

Anatomic Dead Space Cannot Be Predicted by Body Weight

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Abstract

Anatomic, airway, or tracheal, dead space is the part of the tidal volume that does not participate in gas exchange. Knowledge of the size of the dead space is important for proper mechanical ventilation, especially if small tidal volumes are used. Respiratory and medical textbooks state that anatomic dead space can be estimated from the patient's body weight. Specifically, these references suggest dead space can be predicted using a relationship of one milliliter per pound of body weight. Using a volumetric capnography monitor that incorporates on-airway flow and CO₂ monitoring (NICO₂, Respironics, Wallingford CT), anatomic dead space can be automatically and directly measured using Fowler's method in which dead space equals the exhaled volume up to the point when CO₂ rises above a threshold [4]. We retrospectively analyzed data collected in 58 (43 male, 15 female) patients to assess the accuracy of weight-based estimation of anatomic dead space. It appears that the average anatomic dead space roughly corresponds to the average body weight for the overall population; however, the poor correlation between individual patient weight and dead space contradicts the suggestion that dead space can be estimated from body weight.

Introduction

Anatomic dead space volume is the part of the tidal volume that remains in the conducting passages at the end of inspiration and therefore does not participate in gas exchange. Upon expiration, the gas from the conducting passages has the same composition as it did in inspiration; it is commonly referred to as wasted ventilation. Anatomic dead space is also called airway, tracheal or series dead space. Anatomic dead space was first measured using a fast nitrogen analyzer by Fowler¹ in 1948. By 1952, DuBois² had described anatomic dead space measurement technique using a rapid CO₂ analyzer, and by 1954, Bartels³ had shown that several indicator gases including oxygen and carbon dioxide all gave the same value for anatomic dead space and could therefore be used interchangeably.

Anatomic dead space is not a fixed value for each individual, as it is known to be influenced by several factors, most notably: anesthesia, lung volume at the end of

inspiration, posture, position of the neck and jaw, drugs acting on the bronchiolar musculature, tracheal intubation, tracheotomy, and tidal volume and respiratory rate⁴.

Many current text books⁴⁻⁷ suggest a simple estimate of anatomic dead space based on the patient's body weight or predicted body weight. Specifically, these references suggest anatomic dead space can be approximated by one milliliter per pound (or 2.2 ml per kg) of body weight. Because this dead space estimation technique has been so widely disseminated, many clinicians apply the 1 lb = 1 ml rule in clinical practice.

The observation that anatomic dead space in ml is roughly correlated with body weight in lbs seems to have been first put forth by Radford⁸ in 1955. In his article, Radford described ventilation standards he had developed to predict an individual's required ventilation based on their body weight. He presented a summary of anatomic dead

space data from eleven patient groups obtained from several researchers that included a total of 131 subjects aged newborn to 59.6 ± 6.3 years and having mean body weights ranging from about 8 to 170 pounds. Radford plotted the mean values of dead space against the mean values of body weight for each group. He observed a “remarkable, but approximate, rule that the respiratory dead space in milliliters (BTPS) equals the body weight in pounds”. This approximation served Radford’s needs well since he proposed tidal volumes that were relative to any error in dead space estimation.

Contemporary ventilation protocols such as the ARDS network⁹, which call for the use of smaller tidal volumes as part of a lung protection strategy for some patient populations, result in a larger percentage of each breath being wasted in the anatomic dead space volume. When weight-based estimates of anatomic dead space are incorrect, assumed alveolar minute ventilation may be much different from actual alveolar minute volume for patients ventilated with smaller tidal volumes and higher respiratory rates. This leads to unintentional hyperventilation or hypoventilation. The case of hypoventilation could be made worse in breathing circuits that include excessive apparatus dead space^{10, 11}.

Anatomic dead space can be directly measured using Fowler’s equal area method, which is based on volumetric capnometry¹. We analyzed data collected using a respiratory profile monitor that includes volumetric CO₂ analysis to retrospectively study how well estimated anatomic dead space predicts measured anatomic dead space for a set of mechanically ventilated patients.

Methods

We retrospectively analyzed data collected in 58 (43 male, 15 female) tracheally intubated, mechanically ventilated

patients in the operating room and ICU. These patients were monitored using a volumetric CO₂ monitor that utilizes a combination CO₂/flow sensor (NICO₂, Respirationics, Wallingford CT). This monitor calculates anatomic dead space on a breath-to-breath basis by analyzing the expiratory volume at which the CO₂ signal transitions from anatomic to alveolar CO₂ by implementing the method described by Fowler¹. For each patient, the average anatomic dead space was measured using data collected during the first 10 minutes of monitoring and compared to the values predicted using five published prediction methods, which were based on patient body weight, height, and ideal body weight. The difference, standard deviation of the difference and correlation between the measured and estimated values were calculated for each of the published prediction methods.

For 21 patients, there was an elbow placed in the breathing circuit between the endotracheal tube and the volumetric capnometry sensor. For those patients, we subtracted a volume of 6 ml from the measured anatomic dead space to compensate for the extra dead space added by the elbow. For all other patients, the endotracheal tube was connected directly to the volumetric capnometry sensor and no compensations were required.

The first, most common published anatomic dead space prediction equation is cited in many general and respiratory physiology texts⁴⁻⁷. This method simply states that anatomic dead space in ml is equal to body weight in pounds, as Radford⁸ recognized. Alternatively, this can be stated as body weight in kg multiplied by 2.2 is equal to anatomic dead space in ml. A second method commonly in use¹² uses the ideal body weight (lbs) based on the patient’s height to predict the anatomic dead space (ml). A refinement¹³ of the 1 lb = 1 ml method states that estimated anatomic dead space should be decreased by 72 ml when patients are intubated to account for the extrathoracic volume bypassed by the

endotracheal tube. Others^{13, 14} proposed reducing the estimate of 1 lb = 1 ml by 50% to account for the volume bypassed by the airway maintenance devices. The Suwa¹⁵ method is a similar but related approach that estimates dead space (ml) as 2/3 of the patient weight (lbs).

Results

The mean patient age was 63.2 ± 13.8 years (range 14-81 yrs.). The mean patient body weight was 85.3 ± 19.1 kg (188 ± 42 lbs) (range 49.9 - 136.5 kg). The mean height was 172.9 ± 9.8 cm (range 149-198 cm), the mean predicted ideal body weight was 67.6 kg (149 lbs) and the mean BSA was 2.01 ± 0.26 m². Figures 1 and 2 illustrate the correlation of measured anatomic dead space with body weight and ideal body weight

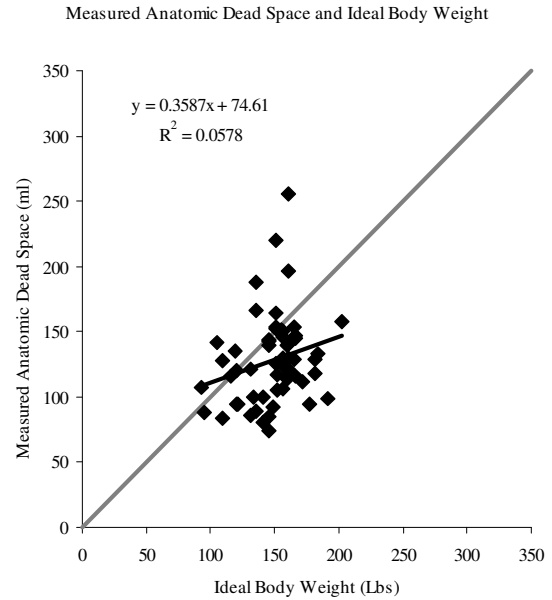


Fig. 2: Regression analysis of measured anatomic dead space and ideal body weight.

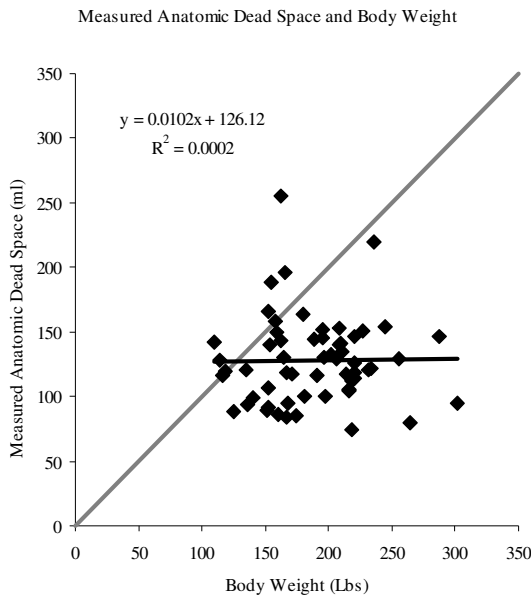


Fig 1: Correlation between measured anatomic dead space and body weight.

Table 1 reports the correlation, average difference and standard deviation of the difference when comparing each of the estimation methods described above to the measured anatomic dead space.

Method	Reference	r ²	Ave difference (ml)	SD difference (ml)
a	8	0.0002	59.9	53.9
b	12	0.058	20.9	35.9
a - 72 ml	13	0.0002	-12.1	53.9
1/2a	14	0.0002	-34.1	39.7
2/3a	15	0.0002	-2.7	43.8

Table 1: Results for each of the standard methods analyzed: method "a" (weight in pounds = anatomic dead space in milliliters)⁸, method "b" (ideal weight in pounds = anatomic dead space in milliliters)¹², method "a" - 72 ml¹³, 50% of "a"¹⁴, 66% of "a"¹⁵.

If the ideal body weight was used in each of the last three equations instead of the actual body weight, the results would be those reported in Table 2.

Method	r ²	Ave difference (ml)	SD difference (ml)
b - 72 ml	0.058	-51.1	35.9
1/2b	0.058	-53.6	33.0
2/3b	0.058	-28.7	33.6

Table 2: Results for each of the standard methods when ideal body weight is used rather than actual weight: method “b” – 72 ml, 50% of “b”, 2/3 of “b”.

The ratios of mean anatomic dead space to mean predicted dead space were 1:1.10 for “weight - 72”, Nunn’s classic method, and 1:1.7 for “ideal body weight - 72”. The ratios that were the closest to 1:1 were from the Suwa method: 1:1.02 (weight) and 1:1.29 (ideal body weight).

Discussion

The poor correlation in this data set between patient weight and measured anatomic dead space appears to contradict the common practice of estimating anatomic dead space from body weight. It appears the average anatomic dead space in milliliters corresponds to the average body weight in pounds for the overall population since the line of identity passes through the data cluster. However, based on the variability of the actual value observed in our data, there is no basis for estimating an individual patient’s anatomic dead space volume from the body weight or ideal body weight.

The 1 pound = 1 ml rule was first proposed by Radford⁸. In Radford’s original paper, he plotted anatomic dead space versus body weight in lbs. On his plot, the

error bars indicate the standard deviation of his anatomic dead space predictions were similar to those we observed. Radford emphasized that the rule of 1 ml dead space for every pound of body weight gives only a rough approximation of anatomic dead space, as evidenced by the large standard deviations of the data he presented. He warned that it is probably not justifiable to extend the dead space-to-body weight relationship in patients weighting more than 200 pounds (91 kg). Radford also elected to ignore the evidence that anatomic dead space increased with age for the purpose of his ventilation guidelines since it was a small effect and was offset by a fall in VCO₂ with age. In fact, Radford did not advocate the use of a dead space estimate for anything but a way to simplify the ventilation guidelines he was proposing. It appears that the practice of estimating dead space from body weight has become a matter of convenience, but it was not Radford’s intended message. His proposed ventilation guidelines, on the other hand, have stood the test of time and are still in wide use today as a starting point for setting automatic support ventilation and weaning protocols^{16, 17}.

Radford’s ventilation nomogram, which was based on body weight, sex and breathing frequency, required adjustment for changes in anatomic dead space associated with endotracheal intubation. He recommended a rough correction, which was defined by subtracting a volume equal to one-half the body weight from the total tidal volume. He based this recommendation on the observation that the volume of the oronasal dead space and upper part of the trachea are approximately 50% of the total anatomic dead space¹⁸. Clearly, the contemporary use of Radford’s 1:1 rule for estimating anatomic dead space was not intended by Radford to be used as an independent estimate of an intubated patient’s anatomic dead space.

Precise knowledge of the anatomic dead space becomes more important when a patient is ventilated using smaller tidal volumes as suggested by the ARDSnet⁹

ventilation recommendations. The percentage of each breath lost to anatomic dead space ventilation increases as the tidal volume decreases. As an example, consider the average patient weighing 85.3 kg in our data set. With the ARDSnet tidal volume suggestion of 6 ml/kg, the tidal volume would be set to 512 ml; since the average measured anatomic dead space is 128 ml, 25% of every breath is lost to dead space ventilation. If tidal volume were set using a rule of 10 ml/kg, only 15% of each breath would be lost to dead space; at 12 ml/kg, only 12.5% of the breath is wasted.

In our average patient with an assumed ventilation of 6 ml/kg, the predicted alveolar tidal volume (tidal volume – predicted anatomic dead space) is 324 ml based on body weight. The measured range of dead space volumes (mean ± 2 standard deviations) for this patient pool was 60 to 196 ml, which is a change in expected alveolar volume of $\pm 21\%$. The measured range of alveolar tidal volumes observed for this group of patients is 316 to 452 ml, a -3% to +40% change from the assumed alveolar tidal volume. These average numbers reveal that the effective ventilation delivered to patients on the ARDSnet protocol can be greater or less than the expected value if the individual to individual variation in anatomic dead space is not considered.

The alveolar tidal volume predicted by ideal body weight (363 ml) would lead to an erroneous estimate of alveolar minute ventilation of between -13% and +25% compared to the assumed value. Even the more complicated (and less common) method of body weight minus 72 ml gives poor estimation of actual alveolar ventilation: -20% to 14%. Given these data, direct measurement of an individual's anatomic dead space appears to be the only reliable method of assessing true dead space and therefore true alveolar ventilation.

Quantification of physiologic dead space is clinically important. Nuckton observed that an increased dead space fraction

(V_D/V_T) is independently associated with mortality in ARDS patients¹⁹. Unfortunately, in their study, Nuckton and colleagues only reported the total pulmonary dead space, so it is not possible to reanalyze their results such that anatomic dead space and alveolar dead space are separated. In a subsequent paper, Kallet et al²⁰ found that the ARDS patients with lower V_D/V_T had better survival rates. They found that the difference in V_D/V_T between survivors and non-survivors was about 0.1. A large portion of total dead space is anatomic dead space. Our data show that when the contribution of the variability in the anatomic dead space is considered, the V_D/V_T can change by ± 0.13 based solely on patient-to-patient differences in anatomic dead space. This means that the variability in anatomic dead space contributes to V_D/V_T measurements by a similar magnitude as the difference observed between survivors and non-survivors. It is likely that the prognostic value of V_D/V_T measurements is related to ventilation perfusion mismatch and not to the percent of each breath lost in anatomic dead space. However, if anatomic dead space variability is not considered, then the relationship between V_D/V_T and V/Q mismatch is weakened. Consider a patient with a low V_D/V_T and an abnormally small anatomic dead space. Based on the V_D/V_T , this patient might be considered to have a favorable prognosis when in fact serious V/Q mismatch problems are masked by a small anatomic dead space. The solution, as proposed by Moppett²¹, is to calculate the ratio of alveolar dead space to alveolar tidal volume rather than the total V_D/V_T . That is, one should measure the anatomic dead space, then subtract the anatomic dead space from both the total dead space and the tidal volume before calculating the ratio. The resulting V_D/V_T would be a ratio of alveolar dead space to alveolar tidal volume. Moppett et al. speculated that the association Nuckton and Kallet observed between dead space ratio and mortality was likely due to disturbed VQ matching, and that the alveolar dead space ratio would be even more strongly associated with mortality.

Drummond²² pointed out that right-left shunting (intra-pulmonary or intra-cardiac) affects the total dead space measurement, but not the anatomic dead space measurement. The idea of measuring anatomic dead space in order to estimate the uniformity of alveolar ventilation goes back to 1944²³⁻²⁵. Anatomic dead space volume was also used to evaluate alveolar ventilation-perfusion relationships in patients with pulmonary disease in 1949²⁶. We suggest the use of direct anatomic dead space measurement in future studies in order to develop better descriptions of the changes that occur in the alveolar dead space with lung injury.

It is important to ensure patients receive adequate tidal volume to overcome the apparatus dead space¹⁰⁻¹¹. Apparatus dead space affects both the alveolar tidal volume and V_D/V_T , and Nuckton and Kallet ensured their V_D/V_T analyses were carried out using minimal apparatus dead space. Correct assessment of the effect of all series dead space (anatomic and apparatus) requires a calculation of the apparatus dead space and addition of this volume to an estimate of anatomic dead space. Direct measurement using volumetric capnography should combine both anatomic and apparatus dead volume into a single volume.

As stated previously, the anatomic dead space is known to change with the size of the tidal volume. We made no effort in this analysis to control for the tidal volume effect. In fact, tidal volumes can be generally assumed to vary widely from patient to patient, so it is not reasonable to assume a specific tidal volume to anatomic dead space relationship. A need for assumptions about this relationship points out another significant drawback of using weight-based estimates of dead space rather than the actual measured value. Since the conducting airways are somewhat compliant the anatomic dead volume can be expected to change with time in a single individual, especially in the presence of changed ventilator settings, inhaled anesthetics, change in posture⁴ and PEEP. We tested the

effect of PEEP on anatomic dead space and found a strong correlation between increased PEEP and increased measured anatomic dead space²⁷.

Conclusion

All these issues point to the need to use direct measurements of anatomic dead space rather than estimation for proper mechanical ventilation. The errors associated with estimations were less significant when larger tidal volumes were used; however, when smaller tidal volumes are used, the percentage of each breath lost to anatomic dead space ventilation becomes greater. With volumetric capnography, it is simple to directly measure anatomic dead space under every condition and use its measure to inform treatment.

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