HIGH FREQUENCY JET VENTILATION
AND ASPIRIN POISONING

by

W. I. CAMPBELL,¹ D. L. COPPEL,² B. F. McLAUGHLIN³

Respiratory Intensive Care Unit, Royal Victoria Hospital, Belfast

ASPIRIN poisoning is responsible for over 200 deaths per annum in England and Wales. Forced alkaline diuresis is advocated to hasten renal elimination of the drug when plasma salicylate levels exceed 500 mg/l, whilst charcoal haemoperfusion is the treatment of choice when plasma salicylate levels exceed 900 mg/kg.¹ This severity of poisoning causes gross respiratory, metabolic and coagulation disturbances, usually requiring cardiovascular and respiratory support.² Conventional low frequency, high volume ventilation is known to impair venous return and cardiac output, so that if respiratory support is needed this type of ventilation may further compromise the cardiovascular system.³,⁴ High frequency jet ventilation (HFJV) is an effective means of controlling ventilation without impairing cardiac output and was successfully used in this case to support respiration in a hypotensive patient suffering from aspirin poisoning.⁵

CASE HISTORY

Day 1: A 55 year old man suffering from a depressive illness was admitted to Craigavon Area Hospital, having consumed 240 aspirin tablets (72g). He was sweating and unresponsive to verbal commands. Respirations were 46 per minute, blood pressure 150/100 mmHg and pulse rate 100 per minute. He had a marked respiratory alkalosis with compensatory metabolic acidosis — Table (line a). Blood was taken for salicylate levels and a stomach washout carried out. A forced alkaline diuresis was commenced using 8.51 of crystalloid fluid intravenously over 24 hours. 100 ml of this was 8.4 per cent sodium bicarbonate. Despite the use of diuretics

| Table |
|-------|
| **High Frequency Jet Ventilation** |

|   | pH   | PaCO₂ | PaO₂ | Base Deficit |
|---|------|-------|------|-------------|
| (a) | 7.406 | 2.44  | 13.03 | 10.2        |
| (b) | 7.385 | 2.87  | 12.21 | 9.0         |
| (c) | 7.11  | 7.41  | 15.87 | 11.5        |
| (d) | 7.45  | 3.92  | 18.53 | 1.4         |
| (e) | 7.27  | 5.57  | 26.40 | 7.0         |
| (f) | 7.35  | 5.53  | 9.47  | 1.8         |
| (g) | 7.38  | 5.23  | 10.67 | 1.0         |

¹ Senior Registrar. ² Consultant Anaesthetist. ³ Medical Physics Technician.
pulmonary oedema occurred so a venesection was carried out, 600 ml of blood being
drawn. At this stage the initial plasma salicylate level became available, it was 940
mg/l. The patient was therefore transferred to this hospital for charcoal haemo-
perfusion. Just prior to transfer, the patient became aggressive and was given
chlorpromazine 50 mg intramuscularly.

**Day 2:** On arrival at the Royal Victoria Hospital, Respiratory Intensive Care
Unit he was unresponsive to verbal commands and tachypnoeic (60 breaths per
minute). Blood pressure was 90/65 mmHg and pulse rate 120 per minute.
Widespread fine crepitations were heard on auscultation of the chest and chest x-ray
was consistent with pulmonary oedema. However, blood gas analysis showed a well
compensated respiratory alkalosis — Table (line b). Arterial and central venous
pressure (CVP) lines were established in addition to electrocardiograph and urinary
output monitoring. Ventilation was with a tidal volume of 800 ml 16 times per
minute but this resulted in the blood pressure falling from 90/65 to 60/40 mmHg,
with heart rate 120 per minute and CVP + 8 cm H20. Transfusion with 80 g of
human albumin and a dopamine infusion (40 µg/kg) only increased the blood
pressure to 66/48 mmHg and respiratory acidosis developed due to inadequate
ventilation — Table (line c). Since increasing the tidal volume would have a further
deleterious effect on cardiac performance, HFJV was commenced via a Mallinckrodt
Hi Lo Jet endotracheal tube using an Acutronic MK800 high frequency jet ventilator.
This was set to deliver 100 breaths per minute at a driving pressure of 1.2 bar at FiO2
0.6. The blood pressure rose and heart rate fell. The dopamine infusion was reduced
and a further blood gas analysis was satisfactory — Table (line d). HFJV was
continued over the next 15 hours with a 5 minute period of 20 breaths per minute
each hour, to prevent airway closure.

The right femoral vein and artery were cannulated in turn, the patient heparinized
(2000 units heparin) and haemoperfusion started. Monitoring of coagulation,
salicylate levels, electrolytes and blood gases were carried out regularly. Shortly
after starting haemoperfusion platelets were transfused. In all, three charcoal
columns were used during haemoperfusion, each for about three hours. Twelve
hours after commencing haemoperfusion the patient was conscious, blood pressure
80/50 mmHg (dopamine 10 µg/kg/min), pulse 100 per minute, CVP 13 cm H20 and
urinary output 100 ml/hour. Salicylate levels had now fallen to 239 mg/l and blood
gas analysis was satisfactory — Table (line e).

**Day 3:** Weaning from the ventilator was commenced by gradually increasing the
frequency of HFJV. Subsequently the patient was able to breathe on a T-piece and
blood gas analysis was satisfactory — Table (line f).

**Day 4:** The patient was now awake and coughing so the endotracheal tube was
removed and oxygen administered by face mask — Table (line g). Blood pressure
was 120/60 mmHg and pulse 84 per minute. The patient’s condition continued to
improve over the next two days after which he was transferred to the referring
hospital.
COMMENT

During forced alkaline diuresis there is a risk of electrolyte imbalance and fluid overload. Pulmonary oedema may occur due to increased pulmonary vascular permeability as a result of the poisoning but is more frequently due to fluid overload. For these reasons central venous pressure monitoring is mandatory in addition to regular serum electrolyte and blood gas estimations.

Should pulmonary oedema occur forced alkaline diuresis should be discontinued and the oedema controlled with either diuretics or venesection. Since these methods failed to alleviate the pulmonary oedema in this patient, intravenous colloids and controlled ventilation were used. In this case the blood pressure fell during controlled ventilation and a respiratory acidosis occurred. HFJV was used in this case since it was considered a rapid means of reducing the PaCO₂ without further compromising cardiac output. HFJV does not significantly alter the cardiac output or mean arterial blood pressure, yet arterial CO₂ levels can be rapidly reduced by this means of ventilation. Since mean airway and intrathoracic pressure are not increased, venous return to the heart is not impaired. Conventional low frequency, high volume ventilation is known to impair venous return and cardiac output, due to the rise in intrathoracic pressure. In this case the patient was able to eliminate a considerable amount of CO₂ during spontaneous breathing, yet with conventional IPPV (800 mg at 16 times per minute) ventilation was inadequate. During HFJV the small volume (80 ml) delivered close to the carina at 100 times per minute were very efficient at lowering the PaCO₂ without impeding cardiac output. However, the use of small volumes over prolonged periods causes airway closure with resulting atelectasis. This was overcome by delivering larger volumes at slower rates for five minutes per hour. Weaning can be facilitated by increasing respiratory rate which results in a smaller tidal volume, allowing PaCO₂ to rise.

SUMMARY

A severe case of aspirin poisoning required charcoal haemoperfusion. A forced alkaline diuresis was attempted but led to fluid overload which did not respond to diuretic therapy. The patient subsequently became hypotensive and developed pulmonary oedema. Attempted control of respiration with conventional low frequency, high volume ventilation caused a further fall in blood pressure, despite inotropic support.

A respiratory acidosis also developed, so high frequency jet ventilation was substituted. The blood pressure rose and the respiratory acidosis rapidly disappeared. It was then safe for charcoal haemoperfusion to proceed.

The patient recovered over the following four days. We believe that the use of high frequency jet ventilation had a major role in the management of this patient.

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