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# The influence of physiotherapy and suction on respiratory deadspace in ventilated children

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**Abstract** *Objective:* To assess and compare the effects of respiratory physiotherapy and suction on deadspace volumes, carbon dioxide elimination ($VCO_2$), end tidal $CO_2$ ($ETCO_2$), and arterial partial pressure of carbon dioxide ($PaCO_2$) in ventilated infants and children. *Design:* Randomised crossover study. Participants received both treatments with a washout interval of more than 90 min. *Setting:* Intensive tertiary care units, Great Ormond Street Hospital, London. *Patients:* Eighty-seven fully ventilated children, requiring physiotherapy, with arterial lines in situ. Paired measurements were obtained in 81 patients, of whom 6 were excluded because of tracheal tube leak greater than 20%. *Interventions:* Respiratory physiotherapy and suction. *Measurements and results:* Data were collected April 1998–March 2000. The "$CO_2$SMO Plus" respiratory monitor was used to calculate parameters before and 30 min after both interventions. Physiotherapy lasted longer and required more saline and catheters per treatment ($p<0.005$). There were significant increases in physiological deadspace ($VD_{phys}$)/kg ($p<0.0001$), alveolar deadspace ($VD_{alv}$)/kg ($p<0.0001$) and $VD_{phys}$/tidal volume ($V_T$) ($p<0.05$) following physiotherapy that were not observed following suction. There were no significant changes following either treatment with respect to airway deadspace ($VD_{airway}$), $VCO_2$ or $PaCO_2$. Comparison of the mean differences following treatments indicated significant differences between physiotherapy and suction in terms of $VD_{phys}$/kg ($p<0.005$), $VD_{alv}$/kg ($p<0.005$), expired tidal volumes ($V_{TE}$) ($p<0.05$), mixed expired $CO_2$ ($PeCO_2$) ($p<0.04$) and $ETCO_2$ ($p<0.03$). *Conclusions:* Differences between physiotherapy and suction techniques probably accounted for their statistically distinguishable effects on deadspace. $VD_{phys}$ and $VD_{alv}$ may be more sensitive indicators of subtle changes in gas exchange and regional ventilation than $VCO_2$ or $PaCO_2$. However, interpretation of these outcomes is dependent on concurrent examination of the parameters from which they are derived.

**Keywords** Physiotherapy · Intensive care unit · Respiratory function tests · Child · Respiratory deadspace · Treatment outcome

## Introduction

Respiratory physiotherapy and suction are regularly performed in mechanically ventilated children to prevent and resolve respiratory complications. The techniques frequently used by physiotherapists for ventilated children include postural drainage, manual hyperinflation, chest wall percussion and vibration, saline instillation and tracheal suction. Routine suction does not usually include manual techniques and is usually performed by nursing staff.

Few researchers have compared physiological responses to physiotherapy or suction and none have reported changes in respiratory deadspace, although a major cause of impaired gas exchange in ventilated patients is atelectasis, causing intrapulmonary shunt. One case study reported that the alveolar deadspace ($VD_{alv}$) was significantly reduced after resolution of a partially atelectatic lung [1]. Other studies indirectly attributed improvements in hypoxaemia and compliance following physiotherapy to improved matching between alveolar ventilation and pulmonary perfusion (V/Q balance) and reduced intrapulmonary shunt [2, 3]. Measurements of respiratory deadspace have shown potential clinical benefit in diagnosis, prognosis, estimation of cardiac output and efficiency of pulmonary perfusion in some adult and paediatric patients [4, 5, 6].

However, deadspace volumes are difficult to identify anatomically and to measure directly. Methods to measure it are generally based on functional rather than morphological definitions, and theoretical models for calculating deadspace volumes from expired carbon dioxide ($CO_2$) analysis have been proposed since 1928 [7].

The principal aims of this study were to assess the effects of physiotherapy treatments and suction on deadspace volumes, carbon dioxide elimination ($VCO_2$), end tidal $CO_2$ ($ETCO_2$) and arterial partial pressure of carbon dioxide ($PaCO_2$) in fully ventilated infants and children and compare the differences between these treatments. The effects of physiotherapy and suction on respiratory mechanics, arterial blood gases and tidal volume ($V_T$) in the same population of children have been described in the accompanying manuscript [8].

## Materials and methods

### Equipment

A portable respiratory monitor ("$CO_2$SMO Plus", Novametrix Medical Systems, USA) was used to measure respiratory volumes and expired $CO_2$ concentration before and after physiotherapy and suction in the same ventilated children. The monitor measured pressure, flow and $CO_2$ concentration continuously and instantaneously via a disposable, fixed-orifice, differential flow sensor with incorporated mainstream infrared absorption capnograph. The sensor was inserted at the airway opening between the tracheal tube and the ventilator circuit. Volumes were integrated from flow and the calculations were based on expired tidal volumes ($V_{TE}$) to minimise any effects of tracheal tube leak [9, 10].

In vitro assessment of $CO_2$ measurement accuracy revealed a small but systematic underestimation of gas concentration with negligible between-sensor differences (measurement error <4% and coefficient of variation <2%). In vitro and in vivo assessments of deadspace measurements by the Ventrak 1550/Capnogard 1265 (now the "$CO_2$SMO Plus") suggested that this was a reliable and accurate alternative to other methods of calculating deadspace [11, 12].

A neonatal sensor (deadspace: 0.8 ml), was used in children under 2 years, whereas a paediatric sensor (deadspace: <4 ml) was used in older children. Apparatus deadspace was less than 1 ml/kg in all subjects and the combined apparatus resistance was less than 20% of the patient's intrinsic resistance at the mean flows likely to be encountered [13].

The clinical and practical validity of the "$CO_2$SMO Plus" in measuring $V_{TE}$, respiratory system compliance ($C_{rs}$) and respiratory system resistance ($R_{rs}$) has previously been established in this population provided that tracheal tube leak is less than 20% [9, 10, 14, 15]. Further details on the accuracy of volume measurements is described in the electronic supplementary material (S1). Ventilator settings were held constant during the measurements and the tubing was cleared of condensed water prior to data collection.

### Protocol

Data were collected in the intensive care units at Great Ormond Street Hospital for Children NHS Trust, London between April, 1998, and March, 2000. Infants and children were eligible for recruitment if they were pharmacologically paralysed, mechanically ventilated, had an arterial line in situ and were deemed by the physiotherapist to require physiotherapy. The local research ethics committee approved the study and written informed consent was obtained from parents.

This randomised crossover study involved assigning patients to receive either physiotherapy or suction in the morning and the alternative intervention in the afternoon with at least 90 min between treatments in individual patients. The order of treatment was randomised as soon as possible after recruitment by witnessed coin toss at the bedside. Arterial bloods were sampled immediately before and 30 min after each treatment using the Hewlett Packard i-Stat portable clinical analyser (i-STAT Corporation, New Jersey) [16].

The "$CO_2$SMO Plus" respiratory monitor calculated physiological deadspace ($VD_{phys}$)/kg, $VD_{alv}$/kg and $VD_{phys}/V_T$ from simultaneous $PaCO_2$ blood gas analysis. In addition, the monitor was used to measure $V_{TE}$/kg, $CO_2$ elimination ($VCO_2$/kg), $ETCO_2$, mixed expired $CO_2$ ($PeCO_2$) and anatomical deadspace ($VD_{ana}$)/kg continuously for 15 min before each treatment and 30 min after each treatment. Continuous data were sampled at 100 Hz and averaged over each minute, with cumulative mean values summarised immediately before and 30 min after treatment in 15-min epochs for comparison during analysis.

Patients were muscle relaxed, sedated and had analgesia, according to standard infusion protocols, of vecuronium, midazolam and morphine, respectively. The changes in parameters were thus likely to be related to treatment rather than changes in respiratory effort. Ventilation modality, age and weight were recorded for consideration during sub-group analysis.

The staff performed the physiotherapy and suction procedures considered appropriate for the individual patients. Both procedures involved any combination of pre-oxygenation, saline instillation, manual hyperinflation and suction, with as many or few cycles as necessary. Physiotherapy procedures were distinguishable from suction by the addition of chest wall vibrations, percussion or compression and postural drainage.

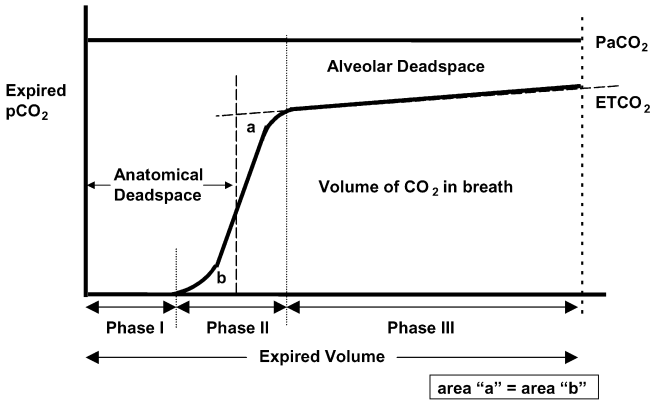


**Fig. 1** The single breath carbon dioxide ($CO_2$) curve. Phase I is the expired volume of the proximal conducting airways containing negligible amounts of $CO_2$. Phase II is the volume of the transitional region between alveolar gas and the conducting airways, characterised by a sharp increase in $CO_2$ concentration as gas from the alveoli mixes with gas from the conducting airways. Phase III primarily contains gas from alveoli and provides most of the expired $CO_2$ volume. It is usually characterised by a gently increasing slope

### Theoretical background

Physiological deadspace ($VD_{phys}$) encompasses the ventilated areas of lung and upper respiratory tract which do not participate in gas exchange. It comprises the sum of alveolar ($VD_{alv}$) and anatomical deadspace ($VD_{ana}$). The "$CO_2$SMO Plus" included ventilator apparatus deadspace (between the patient and the flow sensor) as an extension of anatomical deadspace. Apparatus deadspace was not measured or subtracted from $VD_{ana}$ since it remained constant within each patient.

Figure 1 illustrates the $CO_2$ concentration in the expired breath plotted against expired volume. The "$CO_2$SMO Plus" used a functional estimation of $VD_{ana}$ (or "Fowler's deadspace") by defining it as the volume of conducting airways at the midpoint of the $CO_2$ concentration transition between conducting airways and alveolar gas. The extrapolated phase III slope determined the point at which volumes of $CO_2$ (represented by areas ***a*** and ***b*** in Fig. 1) were equal.

The "$CO_2$SMO Plus" used the Enghoff modification of the Bohr equation to calculate $VD_{phys}/V_{TE}$:

$$VD_{phys}/V_{TE} = (PaCO_2 - PeCO_2)/PaCO_2$$

$VD_{phys}/V_{TE}$ was then multiplied by $V_{TE}$ to obtain $VD_{phys}$. $PeCO_2$ was calculated from the concentration of $CO_2$ in the expired breath. $PaCO_2$ was obtained from simultaneous arterial blood gas analysis. This method compares favourably with the traditional Douglas bag method [17].

Since $VD_{phys}$ refers to the sum of $VD_{alv}$ and $VD_{ana}$, $VD_{alv}$ was calculated by subtracting $VD_{ana}$ volume (Fowler's method) from $VD_{phys}$ (Bohr equation). $VD_{alv}$, or "wasted ventilation", is the volume of gas that enters the functional gas exchange units but does not participate in gas exchange, either because the alveoli are relatively over-ventilated or under-perfused.

The volume of $CO_2$ eliminated was the net volume of exhaled $CO_2$ measured at the tracheal tube over each minute, divided by the weight of the infant and expressed in ml/min per kg ($VCO_2$). Mixed expired $CO_2$ ($PeCO_2$), expressed in kPa, was the partial pressure of $CO_2$ in expired gas. The peak concentration of $CO_2$ averaged over eight expired breaths was reported as the $ETCO_2$.

### Statistical analysis

Paired *t*-tests with 95% confidence intervals (CI) of the difference were performed to assess the effects of respiratory physiotherapy and suction on deadspace parameters and to compare group differences [18]. In addition, the proportion of individuals in whom changes in deadspace parameters exceeded 10 and 20% following either treatment were examined and compared. There were no data available from previous studies to illustrate the extent of normal variability in $VD_{phys}$ and $VD_{alv}$ in paralysed, ventilated children in the absence of any intervention. In this study of ventilated, paralysed children, changes in excess of 10% were considered to have potentially important clinical consequences.

## Results

### Study population

Of the 100 fully ventilated infants and children recruited to the study and described in the accompanying manuscript [8], 87 had arterial lines in situ and were therefore eligible. Paired measurements of both treatments under similar measurement conditions were only obtained in 81 patients because of changes in ventilation or clinical circumstances between treatments and six further patients had to be excluded from data analysis because tracheal tube leak exceeded 20% during the measurement period. Forty-three of the remaining 75 children had a primary cardiac diagnosis (49% male: median age 8 weeks, range 3 days–16 years, median weight: 4 kg, range 2–37 kg) and 32 a primary respiratory diagnosis (65% male, median age: 22 months, range 3 days–16 years, median weight: 10 kg, range 3–87 kg). Pressure pre-set ventilation modes were used in 56 children, while 19 received volume pre-set modalities.

Infants and children with a primary cardiac diagnosis included those who had undergone surgery for congenital cardiac defects such as transposition of the great arteries, truncus arteriosus and septal defects, but also those with pulmonary hypertension and those who had undergone cardiac transplantation. Six infants, all in the cardiac population, were receiving nitric oxide (NO) therapy for suspected pulmonary hypertension. In these patients, physiotherapy and suction treatments were, as per normal practice, modified to ensure continued delivery of NO by manual inflation via the ventilation circuit.

Patients with primary or secondary respiratory problems included those with respiratory failure, head injury, abdominal surgery, bone marrow transplantation, asthma, gastric transposition surgery, inhalation injuries and tracheal reconstruction. Both groups included medical and surgical patients.

There was no difference in baseline $PaCO_2$, $PeCO_2$, $VD_{phys}$ or $VD_{alv}$ between medical and surgical groups. However, baseline $PaCO_2$ and $PeCO_2$ values in the younger cardiac patients were lower than in the respiratory group (4.85 vs 5.85 and 2.7 vs 3.8, respectively). As a

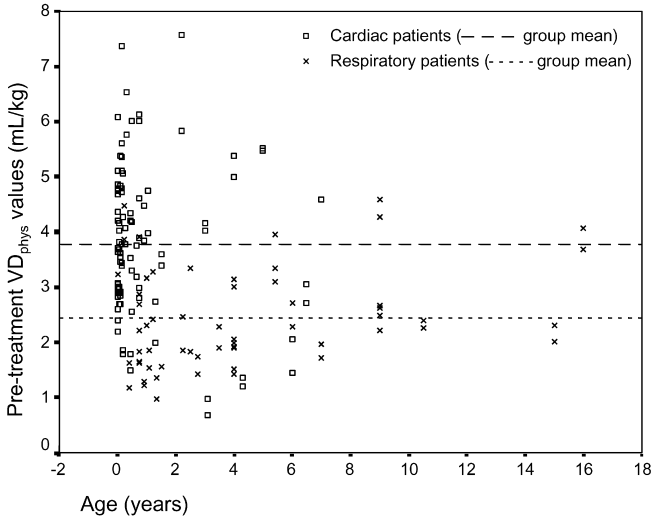


**Fig. 2** Baseline values of physiological deadspace ($VD_{phys}$) marked by cardiac or respiratory diagnosis

consequence, the median weight-corrected $VD_{phys}$ and $VD_{alv}$ were higher in the cardiac patients than the respiratory group ($VD_{phys}$: 3.9 ml/kg, range 0.7–7.6 versus 2.3 ml/kg, range 1.0–4.6 and $VD_{alv}$: 2.0 ml/kg, range: 0.2–4.3 versus 1.3 ml/kg, range: 0.3–3.7) (Fig. 2).

The median baseline oxygenation index (OI) for the group was 4.4 with a range between 2.2 and 36.4. The median OI for patients with cardiac diagnoses was marginally higher than that in the older respiratory group [4.5 (range: 2.3–36.4) versus 3.7 (2.2–10.5)]. Mean tracheal tube leak, assessed on a breath by breath basis, was less than 10% in both groups throughout the measurement period with no significant change in leak as a result of intervention in either group. The median change in percent of leak following treatment was 0.8% (−10.2 to 8%).

## Differences between treatments

As described in the accompanying manuscript [8], the physiotherapy treatments were significantly longer, involved more saline instillation and required more suction catheters per treatment ($p<0.005$).

## Effects of physiotherapy and suction

There were significant increases in $VD_{phys}$/kg ($p<0.0005$), $VD_{alv}$/kg ($p<0.0001$) and $VD_{phys}/V_T$ ($p<0.05$) following physiotherapy (Tables 1 and 2). There was also a tendency for $V_{TE}$ to increase ($p=0.08$) and for $PeCO_2$ to decrease ($p=0.06$) following physiotherapy (Table 1). There were no changes in $VD_{ana}$, $VCO_2$ or $PaCO_2$ following either intervention (Tables 1 and 2).

## Comparison of treatments

The significant changes in mean within-subject values following physiotherapy and suction and the differences between treatments are summarised in Table 3. There were significant differences between treatments for $VD_{phys}$/kg and $VD_{alv}$/kg ($p\leq0.005$). In addition, there were significant differences in $V_{TE}$, $PeCO_2$ and $ETCO_2$ ($p<0.05$) because of the difference in direction of change following physiotherapy and suction. There were no significant differences between treatments with respect to $VD_{airway}$, $VD_{phys}/V_T$, $VCO_2$, pH, $PaO_2$ or $PaCO_2$.

## Within-subject changes

There was considerable individual variation in response to both physiotherapy and suction with respect to expired $CO_2$ and deadspace parameters. The proportions of individuals demonstrating changes in $VD_{phys}$ in excess of

**Table 1** Effect of physiotherapy on deadspace and blood gas parameters

| Parameter | Before physiotherapy (B) | After physiotherapy (A) | 95% CI (A–B) | Mean % change |
|---|---|---|---|---|
| $VD_{phys}$ (ml/kg) | 3.21 (1.36) | 3.51 (1.60) | 0.15 to 0.42 | 9.4[a] |
| $VD_{alv}$ (ml/kg) | 1.64 (1.05) | 1.92 (1.33) | 0.16 to 0.41 | 17.1[a] |
| $VD_{ana}$ (ml/kg) | 1.61 (0.54) | 1.60 (0.55) | –0.05 to 0.03 | –0.6 |
| $VD_{phys}/V_T$ | 0.41 (0.14) | 0.42 (0.14) | 0.00 to 0.03 | 2.4[b] |
| $V_{TE}$ (ml/kg) | 8.21 (2.66) | 8.41 (2.78) | –0.02 to 0.42 | 2.4 |
| $PeCO_2$ (kPa) | 3.13 (0.99) | 3.05 (0.87) | –0.16 to 0.005 | –2.6 |
| $VCO_2$ (ml/min per kg) | 4.26 (1.68) | 4.27 (1.55) | –0.12 to 0.13 | 0.2 |
| $ETCO_2$ (kPa) | 5.03 (1.40) | 4.90 (1.15) | –0.29 to 0.03 | –2.6 |
| $PaCO_2$ (kPa) | 5.32 (1.47) | 5.26 (1.10) | –0.29 to 0.16 | –1.1 |

*$VD_{phys}$* physiological deadspace, *$VD_{alv}$* alveolar deadspace, *$VD_{ana}$* anatomical deadspace, *$V_T$* tidal volume, *$V_{TE}$* expired tidal volume, *$PeCO_2$* mixed expired carbon dioxide, *$VCO_2$* carbon dioxide elimination, *$ETCO_2$* end tidal carbon dioxide, *$PaCO_2$* arterial partial pressure of carbon dioxide
Results are expressed as means (SD)
[a] $p<0.0001$, [b] $p<0.05$

**Table 2** Effect of suction on deadspace and blood gas parameters

| Parameter | Before suction (B) | After suction (A) | 95% CI (A–B) | Mean % change |
|---|---|---|---|---|
| $VD_{phys}$ (ml/kg) | 3.32 (1.46) | 3.32 (1.32) | –0.14 to 0.14 | 0.05 |
| $VD_{alv}$ (ml/kg) | 1.74 (1.15) | 1.73 (1.07) | –0.12 to 0.15 | –0.6 |
| $VD_{ana}$ (ml/kg) | 1.59 (0.55) | 1.59 (0.53) | –0.03 to 0.03 | 0.0 |
| $VD_{phys}/V_T$ | 0.40 (0.14) | 0.41 (0.13) | –0.01 to 0.02 | 2.5 |
| $V_{TE}$/kg (ml/kg) | 8.52 (2.89) | 8.31 (2.48) | –0.52 to 0.09 | –2.5 |
| $PeCO_2$ (kPa) | 3.08 (0.81) | 3.11 (0.81) | –0.04 to 0.09 | 1.0 |
| $VCO_2$ (ml/min per kg) | 4.19 (1.21) | 4.23 (1.30) | –0.09 to 0.17 | 1.0 |
| $ETCO_2$ (kPa) | 4.94 (1.23) | 5.02 (1.13) | –0.03 to 0.18 | 1.6 |
| $PaCO_2$ (kPa) | 5.20 (1.20) | 5.24 (1.05) | –0.12 to 0.21 | 0.8 |

*$VD_{phys}$* physiological deadspace, *$VD_{alv}$* alveolar deadspace, *$VD_{ana}$* anatomical deadspace, *$V_T$* tidal volume, *$V_{TE}$* expired tidal volume, *$PeCO_2$* mixed expired carbon dioxide, *$VCO_2$* carbon dioxide elimination, *$ETCO_2$* end tidal carbon dioxide, *$PaCO_2$* arterial partial pressure of carbon dioxide
Results are expressed as means (SD). There were no significant changes

**Table 3** Significant differences between physiotherapy and suction (paired data)

| Parameter | Mean Δ after physiotherapy (P) | Mean Δ after suction (S) | 95% CI (S–P) |
|---|---|---|---|
| $VD_{phys}$/kg (ml/kg) | 0.29 (0.58) | –0.01 (0.60) | 0.09 to 0.49[a] |
| $VD_{alv}$/kg (ml/kg) | 0.29 (0.55) | –0.03 (0.57) | 0.12 to 0.51[a] |
| $V_{TE}$/kg (ml/kg) | 0.20 (0.95) | –0.21 (1.29) | –0.80 to –0.03[b] |
| $PeCO_2$ (kPa) | –0.08 (0.34) | 0.03 (0.27) | 0.01 to 0.20[b] |
| $ETCO_2$ (kPa) | –0.13 (0.66) | 0.08 (0.45) | 0.03 to 0.39[b] |

*$VD_{phys}$* physiological deadspace, *$VD_{alv}$* alveolar deadspace, *$V_{TE}$* expired tidal volume, *$PeCO_2$* mixed expired carbon dioxide, *$ETCO_2$* end tidal carbon dioxide
Results are expressed as means (SD)
↖ refers to 'change'
[a] $p \leq 0.005$, [b] $p < 0.05$

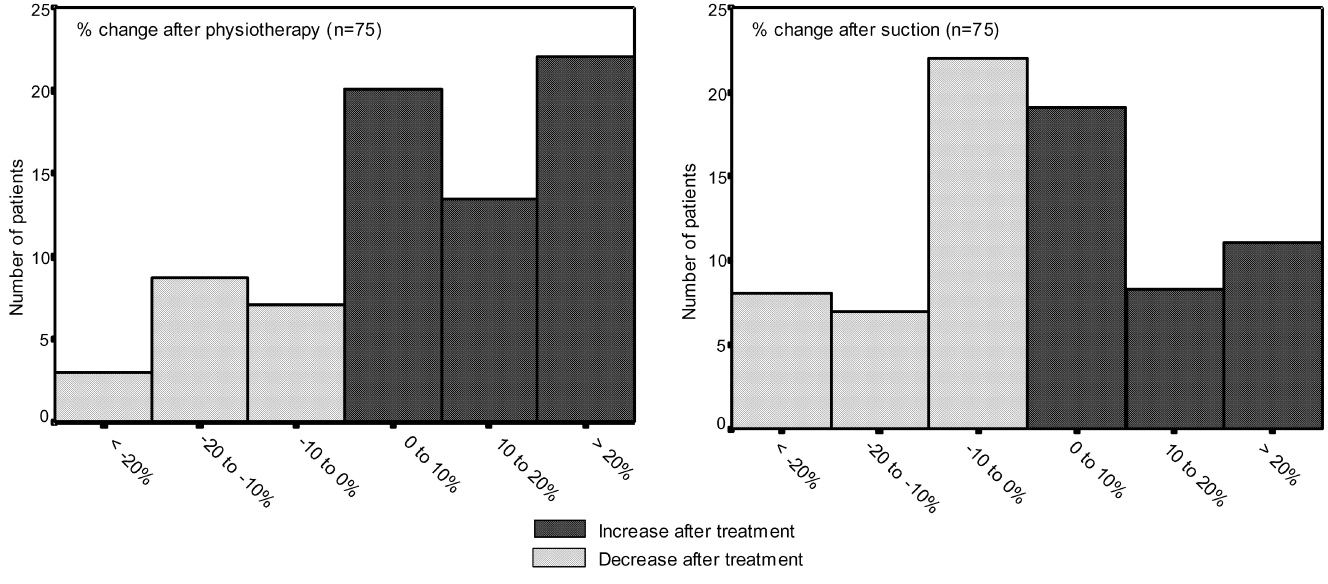


**Fig. 3** Relative changes in physiological deadspace (*$VD_{phys}$*) following physiotherapy and suction

10% and 20% in response to either treatment are shown in Fig. 3. $VD_{phys}$ and $VD_{alv}$ increased by more than 10% in 36/75 (48%) patients undergoing physiotherapy compared with only 18/75 (24%) of those following suction ($p>0.002$).

There was no relationship between OI and adverse events or response to treatment for either physiotherapy or suction. Despite the differences in baseline $VD_{phys}$ and $VD_{alv}$, both cardiac and respiratory groups independently showed significant increases in $VD_{phys}$ and $VD_{alv}$ in re-

sponse to physiotherapy while neither responded to suction alone.

## Discussion

The significant increases in $VD_{phys}$, $VD_{alv}$ and $VD_{phys}/V_T$ following physiotherapy and significant differences between treatments with respect to $VD_{phys}$, $VD_{alv,}$ $V_{TE}$, $PeCO_2$ and $ETCO_2$ were likely to be related to some or all of the physical differences between physiotherapy and suction delivery including factors such as treatment duration, saline instillation, suction catheters used and the additional performance of chest wall vibrations.

### Factors influencing deadspace volumes

Deadspace is proportional to body weight, height and body surface area [19] and affected by body and neck position, alveolar volume at the end of expiration, $V_{TE}$, intubation status, ventilation modality and length of tracheal tube [20]. Since most of these variables remained constant, changes were likely to reflect changes in alveolar ventilation, $V_{TE}$ or pulmonary perfusion. Increases in $VD_{alv}$ reflect the degree to which alveolar ventilation and perfusion fail to match each other, regionally or globally in the lung [21]. This may be caused by any condition that abnormally elevates the V/Q ratio such as pulmonary hypotension, pulmonary embolus, hyperventilation, obstruction of pulmonary arterioles and alveolar hyperinflation. In children both gross hypo- and hyper-perfusion cause an increase in $VD_{alv}$ [22].

The modified Bohr equation requires $PaCO_2$, $PeCO_2$ and $V_{TE}$ and $VD_{airway}$ to derive $VD_{phys}$. These parameters fluctuate in a ventilated child and $VD_{phys}$ is thus dependent on the relative magnitude of each of these variables at any specific time. The tendency for $V_{TE}$ to increase and $PeCO_2$ to decrease following physiotherapy resulted in an amplification of $VD_{phys}$ and $VD_{alv}$ (since group changes in $PaCO_2$ and $VD_{airway}$ were negligible).

An increase in $VD_{phys}$ or $VD_{alv}$ appears at face value to be a cause for concern. However, since $VD_{alv}$ is derived from four dynamically fluctuating variables, changes may reflect a transient pulmonary state, before a new equilibrium between ventilation and perfusion is reached. One case report indicated a reduction in $VD_{alv}$ after manual hyperventilation and tracheal suction in a child with acute lobar atelectasis [1], perhaps reflecting a rapid reduction in the $PaCO_2/PeCO_2$ gradient following resolution of atelectasis, and a relatively smaller increase in $V_{TE}$. If this child had presented with a chronic lobar atelectasis, in which there was substantial hypoxic pulmonary vasoconstriction (HPV), a rapid recruitment of alveoli may not immediately have been matched by reperfusion. In this example, the increase in $V_T$ would, at least temporarily, have been reflected as "wasted ventilation" with increased $VD_{alv}$.

The time taken to restore V/Q equilibrium following the recovery of atelectasis is variable and may depend on the resolution of HPV. Research suggests that there is a time continuum in which HPV occurs and in which it is relieved [23]. Resolution depends on the proportion of lung involved, age, gravity and posture, period of localised atelectasis [24], mixed venous oxygen saturation [25], vascular distending pressure [26] and presence of NO or other pharmacological factors influencing the reactivity of pulmonary vasculature [27]. Disconnection of NO during treatment might result in pulmonary vasoconstriction and increased deadspace. However, routine modification of physiotherapy and suction ensured this did not occur. Finally, pulmonary microvascular networks are extremely complex in nature and the reactivity to hypoxia is perceptibly different in different lung regions [28].

Within the clinical environment, therefore, it is impractical to predict the length of time required for pulmonary reperfusion following the resolution of atelectatic areas. It is possible that sampling arterial blood gases only 30 min after treatment would not have allowed sufficient time for V/Q equilibrium. By contrast, the tendencies for $V_{TE}$ to increase and $PeCO_2$ to decrease following physiotherapy were likely to be immediate and may have indicated some recruitment of atelectatic areas. In the absence of a concurrent increase in pulmonary perfusion, $VD_{phys}$ would have increased as a result of the increased gradient between $PeCO_2$ and $PaCO_2$ [22].

In this study, there were no group changes in the "gold standards" $VCO_2$ or $PaCO_2$ after either treatment, indicating that pulmonary homeostasis was preserved despite the change in V/Q balance suggested by the increase in $VD_{phys}$ and $VD_{alv}$. A compromise in $CO_2$ elimination would be reflected by a rise in $PaCO_2$, which was not observed. This may illustrate the relative insensitivity of these standard measures in identifying subtle changes in regional ventilation and V/Q balance. $VD_{alv}$ and $VD_{phys}$ could offer a more sensitive measure of subtle changes in respiratory physiology following interventions such as physiotherapy, but have not commonly been used as outcomes in clinical research. While measurements of $VD_{phys}$ and $VD_{alv}$ are not dependent on pharmacological paralysis, they do require minimal tracheal tube leak, adequate expiration time ($t_E$), an arterial line in situ for blood gas analysis and $PeCO_2$ calculation. This may make it an impractical measure for many patients in the intensive care unit.

Volume or pressure pre-set ventilation modalities may have had the potential to influence deadspace measurements. There were no group changes in $V_{TE}$ in the subgroup of patients receiving volume pre-set ventilation. However, even in this subgroup, $VD_{alv}$ increased significantly following physiotherapy alone, suggesting that

changes in the distribution of alveolar ventilation occurred, rather than gross changes in $V_{TE}$. A proportion of the ventilation delivered could, for example, have been re-directed from hyper-expanded regions to previously atelectatic regions, recruited during treatment. In this subgroup, there was also a significant reduction in PIP ($p<0.05$) after physiotherapy, indicating an improvement in respiratory compliance. The recently proposed "avalanche" model of inflation of collapsed regions of the lung suggests that rapid and variable pressure changes within the lung (such as those that may be generated during physiotherapy) may open up a successive stream of previously closed airways [29]. This theory may account for changes in regional ventilation reflected by the increase in $VD_{phys}$ and $VD_{alv}$, despite the absence of gross changes in $V_T$ or $PaCO_2$.

### *Cyanotic heart disease*

Children with cyanotic congenital heart disease have significantly larger $VD_{phys}/V_T$ than normal children or those with acyanotic heart disease ($p<0.01$) [30], as a consequence of intra-pulmonary shunt. This was confirmed by the increased baseline values of $VD_{phys}$ and $VD_{alv}$ in the cardiac population, though most infants had already undergone corrective surgery. The changes observed after physiotherapy, however, could not be attributed to an increase in right-left shunt, since this would very likely be accompanied by an increase in the arterial-alveolar gradient (reflected by an increase in $PaCO_2$). This was not supported by the data. In addition, suction alone did not result in any change in $VD_{phys}/V_T$ in the cardiac population and significant increases in $VD_{phys}$ were also demonstrated in the non-cardiac population after physiotherapy.

### *Broncho-constriction*

Some physiotherapy techniques may cause broncho-constriction [31] and $VD_{ana}$ decreases in response to bronchial constriction [32]. In this study, $VD_{ana}$ did not change in either group, indicating that broncho-constriction was not an important consequence of treatment in this sedated and muscle-relaxed patient population. $VD_{ana}$ may be a more important outcome in self-ventilating patients or those with underlying bronchial hyper-responsiveness.

### *$ETCO_2$ vs $PaCO_2$*

End tidal carbon dioxide monitoring is easily available, non-invasive and has been investigated at length as an alternative to $PaCO_2$. While some studies suggest that $ETCO_2$ and $PaCO_2$ may sometimes be used interchangeably [33, 34], others have noted that there are many clinical scenarios in which $ETCO_2$ cannot accurately predict $PaCO_2$ [35, 36, 37]. The current study confirmed that dynamic and sometimes rapid changes in deadspace volumes following clinical interventions make $ETCO_2$ an extremely unreliable substitute for $PaCO_2$ in an intensive care unit.

## Conclusion

We have previously reported material differences in the physical delivery of physiotherapy and suction treatments with subtle differences in their effects on respiratory function [8]. The findings presented in this study confirm that the interventions have small but distinguishable effects on regional ventilation and V/Q balance, with physiotherapy resulting in significant increases in $VD_{alv}$ and $VD_{phys}$.

Measures of $VD_{phys}$ and $VD_{alv}$ show promise as sensitive indicators of subtle changes in gas exchange and regional ventilation. However, changes due to clinical interventions such as physiotherapy are likely to be complex. Meaningful interpretation of these outcomes is absolutely dependent on concurrent evaluation of changes in the parameters from which they are derived ($V_{TE}$, $VD_{ana}$, $PeCO_2$ and $PaCO_2$).

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