Acute Effects of Ethanol Ingestion on the Response to Submaximal and Maximal Exercise in Man

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SUMMARY

The acute effects of ingestion of ethanol on the response to submaximal and maximal exercise were studied by noninvasive technics in a group of eight healthy men, ages 21 to 33 (series I). Cardiac output (dye-dilution technic) and intra-arterial pressures were measured in a separate series of experiments in a subgroup of four subjects (series II). Mean concentration of ethanol in the blood at the end of the experiment was 156 mg/100 ml in series I and 125 mg/100 ml in series II. Heart rates at rest and during submaximal exercise were higher after ingestion of ethanol, but there was no effect on stroke volume. After ingestion of ethanol cardiac output at rest and during submaximal exercise increased, and total A-V difference and total peripheral resistance decreased. The circulatory response to maximal work was not affected by ethanol. Maximal oxygen uptake did not change. Pulmonary ventilation was not altered during submaximal exercise but was reduced during maximal work.

These findings are in agreement with data from animal experiments suggesting that ethanol in blood concentrations below 200 mg/100 ml has no significant depressive effect on performance of the normal heart.

Additional Indexing Words:
Physical performance capacity  Work time  Mechanical efficiency
Lactate concentration

CLINICAL evidence links excessive intake of alcohol to myocardial disease, but several aspects of the pathogenesis of alcoholic cardiomyopathy remain controversial.\(^5\) Conflicting results have been presented from studies on the acute cardiovascular effects,\(^5\) including the response to exercise.\(^8,^9\) There are no data on the effect of ethanol on maximal oxygen uptake and on cardiac output, stroke volume, and blood pressure during maximal work.

The purpose of the present investigation was to evaluate the acute effects of ethanol concentrations in the blood in the range of 100 to 200 mg/100 ml on the response to submaximal and maximal exercise in normal subjects. Maximal oxygen uptake was used as a measure of the functional capacity of the oxygen transport system, and maximal stroke volume and cardiac output were used as indices of cardiac performance. Total A-V oxygen difference and peripheral resistance were determined to document any occurrence
of gross changes in the regulation of peripheral blood flow.

Methods

Basic data on the eight subjects studied appear in table 1. All were men in good health. Five of the men were medical students, two were laboratory technicians, and one was a physician. They represented a wide range with respect to habitual physical activity and performance capacity with maximal oxygen uptakes between 33 and 56 ml/kg × min. All were moderate users of alcohol.

Procedures

Oxygen uptake was determined by the Douglas bag technic, using a Collins triple J valve. The volume of expired air was measured in a Tissot spirometer. Samples were stored in mercury tonometers and were then analyzed by the Scholander technic. Cardiac output was determined by the dye-dilution technic. Indocyanine green was injected through a 25-cm long catheter placed in an antecubital vein. Arterial blood was drawn from a 15-cm long catheter in the brachial artery at a constant rate of 35.6 ml/min using a Harvard withdrawal-infusion pump. A Beckman cardiopulmonary meter with an automatic integrator was used for recording the dilution curve and for calculating the area under the curve. Arterial pressure was measured by connecting the catheter in the brachial artery to a Statham P23Db strain gauge using semi-rigid polyethylene tubing (internal diameter, 3 mm; length, 50 cm). Pressures were recorded on an Electronics for Medicine DR-8 photographic recorder. Heart rate was obtained from electrocardiographic recordings, utilizing at least 30 R-R intervals. Lactic acid was determined from capillary blood by a micromodification of an enzymatic method. Details on the methods listed above have been presented elsewhere.10 Blood alcohol concentrations were measured according to Kingsley and Current.11 A paired t-test was used for statistical evaluation of the results.

Experimental Plan

Maximal oxygen uptake was defined as the level at which an increase in work load failed to produce an increase in oxygen uptake. This level was established in each subject at trial runs prior to the study. Two series of experiments were then performed.

Series I

All eight subjects participated. Control experiments and studies after the ingestion of ethanol were conducted on separate days within an interval of 1 week. The subjects reported to the laboratory in the morning after a light breakfast and performed exercise on a bicycle ergometer at two submaximal work loads and one maximal work load. Submaximal work load levels were selected that required approximately 50 and 75% of each subject’s maximal oxygen uptake. The duration of work at both submaximal exercise levels was 12 min. Each work load was followed by a rest period of 10 to 15 min. The maximal work load was chosen so that the subject was able to perform for 2½ to 5 min. The maximal run was preceded by a 2-min warm-up period at the 75% level. Duplicate measurements of oxygen uptake and heart rate were made during the last minute of exercise at each level. Two blood samples for determination of lactic acid were drawn 1 to 3 min after completion of maximal work. The exercise protocol was identical for the control and ethanol experiments. Ethanol was administered in the form of 150 ml of 86 proof whiskey, rum, or gin, during a 20-min period starting 60 min before exercise at the initial work load. Samples for determination of the blood concentration of ethanol were drawn before exercise started and immediately after maximal work.

Series II

Four of the subjects took part in a second series of experiments including hemodynamic measurements. The interval between individual studies in series I and II was less than 1 mo. The experiments were begun in the morning. Venous and arterial polyethylene catheters were introduced percutaneously. The subject then rested in supine position for 1 hour. Duplicate measurements of oxygen uptake, heart rate, cardiac output, and intra-arterial pressures were then made with the subject resting in sitting position on the bicycle ergometer. All measurements were

Table 1

Basic Data on Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Maximal oxygen uptake (ml/kg × min)</th>
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<td>173</td>
<td>73</td>
<td>33</td>
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</table>

*Indicates subject participating also in series II experiments including measurement of cardiac output and intra-arterial pressures.
repeated during the last minute of exercise at two submaximal and one maximal work load levels as described in series I. Samples for determination of lactate concentration after maximal work were drawn as above. Completion of the control study was followed by another 1-hour period of rest in the supine position. Ethanol was then administered, and samples for determination of blood levels were drawn in the same manner as in series I. The sequence, including measurements at rest and during two levels of submaximal and one level of maximal exercise, was repeated. The interval between completion of maximal work during the control study and the beginning of exercise at the first submaximal load after alcohol intake was 2 hours.

Results

Mean data from series I are presented in table 2 and individual data recorded during maximal work in figure 1. Individual and mean data from series II appear in table 3. Circulatory findings during submaximal and maximal work are displayed in figure 2.

Mean ethanol concentration in series I was 165 mg/100 ml before exercise started and 156 mg/100 ml immediately after completion of maximal work (range, 125 to 200 mg/100 ml). Corresponding figures in series II were 134 and 125 mg/100 ml (range, 89 to 168 mg/100 ml).

In both series oxygen uptake at rest and during submaximal exercise at corresponding work loads was 0.05 to 0.15 L/min higher after administration of alcohol than during the control study, that is, mechanical efficiency was lower after alcohol. Maximal oxygen uptake did not change significantly. Mean values in series I were 3.26 L/min at the control study and 3.14 after alcohol, and in series II, 2.85 and 2.79 L/min.

![Effect of ingestion of ethanol on the response to maximal work, series I. Individual data, n = 8.](http://circ.ahajournals.org/content/465/f1)

Significant differences were present only with respect to ventilation (P < 0.001).
Mean Data, Series I (Eight Subjects)

<table>
<thead>
<tr>
<th></th>
<th>Blood ethanol (mg/100 ml)</th>
<th>Work load (kpm/min)</th>
<th>Oxygen uptake (L/min)</th>
<th>Heart rate (beats/min)</th>
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<td>Control</td>
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<td>1.39 ± 0.21</td>
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<td>&lt;0.40</td>
<td>&lt;0.001</td>
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<tr>
<td>Maximal exercise</td>
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<td></td>
</tr>
<tr>
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<td>3.26 ± 0.38</td>
<td>193.6 ± 2.1</td>
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<tr>
<td>Ethanol</td>
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<td>1472 ± 124</td>
<td>3.14 ± 0.38</td>
<td>193.3 ± 2.2</td>
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<tr>
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<td>&lt;0.80</td>
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</table>

*Ventilatory exchange ratio.

Heart rates at rest and during submaximal exercise were significantly higher after alcohol in both series. The difference decreased with increasing work loads. Heart rates during maximal exercise were the same before and after alcohol ingestion. Alcohol had no effect on the stroke volume (fig. 2) at rest or during exercise. Thus, the higher heart rate at rest and during submaximal exercise after alcohol ingestion was associated with increased cardiac output (fig. 2). The increase in cardiac output at levels below maximal exercise was in excess of the increase in oxygen uptake. The arteriovenous oxygen difference (calculated from measurements of cardiac output and oxygen uptake) was smaller both at rest and during exercise at the lowest of the two submaximal work loads, but the differences were not significant. Mean cardiac output during maximal work in the alcohol experiment equaled the control value of 20.6 L/min.

Mean arterial pressure was slightly lower both at rest and during submaximal exercise during the ethanol runs. Total peripheral resistance fell markedly (fig. 2) since the decrease in pressure was associated with an increase in cardiac output. The difference in peripheral resistance was significant at both submaximal levels (P < 0.02). Ethanol had no effect on mean arterial pressure or peripheral resistance during maximal work.

Ethanol did not affect pulmonary ventilation at rest and during submaximal exercise but caused a significant reduction during maximal work from 132 to 115 L/min in series I (P < 0.001). A decrease of the same magnitude was observed in series II. The respiratory rate did not change. Thus, tidal volumes were unaffected at rest and during submaximal work but decreased during maximal work.

There were no significant differences in ventilatory exchange ratio (RQ) at rest and during submaximal exercise. The reduction in ventilation during the maximal work load of the ethanol experiment was associated with a significantly lower (P < 0.001) value than at the control experiment in series I, but RQ was still above 1.00. Peak lactate concentration after maximal exercise did not change. Work time at the maximal level was slightly shorter at the ethanol run, but the difference was not significant.

Discussion

Ingestion of ethanol had significant circulatory effects both at rest and during submaximal exercise but not during exercise at the maximal level. In contrast, ingestion of ethanol had significant effects on ventilation.
ETHANOL INGESTION DURING EXERCISE

<table>
<thead>
<tr>
<th>Pulmonary ventilation (L/min, BTPS)</th>
<th>Respiratory rate (breaths/min)</th>
<th>Tidal volume (L)</th>
<th>RQ*</th>
<th>Blood lactate (mm/L)</th>
<th>Work time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>39.7 ± 6.0</td>
<td>21.6 ± 0.8</td>
<td>1.73 ± 0.26</td>
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<td>12.0</td>
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<tr>
<td>&lt;0.70</td>
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<td>&lt;0.90</td>
<td>&lt;0.60</td>
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<td>81.5 ± 8.8</td>
<td>35.8 ± 3.3</td>
<td>2.26 ± 0.21</td>
<td>0.97 ± 0.01</td>
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<td>79.5 ± 6.5</td>
<td>38.8 ± 2.6</td>
<td>2.15 ± 0.20</td>
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<td>&lt;0.70</td>
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<td>&lt;0.50</td>
<td>&lt;0.20</td>
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<td>—</td>
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<tr>
<td>132.5 ± 6.0</td>
<td>52.3 ± 1.1</td>
<td>2.55 ± 0.10</td>
<td>1.22 ± 0.02</td>
<td>13.5 ± 0.5</td>
<td>3.7 ± 0.3</td>
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<td>114.7 ± 5.7</td>
<td>52.9 ± 1.7</td>
<td>2.21 ± 0.12</td>
<td>1.07 ± 0.02</td>
<td>13.6 ± 0.5</td>
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<tr>
<td>&lt;0.001</td>
<td>&lt;0.90</td>
<td>&lt;0.005</td>
<td>&lt;0.001</td>
<td>&lt;0.90</td>
<td>&lt;0.20</td>
</tr>
</tbody>
</table>

during maximal work only. Maximal oxygen uptake did not change.

Series I, the larger series of subjects studied only with noninvasive technics, served two purposes: (1) extended the number of observations on the effect of ethanol on oxygen uptake, heart rate, and ventilation, and (2) provided information needed to evaluate to what extent the experimental design might have affected the results during the alcohol run in series II. The alcohol studies in series II were preceded by exercise at two submaximal and one maximal work loads during the control experiment. Hartley and Saltin\textsuperscript{12} have reported a relative increase in heart rate and a decrease in stroke volume during light submaximal work if preceded by exercise at a heavy work load. However, stroke volumes in the control and alcohol experiments in our series were identical. Systematic differences between series I and II data for the four subjects who participated in both series were observed only with respect to heart rate during submaximal exercise. Heart rates were, as expected,\textsuperscript{10} slightly higher during submaximal work with catheters in place. The average increase during the control experiment was 5.5 beats/min. The effect of alcohol on heart rates during submaximal work was similar in both series with an average increase above control levels of 11.9 beats/min in series I compared to 13.8 beats/min in series II. Thus, it seems unlikely that the experimental design in series II introduced any significant bias.

The fact that maximal cardiac output and stroke volume did not change with exercise strongly suggests that ethanol caused no significant acute depression of cardiac performance. However, the possibility that the unchanged maximal cardiac output and stroke volume were achieved at the expense of increased ventricular end-diastolic volumes and pressures cannot be ruled out. The data from the only previous study including hemodynamic measurements at rest and during exercise in normal subjects\textsuperscript{9} and results from carefully controlled animal experiments are consistent with little or no effect on myocardial function at blood levels below 200 mg/100 ml.

Riff and associates\textsuperscript{9} found that blood concentrations of ethanol between 85 and 136 mg/100 ml in normal subjects had no effect on stroke volume during submaximal exercise. Mierzwiaik and associates\textsuperscript{5} used a dog preparation in which cardiac output, heart rate, and aortic pressure were controlled. They found no significant change in left ventricular function at an ethanol blood level of 100 mg/100 ml but did demonstrate a depression at 300 mg/100 ml. A progressive depression with increasing blood concentrations of ethanol above 200 mg/100 ml has been reported.
by others.\textsuperscript{2,3,6} However, Webb and associates\textsuperscript{4} in a later study found no depressive effects at blood concentrations as high as 900 mg/100 ml.

Conway\textsuperscript{8} on the other hand, reported a decrease in cardiac output and stroke volume during mild exercise at ethanol concentrations of 45 to 49 mg/100 ml in eight patients with angina. The patients showed a more prominent ECG change after alcohol administration despite a decrease in tension-time index compared to the control study. Regan and associates\textsuperscript{7} demonstrated a decrease in stroke output, an increase in end-diastolic pressure at rest, and an abnormal response to an induced increase in aortic pressure with blood levels of about 150 mg/100 ml in a series of eight alcoholics with fatty liver but no clinical evidence of a heart disease. It is possible that the alcoholic patients studied by Regan and associates had some degree of myocardial damage and that the depressive effect of ethanol is apparent at lower concentrations in patients with myocardial disease than in normal subjects.

The circulatory effects of ethanol that were observed at submaximal work load levels in the present study, that is, an increase in cardiac output, a reduction in A-V difference, and a decrease in arterial blood pressure and peripheral resistance, are compatible with increased blood flow to nonactive tissue, presumably skin. An ethanol-induced increase in skin flow at rest and during submaximal exercise may be associated with a decrease in flow to nonactive muscle.\textsuperscript{13,14}

Both series showed a significant depressive effect on ventilation during maximal work. The absence of any changes in maximal oxygen uptake indicates that the relative hypoventilation induced by ethanol had no significant effect on oxygen transport.

The ability to perform physical work on the bicycle ergometer was only affected to a small extent. The expected ethanol effect on neuromuscular performance\textsuperscript{15} was reflected by a small increase in oxygen uptake at submaximal loads. Work time was also slightly shorter at the maximal level in the absence of any changes in oxygen uptake or lactate levels. The difference was not significant but was of the same order of magnitude as that reported by Asmussen and Bøje\textsuperscript{16} who measured the time required to perform a given amount of heavy work before and after ethanol.

The pattern of response observed in this study, that is, an intervention resulting in a modified circulatory response to submaximal work loads but an essentially normal response to a maximal load, is by no means unique to ethanol. Comparable findings have been reported from experiments on pyrogen-induced fever\textsuperscript{17} and work in a moderately hot environment.\textsuperscript{18} Another example of the normalizing effect of a maximal effort is found in

\hspace{1cm}Figure 2

*Individual and mean circulatory data at rest, and during submaximal and maximal exercise, series II.*
ETHANOL INGESTION DURING EXERCISE

Table 3

<table>
<thead>
<tr>
<th>Subject</th>
<th>Condition*</th>
<th>Blood ethanol conc. (mg/100 ml)</th>
<th>Oxygen uptake (L/min STPD)</th>
<th>Pulmonary ventilation (L/min BTPS)</th>
<th>Heart rate (beats/min)</th>
<th>Cardiac output (L/min)</th>
<th>Mean arterial pressure (mm Hg)</th>
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<td>&lt;0.98</td>
<td>&lt;0.90</td>
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</table>

*C = control; A = alcohol experiments; numbers under "Condition" refer to work load in kpm/min.

dehydration. Loss of as much as 20% of the extracellular fluid volume is associated with an increased heart rate, a decreased stroke volume at submaximal loads, and a decreased physical work capacity, but the circulatory response during maximal work is normal.19

Atropine has a significant effect on the cardiovascular response during exercise at

*Circulation, Volume XLII, September 1970
submaximal levels but has no apparent effect at the maximal level.\textsuperscript{20} By contrast, beta-
adrenergic blockade significantly alters the
response to both submaximal and maximal
exercise and causes a decrease in maximal
oxygen uptake.\textsuperscript{21} The cardiovascular response
pattern during maximal exercise of short
duration may largely be determined by a
balance between maximal rate of sympathetic
discharge and local metabolic vasodilatation.
Such a combination would explain a stereotypic response pattern characterized by
neutralization of agents and conditions interfering
with the regulation of circulation during work
at lower intensities, and perhaps also by
activation of compensatory mechanisms that
are not evoked at submaximal levels.

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