LETTER TO THE EDITOR

HAEMODYNAMIC RESPONSES TO SCIATIC NERVE STIMULATION DURING ACUTE EXPERIMENTAL ANAEMIA IN CATS

Sir,

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The oxygen carrying capacity of blood is severely reduced by rapid induction of an anaemic state (1). The performance of the left ventricle may also be disturbed due to inadequate myocardial oxygen supply (2). Studies in anaesthetized dogs have demonstrated that haemodilution reduces cardiovascular responsiveness to selective α and β adrenergic receptor stimulation and to certain drugs (3, 4, 5). It has been demonstrated that responsiveness to both vasopressor and vasodepressor drugs reduces during acute normovolaemic anaemia (6, 7). Effect of sciatic nerve stimulation during acute experimental anaemia is reported here.

Experiments were performed on adult cats of either sex weighing 3–6 kg anaesthetized with 70 mg/kg chloralose. The trachea was cannulated and cats were ventilated with respiratory pump. A polyethylene catheter was placed in the descending aorta through a femoral artery to record arterial blood pressure with a pressure transducer (Statham P23Db). The right femoral vein was cannulated for intravenous injections and infusion of dextran.

Normovolaemic anaemia was induced by dextran for blood exchange. Blood was replaced by dextran in steps of 30% each of the total estimated blood volume (5% of body weight) (8). Cardiovascular variables were recorded after a stabilizing period of 30 min following each exchange. The body temperature of the animal was maintained at 37–38°C by surface heating with the help of heating pads. Sciatic nerve was exposed and stimulated with the help of physiograph stimulator. The effect of sciatic nerve stimulation on blood pressure (BP) and heart rate (HR) was studied at three stages i.e. before and after 1st and 2nd exchange of blood by dextran.

TABLE I: Effect of sciatic nerve stimulation on blood pressure and heart rate during graded exchange of blood by dextran.

<table>
<thead>
<tr>
<th>Replacement of blood by dextran</th>
<th>Control</th>
<th>After 1st exchange</th>
<th>After 2nd exchange</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C</td>
<td>S</td>
<td>C</td>
</tr>
<tr>
<td>SAP mm Hg</td>
<td>117 ± 3.0</td>
<td>120 ± 2.0</td>
<td>116 ± 2.0</td>
</tr>
<tr>
<td>DAP mm Hg</td>
<td>60 ± 2.0</td>
<td>80 ± 1.0</td>
<td>62 ± 2.0</td>
</tr>
<tr>
<td>HR beats/min</td>
<td>144 ± 1.0</td>
<td>160 ± 2.0</td>
<td>150 ± 1.0</td>
</tr>
</tbody>
</table>

Values are mean ± SEM : C- Control, S- after sciatic nerve stimulation.
SAP- systolic arterial pressure, DAP – diastolic arterial pressure, HR – heart rate.
On induction of acute normovolaemic anaemia, the haemoglobin (Hb) dropped from baseline level of 14.5 g/dl to 7.2 g/dl and 4.3 g/dl after 1st and 2nd exchange of blood respectively. BP did not show any significant change (P>0.05) following exchange of blood. With fall in Hb, there was corresponding significant increase in HR.

Sciatic nerve stimulation at control Hb produced a rapid rise in BP and simultaneous increase in HR (Table I). However, this vasopressor response was significantly reduced on induction of acute anaemia (Table I).

Acute normovolaemic anaemia produced an increase in HR, which is in agreement with earlier findings in anaesthetized and conscious animals (9, 10). In our study the haemodynamic changes due to sciatic nerve simulation were attenuated on induction of anaemia which could be due to reduced O₂ supply to the myocardial and vascular system due to a sharp drop in the oxygen carrying capacity of the blood. The possibility of involvement of autonomic nervous centres can not be ruled out as in earlier studies it has been demonstrated that sympathetic innervation of the heart is necessary to achieve and/or maintain the usual cardiac output response during acute anaemia (11). This could also imply that responsiveness of chemoreceptor or vasomotor centres to sciatic nerve pressor response is decreased in acute normovolemic anaemia.

O. P. TANDON* AND A. TALWAR
Department of Physiology,
University College of Medical Sciences
and G. T. B. Hospital,
Delhi - 110 095

REFERENCES


*Corresponding Author