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# A systematic review on the use of sevoflurane in the management of status asthmaticus in adults

Gerald Wai Kit Ho[1*], Thirumeninathan Thaarun[2], Neo Jean Ee[2], Teo Chong Boon[3], Koh Zheng Ning[1], Matthew Edward Cove[4] and Will Ne-Hooi Loh[1,5]

## Abstract

**Background** To conduct a systematic review looking into the use of sevoflurane in the management of status asthmaticus (SA) in adults.

**Methods** We performed a systematic search on PubMed, EMBASE, and The Cochrane Library – CENTRAL through 23rd August 2023, restricting to studies reported in English. We included studies reporting use of sevoflurane in asthmatics beyond its use as an anaesthetic agent in surgeries i.e. in the emergency department (ED) and critical care setting, and focused on patient's clinical parameters, ventilation pressures and weaning of invasive ventilation.

**Results** A total of 13 publications fulfilled the inclusion criteria, comprising of 18 cases. All publications were of case reports/ series and conference abstracts, and no randomised trials were available. Most patients required intubation despite best medical management before sevoflurane administration, and high airway pressures and respiratory acidosis were apparent. There was significant heterogeneity regarding severity of asthma, treatment instituted, and the delivery, duration and concentration of sevoflurane administered. Many of the studies also did not quantify the changes in parameters pre- and post-sevoflurane. Sixteen patients experienced improvements in clinical status with sevoflurane administration—one required escalation to extracorporeal membrane oxygenation (ECMO), and another did not survive.

**Conclusion** The systematic review suggests sevoflurane can be a valuable treatment option in SA. As these cases are rare and heterogenous, further prospective case series are needed to support this.

**Keywords** Asthma, Bronchospasm, Intubation, Respiratory acidosis, Sevoflurane, Status asthmaticus, Volatile agent

## Background

Asthma is a chronic disease that is reported to affect more than 250 million people a year, with exacerbations responsible for nearly half a million deaths [1] in both adults and children. Most patients die as a result of SA, a severe life-threatening form of asthma which is refractory to conventional therapy [2], where impending hypoxaemia and respiratory failure [3] necessitates transfer to the intensive care unit (ICU) and initiation of mechanical ventilation.

*Correspondence:
Gerald Wai Kit Ho
gerald.ho@mohh.com.sg
[1] Department of Anaesthesia, National University Hospital, 5 Lower Kent Ridge Rd, Singapore 119074, Singapore
[2] Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore
[3] Department of Medicine, National University Hospital, Singapore, Singapore
[4] Division of Respiratory and Critical Care Medicine, Department of Medicine, National University Hospital, Singapore, Singapore
[5] Department of Anaesthesia, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore

ICU patients receiving mechanical ventilation also receive sedation with anaesthetic agents such as midazolam or propofol. Recently, there has been interest in using volatile agents for sedation in the ICU, especially for patients with SA. Sevoflurane is one such agent, it is commonly used for the induction and maintenance of anaesthesia [4]. In animal studies [5, 6], it has been demonstrated to produce a bronchodilatory effect and out of all the volatile agents it causes the least airway irritation, making it a popular choice for anaesthesia in asthmatic patients undergoing surgery.

Although commonly used in the operating room, volatile anaesthetic agents are not often used in the ICU due to logistical issues and the need for well-trained staff. Therefore, despite their potential to reverse bronchoconstriction, there is currently no consensus or guidelines for their use in the ICU. To address this, we performed a systematic review of the existing literature to investigate the efficacy of sevoflurane in SA.

## Methods

Our systematic review aimed to evaluate the literature to determine the effects of sevoflurane on patients with SA who had failed initial treatment and had worsening clinical status (hypoxia, hypercarbia, peak airway pressures, lung compliance) despite invasive ventilation.

### Search strategy

We searched for literature through electronic databases (PubMed, EMBASE, and The Cochrane Library—CENTRAL) with restrictions to studies reported in English only. The search was conducted from inception until 23rd August 2023, using the search terms "sevoflurane", "asthma", "severe asthma", "refractory asthma", "life-threatening asthma" and "status asthmaticus". We also searched through grey literature, such as conference papers and abstracts. A manual search of references from included articles was also performed to identify additional studies.

We registered our protocol on PROSPERO (identification record number CRD42023467517) and updated it regularly. We used the PRISMA 2020 Checklist to report our systematic review and have included this checklist separately (see Additional file 1).

### Study selection

Study selection and screening were carried out independently by two individuals (TT and JN), with titles and abstracts of all identified studies reviewed, after which full texts of relevant studies were reviewed. In the event of discrepancies, the two individuals would resolve them through consensus with a third senior reviewer (GH). Cases were only included if the asthmatic patients were adults (≥ 18 years of age) and treated in either the ED or the ICU. We excluded studies where patients presented with asthma exacerbation during the induction/maintenance of anaesthesia in the operating room, as sevoflurane would have been administered before conventional treatment options, and patients may have already been intubated. Studies that initiated treatment with volatile agents other than sevoflurane were also excluded from the review. The PICO criteria is provided in Table 1 for clarity.

### Data extraction

Data was extracted independently (TT) and cross-checked by a second reviewer (JN).

### Quality of studies

Since we mainly expected case reports with inherent bias, we used a modified version of the Newcastle–Ottawa Scale (NOS) [7] to assess the methodological quality of such reports [8]. Two authors (TT and JN) independently carried out the assessment, which included (i) ascertaining adequate exposure, (ii) ascertaining of adequate outcome, (iii) ruling out alternative causes, and (iv) whether the case was sufficiently detailed. Items were rated yes or no, and the risk of bias was reported as low, moderate or high (see Additional File 2). A third senior reviewer (GH) was consulted to resolve any dispute.

### Quantitative data synthesis

There was anticipation that the quality of the reports and the nature of data representation would make qualitative data synthesis impractical. Data was analysed and presented using descriptive statistics – medians for non-continuous variables, and percentages for dichotomous variables.

**Table 1** PICO criteria

| Participants | Adult patients (age ≥ 18 years) with status asthmaticus refractory to conventional therapy |
|---|---|
| Intervention | Sevoflurane |
| Comparison | Placebo or other pharmacological strategies |
| Outcome | Reduction in wheeze; improvement in oxygenation and respiratory acidosis; reduction in peak airway pressures; increase in lung compliance; weaning off invasive ventilation |

## Results

### Publication characteristics

From our search, 29 articles were retrieved from PubMed, 21 from EMBASE and one from Cochrane CENTRAL Library (Fig. 1). An additional five articles were found through citation searching and four from grey literature. We screened through 59 potentially relevant articles based on our PICO criteria after removing one duplicate and subsequently assessed their full text for eligibility. In total, 13 articles were excluded, leaving 13 uncontrolled studies accounting for 18 distinct clinical cases [9–21].

### Clinical parameters and treatment prior to sevoflurane use

Most patients presented with acute respiratory distress, clinically characterised by tachycardia (heart rate > 100), tachypnoea (respiratory rate > 20), dyspnoea, hypertension, bilateral diffuse wheeze with poor air entry on auscultation and low oxygen saturation as measured by pulse oximetry ($SpO_2$ < 92%). Most patients were intubated and initiated on mechanical ventilation with significant respiratory acidosis and high airway pressures before the initiation of sevoflurane. Standard asthma therapy was given from the start to all patients, consisting of bronchodilator therapy (nebulized salbutamol, albuterol and/or ipratropium bromide), high dose systemic corticosteroids and IV magnesium sulfate. Salvage options used before initiation of sevoflurane included ketamine, epinephrine, neuromuscular blocker infusions and Heliox.

### Delivery of sevoflurane

Patients received sevoflurane therapy either via an anaesthesia workstation (11/16 reported, 68.8%) [9–21] or via an Anaesthetic Conserving Device (ACD) (5/16 reported, 31.2%) [12, 17, 18]. Two reports did not describe the mode of delivery of the volatile agent. The concentration of sevoflurane delivered, where reported, ranged from 0.25% to 8% (median 2.6%) [10, 11, 13–15, 17, 19, 21] (Table 2). Two reports mentioned a gradual reduction in sevoflurane delivery as the patient's condition improved [13]. One patient had sevoflurane delivery of 0.25% via a face mask [21]. Patients who were on an ACD were described to have a sevoflurane flow rate ranging from 3 to 10 mL/hr [12, 17] (median 7.5 ml/hr) with a target Minimum Alveolar Concentration (MAC) of 0.5. Ruszkai et al. reported an end-tidal sevoflurane concentration of 0.5–0.8% while achieving a MAC of 0.4–0.6 on the ACD

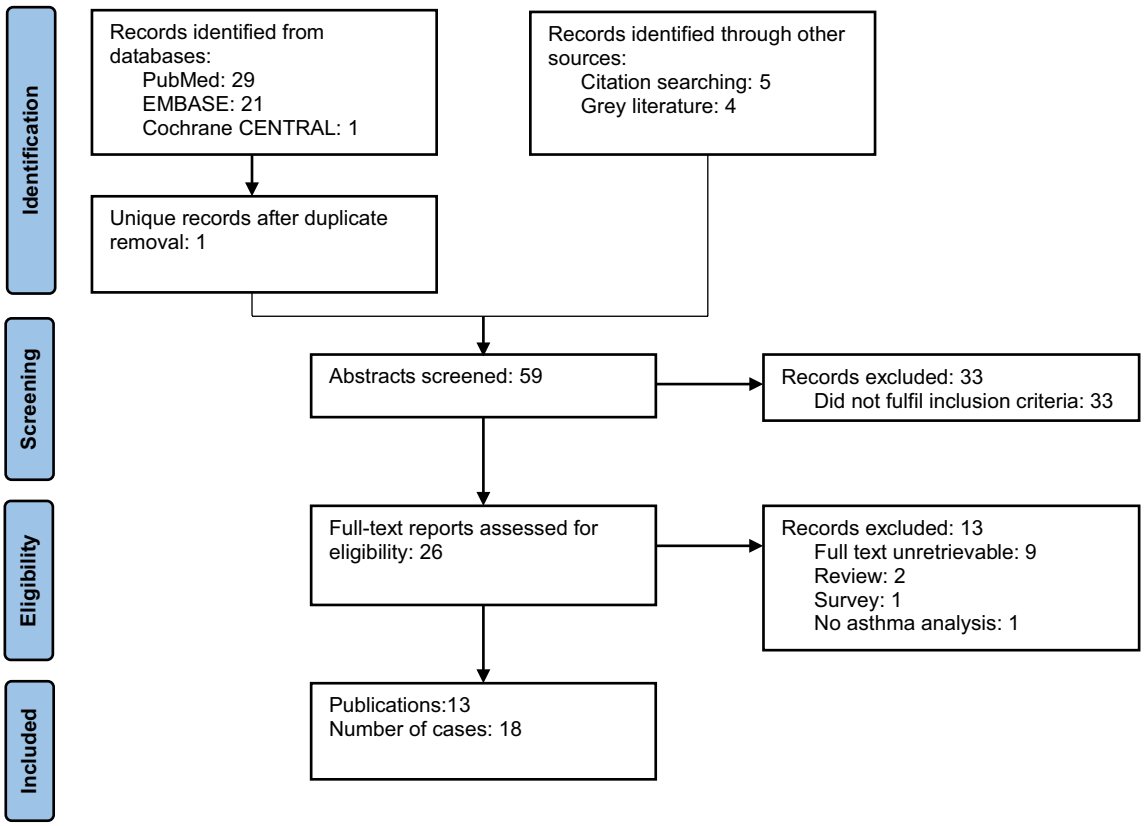


**Fig. 1** PRISMA 2020 flow diagram

**Table 2** Summary of studies and their results

| Author | Patient demographics | Sevoflurane administered | Pre-sevoflurane clinical parameters | Post-sevoflurane clinical parameters |
|---|---|---|---|---|
| Carmel et al. [9] | 45yo male | % not reported; several hours | $PaCO_2$: > 100 mmHg<br>pH: 7.1<br>Ppeak: 136 $cmH_2O$<br>Pplat: < 41 $cmH_2O$ | $PaCO_2$: < 70 mmHg<br>pH: > 7.3 |
| Munusamy et al. [10] | 38yo male | Fi 0.5%-2%; 48 h | $PaCO_2$: 83 mmHg<br>pH: 7.13<br>Ppeak: 48 $cmH_2O$<br>Pplat: 22 $cmH_2O$<br>autoPEEP: 4 $cmH_2O$ | $PaCO_2$: 38 mmHg<br>pH: 7.44<br>Ppeak: 30 $cmH_2O$<br>Pplat: 23 $cmH_2O$<br>autoPEEP: 0 $cmH_2O$ |
| Maqsood et al. [11] | 29yo female | Fi 8%; duration not reported | $PaCO_2$: 172 mmHg<br>pH: 6.8<br>Ppeak: 55 $cmH_2O$<br>autoPEEP: 14 $cmH_2O$ | $PaCO_2$: minimal change<br>pH: minimal change |
| Adi et al. [12] | 25yo male | 5-10 ml/hr for MAC 0.5; 24 h | $PaCO_2$: 96 mmHg<br>pH: 6.96<br>Ppeak: 60–70 $cmH_2O$<br>Pplat: 30–34 $cmH_2O$<br>autoPEEP: 12 $cmH_2O$ | $PaCO_2$: 64 mmHg<br>pH: 7.23<br>Ppeak: 25–30 $cmH_2O$<br>Pplat: 20–25 $cmH_2O$<br>autoPEEP: < 5 $cmH_2O$ |
| Adi et al. [12] | 34yo male | 5-10 ml/hr for MAC 0.5; 6 h | $PaCO_2$: 67 mmHg<br>pH: 7.21<br>Ppeak: 60 $cmH_2O$<br>Pplat: 30 $cmH_2O$<br>autoPEEP: 7 $cmH_2O$ | $PaCO_2$: 50 mmHg<br>pH: 7.3<br>Ppeak: 25–35 $cmH_2O$<br>Pplat: 20–25 $cmH_2O$<br>autoPEEP: 7 $cmH_2O$ |
| Adi et al. [12] | 37yo male | 8 ml/hr for MAC 0.5; 8 h | $PaCO_2$: 94 mmHg<br>pH: 7.09<br>Ppeak: 60 $cmH_2O$<br>Pplat: 32 $cmH_2O$<br>autoPEEP: 12 $cmH_2O$ | $PaCO_2$: 68 mmHg<br>pH: 7.2 |
| Suzuki et al. [13] | 29yo male | Fi 2–4%, gradually reduced on day 4; 104 h (152.5 MAC-h) | $PaCO_2$: 69.7 mmHg<br>pH: 7.24<br>Ppeak: 30 $cmH_2O$ | $PaCO_2$: 59.3 mmHg @ 11 h<br>49.4 mmHg @ 24 h<br>41.3 mmHg @ 4 days<br>pH: 7.47 |
| Suzuki et al. [13] | 55yo male | Fi 1–3%, gradual reduction; 30 h | Ppeak: 16–44 $cmH_2O$ | Ppeak: 16–20 $cmH_2O$ |
| Keenan et al. [14] | 55yo female | Fi 1.5%; 72 h | $PaCO_2$: 90 mmHg<br>pH: 7.22<br>Ppeak: > 77 $cmH_2O$<br>Pplat: 17 $cmH_2O$<br>autoPEEP: 16 $cmH_2O$ | $PaCO_2$: 52 mmHg<br>pH: 7.44<br>Ppeak: 41 $cmH_2O$ |
| Najout et al. [15] | 42yo male | Fi 3%; 1 h twice daily | $PaCO_2$: 63 mmHg<br>pH: 7.09<br>Ppeak: 45 $cmH_2O$ | Ppeak: 28 $cmH_2O$ |
| Ng D et al. [16] | Early 20 s, male | % not reported; 2.5 h | $PaCO_2$: 120 mmHg<br>pH: 7<br>Ppeak: 50 cmH2O | 35 min<br>$PaCO_2$: 105 mmHg<br>pH: 7.1<br>Ppeak: 35 $cmH_2O$<br>2.5 h<br>$PaCO_2$: 53 mmHg<br>pH: 7.26<br>Ppeak: 29 $cmH_2O$ |
| Ruszkai et al. [17] | 67yo male | ETsevo 0.5–0.8% (syringe pump 3–7 ml/hr), MAC 0.4–0.6; 28 h | $PaCO_2$: 71 mmHg<br>pH: 7.23<br>Ppeak: 60 $cmH_2O$<br>autoPEEP: 11 $cmH_2O$ | 30 min<br>Ppeak: 36 $cmH_2O$<br>autoPEEP: 3.2 cm $H_2O$<br>28 h<br>Ppeak: 15 cm $H_2O$<br>autoPEEP: 3 cm $H_2O$ |
| Sorour et al. [18] | 32yo male | % not reported; 2 h | $PaCO_2$: 112 mmHg<br>pH: 6.9 | - |
| Littlefield et al. [19] | 45yo female | Fi 2.2–4.0%; 16.5 h | pH: Acidosis<br>Ppeak: Elevated<br>Pplat: Elevated<br>autoPEEP: Elevated | $PaCO_2$: Improved<br>pH: Improved |

**Table 2** (continued)

| Author | Patient demographics | Sevoflurane administered | Pre-sevoflurane clinical parameters | Post-sevoflurane clinical parameters |
|---|---|---|---|---|
| Littlefield et al. [19] | 19yo male | Fi 2.2%; 22.5 h | Ppeak: Elevated<br>Pplat: Elevated<br>autoPEEP: Elevated | $PaCO_2$: Improved<br>pH: Improved |
| Littlefield et al. [19] | 19yo male | % not reported; 16 h | Ppeak: Elevated Pplat: Elevated<br>autoPEEP: Elevated | $PaCO_2$: Improved<br>pH: Improved |
| Sinniah et al. [20] | 41yo female | % and duration not reported | $PaCO_2$: 141.7 mmHg<br>pH: 6.85 Ppeak: 80 $cmH_2O$<br>autoPEEP: Present | - |
| Baigel et al. [21] | NA | Fi 0.25%; 35 min of self mask holding | $PaCO_2$: 22.8 mmHg | $PaCO_2$: 37.2 mmHg |
| ***Median*** | **-** | ***Fi 2.2%* via *workstation or 7.5 ml/hr* via *ACD*** | ***$PaCO_2$: 92 mmHg***<br>pH: 7.09<br>Ppeak: 60 $cmH_2O$<br>Pplat: 31 $cmH_2O$<br>autoPEEP: 12 $cmH_2O$ | ***$PaCO_2$: 56.2 mmHg***<br>pH: 7.3<br>Ppeak: 30 $cmH_2O$<br>Pplat: 25 $cmH_2O$<br>autoPEEP: 3.6 $cmH_2O$ |

[17]. Sevoflurane concentration/ delivery rate was not described in five of the reports [9, 16, 18–20].

The duration of sevoflurane treatment was reported in 16 patients, ranging from 35 min to 104 h (median of 22.5 h) [9–21]. One patient was described as having received intermittent sevoflurane treatment of 1 h twice a day for two days [15].

### Outcome of sevoflurane

After sevoflurane was instituted, 16 out of 18 patients (88.9%) reported improvement. Four patients were described to have resolution of wheeze [14, 15, 18, 21], and another report mentioned improved air entry in the patient [12].

Acid base status and airway pressures largely showed improvement (see Table 1), as early as 30 min post sevoflurane in some cases [16, 17]. One author mentioned improved lung compliance at 30 min and 28 h post sevoflurane (17.2 > 47.7 > 116 mL/$cmH_2O$) [17]. Another author described a "drastic improvement" in both respiratory acidosis and airway pressures without providing actual values [19]. The median time until extubation following administration of sevoflurane was 3.7 days [9, 10, 13–17].

Only three reports mentioned any secondary complications. One patient had hypotension during sevoflurane administration, requiring noradrenaline up to 0.2mcg/kg/min [12]. Another two patients required a tracheostomy due to critical illness myopathy [14] and delirium [19].

There were two reports for which sevoflurane was ineffective in the treatment of SA. Maqsood et al. described of a young patient who had minimal improvement in her asthma despite giving sevoflurane of 8%, of which the duration was not specified [11]. She had persistent high intrinsic peak end expiratory pressure (PEEP) and respiratory acidosis. The patient eventually required transfer to another hospital for initiation of ECMO for 48 h. She was extubated the following day. Sinniah et al. described of a multiparous patient in her second trimester of pregnancy (13 weeks gestation) who presented with SA refractory to conventional treatment and had subsequently failed a trial of sevoflurane and ECMO [20]. Her demise was ultimately due to the development of multiple complications (ischemia of the lower limb, status epilepticus, vaginal bleeding) that conferred a poor prognosis for the mother and child.

### Quality appraisal

The overall quality of the cases was fair, 50% of the cases were rated as having low risk of bias, 16.7% having moderate risk, and 33.3% having high risk. Most cases were ascertained to have had an adequate exposure to sevoflurane (83.3%). 72.2% were deemed to have a reliable outcome measure, while two thirds (66.7%) adequately ruled out alternative causes of SA responding to sevoflurane. Just over half of the cases were sufficiently detailed (55.6%).

## Discussion

Our systematic review aimed to consolidate the clinical evidence in the current literature regarding the administration of sevoflurane in adult patients with SA. In our literature review we only found reports that were case studies or case series, there were no clinical trials. We identified 18 patients treated with sevoflurane, and all but 2 of these were reported to improve after initiation of sevoflurane. Only 1 case of hypotension during

sevoflurane administration requiring vasopressor support was reported. Although further research is required, our results suggest that sevoflurane is a potentially effective management strategy that can be instituted for adults with SA.

Sevoflurane is a fluorinated methyl isopropyl ether popular for anaesthesia in the surgery setting because of its low blood:gas partition coefficient, allowing rapid induction and emergence from anaesthesia once turned off [4]. When used in the ICU setting as a sedative, quality of sedation has been shown to be comparable to propofol, with a reduction in time to spontaneous breathing after termination of sedation [38]. Wake-up time and extubation delay have also been shown to be significantly shorter when sevoflurane was used for sedation compared to propofol or midazolam [22].

Apart from its sedative effects, sevoflurane has been shown in animal studies to cause relaxation of airway smooth muscle [5] and mitigation of allergic airway inflammation [23]. The inhibitory action of sevoflurane on porcine tracheal smooth muscle contraction occurs by blocking T-type voltage-dependent $Ca^{2+}$ channels, thereby decreasing the concentration of intracellular free $Ca^{2+}$ at clinically significant levels [24, 25]. Sevoflurane also significantly inhibits $Cl^{-}$ currents through Cl-Ca channels, and has variable inhibition on $K^{+}$ channel subtypes, within porcine bronchial and tracheal smooth muscles [25, 26]. This rapid bronchodilatory effect has been elucidated in a study by Rooke et al. [27], which showed a significant drop in respiratory system resistance after 5 min of treatment in healthy human volunteers. As the action of sevoflurane on tracheal smooth muscles require diffusion of the inhalational agent from the bronchial and tracheal lumen through the airway wall to reach the smooth muscle, the speed of onset is dependent on the concentration gradient of sevoflurane [28]. Other known mechanisms by which sevoflurane causes bronchodilation include inhibition of postganglionic cholinergic neuroeffector transmission in airway smooth muscle [29]. It is also hypothesised that sevoflurane exhibits its bronchodilatory effects through systemic absorption via the bronchial/ pulmonary vessels to reach poorly ventilated regions [30, 31], as well as neurally-mediated actions such as a reduction in vagal tone and reflexes [32–34]. It is thus recommended as a maintenance agent for asthmatic patients undergoing anaesthesia [38]. These properties also make it a useful drug for the critically ill patient with unresponsive reactive airway disease. Patients with SA have a failure of medical therapy and often require high ventilatory settings. Complications of invasive positive pressure ventilation in patients with SA include air trapping, barotrauma, and hypotension [35]. All 8 studies in our systematic review that reported post-sevoflurane airway pressures showed decreased airway pressure. The median autoPEEP also decreased from $12cmH_2O$ to $3.6cmH_2O$, suggesting sevoflurane provides a dual benefit of sedation and bronchodilation in adult SA patients after failed salvage options.

The use of sevoflurane for SA has also been studied in the paediatric population. A multicentre retrospective case series conducted in the Netherlands in 2013 showcased the efficacy of sevoflurane for life-threatening asthma in children [36]. Seven children (aged between 4 to 13 years) were included in the study. Sevoflurane use was shown to reduce $PCO_2$ (median of $14 > 9.8 > 6.2$ kPa) ($p = 0.05$), improve pH (median of $7.02 > 7.18 > 7.43$) ($p = 0.01$), and reduce peak pressures (median of $30 > 20.4$) ($p = 0.03$), when comparing levels at the start, after 2 h of sevoflurane and at the end of treatment. Peak concentration ranged from 1–8% and duration ranged from 0.5 – 90 h (median of 24 h). One patient did not improve with sevoflurane and was deemed to have ARDS related to pneumonia. The study concluded that children with life-threatening asthma could be treated with sevoflurane without serious side effects.

At present, there is limited evidence for long term use of sevoflurane for SA in the adult ICU setting. Our systematic review highlighted one case of hypotension requiring vasopressor support during sevoflurane administration [11]. Sevoflurane produces dose-dependent effects on the circulatory system most notably a drop in systemic blood pressure and cardiac output [4]. This effect may be more pronounced in more critically ill patients [37] but can be mitigated with vasopressor support. A randomised controlled trial ($n = 79$) conducted by Soukup and colleagues comparing sevoflurane sedation to propofol in the ICU (> 48 h) showed no significant difference in hemodynamics ($p < 0.05$) or adverse events [38]. Other concerns include the buildup of inorganic fluoride metabolite as well as the production of Compound A with low gas flow, causing renal insufficiency, especially in those with chronic renal disease, although they pose an unlikely risk of renal injury in humans [39, 40].

A series of case reports have demonstrated a possible association between prolonged sevoflurane use and reversible nephrogenic diabetes insipidus (NDI) in ICU patients [41–43]. This temporary reduction in renal concentrating ability is hypothesized to be due to a decrease in aquaporin-2 [44], and is not considered to be due to fluoride ions. An alternative agent would be isoflurane, which has been shown to have a lower incidence of NDI [45], and is well documented in the treatment of SA [46, 47]. Lastly, there is a risk patients may develop malignant hyperthermia and it is imperative for staff to have knowledge of handling this crisis scenario when dealing with inhalational agents.

Despite its availability in most hospitals, there exists a set of challenges that one must consider before utilising sevoflurane. Modern anaesthesia workstations come equipped with vapourisers, but are bulky and can be difficult to manoeuvre around, especially in space-constrained settings like the ED or ICU [48, 49]. Scavenging systems may not be available outside of the operating theatre (OT), where reduced air changes per hour increases the exposure of healthcare workers to the leaked gas [50, 51]. Volatile capture systems like CONTRAfluran™ and FlurAbsorb, which are compatible with ICU ventilators, should be used to limit this occupational exposure [52]. Monitoring of volatile agent delivery to the patient (both inspired and end-tidal concentrations) via commercially available anaesthesia gas analysers is also important to ensure a steady state of gas concentration to the patient, avoiding unnecessary prolonged exposure to high anaesthetic levels with its unwanted side effects, as well as wastage of the agent [4].

Conversely, occupying an operating room for the sole purpose of sevoflurane administration in patients with SA would compromise on space and manpower that could otherwise be utilised for elective surgeries [53]. Transport of a critically ill patient to the OT presents its own set of challenges, where anaesthesia workstations may not be equipped with complex ventilation modes that patients with lung pathologies might require [54–56].

An ACD circumvents some of these issues. The device is compatible with ICU ventilators being connected to the ventilator circuit and patient's endotracheal tube, preventing the need for an anaesthesia workstation [54]. It allows the introduction of volatile agents and acts as a heat and moisture filter. This prevents ambient volatile gas levels from exceeding safe workplace exposure values even in the absence of scavenging [57, 58]. The use of an ACD alone has been shown to worsen hypercapnia and increase work of breathing [59, 60] through increasing apparent dead space [59]–these effects are attenuated but not abolished with the use of sevflurane [59, 61], hence limiting its use in patients who require low tidal volume ventilatory strategies [61]. Another option is to connect the ACD on the inspiratory limb which eliminates this dead space and additional work of breathing [62].

At present, there is limited evidence for long term use of sevoflurane for SA in the adult ICU setting as shown by the lack of large randomised trials. This may be due to sevoflurane being used as a salvage strategy after a patient with SA has failed other medical therapy. Our findings were limited by the low quality and breadth of data available, which was heterogenous, and prevented conduct of a meta-analysis. These patients are rare and heterogenous, therefore better prospective case series may be a viable model to accurately characterise the effects of sevoflurane in this group.

## Conclusion

In conclusion, our systematic review suggests that the use of sevoflurane has the potential to treat patient with SA through an improvement in respiratory acidosis and airway resistance. Its bronchodilatory and anti-inflammatory properties, combined with its easily titratable and its short-term effects, make it a potentially useful secondary therapy. Further research is required to establish its use in this subgroup of patients.

### Abbreviations

| | |
|---|---|
| ACD | Anaesthetic Conserving Device |
| ECMO | Extracorporeal membrane oxygenation |
| ED | Emergency department |
| ICU | Intensive care unit |
| MAC | Minimum Alveolar Concentration |
| OT | Operating theatre |
| PEEP | Peak end expiratory pressure |
| SA | Status asthmaticus |

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13054-024-05122-8.

**Additional file 1**: PRISMA 2020 Checklist.

**Additional file 2**: Quality appraisal for reported cases.

### Acknowledgements

Not applicable.

### Author contributions

GH made contributions to the conception and design of the review, and was a major contributor in writing the manuscript. TT and JN acquired, analysed and interpreted the data, and played a role in writing the results of the manuscript. CB contributed to the research design and data analysis. ZN, MC, and WL made substantial revisions to the manuscript leading to the final report. All authors have read and approved the final manuscript.

### Funding

No sources of funding in the conduct of the study.

### Availability of data and materials

No datasets were generated or analysed during the current study.

## Declarations

### Ethics approval and consent to participate

Not applicable.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

Received: 11 July 2024 Accepted: 5 October 2024
Published online: 14 October 2024

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