on ketamine requirement. Following atropine premedication ketamine 5 mg lb⁻¹ was administered i.m. They found that i.v. supplementation was required to prevent limb movement before surgery in 70% of patients less than 1 year of age and showed that i.v. ketamine requirements for surgical anaesthesia were associated with age, weight and surface area, age being most significant:

\[ \text{i.v. ketamine (mg lb}^{-1} \text{min}^{-1}) = 70.4 - 11.5 \times \text{age (yr)} \]

Other authors have described an increased MAC value for halothane in this age group (Gregory, Eger and Munson, 1969; Nicodemus et al., 1969).

Alternative possibilities for the greater ketamine requirement of younger children include differences in cerebral blood flow and neuronal density, incomplete myelination with impaired transmission, differences in cardiac output and larger extracellular fluid volumes. Regardless of the possibility of abnormal neuronal pathways within the brain, the ketamine requirement of young children should be expected to exceed that of adults.

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REFERENCES


**RESPIRATORY DEPRESSION AFTER EXTRADURAL MORPHINE**

_Sir,—_The editorial by Spence (1980) advocates the use of extradural narcotics as a new and efficient method of relieving pain after operation. It also mentions the risk of inadvertent ventilatory depression from excessive doses of narcotics entering the spinal fluid. Magora and colleagues (1980) reported their experiences of extradural morphine 2–3 mg in 10 ml of 10% dextrose in 98 adult patients without any haemodynamic or respiratory complications.

Respiratory depression has been reported after the use of extradural morphine in both large (15 mg) (Liolios and Andersen, 1979) and smaller doses (3–5 mg) (Glynn et al., 1979), and after extradural pethidine 50–100 mg (Scott and McClure, 1979). It seems pertinent to report respiratory depression after extradural morphine 4 mg in 10 ml normal saline.

A healthy 83-yr-old man had no premedication before transurethral resection of the prostate gland; extradural analgesia was achieved with carbocaine injected through an extradural catheter inserted at L2–3. Postoperative analgesia was provided by morphine 4 mg in 10 ml normal saline administered through the catheter before it was removed shortly after the cessation of surgery. Six hours later, the patient was comatose with inadequate ventilation and pin-point pupils.

He responded to naloxone 0.4 mg i.v., his arterial \( P_{CO_2} \) changing from 13.0 to 6.9 kPa and \( P_{O_2} \) from 3.2 to 8.8 kPa. Three further doses of naloxone were given to maintain adequate ventilation during the following 4 h.

Ventilatory depression after extradural pethidine has been reported as occurring approximately 30 min after extradural administration (Scott and McClure, 1979). In contrast, the time of onset in the patient described was 6 h later, about the same time observed after intrathecal morphine: 6 h and 11 h (Glynn et al., 1979) and 7 h (Liolios and Andersen, 1979).

Prolonged observation of the patient may be required to detect late respiratory complications.

The safe limits of dosage and volume of extradural morphine are still to be defined. It would be interesting to know to what extent the age of the patient may influence these limits.

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**REFERENCES**


**OXYGEN TRANSPORT DURING DOPAMINE INFUSION**

_Sir,—_Dr Scott and his colleagues (1979) contribute a potentially valuable paper on the effect of dopamine on whole body oxygen transport. However, their conclusions do not seem to be borne out by their data.

**Oxygen and carbon dioxide production.** Their increases in \( V_{O_2} \) and \( V_{CO_2} \) were only significant after 60 min at the greater dose of dopamine (30 \( \mu \)g kg⁻¹ min⁻¹) when compared with initial control. The smaller dose of dopamine produced no significant changes.

**Oxygen availability and ratio.** At no time was oxygen availability significantly changed compared with initial control, and the oxygen availability ratio did not change under any of the test conditions.

Their conclusion that dopamine increased oxygen delivery is not correct. Comparison with the post-dopamine state appears inappropriate as the haemodynamic and metabolic state is very different. Oxygen utilization only changed in one test condition and so any inferences about the relationship between oxygen transport and utilization are not possible.

I question the value of giving percentage changes when the absolute value of two control means are different. If we take the example of carbon dioxide production, a change from 4.9 to 7.30 represents a change of 49%. If the same absolute change (i.e. 2.4) had occurred from the initial control of 6.0, then this would be a 40% change.

Is the second change any less significant biologically than the first?

Last, the technique of comparing each of the means of

not the sole cause of Mendelson's Syndrome. These findings (Schwartz et al., 1980). Acidification of foodstuff causes as severe as those caused by hydrochloric acid at pH 1.8 neutralization of gastric acid is a worthwhile objective. Although the physiological changes were similar, the pathological abnormalities were much less severe for the digested food even at pH greater than 2.5 produces physiological, histological and x-ray derangements, at least as severe as those caused by hydrochloric acid at pH 1.8 (Schwartz et al., 1980). Acidification of foodstuff causes death in the same experimental model. These findings support Dr Moir's implication that aspiration of acid is not the sole cause of Mendelson's Syndrome.

We compared subsequently the consequences of emulsion antacid with clear antacid sodium citrate 0.3 mol litre⁻¹. Although the physiological changes were similar, the pathological abnormalities were much less severe for the citrate aspirate (Gibbs, Hempling et al., 1979). Therefore, we are currently investigating the effectiveness of sodium citrate as an antacid, since we still believe that neutralization of gastric acid is a worthwhile objective. Finally, we have also shown that aspiration of partially digested food even at pH greater than 2.5 produces physiological, histological and x-ray derangements, at least as severe as those caused by hydrochloric acid at pH 1.8 (Schwartz et al., 1980). Acidification of foodstuff causes death in the same experimental model. These findings support Dr Moir's implication that aspiration of acid is not the sole cause of Mendelson's Syndrome.

REFERENCES


MATERNAL MORTALITY AND ANAESTHESIA

Sir,—Dr Moir, in his editorial, Maternal Mortality and Anaesthesia, correctly emphasized the continuing problem of aspiration despite the use of antacids (Moir, 1980). He speculated that "... particles of magnesium trisilicate might be a pulmonary irritant". We examined the question in dogs (Gibbs, Schwartz et al., 1979). Kolantyl-Gel, an antacid preparation commercially available in U.S.A. was compared with hydrochloric acid; after instillation to the lung it produced comparably severe decreases in Pao₂, and increases in pulmonary shunt. Histologically, the acid produced the expected haemorrhage, exudate and oedema, while the antacid produced severe bronchopneumonia that did not totally disappear within 1 month. Five other commonly used antacid preparations caused similar histological changes when introduced to the lungs of rabbits (Wynne, Gibbs and Hood, 1979). Magnesium trisilicate, the preparation most widely used in the United Kingdom, is likely to cause similar findings, since all these antacids are of the emulsion-type and contain a variety of globules and particles.

We compared subsequently the consequences of emulsion antacid with clear antacid sodium citrate 0.3 mol litre⁻¹. Although the physiological changes were similar, the pathological abnormalities were much less severe for the citrate aspirate (Gibbs, Hempling et al., 1979). Therefore, we are currently investigating the effectiveness of sodium citrate as an antacid, since we still believe that neutralization of gastric acid is a worthwhile objective. Finally, we have also shown that aspiration of partially digested food even at pH greater than 2.5 produces physiological, histological and x-ray derangements, at least as severe as those caused by hydrochloric acid at pH 1.8 (Schwartz et al., 1980). Acidification of foodstuff causes death in the same experimental model. These findings support Dr Moir's implication that aspiration of acid is not the sole cause of Mendelson's Syndrome.

Johannesburg A-D circuit switch

Sir,—We have read with great interest the paper on the Johannesburg A-D circuit switch by Drs Manicom and Schoonbee (1979). They introduced the concept of combining in a single system the principles and advantages of the Mapleson A (Lack) system for spontaneous respiration and the Mapleson D (Bain) system for controlled ventilation.

Have Drs Manicom and Schoonbee considered the rebreathing that will occur with the new system during change-over from spontaneous to controlled ventilation or vice versa? At the moment of change-over the gases that were in the expiratory limb now become the gases that will be inspired at the next breath, causing an increase in PICO₂. The problem is exacerbated when the reservoir bag in the Mapleson D position becomes the reservoir bag in the Mapleson A position; in the former position it would contain up to 2 litre of expired gas.

In 20 anaesthetized patients the authors, while studying a similar system, investigated the effect of this rebreathing and found initial increases to 4% in PICO₂ and 1% in PICO₂, using a constant fresh gas flow of 70 ml kg⁻¹ min⁻¹. It took more than 10 min for the effect of this rebreathing to be eliminated. In the healthy patient this would not be a hazard; in the seriously ill, it might be.

REFERENCES


MUSCULAR PARALYSIS FOLLOWING I.V. REGIONAL SUXAMETHONIUM TEST

Sir,—Baraka (1978) administered regionally suxamethonium 5 mg i.v. for clinical studies on conscious patients with traumatic peripheral nerve lesions. A contraction of the de-nervated muscle took place with a painful cramp-like sensation. Systemically, the only sign observed was a mild ptosis following the release of the tourniquet. Baraka recommended the use of the i.v. regional suxamethonium test in conscious patients to confirm the diagnosis of nerve injury before suturing is attempted and as a diagnostic aid in other clinical situations.

Experimenting on himself, one of us (J. P.) used Baraka's method by injecting suxamethonium 5 mg diluted in normal saline 40 ml over a period of 30 s via an i.v. indwelling cannula placed between the fourth and fifth metacarpal on the dorsum.