### TABLE III—Comparison of Effect of Practolol and Atenolol on Cardiac Output (measured in 11 Patients) and Blood Gases. Results are Mean ± SE

<table>
<thead>
<tr>
<th></th>
<th>Cardiac output (l/min)</th>
<th>( \text{PaO}_2 ) (kPa)</th>
<th>( \text{PaCO}_2 ) (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At rest (5 minutes)</strong></td>
<td></td>
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<tr>
<td>Practolol 6.66</td>
<td>6.66 ± 0.53</td>
<td>11.3 ± 0.36</td>
<td>4.8 ± 0.15</td>
</tr>
<tr>
<td>Atenolol 4.60</td>
<td>4.60 ± 0.48</td>
<td>11.5 ± 0.44</td>
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<tr>
<td><strong>Exercise</strong></td>
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<tr>
<td>Practolol 5.02</td>
<td>5.02 ± 0.21</td>
<td>11.9 ± 0.44</td>
<td>5.1 ± 0.16</td>
</tr>
<tr>
<td>Atenolol 4.60</td>
<td>4.60 ± 0.16</td>
<td>11.9 ± 0.44</td>
<td></td>
</tr>
<tr>
<td><strong>At 45 minutes</strong></td>
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<tr>
<td>Practolol 7.35</td>
<td>7.35 ± 0.72</td>
<td>11.2 ± 0.44</td>
<td>4.7 ± 0.15</td>
</tr>
<tr>
<td>Atenolol 6.23</td>
<td>6.23 ± 0.40</td>
<td>11.2 ± 0.36</td>
<td>4.8 ± 0.11</td>
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<tr>
<td><strong>5 minutes after isoprenaline (at 50 minutes)</strong></td>
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<td></td>
</tr>
<tr>
<td>Practolol 7.65</td>
<td>7.65 ± 0.76</td>
<td>10.6 ± 0.55</td>
<td>4.5 ± 0.09</td>
</tr>
<tr>
<td>Atenolol 6.97</td>
<td>6.97 ± 0.72</td>
<td>10.7 ± 0.40</td>
<td>4.5 ± 0.09</td>
</tr>
<tr>
<td><strong>20 minutes after isoprenaline (at 65 minutes)</strong></td>
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<td></td>
</tr>
<tr>
<td>Practolol 4.5</td>
<td>4.5 ± 0.16</td>
<td>10.7 ± 0.40</td>
<td>4.5 ± 0.09</td>
</tr>
<tr>
<td>Atenolol 4.5</td>
<td>4.5 ± 0.09</td>
<td>11.5 ± 0.35</td>
<td></td>
</tr>
</tbody>
</table>

Conversion: SI to traditional units—1 kPa = 7.55 mm Hg.

\( \text{PaO}_2 \) after isoprenaline when on practolol that was not present on atenolol, but the difference was not significant.

The pattern of variation of the values was similar in all patients.

### Discussion

The cardioselectivity of practolol has recently been shown by many workers.\(^1\)\(^{15}\) It therefore seemed reasonable to compare the effects of a new beta-adrenergic blocking agent with those of practolol.

Marlin et al\(^13\) suggested that the bronchopulmonary effect of atenolol was intermediate to those of propranolol and practolol. Our results indicate that atenolol is at least as selective for cardiac beta-adrenergic receptors as practolol. This claim can be made with some confidence since tests for blockade of cardiac beta-receptors (exercise tachycardia) and for blockade of bronchial receptors (bronchodilator response to inhaled isoprenaline) were both carried out. Cardioselectivity is a relative concept, and, clearly, every cardioselective beta-blocker may have unwanted effects on ventilatory function. These drugs should accordingly be used with caution in patients with obstructive airways diseases.

Atenolol did not reduce FEV\(_1\), and PEFR substantially. This confirms the findings of Aström,\(^9\) who gave atenolol intravenously to five patients with asthma without observing an increase in airways resistance. We observed a slight decrease in FEV\(_1\), and PEFR after exercise, but none of the patients complained of any discomfort.

Neither atenolol nor practolol interfered significantly with the bronchodilator effect of inhaled isoprenaline. This non-interference with the effect of exogenous, and presumably endogenous, beta-agonists on bronchial smooth muscle may be one of the more clinically valuable characteristics of the selective compared with the non-selective blockers.

The dose of atenolol we used was derived from animal studies. It might have been too high compared with that of practolol as judged from the effect on the heart rate. But the fact that its effect on the airways was still small indicates that atenolol is even more cardioselective than practolol. Atenolol therefore seems to be an appropriate drug to use when practolol would formerly have been chosen because of its cardioselectivity.

### References


### SHORT REPORTS

**Hodgkin’s disease presenting with hypoadrenalism**

Kaplan\(^1\) and Smithers\(^2\) reported that although 8% of patients with Hodgkin’s disease will be found at necropsy to have invasion of the adrenal glands, clinical evidence of hypoadrenalism is “seldom if ever recognisable during life.” No such case has, in fact, been recorded, and the following report is therefore unique.

**Case report**

A 52-year-old woman presented in September 1974 with a three-week history of severe epigastric pain, watery diarrhoea, and vomiting. She had felt well up to eight months previously but had then noticed lassitude, anorexia, and weight loss and had bouts of abdominal pain. An appendicectomy had been performed in 1945. Excision biopsy of an enlarged tuberculous inguinal lymph node in 1970 was followed by eight months’ treatment with streptomycin para-aminosalicylic acid, and isoniazid.

The patient was pale, cyanosed, and moderately dehydrated but apyrexic and normotensive (110/75 mm Hg). Her abdomen was soft with normal bowel sounds but there was some epigastric tenderness. No other abnormality was detected. Haemoglobin was 16.0 g/dl, packed cell volume 0.50 (50%), and white cell count 8.5 × 10\(^3\) (8500/mm\(^3\)). Despite evidence of dehydration, supported by a blood urea of 15 mmol/l (90 mg/100 ml), the serum Na\(^+\) was 121 mmol (mEq)/l, CI\(^-\) 92 mmol (mEq)/l, and K\(^+\) 5.7 mmol (mEq)/l. Intravenous dextrose 5% and normal saline were given and she rapidly improved.

Forty-eight hours after admission her condition suddenly deteriorated and she had an epileptiform fit. Serum electrolytes suggested water intoxication, which recurred on several occasions. Pigmentation was noted in the palmar creases, and hypoadrenalism was provisionally diagnosed. A tetra- carbon test showed low plasma cortisol levels with an inadequate response to stimulation. There was a good clinical response to cortisone acetate 10 mg four times a day, fluoroconisone 0.2 mg daily, and Slow-K 600 mg three times a day. The electrolytes returned to normal and she was discharged.

Three weeks later she was readmitted with signs of peritonitis after the sudden onset of agonising abdominal pain. Laparotomy showed a small perforation in the jejunum distal to a neoplastic stricture, the rest of the gut being normal. The affected bowel was resected. Histologically the jejunum showed lymphocyte-depletion Hodgkin’s tumour tissue (see figure) forming the floor of an annular ulcer, which had perforated. Seeding of the jejunum gave rise to an ulcer 10 mm long, with a nodular submucosal infiltrate. A further perforation occurred three weeks later, and a second laparotomy showed multiple Hodgkin’s tumours of the small bowel, confirmed histologically. The patient died three weeks later from pneumonia, shortly after starting combination chemotherapy.

Necropsy showed bilateral bronchopneumonia and multiple small perforations throughout the small bowel. The retroperitoneal lymph nodes were extensively enveloped by tumour which had surrounded the inferior vena cava, invading and almost obliterating the adrenal glands. The mediastinal and supraclavicular nodes were affected, and there were deposits in the thyroid and gall bladder. Histological examination confirmed the presence of lymphocyte-depletion Hodgkin’s disease in all affected areas. Ane-mortem thrombus was present in the central adrenal veins, giving rise to infarction of the adrenal glands, which were surrounded and almost obliterated by tumour. The adrenals were not tuberculous and the pituitary gland was normal.
Hodgkin's tissue in floor of jejunal ulcer. (× 200.)

Comment

Despite its rarity, adrenal hypofunction must be remembered when patients with poorly differentiated Hodgkin's tumours show electrolyte disturbances.

We thank Mr A S Boulough, FRCS, lately consultant surgeon, Hope Hospital, for referring this patient to the professorial surgical unit.


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Neonatal pneumothorax: survey and prevention

Neonatal pneumothorax is still an important cause of morbidity and mortality, recent reports giving an incidence of 3 cases per 1000 live births. Over a six-year period in this unit the incidence has been reduced from 5 cases to 1 case per 1000 live births, the perinatal mortality rate having fallen from 25 to 18 per 1000 in the same period. Thirty-two symptomatic cases occurred, of which 18 were associated with labour and resuscitation, four with prolonged assisted ventilation, and eight with severe congenital malformations.

Present series

Ten of the cases associated with resuscitation resulted from the delivery of excessive pressures from the Cardiff Penlon bag used with an endotracheal tube. Since the bag was modified in 1970 no case has been noted when it was used with either a facemask or endotracheal tube. Six cases were due to misuse of the soft rubber McIlroy funnel. If the vent hole is occluded by a fingertip when the funnel is applied to the baby's face the airway is faced with an oxygen-line pressure of up to 350 kPa (3600 cm water). The funnels have been successfully modified by making four similar vent holes around the cone. Unmodified funnels designed for ventilation should be covered by a deadweight release valve (see figure). One baby suffered gross lung damage when an endotracheal tube was connected to the oxygen supply via a T piece without a pressure-relief system.

Two deaths from tension pneumothorax were caused by continuous positive airway pressure, which is now used only with a deadweight release valve that opens at 0.5 to 1.0 kPa (5 to 10 cm water). The inertia of water manometers is inadequate to compensate for surges of pressure that may occur in this and other apparatus used for ventilatory support. Intermittent positive-pressure ventilation with a prolonged inspiratory phase was associated with two deaths from tension pneumothorax, suggesting that prolonged inspiration should be cautiously applied. Only one baby with idiopathic respiratory distress syndrome of the newborn developed spontaneous symptomatic pneumothorax.

Congenital abnormalities associated with symptomatic pneumothorax included three babies with polycystic kidneys, three with diaphragmatic hernia, and one each with misplaced intratubal transfusion and situs inversus. Diagnostic lung aspiration produced one pneumothorax, and another occurred after meconium aspiration, which may be preventable by tracheal aspiration with a wide-bore endotracheal tube.

Comment

Interstitial emphysema, pneumomediastinum, surgical emphysema, pneumothorax, and even pneumopericardium may coexist in the same patient, since the causative mechanisms are similar. Many hours may pass after the initial lung damage before a bout of crying causes the final lung rupture and tension pneumothorax.

Careful investigation should determine the cause of any case of neonatal symptomatic pneumothorax and appropriate changes made to equipment and staff training schedules. A deadweight release valve and safe mechanical valves should replace water manometers, and enough simple safe resuscitation sets should be available wherever there are newborn infants. The results suggest that attention to technical detail can diminish potentially lethal pneumothorax while still providing full resuscitation and ventilatory support.

I thank Professor O P Gray and the many members of staff for advice. Initial results were presented to the Neonatal and Obstetric Anaesthetists' Societies in 1971-2.

1 British Medical Journal, 1975, 4, 310.
2 Lancet, 1973, 2, 1304.
5 Macklin, M T, and Macklin, C C, Medicine (Baltimore), 1944, 22, 281.

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