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Halothane treatment in life-threatening asthma

P. Rosseel, L. F. Lauwers and L. Baute

Department of Anesthesia and Intensive Care, A. Z. Stuivenberg, Antwerp, Belgium

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Abstract. We analysed the case histories of 22 patients with life threatening asthma retrospectively. Fifteen patients needed mechanical ventilation. Three patients, all of them in cardiac arrest before or at the moment of admission, died. In five patients, halothane therapy was successfully used as a last resort. No major complications occurred. Two case-reports are presented.

Key words: Life threatening asthma – Halothane

Life threatening asthma (LTA) should be defined as a severe asthmatic attack unresponsive to conservative therapy with methylxanthines, sympathomimetics, corticosteroids and intensive respiratory care, while rapid progression to death is expected.

Although the side-effects and complications of artificial ventilation in this condition are prominent, it will be needed frequently [1, 8, 10, 15, 24, 28].

General [17, 22, 23, 28] and epidural anesthesia [32] and bronchial lavage [1, 19, 37] have been advocated in LTA. Halothane anesthesia has sporadically been reported to be effective although all reports stress the risks and dangers [7, 15, 17, 30, 31].

Table 1. Criteria for entering in the study group

1. Positive history of asthma or asthmatiform bronchitis
2. Asthma crisis or its complications as the indication for admission to the intensive care unit
3. Respiratory failure: $PaO_2 < 70$ mmHg (without O_2-therapy); $PaCO_2 > 50$ mmHg
4. Acidosis: pH < 7.15
5. Tachyocardia > 130 beats/min or bradycardia < 60 beats/min
6. Clinical impression of exhaustion
7. No response to standard therapy (Table 2) within 1 hour

Patients and methods

All patients admitted to our ICU with the diagnosis of LTA in a 4-year period (1980 – 1983) were analysed retrospectively. The criteria for entering the study are summarized in Table 1. All patients received standard therapy as outlined in Table 2. IPPV was started whenever the clinical condition was precarious and deteriorating. Abnormal blood-gas values were not the only criteria to start IPPV. A flow generated ventilator was used (Servo Siemens 900, UV-1 Dräger or Veriflo 2000). Ventilator settings were chosen to provide an optimal match between blood-gas values, airway pressures and hemodynamic function. The FiO_2 was selected to assure an arterial oxygen saturation of 90%.

Inotropic support with dopamine 2 – 10 μg/kg/min was provided whenever systolic bloodpressure was lower than 100 mm Hg or urinary output was inadequate in normovolemic patients. Indications for halothane treatment are summarized in Table 3. Halothane treatment was only started after obtaining a general consensus of the staff of our ICU.

Table 2. Summary of the standard therapy given to all patients with life-threatening asthma

1. Nursing in a semi-sitting position
2. Oxygen by face mask (humidified and warmed)
3. Methylprednisolone 30 mg/kg IV, followed by 6 × 40 mg IV daily
4. Aminophylline therapy assuring constant blood-levels (10 – 20 ng/ml)
5. Sympathomimetics when there was no evidence of existing overdosage
6. Digitalisation, when tachycardia persisted and when no contra-indications existed
7. Sodium bicarbonate, to correct pH values below 7.2 – 7.35

Table 3. Criteria for starting halothane treatment

Persistance of the following conditions 4 to 6 h after full standard intensive therapy:
1. $PaO_2 < 60$ mmHg and/or $PCO_2 > 50$ mmHg under IPPV with FiO_2 of at least 0.6 and a tidal volume of 10–12 ml/kg
2. Persisting high inspiratory peak pressures > 50 cm H_2O
3. Persisting circulatory instability, hypotension, oliguria, tachycardia or bradycardia

Results

Twenty-two patients entered the study. Fifteen needed IPPV and in five halothane was administered. Three patients died, all of them having developed cardiopulmonary arrest before or during their admission to the ICU.

Standard therapy group (Table 4)

In this group two patients, both of them initially successfully resuscitated from asystole at admission died of cerebral complications. One patient was successfully resuscitated from ventricular fibrillation and was discharged without sequelae. Mechanical ventilation was instituted in 10 patients and only in three patients did the duration exceed 24 h, due to concomitant bronchopneumonia.

Halothane-treated group (Table 5)

All patients treated with halothane showed marked improvement of respiratory pressures and resistance within 30 to 60 min of starting treatment. In all patients, except one (case report 2), halothane could be stopped within 24 h and there was no need for inspiratory concentrations in excess of 0.75%.

In one patient, multiple ventricular beats developed but were easily controlled with a lignocaine infusion (2 mg/min). Positive inotropic support was necessary in three patients. Two patients were already receiving dopamine before the halothane therapy. There was a moderate increase in hepatocellular enzymes without jaundice in three patients. The levels returned to normal within 30 days. One patient in the halothane group died 36 h after stopping halothane

Table 4. Schematic overview of 17 patients with life threatening asthma and treated with standard intensive therapy

Patient	Sex	Age (years)	pH	PCO_2 (mmHg)	PO_2 (mmHg)	CPR, before admission	CPR after admission	Impaired consciousness	IPPV (hours)	Outcome (leaving hospital)
BP	M	70	7.1	77	64	–	–	–	24	Alive
RL	F	45	7.3[a]	49[a]	68[a]	–	–	+	2	Alive
VBH	F	46	7.15	58	60	–	–	–	–	Alive
VDBB	F	74	7.1	60	62	–	–	–	–	Alive
VOJ	F	22	7.0	85	50	–	–	+	8	Alive
VDBI	F	36	7.08[a]	58[a]	75[a]	–	&	+	4	Alive
DA	M	48	7.12	51	50	–	–	–	–	Alive
SM	F	20	7.1	54	43	–	–	–	–	Alive
MM	F	29	7.15	54	85	–	–	–	–	Alive
SM	F	37	7.16	67	46	–	–	+	4	Alive
MI	F	39	7.1[a]	49[a]	80[a]	§	§	+	8	Dead
DMC	F	53	7.2	52	59	–	–	+	–	Alive
DG	M	63	7.2	57	59	–	–	–	–	Alive
SA	M	69	7.16[a]	89[a]	151[a]	–	–	+	60	Alive
VA	M	68	7.15	115	59	–	–	+	6	Alive
GL	F	34	7.35	51	51	§	§	+	11d	Dead
DV	F	62	6.95	104	68	–	–	+	12d	Alive

[a] Measured after installation of IPPV because of the critical condition, & = ventricular fibrillation, § = asystoly)

Table 5. Schematic overview of five patients with life threatening asthma and treated with halothane

Patient	Sex	Age (years)	pH	PCO_2 (mmHg)	PO_2 (mmHg)	CPR, before admission	CPR after admission	Impaired consciousness	IPPV (hours)	Halothane %	Admin. (hours)	Outcome (hosp.)
HJL	M	76	7.03	102	60	–	–	+	12	0.4	6	Alive
CD	F	50	6.9	55	64	–	–	+	48	0.3	20	Alive
PE	M	71	7.1[a]	57[a]	98[a]	&	&	+	54	0.2–0.5	12	Dead
KP	M	18	7.1[a]	85[a]	223[a]	–	–	+	24	0.5	20	Alive
DH	F	27	7.1	61	48	–	–	+	120	0.5–1.5	36	Alive

[a] Measured after installation of IPPV because of the critical condition (& = ventricular fibrillation, § = asystoly)

treatment. At that time, the patient was in good respiratory condition but developed refractory ventricular fibrillation, not related to halothane.

Case report 1

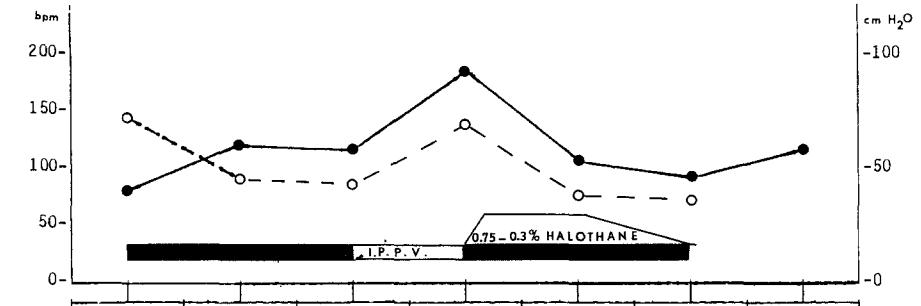
An 18-year-old male was admitted after a road accident. Examination revealed a comatose patient (3/15 on the Glasgow Coma Scale) with slowly light-reactive, dilated pupils. There were no signs of lateralisation. The patient was in severe respiratory distress with central cyanosis, and paradoxical respiratory movements at a rate of 8/min. On auscultation, weak and wheezing breath sounds were detected over the left lung while the right lung was silent. The pulse was weak at a rate of 80 beats/min. Blood pressure was 80/50 mm Hg. The jugular veins were markedly distended. Several bruises and multiple wounds covered face, left arm and leg. Endotracheal intubation was performed. Artificial ventilation with a flow generator (pressure limited at 70 cm H_2O) at an $FiO_2 = 1.0$ and standard therapy was started. High peak pressures resulted in almost ineffective ventilation.

The chest X-ray showed marked hyperinflation of both lungs. At this moment, the diagnosis of life-threatening asthma, rather than chest injury, was likely. Subsequent history revealed a history of asthma but no respiratory distress existed at the moment of the accident. Instillation of 0.25 mg epinephrine endotracheally resulted in decreasing peak pressures to 40 – 50 cm H_2O and an increased expired minute volume. Six hours after admission continuous pleural drainage was instituted for a right pneumothorax with accompanying subcutaneous emphysema. Maintenance therapy consisted of methylprednisolone 250 mg/24 h I.V., aminophylline 60 mg/h I.V., epinephrine 4 × 0.25 mg SC, tiazinamium 4 × 50 mg/24 h I.M. The patient was sedated with fentanyl and promethazine. Artificial ventilation was continued with a Servo-ventilator (Tidal volume 600 ml, frequency 20/min, I/E ratio 1/1, $FiO_2 = 0.4$). Fifteen hours after admission respiratory status deteriorated (Fig. 1) with increasing inflation pressures and hypercapnia. Oxygenation had to be maintained by increasing FiO_2 to 1.0. Arterial hypotension with oliguria and tachycardia of 180 bpm could be controlled with a dopamine infusion at 5 μg/kg/min. There was no benefit in changing the ventilator settings. Halothane was then delivered from a Fluotec Mark II Vaporiser, mounted on the fresh gas line of a Spiromat Dräger. An inspiratory concentration of 0.75% was gradually reached over 5 min. Epinephrine therapy was stopped 3 h before halothane administration while dopamine and aminophylline infusions were continued. Thirty min after starting halothane administration a substantial improvement of all parameters was noted (Fig. 1).

The halothane administration was continued for 20 h decreasing the inspiratory concentrations gradually to 0.3% within the first 2 h. The patient was easily weaned and was extubated 24 h after starting halothane. The patient was awake and fully responsive to commands. The hepatocellular enzymes increased in the days after halothane treatment. No clinical symptoms accompanied this chemical 'hepatitis' which normalized in 20 days. He was transferred to the ward after 6 days and discharged from hospital on day 24 in good health.

Case report 2

A 27-year-old pregnant female (gestational age 105 days) was admitted with life-threatening asthma. She



	admission	3 h	5 h	15 h	15 1/2 h	35 h	40 h
pH	7.1	7.22	7.29	7.20	7.34	7.45	7.49
PCO2 (mmHg)	85	65	56.4	71	57	39	32
PO2 (mmHg)	223	67	120	58	66	95	75
FiO2	1.0	0.5	0.35	1.0	0.5	0.25	0.25

Fig. 1. Evolution of blood-gas values, heart rate, arterial pressure and peak inspiration pressure in a patient with life threatening asthma, treated with halothane 0.3 – 0.75% ●——● heart rate; ○ – – ○ peak pressures

had had a respiratory infection for a few days. She was treated by the family physician with antibiotics, methylxanthines orally, and nebulized β-agonists. On admission she was comatose, cyanotic, with a pulse rate of 170 beat/min. Artificial ventilation was immediately instituted resulting in peak pressures of 75 cm H_2O. As her precarious condition did not improve with full standard therapy, halothane in a concentration of 0.5% was administered 4 h after admission. Blood-gas values and inspiratory pressures normalized within 30 min. However, when halothane was stopped, after 5 h of treatment, LTA recurred. Intermittent halothane administration at concentrations from 0.2 to 1.2% was necessary for 3 days for recurring LTA-attacks. IPPV had to be continued for 5 days because of delayed awakening and marked muscle weakness.

The bronchopneumonia was successfully treated and the patient was discharged after full recovery on the 20th day. She delivered a healthy boy at term.

Discussion

The cases are an illustration of LTA not responding to conventional intensive therapy and rapidly deteriorating with artificial ventilation. Various reports recommend different methods for handling this situation.

It is very difficult to obtain representative figures about incidence and mortality of life-threatening asthma in an asthma population. In the report of Santiago et al., two fatalities out of 86 patients in status asthmaticus over a 9-year period were described [34]. Williams [39] had 80 patients with 111 admissions for LTA in a 10-year-period, of a total of 851 admissions for asthma. Fifteen patients died. He estimated that 1 in 600 asthmatics would experience a life threatening asthma attack in the course of 1 year. He pointed out that there was a greater chance for recurrence after LTA in comparison with other asthma patients. Scoggin reports a mortality range from 0 to 38% in asthma patients requiring MV [35].

When the disease cannot be controlled with conventional therapy, mechanical ventilation (MV) should be considered [12, 17, 18, 28].

Clinical features such as unconsciousness, exhaustion, hemodynamic impairment and characteristics of asthmatic breathing are more important in deciding upon MV than some arbitrary blood-gas values. The heart of the matter is to provide an optimum matching of oxygenation, ventilator settings and hemodynamic state. Endotracheal intubation and MV can trigger or worsen bronchospasm through reflex vagal activity [1, 2] and sedation, local and general anesthesia are important issues in controlling this problem [2, 7, 15, 17, 22, 23]. In our opinion the use of muscle relaxants should be restricted, since many of them may provoke histamine release. An increased incidence of barotrauma is the consequence of high inflation pressures [28]. Efforts should be made to treat bronchospasm and mucous plugging [1, 2, 16, 19].

Failure to recognise LTA or lack of aggressive treatment will almost inevitably lead to death [10]. Several therapeutic measures have been proposed in clinical and experimental work with limited success. Bronchial lavage and bronchoscopic removal of plugs is indicated for atelectasis [1, 19, 36]. However, it should not be used routinely since it can worsen bronchospasm. Also bronchial lavage with saline can wash out surface active material and cause ARDS in experimental models.

Epidural anesthesia has been reported for the relief of bronchospasm [5, 33] but the mechanism of its action is not understood. Changes in lung-compliance and surface active material, afferent sensory blockade and vascular absorption of local anesthetics could be involved [5]. On the contrary, inadvertent high spinal anesthesia was reported to trigger bronchospasm [26].

Thirdly, general anesthesia with ketamine [22], hydroxybutyrate [23] and halothane [2, 7, 17, 30] has been shown to be successful. Several reports indicate that halothane anesthesia is a safe practice for anesthetizing asthma patients wheter they are bronchospastic or not. However, to our knowledge it is only used sporadically in the treatment of LTA [7, 15, 17, 30, 31].

Halothane has a distinct bronchodilatory effect decreasing pulmonary resistance and increasing pulmonary compliance [8, 20, 24]. Different mechanisms of action have been proposed such as a relaxant effect on bronchial smooth muscle, either directly [14], or through non-specific musculotropic reactors [2]. Halothane has been shown to block various bronchospasm inducing stimuli e.g. acetylcholine, histamine [20], hypocapnia [9], and extrinsic allergen administration [4, 21] in the guinea pig, dog, and man. A direct β-adrenergic receptor stimulation has also been implicated [24]. Halothane has no stimulatory effect upon respiratory secretions, nor does it cause laryngeal irritation.

All investigators using halothane in severe asthma, have reported a rapid and persistent amelioration of the respiratory condition. Arrhythmias and hypotension were encountered, although they have never caused severe problems [7, 15, 17, 30, 31].

Drawbacks to the use of halothane in LTA

Halothane administration in LTA must necessitate mechanical ventilation because of possible CO_2 retention [11, 12, 16]. The CO_2 response curve is shifted to the right by many anesthetics including halothane.

Depression of myocardial contractility by halothane [29] is well known. Moreover, myocardial depression due to acidosis, hypercapnia, hypoxia and high inflation pressures already exists in LTA. In our LTA patients treated with halothane the consistent improvement in lungmechanics resulted in a rapid circulatory improvement and hence inotropic support could be reduced and even stopped.

Arrhythmias are one of the main hazards in LTA. Hypoxia, hypercapnia, acidosis and treatment or even overdosage with methylxanthines and adrenergic drugs predipose to the occurrence of serious arrhythmias [11, 25, 32, 40]. Halothane should be administered under careful hemodynamic and electrocardiographic monitoring in low concentrations, gradually increasing up to a concentration of 1%. In our patients arrhythmias were encountered but could always be controlled. We were never obliged to discontinue halothane prematurely.

Although halothane has been shown to inhibit pulmonary hypoxic vasoconstriction [3, 38], this mechanism is probably of minor importance in LTA, a disease affecting both lungs to the same extent, and offset by the beneficial effects of bronchodilatation.

Recently much attention has been paid to the immunodepressive effects of anesthetics. Halothane depresses ciliary activity at high concentrations [27] but the inhibition of normal host defense by halothane remains controversial [13].

Although the reported evidence of halothane hepatitis is low it could represent an additional risk in these patients. An extensive review of this multidebated and contradictory subject is beyound the scope of this paper [6]. Nevertheless it is important to notice that recent investigations concentrate upon the interaction of halothane and hypoxia. In three patients out of five we recorded an asymptomatic temporary chemical hepatitis. However, other factors than halothane could be involved.

Halothane is a powerful cerebral vasodilatator and can aggravate or trigger intracranial hypertension when low intracranial compliance exists as after cardiopulmonary arrest.

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Dr. P. Rosseel
Department of Anesthesia and
Intensive Care
A. Z. Stuivenberg
B-2008 Antwerpen
Belgium

Book review

Paediatric Intensive Care. E. Nussbaum (ed). Futura Publishing Company 1984. $ 65.00

There has been no comprehensive textbook on this subject and I approached this volume with enthusiasm. The Editor states that this is an attempt to present the "state of the art" on paediatric intensive care and is geared to a wide range of readers. There are 26 contributors from various centres in the United States of America. The text is divided into seven sections, is easy to read and there are references at the end of each chapter. The Index is comprehensive and the book well produced. Section I on Central Nervous System Failure begins with a chapter on the normal anatomy and physiology of the brain and the pathophysiology of intracranial disorders. This is followed by a chapter on the neurological aspects of drowning. It supports the treatment regimen advocated by Dr. A. W. Conn (who contributes a Foreword to the book). Care of the unconscious child, head injuries and status epilepticus are covered in the next chapters. A review of CNS infections follows, but six pages of references seem excessive! A discussion of Reye's syndrome completes this section which is a good presentation of these acute cerebral problems. A chapter on cardiovascular monitoring begins Section II. The parameters and techniques are described but discussion of the equipment required would have been useful. Respiratory monitoring is barely mentioned here or elsewhere. The chapter on shock gives a good outline of this complicated subject. Cardiopulmonary resuscitation is well covered with a discussion of the theories of blood flow during external cardiac massage. A clear statement of the procedure following cardiac arrest, drug dosages and practical tips on performing direct current countershock are given. Cor pulmonale is discussed in isolation as congenital cardiac lesions are not included. Section III starts with a comprehensive chapter on radiological evaluation of respiratory emergencies. The 93 pages include 63 X-ray plates which are well produced with the abnormality clearly seen and described. Subjects covered include lesions of the nasopharynx and larynx, foreign bodies, pulmonary oedema, asthma and chest trauma. The chapter on epiglottitis and croup covers these topics well until the point of intubation. Status asthmaticus is adequately discussed though salbutamol is considered experimental! Again, there is no mention of management of respiratory failure. The Adult Respiratory Distress Syndrome in children has a chapter which gives a broad presentation of this subject. Unfortunately, there is a disastrous error on page 318: the fluid regimen is given as 700 – 1500 cc/kg per 24 h. The chapter on respiratory care is written by a respiratory therapist. Ventilatory techniques, the equipment for oxygen therapy and physiotherapy are described. The glaring omission in this section is any mention of intubation and ventilatory support. Several chapters on lung mechanics and physiology are needed, along with guidelines on intubation and ventilation in different disorders. Two chapters on trauma start the next section and include a reminder that 50% of all spinal cord injuries in young children show no X-ray abnormality. There follow useful chapters on burns and haematological problems, and one on poisoning which gives lists of toxic and non-toxic substances with the appropriate management. The subject of infection is next, including aspects of neonatal sepsis, septicaemic shock and pneumonia. The chapter on foreign bodies repeats part of the radiology section. Complications such as tracheo-oesophageal fistula are described and there is good advice on limiting attempts at removal until expert assistance is available. Finally there is a chapter on the Sudden Infant Death Syndrome mainly describing research, which is out of place here. The major criticism of this book is inadequate coverage of respiratory support, but neither is there mention of acute renal failure, nutrition, congenital cardiac lesions or neuromuscular problems. I would have been interested too in topics such as sedation of ventilated patients, the emotional support of the patient and family and the fraught question of organ donation. This is a less than comprehensive text on paediatric intensive care, but there is something for everyone and junior medical staff will find an enormous amount of information provided. It is expensive, but I would attempt to find the money for the chapter on radiology alone.

J. S. Catling (Carshalton, Surrey, UK)