BLOOD ALCOHOL CONCENTRATIONS AS AFFECTED BY COMBINATIONS OF ALCOHOLIC BEVERAGE DOSAGES AND ALTITUDES

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April 1970

Department of Transportation
FEDERAL AVIATION ADMINISTRATION
Office of Aviation Medicine
ACKNOWLEDGMENTS

The authors thank the physicians of CAMI for providing medical coverage during the chamber flights, and to the personnel of the Physiological Operations and Training Section for operating the altitude chamber. We also are grateful to Mr. Floyd Passmore for his technical assistance and to Mr. William V. Flores for the technical illustrations of this report. We also thank Dr. P. F. Iampietro for his review of the manuscript and helpful suggestions.
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I. Introduction.

In an earlier study human subjects ingesting 2.50 ml. of 100 proof bourbon/kg. body weight had a significantly higher blood alcohol level at 20,000 ft. than those ingesting this amount of alcohol at 12,000 ft. or at ground level. The altitude-related difference was not apparent with a lower dose of alcohol (1.25 ml. of 100 proof bourbon/kg. body weight).

This report includes the results from subsequent experiments designed to: (1) Verify the altitude-dose interaction; (2) examine the possibility that the principle mechanism involves dehydration; and (3) determine if breathing oxygen was a factor.

II. Methods.

Sixty male paid volunteer subjects, 21 to 31 years old (mean age, 23 years), were divided into three groups. One of the two groups exposed to 12,000 ft. breathed ambient air; the other group was given a normal oxygen mixture by a demand-type regulator system. The third group was exposed to 20,000 ft. and also breathed the normal oxygen mixture.

In each experiment, two to four subjects were tested for 3 hours during which time physiological measurements were made at intervals (see Table I).

Table I—Schedule of events

A. Subjects report to Employee Health Clinic at 8:30 a.m. (no breakfast):

Execution of forms.
Physical examination by flight surgeon.
Consumption of 8 oz. milk and two slices of dry toast.

B. In Laboratory:

Pre-exposure nude body weight.

C. In Altitude Chamber:

- 30 minutes: measurement of oxygen saturation and heart rate, draw 5 ml. blood sample.
- 30 minutes to time zero: consumption of alcoholic beverage over 30 minutes (½ per 5 minutes).
- 0 minutes: ascend to altitude.
- + 30 minutes: repeat above measurements, blood sample.
- + 60 minutes: repeat above measurements, blood sample.
- +120 minutes: repeat above measurements, blood sample.
- +180 minutes: repeat above measurements, blood sample.

End of exposure to altitude.

D. In Laboratory:

Post-exposure urine collection followed by nude body weight.

Blood oxygen saturation was determined by a Waters (Model XE-350) ear oximeter and heart rate was obtained by counting radial artery pulses.

Nude body weights were taken before and after the altitude exposure on a 125 kg. balance, sensitive to 10 gm.; corrections were made for fluid intake and urine excretion (see Table II).

A 5-ml. sample of venous blood was drawn in a heparinized syringe. Hematocrit was measured by a microcapillary centrifuge and reader. The remainder of the blood was mixed with desiccated sodium and ammonium oxalate, sealed and stored in a refrigerator (0.6 to 3.3°C). Blood alcohol levels were measured in these stored samples by the automated method of Syed.7

Twenty-four of the subjects (eight per condition) received 1.25 ml./kg. body weight (low dose) and the remaining 36 men (12 per condition) each received 2.50 ml./kg. (high dose) of
Table II.—Changes in body weight for all conditions

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Pre-exposure weight</th>
<th>Fluid intake</th>
<th>Urine output</th>
<th>Post-exposure weight</th>
<th>Change in body weight (corrected)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(kg.)</td>
<td>(kg.)</td>
<td>(g./kg.)</td>
<td>(kg.)</td>
<td>(g./kg.)</td>
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<tr>
<td>Low Dose—</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>12,000 ft. without sup. O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>72.354 ± 2.160</td>
<td>0.601 ± 0.005</td>
<td>8.361 ± 0.266</td>
<td>0.709 ± 0.092</td>
<td>10.010 ± 1.397</td>
</tr>
<tr>
<td>12,000 ft. with supp. O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>74.686 ± 4.269</td>
<td>0.610 ± 0.006</td>
<td>8.338 ± 0.422</td>
<td>0.454 ± 0.089</td>
<td>5.904 ± 0.812</td>
</tr>
<tr>
<td>20,000 ft. with supp. O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>68.758 ± 4.938</td>
<td>0.602 ± 0.002</td>
<td>9.075 ± 0.634</td>
<td>0.580 ± 0.114</td>
<td>8.786 ± 1.698</td>
</tr>
<tr>
<td>High Dose—</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>12,000 ft. without sup. O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>72.371 ± 2.259</td>
<td>0.595 ± 0.008</td>
<td>8.301 ± 0.252</td>
<td>1.148 ± 0.117</td>
<td>16.085 ± 1.724</td>
</tr>
<tr>
<td>12,000 ft. with supp. O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>72.766 ± 2.997</td>
<td>0.596 ± 0.005</td>
<td>8.342 ± 0.348</td>
<td>1.095 ± 0.069</td>
<td>15.135 ± 0.858</td>
</tr>
<tr>
<td>20,000 ft. with supp. O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>72.021 ± 2.594</td>
<td>0.602 ± 0.008</td>
<td>8.479 ± 0.320</td>
<td>0.889 ± 0.107</td>
<td>13.083 ± 1.675</td>
</tr>
</tbody>
</table>

*Reported as means ± standard errors.

100 proof bourbon.* Each dose was mixed with a cola to a total volume of about 600 ml.

Data from repeated measures were subjected to an analysis of variance (repeated measures technique) and the Student multiple range test.* Other data were subjected to “t” tests.

III. Results.

Blood alcohol concentration at both dose levels showed significant differences with time (P ≤ .01); peak values were attained between 30 and 60 minutes after ingestion (Figure 1, A & B). Differences attributable to cabin conditions were evident only at the high dose (P ≤ .05).

Changes in hematocrit at the two alcohol levels are shown in Figure 2, A & B. There was a significant increase in hematocrit with time in each case (P ≤ .01); generally, a maximum level was attained in or near the second hour. Hematocrits did not vary significantly with cabin condition, nor did they demonstrate any significant interactions.

At both alcohol levels time significantly influenced heart rate (P ≤ .01) (Figure 3, A & B). Maximum heart rates occurred at about 60 minutes, after which they gradually declined during the remaining 120 minutes.

Blood oxygen saturation measurements (Figure 4, A & B) demonstrated significant effects for both time and cabin condition as well as for the interaction between the two (P ≤ .01).

Subjects receiving the high alcohol dose had greater urine volumes than those receiving the low dose (P ≤ .01). Mean volume with the high dose was 1,044 ml.; mean volume with the low dose was 581 ml. Body weight change, corrected for fluid intake and urine loss, was not significantly affected by altitude (Table II).

IV. Discussion.

By Analysis of Variance the data revealed that a significant difference existed when considering the effect of cabin condition on blood alcohol con-
Figure 1. Blood alcohol concentration expressed as mg % as a function of time. Low dose, n=8; High dose, n=12. Means ± SE.
Figure 2. Hematocrit expressed as a function of time. Low dose, n=8; High dose, n=12. Means ± SE.
Heart rate expressed in beats per minute as a function of time. Low dose, n=8; High dose n=12. Means ± SE.
Figure 4. Blood oxygen saturation expressed in per cent as a function of time. Low dose, n=8; High dose, n=12. Means ± SE.
centration for the high alcohol dose ($P \leq .05$). Using the Student Multiple Range test, this study did verify the finding of the previous study\(^2\) that blood alcohol concentrations were significantly higher for subjects at 20,000 ft. than for those at 12,000 ft. without supplemental oxygen ($P \leq .05$).

McFarland and Forbes\(^3\) also have reported that the concentration of alcohol in the blood rose more rapidly and reached a higher level at high altitudes than at sea level.

Results for the two 12,000 ft. altitude conditions did not approach significance for difference. Although blood alcohol concentrations at 20,000 ft. (high dose) were significantly higher than those found at 12,000 ft. without supplemental oxygen, the statistical comparison between the 20,000 ft. values and those for 12,000 ft. with supplemental oxygen was not significant ($P \leq .10$). Therefore, the effects of breathing oxygen on blood alcohol concentrations cannot be ascertained from the data obtained in this study.

It was considered that dehydration would influence the findings because alcohol enhances diuresis and Miller\(^4\) reported an increased human body water loss at reduced ambient pressures. If the diuretic loss were much greater with the high dose of alcohol, then an additional water loss due to reduced ambient pressure might prove to be of greater significance for the high dose than for the low dose.

Although hematocrit changes with time were very small, they were significant ($P \leq .01$). Lowest values were for the control samples with peak values at 2 hours. Subjects receiving the high dose of alcohol had mean hematocrit values increasing from 46.0 to 47.8. Mean hematocrit values for low-dose subjects were 45.6 to 46.9. These were increases of 1.8 and 1.3 respectively, which might reflect the diuretic effect of the alcohol demonstrated by the urine volumes. Although individual variation was quite large, those receiving the high alcohol dose produced statistically higher urine volumes. Using Miller's method for estimating evaporative water loss, the losses in our subjects would be 186.7 gm./hr. and 195.6 gm./hr. for 12,000 ft. and 20,000 ft. respectively. This small difference of less than 10 gm./hr. would not be sufficient to account for the difference in blood alcohol concentration. None of the measured corrected weight losses were as high as would have been predicted by Miller's method. There were no significant differences for weight loss. It does not appear, from the data collected in this study, that differences in dehydration levels can account for the observed phenomenon.

There are other possible explanations for the blood alcohol concentration findings which, however, could not be tested with this experimental design. A reduced rate of metabolism at 20,000 ft. could explain the findings for the high dose of alcohol. If this were the case, however, one would expect a similar finding for the low dose. Furthermore, alcohol oxidation (in a healthy individual) appears to be fairly constant. Many attempts have been made to alter the rate of metabolism of alcohol (e.g., administration of thyroid extract, inhalation of oxygen, and exercise) without significant results. Once peak blood alcohol levels were attained, the rate of decline of blood alcohol levels at 20,000 ft. was quite similar to the rate of decline at lower altitudes. Therefore, a reduced rate of alcohol metabolism at 20,000 ft. does not appear to be a logical explanation.

Another explanation might be a greater absorption rate at 20,000 ft. facilitated by the high dose of alcohol. A greater absorption rate at 20,000 ft. might be considered possible because of a reported hypermotility of the intestinal tract at lowered ambient pressures.\(^1\) The increase in motility appears to be related to the expansion of gases contained in the gastrointestinal tract. Because these gases are saturated with water vapor they do not expand in exact proportion to the decrease of barometric pressure. The pressure changes from ground level (just over 1,200 ft.) to 12,000 ft. and to 20,000 ft. may be expressed as: (A) 727-47/484-47 or 1.55, and (B) 727-47/350-47 or 2.25. Gas expansion at 20,000 ft. is almost one and one-half times as great as at 12,000 ft. At slow rates of ascent (less than 300 ft./min.) the distention persists for an hour or two when the gas volume again becomes approximately equal to ground level. At higher rates of ascent (1,000 ft./min. or more), as in this study, gas tends to become more localized in pockets in the intestinal loop instead of being passed off. Under these conditions hypermotility probably persists even longer.
Hulpieu and Harger report that alcohol may serve as a stimulant for motility of the stomach and intestines. Alcohol concentrations (in the ingested drink) below 10% have little effect on the motility of the stomach and intestines but concentrations above 15% are definitely irritating. As presented to our subjects (average weight near 72 kg.) the average high dose of alcohol was approximately 15.8% and the average low dose was approximately 7.9%. It is possible, therefore, that increased motility caused by the alcohol concentration combined with the increased motility attributed to the lowered barometric pressure were responsible for the significant increase in blood alcohol level at 20,000 ft. for the high dose but not for the low dose.

Additional studies which would include intermediate altitudes, with and without supplemental oxygen, and the administration of the high dose of alcohol in a more dilute drink concentration (less than 10%) could further clarify these findings. However, none are planned at this time.

V. Summary.

This study established blood alcohol levels in man at 12,000 ft. with and without supplemental oxygen and at 20,000 ft. with supplemental oxygen. At 2.50 ml. of 100 proof bourbon/kg. body weight, subjects exhibited a lower blood alcohol level at 12,000 ft. without supplemental oxygen than at 20,000 ft. with supplemental oxygen. A difference in blood levels was not seen with 1.25 ml. of 100 proof bourbon/kg. body weight.

It was established that dehydration effects alone could not account for these findings.

The effect of breathing a normal oxygen mixture could not be ascertained with the data collected.

An increased motility of the gastrointestinal tract caused by the high alcohol concentration and the increased motility attributable to the lowered barometric pressure could increase the absorption rate of the alcohol at 20,000 ft. with the high dose, thereby contributing to higher blood alcohol levels.

REFERENCES


