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

Arnold E. D. van de Vijver M.D. (Utrecht) Senior Lecturer, Department of Anaesthesia, University of Stellenbosch, Senior Anaesthetist, Karl Bremer Hospital, Bellville.

A reduction in post-operative pulmonary ventilation is almost invariably present amongst patients undergoing upper abdominal surgery. It is very common amongst those having lower abdominal surgery and very rare following orthopaedic operations.

Nichols and Howell (1970) 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 this 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 (1970) 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 tidal 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) 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 Vt. 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 abovenoted 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) 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 H2O and the inhalation of 5% CO2 were methods of intermediate value. They were, however, infrequently equal or superior to voluntary efforts in producing significant increases in Vt.

3. Voluntary deep breathing was frequently quite effective. Increases in Vt of 80% or more occurred in two-thirds of the subjects, while Vt 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 H2O was clearly the most superior method. When Vt greater than those due to voluntary deep breathing were observed, they were almost always produced by I.P.P.B. of 25 cm H2O.

However, the author stresses the limitation of this study. A comparative evaluation of these methods in a larger series of patients, wherein the augmentation of Vt 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).

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 CO2 requires CO2 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) 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 PaCO₂ could be maintained, has been described by Gad (1880). He constructed 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 CO₂ content of the inspiratory air would increase to the same degree, as the amount of CO₂ present in the physiological dead space from the previous expiration.

Scott and Cutler (1928) described the use of hyperventilation by rebreathing of carbon dioxide from a paper bag for the treatment of post-operative massive atelectasis.

Diomacito and Diaz-Romero (1937) 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 VT. Under these conditions no hypocapnia occurs because, notwithstanding the increase of Respiratory Minute Volume (R.M.V.), the PaCO₂ is kept constant by the increase in physiological dead space.

Schwartz and Dale (1955) described the addition of dead space in order to produce hyperventilation for the prevention of atelectasis. They give evidence that there is a direct relation between the size of PaCO₂ and the VT i.e. the PaCO₂ and VT curves closely parallel each other. The greatest increment in “stimulated Tidal Volume” occurred during the first two minutes: in this time the VT increased from a control value of 596 to 1798 ml. During the third minute there was another significant increase from 1778 to 1334 ml. From the 3rd to the 10th minute there was little change. Similar results were obtained in patients with a dead space added. As long as 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 hypocapnia.

In all groups of patients there is a decrease of AaDO₂ (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 VT induced by the addition of dead space therefore not only improves the pulmonary ventilation, but also the total pulmonary perfusion.

The decrease of AaDO₂ parallels closely the increase in PaO₂.

The PaCO₂ 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 PCO₂ in the inspired air is auto regulated in such a way that the PaCO₂ remains within normal limits.

During respiration with artificial increase of dead space there is neither increase of the blood pressure nor of the pulse rate.

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.

If correctly applied, the method of artificial increase of dead space provided an efficient treatment of subacute or chronic atelectasis and 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 the post rebreathing PaO₂ elevated in comparison with pre-rebreathing PaO₂. In the remainder, there was no change, or an actual decrease of PaO₂. 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 ateleastic areas of the lungs are perfused, but not ventilated.

2. Maldistribution: Different areas of the lungs have different ventilation/perfusion ratios. The ratio of dead space there is neither increase of the blood pressure nor of the pulse rate.

Reproduced by Subnet Gateway under licence granted by the Publisher (dated 2013).
normal, increases of pulmonary ventilation do not imply changes of Pa $O_2$, at least if the $O_2$ concentration of the inspired gas mixture remains unchanged. Edelist and Orkin (1967) 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 $O_2$ 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 Johansen (1970) exposed 23 patients to inhalation of 2% $CO_2$ in room air for 20 minutes after the completion of surgery. During this administration period an increase in Pa $O_2$ and pulmonary ventilation was observed, whereas the Pa $CO_2$ remained constant.

The increasing Pa $O_2$ 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 $O_2$ 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 500-1 000 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 the 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.

References