80% or more occurred in two-thirds of the subjects, while  $V_T$  as large or larger than those produced by any other method were observed in about 50% of the patients.
4. I.P.P.B. at the relatively high inspiratory pressure of 25 cm H<sub>2</sub>O was clearly the most superior method. When  $V_T$  greater than those due to voluntary deep breathing were observed, they were almost always produced by I.P.P.B. of 25 cm H<sub>2</sub>O.

However, the author stresses the limitation of the study. A comparative evaluation of these methods in a larger series of patients, wherein the augmentation of  $V_T$  is the only variable, is recommended.

"The voluntary deep breathing" mentioned in 3 can be compared with "the elicitation of the latent encouragement potential" of Finer (1970)<sup>(5)</sup>.

In a department of physiotherapy one needs for the average daily hospital practice "efficient" methods for the improvement of post-operative ventilation. At the same time, the methods applied should be as simple as possible, require a minimum of routine instrumentation and a minimum of patient conveyance.

Inhalation of CO<sub>2</sub> requires CO<sub>2</sub> supply, oxygen supply, a gas flow measuring device and a well-fitting gas administration set. I.P.P.B. requires a rather expensive and complicated equipment for artificially assisted respiration.

One wonders whether Jones (1968)<sup>(6)</sup> has paid sufficient attention to the clinical possibilities of rebreathing methods for the improvement of post-operative pulmonary ventilation.

In experimental physiology the principle of a simple procedure, by which at the same time pulmonary ventila-

tion could be increased and a normal  $P_A CO_2$  could be maintained, has been described by *Gad* (1880). He connected a large tube with a diameter of 0,4 cm and a volume of about 108 ml to the trachea of a rabbit thus provoking by this manoeuvre an increase of respiratory volume and respiratory rate. *Liljestrand* (1918) applied tubes with a diameter of 1,8 cm and a volume of 1740-1950 ml to human beings. He discovered that the  $CO_2$  content of the inspiratory air would increase to the same degree, as the amount of  $CO_2$  present in the physiological dead space from the previous expiration.

*Scott and Cutler* (1928)<sup>(5)</sup> described the use of hyper-ventilation by rebreathing of carbon dioxide from a paper bag for the treatment of post-operative massive atelectasis.

*Duomacio and Diaz-Romero* (1937)<sup>(6)</sup> described the artificial increase of respiratory dead space as a practical method for the improvement of pulmonary ventilation. The artificial dead space is increased to a volume, whereby it may still be compensated by the corresponding increase of  $V_T$ . Under these conditions no hypocapnia occurs because, notwithstanding the increase of Respiratory Minute Volume (R.M.V.), the  $P_A CO_2$  is kept constant by the increase in physiological dead space.

*Schwartz and Dale* (1955)<sup>(7)</sup> describe the addition of dead space in order to produce hyperventilation for the prevention of atelectasis. They give evidence that there is a direct relationship between the size of  $P_A CO_2$  and the  $V_T$  i.e. the  $P_A CO_2$  and  $V_T$  -curves closely parallel each other. The greatest increment in "stimulated Tidal Volume" occurred during the first two minutes: in this time the  $V_T$  increased from a control value of 596 to 1178 ml. During the third minute there was another significant increase from 1178 to 1334 ml. From the 3rd to the 10th minute there was little change. Similarly the greatest rise in  $P_A CO_2$  from a control value of 39,44 to 44,17 mm of Hg occurred during the first two minutes. A second increase from 44,17 to 47,20 mm Hg was noted between the 2nd and the 5th minutes. A concomitant decrease of  $P_A O_2$  was also demonstrated during the first two minutes of hyperventilation (100,5 mm Hg control value, sharply decreasing to 77,61 mm Hg); over the following 8 min  $P_A O_2$  rose steadily and gradually to 88,93 mm Hg. Although the lowered  $P_A O_2$  was generally well tolerated by the patients, this eventual  $P_A O_2$  decrease could be prevented by directing a flow of oxygen into the distal end of the dead space tube. A dead space of 1 000 ml gave a more than 100% increase of  $V_T$  in 14 patients during the first three post-operative days without any significant alteration of respiratory rate.

The same study was repeated, but this time a flow of 3-4 litres of oxygen per minute was introduced into the distal end of the rebreathing tube. In the three patients involved the  $P_A O_2$  was maintained above the control value of 100,5 mm Hg. The average increases in  $V_T$  and  $P_A CO_2$  were similar to the changes noted in 15 patients breathing room air. This clearly indicates that while subjects rebreathe through the 1 000 ml dead space tube, the elevation of  $P_A CO_2$  is the critical factor that effects the increase of  $V_T$ .

*Schwartz-Dale-Rahn* (1957)<sup>(8)</sup> evaluated their clinical experience in over 1 000 patients with a dead space rebreathing tube as a post-operative routine. The tube was used for a period of five minutes at two hourly intervals. Its use caused an apparent decrease in the incidence of post-operative atelectasis and was thus established as an effective therapy in the treatment of atelectasis. No difficulties due to hypoxia were observed, although the occasional patient had to receive additional oxygen through the distal end of the tube.

*Giebel* (1968)<sup>(9)</sup> investigated five groups totalling 52

patients in ranges of normal, subnormal and abnormal ventilation/perfusion-ratios. Twelve of the patients reviewed were immediate post-operative cases. A complete radiological documentation of six of the patients, with severe forms of atelectasis and pulmonary collapse, was presented both before and after the treatment by means of artificial dead-space-increased ventilation. From his investigations the following conclusions emerge:

1. The addition of artificial dead space induces an increase of R.M.V. in all patients. The increase of R.M.V. is proportional to the volume of dead space added. As long as alveolar ventilation does not decrease, respiratory rate does not increase significantly. However, as soon as respiratory rate in adults increases to more than 20-24 per minute, dead space ventilation increases and alveolar ventilation decreases, leading to consequent hypercapnia.
2. In all groups of patients there is a decrease of  $AaDO_2$  (alveoloarterial oxygen difference) during respiration with artificial increase of dead space. This may only be explained as an overall improvement and normalisation of the pre-existing ventilation/perfusion disturbances. The increase of  $V_T$  induced by the addition of dead space therefore not only improves the pulmonary ventilation, but also the total pulmonary perfusion.
3. The decrease of  $AaDO_2$  parallels closely the increase in  $Pa O_2$ .
4. The  $PaCO_2$  remains within normal limits during the efficient addition of volume to the artificial dead space. The increase in pulmonary ventilation as a result of the increased  $PCO_2$  in the inspired air is autoregulated in such a way that the  $PaCO_2$  remains within normal limits.
5. During respiration with artificial increase of dead space there is neither increase of the blood pressure nor of the pulse rate.
6. The best criterion for testing whether the volume added to the dead space is correct is the behaviour of the respiratory rate. In adult patients the respiratory rate should not surpass 20-24 per minute.
7. If correctly applied, the method of artificial increase of dead space provided an efficient treatment of subacute and chronic atelectasis.

*Edelst and Orkin* (1967)<sup>(10)</sup> produced, with dead space increase of 750 ml and an oxygen flow of 300 ml per minute into the rebreathing tube, an increase of the inspiratory  $PCO_2$ , which was sufficient to increase pulmonary ventilation at least 2½ times. The authors' assumption was, that by the significantly increased pulmonary ventilation areas of miliary atelectasis would be opened and thus venous admixture would decrease. They could, however, not support these assumptions with their results. In only two of the eight patients was the post rebreathing  $Pa O_2$  elevated in comparison with pre-rebreathing  $Pa O_2$ . In the remainder, there was no change, or an actual decrease of  $Pa O_2$ . This seems illogical, but it must be remembered that their patients were not suffering from any known cardio-pulmonary disease. Venous admixture, with the exclusion of actual anatomical shunts from the right to the left heart, may be caused by two pulmonary mechanisms:

1. *Atelectasis*: In this case the atelectatic areas of the lungs are perfused, but not ventilated.
2. *Maldistribution*: Different areas of the lungs have different ventilation/perfusion ratios.

It is clear that none of these conditions are present in patients not suffering from cardio-pulmonary disease. This means that, as ventilation/perfusion ratios are

normal, increases of pulmonary ventilation do not imply changes of Pa O<sub>2</sub>, at least if the O<sub>2</sub> concentration of the inspired gas mixture remains unchanged. *Edelist and Orkin* (1967<sup>(10)</sup>) should first have selected patients in two groups: a group with and a group without pulmonary ventilation/perfusion disturbances. They should then have tested their assumption on the first group in order to make sure that the Pa O<sub>2</sub> would have increased, proportionately to the decrease of venous admixture, by the improvement of ventilation in either the atelectatic areas or in the areas of maldistribution.

*Hald and Johanson* (1970)<sup>(11)</sup> exposed 23 patients to inhalation of 2% CO<sub>2</sub> in room air for 20 minutes after the completion of surgery. During this administration period an increase in Pa O<sub>2</sub> and pulmonary ventilation was observed, whereas the Pa CO<sub>2</sub> remained constant.

The increasing Pa O<sub>2</sub> contrasts with the data of *Edelist and Orkin*, but once again I should like to refer to the importance of patient selection. In post-anaesthetic patients especially there is a known high ratio of micro-atelectatic changes and consequent arteriovenous pulmonary shunting. This correlates well with the favourable effect of pulmonary hyperventilation on the Pa O<sub>2</sub> in these cases.

### Summary

From the literature reviewed, as regards the value of methods used for improvement of pulmonary ventilation, several concepts have emerged.

1. Post-operative ventilatory disturbances are prevalent especially in thoracic and abdominal (both higher and lower) surgery.
2. In patients without pre-existing cardio-pulmonary disease these ventilatory disturbances are never of the obstructive type, but always of the restrictive type.
3. Classical physiotherapy procedures are of great value for the prevention and treatment of pulmonary complications in the patient suffering from obstructive lung disease. However, the value of these procedures as regards restrictive ventilatory disturbances is questionable.
4. It is of prime importance that post-operative ventilatory disturbances be treated in order to prevent incipient atelectasis, bronchopneumonia and massive lung-collapse.
5. Among the methods described for the treatment of post-operative ventilatory disturbances and their complications, different grades of complexity and efficiency may be distinguished.
6. It is generally acceptable that the best method of treatment should be an ideal combination of efficiency and simplicity capable of covering the widest possible clinical field. It seems that the data derived from the literature reviewed indicates that the method of choice is the artificial increase of dead space; subject to certain conditions. These conditions are:
  - (a) The method should be applied for a maximum time of five minutes.
  - (b) It should be repeated two hourly.
  - (c) The additional dead space volume should be guided by the respiratory rate. A respiratory rate of 20-24 per minute should not be exceeded in the case of adults. For a start an optional dead space increase of 500-1000 ml for adults and of 250-500 ml for children should be selected.
7. Pain is an important contributory factor to post-operative ventilatory impairment. It results directly in diaphragmatic inhibition. Sufficient attention and skill should be spent in providing adequate post-operative analgesia, the degree of success of which should be evident from a favourable relationship

between the degree of analgesia and the amount of respiratory depression.

8. The simultaneous combination of an artificial increase of dead space and adequate analgesia merits further study. A comparison should therefore be made between the effects of rebreathing methods under painless, pre-operative conditions and the effects of rebreathing methods under painful, post-operative conditions. Thereafter analgesia should be administered and pulmonary ventilation with rebreathing methods retested.
9. This study could be completed by aiding rebreathing with an increase of artificial dead space and adequate analgesia-therapy with the psycho-therapy of encouragement (*Finer*: "the elicitation of the latent encouragement potential!").

Voluntary deep, painless breathing supported by the stimulation of the respiratory centre by an artificially increased dead space would then be the sum total of therapeutic achievement towards the difficult task of overcoming post-operative pulmonary ventilation disturbances. This achievement would thus have been obtained with a minimum of costs and without need for sophisticated equipment.

### Conclusion

The artificial increase of dead space is a most promising, but as yet incompletely evaluated method for the improvement of post-operative pulmonary ventilation. The majority of the available experimental studies report favourable results.

Some of the conflicting points regarding this method could possibly be better understood and improved significantly if more attention could be paid simultaneously both to the relief of post-operative somatic pain and to the personal encouragement of the patient.

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