DIAZEPAM AND INTRAOCULAR PRESSURE

Sir,—The effect of diazepam on intraocular pressure (i.o.p.) is unknown. A study is described demonstrating the effect of the i.v. injection of diazepam 10 mg on i.o.p. in 10 unpremedicated adult patients awaiting surgery. I.o.p. was measured with a Perkins hand-held applanation tonometer which may be used with the patient in the supine position (Perkins, 1965). This instrument was chosen because it uses the principle of applanation which is more accurate than that of indentation tonometry with the Schiötz tonometer (Kaufman, 1972). Systolic arterial pressure was measured with an oscillotonometer. The heart rate was counted digitally. Expired air was collected via a face-mask and a Ruben non-return valve into a bag and the carbon dioxide concentrations measured (Lode Gasanalyzer L RA-69).

Measurements were made before the injection of diazepam and at 2, 5 and 10 min after injection.

In every patient i.o.p. decreased significantly after injection of diazepam and returned near to the initial value after 10 min (table I). The mean percentage change was $-27.1 \pm 7.8$ ($P < 0.001$) and $-45.6 \pm 9.8$ ($P < 0.001$) at 2 and 5 min respectively.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Before injection</th>
<th>After injection</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I.o.p.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>mean</td>
<td>10.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>2.8</td>
</tr>
<tr>
<td>% change in i.o.p.</td>
<td>mean</td>
<td>$-27.1$</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>7.8</td>
</tr>
<tr>
<td></td>
<td>$t$</td>
<td>10.99</td>
</tr>
<tr>
<td></td>
<td>$P$ on % changes</td>
<td>$&lt;0.001$</td>
</tr>
</tbody>
</table>

All the patients had normal values of i.o.p. before injection. The mean value was $10.2 \pm 2.8$ mm Hg, which is within the normal range of 10.5-20.6 mm Hg, quoted by Leydhecker, Akiyama, and Neumann (1958). After injection mean i.o.p. was $7.4 \pm 2.0$ and $5.6 \pm 2.0$ at 2 and 5 min after injection, respectively, and these values are outside the normal range.

The systolic arterial pressure decreased significantly. However, this decrease was small and the change did not correlate with the decrease in i.o.p. Heart rate and the expired carbon dioxide concentrations did not change significantly.

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REFERENCES


HYPERSENSITIVITY TO ATROPINE

Sir,—We were interested in the case report of hypersensitivity to atropine by Giala and Tzovairi-Tsakona (1978). A safer and appropriate alternative to atropine is glycopyrrolate (Glycopyrrolate USNF). The advantageous use of this drug has already been demonstrated (Ramamurthy, Shaker and Winnie, 1972; Mirakhur, Dundee and Clarke, 1977; Ostheimer, 1977). Isoprenaline possesses other adrenergic properties perhaps undesirable at the time of the antagonism of neuromuscular block and hyoscine is often associated with a secondary bradycardia.

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REFERENCES


NEUROMUSCULAR BLOCKADE BY NEOSTIGMINE

Sir,—We have been unable to show any enhancement of non-depolarizing blockade following a second injection of neostigmine 2.5 mg, and recovery was unimpaired.

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