Use of helium-oxygen mixture in adult patients presenting with exacerbations of asthma and chronic obstructive pulmonary disease: a systematic review

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Summary
We examined systematically all controlled and cross-over randomised trials in patients with acute exacerbations of asthma and chronic obstructive pulmonary disease comparing Heliox against air-oxygen mixtures. Fourteen studies were identified. In asthma studies, peak expiratory flow rate (PEFR) was increased by an average of 29.6% (95% CI 16.6–42.6) by Heliox-driven nebulisers, or by 13.3 l.min⁻¹ (95% CI 3.71–22.81) absolute. In studies of patients with chronic obstructive pulmonary disease receiving non-invasive ventilation the arterial carbon dioxide tension (PₐCO₂) and respiratory rate were unchanged: weighted mean difference for PₐCO₂ = 0.29kPa (95% CI –0.64–0.07) favoured Heliox, and for respiratory rate 1.6 breaths.min⁻¹ (95% CI –0.93, 4.14) favoured control. Heliox minimally reduced the work of breathing in intubated patients, and reduced intrinsic positive end expiratory pressure (iPEEP). The use of Heliox to drive nebulisers in patients with acute asthma slightly improves airflow measures. We were unable to determine whether this improved recovery.

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Helium is an inert and pharmacologically inactive gas with a density one eighth that of nitrogen. Helium (70–80%) blended with oxygen (Heliox) has a density one-third that of room air. In the first to tenth generations of human airways, gas flow is predominantly turbulent due to the branching structure of the bronchial tree. Resistance to gas flow is density-dependent and so gases with a lower density than air should reduce the work of breathing. Gas flow beyond the tenth generation is laminar and hence is density-independent [1].

Heliox was first used in patients with acute asthma and upper airway obstruction by Barach in 1934, who observed some relief of dyspnoea [2]. However, despite the theoretical benefits of Heliox where the work of breathing overloads respiratory capacity, such as acute exacerbations of asthma and chronic obstructive pulmonary disease (COPD), it has not been widely adopted in the seven decades since its clinical use was first described. Our aim was to determine whether this was due to a lack of evidence or a lack of efficacy.

Systematic reviews of the use of Heliox include a Cochrane review in spontaneously breathing patients, without artificial airways, with exacerbations of asthma. That review combines adult and paediatric data and was last updated in 2002 [3]. A Cochrane review was also carried out for Heliox use in exacerbations of COPD in 2000 [4]. Only two studies were included due to a lack of data availability. Further studies on this subject have since been published.

We therefore report an up-to-date systematic review of the use of Heliox in adult patients presenting with exacerbations of asthma and COPD.

Methods
We searched Medline, EMBASE and the National Research Register in November 2005. Search terms
were those recommended by the Cochrane collaboration to identify randomised trials together with the terms ‘helium OR Heliox OR helium-oxygen’. We identified relevant studies initially by title and abstract, and then reviewed the full text of potentially relevant studies. We excluded studies on children (< 16 years) and basic physiology studies on healthy volunteers. Language or year of publication was not used as a restriction. We searched the bibliographies of identified reviews, including the Cochrane reviews, and consulted expert colleagues.

We included randomised controlled trials and crossover trials where the treatment sequence was randomly determined, involving patients with acute exacerbations of asthma or COPD.

The study intervention was the use of Heliox compared to air-oxygen. Outcome measures for use in the review were selected after preliminary screening of the identified studies. In the studies on patients with asthma these were peak expiratory flow rate (PEFR) and forced expiratory volume in 1 s (FEV$_1$). In the studies in patients with exacerbations of COPD, we looked at arterial partial pressure of carbon dioxide ($P_aCO_2$), respiratory rate, work of breathing and intrinsic positive end expiratory pressure (iPEEP).

Study characteristics and the outcomes listed above were collected for all identified studies. Study quality was assessed using the Jadad score, a score of zero being the lowest quality and five being the highest [5].

$I^2 > 50\%$ was used to indicate significant heterogeneity ($I^2$ describes the percentage of the variability in effect estimates that is due to heterogeneity rather than chance. A value greater than 50\% is usually considered to represent substantial heterogeneity).

All outcome measures selected were continuous variables; we therefore calculated weighted mean difference and 95\% confidence intervals with either fixed and random effects models (depending on heterogeneity) using RevMan 4.2 software (Cochrane Collaboration, Oxford, UK). For cross-over studies the two treatments were analysed as if they were separate patient groups in a randomised controlled trial.

**Results**

Figure 1 illustrates the process used to identify studies and the reasons for exclusion.

Two further studies were identified from the reference lists of reviews (Jaber et al. [6] and Esquinas et al. [7]). A single citation was obtained from expert colleagues (COPD [8]).

The 14 identified studies were divided into groups based on the condition studied (asthma or COPD) and the use made of Heliox (nebuliser driving gas, breathing gas for non-invasive ventilation, breathing gas in spontaneously breathing intubated patients). The Heliox mixtures varied from 20\% to 30\% oxygen, presumably reflecting local availability.

Amongst the asthma studies Dorfman et al. [9] published his results only as a research letter; however, it had a Jadad score of 2/5 and was therefore included. One study by Lee et al. [10] contained two datasets; one was restricted to patients over 40 years of age and was excluded as it did not comprise part of any randomised comparison. One report by Xie et al. [8] (COPD) contained data on both COPD and asthma patients. The study on asthma patients contained only eight patients and treatments were inconsistent, these data were not included.

Studies variously reported absolute values for outcome variables, or relative changes as percentages. All the
authors of the latter studies were sent e-mail requests for further data to allow absolute changes to be calculated, but none replied. As a result the two different measures of a change in an outcome variable had to be analysed separately.

**Study quality**

Tables 1–3 (study characteristics) demonstrate the overall deficiency in blinding. It is difficult to blind these trials in un-intubated patients due to the ‘Donald Duck’ voice change which occurs with the use of Heliox.

**Results in the asthma group**

**Heliox driven nebulisers**

The forest plots in Fig. 2 illustrate meta-analyses of the effect of Heliox on PEFR by absolute and percentage change in patients with asthma receiving bronchodilators, using Heliox or air–oxygen driven nebulisers. There is a positive effect seen in favour of Heliox, with a weighted mean difference of 13.29 l.min$^{-1}$ (95% CI 3.76–22.81) in the analysis of absolute improvement in PEFR from base-line (Rose et al. [11] and Xie et al. (asthma) [12]). In the two studies examining percentage increase in PEFR (Lee et al. [10] and Dorfman et al. [9]) the weighted mean difference of 29.6% (95% CI 16.6–42.6) favours Heliox.

Three studies examining changes in FEV$_1$ after bronchodilators were nebulised using Heliox or air–oxygen driven nebulisers. There is a positive effect seen in favour of Heliox, with a mean change (SD) of 1.43 (0.5) vs 1.26 (0.4) l.min$^{-1}$ in Heliox and control groups, respectively. Rose et al. [11] reported similar findings (mean change (SD) for Heliox and control groups, respectively (1.43 (0.5) vs 1.26 (0.4) l.min$^{-1}$). The third paper by Kress et al. [13] reported the percentage change in median FEV$_1$ from baseline. His study suggested a benefit for Heliox with an increase in median FEV$_1$ from baseline of 65.1% (interquartile range 27.2%) with Heliox against an increase of 26.6% (interquartile range 34%) with air–oxygen breathing (p < 0.016).

Only two studies report ‘patient centred’ outcomes. Dorfman et al. [9] found 5/20 vs 0/19 (Heliox vs air–oxygen breathing) patients required hospital admission after treatment. Lee et al. [10] reported 18/40 vs 12/40 admissions to hospital (Heliox vs air–oxygen breathing). Neither group found any statistically significant difference.

**Heliox breathing**

A single paper Kass and Terregino [14] examined the effect of using Heliox as the breathing gas in asthma, compared to air–oxygen. He found a significant improvement in mean PEFR where patients breathed Heliox (Heliox 270.6 (SD 31.3) vs 235.0 (SD 25.2) l.min$^{-1}$, p < 0.001).

**Subjective effects of Heliox breathing and Heliox driven nebulisers**

Two papers reported the effect of Heliox on patients’ sensation of breathlessness. Rose et al. [11] reported a significant improvement in the patient’s perceived dyspnoea score (Borg scale 1–10 points) with Heliox driven nebulisation. Two hours from baseline there was a 1.6 point greater improvement in reported scores (95% CI 0.3–3.0). Kass and Terregino [14] reported an earlier significant fall in reported breathlessness in the group breathing Heliox (visual analogue breathlessness scale 0–10) 20 min from the outset of treatment (mean (standard error of the mean) 6.18 (0.76) at time 0 and 3.81 (0.80) at 20 min). However, at the end of the trial period of 480 min both Heliox and air–oxygen breathing groups had achieved significant reductions in shortness of breath scores (respectively mean (standard error of the mean): 1.56 (0.31), air–oxygen group 1.22 (0.37)). There was no significant difference between the two groups at 20 min or at 480 min.

**Differential effects depending on patient characteristics**

In one study (Lee et al. [10]) a post-hoc analysis was performed to determine patient characteristics associated with a larger effect size following Heliox use for nebulising bronchodilators. The authors found age > 40 years and a lower PEFR at baseline were associated with a greater response. They went on to study a cohort of 80 patients with acute exacerbations of asthma over the age of 40 with PEFR ranging from 18.1% to 37.3% of predicted. They found a significantly greater response to nebulisation with Heliox in the patients in the lowest quartile of baseline PEFR (mean and standard deviation of change in predicted PEFR 23% (SD 7%) compared with patients in the highest quartile of baseline PEFR (mean and standard deviation of change in predicted PEFR 15% (SD 10%), p = 0.05).

**Results in the COPD group**

**Heliox use with non-invasive ventilation (NIV)**

The forest plots in Fig. 3 illustrate the results from the studies examining Heliox use in the treatment of acute exacerbations of COPD in conjunction with NIV and invasive ventilation.

In the four studies [6, 7, 15, 16] that examined Heliox as the breathing gas during NIV there was no significant
Table 1 Asthma studies included (all randomised controlled trials).

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Intervention</th>
<th>Primary outcome</th>
<th>Secondary outcome</th>
<th>Allocation concealment</th>
<th>Blinding</th>
<th>Group sizes (ITT) and withdrawals</th>
<th>Power</th>
<th>Jadad score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kass &amp; Terregino [14] 1999</td>
<td>Patients with moderate-severe asthma presenting to an emergency department</td>
<td>Heliox 70 (70% helium) added to standard treatment</td>
<td>PEFR [14]</td>
<td>Respiratory rate Dyspnoea score</td>
<td>Unable to ascertain</td>
<td>Open</td>
<td>Treatment 11 Control 12</td>
<td>80%</td>
<td>0.05</td>
</tr>
<tr>
<td>Dorfmam et al. [9] 2000</td>
<td>Patients with moderate-severe asthma presenting to an emergency department</td>
<td>Nebulisers driven by Heliox 80 (80% helium) vs air-oxygen</td>
<td>% change in PEFR</td>
<td>Admission rate</td>
<td>Unable to ascertain</td>
<td>Open</td>
<td>Treatment 21 Control 19</td>
<td>80%</td>
<td>0.05</td>
</tr>
<tr>
<td>Rose et al. [11] 2001</td>
<td>Patients with moderate-severe asthma presenting to an emergency department</td>
<td>Nebulisers driven by Heliox 70 (70% helium) vs air-oxygen</td>
<td>PEFR FEV₁</td>
<td>Respiratory rate Arterial oxygen saturation Dyspnoea score</td>
<td>Adequate</td>
<td>Blinded by shrouding apparatus Double blinded</td>
<td>Treatment 20 Control 19</td>
<td>80%</td>
<td>0.05</td>
</tr>
<tr>
<td>Kress et al. [13] 2002</td>
<td>Patients with moderate-severe asthma presenting to an emergency department</td>
<td>Nebulisers driven by Heliox 80 (80% helium) vs air-oxygen</td>
<td>FEV₁</td>
<td>Admission rate</td>
<td>Unable to ascertain</td>
<td>Patients unblinded due to voice changes</td>
<td>Treatment 23 Control 22</td>
<td>80%</td>
<td>0.05</td>
</tr>
<tr>
<td>Xie et al. [12] 2003 (Part I)</td>
<td>Patients with mild- moderate asthma in the out-patient department</td>
<td>Nebulisers driven by Heliox 79 (79% helium) vs air-oxygen</td>
<td>PEFR FEV₁ FVC</td>
<td>Unable to ascertain</td>
<td>Personnel blinded Patients not explicitly told Single blinded</td>
<td>Treatment 13 Control 11</td>
<td>Not stated</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Lee et al. [10] 2005 (Part I)</td>
<td>Patients with moderate-severe asthma presenting to an emergency department</td>
<td>Nebulisers driven by Heliox 80 (80% helium) vs air-oxygen</td>
<td>% change in PEFR</td>
<td>Admission rate</td>
<td>Adequate</td>
<td>Blinded by shrouding the apparatus Double blinded</td>
<td>Treatment 40 Control 40</td>
<td>80%</td>
<td>0.01</td>
</tr>
</tbody>
</table>

PEFR, peak expiratory flow rate; FEV₁, forced expiratory volume in 1 s; FVC, forced vital capacity.
Table 2 COPD studies: Heliox driven nebulisers or non-invasive ventilation with Heliox.

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Intervention</th>
<th>Primary outcome</th>
<th>Secondary outcomes</th>
<th>Allocation concealment</th>
<th>Blinding</th>
<th>Group sizes and withdrawals</th>
<th>Power</th>
<th>Jadad score (0–5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jolliet et al. [15] 1999</td>
<td>Decompensated patients with COPD on a medical ICU</td>
<td>Non-invasive ventilation with Heliox 70 (70% helium) vs air-oxygen</td>
<td>$P_aCO_2$</td>
<td>$P_aO_2$</td>
<td>Adequate</td>
<td>Patients blinded</td>
<td>20 patients included in a cross-over design; one patient was removed due to technical problems</td>
<td>Not stated</td>
<td>3</td>
</tr>
<tr>
<td>DeBloisblanc et al. [19] 2000</td>
<td>Patients with COPD presenting to an emergency department</td>
<td>Nebulisers driven by Heliox 80 (80% helium) vs air-oxygen</td>
<td>% change in FEV$_1$</td>
<td></td>
<td>Adequate</td>
<td>Open</td>
<td>Treatment 25 Control 25</td>
<td>Not stated</td>
<td>3</td>
</tr>
<tr>
<td>Jaber et al. [6] 2000</td>
<td>7 patients with mild-moderate COPD</td>
<td>Non-invasive ventilation with Heliox 78 (78% helium) vs air-oxygen</td>
<td>Effort to breathe</td>
<td>Work of breathing $P_aCO_2$ $P_aO_2$</td>
<td>Unable to ascertain</td>
<td>Open</td>
<td>10 patients included in a cross-over design; one data set was excluded at 9 cm H$_2$O due to incompleteness</td>
<td>Not stated</td>
<td>2</td>
</tr>
<tr>
<td>Esquinas et al. [7] 2001</td>
<td>Patients with COPD with respiratory distress post-extubation</td>
<td>Non-invasive ventilation with Heliox 72 (72% helium) vs air-oxygen</td>
<td>$P_aCO_2$</td>
<td>pH Respiratory rate Mortality</td>
<td>Unable to ascertain</td>
<td>Open</td>
<td>Treatment 25 Control 25</td>
<td>Not stated</td>
<td>2</td>
</tr>
<tr>
<td>Jolliet et al. [16] 2003</td>
<td>Patients in ICU with uncompensated COPD</td>
<td>Non-invasive ventilation with Heliox 78 (78% helium) vs air-oxygen</td>
<td>Intubation rate</td>
<td>Length of stay in ICU and in hospital $P_aCO_2$ Respiratory rate Dyspnoea score</td>
<td>Unable to ascertain</td>
<td>Patients blinded single blinded</td>
<td>Treatment 59 Control 64</td>
<td>Not stated</td>
<td>2</td>
</tr>
</tbody>
</table>

$P_aCO_2$, arterial partial pressure of carbon dioxide; $P_aO_2$, arterial partial pressure of oxygen; FEV$_1$, forced expiratory volume in 1 s.
difference in $P_{a\text{CO}_2}$ between Heliox and air-oxygen. Weighted mean difference was $-0.29\text{ kPa}$ in favour of Heliox (95% CI $-0.64, 0.07$).

Meta-analysis of the effect of Heliox and air-oxygen with NIV on respiratory rate in the same papers gave a weighted mean difference of $1.6\text{ breaths.min}^{-1}$ in favour of control (95% CI $-0.93, 4.14$) indicating no clinically significant effect.

Jolliet et al. [16] also looked at clinical outcomes as a primary end-point and found no significant difference in intubation rates (Heliox vs air-oxygen 13% and 20%, respectively) or length of intensive care stay (Heliox vs air-oxygen, mean of 5.1 days and 6.2 days, respectively). They did, however, report reduced overall costs in the Heliox-treated group due to a significant reduction in the overall length of hospital stay (Heliox vs air-oxygen 13 and 19 days, respectively, $p = 0.002$).

**Heliox use in ventilated patients**

Three studies [8, 17, 18] investigated the effect of Heliox on iPEEP in invasively ventilated (intubated) patients. Meta-analysis gave a weighted mean difference of $-2.23\text{ cmH}_2\text{O}$ in favour of Heliox (95% CI $-4.23, -0.24$). Diehl et al. [17] and Gainnier et al. [18] also looked at the effect of Heliox on work of breathing and found a weighted mean difference of $0.38\text{ J.l}^{-1}$ (95% CI $0.75, -0.01$) in favour of Heliox.

**Heliox as the nebuliser driving gas in acute exacerbations of COPD**

A single paper [19] examined percentage changes from baseline in FEV$_1$. DeBloisblanc et al. [19] found no significant difference in the mean percentage change in FEV$_1$ from baseline between the Heliox and the air-oxygen group (mean (range) respectively 10% (6–15%) and 10% (6–14%)).

**Discussion**

**Limitations of this review**

The main limitation of this review is the heterogeneity of the study designs. All the studies in asthma were true randomised controlled clinical trials. However, amongst the COPD studies there are some randomised controlled studies and some cross-over design trials. We only included cross-over trials with randomised allocation of treatment order as airflow obstruction improves with time during treatment. Without random allocation, time-dependent changes might be interpreted as treatment-dependent changes.

Even so, this approach is not without its problems. Analysing the two treatment periods in cross-over studies...
Figure 2 (a) Forest plot illustrating weighted mean difference in absolute value of PEFR in patients receiving Heliox or air-oxygen driven nebulisers. (b) Forest plot illustrating weighted mean difference in percentage change in PEFR in patients receiving Heliox or air-oxygen driven nebulisers.

Figure 3 (a) Forest plot illustrating weighted mean difference of change in $P_{aCO_2}$ in patients with COPD treated with non-invasive ventilation using Heliox or air-oxygen. (b) Forest plot illustrating weighted mean difference for change in respiratory rate in patients with COPD treated with non-invasive ventilation using Heliox or air-oxygen. (c) Forest plot illustrating weighted mean difference in iPEEP in invasively ventilated patients with COPD receiving Heliox or air-oxygen.
as if they were two arms in a clinical trial is not strictly correct, as the patients in each period are neither independent nor randomly selected. In addition, the cross-over trials receive an undue weight in meta-analyses because they are represented as if they had recruited twice as many patients as they actually studied. As a result there will be a tendency to overestimate any treatment effect they demonstrate [20]. Ideally the patients in a cross-over trial should be compared using paired statistics or the first treatment period only should be included as a ‘randomised controlled trial’. Unfortunately this is only feasible where individual patient data are available. We were unable to obtain individual data and therefore were compelled to analyse the results as we did.

The studies we identified were all small, and most used changes in pulmonary function tests as surrogates for ‘patient-centred’ outcomes. They generally had poor quality scores (Jadad scores), in part because blinding is almost impossible.

All the asthma studies using devices to measure airflow limitation (FEV1, PEFR) specifically calibrated them for use with Heliox as well as air, so there should be no systematic measurement bias. In the asthma and non-ventilated COPD patient studies the maintenance of a closed breathing circuit is vital for Heliox breathing or delivering Heliox driven nebulisers to prevent the patients inhaling room air. In practice this is very difficult to achieve, and dilution of the Heliox with air would produce an apparent beneficial treatment effect in measurement devices calibrated for Heliox. No studies used exhaled gas analysers to ensure gas-tight breathing circuits, so the adequacy of the apparatus has to be assumed.

Clinical significance of outcome data from asthma studies
Heliox used as a driving gas for nebulisers delivering bronchodilators improved the PEFR by an average of 13 L.min\(^{-1}\) in the two studies looking at absolute changes, or by 30% of predicted in the other two studies in patients with acute exacerbations of asthma. These are somewhat disparate results, 30% of predicted PEFR for 40-year-old adult males and females is 190 and 150 L.min\(^{-1}\) respectively, considerably greater than the calculated weighted mean improvement of 13 L.min\(^{-1}\). In both meta-analyses, one study of the pair contributes more than 90% of the weight. In the three studies examining changes in FEV1 after similar treatment, a benefit for Heliox was seen in only one study. Thus the efficacy of Heliox as a driving gas for nebulisers remains in doubt.

Even if Heliox does confer a benefit, it is unclear if the changes in pulmonary function tests seen when Heliox is used as a driving gas for nebulising bronchodilators arise from deeper penetration of aerosol particles into the airways [21] or from a reduction in the resistance to gas flow arising from Heliox breathing. The results from the single identified study of Heliox breathing alone in asthma [14] (without nebulisation using Heliox as a driving gas) suggest that the effect is at least in part due the reduction in gas flow resistance due to the low density of helium. There are limited data to suggest that older asthmatics with more severe exacerbations may derive a greater benefit from Heliox driven nebulisation of bronchodilators [10]. We found virtually no data to determine whether these changes in pulmonary function translate into significant benefit for patients or hospitals. Two studies suggested admission rates from emergency departments were unchanged, but these were small studies and so would have only detected gross changes [9, 10]. Two studies examined the patients’ sensation of breathlessness, finding initially small but significant differences between Heliox and air-oxygen breathing patients [11, 14]. However, the trial which continued to measure these differences for a greater period of time found the difference to reduce to an insignificant level [14].

Clinical significance of the outcome data from the COPD studies
We were unable to demonstrate significant benefit using Heliox in conjunction with NIV in patients with COPD. The mean reductions in \(P_{\text{ACO}_2}\) (0.29 kPa) and respiratory rate (1.6 breaths.min\(^{-1}\)) are both clinically and statistically insignificant. Using Heliox as the driving gas for nebulisation of bronchodilators in the COPD patient group also did not demonstrate benefit.

Intubated, artificially ventilated patients with COPD had a reduced average iPEEP (mean 2.2 cmH\(_2\)O reduction) when ventilated with Heliox as the breathing gas, though this occurred only in patients with high iPEEP. Given that helium would be expected to reduce the gas flow resistance through an endotracheal tube, the reduction in work of breathing using Heliox in intubated patients is not surprising. There are no data on the effects of this reduction on clinical outcomes such as duration of mechanical ventilation, re-intubation rates or survival.

Conclusions
This systematic review did not find data to support the use of Heliox in the routine care of patients with acute exacerbations of asthma, either as a nebuliser driving gas or a breathing gas, or as a breathing gas in patients receiving NIV for exacerbations of COPD. However, the studies identified are all small, with methodological shortcomings, and the inclusion of cross-over studies further reduces the chance of a treatment effect being detected in patients with COPD. Thus the data to
determine the true utility or otherwise of Heliox have not been generated, and there is a need for well designed efficacy studies with clear physiological outcome measures. Effectiveness studies of Heliox in these patients could determine whether efficacy could be demonstrated.

There are data to support the use of Heliox in ventilated patients with COPD to reduce iPEEP in patients with marked air trapping. Whether the reduced air trapping translates to a clinical advantage is unknown.

Treatment with Heliox is both complex and costly. Standard artificial ventilators are not designed for use with Heliox, and so ventilators for both invasive and non-invasive ventilation with Heliox have to be custom-built or modified from standard models. Breathing circuits for Heliox-driven nebulisation have to be either semi-closed or of high flow to prevent air entrainment. In the UK, Heliox is usually supplied in 9.5-l cylinders containing 1200 l of Heliox (21%⁄79%) at ambient pressure. The minimum gas consumption would be a patient’s minute volume for intubated, artificially ventilated patients. This would last about 2 h, at a cost of approximately £26 per hour. Non-invasive ventilators and semi-closed breathing circuits require gas flows in excess of minute volume. We suggest that the data on the efficacy and effectiveness of Heliox are insufficient to justify this investment.

References


