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# *Beware the airway filter: deadspace effect in children under 2 years*

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## *Summary*

*Background*: Filters are increasingly used in breathing circuits as they protect the circuit from contamination and facilitate humidification of inspired gas. The use of filters, however, can augment the anatomical deadspace. This can be significant in children because they have much smaller tidal volumes.

*Methods*: Following institutional ethical approval, 20 healthy children <2 years of age who required tracheal intubation were recruited. Ventilation was adjusted to achieve an endtidal carbon dioxide ($P_ECO_2$) of 4.6 kPa (35 mmHg) when sampled at the tracheal tube (TT) adapter. Following a 10-min period of stabilization, an airway filter (22 ml) was introduced into the circuit. The respiratory rate (RR) was then adjusted to return $P_ECO_2$ to 4.6 kPa (35 mmHg).

*Results*: A mean increase in ventilation of 1.42 (0.38) $l \cdot min^{-1}$ was required to maintain a normal $P_ECO_2$ level. Airway pressure and respiratory rate increased by 7.9 mmHg (4.6) and 19.8 $breath \cdot min^{-1}$ (8.7) respectively. The $P_ECO_2$ and partial pressure of inspired carbon-di-oxide ($PiCO_2$) measured from the TT adapter were higher than measured from the filter port. The mean increase was 3.6 (1.6) mmHg for $P_ECO_2$ and 5.9 (3.9) mmHg for $PiCO_2$.

*Conclusion*: Amplified deadspace from airway filters results in a significant increase in ventilation needed to maintain a normal $P_ECO_2$ in children <2 years of age with normal lungs. Sampling of $P_ECO_2$ and $PiCO_2$ from the filter significantly underestimates the effect of increased deadspace. The effect of increased deadspace may be predicted using a proposed mathematical model.

***Keywords:*** anesthesia; breathing circuit; deadspace; filters

## Introduction

During ventilation, there is an appreciable fraction of the tidal volume that does not participate in gas exchange, and this has long been known as dead-

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space (1). Deadspace may be subdivided into four different types: anatomical, alveolar, physiologic and apparatus.

The first portion of the exhaled breath contains no carbon dioxide ($CO_2$), as it comes from the anatomical deadspace, where no gas exchange occurs. However, in the latter part of exhalation, the $CO_2$ concentration rises sharply as gas from areas of active gas exchange is breathed out. Although diluted by fresh gas drawn in with the remainder of the tidal intake, anatomical deadspace still amounts to approximately 2 $ml \cdot kg^{-1}$, or 25–35% of tidal volume (2).

Alveolar deadspace results from ventilation-perfusion inequalities. The sum of alveolar and anatomical deadspaces is referred to as the physiological deadspace. It is common to express physiological deadspace as a fraction of the tidal volume, better known as the physiological deadspace ratio ($V_D/V_T$; 1).

The use of any external breathing apparatus, such as a breathing circuit, increases the distance that gas must travel before it reaches the gas exchange zone of the lungs. This additional conducting segment is the apparatus deadspace. Apparatus deadspace is in series with anatomical deadspace and can significantly augment physiological deadspace volume. The augmentation in deadspace can potentially increase the $CO_2$ that is rebreathed, which subsequently results in a rise in arterial $pCO_2$ unless the volume of ventilation is increased (3). The clinical impact of the expanded deadspace depends on the magnitude of the $V_D/V_T$ ratio. The volume of apparatus deadspace is often similar in adults and children; however, the impact in children, with smaller tidal volumes, can be very significant (4).

Breathing circuits used in anesthesia are designed to minimize deadspace, for example, by removing $CO_2$ via soda lime absorption. Their overall impact depends on their design, fresh gas flow-rate and the patient's respiratory mechanics (5).

Contaminated anesthesia breathing circuits have been implicated as a causative factor of cross-infections in hospital patients (6). Filters are designed to help prevent microbial contamination of the breathing circuit, hence allowing it to be reused for more than one patient. The practice of using filters and reusing the same anesthesia breathing circuit for multiple patients is prevalent across Canada (7). However, current recommendations of North American regulatory agencies do not support the use of filters in order to reuse anesthesia breathing circuits. The impact that these filters have on respiratory mechanics in adults has been described (8). Filters have been shown to affect deadspace in adults, yet data for children have not previously been reported.

When the filter is placed in the expiratory limb of the circuit distal to the patient before gas enters the $CO_2$ absorber, it protects the absorber from contamination but not the breathing circuit itself (see Figure 1, Configuration A). Alternatively, the filter may also be placed as close to the tracheal tube (TT) adapter and patient as possible (see Figure 1, Configuration B). The breathing circuit is protected in this configuration but the main disadvantage is that the filter increases apparatus deadspace.

When a filter is used, sampling of endtidal partial pressure of $CO_2$ ($P_ECO_2$) may be performed at the TT adapter or at the filter port. Given that filters induce a deadspace effect, location of sampling will influence $CO_2$ measurement.

The purpose of this study was to quantify the increase in ventilation required to overcome the deadspace effect introduced by inserting a filter close to the patient (Configuration B) in small children. The secondary objective was to quantify the discrepancy in $P_ECO_2$ and $PiCO_2$ estimation by sampling at the end of the filter (Configuration B) rather than at the end of the TT (Configuration A).

## Methods

### *Study patients*

After approval by the institutional ethics board, healthy children under 2 years of age weighing between 4 and 15 kg, undergoing general anesthesia and requiring tracheal intubation were recruited. Patients who met these criteria for inclusion were ineligible if they had a history of abnormal lung functions, significant cardiac disease, significant metabolic disorders, open chest or abdominal cavity during the period of measurement, increased intracranial pressure or complicated induction of anesthesia.

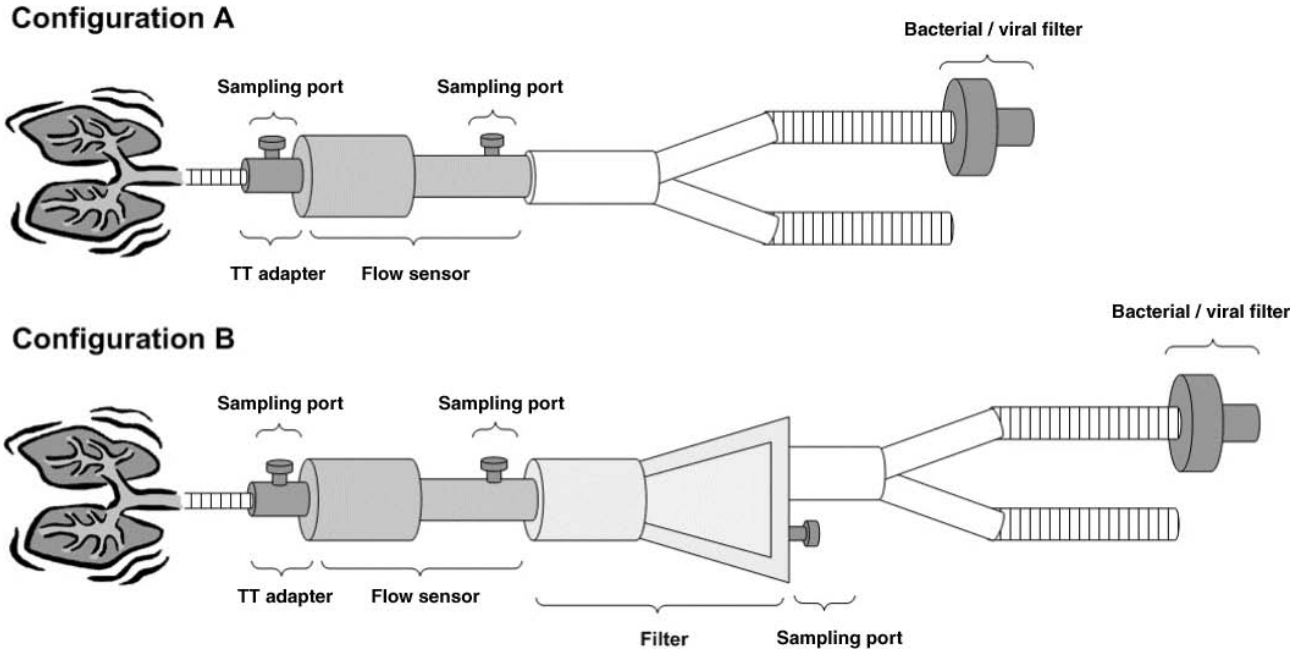


**Figure 1**
Breathing circuits with and without insertion of filter.

## *Study procedures*

After obtaining informed consent, anesthesia was induced and tracheal intubation performed as is routinely practiced using a nonstandardized protocol. Mechanical ventilation was initiated and anesthesia was maintained with an inhaled anesthetic. Cuffed tracheal tubes were used to ensure no leak.

A circuit configuration with the Aquesure filter (ICORAB, Bromma, Sweden) situated close to the $CO_2$ absorber (Configuration A) and a Datex Pedilite® (Datex, Helsinki, Finland) spirometry flow sensor close to the TT were initially used (see Figures 1 and 2). Gas analysis was performed using the Datex-Ohmeda® M-CAiOV gas analyzer with a sampling rate of 200 ml·min$^{-1}$. Fresh gas flow was adjusted to 2 l at a fractional inspired oxygen concentration ($FiO_2$) of 0.5. The tracheal connector with side sampling port was used to measure $P_ECO_2$. Volume controlled ventilation (Datex-Ohmeda® (Datex, Helsinki, Finland) 7810), with an inspiratory to expiratory ratio (I : E) of one to two was selected as the mode of ventilation. Tidal volume was set at 10 ml·kg$^{-1}$ and respiratory rate was adjusted until $P_ECO_2$ reached 4.6 kPa (35 mmHg) when measured at the TT adapter. Once the target $P_ECO_2$ was achieved, tidal volume was not changed during the study. A minimum of 10 min was then allowed for stabilization. Baseline values including airway pressure, minute ventilation, respiratory rate and $P_ECO_2$ were recorded at the TT adapter.

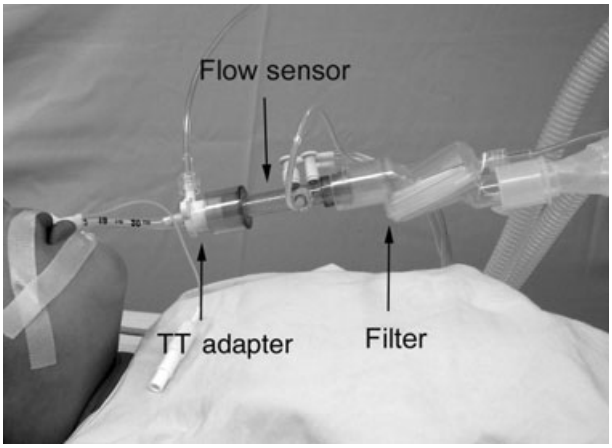


**Figure 2**
Components of a breathing circuit on an actual patient.

After the first set of data was recorded, a filter (Brathwaites Oliver Medical Aquesure Anesthesia Bacterial/Viral Filter with Gas Sampling Port, 22 ml volume) was inserted between the TT and flow sensor to create Configuration B. Following a 10-min period of stabilization, a second set of data was recorded at the TT adapter and distal end of the filter.

The respiratory rate was then increased to return the $P_{E}CO_2$ as measured at the TT to 4.6 kPa (35 mmHg) while the patient remained on Configuration B. After stabilization for 10 min, a final set of data was recorded.

### *Statistical analysis*

This study was designed to test the hypothesis that the insertion of the airway filter at the proximal end of the breathing circuit results in a 10-mmHg increase in $P_{E}CO_2$. Based on the result of a small pilot study, we assumed that the standard deviation of the difference in the $P_{E}CO_2$ is 1.6 kPa (12 mmHg). Hence, we determined that 14 patients were required to detect a treatment difference in $P_{E}CO_2$ with $\alpha = 0.05$ and $\beta = 0.2$. Our sample size target was 20 patients in order to account for withdrawals or dropouts during the study.

The analysis plan excluded children who were withdrawn from the study. Paired student's *t*-tests were used to investigate differences in $P_{E}CO_2$ and minute ventilation between Configurations A and B. A *P*-value of 0.05 or less was considered significant.

## Results

All patients were recruited at the British Columbia Children's Hospital from July 2004 to December 2004. A total of 32 patients were assessed for eligibility. Of these, two patients did not meet the inclusion criteria and 10 refused to participate. A total of 20 patients were left to allocate to intervention, but two of the remaining patients were withdrawn because of abnormal pulmonary compliance that was not apparent during induction of anesthesia. Demographic data of the 18 children who were included in the final analysis are presented in Table 1.

A mean increase in minute ventilation (MV) of 112.6 ± 60.5% was required to maintain a $P_{E}CO_2$ of 4.6 kPa (35 mmHg; Figure 3). It was observed that the $P_{E}CO_2$ and $PiCO_2$ measured from the TT adapter were higher than measured from the filter port. The mean difference was 3.6 ± 1.6 mmHg for $P_{E}CO_2$ and 5.9 ± 3.9 mmHg for $PiCO_2$. The relationship between percent increase in MV and body weight is shown in Figure 4.

**Table 1**
Patient demographics

| *Characteristics* | *Treated patients* (n = *18*) |
|---|---|
| Age [months; median (range)] | 5.5 (2–24) |
| Weight [kg; median (range)] | 7.4 (5–15) |
| Gender [male/female] | 11/7 |

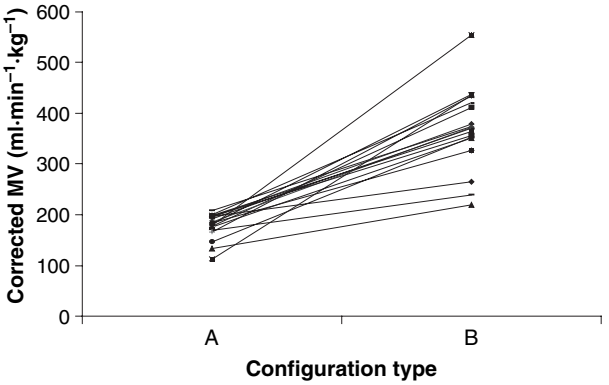


**Figure 3**
Increase in minute ventilation corrected for weight to return end-tidal $CO_2$ levels to 35 mmHg after insertion of an airway filter.

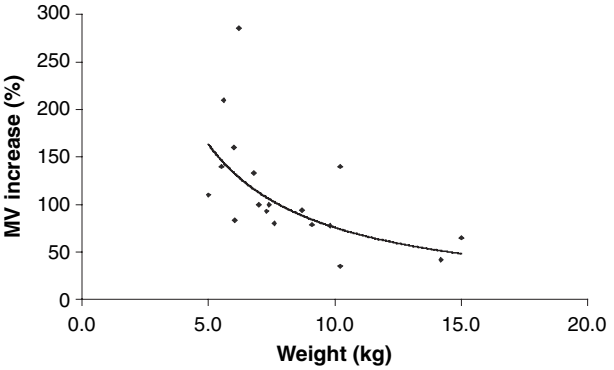


**Figure 4**
Percent increased in minute ventilation required to return end-tidal $CO_2$ levels to 4.6 kPa (35 mmHg) after insertion of an airway filter vs weight.

## Discussion

Introducing a filter into the breathing circuit helps to prevent contamination and increase humidification. However, in exchange for the protection, inserting a filter augments the anatomical deadspace and decreases the efficiency of $CO_2$ removal. This will increase the amount of $CO_2$ that is rebreathed, leading to a rise in arterial $pCO_2$. An acute rise in arterial $pCO_2$ has multiple systemic effects that may not be well tolerated. For example, intracranial pressure tends to rise

 

with increasing $pCO_2$, likely as a result of cerebral vasodilatation (1). Elevated $pCO_2$ also causes vasoconstriction in the pulmonary circulation. Physiologically, the cardinal sign of elevated $pCO_2$ is increased spontaneous ventilation; however, this sign is often absent in patients undergoing anesthesia. The arterial $pCO_2$ in an anesthetized patient will continue to rise unless ventilation is assisted.

In this study, we established that the ventilation increase required to maintain normal arterial $pCO_2$ values after insertion of a filter was significant in children under 2 years of age. The clinical impact of increasing ventilation to maintain $P_{E}CO_2$ in children with normal lungs is unknown. However, empirical evidence suggests that an increase in ventilation in patients with altered lung mechanics could pose a considerable risk (5). Patients with reduced lung compliance will also be subjected to greater deadspace effects (9). Further studies will be needed to clarify the clinical impact of increasing MV in patients with abnormal pulmonary function.

A large filter with a deadspace volume of 22 ml was used in this study, which might not be the standard practice in other institutions. However, this was the smallest bacterial/viral filter available to our institution at the time of this study's design. It was our routine practice to place a filter at the expiratory port of the circle system and dispose of the entire circuit when anesthetizing infants. In larger patients, it was our routine practice to place the filter at the more distal location (between the TT connector and the circuit). In some cases when the latter configuration was used in smaller patients we experienced difficulties with ventilation. This stimulated us to conduct this investigation.

The physiological deadspace volume is approximately 30% of the tidal volume in adults and children, but the absolute volume is actually quite small (1). However, as the tidal volume ($V_T$) is much smaller in children, any increase in physiological deadspace volume ($V_D$) caused by apparatus deadspace has a proportionally greater effect in small children. In this study, tidal volume was kept constant and hence, as the weight of a patient was increased, their tidal volume also increased. Thus, as $V_D/V_T$ decreases, a smaller increase in MV is needed to maintain normal $P_{ET}CO_2$. This is illustrated in Figure 4.

Accurate measurement of $P_{E}CO_2$ is critical to the anesthesiologist in monitoring respiratory function in any patient undergoing anesthesia. We have demonstrated that sampling at the distal end of the filter will produce an artificially low $P_{E}CO_2$ value, rendering the anesthesiologist unaware of the effect of the increased deadspace and resulting in significant hypoventilation. The falsely low $P_{E}CO_2$ is likely a result of three factors: (i) rebreathing, (ii) increased mixing and (iii) dilution due to aspiration. The endtidal gas might not have reached the end of the filter as the deadspace volume was similar to the alveolar ventilation. Some patients may have been ventilated mainly by deadspace gas. Also, as a result of the large volume of deadspace, there is increased opportunity for exhaled gas to mix with fresh gas. Moreover, dilution due to aspiration of fresh gas by the gas analyzer is possible. In certain situations such as elevated intracranial pressure and altered pulmonary function, underestimation of the actual $P_{E}CO_2$ will be clinically significant (6).

We have proposed a mathematical model in an attempt to predict the clinical effect of increased deadspace. When tidal volume is kept constant, the increase in respiratory rate (RR) after insertion of a filter may be predicted using the following equation.

$$\mathrm{RR}(B) = \frac{\mathrm{RR}(A) \times \mathrm{TV} \times \mathrm{Wt} \times (1 - \mathrm{PDSR})}{[\mathrm{TV} \times \mathrm{Wt} \times (1 - \mathrm{PDSR})] - \mathrm{DV}}$$

where RR($B$), predicted respiratory rate postinsertion of the filter; RR($A$), baseline respiratory rate; TV, tidal volume ($ml \cdot kg^{-1}$); Wt, weight (kg); PDSR, physiological deadspace ratio; DV, filter deadspace volume.

Detailed explanations on the derivation of the equation are provided at the end of this paper (see Appendix). In our study, the TV was maintained at approximately 10 $ml \cdot kg^{-1}$ once normal $P_{E}CO_2$ was reached and the filter deadspace volume was 22 ml. Based on the assumption that the physiological deadspace ratio is 0.3, we have calculated the respiratory rates for each child and plotted these values against the actual measured values observed (see Figure 5).

Alternatively, if we had kept the respiratory rate constant and varied the tidal volume, the following equation could be used to predict the new tidal volume after insertion of a filter:

$$\mathrm{TV}(B) = \frac{\mathrm{DV}}{[\mathrm{Wt} \times (1 - \mathrm{PDSR})]} + \mathrm{TV}(A)$$

 

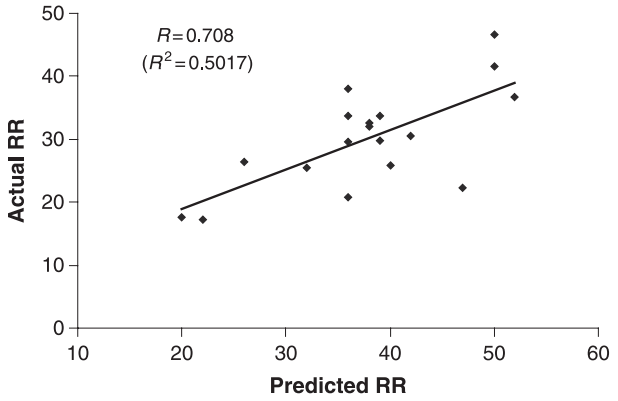


**Figure 5**
Relationship between predicted respiratory rate (RR) calculated using a proposed mathematical model vs actual RR observed after insertion of a filter.

**Table 2**
Changes after insertion of filter

| *Data* | *Value* (n = *18*) | *95% confidence interval* |
|---|---|---|
| Increased $P_{E}CO_2$ [mmHg; mean (SD)] | 10.3 (3.22) | 8.7–11.9 |
| Increased $PiCO_2$ [mmHg; median (range)] | 4 (1–9) | 3.0–5.0 |
| Increased MV [l·min$^{-1}$; mean (SD)] | 1.42 (0.38) | 1.2–1.6 |
| Increased PAW [cm$H_2O$; mean (SD)] | 7.9 (4.6) | 5.7–10.2 |
| Increased RR [breath·min$^{-1}$; mean (SD)] | 19.8 (8.7) | 15.5–24.2 |

where TV(*B*), predicted tidal volume postinsertion of the filter (ml·kg$^{-1}$); TV(*A*), tidal volume at baseline (ml·kg$^{-1}$); Wt, weight (kg); PDSR, physiological deadspace ratio; DV, filter deadpace volume.

One of the limitations in this study is that arterial $pCO_2$ values were not directly measured. Insertion of arterial catheters to measure arterial $pCO_2$ would have made this study unnecessarily invasive. Hence, we assumed that the values measured at the TT adapter were a true reflection of arterial values. Previous studies have concluded that this is a reasonable assumption (10,11). In addition, the value measured at the TT adapter is usually the value used in clinical practice. Other limitations in this study include the fact that the flow sensor itself also acts as an apparatus deadspace. We believe the augmentation in deadspace from the flow sensor is relatively small in comparsion to the filter (2 vs 22 ml). In fact, using a flow sensor, measured mean $P_{E}CO_2$ difference was 0.3 ± 0.8 mmHg. As such, the impact of inserting a flow sensor on the increase in MV is minimal compared with that of the filter (Table 2).

Amplified deadspace from airway filters results in a significant increase in ventilation needed to maintain a normal $P_{E}CO_2$ in children <2 years of age with normal lungs. We demonstrated that sampling at the distal end of the filter will produce artificially low $P_{E}CO_2$ and $PiCO_2$ values, rendering the anesthesiologist unaware of the effect of increased deadspace, potentially resulting in significant hypoventilation. The effect of increased deadspace may be predicted using the proposed mathematical model.

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## Appendix: mathematical modeling of deadspace

The volume of gas ventilated in 1 min is equal to the sum of the volume of gas in the alveoli that participate in gas exchange and the volume of gas in the anatomical, alveolar, and apparatus deadspaces that do not participate in gas exchange. Hence,

$$\mathrm{MV} = \mathrm{DSV_{Anat}} + \mathrm{DSV_{Alve}} + \mathrm{DSV_{Appa}} + V_{\mathrm{Alve}} \quad (1)$$

As physiological deadspace is the sum of anatomical and alveolar deadspaces,

$$\mathrm{MV} = \mathrm{DSV_{Anat}} + \mathrm{DSV_{phys}} + V_{\mathrm{Alve}} \quad (2)$$

The minute ventilation before the insertion of a filter (Configuration A) and after insertion of a filter (Configuration B) can be expressed, respectively, as follows:

$$\mathrm{MV}(A) = \mathrm{DSV_{phys}}(A) + \mathrm{DSV_{Appa}}(A) + V_{\mathrm{Alve}}(A) \quad (3)$$

$$\mathrm{MV}(B) = \mathrm{DSV_{phys}}(B) + \mathrm{DSV_{Appa}}(B) + V_{\mathrm{Alve}}(B) \quad (4)$$

The change in minute ventilation is hence:

$$\Delta\mathrm{MV} = \mathrm{DSV_{phys}}(B-A) + \mathrm{DSV_{Appa}}(B-A) + V_{\mathrm{Alve}}(B-A) \quad (5)$$

$\mathrm{DSV_{Phys}}$ can be estimated by taking the product of tidal volume, weight, physiological deadspace ratio and the change in respiratory rate (1).

$$\mathrm{DSV_{phys}} = \mathrm{TV} \times \mathrm{Wt} \times \mathrm{PDSR} \times \Delta\mathrm{RR} \quad (6)$$

If we assume that the deadspace volume before introducing the filter is 0 ml, $\mathrm{DSV_{Appa}}(B–A)$ may be determined as follows:

$$\mathrm{DSV_{Appa}} = [\mathrm{DV} \times \mathrm{RR}(B)] - [0 \times \mathrm{RR}(A)] = \mathrm{DV} \times \mathrm{RR}(B) \quad (7)$$

If we assume the change in alveolar ventilation to be very small, $V_{\mathrm{Alve}}\,(B - A) \approx 0$ and Eqn 5 can now be rewritten as follows:

$$\Delta\mathrm{MV} = [\mathrm{TV} \times \mathrm{Wt} \times \mathrm{PDSR} \times \Delta\mathrm{RR}] + [\mathrm{DV} \times \mathrm{RR}(B)] \quad (8)$$

The change in minute ventilation can be rewritten in terms of the change in respiratory rates:

$$[\mathrm{TV} \times \mathrm{Wt} \times \Delta\mathrm{RR}] = [\mathrm{TV} \times \mathrm{Wt} \times \mathrm{PDSR} \times \Delta\mathrm{RR}] + [\mathrm{DV} \times \mathrm{RR}(B)] \quad (9)$$

Rearranging Eqn 9 to solve for RR($B$), we get the following:

$$\mathrm{RR}(B) = \frac{\mathrm{RR}(A) \times \mathrm{TV} \times \mathrm{Wt} \times (1 - \mathrm{PDSR})}{[\mathrm{TV} \times \mathrm{Wt} \times (1 - \mathrm{PDSR})] - \mathrm{DV}} \quad (10)$$

Alternatively, if respiratory rate was kept constant instead of tidal volume, Eqn 8 would be rewritten as:

$$[\Delta\mathrm{TV} \times \mathrm{Wt} \times \mathrm{RR}] = [\Delta\mathrm{TV} \times \mathrm{Wt} \times \mathrm{PDSR} \times \mathrm{RR}] + [\mathrm{DV} \times \mathrm{RR}] \quad (11)$$

Simplifying Eqn 11 yields the following equation for predicting tidal volume postinsertion of filter:

$$\mathrm{TV}(B) = \frac{\mathrm{DV}}{[\mathrm{Wt} \times (1 - \mathrm{PDSR})]} + \mathrm{TV}(A) \quad (12)$$

DV, filter deadspace volume (ml); $\mathrm{DSV_{Anat}}$, anatomical deadspace ventilation (ml·min$^{-1}$); $\mathrm{DSV_{Alve}}$, alveolar deadspace ventilation (ml·min$^{-1}$); $\mathrm{DSV_{Appa}}$, apparatus deadspace ventilation (ml·min$^{-1}$); $\mathrm{DSV_{Phys}}$, physiological deadspace ventilation (ml·min$^{-1}$), PDSR, physiological deadspace ratio. Physiological deadspace ratio is the deadspace volume to tidal volume ratio, often abbreviated to $V_{\mathrm{D}}/V_{\mathrm{T}}$ and expressed as a percentage (1); MV, minute ventilation (ml·min$^{-1}$); RR, respiratory rate (breath·min$^{-1}$); RR ($A$), baseline respiratory rate (breath·min$^{-1}$); RR ($B$), predicted respiratory rate postinsertion of the filter (breath·min$^{-1}$); TV, tidal volume (ml·kg$^{-1}$); TV ($A$), tidal volume at baseline (ml·kg$^{-1}$); TV ($B$), predicted tidal volume postinsertion of the filter (ml·kg$^{-1}$); $\mathrm{V_{Alve}}$, alveolar ventilation (ml·min$^{-1}$); Wt, weight (kg).

 