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Ketamine in severe acute asthma

Sir

We recently treated a 35-year-old female asthmatic patient with intravenous bolus ketamine. She is known to be steroid dependent and her prednisolone had been ill-advisedly stopped. She arrived at hospital in severe status asthmaticus and failed to respond in the emergency room to nebulized salbutamol, intravenous aminophylline and hydrocortisone, and blood gases deteriorated. One hour after initiating therapy it was decided to ventilate her mechanically and blood gases improved as a result. However, with the patient very restless and peak airway pressures reaching 60–80 cm H₂O, ketamine was given by intravenous bolus with the aim of achieving sedation without compromising cardiovascular parameters. We were also aware of a report in which ketamine had been used successfully in the treatment of bronchospasm. Peak airway pressures dropped to 40 cm H₂O and it was necessary to give two more smaller bolus doses of ketamine over the next 75 min. What subsequently impressed us was the fact that it was possible to extubate her after only 3¼ h, before any effect from the hydrocortisone, administered 4½ h earlier, would have been expected. She subsequently made an uneventful recovery.

This has led us to question whether ketamine might have a wider role in the treatment of severe acute asthma, given that asthmatic deaths have been increasing in recent years (Burrey, 1974; Jackson *et al.*, 1982), and that a significant number of these still occur in hospital (Sears *et al.*, 1985; Rothwell *et al.*, 1987) where it has been shown that management is variable and treatment sometimes delayed due to the severity of the attack being underestimated (Rothwell *et al.*, 1987; Bucknall *et al.*, 1988).

Ketamine has known bronchodilator effects, although its mechanism of action is unclear (White *et al.*, 1982), and it has been implicated as beneficial in the treatment of bronchospasm in the elderly (Sheref, 1985), the young (Belts *et al.*, 1971; Fisher, 1977; Strube *et al.*, 1986), by continuous infusion (Sheref, 1985; Strube *et al.*, 1986; Park *et al.*, 1987), in ventilated (Fisher, 1977; Rajanna *et al.*, 1982; Park *et al.*, 1987) and non-ventilated (Belts *et al.*, 1971; Strube *et al.*, 1986) subjects, in asthmatics (Fisher, 1977; Strube *et al.* 1986; Park *et al.*, 1987) and non-asthmatics (Rajanna *et al.*, 1982). However, no clinical trial has ever been conducted. Although ether has also been shown to benefit severe acute asthma (Robertson *et al.*, 1985) this is neither widely available nor easily administered, and is less free of side effects.

We therefore suggest that ketamine deserves to be evaluated as an agent which may prove to be a valuable addition to the therapeutic measures available for the fight to reduce hospital deaths from life threatening asthma.

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Migration of a Kirschner Wire from the clavicle into the abdominal aorta

Sir

A 33-year-old male presented to the Accident and Emergency Department on 30 April, 1987, because of back pain which he claimed was caused by lifting.

The pain was described as constant, discomforting, and mostly in the lower back.

Significant past history included a motor vehicle accident in 1985 during which he sustained a fractured right ankle and a fracture of the left clavicle. The fractured clavicle was corrected surgically by open reduction and fixation with Kirschner Wires (Fig. 1). Physical examination demonstrated some tenderness in the region of the second lumbar vertebrae and limitation of straight leg raising in both legs. Power, tone, sensation and reflex were normal.