Severe ventilatory failure in asthma in children

Experience of 13 episodes over 6 years

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SUMMARY During the 6-year period from 1 October 1971 to 30 September 1977, 13 (about 1%) of 1225 admissions to hospital with asthma developed severe ventilatory failure (peak arterial $\text{PCO}_2 > 8$ kPa). Mean age was 4.1 years (2.3-7.9), and on average each patient had been admitted to hospital on 5 occasions during the preceding year. 11 gave a family history of asthma or a personal history of associated allergies. A viral upper respiratory tract infection was the commonest precipitant of wheeze, and in 7 patients the duration of wheeziness before admission to hospital was 12 hours or less. Six (0.5%) patients were treated by mechanical ventilation and all survived. The changing patterns of management during the study period are reviewed.

Acute attacks of asthma in children can generally be relieved by sympathomimetic drugs or corticosteroids. Some patients do not respond satisfactorily and various treatments have been suggested for them. Recommendations include the use of aminophylline (Maselli *et al.*, 1970), salbutamol by inhalation (Berg, 1973), or IV infusion (Phelan and Stocks, 1974), or isoprenaline by constant IV infusion (Wood *et al.*, 1972; Parry *et al.*, 1976). Mechanical ventilation is advocated for the occasional patient who deteriorates despite these measures (Downes and Striker, 1966; Wood *et al.*, 1968). We have attempted to characterise the small group of asthmatic patients who develop severe ventilatory failure during attacks despite conventional medical treatment, with particular reference to age, previous history, mode of presentation, and the relation of clinical and blood gas findings, from experience gained during a 6-year period.

Patients

During the period 1 October 1971 to 30 September 1977 acute asthma and 'wheezy bronchitis' in children over the age of 2 accounted for 1225 admissions to the general medical wards at the Royal Hospital for Sick Children, Edinburgh—i.e. 5.6% of all admissions to these wards. Monitoring of blood gas tension and $[\text{H}^+]$ was considered necessary for management in some 30% of patients

and this proportion varied little from year to year. CO_2 retention ($\text{PCO}_2 > 6$ kPa) was uncommon (14%) and severe ventilatory failure with peak $\text{PCO}_2 > 8$ kPa (≈ 60 mmHg) was present in only 11 (3%) patients. These 11 patients (13 admissions) are reported. They are at one end of the severity spectrum for asthmatic children admitted to hospital and represented only 1% of all asthma admissions to the hospital during the period.

Table 1 gives the clinical details of these patients. They were mainly preschool children, 9 of the 11 being less than five, and had a history of eczema or other allergies, and a strong family history of asthma. Eosinophilia ($> 10.0 \times 10^9/\text{l}$) in peripheral blood had been demonstrated previously in all but 2 patients (Cases 8 and 9). Positive skin prick reactions (weal > 5 mm) to one or more allergens were obtained in 5 of 8 patients. Ten patients had been admitted to hospital with asthma previously and were on regular prophylactic medication.

Table 2 summarises details of asthmatic attacks. Most patients on arrival in hospital were distressed with central cyanosis, sweating, tachypnoea, and tachycardia. Three were semicomatose with diminished conscious level and 3 were comatose with little or no response to painful stimuli. In fully conscious patients the respiration rate varied between 40 and 80/min and pulse rate between 130 and 180/min. Arterial blood samples were obtained immediately or within one hour of arrival in hospital in all but 2 (Cases 1 and 6). Wheeziness had usually built up

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Table 1 Clinical details of patients

Case no.	Sex	Age (years)	Nearest growth centile		Eczema and/or other allergies	First-degree relatives asthma and/or other allergies	No. of previous hospital admissions	Regular prestudy treatment
			Height	Weight				
1	M	3.0	50	90	—	+	8	Salbutamol (O)
2	M	4.7	50	75	+	+	1	Salbutamol (O)
3	M	7.9	3	3	+	+	5	Salbutamol (O)
4	F	3.3	10	25	+	+	4	Beclomethasone (I) Salbutamol (O)
5	F	4.0	50	3	—	+	4	Cromoglycate (I) Salbutamol (O)
6a	M	2.5	75	75	+	+	9	Theophylline (O)
6b	M	3.0	90	97	+	+	12	Salbutamol (O)
6c	M	3.2	90	97	+	+	13	Salbutamol (O) Cromoglycate (IN)
7	F	2.5	75	97	+	+	4	Salbutamol (O)
8	F	2.3	75	75	—	+	1	Salbutamol (O)
9	M	6.7	75	75	—	—	0	—
10	M	6.1	75	50	+	+	10	Salbutamol (O)
11	F	3.5	3	3	+	+	7	Cromoglycate (I) Orciprenaline (O)

O=oral; I=inhalation; IN=inhalation (via nebuliser).

quickly and in several patients a life-threatening attack of asthma developed within a few hours of the onset of wheeze. An upper respiratory tract infection (cold) was the commonest apparent precipitant, with coryzal symptoms preceding wheeziness by one or 2 days.

On admission Hb levels ranged from 10.5 to 14.2 g/dl (mean 12.1) and white cell counts from 10.4 to $27.9 \times 10^9/l$ (10.4 to $27.9 \times 10^9/mm^3$) (mean $17.4 \times 10^9/l$; $17.4 \times 10^9/mm^3$) with a marked neutrophil preponderance. Serum Na, K, Cl, and urea concentrations were within normal limits in measurements made before 'rehydration', and serum osmolality varied between 277 and 288 mOsm/kg except in one patient (Case 3, serum osmolality 305 mOsm/kg). Chest x-rays all showed signs of over-inflation, associated with 'probable inflammatory changes' at one or other lung base in 8 cases. *Staphylococcus pyogenes* (Case 6a) was the sole pathogen from nasal and throat swabs, and organisms (pneumococci) were isolated from bronchial secretions obtained at the start of mechanical ventilation in only one patient (Case 8). Blood culture in 8 admissions was negative. Viruses (RSV, rhinovirus, or parainfluenza virus) were isolated from nasopharyngeal secretions, or infection inferred serologically from a 4-fold rise in antibody titre in 7 of 12 admissions (Table 2).

Our plan of treatment was not static during the 6-year study period. The use of isoprenaline by constant IV infusion, and its subsequent replacement by IV salbutamol in patients with ventilatory failure who otherwise would have been treated by mechanical ventilation, were the main innovations.

We used the following general regimen (Tables 2 and 3).

1. IV fluids, 110–150 ml/kg per day (5% dextrose in 0.45% saline). There was no severe dehydration in our cases as the duration of illnesses had been short. Potassium supplements (3 mEq/kg per day) were given during recovery.
2. Systemic bronchodilators: IV aminophylline 4 mg/kg for 20–30 minutes every 6 hours.
3. β -Receptor stimulants: salbutamol respirator solution (0.5%), was administered via a face mask and Wright's nebuliser in a dosage of 0.03–0.05 ml/kg 4-hourly to four patients (Cases 5, 6c, 10, and 11). Salbutamol, 3–4 μ g/kg, was infused IV for 5 min every 3 or 4 hours in four patients (Cases 2, 4, 6a, and 9). Salbutamol, 5–6 μ g/kg (loading dose), IV over 5 min, followed by continuous IV infusion, 0.1–0.3 μ g/kg per min was given to two patients (Cases 5 and 10).
4. Corticosteroids: IV hydrocortisone 100 mg 2- to 6-hourly.
5. Sodium bicarbonate (8.3%), IV in a dosage of 1.5–6.0 mEq/kg to correct metabolic acidosis or 'defend' hydrogen ion $[H^+]$ during preparations for mechanical ventilation (Cases 6b, 7–9, and 11).
6. Oxygen, given by face mask or tent. A concentration of 40% was usually adequate to correct arterial hypoxaemia and maintain arterial P_{O_2} between 12 and 14 kPa. P_{CO_2} was monitored during treatment.
7. Antibiotics: ampicillin 50 mg/kg per day in 4 doses, pending the results of sputum or bronchial secretion cultures.

Table 2 Details of asthmatic attacks

Case no.	Clinical state on admission	Duration of wheeze before admission (h)	'Precipitating' cause		Virology		Treatment	
					Isolates	Paired sera	Drugs	IPPV
1	Cyanosed; restless	4	'Cold'		Negative	Negative	A, HC, NaHCO ₃	—
2	Pink; restless	12-18	Not known		Negative	Paraflu 1	A, S, HC	—
3	Cyanosed; semicomatose	12	'Soaking'		Negative	Not attempted	A, HC	—
4	Cyanosed; semicomatose	12	'Cold'		Negative	Negative	A, S, HC, I	—
5	Pink	12-18	'Cold'		Rhinovirus	Not attempted	A, HC, S*	—
6a	Pink; restless	2	'Cold'		Rhinovirus	Not attempted	A, S, HC, I	—
6b	Cyanosed; semicomatose	Several days	'Cold'		Negative	RSV	A, S, HC, I, NaHCO ₃	—
6c	Pink; restless	Several days	Eggs		Negative	RSV	A, S, HC	—
7	Cyanosed; comatose	12	'Cold'		Rhinovirus, RSV	RSV	A, HC, NaHCO ₃	+
8	Cyanosed; restless	14	Earsache		Paraflu 2	Not attempted	A, HC, NaHCO ₃	+
9	Cyanosed; comatose	2	Sore throat; exercise		Negative	Negative	A, HC, NaHCO ₃ , S	+
10	Pink; restless	Low grade 1-2 weeks	Corticosteroid withdrawal		Not attempted	Not attempted	A, S*, HC, I	+
11	Cyanosed; comatose	4	'Cold'		Not attempted	Not attempted	A, HC, NaHCO ₃	+

A = Aminophylline IV; HC = hydrocortisone IV; NaHCO₃ = sodium bicarbonate (8.3%) IV; S = salbutamol by nebuliser and/or IV; S* = salbutamol by constant IV infusion; I = isoprenaline by constant IV infusion. RSV = respiratory syncytial virus. Case 10 was transferred from another hospital.

Table 3 Sequential acid-base measurements

Case no.	Initial		Peak*		Recovery		Recovery	
	F _i O ₂ %	P _a O ₂ kPa	[H ⁺] (nmol/l) (pH)	Time† (hours)	P _a O ₂ kPa	Base excess (mmol/l)	[H ⁺] (nmol/l) (pH)	Time‡ (hours)
1	25	8.9	71	9	71	3.5	35	—
2	21	8.3	15	< 0.5	69	3.5	35	—
3	FM	9.4	60	< 1	64	3.5	36	5
4	21	7.3	57	< 0.5	64	3.5	36	1
5	21	7.7	46	1	66	3.5	36	14
6a	FM	7.7	56	5.5	57	3.5	36	5.5
6b	FM	10.1	85	3.2	73	3.5	36	6
6c	21	—	44	15.8	73	3.5	36	1.5
7	FM	16.1	66	1.0	72	3.5	36	3.5
8	FM	12.2	62	2.2	68	3.5	36	5
9	FM	13.4	89	7.5	62	3.5	36	3.5
10	31	9.6	64	5.0	72	3.5	36	0.5
11	30	7.2	66	9.0	83	3.5	36	3

Initial data in Cases 5 and 6c, and all recovery data were obtained from arterialised capillary blood samples.

* = PreIPPV in cases 9c-11; † = from admission to hospital to first arterial sample; ‡ = from initial to peak measurements; § = from initial measurements; FM = face mask.

Conversion: SI to traditional units—P_{CO₂}: 1 kPa = 7.5006 mmHg.

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8. Isoprenaline, constant IV infusion of 0.1–0.3 $\mu\text{g/kg}$ per min in patients with progressive ventilatory failure despite other treatments (Cases 4, 6a, 6b, and 10).

The importance of monitoring arterial pH, PCO_2 , and PO_2 in severe acute asthma has been stressed (Simpson *et al.*, 1968). Our patients were treated in a respiratory intensive care unit and arterial or arteriased capillary blood gas tensions were monitored repeatedly. Percutaneous catheterisation of the radial artery (for blood sampling and direct arterial pressure recording) was invaluable in 3 patients (Cases 6a, 9, and 10). Intermittent positive pressure ventilation (IPPV) using a mechanical ventilator was used according to clinical assessment and measurement of blood gas tensions when medical treatment 'failed'. Our techniques were similar in principle to those described for the management of ventilatory failure in bronchiolitis and pneumonia (Simpson *et al.*, 1974a) with minor practical differences. The transition from spontaneous breathing to mechanical ventilation was greatly helped by using ketamine 2 mg/kg IV and atropine 0.12 mg/kg before intubation. Oral intubation preceded nasotracheal intubation (Portex Blue-line tube) and muscle relaxation was achieved by pancuronium bromide 0.1 mg/kg IV, repeated every 2 or 3 hours. Diazepam 0.1–0.2 mg/kg IV was given 4-hourly to maintain sedation. Mechanical ventilation was provided by a volume-cycled ventilator (latterly the Dräger Spiromat 760) at a respiratory rate of 15–30/min and inspiratory:expiratory ratio of 1:2. In each case there was adequate ventilation with a mean airway pressure <60 cm H_2O . The average duration of mechanical ventilation was 15 hours (11–18) in the 6 patients (Cases 6c–11) treated by this means.

Virological and blood gas analytical methods have been described (Simpson *et al.*, 1974b).

Results

Six patients (Cases 6c–11) were treated by mechanical ventilation. Arterial blood gas analysis was delayed for several hours in 2 (Cases 1 and 6a) who did not at first seem unduly ill, but in whom clinical severity increased after admission to hospital; ventilatory failure was diagnosed when PCO_2 was measured. Table 3 gives individual acid-base values for all patients at various stages of their illnesses. Peak arterial PCO_2 values obtained within 6 hours of initial measurements show that CO_2 retention increased in most patients after admission to hospital. Recovery data were obtained at a time when patients were obviously improving clinically

after medical treatment alone or in conjunction with IPPV (Cases 6c–11).

The use of isoprenaline, and latterly salbutamol, by constant infusion probably obviated the need for mechanical ventilation in 4 patients (Cases 4, 5, 6a, and 6b). IPPV was used in patients in whom the clinical state deteriorated and ventilatory failure increased despite medical treatment (Cases 6c–11). The mean arterial PCO_2 of these patients just before IPPV was 12 kPa (8.7–14.2). Our complication rate was low. Subcutaneous emphysema around the neck was noted in one patient (Case 11) and another (Case 10) developed mild postextubation croup. Secondary bacterial infection during mechanical ventilation was avoided.

The different modes of presentation and the effectiveness of treatment in restoring normal ventilation and preventing asphyxia are illustrated by the following case reports. The corresponding acid-base data are presented graphically on the acid-base diagram of Flenley (1971). All patients in the series recovered.

Case reports

Case 2. A 4-year-old boy admitted with severe acute asthma, with hypoxaemia (arterial PO_2 7 kPa), respiratory acidosis, and mild metabolic acidosis when breathing air (Fig. 1). Within 6 hours PCO_2

X Initial arterial data. Peak PCO_2 after 5 hours.

Fig. 1 Case 2. Acid-base state of arterial or arteriased capillary blood over 48 hours.

exceeded 10 kPa despite salbutamol 4 $\mu\text{g/kg}$ IV 3-hourly, aminophylline 4 mg/kg IV 6-hourly, hydrocortisone 100 mg IV 2-hourly, and oxygen (40–50%). This drug regimen was continued and inspired O_2 reduced to 28–30%. Within a further 12 hours PCO_2 had fallen to near 7 kPa. Normal data were obtained after 48 hours. Throughout his arterial PCO_2 /hydrogen ion $[\text{H}^+]$ relationship moved along the acute whole body CO_2 titration line.

Case 4. A 3-year-old girl was admitted with severe acute asthma with hypoxaemia (arterial PO_2 7.3 kPa), respiratory acidosis, and mild metabolic acidosis when breathing air (Fig. 2). She was treated initially with aminophylline 4 mg/kg IV 6-hourly, salbutamol 3 $\mu\text{g/kg}$ IV 4-hourly, and hydrocortisone 100 mg 2-hourly. During the first 12 hours improvement was followed by deterioration in her acid-base state. At peak PCO_2 (8.5 kPa) constant infusion of isoprenaline 0.1–0.3 $\mu\text{g/kg}$ per min was started. Improvement was dramatic; her conscious level improved and within 4 hours PCO_2 and $[\text{H}^+]$ had returned to within the normal range. Her peak pulse rate was 190–200/min during the isoprenaline infusion which was continued for 24 hours. Final acid-base values were obtained after 72 hours.

X Initial arterial data.
I Isoprenaline infusion started after 14 hours—at peak PCO_2 .

Fig. 2 Case 4. Acid-base state of arterial or arterIALIZED capillary blood over 72 hours.

Case 5. A 4-year-old girl was admitted with acute asthma, with a PCO_2 of 5.5 kPa and mild metabolic acidosis breathing air (Fig. 3). She was treated initially with salbutamol respirator solution (0.5% in a dosage of 0.5 ml in 2.0 ml saline nebulised via a face mask for 10 minutes. This was followed by aminophylline 4 mg/kg IV 6-hourly, and hydrocortisone 100 mg IV 4-hourly. During the first 5 hours there was gradual clinical deterioration and increasing CO_2 retention, breathing 30–40% O_2 . At peak PCO_2 (10.0 kPa) she was given salbutamol 5 $\mu\text{g/kg}$ IV (loading dose), followed by 0.1–0.3 $\mu\text{g/kg}$ per min by constant IV infusion. Within 30 min PCO_2 had fallen to 8.3 kPa and after 9 hours PCO_2 and $[\text{H}^+]$ had returned to within the normal range. Her peak pulse rate was 160/min during the salbutamol infusion which was continued for 30 hours. Final acid-base values were obtained after 38 hours.

Case 10. A 6-year-old boy was admitted from a neighbouring hospital where he had been treated for some 12 hours for severe acute asthma, with little response to corticosteroids, aminophylline, and terbutaline IV. His respiration rate was 50/min and pulse rate 160/min. He had severe respiratory acidosis and mild metabolic acidosis (Fig. 4) and PO_2 was 9.6 kPa breathing 30% O_2 . He was given 1.0 ml nebulised salbutamol (0.5% respirator solution) on admission and hydrocortisone 100 mg IV

X Initial data. Peak PCO_2 after 5 hours.
S Salbutamol infusion started within 6 hours—at peak PCO_2 .

Fig. 3 Case 5. Acid-base state of arterial or arterIALIZED capillary blood over 38 hours.

Fig. 4 Case 10. Acid-base state of arterial or arteriatised capillary blood over 24 hours.

4-hourly was continued. Salbutamol was infused continuously during the first hour in a dosage of $10 \mu\text{g/kg}$ per hour. This was followed by isoprenaline infusion, starting with $0.1 \mu\text{g/kg}$ per min and increasing to $0.3 \mu\text{g/kg}$ per min. Peak PCO_2 was 11.4 kPa and pulse rate $190/\text{min}$ after 2 hours of this treatment. A decision was then made to proceed to mechanical ventilation using the Dräger Spiromat ventilator. PCO_2 and $[\text{H}^+]$ returned to within normal limits within 6 hours and weaning was completed after 18 hours. Throughout he remained on aminophylline 4 mg/kg IV 6-hourly and hydrocortisone 100 mg/kg IV 4-hourly.

Case 9. A 6-year-old boy was admitted with severe acute asthma induced by exercise. He was cyanosed, unresponsive to pin-prick, with minimal wheeze, and markedly diminished breath sounds. He had severe respiratory acidosis and mild metabolic acidosis (Fig. 5). Arterial PO_2 was 13.4 kPa breathing O_2 by face mask. He was given hydrocortisone 150 mg IV and sodium bicarbonate 6 mEq/kg IV. Within 30 min $[\text{H}^+]$ had fallen to 62 nmol/l (7.21 pH) with little change in PCO_2 . Mechanical ventilation was started within one hour of admission using the Dräger Spiromat ventilator and an hour later acid-base variables were within normal limits. He was allowed to breathe spontaneously after 3 hours of mechanical

Fig. 5 Case 9. Acid-base state of arterial or arteriatised capillary blood over 22 hours (see text).

ventilation and again developed severe respiratory acidosis ($\text{PCO}_2 \sim 10 \text{ kPa}$). Mechanical ventilation was restarted with a return to normal acid-base state within 2 hours. The total period of mechanical ventilation was under 12 hours. Hydrocortisone 100 mg and salbutamol $4 \mu\text{g/kg}$ were continued IV 4-hourly throughout. Final acid-base measurements were made after 24 hours.

Discussion

The patients in this series constituted only 1% of all admissions to hospital with asthma or 'wheezy bronchitis' over the age of 2 during the 6-year observation period. Most were known asthmatics of preschool age in whom maintenance drug treatment had proved inadequate to prevent life-threatening attacks of asthma. The prophylactic value of drugs such as cromoglycic acid (Intal) (Jones and Blackhall, 1970) and beclomethasone (Becotide) by metered aerosol (Morrow Brown *et al.*, 1972) is well recognised in children old enough to use them properly; lack of co-operation preclude their use in many younger children. Most doctors are also reluctant to use systemic corticosteroids on a long-term basis in these patients, and oral preparations of drugs—such as orciprenaline, salbutamol, or theophylline—singly or in combination, have tended

to become the mainstays of long-term drug management (Williams and Phelan, 1975). Unfortunately these do not always confer adequate protection against further attacks.

We have recently been impressed with the effectiveness of salbutamol respirator solution 0.5% in the home management of selected patients. This involves the use of a portable air compressor and the inhalation of nebulised salbutamol through a closely fitting face mask. The dose recommended is 0.03–0.05 ml/kg body weight (P. D. Phelan, personal communication), the volume being adjusted to 2 ml by adding normal saline. This treatment started at the onset of coryzal symptoms and repeated 3- to 4-hourly may abort an attack at an early stage. Persistent wheeziness despite this treatment is an indication for hospitalisation.

The duration of wheeziness before admission was 12 hours in fewer than 7 patients. Attacks of asthma had built up quickly and ventilatory failure ensued within hours of the onset of wheeze, and not after several days as is usual in potentially fatal asthma in the adult (Macdonald *et al.*, 1976). In these circumstances treatment must be available quickly and if a delay is likely, direct access to hospital is essential. This arrangement, agreed by parents and general practitioners, has proved most satisfactory and 3 of our patients were admitted without first being seen by their own doctors.

The severity of asthmatic attacks was recognised in the emergency admitting department in 11 of our admissions and blood gas analysis quickly carried out. Initial severity may however have been underestimated in 2 patients (Cases 1 and 6a) in whom ventilatory failure was diagnosed when arterial PCO_2 was measured several hours after admission. IV corticosteroids and aminophylline were prescribed immediately in each case, yet arterial PCO_2 generally increased during the next 6 hours. This may have been owing to the apprehension and panic which sometimes accompany severe respiratory distress, or to increasing tiredness and impending exhaustion. The question of the adequacy of initial treatment also arises. Our dosage schedule for aminophylline was slightly lower than that considered optimal (Piafsky and Ogilvie, 1975) when facilities for monitoring blood levels of theophylline are available, whereas our corticosteroid regimen was higher than that recommended by Pierson *et al.* (1974). Salbutamol dosage accorded with earlier recommendations (Berg, 1973; Phelan and Stocks, 1974). Uncontrolled O_2 may have been a factor in aggravating CO_2 retention (Simpson *et al.*, 1968), especially in 3 patients (Cases 2, 5, and 6c) who were treated with 30–50% O_2 initially. The use of sodium

bicarbonate in the treatment of status asthmaticus has been described by Mithoefer *et al.* (1968). It is indicated for the correction of metabolic acidosis, especially in the resuscitation of near-moribund patients (e.g. Case 9, Fig. 5) and to prevent a further rise in $[\text{H}^+]$ while preparations are made for mechanical ventilation (Simpson, 1973). The infusion of sodium bicarbonate almost certainly contributed to an increase in PCO_2 in 3 patients (Cases 6b, 7, and 11) although in one there was a concomitant fall in $[\text{H}^+]$.

Wood *et al.* (1972) and Parry *et al.* (1976) have reported their experience with the constant IV infusion of isoprenaline in the treatment of life-threatening acute asthma. This treatment has been used with success in patients with a rising PCO_2 at a stage when mechanical ventilation seemed the next logical step in management. Parry *et al.* (1976) reported a mean decrease in PCO_2 of 2.4 kPa (18 mmHg) in 27 patients with CO_2 retention (mean PCO_2 7 kPa) within $\frac{1}{2}$ to 4 hours of starting treatment. The efficacy of this treatment was demonstrated in 3 of our patients (Cases 4, 6a, and 6b) in whom arterial PCO_2 was rapidly restored to normal, but in a 4th (Case 10, Fig. 4) CO_2 retention increased despite isoprenaline, and IPPV was used. In the present series the use of salbutamol by inhalation or intermittent IV infusion (for 5 minutes) was without lasting benefit. However in one patient (Case 9) salbutamol by constant IV infusion was effective in restoring normal ventilation without the tachycardia invariably present with isoprenaline. Where equivalent doses of isoprenaline and salbutamol IV in man are compared, the former is 5 times more potent as a bronchodilator, and has a 10 times greater effect on heart rate (Marlin and Turner, 1975). Salbutamol by constant IV infusion has therefore fewer cardiac side effects and is now our first choice β -stimulant.

We adopted the general criteria for mechanical ventilation suggested by Wood *et al.* (1968). In deciding on this means of treatment, the risks of continuing conservative medical management have to be weighed against the possible hazards of mechanical ventilation. The decision to ventilate is always difficult as there are no precise criteria. This treatment should be limited to well equipped centres with experience in dealing with such problems. Fortunately our complication rate was low and hazards—such as pneumothorax, vomiting, and aspiration, subglottic stenosis, and long-term neurological sequelae—were avoided.

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