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Combined effects of ethanol and hyperbaric air on body sway and heart rate in man

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Jones, A. W., R. D. Jennings, J. Adolfson, and C. M. Hesser. 1979. Combined effects of ethanol and hyperbaric air on body sway and heart rate in man. Undersea Biomed. Res. 6(1): 15–25.— Eight amateur divers took part in crossover experiments to study the combined effects of ethanol (0.72 g/kg b.wt.) and hyperbaric air (4 and 6 ATA) on heart rate and body-sway movements. Body sway with open and closed eyes was measured in lateral and sagittal directions by a statometer device. In the alcohol condition, there was an initial increase in body sway corresponding to the acute phase of ethanol intoxication. At a mean blood alcohol concentration of 0.77 mg/ml, this increase in body sway was statistically significant ($P < 0.01$) compared with the alcohol-free condition. At 90 min from start of drinking, body sway scores at 1 ATA were not significantly different from alcohol-free measurements. On raising the pressure to 4 and 6 ATA, increased body sway occurred in both alcohol and alcohol-free conditions and, moreover, the rate of increase was more extreme in the alcohol condition. Significant pressure-alcohol interactions were established, suggesting a potentiating action of alcohol on the increase in body sway induced by acute exposure to high pressures of air. Heart-rate measurements with and without alcohol were not significantly different, although increases in ambient pressure caused a drop in heart rate in both conditions.

alcohol	hyperbaric air
ethanol	interaction
impairment	performance
body sway	heart rate

There is a certain similarity between the signs and symptoms of ethanol intoxication and those of inert gas narcosis. Whereas changes in performance and behavior under the influence of ethanol (for reviews, see Goldberg 1966; Kissin and Begleiter 1972) and of a hyperbaric air environment (for reviews, see Adolfson and Berghage 1974; Bennett 1975) have been extensively studied separately, nothing is known about the combined action of these influences.

A sensitive test for both ethanol and hyperbaric air intoxication is the evaluation of subjects' recorded body-sway movements (Adolfson, Goldberg, and Berghage 1972; Franks, Hensley, Hensley, Starmer, and Teo 1976). In both conditions, the involuntary body-sway motions increase with the degree of impairment.

The aim of the present investigation was to study the possible interaction between inert gas narcosis and ethanol intoxication. This was done by measuring changes in body sway and heart rate induced by acute exposure to raised air pressures and intake of a moderate dose of alcohol.

MATERIAL AND METHODS

Subjects and conditions

Eight male subjects, all amateur divers, took part in the experiments. They had a mean age of 31 (range 26–42) years, mean body weight of 69 (range 69–76) kg, and mean height of 174 (range 167–179) cm. They were all accustomed to moderate drinking. In the present study whisky was given in a standard dose of 2 ml/kg b.wt., equivalent to 0.72 g absolute alcohol per kg b.wt. The calculated volume was consumed neat, within a 20-min period, after the subjects had fasted 10 to 12 h.

All experiments were carried out in a dry compression chamber at normal or at raised air pressures. For comfort and to preclude inhalation of CO_2-enriched chamber air, the subjects inspired air saturated with water from a 200-liter Douglas bag via a mouthpiece, a low dead-space breathing valve, and flexible large-caliber tubing. Chamber temperature was controlled at about 23°C by means of a fan and heat exchanger.

Blood sampling and ethanol analysis

Capillary blood samples were taken from a fingertip at 30, 60, 90, 108, 128, 156, 180, 240, 300, and 360 min from start of drinking. The samples were taken in triplicate, using 10-μl disposable blood pipettes, and immediately diluted with 1 ml of 0.2 M sodium fluoride solution in Auto Analyzer cups. The cups used when blood samples were taken under pressure had pin holes in their caps to avoid difficulties caused by pressurization of the air space above the liquid. Preliminary experiments with standard alcohol solutions showed that no loss of ethanol occurred during the time under raised pressure. The blood alcohol concentration (BAC) was determined by an automated enzymatic alcohol dehydrogenase technique. The standard deviation of a single determination with this method of analysis is $\pm$ 0.012 mg/ml at a mean ethanol concentration of 0.51 mg/ml (Buijten 1975).

Subjective estimates of intoxication

The degree of subjective intoxication was assessed by a magnitude estimation technique (Ekman, Frankenhaeuser, Goldberg, Bjerver, Järpe, and Myrsten 1963). A subjective feeling of mild intoxication, to the degree of being "a little high" (tipsy), was arbitrarily assigned a value of 10, a higher or lower level of intoxication being given a number greater or less than 10, respectively. On this scale, a completely normal (sober) condition would be estimated as zero. The subjects were required to estimate their degree of intoxication based on this scale at 30- to 60-min intervals throughout the experiments in both alcohol and sober conditions. Subjects with estimates of zero were not included when calculating group averages for the two conditions (compare Ekman, Frankenhaeuser, Goldberg, Hagdahl, and Myrsten 1964).

Measurements of body sway and heart rate

Body sway was quantitatively measured by statometry (Adolfson, Bjerver, Fluur, and Goldberg 1974). With the subject standing on the statometer platform in a Romberg position,

recordings were made over two 70-s periods. The first recording was made with open eyes, the subject looking directly ahead with his gaze fixed on a distant mark at eye level. After a 30-s rest period, the second recording was made with closed eyes. The parameter used for evaluation purposes was the mean amplitude of sway. This was calculated from the body-sway recordings stored on magnetic tape after analog-to-digital signal conversion. The digital impulses were counted once per second for 60-s periods. Mean amplitudes of lateral and sagittal sway movements with both open and closed eyes were used as dependent variables.

Heart rate was recorded beat-by-beat from chest electrodes and a linear cardiotachometer (Lindborg, Wigertz, and Ödman 1969) and computed as time-averages over each period of body-sway measurement by means of an integrating voltmeter.

Experimental procedure

Each subject served in two experimental sessions, using a randomized cross-over design. Two subjects were tested on each occasion; one subject was alcohol-free, the other had consumed alcohol. On their return visit, 3 to 7 days later, the conditions were reversed.

After a medical examination on the morning of the experimental day, the subjects performed preliminary practice tests on the balance stand to reduce possible effects of training. The experiments started at 8:30 a.m. with control statometer recordings, two eyes-open and two eyes-closed runs. Alcohol was given at 9:00 a.m. The first blood sampling and post-alcohol body-sway recording were made at 9:30 a.m. The subject in the alcohol condition was tested first at all times. The statometry recording was made immediately after each blood sampling.

The response of the subjects to specific instructions, e.g., eyes open or eyes closed, was monitored from outside the chamber on a television screen. The air pressure was increased to 4 ATA at a rate of 1.5 atm/min. This pressure was maintained for 20 min, followed by a further increase to 6 ATA that was held for another 20 min. The decompression was carried out in stages involving stops at 12, 9, 6, and 3 meters, corresponding to a total decompression time of 110 min. Blood sampling and body-sway recordings were made on reaching 4 and 6 ATA, and also during decompression stops. Two final blood samples and recordings of body sway were taken on reaching atmospheric pressure. The blood-sampling times and pressure profile in relation to start of drinking are shown in Fig. 1C.

Statistical analysis

The mean amplitudes of sway at 1 ATA, immediately before the pressure was raised, and at 4 and 6 ATA for both alcohol and alcohol-free conditions, have been evaluated by the three-way analysis of variance (Brownlee 1965). The conditions alcohol and alcohol-free, and the pressures 1, 4, and 6 ATA served as fixed effects, and the eight subjects as random effects. Group means and variabilities were calculated by standard formulas, and the significance of intra-individual mean differences was tested with the Student t-test.

RESULTS

Blood alcohol concentration

The mean values for BAC obtained at successive intervals are shown in Fig. 1A. Very soon after drinking, BAC rises rapidly because alcohol is being absorbed from the stomach and intestines and reaches a maximum level between 30 and 60 min from start of drinking, corre-

sponding to the distribution of alcohol into the body water compartment. After 90 min, BAC gradually decreases as a result of biotransformation by enzymatic oxidation occurring primarily in the liver (Kalant 1971). In the present study BAC reached a peak value of 0.77 mg/ml 60 min from start of drinking, and decreased to 0.73 mg/ml immediately prior to the first increase in ambient pressure. The alcohol was eliminated in about 420 min, corresponding to a turnover of 0.103 g/kg/h, in good agreement with previous work on ethanol metabolism after the same dose of alcohol (Goldberg 1943).

Subjective estimates

The mean time course of subjective intoxication is shown in Fig. 1B. The maximum degree of intoxication occurred 60 min from start of drinking, i.e., when BAC attained its peak level. The mean subjective estimate was 7, and the range was from 3 to 11, indicating the wide variation of subjective intoxication in different subjects even after intake of the same standard dose of alcohol (0.72 g/kg b.wt.). Only 2 subjects exceeded the feeling "a little high" (estimate = 10) on this dose; they both estimated a score of 11.

The curve of subjective intoxication followed closely the blood alcohol curve, with two notable exceptions. The first exception occurred when the pressure reached 6 ATA and the curve of subjective intoxication increased and deviated from the previous downward trend. On beginning the decompression, the feeling of intoxication rapidly dropped off, the curve returning to a downward path. Second, the subjects considered themselves normal, i.e., sober, 360 min from start of drinking, when BAC averaged 0.13 mg/ml.

In the alcohol-free condition, three subjects experienced mild symptoms of intoxication when the pressure was raised to 4 ATA, the mean score being 1.7. A further increase in pressure to 6 ATA produced symptoms of intoxication in 6 subjects; the mean score was 2.7.

Body sway movements

Standing steadiness (body sway) scores were worked out for each subject in both lateral and sagittal directions, and with open and closed eyes. In the control tests (pretreatment recordings) body sway scores were greater in the sagittal than in the lateral directions ($P < 0.05$), suggesting that directional differences in sway exist and were also greater with eyes closed than with eyes open ($P < 0.001$), pointing to the importance of visual cues for maintaining balance (compare Adolfson et al. 1972).

To allow for the large interindividual variations in mean amplitude of sway in the control recordings, changes in body sway scores were computed as percentages of the pretreatment scores when evaluating the mean time course of balance disturbances induced by alcohol and/or pressure (Fig. 2). On this scale, a figure greater than 100 implies an increase in body sway (impairment), and a figure less than 100 implies a decrease in sway (improvement).

Alcohol-free runs

In the alcohol-free condition, there was an initial improvement in body sway (Fig. 2), probably due to a prolonged training effect. The mean body sway later became more or less constant until the pressure was raised to 4 ATA, which caused an increase in body sway. On raising the pressure to 6 ATA, a further increase in sway was noted.

On reducing the pressure, there was a rapid recovery in all body sway measurements, and at 1.9 ATA the body sway scores had returned to prepressure levels. Subsequent recordings

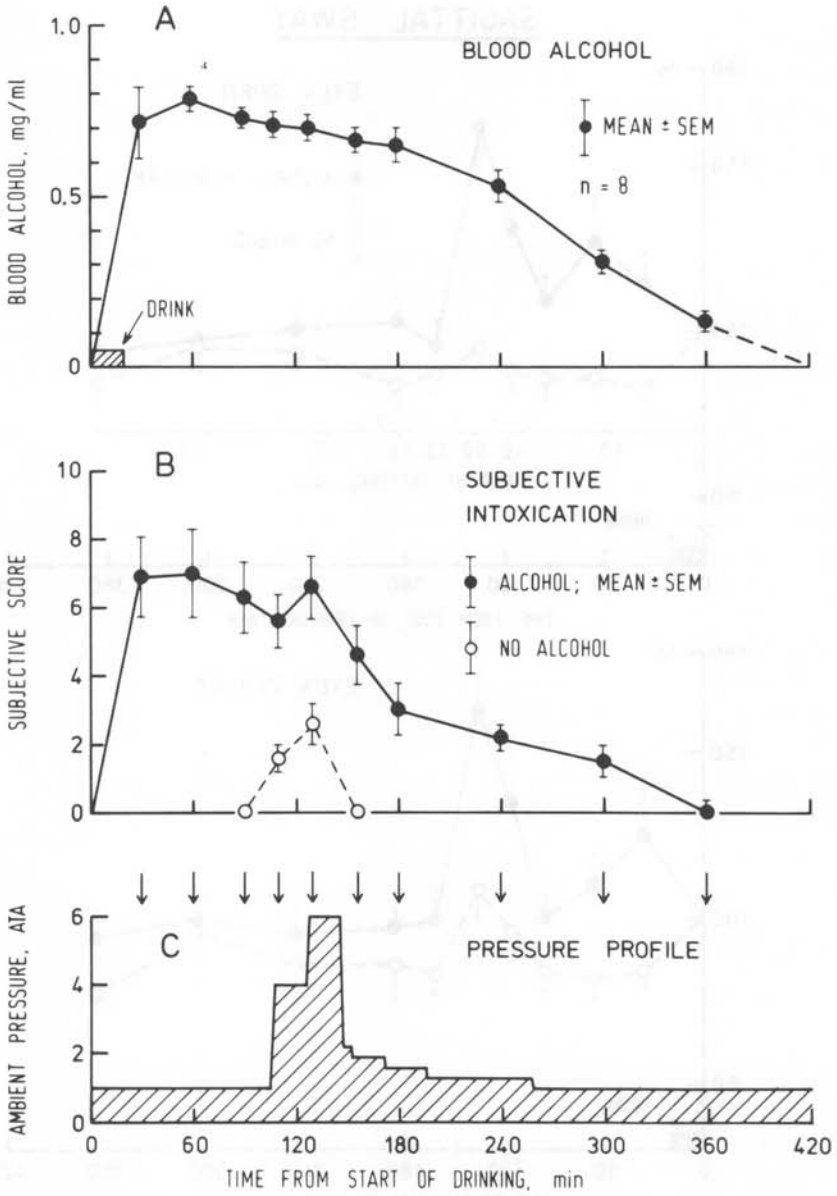

Fig. 1. Changes in blood alcohol concentration (*A*) and subjective evaluation of intoxication (*B*) after intake of moderate dose of alcohol (0.72 g/kg b.wt.) and changes in ambient air pressure (*C*). Arrows in *C* indicate blood-sampling times.

were more or less constant, although in some measurements a tendency to increased sway was seen 5 to 6 h after the start of the experiments, probably because of fatigue or tiredness on the part of the subjects.

Alcohol runs

During the initial phase of alcohol intoxication, body sway increased in all conditions tested (compare Fig. 2), in parallel with BAC and the subjective estimate of intoxication. The dif-

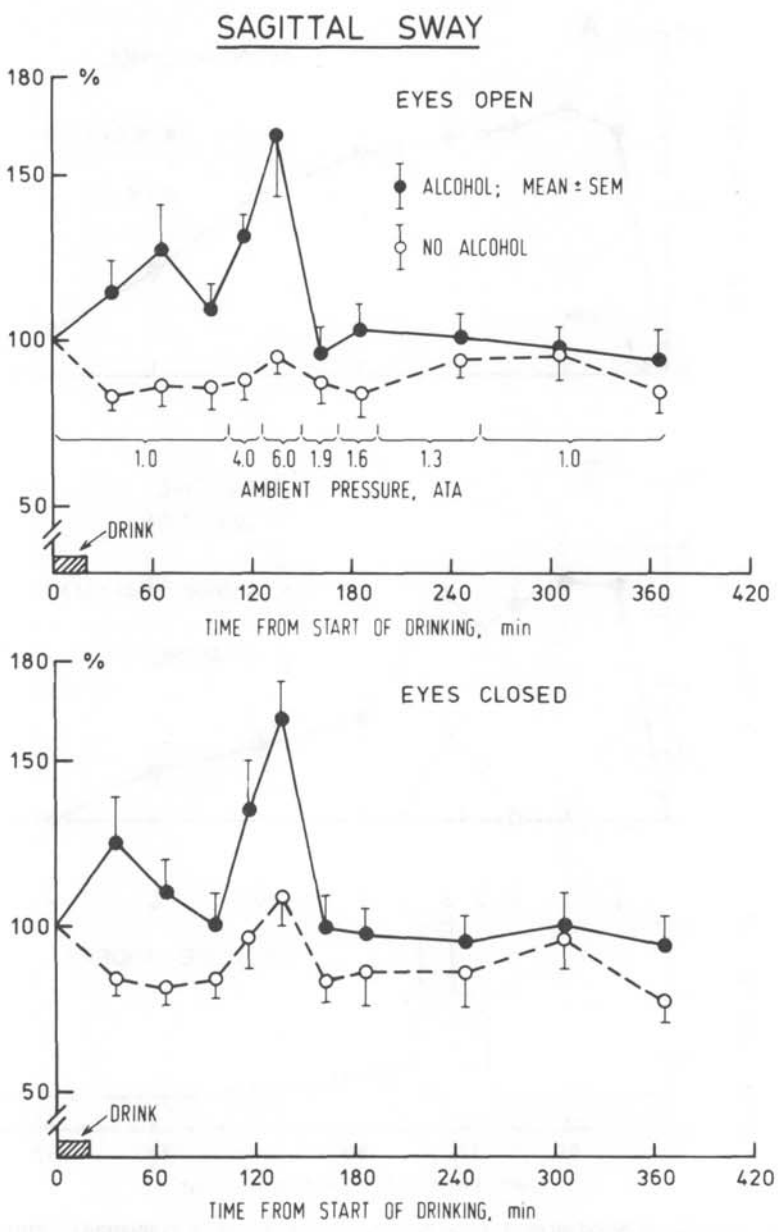

Fig. 2. Effects of alcohol and raised air pressures on body sway in sagittal direction with eyes open (*upper graph*) and eyes closed (*lower graph*). Changes in body sway scores computed as percentages of control (pretreatment) values and given as mean values ± SEM; $n = 8$.

ferences in sway between the alcohol and alcohol-free runs were statistically significant during this phase ($P < 0.01$). At 90 min from start of drinking, there was a clear recovery from the acute alcohol effect and, at this time, there were no significant differences ($P > 0.05$), i.e., the alcohol effect on body sway was no longer evident.

On raising the ambient pressure to 4 and 6 ATA, marked increases of body sway, which were greatest in the sagittal direction, occurred. This is in contrast to the pressure effect in the

alcohol-free runs, in which body sway increased more in the lateral than in the sagittal direction. During decompression, there was a rapid recovery of body sway, as was the case also in the alcohol-free runs. The mean sway scores after alcohol remained above the alcohol-free scores for several hours after the pressure was reduced, although the differences were not statistically significant ($P > 0.05$).

Combined alcohol and pressure effects

To elucidate the combined effects of alcohol and pressure on body sway in comparison with the effects of pressure alone, an analysis of variance was made using the scores obtained at 1, 4, and 6 ATA. The mean amplitudes of sway are illustrated in Fig. 3 for sagittal and lateral directions and with open and closed eyes. It may be seen that the differences in amplitude of sway between the alcohol and alcohol-free conditions are small at 1 ATA, and then clearly increase as the pressure is raised to 4 and 6 ATA. The rate of increase is more dramatic for body sway in the sagittal direction, particularly with eyes closed.

The results of the analysis of variance are presented in Table 1. There is a statistically significant difference in body sway among different subjects ($P < 0.01$), and also a highly significant effect of pressure on body sway ($P < 0.001$). This confirms the high interindividual variations in mean amplitude of body sway and the marked effects of pressure alone in producing body sway disturbances (Adolfson et al. 1972; Braithwaite, Berghage, and Crothers 1974). The main effect of the condition, i.e., the effect of alcohol independent of subject and pressure effects, was statistically significant in the sagittal direction with eyes open ($P < 0.01$) and in the lateral direction with eyes closed ($P < 0.05$).

The subject x condition interaction was statistically significant, indicating that large interindividual variations in sway existed in both the alcohol and alcohol-free conditions. The subject x pressure interaction effects were significant only with eyes closed. Thus, with increasing

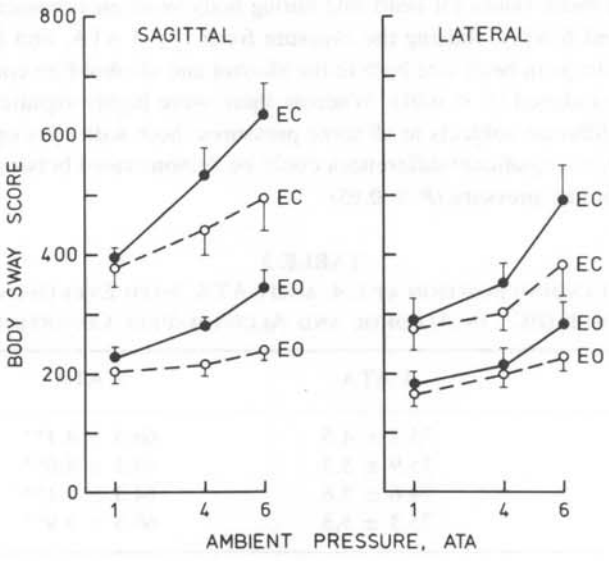

Fig. 3. Body sway scores with eyes open (EO) and eyes closed (EC) in sagittal (*left graph*) and lateral (*right graph*) directions at air pressures of 1, 4, and 6 ATA, in alcohol-free condition (*dashed lines*) and 90–130 min after intake of moderate dose of alcohol (*solid lines*). Mean values ± SEM; $n = 8$.

TABLE 1

F-RATIOS COMPUTED BY ANALYSIS OF VARIANCE BASED ON BODY SWAY SCORES IN ALCOHOL AND ALCOHOL-FREE CONDITIONS AT AIR PRESSURES OF 1, 4, AND 6 ATA.

Source of Variation	Degrees of Freedom	Sagittal Sway		Lateral Sway	
		EO	EC	EO	EC
Subjects (S)	7/14	6.55**	15.66†	11.74†	21.93†
Pressures (P)	2/14	15.91†	16.91†	12.78†	17.04†
Conditions (C)	1/7	13.51**	2.57	1.25	5.69*
Interactions					
S × P	14/14	1.44	2.50*	1.46	2.94*
S × C	7/14	4.39**	10.20†	5.93**	3.54*
P × C	2/14	9.31**	5.55*	1.94	4.43*

Recordings made with 8 subjects; body sway measured in lateral and sagittal directions with eyes open (EO) and eyes closed (EC). *$P < 0.05$; **$P < 0.01$; †$P < 0.001$.

pressures and open eyes, subjects maintained their relative position in the group, i.e., a subject with a low body sway at 1 ATA also showed a low sway at 4 and 6 ATA. With eyes closed (nonvisibility), on the other hand, increasing pressure disrupts an individual's own level of sway, and hence the significant interaction. The analysis of variance also showed that there was a significant pressure *x* condition interaction in the sagittal direction with eyes open ($P < 0.01$) and eyes closed ($P < 0.05$), and in the lateral direction with eyes closed ($P < 0.05$).

Heart rate

Table 2 presents mean values for heart rate during body sway measurements in the standing position at 1, 4, and 6 ATA. Raising the pressure from 1 to 4 ATA, and further to 6 ATA, caused significant drops in heart rate both in the alcohol and alcohol-free conditions, and with eyes open and eyes closed ($P < 0.01$). Whereas there were highly significant differences in heart rate among different subjects at all three pressures, both with eyes open and with eyes closed ($P < 0.001$), no significant differences could be demonstrated between the alcohol and alcohol-free runs at any pressure ($P > 0.05$).

TABLE 2

HEART RATE IN STANDING POSITION AT 1, 4, AND 6 ATA, WITH EYES OPEN (EO) AND EYES CLOSED (EC), IN ALCOHOL AND ALCOHOL-FREE CONDITIONS

Condition		1 ATA	4 ATA	6 ATA
Alcohol-free	EO	75.2 ± 4.5	66.5 ± 4.1**	62.3 ± 3.8†
	EC	75.9 ± 5.3	69.1 ± 4.0**	64.2 ± 4.1†
Alcohol*	EO	74.6 ± 5.6	64.3 ± 4.1**	60.5 ± 3.2†
	EC	75.3 ± 5.3	66.8 ± 3.9**	61.1 ± 2.7†

Values are means ± SEM; $n = 8$; *90–130 min after start of drinking; **significantly different from 1.0 ATA, $P < 0.01$; †significantly different from 4.0 ATA, $P < 0.01$.

DISCUSSION

The present investigation appears to be the first attempt to study the effects of ethanol on performance and behavior in man under hyperbaric conditions. The statometer technique used to evaluate the degree of performance decrement has been shown to be sensitive to elevated blood alcohol (Dussault and Chappel 1975), certain drugs (Orr, Dussault, Chappel, Goldberg and Reggiani 1976), hypoxia (Bjerver and Persson 1957) and increased pressures of air and He-O_2 (Adolfson et al. 1972; Adolfson, Fagraeus, and Hesser 1973; Braithwaite, Berghage, and Crothers 1974). The results obtained with the statometer technique are both objective and reproducible and, therefore, this method should be appropriate for the study of possible interactions between alcohol intoxication and inert gas narcosis.

It was found that increasing the ambient air pressure to 4 and 6 ATA had no significant influence on either the general shape of the blood alcohol curve or on the ethanol turnover rate normally observed after a single moderate dose of alcohol. This extends the previous finding of Senior (1972) that increased oxygen pressure exerts no effect on ethanol metabolism.

Body sway scores at 30 and 60 min after the start of alcohol consumption were significantly higher than those during the sober condition, although at 90 min, i.e., the last prepressure test, sway did not differ significantly between the two conditions. The most interesting finding from the analysis of variance was the significant pressure x condition interaction. As illustrated by Fig. 3, this significant pressure-condition interaction may be interpreted to mean that an elevated blood alcohol level potentiates the increase in body sway caused by acute exposure to high pressures of air.

At the 90-min prepressure recording, the mean BAC was 0.73 mg/ml. The subjects, however, did not consider themselves particularly intoxicated; the mean subjective rating was a score of 6. It is well known that ethanol intoxication is not completely linked to the underlying blood alcohol concentration, but is also dependent on certain psychological factors, acute adaptation, and tolerance phenomena (Wallgren and Barry 1970). Furthermore, it has been shown that younger people (20 to 30 years) consider themselves less intoxicated, and also have less body sway, than older people (40 to 60 years) for the same alcohol dose (Goldberg, Jones, and Neri 1974).

In accordance with previous observations (Hesser, Fagraeus, and Linnarsson 1978), it was found that with air breathing the heart rate in the standing position decreased with a rise in ambient pressure. Evidence has been presented that three mechanisms are predominantly responsible for this reduction in heart rate, namely, an oxygen-dependent increase in vagal tone, a direct myocardial effect of increased oxygen pressure, and a nitrogen-dependent beta-blockade of the heart. The observation that heart rate was uninfluenced by alcohol at normal ambient pressure is at variance with the findings of Myrsten, Hollstedt, and Holmberg (1975), who found significantly higher heart rate after intake of the same dose of alcohol as used in the present investigation. This discrepancy in results may be attributed to the fact that in the present study heart rate was recorded in the standing position, whereas in the study of Myrsten et al. (1975) it was measured in the seated position. The fact that no significant differences in heart rate could be demonstrated at any pressure between the alcohol and alcohol-free conditions suggests that alcohol had no apparent effect on the systemic circulation and, hence, that the greater body sway after alcohol intake was not to any significant degree due to circulatory disturbances.

In conclusion, it may be stated that a moderate dose of alcohol potentiates the increase in body sway and hence in the performance decrement caused by acute exposure to high

pressures of air. These findings could be of practical significance in relation to alcohol use by sport divers before and between dives and may be one explanation of underwater accidents.

This study was supported by the Swedish Medical Research Council, Projects No. 5020 and 5021, and by the National Defence Research Institute, Project No. H 305. R. D. Jennings was on sabbatical leave from Portland State University. A preliminary account of part of the results was given at the 1977 Annual Scientific Meeting of the Undersea Medical Society, Toronto, Ontario, Canada. R. D. Jennings's address is: Department of Psychology, Portland State University, P.O. Box 751, Portland, OR 97207, USA.—*Manuscript received for publication June 1978; revision received September 1978.*

Jones, A. W., R. D. Jennings, J. Adolfson, and C. M. Hesser. 1979. Effets combinés de l'alcool éthylique et de l'air hyperbarique sur le mouvement corporel et sur la fréquence cardiaque chez l'homme. Undersea Biomed. Res. 6(1): 15–25.— Huit plongeurs amateurs ont participé an expériences pour préciser les effets de l'alcool éthylique (0,72 g/kg de poids corporel) et de l'air hyperbare (4 et 6 ATA) sur la fréquence cardiaque et sur les mouvements corporels physiologiques. Les mouvements latéraux et sagittaux ont été mesurés (les yeux des sujets ouverts ou fermés) par un statokinésimètre. Sous alcool, on a remarqué une augmentation initiale de mouvement, ce qui correspondrait au phase aigu de l'intoxication. A la concentration moyenne de 0,77 mg/ml cette augmentation de mouvement est significative ($P < 0{,}01$) par comparaison au mouvement sans alcool. Une heure et demie après l'administration de l'alcool, le mouvement n'est guère différent de celui qu'on observe chez les sujets non-intoxiqués. Aux pressions de 4 et 6 ATA, les mouvements s'augmentent chez les deux groups de sujets; la vitesse de l'augmentation est plus grande chez les sujets intoxiqués. On a pu établir des rapports significatifs entre la pression et l'intoxication, ce qui permet de conclure a une activité de potentiation de l'alcool sur l'augmentation de mouvement provoquée par l'exposition aigue à l'air à hautes pressions. La fréquence cardiaque n'a pas subi d'altération significative chez le sujet intoxiqué, mais la pression ambiante accrue a provoqué une baisse de la fréquence cardiaque chez les deux groupes de sujets.

alcool
alcool éthylique
mouvement corporel
performance
air hyperbarique
synergisme
fréquence cardiaque

REFERENCES

Adolfson, J. A., and T. E. Berghage. 1974. Perception and performance under water. John Wiley & Sons, New York. 359 pp.

Adolfson, J., K. Bjerver, E. Fluur, and L. Goldberg. 1974. Balance disturbance in man at 10 ATA ambient air pressure. Swed. J. Defence Med. (Försvarsmedicin, Stockholm) 10: 148–156.

Adolfson, J., L. Fagraeus, and C. M. Hesser. 1973. Deterioration of standing steadiness in hyperbaric environment. Swed. J. Defence Med. (Försvarsmedicin, Stockholm) 9: 253–256.

Adolfson, J. A., L. Goldberg, and T. Berghage. 1972. Effects of increased ambient air pressures on standing steadiness in man. Aerosp. Med. 43: 520–524.

Bennett, P. B. 1975. Inert gas narcosis. Pages 207–230, *in* P. B. Bennett and D. H. Elliott, Eds. The physiology and medicine of diving and compressed air work. 2nd ed. Baillière, Tindall, London.

Bjerver, K., and P.-A. Persson. 1957. The effect of hypoxia on standing steadiness. Arch. Int. Pharmacodyn. Ther. 112: 247–263.

Braithwaite, W. R., T. E. Berghage, and J. C. Crothers. 1974. Postural equilibrium and vestibular response at 49.5 ATA. Undersea Biomed. Res. 1: 309–323.

Brownlee, K. A. 1965. Statistical theory and methodology in science and engineering. 2nd ed. John Wiley & Sons, New York. Chap. 15.

Buijten, J. C. 1975. An automatic ultra-micro distillation technique for determination of ethanol in blood and urine. Blutalkohol 12: 393–398.

Dussault, P., and C. I. Chappel. 1975. Difference in blood alcohol levels following consumption of whisky and beer in man. Pages 365–370, *in* S. Israelstam and S. Lambert, Eds. Alcohol, drugs, and traffic safety. Addiction Research Foundation of Ontario, Toronto, Canada.

Ekman, G., M. Frankenhaeuser, L. Goldberg, K. Bjerver, G. Järpe, and A.-L. Myrsten. 1963. Effects of alcohol intake on subjective and objective variables over a five-hour period. Psychopharmacologia 4: 28–38.

Ekman, G., M. Frankenhaeuser, L. Goldberg, R. Hagdahl, and A.-L. Myrsten. 1964. Subjective and objective effects of alcohol as functions of dosage and time. Psychopharmacologia 6: 399–409.

Franks, H. M., V. R. Hensley, W. J. Hensley, G. A. Starmer, and R. K. C. Teo. 1976. The relationship between alcohol dosage and performance decrement in humans. J. Stud. Alcohol 37: 284–297.

Goldberg, L. 1943. Quantitative studies on alcohol tolerance in man. Acta Physiol. Scand. Suppl. 16, 5: 1–128.

Goldberg, L. 1966. Behavioral and physiological effects of alcohol on man. Psychosom. Med. 28: 570–595.

Goldberg, L., A. W. Jones, and A. Neri. 1974. Age dependent differences in blood alcohol and subjective mood estimates. Communication, Department of Alcohol Research, Karolinska Institutet, Stockholm, Sweden, No. 740129.

Hesser, C. M., L. Fagraeus, and D. Linnarsson. 1978. Respiratory gases and hyperbaric bradycardia. Proceedings of the third annual scientific meeting of the European undersea biomedical society, Toulon, France, 15–16 July 1977. Méd. Aéronaut. Spat. Subaquat. Hyp. 17: 110–113.

Kalant, H. 1971. Absorption, diffusion, distribution, and elimination of ethanol: effects on biological membranes. Pages 1–62, *in* B. Kissin and H. Begleiter, Eds. The biology of alcoholism. Vol. 1. Biochemistry, Plenum Press, New York.

Kissin, B., and H. Begleiter, Eds. 1972. The biology of alcoholism. Vol. 2. Physiology and behaviour. Plenum Press, New York, 551 pp.

Lindborg, B., O. Wigertz, and Th. Ödman. 1969. A beat-by-beat heart-rate meter with linear analog output for muscular exercise studies. Karolinska Institutet Lab. Aviat. Naval Med. Report.

Myrsten, A.-L., C. Hollstedt, and L. Holmberg. 1975. Alcohol-induced changes in mood and activation in males and females as related to catecholamine excretion and blood-alcohol level. Scand. J. Psychol. 16: 303–310.

Orr, J., P. Dussault, C. Chappel, L. Goldberg, and G. Reggiani. 1976. Relation between drug-induced central nervous system effects and plasma levels of diazepam in man. Mod. Probl. Pharmacopsychiatr. 11: 57–67.

Senior, J. R. 1972. Studies on the effect of hyperbaric oxygen breathing on the rate of ethanol metabolism in man. Nav. Sub. Med. Res. Lab., Submarine Base, Groton, Conn., Report No. 717. 11 pp.

Wallgren, H., and H. Barry. 1970. Actions of alcohol. Vol. 1. Biochemical, physiological, and psychological aspects. Elsevier, Amsterdam. 400 pp.