COMPARISON OF HAEMODYNAMIC EFFECTS OF METOCURINE AND PANCURONIUM IN PATIENTS WITH CORONARY ARTERY DISEASE

J. Heinonen and Yrjölä

SUMMARY

The haemodynamic effects of large bolus doses of metocurine 0.45 mg kg\(^{-1}\) and pancuronium 0.1 mg kg\(^{-1}\) were compared in patients with coronary artery disease anaesthetized with diazepam, anileridine and nitrous oxide. Hypotension occurred more frequently after metocurine and was a result of a decrease in systemic vascular resistance. After pancuronium there was no increase in arterial pressure or heart rate, but a small increase in cardiac index. The decrease in heart rate after metocurine was the principal cause of the statistically significant difference in cardiac index between the two groups. Contrary to previous findings, metocurine did not attenuate circulatory responses to tracheal intubation. During surgical stimulation, two of the 10 patients of the pancuronium group developed significant ST segment depression and three patients had a rate-pressure product greater than 12 000 mm Hg beat min\(^{-1}\). However, the difference in rate-pressure product between the groups was not statistically significant.

The myoneural blocking drug metocurine (dimethyl-tubocurarine) was introduced to clinical practice in 1948, but has not been used widely by anaesthetists. Recent studies have revealed that it has no vagolytic effect at cholinergic receptors in the heart and that its ganglion-blocking and histamine-releasing actions are weak (McCullough et al., 1972; Hughes and Chapple, 1976; Savarese, 1979). Stoelting (1974) and Hughes, Ingram and Payne (1976) have reported minimal circulatory effects in patients without cardiovascular disease.

Hypertension, tachycardia and episodes of hypotension should be avoided during anaesthesia for patients with coronary artery disease (Waller, Kaplan and Jones, 1979). Thus, pancuronium has been considered to be the neuromuscular blocker of choice in patients undergoing cardiac surgery (Harrison, 1972; Branthwaite, 1977). However, increases in heart rate and arterial pressure may occur after its administration (Stoelting, 1972). In this study we report a comparison of the cardiovascular effects of large doses of metocurine and pancuronium in patients with coronary artery disease.

PATIENTS AND METHODS

Twenty patients undergoing elective coronary artery bypass surgery were studied (table I). Patients with congestive heart failure, valvular heart disease or an ejection fraction of less than 0.5 were excluded. The purpose of the study and the procedure were explained to the patients, who gave consent. The interval between the last dose of beta-adrenergic blocking drug and induction of anaesthesia was 36-48 h.

<table>
<thead>
<tr>
<th>Sex (M/F)</th>
<th>Pancuronium</th>
<th>Metocurine</th>
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<tbody>
<tr>
<td>7/3</td>
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<table>
<thead>
<tr>
<th>Age (yr)</th>
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<tr>
<td>46.3 ± 2.40</td>
<td>47.9 ± 2.30</td>
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<tr>
<th>Weight (kg)</th>
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<td>72.6 ± 3.40</td>
<td>78.9 ± 2.90</td>
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<table>
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<tr>
<th>Body surface area (m(^{2}))</th>
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<th>Metocurine</th>
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<tr>
<td>1.82 ± 0.05</td>
<td>1.91 ± 0.05</td>
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<table>
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<td>6</td>
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<tr>
<td>&gt;500</td>
<td>2</td>
<td>1</td>
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<th>Metocurine</th>
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<th>Metocurine</th>
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<table>
<thead>
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<th>Beta-adrenergic blocking drug</th>
<th>Pancuronium</th>
<th>Metocurine</th>
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<tr>
<td>9</td>
<td>9</td>
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</table>

<table>
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<tr>
<th>Ejection fraction</th>
<th>Pancuronium</th>
<th>Metocurine</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.65 ± 0.04</td>
<td>0.67 ± 0.04</td>
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</table>

Anaesthesia and plan of investigation

On the evening before operation the patients were given one to two tablets, each containing pentobarbitone 100 mg and promethazine 25 mg, and 1 h before arrival at the operating theatre, diazepam...
0.2 mg kg⁻¹ was given orally. On arrival an i.v. infusion of 5% glucose solution was commenced and diazepam 0.1 mg kg⁻¹ given i.v. Intravascular catheters were inserted under local anaesthesia (1% lignocaine) and the patient was allowed to rest for a few minutes. Control measurements of cardiac output and intravascular pressures were made and blood samples were obtained for analyses of blood-gas tensions, acid–base balance and haemoglobin concentration while the patient was breathing room air.

Anaesthesia was induced with diazepam 0.3 mg kg⁻¹, followed by anileridine 0.4 mg kg⁻¹ (Leritine: Merck, Sharp & Dohme) and thiopentone 1 mg kg⁻¹. The injection rate of diazepam was 5 mg min⁻¹ and that of anileridine 10 mg min⁻¹, whereas thiopentone was administered within 1 min (Anileridine is approximately four times as potent as pethidine with minimal cardiovascular effects in unanaesthetized patients (Tuominen, Heinonen and Heikkila, 1976)). From the start of induction, the patients breathed a mixture of 60% nitrous oxide in oxygen via a face-mask and a non-rebreathing valve and, when needed, ventilation was assisted or controlled manually.

After a second series of measurements, one of the test drugs was given in double-blind fashion. Pancuronium (Pavulon, Organon) 0.1 mg kg⁻¹ or metocurine (Metubine iodide, Lilly) 0.45 mg kg⁻¹ was injected rapidly into a peripheral vein and a third set of measurements was made 5 min after the injection. Thereafter, the larynx and trachea were sprayed with 4% lignocaine 3 ml, endotracheal intubation was performed and the patient's lungs were ventilated using an Engström respirator (model 200). At this stage the fourth set of measurements was made.

Artificial ventilation with a 60% nitrous oxide in oxygen mixture was performed at a frequency of 16 b.p.m. In our experience, ventilation with the minute volume derived from the Engström–Herzog nomogram usually produces hypocapnia and so ventilation was performed with a minute volume 10% smaller than that indicated by the nomogram. After endotracheal intubation a further dose of anileridine was given (1.6 mg kg⁻¹, 10 mg min⁻¹). The degree of neuromuscular block was estimated according to the train-of-four principle, using 2 Hz supramaximal stimulation (Wakeling Instruments Ltd) of the ulnar nerve (Lee, 1975). The aim was to maintain a block of at least 80–90%.

A fifth series of measurements was made immediately before surgery commenced and a sixth after sternotomy.

**Techniques of measurement**

A Swan–Ganz flow-directed thermodilution catheter size 7 F (Edwards Laboratories) was inserted via the right internal jugular vein and its tip was positioned in a branch of the pulmonary artery under pressure and e.c.g. monitoring. This was used for measurement of pulmonary arterial pressure (p.a.p.), pulmonary capillary wedge pressure (p.c.w.p.) and central venous pressure (c.v.p.) and sampling of mixed venous blood. A cannula was inserted to the radial artery for arterial blood sampling and measurement of systemic arterial pressure (AP). The catheters were connected to appropriate transducers and pressures were recorded with an ink-jet recorder (Mingograf 81, Elema–Schöniander). The zero reference point was taken as 5 cm posterior to the sternal angle and intravascular pressures were recorded at end-expiration. Leads V₅ and aVF of the e.c.g. were monitored continuously and recorded during the periods of measurement. Cardiac output (CO) was measured using the Swan–Ganz thermodilution catheter and Devices cardiac output computer 3750 (Branthwaite and Bradley, 1968; Aalto-Seitälä, Heinonen and Salorinne, 1975).

The blood samples were drawn into heparinized glass syringes and immersed in ice water until analysed. PO₂, pH and PCO₂ were measured and base excess (BE) was derived by an automatic analyser (ABL 1, Radiometer). Corrections for temperature were made, using the nomograms of Kelman and Nunn (1966), if the rectal temperature of the patient was less than 36.5 °C.

**Calculations**

Systemic vascular resistance (SVR) was calculated as follows:

\[
SVR = \frac{m.a.p. - c.v.p.}{CO}
\]

Rate–pressure product (RPP) was calculated by multiplication of the heart rate by systolic arterial pressure; oxygen consumption was derived from CO × arterial–mixed venous oxygen content difference. Blood oxhaemoglobin saturation was derived from the dissociation curve line charts of Kelman and Nunn (1966).

**Statistical analysis**

The paired t test was used when the statistical significance of changes within a group was analysed. This test was applied only when the immediate haemodynamic effects of the neuromuscular blocking
drugs were studied (stage 3 as compared with stage 2). The t test for two independent samples was used for analysis of the statistical significance between the changes within the two groups. The difference was considered significant when \( P \leq 0.05 \).

**RESULTS**

Before induction of anaesthesia (stage 1) there was a statistically significant difference in stroke index (SI) and SVR between the two groups (table II). These differences disappeared after induction (stage 2) when a significant difference in c.v.p. was noted.

**TABLE II.** Mean systemic arterial pressure (MAP), mean pulmonary artery pressure (MPAP), pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), heart rate (HR), cardiac index (CI), stroke index (SI), systemic vascular resistance (SVR) and rate-pressure product (RPP) during stage 1 (awake) and stage 2 (asleep). Mean values ± SEM. *\( P \leq 0.05 \); **\( P < 0.01 \); ***\( P < 0.001 \) = significant difference between the two groups

<table>
<thead>
<tr>
<th></th>
<th>Awake</th>
<th>Asleep</th>
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<tbody>
<tr>
<td><strong>MAP (mm Hg)</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pancuronium</td>
<td>91 ± 2.1</td>
<td>84 ± 4.3</td>
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<tr>
<td>Metocurine</td>
<td>86 ± 3.5</td>
<td>78 ± 3.9</td>
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<tr>
<td><strong>MPAP (mm Hg)</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pancuronium</td>
<td>9.4 ± 0.7</td>
<td>14.2 ± 1.4</td>
</tr>
<tr>
<td>Metocurine</td>
<td>10.3 ± 1.0</td>
<td>16.1 ± 1.4</td>
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<tr>
<td><strong>PCWP (mm Hg)</strong></td>
<td></td>
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<tr>
<td>Pancuronium</td>
<td>3.9 ± 0.9</td>
<td>7.8 ± 0.9</td>
</tr>
<tr>
<td>Metocurine</td>
<td>1.9 ± 0.5</td>
<td>9.8 ± 1.5</td>
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<tr>
<td><strong>CVP (mm Hg)</strong></td>
<td></td>
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<tr>
<td>Pancuronium</td>
<td>2.2 ± 0.5</td>
<td>6.8 ± 1.0</td>
</tr>
<tr>
<td>Metocurine</td>
<td>2.6 ± 0.9</td>
<td>9.9 ± 1.3***</td>
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<tr>
<td><strong>HR (beat min^-1)</strong></td>
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<tr>
<td>Pancuronium</td>
<td>72 ± 2.1</td>
<td>70 ± 3.5</td>
</tr>
<tr>
<td>Metocurine</td>
<td>71 ± 3.2</td>
<td>72 ± 5.4</td>
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<tr>
<td><strong>CI (litre m^-2)</strong></td>
<td></td>
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<tr>
<td>Pancuronium</td>
<td>2.88 ± 0.16</td>
<td>2.32 ± 0.10</td>
</tr>
<tr>
<td>Metocurine</td>
<td>3.28 ± 0.17</td>
<td>2.37 ± 0.14</td>
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<tr>
<td><strong>SI (ml m^-2)</strong></td>
<td></td>
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<tr>
<td>Pancuronium</td>
<td>40 ± 2.0</td>
<td>34 ± 2.2</td>
</tr>
<tr>
<td>Metocurine</td>
<td>47 ± 2.3*</td>
<td>34 ± 2.4</td>
</tr>
<tr>
<td><strong>SVR (unit)</strong></td>
<td></td>
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</tr>
<tr>
<td>Pancuronium</td>
<td>18 ± 1.0</td>
<td>19 ± 1.0</td>
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<tr>
<td>Metocurine</td>
<td>13 ± 0.5**</td>
<td>16 ± 1.4</td>
</tr>
<tr>
<td><strong>RPP (mm Hg beat min^-1)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancuronium</td>
<td>9816 ± 360</td>
<td>8326 ± 722</td>
</tr>
<tr>
<td>Metocurine</td>
<td>9070 ± 688</td>
<td>7928 ± 805</td>
</tr>
</tbody>
</table>

Figure 1 shows the changes from the haemodynamic values after induction in stages 3–6 of the study. After metocurine (stage 3), there was a significant decrease in m.a.p., m.p.a.p., c.w.p., c.v.p., RPP (\( P < 0.01 \)), HR and SVR (\( P < 0.05 \)). The decrease in m.a.p. can be attributed to the decrease in SVR. After pancuronium, a small but statistically significant increase in CI (\( P < 0.05 \)) was found and the small decreases in m.p.a.p., c.w.p. (\( P < 0.05 \)) and c.v.p. (\( P < 0.01 \)) were also statistically significant. When the differences between the changes within the two groups were analysed, the only significant difference was that in CI (\( P < 0.05 \)). The decrease in HR in the metocurine group was the principal cause of this difference.

In stage 3, a decrease in arterial systolic pressure (s.a.p.) exceeding 30% of the initial (stage 1) value was found in five patients receiving metocurine compared with only one receiving pancuronium. Two minutes after the administration of pancuronium in two patients s.a.p. was less than 85 mm Hg (70 and 75 mm Hg, respectively).
After tracheal intubation (stage 4) AP, HR, SVR and RPP increased in both groups. No significant differences between the changes within the two groups occurred (stage 4 as compared with stage 2). An increase in s.a.p. exceeding 30% of the initial (stage 1) value was found only in one patient, who had received metocurine. AP and HR of stage 4 shown in figure 1 are those obtained at the time of cardiac output measurement, after the commencement of automatic ventilation. However, most patients exhibited greater values of AP and HR at the time of tracheal intubation. At the time of greatest s.a.p., the mean increases in RPP from the initial (stage 1) values were 14.5% in the pancuronium group and 17.1% in the metocurine group. No difficulties were encountered in intubating the patients and no additional neuromuscular blocking drug was required during the later period of the study.

In stage 5 (before sternotomy) AP, CI, HR and RPP reached the smallest values. There were no significant differences between the changes within the two groups. A decrease in s.a.p. exceeding 30% of the initial value occurred in five patients of the pancuronium group and in eight of the metocurine group. Surgical stimulation produced an increase in AP, HR, SVR and RPP, but there were no significant differences between the changes within the two groups (stage 6 compared with stage 2). One patient treated with pancuronium showed an increase in s.a.p. exceeding 30% of the initial value and three patients of the pancuronium group had RPP greater than 12,000 mm Hg beat min⁻¹, whereas the greatest individual RPP value in the metocurine group was 11,200 mm Hg beat min⁻¹.

Significant depression (>0.1 mV) of the ST segment (precordial lead) was found after intubation in one patient given metocurine and after sternotomy in two patients given pancuronium. No significant differences in blood-gas tensions or acid-base indices were found between the groups (table III). Data for calculation of oxygen consumption were obtained in stages 1 and 5 of the study. In stage 5 oxygen consumption was decreased 24±4 ml min⁻¹ m⁻² in the pancuronium group and 34±6 ml min⁻¹ m⁻² in the metocurine group.

**DISCUSSION**

**Design of the study**

In most of the previous studies of the haemodynamic effects of metocurine, no comparison has been made with other neuromuscular blocking agents. Furthermore, haemodynamic measurements have generally been made during a steady state as suggested by Kennedy and Kelman (1970). Therefore, we compared haemodynamic measurements after the administration of equipotent doses (Savarese, Ali and Antonio, 1977) of metocurine and pancuronium throughout the course of anaesthesia until surgery had started.

Although our patient groups were comparable in other respects, there were some differences in the initial haemodynamic values which decreased after the induction of anaesthesia. Therefore, we used the post-induction values as control when the haemodynamic effects of the neuromuscular blocking drugs were compared.

**Metocurine**

The changes found 5 min after the administration of metocurine differ from those reported by Zaidan and colleagues (1977) in patients with coronary artery disease. Their patients showed no decrease in AP or HR, whereas CO increased significantly. These effects may be a result of light anaesthesia (morphine

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**Table III. Blood-gas tensions and base excess (BE). Mean values ±SEM**

<table>
<thead>
<tr>
<th></th>
<th>1 Awake</th>
<th>3 After relaxant</th>
<th>4 After intubation</th>
<th>5 Before incision</th>
<th>6 After sternotomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{\text{O}_2}$ (kPa)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Pancuronium</td>
<td>10.2±0.4</td>
<td>20.5±1.6</td>
<td>20.9±2.4</td>
<td>15.9±1.6</td>
<td>16.4±1.5</td>
</tr>
<tr>
<td>Metocurine</td>
<td>10.3±0.5</td>
<td>20.8±1.4</td>
<td>21.3±1.7</td>
<td>16.5±1.3</td>
<td>17.5±1.2</td>
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<tr>
<td>$P_{\text{CO}_2}$ (kPa)</td>
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<tr>
<td>Pancuronium</td>
<td>5.0±0.1</td>
<td>5.4±0.2</td>
<td>5.5±0.1</td>
<td>4.5±0.1</td>
<td>4.6±0.1</td>
</tr>
<tr>
<td>Metocurine</td>
<td>5.0±0.1</td>
<td>5.1±0.3</td>
<td>5.5±0.2</td>
<td>4.5±0.2</td>
<td>4.7±0.2</td>
</tr>
<tr>
<td>BE (mmol litre⁻¹)</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Pancuronium</td>
<td>-0.2±0.4</td>
<td>-1.3±0.5</td>
<td>-1.2±0.4</td>
<td>-1.0±0.5</td>
<td>-0.8±0.5</td>
</tr>
<tr>
<td>Metocurine</td>
<td>0.0±0.3</td>
<td>-1.6±0.4</td>
<td>-1.6±0.3</td>
<td>-1.0±0.3</td>
<td>-1.2±0.3</td>
</tr>
</tbody>
</table>
0.5 mg kg\(^{-1}\) and 50\% nitrous oxide) or the smaller
dose (0.35 mg kg\(^{-1}\)) and slower rate of injection of
metocurine in Zaidan's study.

A small decrease in AP and no change in CI have
been reported after metocurine in patients without
cardiovascular disease receiving halothane (Stoelting,
1974) or enflurane (Stanley, 1978). During thiopento-
tone-nitrous oxide-i.v. anaesthetic, meto-
curine 0.2-0.3 mg kg\(^{-1}\) did not produce significant
changes in AP or HR (Hughes, Ingram and Payne,
1976; Savarese, Ali and Antonio, 1977), whereas
greater doses produced hypotension (Savarese, Ali
and Antonio, 1977). Hypotension has been attributed
to histamine release and a slow injection rate is
recommended when large doses of metocurine are
given to patients with poorly compensated circulatory
states (Savarese, Ali and Antonio, 1977).

Like Zaidan and others (1977) and Stanley (1978)
we observed a small decrease in SVR. Fogdall and
DeMaster (1977) have presented evidence to suggest
that metocurine may cause venodilatation. Since the
filling pressures of the heart were decreased in our
patients after metocurine, it is possible that a more
rapid infusion of salt solution had partially prevented
hypotension. The decreases in filling pressures were
not, however, significantly greater after metocurine
than after pancuronium.

**Pancuronium**

Several investigators have reported increases in
HR, CO and AP after pancuronium during steady-
state anaesthesia (Loh, 1970; Kelman and Kennedy,
1971; Stoelting, 1972; Miller et al., 1975). These
changes probably result from the vagolytic action of
pancuronium at the cholinergic receptors in the heart
(Saxena and Bonta, 1970; Hughes and Chapple,
1976) and they are prevented by atropine (Kelman
and Kennedy, 1971; Miller et al., 1975).

Although our patients did not receive atropine, they
showed no increase in AP and HR after pancuronium.
This finding accords with the results of several other
studies in which pancuronium was given to patients
about to undergo cardiac surgery (Harrison, 1972;
Lyons and Clarke, 1972; Clarke and Lyons, 1977; Maunuksela,
1977). In these studies, as in our own,
pancuronium was given immediately after induction
of anaesthesia. Therefore, it is probable that the
measurements were not made during a steady state.
Furthermore, the results may have been influenced by
cardiac disease and residual effects of digoxin or
beta-adrenergic blocking drugs. Whatever the cause
of the absence of increase in HR and AP, it is still
possible that the differences in HR, CI and AP at
stage 3 between our pancuronium and metocurine
groups are a result of the vagolytic effect of
pancuronium.

**Metocurine v. pancuronium**

Savarese, Ali and Antonio (1977) have suggested
that metocurine may be the myoneural blocking drug
of choice during the administration of anaesthesia to
patients with coronary artery disease. In these patients
increases in RPP or episodes of hypotension may pre-
cipitate myocardial ischaemia (Waller, Kaplan and
Jones, 1979). In our patients hypotension occurred
more frequently after metocurine than after pan-
curonium (stage 3). Although this hypotension was
not associated with ischaemic ecg changes, it is
potentially dangerous. In spite of the large doses of
myoneural blocking drugs used, no attenuation was
observed in circulatory response (RPP) to tracheal
intubation in the metocurine group as compared with
the pancuronium group. This finding seems to differ
from that of Basta and Lichtiger (1977), who com-
pared the effects of metocurine and pancuronium on
changes in the tension-time index (TTI) evoked by
endotracheal intubation in patients with coronary
artery disease and anaesthetized with halothane-
nitrous oxide. TTI after intubation was significantly
greater in their pancuronium group and 18% of the
patients given pancuronium showed ST segment
depression.

During surgical stimulation, two patients in our
pancuronium group developed significant ST segment
depression in the precordial lead and RPP was greater
in the pancuronium group than in the metocurine
group, although the difference was not statistically
significant. Further studies are needed to clarify the
efficacy of metocurine in attenuating circulatory
responses to various stimuli.

In patients with coronary artery disease a large
bolus dose of metocurine offered no obvious advantage
over pancuronium. After metocurine, hypotension
occurred more frequently than after pancuronium
and no significant differences were found in circula-
tory responses to tracheal intubation or surgical
stimulation between the metocurine and pancuronium
groups. Our patients were under relatively deep
anaesthesia achieved with diazepam, anileridine,
thiopentone and nitrous oxide and we realize that
the haemodynamics after the administration of
a neuromuscular blocking drug depend greatly
on the milieu into which the drug is adminis-
tered.
ACKNOWLEDGEMENT

Metocurine was kindly provided by Eli Lilly S.A.

REFERENCES


VERGLEICH DER HÄMODYNAMISCHEN WIRKUNGEN VON METOCURIN UND PANCURONIUM BEI PATIENTEN MIT KORONARARTERIENERKRANKUNG

ZUSAMMENFASSUNG
Die hämodynamischen Wirkungen grosser Dosen von Metocurin (0,45 mg kg^{-1}) und Pancuronium (0,1 mg kg^{-1}) wurden bei gefässkranken Patienten verglichen, die mit Diazepam, Anileridin und Stickoxyd narkotisiert waren. Hypotension trat häufiger auf Metocurin ein, und war Resultat eines Fallens des systemischen Gefäßwiderstandes. Nach Pancuronium kam es zu keiner Erhöhung von arteriellem Druck oder Pulszahl, dafür aber des Herzindex. Pulszahlabstieg nach Metocurin war der Hauptgrund des Herzindex-Unterschiedes zwischen den beiden Gruppen. Im Gegensatz zu früheren Ergebnissen erhöhte Metocurin nicht die Kreislaufreaktionen auf tracheale Intubation. Während chirurgischer Stimulierung entwickelten 2 der 10 Pancuronium-Patienten ST-Segmentunterdrückung, und 3 Patienten zeigten ein Pulszahl-Druckprodukt über 12 000 mm Hg Schläge pro Minute. Der diesbezügliche Unterschied zwischen den beiden Gruppen war jedoch statistisch nicht wesentlich.

COMPARACION DE LOS EFECTOS HEMODINAMICOS DE LA METOCURINA Y DEL PANCURONIUM EN PACIENTES CON ENFERMEDAD DE LA ARTERIA CORONARIA

SUMARIO
Los efectos hemodinámicos de grandes dosis de tipo bolus de metocurina (0,45 mg kg^{-1}) y de pancuronium (0,1 mg kg^{-1}) se compararon en pacientes con enfermedades de la arteria coronaria, anestesiados con diazepam, anileridina y óxido nitroso. La hipotensión tuvo lugar con más frecuencia después de la metocurina. No hubo incremento alguno en la presión arterial ni en el ritmo cardíaco, pero se presentó un pequeño incremento del índice cardíaco. La disminución del ritmo cardíaco después de la metocurina fue la principal causa de la diferencia estadísticamente significativa en el índice cardíaco entre los dos grupos. En oposición a los resultados previos, la metocurina no atenuó la respuesta circulatoria a la intubación traqueal. Durante la estimulación quirúrgica, dos de los diez pacientes del grupo de pancuronium desarrollaron una depresión significativa del segmento ST y tres de los pacientes presentaron una relación de ritmo-presión superior a 12 000 mm Hg latidos min^{-1}. No obstante, la diferencia entre la relación de ritmo-presión entre los grupos no fue de un significado estadístico.