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ACUTE MYOCARDIAL INFARCTION IN PATIENTS WITH HYPERTENSION: ASSOCIATION WITH ADVERSE PROGNOSTIC INDICATORS
Andrew J Hogarth, Lee N Graham, Alan F MacKintosh, David ASG Mary. Department of Cardiology, St James’s University Hospital, Leeds, United Kingdom.

An impaired baroreceptor reflex sensitivity controlling the heart period through the vagus nerves (BRS) and sympatho-humoral activation following acute myocardial infarction (AMI) represent adverse prognostic indices. The occurrence of AMI in patients with hypertension (HT-AMI) is not unexpected, and has been associated with a greater mortality than that occurring in normotensive subjects (NT-AMI). This study was planned to determine whether HT-AMI patients have a greater central sympathetic output and BRS impairment than NT-AMI patients.

We examined 11 HT-AMI, 10 NT-AMI patients 2-4 days following AMI and 10 normal control (NC). The groups were matched according to age, body mass index (BMI) and gender (Table). The two AMI groups were also matched according to heart rate (HR). Muscle sympathetic nerve activity (MSNA) was measured by microneurography from the peroneal nerve and quantified in terms of bursts per 100 cardiac beats (b/100b). BRS was obtained from the Valsalva maneuver as the steepest slope between the RR interval (ms) and systolic pressure (mmHg). Data were expressed as mean ± SEM and summarised in the table below.

Mean arterial pressure (MBP) was insignificantly lower in NT-AMI and the HR was higher in NC than NT-AMI (P<0.05; ANOVA post-tests). MSNA hyperactivity relative to NC was greater in HT-AMI than in NT-AMI (at least P<0.05) whilst BRS impairment (at least P<0.01) was insignificantly greater in HT-AMI than NT-AMI.

These results in patients following AMI indicate that pre-existent hypertension leads to an excessive level of sympathetic activation and BRS impairment, and may at least partly explain the adverse prognosis seen in hypertensive patients following AMI.

Table showing data as mean ± SEM for the 3 groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Number (males)</th>
<th>Age years</th>
<th>BMI kg/m2</th>
<th>HR b/min</th>
<th>MAP mmHg</th>
<th>MSNA b/100b</th>
<th>BRS ms/mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>HT-AMI</td>
<td>11(8)</td>
<td>62 ± 2.1</td>
<td>27 ± 1.0</td>
<td>59 ± 2.7</td>
<td>97 ± 4.3</td>
<td>89 ± 3.2</td>
<td>1.7 ± 0.2</td>
</tr>
<tr>
<td>NT-AMI</td>
<td>10(8)</td>
<td>60 ± 2.7</td>
<td>25 ± 0.3</td>
<td>57 ± 1.6</td>
<td>89 ± 2.7</td>
<td>78 ± 3.8</td>
<td>2.9 ± 0.5</td>
</tr>
<tr>
<td>NC</td>
<td>10(7)</td>
<td>60 ± 2.2</td>
<td>27 ± 1.0</td>
<td>60 ± 2.4</td>
<td>99 ± 1.5</td>
<td>52 ± 4.9</td>
<td>4.8 ± 0.4</td>
</tr>
</tbody>
</table>

Key Words: Hypertension, Myocardial Infarction, Sympathetic Nerve Activity

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INHIBITION OF NITRIC OXIDE SYNTHESIS DOES NOT ALTER HYPOGLYCEMIA-INDUCED ACTIVATION OF LUMBAR SYMPATHETIC NERVE ACTIVITY IN RATS
Martin S Mustzel, Onyewere Onwumere, Tayyana Joseph. Biological Sciences, Lehman College, Bronx, NY.

Several studies utilizing nitric oxide (NO) donors or inhibitors of NO synthase have shown that NO generally causes reductions in sympathetic nerve activity (SNA). For example, NO donors usually decrease SNA whereas NO synthase inhibitors produce gradual increases in sympathetic neural output. However, few studies have examined whether NO modulates reflex increases in SNA elicited by sympathoexcitatory stimuli. Given this background, we hypothesized that elevations in SNA, generated by insulin-induced hypoglycemia, would be potentiated by pretreatment with the NO synthase inhibitor, NG-nomomethyl-L-arginine (L-NMMA). We administered an insulin bolus (30U/kg, i.v.) in urethane-anaesthetized rats receiving either no pretreatment (Insulin group; n = 8) or after pretreatment with LNMMA (L-NMMA-Insulin group; 0.35 mg/kg/min, i.v.; n = 13), while measuring blood glucose, mean arterial pressure (MAP), heart rate (HR), and lumbar SNA. We found that insulin caused equivalent blood glucose decreases in the Insulin (from 161±14 mg/dl to 63±3 mg/dl) and L-NMMA-Insulin (from 170±14 mg/dl to 58±15 mg/dl) groups. Insulin-induced hypoglycemia caused expected MAP decreases in the Insulin group (from 113±14 mmHg to 101±14 mmHg) but only expected MAP decreases were abolished by L-NMMA (from 113±11 mmHg to 101±14 mmHg) that were abolished and reversed into elevations in the L-NMMA-Insulin group (from 106±6 mmHg to 112±7 mmHg). Hypoglycemia stimulated substantial HR increases that were not different between the Insulin (from 371±9 bpm to 428±26 bpm) and L-NMMA-Insulin (from 369±10 bpm to 404±21 bpm) groups. Finally, and in contrast to our expectation, increases in SNA to hypoglycemia were not different between the Insulin (273±70 % from 100% baseline) and L-NMMA-Insulin (307±56 % from 100% baseline) groups. These findings suggest that NO does not modulate increases in lumbar SNA induced by severe hypoglycemic stress. Furthermore, our finding that MAP decreases to hypoglycemia were abolished by L-NMMA indicates that the blood pressure decreases may be secondary to epinephrine-induced activation of beta-2-adrenergic receptors as well as NO-induced vasodilation.

Key Words: Blood Pressure, Heart Rate, Insulin