The cost of soda-lime is important, when the economy is illustrated by means of the iso-cost curves.

The iso-cost curves appear very useful for training and are easily computerized from the formula during other conditions, such as change in prices.

Previous publications show a tendency to use fresh-gas flows in circle systems, which are close to the alveolar ventilation in adult patients (5-6 litre min⁻¹). By the use of a Mapleson D system with identical fresh-gas flow, this will always be the cheapest in use and protects the patient against accidental hypocarbia. The use of circle systems for shorter lasting operations thus cannot be justified from an economic point of view.

K. N. CHRISTENSEN
Aalborg
A. THOMSEN
Aarhus
S. JØRGENSEN
Odense

REFERENCES

SINUS ARREST AFTER ALFENTANIL

Sir,—We wish to report two cases of sinus arrest during intubation following administration of alfentanil 30 μg kg⁻¹.

Laryngoscopy and tracheal intubation have been shown to produce a marked increase in heart rate (Millar and Dally, 1970). Pretreatment with alfentanil 30 μg kg⁻¹ has been shown not only to abolish this tachycardia, but also to produce a relative bradycardia significantly lower than control values 4 min after the administration of the alfentanil (Black, Kay and Healy, 1984). This suggests that the alfentanil may have attained a higher level of activity at this time, indicating a more appropriate moment to intubate.

We had intended to study two groups of 20 patients. Patients were premedicated orally with temazepam 10–20 mg. ECG monitoring was commenced on arrival in the anaesthetic room. Anaesthesia was induced with alfentanil 30 μg kg⁻¹ given over 20 s, followed after 1 min by thiopentone 4 mg kg⁻¹ and suxamethonium 1 mg kg⁻¹. The patient's lungs were ventilated by hand with 50% nitrous oxide in oxygen. Laryngeal spray (lignocaine 3 mg kg⁻¹) and intubation were performed in group 1 after 30 s and in group 2 after 2 min.
In group 1 the lowest mean heart rate for the group was 63 beat min\(^{-1}\) and occurred at intubation; the lowest individual recorded heart rate was 42 beat min\(^{-1}\). The study of group 2 was abandoned after six patients had been studied because two patients suffered sinus arrest at the onset of laryngeal spraying. The durations of sinus arrest were 10 and 12 s. In both patients spontaneous reversion to nodal and then sinus rhythm occurred. A third patient developed a bradycardia of 37 beat min\(^{-1}\). All patients recovered uneventfully.

It has been shown in the paralysed, artificially ventilated animal model that stimulation of either the laryngeal or tracheobronchial mucose produces increases in both cervical sympathetic and vagal outflow (Tomori and Widdicombe, 1969). We suggest that similar changes occur in man, and that high-dose opioid pretreatment may attenuate or completely abolish the sympathetic element of this response which allows unopposed vagal activity to produce bradycardia and sinus arrest. Our results suggest that, in the case of alfentanil, this effect is most pronounced if intubation is undertaken 4 min after the administration of the opioid.

There may be a number of reasons for the high incidence of sinus arrest in our patients: First, the use of a potent opioid with a rapid onset of action may result in a greater degree of attenuation of the sympathetic response at the time of laryngeal stimulation. Second, we did not use a neuromuscular blocker with ganglion stimulating properties, such as pancuronium. Third, the presence of increased acetylcholine concentrations as a result of the breakdown of suxamethonium may accentuate the vagal response.

Fortunately, our patients were free from cardiovascular disease and suffered no sequelae. The outcome in patients with cardiac disease might be less favourable.

J. K. Maryniak
V. A. Bishop
London

REFERENCES


ACUTE (TYPE 1) HYPERSENSITIVITY TO I.V. DIAZEMULS

Sir,—Side effects from diazepam formulations are well documented. Pain on injection and thrombophlebitis are the two main disadvantages (Jensen, Huttel and Schou Olesen, 1981). The advent of using fat emulsion as a carrier for diazepam (Diazemuls, Kabi Vitrum, Ltd, London) has reportedly largely done away with these problems (Selander, Curelaru and Stefansson, 1981; Bullimore, 1982; von Dardel et al., 1983) without reducing the efficacy of the drug.

Type 1 hypersensitivity to Diazemuls is unrecorded in the literature, although acute anaphylaxis to Intralipid has been documented (Kamath, Berry and Cummins, 1981). We report a case in which there was a Type 1 hypersensitivity reaction to an i.v. injection of Diazemuls.

A 48-year-old epileptic man presented to our Accident and Emergency Department with a history of a fit, witnessed by his wife. This was probably precipitated by alcohol. He had not received any anti-epileptic treatment for 4 years and had remained fit-free during this time. Whilst being examined he had a focal motor seizure which was controlled by Diazemuls 10 mg i.v. The patient immediately became dyspnoeic and cyanosed with widespread bronchospasm. He also developed an urticarial rash. He had a tachycardia of 120 beat min\(^{-1}\), but his arterial pressure was not recorded at this time. He was resuscitated with oxygen, aminophylline and hydrocortisone, and his bronchospasm resolved over the next 30 min. Further fits were controlled with i.v. chlorpromazine.

As there was no history of asthma, bronchitis or subsequent evidence of foreign body inhalation, his sudden deterioration was attributed to Diazemuls.

Skin pin-prick tests later supported this theory. A local urticarial reaction was produced by Diazemuls, but not by diazepam or 10% Intralipid. Similar pin-prick tests were carried out on 10 control subjects, five of whom had been recently exposed to i.v. Diazemuls and five who had no history of exposure to i.v. sedation or intralipid. In all cases the results were negative. There was no recorded evidence that our patient had been previously exposed to Diazemuls.

We conclude that our patient had an acute (Type 1) hypersensitivity reaction to Diazemuls, and suggest that the reaction was to the lipid vehicle of the particular preparation.

D. J. Deardon
G. L. A. Bird
Swindon

REFERENCES


