Ethanol-Induced Hypertension Involves Impairment of Baroreceptors

Abdel A. Abdel-Rahman and Wallace R. Woolies

Summary: We studied the effect of 12 weeks of ethanol feeding on arterial blood pressure and baroreceptor reflex control of heart rate in Sprague-Dawley and Wistar rats. Baroreceptor reflex sensitivity and pressor responsiveness were evaluated by evoking graded rises in mean arterial pressure with increasing doses of phenylephrine and angiotensin II. After 12 weeks of ethanol feeding, there was a modest increase in mean arterial pressure with no change in heart rate in both strains. When angiotensin II or phenylephrine was used as the pressor agent, baroreceptor reflex curves (relationships between changes in mean arterial pressure and heart rate) of Wistar rats were shifted upward and had a markedly reduced slope compared with those of control rats, suggesting that impairment of baroreceptor reflex control of heart rate had occurred. This effect was less evident in the Sprague-Dawley rats. Ethanol-fed rats had a higher sympathetic activity, since ß-blockade with propranolol decreased heart rate to a greater degree than that seen in control rats. The pressor response curve of phenylephrine was shifted to the right in control rats challenged with ethanol (0.5 g/kg), implying the presence of ß-blockade. This shift was not present in ethanol-fed rats, showing that tolerance had developed to this effect of ethanol. These findings show that attenuation of baroreceptor reflex function is associated with ethanol-induced hypertension but do not establish whether this is a cause or an effect of the developed hypertension. (Hypertension 10: 67-73, 1987)

Key Words: • ethanol • hypertension • baroreceptors • heart rate • phenylephrine • cardiac ß-blockade • sympathetic activity

Numerous epidemiological studies have established that there is a positive correlation between the duration and extent of ethanol intake and the development of hypertension.1-5 Recent controlled clinical trials have shown that ethanol acts as a pressor agent, even in hypertensive patients,4 and that when ethanol intake ceases, blood pressure returns to predrinking levels.4,5 However, whether ethanol is the causative agent cannot be determined from such studies. Similarly, the mechanism (or mechanisms) by which ethanol intake elevates blood pressure is unknown, probably because of the absence of a satisfactory animal model.

Recently, Chan and Sutter and colleagues6-7 developed a rat model for ethanol-induced hypertension in which they showed that ethanol intake for 4 to 12 weeks caused a moderate rise in blood pressure. After 12 weeks of ethanol feeding, elevated blood pressure was associated with a significant increase in plasma norepinephrine,7 which suggested the possibility that sympathetic nervous system activity may have been enhanced by ethanol feeding.

On the other hand, the baroreceptor reflex control of heart rate (HR) is known to be depressed in human8,9 and experimental10,11 hypertension. Impairment of baroreceptors has been shown to precede the development of hypertension in the Dahl salt-sensitive rat,10,11 which suggests that it may be a primary factor in the development of hypertension in that model. We have previously shown that Sprague-Dawley rats maintained on ethanol for 4 weeks did not become hypertensive, even though there was a marked inhibition of baroreceptor reflex activity.12 Therefore, if baroreceptor impairment is a primary factor in ethanol-induced hypertension, then a longer period of ethanol feeding should result in elevated arterial pressure.

In this study, we report the effect of 12 weeks of ethanol feeding on blood pressure, HR, baroreceptor reflex control of HR, and pressor responsiveness to phenylephrine (PE) and angiotensin II (ANG II). We used both Sprague-Dawley and Wistar rats to compare our results with those of Chan et al.6,7 and to determine if the effect of ethanol would be greater in the latter
strain. We also studied the effect of sequential cardiac blockade with propranolol and atropine to evaluate the possibility 1) that a higher sympathetic drive to the heart might be present but masked by a direct negative chronotropic effect of ethanol, 2) that the HR responses secondary to the injection of pressor agents were reflex in origin, and 3) that nonreflex chronotropic changes, if any, were not influenced by ethanol.

Materials and Methods

Male Sprague-Dawley and Wistar rats (Charles River Breeding Laboratories, Research Triangle Park, NC, USA) weighing 180 to 220 g initially were randomly allocated to either the ethanol-fed or control group. The method of ethanol feeding was that described by Chan and Sutter in which rats received 5% ethanol (vol/vol) in the drinking water for the first week, 10% for the next 2 weeks, and 20% from Weeks 4 to 12. The control group was fed tap water ad libitum, and all rats had constant access to Purina Lab Chow (St. Louis, MO, USA). Daily food and water consumption were recorded, but animals were not pair-fed. On this regimen average daily ethanol consumption was 10 to 11 g/kg. Blood pressure was determined by the tail-cuff method at Weeks 0, 2, and 6 in Sprague-Dawley rats and Weeks 0, 2, and 8 in Wistar rats. Blood pressure was measured by direct arterial cannulation at Week 12. Blood ethanol concentration was determined by the method of Bonnichsen and Lundgren from arterial blood samples taken from anesthetized rats at the time of the experiment. All experiments were conducted between 0730 and 1130 to minimize the diurnal variation in ethanol metabolism.

Measurement of Baroreceptor Reflex Sensitivity

The details of this technique have been published elsewhere. Baroreceptor reflex sensitivity (BRS) was assessed as the gain in baroreceptor reflex function according to the method of Komer et al. Graded increases in arterial pressure were produced by bolus injections of PE or ANG II. The peak change in mean arterial pressure (MAP) and the associated reflex bradycardia were used to calculate BRS using the ratio \( \Delta HR/\Delta MAP \) (expressed as beats/min/\( \ \text{mm Hg}^{-1} \)). Since we had previously found that this ratio was independent of the dose of the pressor agent used, the average values of all \( \Delta HR/\Delta MAP \) measurements obtained both before and after administration of saline or ethanol were used as a measure of BRS.

At Week 12 of ethanol feeding, control and ethanol-fed rats of both strains were subdivided into two groups according to the pressor agent used and the subsequent treatment. BRS of Wistar rats was assessed by injection of PE, in doses of 0.25, 0.5, 1, 2, and 4 \( \mu g/kg \), and of ANG II, in doses of 2.5, 5, 10, 20, 40, and 80 ng/kg. Immediately after the assessment of BRS, Wistar rats (ethanol-fed and their controls) were challenged by a short-term intravenous dose of ethanol (0.5 g/kg) to determine if tolerance had developed to the effect of ethanol. BRS was assessed in Sprague-Dawley rats by the same procedure. However, instead of receiving a short-term dose of ethanol, Sprague-Dawley rats were treated with propranolol (1 mg/kg) and 15 minutes later with atropine (1 mg/kg). These doses have been reported to produce complete blockade of \( \beta \)-adrenergic receptors and muscarinic receptors in rats.

Each rat in the control or ethanol-fed groups in both strains served as its own control, and the PE or ANG II dose-response curves were repeated after the short-term injection of ethanol in the Wistar rats or after \( \beta \)-adrenergic and muscarinic blockade in the Sprague-Dawley rats. Either pressor agent was administered at 5-minute intervals as this had previously been found adequate for HR and blood pressure to return to preinjection baseline values. Therefore, all baseline, or resting, MAP and HR values are presented as the mean of all determinations of both variables over the control (pretreatment) period. MAP was calculated as diastolic blood pressure plus one third pulse pressure.

Dose-response curves were constructed by plotting the peak rise in blood pressure evoked by the different doses of each of the pressor agents and were obtained under the following conditions: 1) long-term exposure to ethanol, and 2) short-term administration of ethanol to control and ethanol-fed rats. Peak rises in MAP evoked by either pressor agent together with the corresponding nadir changes in HR were used to construct \( \Delta MAP/\Delta HR \) curves for either pressor agent before and after the short-term treatments. The slope of the linear regression line relating decreases in HR to increases in MAP was taken as a measure of the gain in BRS.

Statistical Analyses

Values presented are the means ± SE. Student’s \( t \) test was used in the analysis of paired and unpaired means, with the level of significance chosen at \( p \) less than 0.05. The stimulus response curves were analyzed by the one-way analysis of variance, and a test for the equality of elevations was used to determine whether the regression lines were identical.

Results

Both Wistar and Sprague-Dawley rats consumed a comparable amount of ethanol, 10 to 11 g/kg/day, which resulted in a similar blood ethanol concentration on the morning of the experiment (0.53 ± 0.04 in Wistar rats and 0.49 ± 0.05 mg/ml in Sprague-Dawley rats). Ethanol was not detectable in the blood of control rats of either strain. The average daily food intake was markedly reduced in ethanol-fed rats of both strains, which was manifested as a significantly \( (p < 0.001) \) lower body weight at the end of the experiment. The weight difference was more prominent in Sprague-Dawley rats (420 ± 11 g in Wistar ethanol-fed rats vs 487 ± 12 g in the appropriate controls and 433 ± 14 g in Sprague-Dawley ethanol-fed rats vs 547 ± 17 g in the control group). The starting body weights of the treatment and control groups of either strain were very similar at Week 0 (Table 1).
Table 1. Measurements Taken at Weeks 0 and 12 on Ethanol-Treated and Control Rats

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Ethanol-treated</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Week 0</td>
<td>Week 12</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wistar rats</td>
<td>10</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Weight (g)</td>
<td>220 ± 3.9</td>
<td>487 ± 12</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>117 ± 3.9</td>
<td>131 ± 5.5</td>
<td></td>
</tr>
<tr>
<td>Estimated ethanol consumption (g/kg/day)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Blood ethanol concentration (mg/ml)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Sprague-Dawley rats</td>
<td>11</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Weight (g)</td>
<td>195 ± 2.7</td>
<td>547 ± 17</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>106 ± 2.6</td>
<td>132 ± 4.7</td>
<td></td>
</tr>
<tr>
<td>Estimated ethanol consumption (g/kg/day)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Blood ethanol concentration (mg/ml)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SEM except for daily ethanol consumption; these values were estimates and not measurements.

Systolic blood pressure was measured by the tail-cuff method at Week 0 in all rats and by the direct method in anesthetized rats at Week 12.

*p < 0.001, †p < 0.05, compared with Week 12 control values.

Blood Pressure and Heart Rate

Baseline systolic blood pressure, measured by the tail-cuff method, was similar in all rats at the beginning of the experiment (Week 0; see Table 1). Over the 12-week experimental period systolic pressure of control rats of both strains increased, but the increase in systolic pressure produced by ethanol was significantly greater than the control levels in both groups (Figure 1). Thirteen Wistar rats received ethanol, and three of these did not show any increase in their blood pressure. Similarly, 10 Sprague-Dawley rats were fed ethanol, and three did not manifest any increase in blood pressure. In fact, after blood pressure began to be elevated three ethanol-fed rats in each strain consistently remained normotensive. The nonresponsive rats of either strain had similar blood ethanol concentrations and body weight values after 12 weeks of ethanol feeding as compared with those of responsive rats. However, the values presented in Figure 1 and Table 1 include all rats initially placed on the ethanol diet. The 12-week values were obtained with the rats under anesthesia and were direct measurements of systolic arterial pressure made before other physiological measurements. Figure 2 shows that the MAPs of ethanol-fed Wistar and Sprague-Dawley rats after 12 weeks were significantly (p < 0.02 and p < 0.001) higher than their respective control values. However, as also shown in Figure 2, HR of both strains was not altered by ethanol feeding.

Baroreceptor Reflex Control of Heart Rate

BRS was attenuated by long-term ethanol feeding particularly, in Wistar rats (Table 2). When BRS was evaluated by using PE as a pressor agent there was a 61% inhibition, and when ANG II was the pressor agent there was a 59% inhibition (see Table 2). As shown in Figure 3, the line relating reflex decreases in HR to evoked increments in MAP is shifted upward in ethanol-fed rats, indicating that for a comparable rise

![Figure 1](http://hyper.ahajournals.org/doi/fig/10.1161/01.HYP.74.1.1)

**Figure 1.** Changes in systolic blood pressure over a 12-week ethanol feeding period in Wistar rats and Sprague-Dawley rats. Systolic blood pressure was measured by the tail-cuff method except at Week 12, where it was measured directly while the rats were under chloralose anesthesia. Values are means ± SEM, and the number of rats in each group of both strains is shown in parentheses. Blood pressure was significantly higher at Week 6 in Sprague-Dawley ethanol-fed rats (p < 0.05, indicated by asterisk); at Week 8 in Wistar ethanol-fed rats (p < 0.001, indicated by double asterisks); and at Week 12 (p < 0.05, indicated by asterisk) in both strains as compared with the appropriate control.
in blood pressure there was a significantly smaller ($p < 0.05$) decrease in HR. On the other hand, in Sprague-Dawley rats no significant change in BRS was observed in ethanol-fed rats (Figure 4; see Table 2).

The effect of a short-term dose of ethanol (0.5 g/kg) on $\Delta$MAP-$\Delta$HR relationship in control and chronically ethanol-fed Wistar rats, evaluated by the effect of PE and ANG II, is shown in Figures 5 and 6. There was no change in this relationship when PE was used as the pressor agent in either the control or ethanol-fed groups (see Figure 5). When ANG II was used as the pressor agent there was a slight upward shift in this line in Wistar control rats, but no change occurred in ethanol-fed animals challenged with a short-term ethanol dose (see Figure 6).

### Cardiac Autonomic Blockade

Cardiac autonomic blockade experiments were conducted in chronically ethanol-fed Sprague-Dawley rats and appropriate controls. As shown in Figure 7A, the MAP of ethanol-fed rats was approximately 20 mm Hg higher than the control value. Propranolol produced the expected drop in MAP; however, this propranolol-evoked decrease in MAP, even though significant ($p < 0.05$) in both groups, was much greater in the ethanol-fed rats (22 vs 11%) and resulted in comparable MAP values in both groups (97 ± 4 vs 94 ± 3.2 mm Hg; see Figure 7A). When atropine was added after propranolol, blood pressure of the control group returned to control values whereas the blood pressure of ethanol-fed rats did not return to predrug levels. As shown in Figure 7B, baseline HR was comparable in control and ethanol-fed Sprague-Dawley rats. However, after propranolol administration, the HR decreased 10% in the control group, whereas there was an 18% decrease in the chronically ethanol-fed group that resulted in significantly ($p < 0.05$) lower HR values in ethanol-fed rats as compared with their controls (302 ± 9.1 vs 332.9 ± 9 beats/min; see Figure 7B). Additive parasympathetic blockade by atropine restored HR of the control group to predrug levels, and a similar but lesser effect was observed in the chronically ethanol-fed group (see Figure 7).

As shown in Figure 4, cardiac autonomic blockade by propranolol and atropine abolished the cardiac-slowing response to evoked increments in MAP produced by both PE and ANG II in control and ethanol-fed rats. These data clearly show that the bradycardia observed before blockade was mainly reflex in origin, the doses of each of the pressor agents used had negligible nonreflex chronotropic effects, and more importantly, long-term ethanol feeding was without effect on nonreflex chronotropy.

### Pressor Responsiveness

Long-term ethanol-feeding had no effect on pressor responsiveness in either strain of rats since the PE

### Table 2: Blood Pressure, Heart Rate, and Baroreceptor Reflex Sensitivity Values

<table>
<thead>
<tr>
<th>Variable</th>
<th>Phentolamine (mg kg$^{-1}$)</th>
<th>ANG II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Ethanol</td>
</tr>
<tr>
<td>Wistar rats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>119.4±3.0</td>
<td>133.8±6.1</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>406.2±10.2</td>
<td>370.6±15.6</td>
</tr>
<tr>
<td>$\Delta$HR/$\Delta$MAP (beats/min mm Hg$^{-1}$)</td>
<td>−1.18±0.19</td>
<td>−0.46±0.26$^+$</td>
</tr>
<tr>
<td>Sprague-Dawley rats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>110.3±2.6</td>
<td>137.8±5.4$^@$</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>376.0±16.3</td>
<td>388.3±20.9</td>
</tr>
<tr>
<td>$\Delta$HR/$\Delta$MAP (beats/min mm Hg$^{-1}$)</td>
<td>−1.23±0.12</td>
<td>−1.08±0.35</td>
</tr>
</tbody>
</table>

Baroreceptor reflex sensitivity was obtained by averaging four to five measurements made by evoking MAP rises by graded doses of the pressor agents. Data are means ± SEM and represent the baseline values for the subgroups that were subsequently treated with short-term ethanol administration (see Figures 5 and 6) or propranolol and atropine (see Figure 4).

$^*$p<0.002, $^@$p<0.05, $^+$p<0.05, $^@$p<0.01, ||p<0.001, compared with control values.
Figure 3. HR response to evoked increments in MAP by phenylephrine (PE; A) or ANG II (All; B) in Wistar ethanol-fed rats and their controls. The upward shift of ΔHR-ΔMAP curve of ethanol-fed rats was significantly different when compared with the control curve; the baroreceptor reflex sensitivity (ΔHR/ΔMAP) values are presented in Table 2. Values are means ± SEM, and values in parentheses indicate the number of rats in each group.

Figure 4. Relationship between changes in HR and MAP in Sprague-Dawley rats fed with ethanol for 12 weeks and their controls. The rises in MAP were evoked by phenylephrine (A) or ANG II (B) before (circles) and after (triangles) cardiac autonomic blockade with propranolol and atropine (1 mg/kg each). The HR responses to evoked rises in MAP were virtually abolished after cardiac autonomic blockade in both ethanol-fed rats and their controls. Values are means ± SEM, and the number of rats in each group is shown in parentheses.

Figure 5. Effect of an acute dose of ethanol (0.5 g/kg i.p.) on ΔMAP-ΔHR relationship in control rats (A) and 12-week ethanol-fed rats (B). The increments in MAP were evoked by phenylephrine. Note that acute ethanol administration had no significant effect on this relationship. Data are means ± SEM, and the numbers in parentheses indicate the number of rats in each group.

FIGURE 5. Effect of an acute dose of ethanol (0.5 g/kg i.p.) on ΔMAP-ΔHR relationship in control rats (A) and 12-week ethanol-fed rats (B). The increments in MAP were evoked by phenylephrine. Note that acute ethanol administration had no significant effect on this relationship. Data are means ± SEM, and the numbers in parentheses indicate the number of rats in each group.

Discussion

The increase in blood pressure after ethanol feeding began within 4 to 6 weeks and continued over the remaining 6 weeks of the study. This finding is in agreement with that of Chan et al., who reported that blood pressure elevation began 4 weeks after ethanol feeding and continued to increase up to 12 weeks. In both studies the method of feeding ethanol was the same. At the end of 12 weeks in our study no correlation (r = -0.03) was observed between the concentration of blood ethanol and the level of hypertension. In fact, in either strain, the rats that did not manifest elevated blood pressure had blood ethanol levels comparable to those that did.

The present study, which establishes that long-term ethanol feeding produces hypertension associated with an impairment of baroreceptor reflex control of HR, is an extension of a previous study, in which we showed that ethanol feeding for 4 weeks produced a significant
The relationship between changes in HR and MAP is shown as in Figure 5 except that increments in MAP were evoked by ANG II. Note that the HR response was slightly attenuated by short-term ethanol administration in the control group.

**Figure 7.** Effect of successive administration of propranolol (1 mg/kg) and atropine (1 mg/kg) on basal MAP (A) and HR (B) values of 12-week ethanol-fed rats (n = 10) and their controls (n = 11). Values are means ± SEM. Asterisk indicates (p < 0.05) significant difference compared with pretreatment values. Note that the HR value of the ethanol-fed rats was significantly smaller as compared with the control value after propranolol administration.

Attenuation of BRS even though the animals remained normotensive. It is tempting to speculate that impairment of baroreceptor reflex control of HR is an important determinant in the development of ethanol-induced hypertension. However, neither our previous nor the present study establishes a cause-and-effect relationship between ethanol-induced impairment of baroreceptor reflex control of HR and the development of hypertension; whether or not such a relationship exists is under investigation.

In their most recent study, Chan et al. suggested that the responses to long-term ethanol consumption may vary according to the strain of rat. For that reason we used both Wistar and Sprague-Dawley rats. Our findings show that a similar degree of hypertension was produced by long-term ethanol feeding in both strains. However, the type of alteration of baroreceptor reflex control of HR was different in each strain. In Wistar rats there was a definite impairment of BRS at the end of 12 weeks of ethanol feeding, whereas in Sprague-Dawley rats there was resetting of baroreceptors. This conclusion is based on the findings that BRS was significantly depressed in ethanol-fed Wistar rats (see Table 2 and Figure 4) as compared with the appropriate control. However, the finding that baseline MAP was significantly higher in ethanol-fed Sprague-Dawley rats as compared with their controls, while the baseline HR values were comparable in both groups suggests a resetting of baroreceptors in this strain following 12 weeks of ethanol feeding. It is possible that Wistar rats are more sensitive to the effects of ethanol. Since ethanol intake was comparable in both strains and since the degree of elevation of arterial pressure was comparable, our findings show that the development of ethanol-induced hypertension is independent of strain. However, the finding that Wistar rats were...
better able to maintain body weight while on a long-term ethanol diet may make this strain more desirable.

Our findings that 12 weeks of ethanol feeding produced resetting of baroreceptor reflex control of HR in Sprague-Dawley rats is somewhat different from our previous finding that 4 weeks of ethanol consumption produced a significant impairment of BRS in the absence of any change in blood pressure. In our previous study, after 4 weeks of ethanol feeding, blood pressure was not changed but HR was decreased. In this study, after 12 weeks of ethanol, blood pressure was elevated while HR was comparable to control values. These differences plus the longer duration of ethanol feeding may explain why ethanol-induced impairment of baroreceptor reflex control of HR was noted earlier and not in this study.

Although our data do not establish a mechanism by which long-term ethanol consumption produces hypertension, it is reasonable to assume that an attenuated baroreceptor reflex mechanism would cause an enhanced sympathetic activity. This is supported by the findings that there is an inverse correlation between BRS and plasma norepinephrine concentration and a positive correlation between plasma norepinephrine levels and sympathetic neural activity. Our data showing that ß-blockade produced a greater reduction in HR in ethanol-fed rats than in controls and the findings of Chan et al. that plasma norepinephrine levels were increased after 12 weeks of ethanol support the view that long-term ethanol administration is associated with enhanced sympathetic activity. Whether or not this increased activity is involved in a cause-and-effect relationship has not been established.

Cardiac autonomic blockade with propranolol and atropine completely prevented the decreases in HR secondary to evoked increments in blood pressure induced by PE or ANG II (see Figure 4). This finding shows that the bradycardia was reflex in origin and further shows that neither pressor agent, in the doses used, had any significant direct chronotropic effect. These data are consistent with the reports of Coleman and Gordon et al., who obtained similar results with PE and ANG II. This finding also eliminated the possibility that long-term ethanol feeding might have altered the nonreflex effects of either pressor agent in any way that could have contributed to the observed alteration of baroreceptor reflex control of HR.

Another factor that should be considered is the α-receptor blocking effect of ethanol. This effect should prevent or at least delay the development of hypertension. We have previously shown that short-term administration of ethanol shifts the dose-response curve of PE, but not ANG II, to the right, suggesting an α-receptor blocking effect. After 4 weeks of ethanol feeding this effect was much less evident than in the short-term situation, suggesting that partial tolerance had developed to this effect. In the present study we found that this effect was completely lost after 12 weeks of ethanol feeding, suggesting that complete tolerance had developed. These data show that the α-receptor blocking activity of ethanol is inversely related to the duration of ethanol treatment and that, when present, it should have a negative effect on the development of ethanol-induced hypertension.

The results presented here and in our previous study strongly suggest that impairment of arterial baroreceptors may play a contributory role in the development of ethanol-induced hypertension. However, these conclusions apply to rats; in humans, impaired BRS associated with long-term alcohol consumption and elevated blood pressure remains to be demonstrated.

References
Ethanol-induced hypertension involves impairment of baroreceptors.
A A Abdel-Rahman and W R Wooles

Hypertension. 1987;10:67-73
doi: 10.1161/01.HYP.10.1.67

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/10/1/67