

# Clinical Workshop

S. G. HERSHEY, M.D., *Editor*

## A Nomogram for Dead-space Requirement during Prolonged Artificial Ventilation

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We have derived a nomogram which permits easy determination of the mechanical dead-space required to prevent hypocapnea during artificial ventilation.

The  $P_{aCO_2}$  usually is abnormally low in patients receiving prolonged ventilation.<sup>1,4</sup> Radford designed a nomogram to predict the tidal volume and respiratory rate needed to achieve normal alveolar ventilation in patients with normal respiratory and circulatory function.<sup>5</sup> Many patients complain of dyspnea, however, when ventilation is provided according to these standards.<sup>4, 6</sup> The sensation of dyspnea may be present despite normal  $P_{aCO_2}$ . A ventilation pattern which satisfies the patient results in an abnormally low  $P_{aCO_2}$  (often in the order of 25–28 mm Hg). Furthermore, a relatively large tidal volume and periodic hyperinflation are required to prevent atelectasis during positive-pressure ventilation.<sup>7, 8</sup> Influenced by the patient's request

for increased ventilation, and considering the need to maintain the lungs well inflated, it has become common practice, in the care of patients on mechanical ventilators, to provide large tidal volumes, approximately twice those predicted from ventilation standards, at a normal respiratory frequency of 12–18 breaths per minute. A contributing consideration may be the mistaken belief that  $P_{aCO_2}$  can and should be maintained below the so-called apnea threshold during prolonged mechanical ventilation.

Although such hypocapnea cannot be blamed conclusively for any increase in morbidity or mortality,<sup>9</sup> several possibly harmful effects must be considered.

Weaning appears more difficult following prolonged hyperventilation, presumably because compensatory changes in cerebrospinal fluid acid-base balance reset the chemoreceptors.<sup>10, 11</sup> Once reset, the central respiratory drive imposes a demand for continued hyperventilation, an unnecessary imposition of an increased ventilation requirement which the weaning patient finds it particularly difficult to meet.

Metabolic acidosis develops to compensate for the respiratory alkalosis. Since renal regulation of acid-base balance takes place slowly, this causes measurable acidemia at the time of successful weaning from the ventilator, when alveolar ventilation is reduced.<sup>4</sup>

A low  $P_{aCO_2}$  and a high  $pH$  cause a shift of the oxygen dissociation curve to the left and thereby impair the release of oxygen from hemoglobin to tissue. Suskind and Rahn showed theoretically and experimentally that

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the mixed venous oxygen tension, as an index of tissue oxygen tension, falls with increased alveolar ventilation, cardiac output being held constant.<sup>12</sup> Astrup *et al.* attribute the appearance of cyanosis in hyperventilated patients to this mechanism.<sup>1</sup>

The cerebral circulation varies with  $P_{aCO_2}$ . A very low  $P_{aCO_2}$  may cause cerebral hypoxia due to decreased cerebral blood flow.<sup>13, 14, 15</sup>

Tetany is another complication of hyperventilation,<sup>1</sup> as is the finding that cardiac output decreased to half the normal value when  $P_{aCO_2}$  was lowered to 20 mm Hg.<sup>16</sup>

With these prospects, normality is certainly preferable to abnormality. Maintenance of  $P_{aCO_2}$  in a normal range is desirable.

The feeling of dyspnea is, at least in part, independent of  $P_{aCO_2}$  and related to the inadequacy of lung expansion.<sup>6, 17</sup> Apparently incompatible goals, normal  $P_{aCO_2}$  and large tidal volumes at a normal frequency, can be achieved by inserting a mechanical deadspace, provided the quantitative relationship between the desired increase in  $P_{aCO_2}$  and the size of mechanical deadspace necessary.

### Theoretical and Experimental Basis

Previous studies<sup>18, 19</sup> provided experimental confirmation of the theoretically derived relationships expressed by the following equation:

$$P_{a_2CO_2} = P_{a_1CO_2} + P_{\bar{a}_1CO_2} \times \frac{V_{DM}}{V_T - V_{D_{an}} - V_{DM}} \quad (1)$$

where  $P_{aCO_2}$  is arterial  $CO_2$  tension,  $P_{\bar{a}CO_2}$  is alveolar  $CO_2$  tension (sometimes called " $P_{CO_2}$  of the alveolar component of expired gas"<sup>20</sup>),  $V_T$  is tidal volume and  $V_{D_{an}}$  is the anatomical deadspace of the patient, including the deadspace of the tracheostomy tube and the ventilator;  $V_{DM}$  is the mechanical deadspace added. Subscripts 1 and 2 refer to conditions before and after  $V_{DM}$  is added. Both tidal volume and respiratory rate were kept constant, and measurements were done under steady-state conditions. Previous work permits the assumption that equation 1 is valid under clinical conditions. Solving equation 1

for  $V_{DM}$ ,

$$V_{DM} = \frac{P_{a_2CO_2} - P_{a_1CO_2}}{P_{a_2CO_2} - (P_{a_1CO_2} - P_{\bar{a}_1CO_2})} \times (V_T - V_{D_{an}}), \quad (2)$$

it is possible to calculate the additional deadspace required to raise  $P_{aCO_2}$  to any desired value.

### Nomogram

A nomogram was constructed on the basis of two modifications of equation 2, as follows:

#### I. NORMAL PULMONARY AND CIRCULATORY STATUS

A relatively normal ventilation-perfusion relationship is assumed. Therefore,  $P_{a_1CO_2} - P_{\bar{a}_1CO_2}$  is less than 10 mm Hg, but cannot take a negative value. We adopted the value of 5 mm Hg.<sup>21</sup>  $P_{a_2CO_2}$  (the desired  $P_{aCO_2}$ , following addition of the mechanical deadspace) is 40 mm Hg, hence:

$$V_{DM} = \frac{40 - P_{a_1CO_2}}{35} \times (V_T - V_{D_{an}}) \quad (3)$$

#### II. ABNORMAL PULMONARY FUNCTION OR CIRCULATORY FAILURE\*

A wide scatter of ventilation-perfusion ratios is assumed,  $P_{a_1CO_2} - P_{\bar{a}_1CO_2}$  being unpredictable and generally greater than 10 mm Hg.<sup>22</sup>  $P_{\bar{E}CO_2}$  ( $PCO_2$  in mixed expired gas) is used instead of the  $P_{\bar{a}_1CO_2}$  on this occasion, because the difference of the two becomes smaller (see Appendix),

$$V_{DM} = \frac{40 - P_{a_1CO_2}}{40 - (P_{a_1CO_2} - P_{\bar{E}CO_2})} \times (V_T - V_{D_{an}}). \quad (4)$$

As can be seen from equations 3 and 4, the parameters which need to be measured are  $V_T$  and  $P_{a_1CO_2}$ .  $V_{D_{an}}$  is estimated from body weight. In the presence of pulmonary disease

\* Hypocapnea, of sufficient degree to require correction by the use of a mechanical deadspace, is unlikely in the presence of severe pulmonary disease; however, hypocapnea is seen in the presence of moderate to moderately severe pulmonary disease; hence the need for correction.

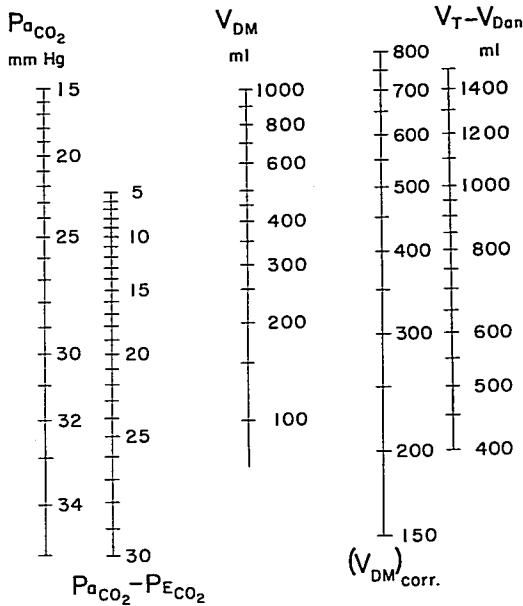


Fig. 1. Mechanical deadspace required to raise  $P_{aCO_2}$  to approximately 40 mm Hg can be predicted from this nomogram.  $P_{aCO_2}$  is the observed arterial carbon dioxide tension before introduction of the mechanical deadspace,  $V_T$  is tidal volume, and  $V_{DM}$  is anatomical deadspace. For the patient with normal pulmonary and circulatory status, the line in the middle gives the required value for  $V_{DM}$ . In the presence of pulmonary disease or circulatory failure, a correction must be made by connecting the points on the ( $P_{aCO_2} - P_{ECO_2}$ ) line and the  $V_{DM}$  line, then extending it to the right. Read the value on the ( $V_{DM}$ ) corr. line.  $V_{DM}$  is two-thirds of body weight (1b) plus deadspace of the tracheostomy tube and the ventilator.

#### Example:

A patient is being ventilated at a given respiratory frequency with a tidal volume of 1130 ml.  $P_{aCO_2}$  is measured and found to be 25 mm Hg.

The patient's body weight is 150 lbs; the deadspace of the tracheostomy tube and the ventilator is 30 ml.  $V_{DM} = 2/3$  of body weight + ventilator and tracheostomy deadspace = 130 ml.  $V_T - V_{DM} = 1000$  ml.

The two points, 1000 for  $V_T - V_{DM}$  and 25 for  $P_{aCO_2}$ , are connected, and we read off the center line that  $V_{DM}$  should be 430 ml. Assuming reasonably

normal lungs, the insertion of a deadspace of 430 ml should raise this patient's  $P_{aCO_2}$  to approximately 40 mm Hg. It is emphasized that respiratory frequency and tidal volume must remain unchanged.

If the patient's lung function is not normal,  $P_{ECO_2}$  is determined also and found to be 10 mm Hg;  $P_{aCO_2} - P_{ECO_2}$  is then 25 mm Hg - 10 mm Hg = 15 mm Hg. Connect this point ( $P_{aCO_2} - P_{ECO_2} = 15$  mm Hg) with the already obtained  $V_{DM}$  of 430 ml; extend the connecting line to the right, and read off the corrected  $V_{DM}$  (on the line ( $V_{DM}$ ) corr.). The corrected value is 600 ml.

$P_{ECO_2}$  must be measured also. For the two situations considered above the nomogram has been constructed (Fig. 1).

#### Discussion

The possible errors in using the nomogram stem from the assumptions made in equation 1, from the measurements and from the construction of the nomogram. The sources of error in equation 1 are the measurements of tidal volume and  $P_{aCO_2}$ , estimation of anatomical

deadspace from body weight, assumption of a constant ventilation-perfusion relationship, and the dimensions of the mechanical deadspace. Tidal volume and  $P_{aCO_2}$  can be measured accurately. The anatomical deadspace is estimated with sufficient accuracy from body weight, the error being an insignificant fraction of the tidal volume. The assumption that the volume of an added mechanical deadspace, measured by water displacement, acts as the deadspace of the same volume may be ques-

tioned. This assumption is accurate only when the tidal volume is considerably larger than the anatomical deadspace; strictly speaking,  $V_T$  should be at least twice as large as the sum of  $V_{D_{an}}$  and  $V_{D_M}$ .<sup>22</sup> The error introduced by this factor would result in a  $P_{aCO_2}$  lower than the predicted 40 mm Hg. This effect was not observed in the experimental study.<sup>19</sup>

When further approximations were made and equation 2 reduced to equations 3 and 4, a new error was introduced. Equation 3 introduces a potential error of maximum  $\pm 5$  mm Hg, because  $(P_{aCO_2} - P_{ACO_2})$  was assumed to be 5 mm Hg instead of between 0 and 10 mm Hg. Similarly, equation 4 introduces a potential error of the same magnitude (see Appendix).

In most cases in which patients are treated with artificial ventilation, ventilation standards based on normal lung function and metabolic rate do not apply.<sup>22</sup> Measurement of  $P_{ACO_2}$  is, then, the only way to assess the adequacy of alveolar ventilation; for this reason, the measurement of  $P_{ACO_2}$  was incorporated as the basis for predicting required deadspace, rather than using the existing normal ventilation standards. The nomogram must be used in conjunction with, not as a substitute for, blood gas measurements.

In practice, we recommend correcting a low  $P_{ACO_2}$  stepwise over several hours, rather than in one step. On two of 20 occasions the addition of mechanical deadspace led to the feeling of dyspnea.<sup>19</sup> This, together with the findings of Opie *et al.*,<sup>8</sup> suggests that such dyspnea is not entirely independent of  $P_{ACO_2}$  or the rate of change of  $P_{ACO_2}$ .

### Summary and Conclusions

A nomogram which permits prediction of the mechanical deadspace to be added in order to raise a low  $P_{ACO_2}$  to the normal 40 mm Hg is described. The purpose is to avoid the excessive hypocapnea often seen in patients receiving prolonged artificial ventilation.

The use of this nomogram requires measurement of tidal volume and  $P_{ACO_2}$ . The anatomical deadspace is estimated from body weight. In the presence of diseased lungs or circulatory failure, the measurement of mixed expired  $P_{CO_2}$  ( $P_{E_{CO_2}}$ ) is required also. The maximum error is in the order of 5 mm Hg.

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### APPENDIX

In the presence of abnormal pulmonary function or circulatory failure,  $P_{E_{CO_2}}$  can be used as the substitute for  $P_{aCO_2}$ , because

$$P_{aCO_2} = \frac{P_{E_{CO_2}}}{1 - \frac{V_{D_{an}}}{V_T}} \quad (\text{modified Bohr equation}).$$

It follows that

$$P_{aCO_2} - P_{E_{CO_2}} = P_{E_{CO_2}} \times \frac{V_{D_{an}}}{V_T - V_{D_{an}}}.$$

This means that the error introduced by substituting  $P_{E_{CO_2}}$  for  $P_{aCO_2}$  is smaller when  $P_{E_{CO_2}}$  is small and/or  $V_T$  is large compared to  $V_{D_{an}}$ .

$P_{aCO_2}$  is smaller than 35 mm Hg (if not, there is no need for  $V_{D_M}$ ).  $V_{D_{an}}$  is not more than 150 ml. In diseased lungs, a large tidal volume, most often larger than 750 ml, is required to produce hyperventilation.<sup>19,22</sup> Assuming that the physiologic deadspace-to-tidal-volume ratio ( $V_{D_b}/V_T$ ) is not smaller than 0.5<sup>22</sup> and that  $P_{E_{CO_2}}$  is no larger than 17.5 mm Hg, the highest value for  $P_{aCO_2} - P_{E_{CO_2}}$  is:

$$(P_{aCO_2} - P_{E_{CO_2}})_{\max} = 17.5 \times \frac{150}{600} = 4.5 \text{ mm Hg.}$$

This means that substitution of  $P_{E_{CO_2}}$  for  $P_{aCO_2}$  is unlikely to introduce an error greater than approximately 5 mm Hg.

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## Technical Note Concerning the Block-Aid Monitor

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Recently we compared the responses of the adductor pollicis muscle to stimulation by the Block-Aid Monitor and by an American Electronics Laboratories No. 104A square-wave pulse generator.

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A healthy man undergoing elective peripheral surgery was anesthetized with nitrous oxide (5 l/min) and oxygen 2 l/min) supplemented with meperidine. Ten mg *d*-tubocurarine had been given during the 70 minutes prior to the measurements. Supramaximal stimuli were applied to the ulnar nerve via two needle electrodes at the wrist. Contraction of the adductor pollicis was measured with a force transducer and recorded on a Grass Polygraph. When a ten-second tetanic train was delivered at 30 cycles per second from the