LETTERS TO THE EDITOR

The haemodynamic effects of morphine

Sir

I read with interest your editorial on the effects of morphine (Archives of Emergency Medicine 4, 131–132). I would agree that the benefits of treatment may not be due to any haemodynamic change produced. I would like to emphasize, however, that changes in blood pressure can occur after the administration of intravenous morphine and other opiates.

Morphine has been shown to produce orthostatic hypotension in some patients (Thomas et al., 1985). In one of the studies mentioned in your editorial (Lee et al., 1976), 10 patients were given 15 mg of morphine intravenously. It is of note that these patients were investigated some hours after initial symptoms, in a coronary care unit, and measurements were made in the supine position. In the United Kingdom, the majority of patients will call their general practitioner soon after the onset of pain and receive analgesia at home. Thereafter, the patient may have to maintain a sitting position during transfer to hospital, for example, to negotiate a staircase, with consequent effects on blood pressure.

Hypotension is more likely to occur if opiates are administered in excessive dosage. Diamorphine 5 mg intravenously has been shown to relieve pain in most patients, rapidly and with few side effects (MacDonald et al., 1969). Morphine has been recommended in an initial bolus dose of 2–4 mg intravenously, if necessary repeated after 5 min (Braunwald et al., 1980). Earlier this year, our department surveyed general practitioners in the Chester area to establish which agents were being used in myocardial infarction. Table 1 gives details of the numbers of doctors administering the most popular preparations at various doses. Fifty-four (70%) of the practitioners who responded were giving 10 mg of opiate or more initially. It was my opinion that many were using doses likely to cause side effects (Chambers, 1987).

Morphine and diamorphine are excellent drugs for relieving pain and anxiety in

Table 1  Numbers of doctors administering various opiates

<table>
<thead>
<tr>
<th>Opiate used</th>
<th>Dose (mg)</th>
<th>Diamorphine</th>
<th>Morphine + cyclizine 50 mg (Cyclimorph*)</th>
<th>Morphine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5</td>
<td>12</td>
<td>1</td>
<td>–</td>
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<tr>
<td></td>
<td>5–10</td>
<td>7</td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>18(3)†</td>
<td>6(2)†</td>
<td>2(1)†</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>–</td>
<td>–</td>
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</tbody>
</table>

†Figures in brackets indicate numbers of doctors specifying intramuscular route.
myocardial infarction and distress in pulmonary oedema. The production of a 'medical phlebotomy' as part of their therapeutic effect has not been proven. It is just as likely that any haemodynamic change and, in particular, severe hypotension which may occur will have an adverse effect.

I would urge caution in relation to dosage of intravenous opiates in the early stages of treatment.

JOHN A. CHAMBERS
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REFERENCES


A severe case of hyponatraemia

Sir

Recently, while working in the Accident and Emergency Department, a 49-year-old man presented in a very agitated state making incomprehensible sounds. His wife knew of no recent fall or trauma of any kind. Examination of the patient proved difficult, but venous access and blood tests were eventually obtained. A history of prolonged alcohol intake and protracted vomiting was eventually obtained from his wife. A serum sodium was 100 meq/l and serum urea was 2.2 mmol/l (Anderson et al., 1985).

He was given hypertonic saline intravenously (Ayres et al., 1982). A chest X-ray showed no gross abnormality. Soon after returning to the Department, having had his X-ray, and while still in an agitated state he aspirated and suffered a respiratory arrest followed by cardiac arrest. He was resuscitated and subsequently transferred to the Intensive Care Unit. Unfortunately, later that evening, he suffered several further episodes of cardiac arrest and died. The serum sodium remained at 113 meq/l in spite of intensive therapy (Levensky, 1983). A post-mortem examination revealed cirrhosis of the liver, oesophageal varices, widespread peptic ulceration, cerebral atrophy and cerebral oedema.