NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

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REPORT NUMBER 717

STUDIES ON THE EFFECT OF HYPERBARIC OXYGEN BREATHING ON THE RATE OF ETHANOL METABOLISM IN MAN

by

John R. Senior, CDR, MC, USNR

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MF099.01.01.09

Released by:

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Officer-in-Charge
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7 July 1972

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SUMMARY PAGE

THE PROBLEM

To investigate the effect of wide variations in partial pressure of inspired oxygen on the rate of ethanol disappearance from body water in man, under controlled laboratory conditions, with the aim of providing further information concerning the rate-limiting steps in the oxidative catabolism of ethanol.

FINDINGS

Over a twenty-five fold range of inspired oxygen pressure, from 91 to 220 mmHg, the linear rate of decrease of blood ethanol concentration with time was demonstrated to be independent of oxygenation. These findings support the concept that the hepatic intracellular and intra-mitochondrial pO_2 is probably not rate-limiting in the removal of electrons derived from the reducing equivalents produced by the oxidation of ethanol in the liver. These data are consistent with the known values for the high affinity of cytochrome oxidase for oxygen, and suggest that an earlier step in the delivery and oxidation of cytoplasmic reducing equivalents is rate-limiting for ethanol oxidation.

APPLICATIONS

These observations represent the first such investigation on humans at three atmospheres absolute pressure, at oxygen concentrations 4%, 7% and 100% for periods approaching the safe limits of tolerance, on the rate of ethanol metabolism. The need for further work on the side of control of ethanol oxidation, and the possible mechanism of ethanol-induced hepatic intracellular membrane injury, is suggested by this work and has been initiated.

ADMINISTRATIVE INFORMATION

This study was performed and reported in partial fulfillment of the requirements for qualification in submarine and diving medicine. It was selected for publication in order to make the information readily available in the School of Submarine Medicine and the Technical Library at the Naval Submarine Medical Center.

The manuscript was approved for publication on 7 July 1972, and designated as NavSubMedRschLab Report Number 717. It is report number 9 on the work unit MF099.01.01.

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ABSTRACT

The initial step in the metabolism of ethyl alcohol in man is the oxidative removal of two hydrogen atoms from the hydroxy-methyl group of the compound, which is accomplished almost exclusively in the liver, and is catalyzed by the well-studied enzyme, alcohol dehydrogenase. The hydrogen removed, along with other hydrogen or equivalent reducing substances from subsequent oxidative reactions on the derived acetaldehyde and acetate, is eventually transferred from the cell sap into the mitochondria where the electrons from the hydrogen are transported to combine ultimately with oxygen: the hydrogen-derived protons then are used to form water. Although the affinity of cytochrome oxidase is so great that a very low partial pressure of oxygen suffices to allow the terminal step in transfer of electrons to oxygen to proceed, it has never been determined whether the overall rate of ethanol oxidation might be accelerated by increasing the whole body, and presumably intrahepatic, pO_2 to the maximal tolerable level.

In two healthy male adult subjects, exposed twice for one hour to 100% oxygen at three atmospheres absolute pressure, no consistent or impressive acceleration in the disappearance of ethanol from whole blood and body water could be demonstrated. Further investigations on the transhepatic oxygen and ethanol concentrations are underway, and additional studies on controlling earlier steps in the electron transport process are being planned. This work provides a significant clue in understanding the mechanism of ethanol-induced liver injury, and represents a merging of the disciplines of underwater physiology and biochemistry-cell biology toward clarification of the nature of clinical disease, under the sponsorship of the Institute for Environmental Medicine of the University of Pennsylvania, the U. S. Naval Reserve Training Program, and the School of Submarine Medicine, Naval Submarine Medical Center, Groton, Connecticut.

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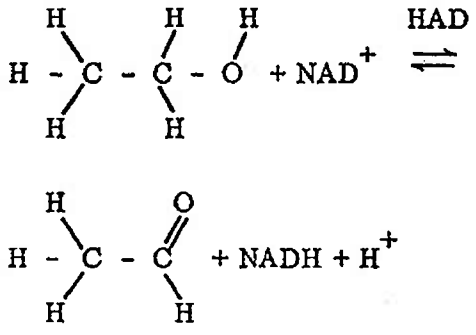
INTRODUCTION

Ethyl alcohol in man is removed from his body almost entirely by oxidation; the amounts lost by exhalation as vapor or in solution in the urine amount to less than a few percent of the total ingested or infused.[1] It has long been known[2] and repeatedly observed that the rate of equilibration of ethanol between blood and the total body water is quite rapid, and that disappearance of ethanol from the body water is independent of its concentration above 0.1 mg/ml. The linear, or zero-order, disappearance rate does not appear to be a function of saturation of alcohol dehydrogenase, the principal enzyme catalyzing the first step in ethanol oxidation.[3] Current thinking holds that reoxidation of reduced nucleotides produced by interaction of ethanol with oxidized nicotinamide adenine dinucleotide (NAD^+) is necessary to permit this reaction to proceed at maximal rates, that NAD^+ is necessary for the ethanol oxidation[4], and that some step in transport of electrons from the reduced nucleotides to oxygen is probably rate-limiting in the overall process[5]. The bulk of the body alcohol dehydrogenase is found in the liver cell cytoplasm, and the principal site for electron transport is within the hepatic cell mitochondria. Most recent evidence points to the rate-limiting step for alcohol metabolism being the transfer of hydrogen from cytoplasmic reduced nicotinamide adenine dinucleotide (NADH) across the hepatic cell mitochondrial membranes and to the electron transport chain inside.[5] The net effect of the whole concerted process is to transfer hydrogen from intra-cellular ethanol to oxygen arriving via the hepatic sinusoidal blood, producing water and carbon dioxide.

The overall reaction is exothermic, and yields about 7 kcal/g. In the mammalian systems known, it takes place

$$CH_3CH_2OH + 3\ O_2 \longrightarrow 2\ CO_2 + 3\ H_2O$$

not as a single step, but in a sequence of enzyme-catalyzed steps, with linkage to the energy transmuting mechanisms to conserve some 35-40% of the heat energy as chemical energy in the form of adenosine triphosphate (ATP). The first step is catalyzed by the well-studied enzyme, hepatic alcohol dehydrogenase (HAD) (I.E.C., 1.1.1.1.), to produce acetaldehyde,



NADH, and a hydrogen ion (H^+). Kinetic data has indicated that the enzyme HAD is not saturated at tolerable body concentrations[3], and that availability of oxidized nicotinamide adenine dinucleotide (NAD^+) might limit the acetaldehyde production rate.[4] More NAD^+ is needed for oxidation of acetaldehyde to acetate,

which is rapid and also carried out in the liver. Even more NAD^+ is needed for oxidation of acetate to CO_2 and H_2O via the Krebs cycle, either in liver or peripheral tissues. The large demand for NAD^+, or excessive conversion of it to NADH, therefore appears to create the bottleneck in the overall process of ethanol oxidation, and can be supplied only by reoxidation of NADH to NAD^+. This process is effectively accomplished in a net sense only within the mitochondrial electron transport system (METS), where electrons are transferred via several steps to cytochrome oxidase (cyt. ox.) and thence to molecular oxygen.

$$NADH + METS \longrightarrow NAD^+ + METS\begin{matrix}H\\ \diagup\\ \diagdown\\ e^-\end{matrix}$$

$$4\ \text{cyt.ox.}^- + O_2 \rightleftharpoons 2O^= + 4\ \text{cyt.ox.}$$

$$4\ H^+ + 2O^= \longrightarrow 2\ H_2O$$

Reoxidation of NADH to NAD^+ occurs also in the cell sap outside mitochondria, using substances such as pyruvate, dihydroxyacetone phosphate, oxaloacetate, etc. as acceptors of hydrogen, but unless reducing equivalents can be transported to oxygen, the accumulation of reduced products such as lactate, glycerol-3-phosphate, malate, etc. will stop the reactions. The ultimate acceptor, therefore, of the 6 hydrogen atoms from ethanol, and of 6 electrons, are three atoms of oxygen. Since NADH cannot itself be readily transported across the mitochondrial membrane to the METS[6], intermediate shuttle substances such as malate and glycerophosphate appear to be extremely important for carrying hydrogen to the METS[7].

The major site of ethanol oxidation, at least the first two oxidations, is the liver, but there has not as yet been advanced any data to suggest that availability of oxygen itself might be rate-limiting. The affinity of cytochrome oxidase for oxygen ($K_m \approx 10^{-6}$ to 10^{-8} molar, depending on the respiratory rate) is so great that, even at very low tissue water pO_2, the transfer of electrons to oxygen is rapid and efficient. However, there have been no data which obviate the possibility that the process might be accelerated by hyperoxygenation of the whole animal, including the intrahepatic, intra-mitochondrial site of electron transfer to oxygen. Some earlier findings[8] did in fact indicate an accelerated reoxidation of NADH to NAD^+ in rats exposed to pure oxygen at 10 ATA. Practical limits of safe exposure to hyperbaric oxygen at 3 ATA for man have been considered to be about 1-2 hours[9], the limiting effects being central nervous system toxicity and the resulting convulsions, or for longer exposures at lower pressures by reduction in pulmonary ventilation. There have been no data at all provided on (1) a possible acceleration in ethanol metabolism, (2) a possible alteration in the NADH/NAD^+ in man, (3) the increase in hepatic venous pO_2, and therefore centrilobular hepatic cell pO_2, and (4) possible reduction in CNS irritability by the depressant effect of ethanol under conditions of hyperbaric oxygenation at or near the limits of oxygen tolerance. Further, there has been no investigation of possible effects of ethanol on inert gas bubble formation,

so that routine decompression schedules cannot be applied without some consideration of correction factors. The present studies were initiated to investigate the first of these points, and perhaps to gain some preliminary insights into some of the others.

MATERIALS AND METHODS

Subjects for these studies were the principal investigator and colleagues, all in apparent good health, all under 45 years of age, and all accustomed to moderate occasional use of ethyl alcohol in social settings. Pure grain alcohol, 200 proof (Pharmco), was used without sterilization for addition to physiologic saline (PSS) for intravenous infusion, or to water for oral or intraduodenal introduction. Doses of 1.0 to 1.5 ethanol/kg body weight were administered to subjects who were fasting or who had had a very light breakfast over periods of 40 to 60 minutes on the morning of the study. Venous blood was taken at frequent intervals from an opposite (in the case of intravenous administration) forearm vein via an indwelling catheter or butterfly needle, kept open by intermittent rinsing with heparin solution diluted by PSS. Measurement of whole blood and plasma ethanol concentrations was carried out by both enzymatic and gas-liquid chromatographic techniques, in duplicate for each specimen. Since the enzymatic method was slow and cumbersome for the number of specimens required, a rapid (3-minute) method was developed for direct chromatographic assay by whole blood injection into a glass wool column in train with a Porapak-S column with a flame ionization detector (Carle Instruments, model 9000), as modified after Baker et al.[10] Satisfactory sensitivity and linearity of standard solutions from 25 to 300 mg ethanol/100 ml aqueous solution (Figure 1) were achieved with column temperature of 130° C, helium carrier gas flow of 32.8 ml/minute (28 psig through 1/16" copper tubing), injection volume of 0.50µl whole heparinized blood, with flame conditions stabilized by hydrogen gas flow of 29.6 ml/minute (35 psig through 1/16" copper tubing) (Figure 2) and air flow of 565 ml/minute (20 psig through 1/8" copper tubing), at attenuations of 20-fold for blood ethanol concentrations below 150 mg/100 ml and 50-fold for higher concentrations. Under these conditions ethanol began to reach the detector in 2.1 minutes, the peak was reached in 2.4 minutes, and the tail reached the baseline by 2.9-3.1 minutes (Figure 3); other alcohols, water acetaldehyde, or acetone did not interfere with or overlap these peaks. Values determined on the same blood samples by the alcohol-dehydrogenase-NAD^+ method the following day after preservation of the 1:20 solution of whole heparinized blood in 2% perchloric acid agreed within 1%. Agreement of duplicate samples, and linearity over the range of expected values, was excellent (Figure 1). In earlier experiments,[14] C-labeled ethanol was included in the administered load of 0.5-0.75 g unlabeled ethanol/kg body weight, and expired air was collected for 3-minute intervals repeatedly for determination of the specific activity of $^{14}CO_2$ exhaled over a prolonged period, by a technique previously described.[11]

Computation of decompression schedules, and detailed planning for the

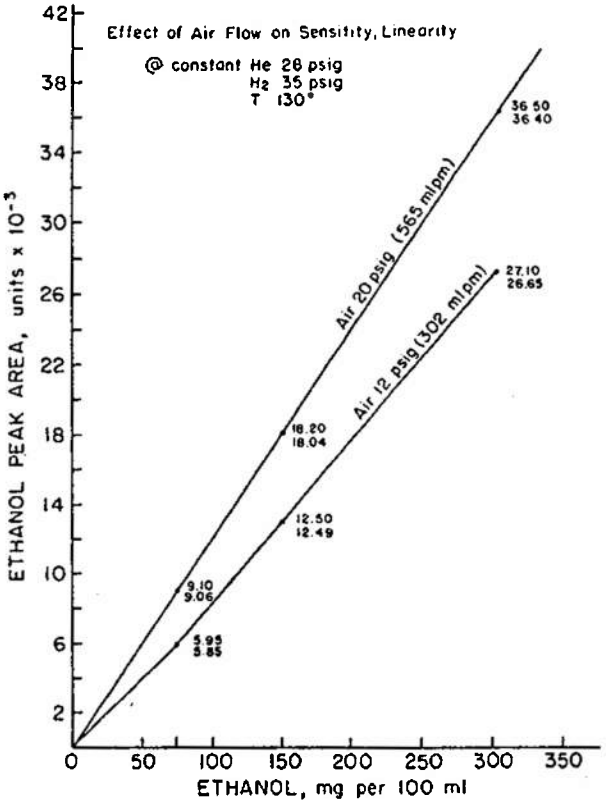


Fig. 1. Linearity and reproducibility of gas-liquid chromatographic determination of whole venous blood ethanol concentration. Column temperature 130°C, flame regulation by hydrogen and air pressures as shown. Note the near-superimposition of duplicate points on the air 20 psig line.

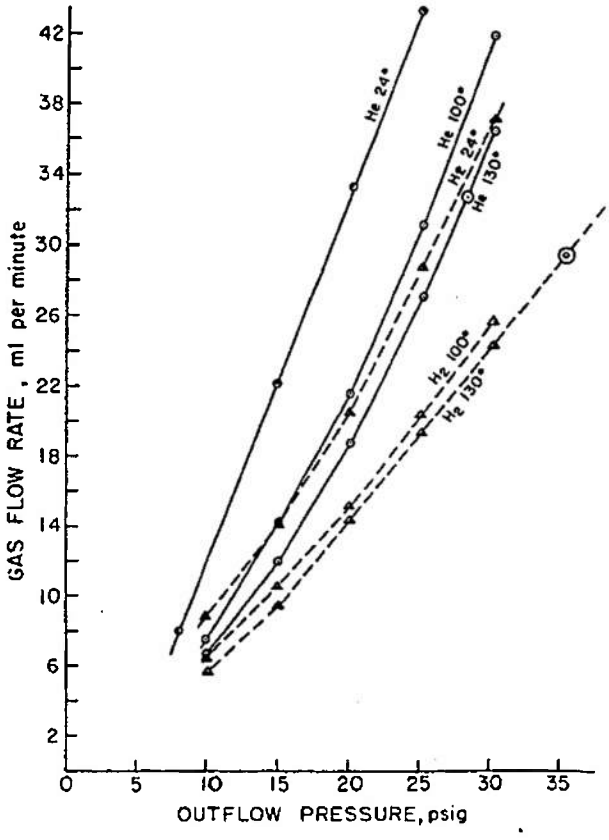


Fig. 2. Calibration of gas flows vs. gauge pressures at temperatures from 100 to 130°C. Points used shown by large circles.

dives was carried out in consultation with the staff, including Drs. C. J. Lambertsen and J. G. Dickson of the Institute for Environmental Medicine of the University of Pennsylvania (IFEM). The compression chamber complex, surrounding biochemistry and physiology laboratory, and supporting technical personnel and assistance were provided by IFEM (Figures 4,5). The time schedule of events, after initial catheterizations, intravenous equipment arrangement, final preparation of subject and materials, and attaining control blood samples, was as follows (Figure 6):

0800 Begin infusion or ingestion of ethanol solutions.

0900 Complete ethanol administration, move into chamber (1 ATA, room air) subject and medical observer.

1000 Compress to 3 ATA, by adding 2 atmospheres of N_2, both subject and medical observer,

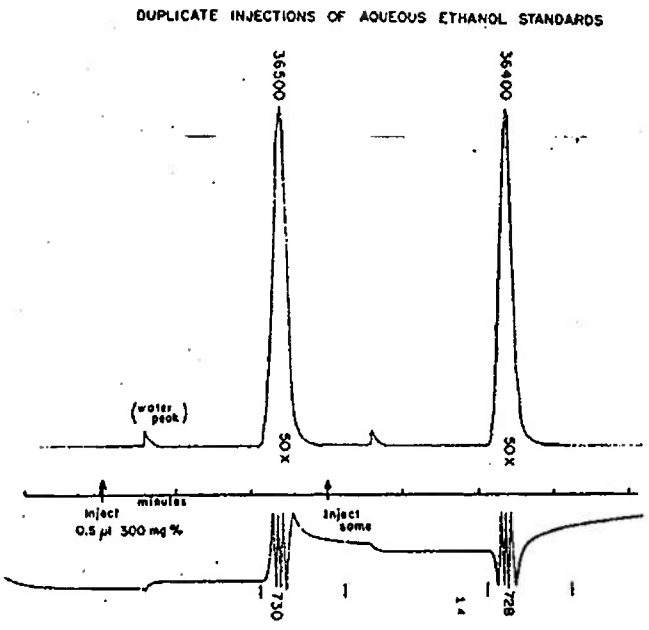


Fig. 3. Peak shape and reproducibility of ethanol injections, using 0.50 µl aqueous solutions of standard ethanol of concentration 3 mg/ml. Time for appearance of water peak 0.6 minutes; for ethanol, 2.2-2.8 minutes. Lower integration curve shows areas under peaks; attenuation, 50-fold.

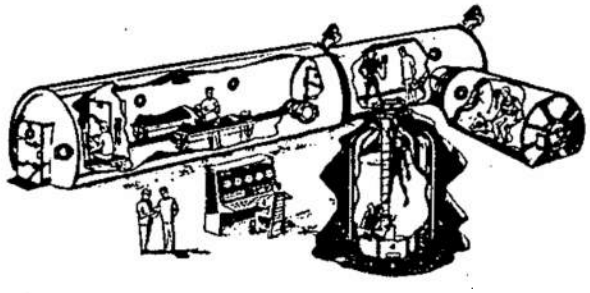


Fig. 4. Diagram of chamber complex at the Institute for Environmental Medicine, University of Pennsylvania Medical Center.

breathing air from masks until equilibration of 7% O_2 atmosphere (Figure 6).

1100 Subject begins breathing 100% O_2 at 3 ATA.

1200 Subject returns to 7% O_2 at 3 ATA.

1300 Subject begins breathing 4% O_2 at 3 ATA.

1400 Subject returns to 7% O_2 at 3 ATA.

1500 Subject again begins breathing 100% O_2 at 3 ATA.

1600 Begin decompression schedule (see appendix I for details), subject and medical observer both on 21% O_2.

1952 Reach "surface".

During this time 1 ml lightly-heparinized blood samples were obtained every 10-20 minutes. These samples were kept pressurized on ice and were removed via a small medical air lock to the adjacent chemistry laboratory for ethanol determinations. Close physical and behavioral observations were made of the subject by the trained diving medical observer inside the chamber, and by another outside the chamber. Gas chromatographic measurements of ethanol were done at once, results becoming available within 5 or 10 minutes; confirming enzymatic determinations of blood ethanol were carried out the following day on the refrigerated perchloric acid extracts of the blood.

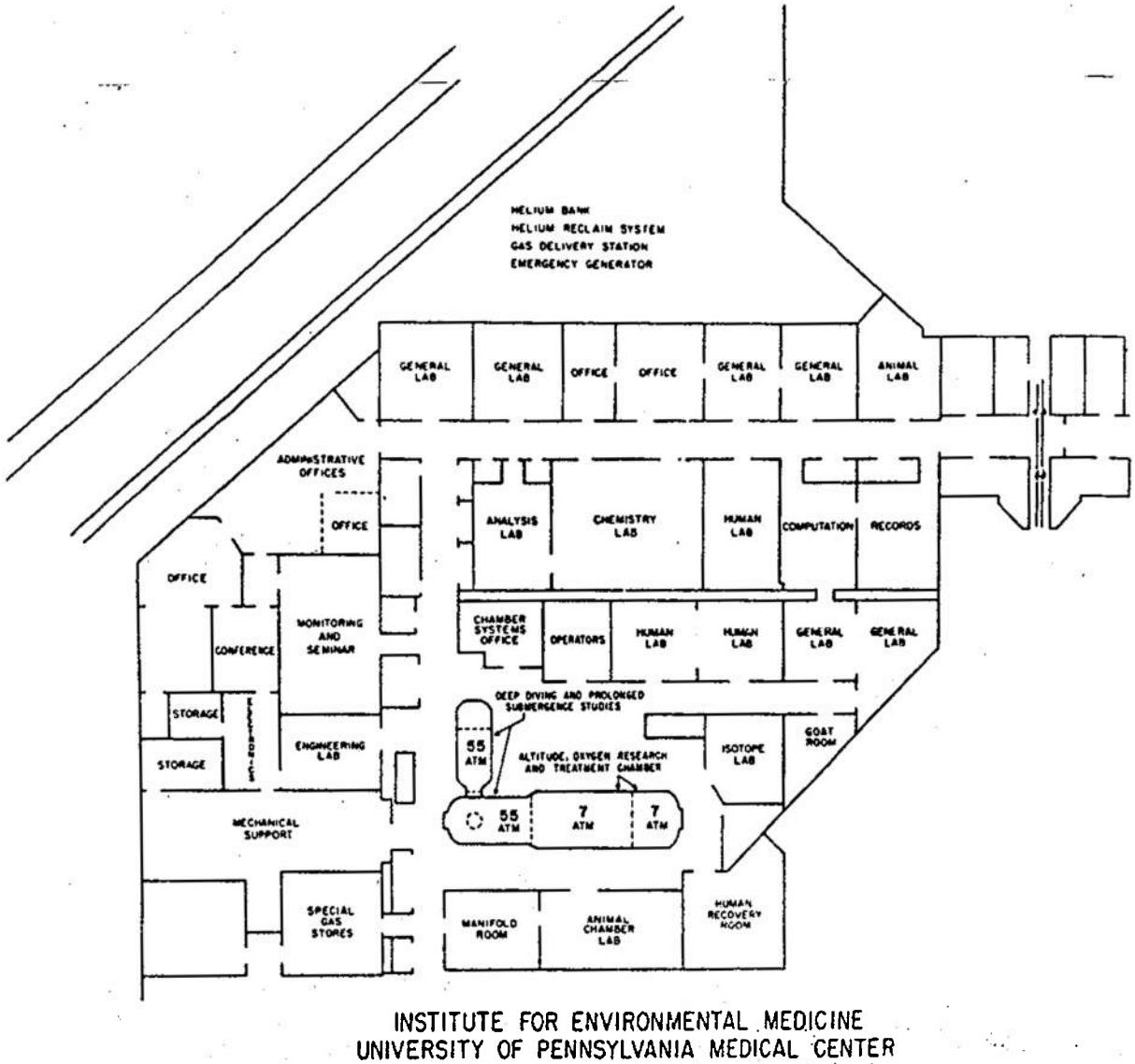


Fig. 5. Supporting laboratories and shops surrounding the chamber complex, Institute for Environmental Medicine, University of Pennsylvania.

RESULTS

The ability of the subjects to estimate their own approximate level of blood ethanol and its rate of change was notable when the log entries of their comments and subjective reactions were compared with the chemical data. This was especially true during the induction period, for subjects generally became drowsy and slept for most of the time while under compression, although they were rousable and could cooperate well in breathing the different gas mixtures and responding to commands. The dose level chosen was sufficient to allow a long, linear, disappearance curve of the blood ethanol with time, extending

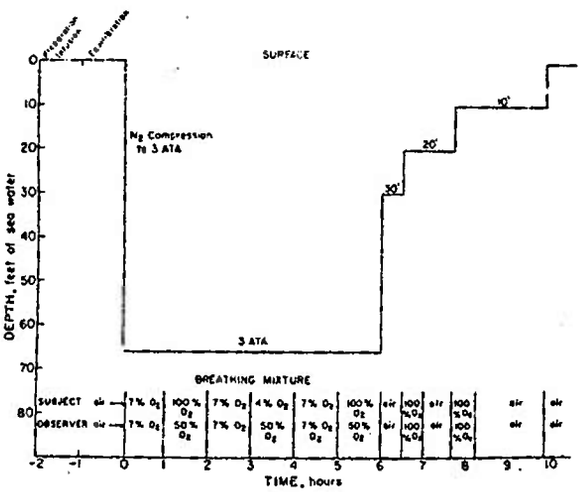


Fig. 6. Dive profile, and gas mixtures breathed by subject and attendant, during six-hour, 3 ATA dive and decompression period. See also appendix I.

over the whole time under compression in the chamber, but not so great as to cause excessively high or dangerous initial peak values. Intravenous administration of ethanol in PSS was well tolerated, and avoided gastric irritation, nausea, and vomiting sometimes encountered with oral administration. Maximal blood ethanol values in the range of 200 mg/100 ml were attained. With these amounts, the subjects were almost entirely free of the effects of ethanol by the end of the decompression period, with very little "hangover" or other residual aftereffects. No decompression effects were observed, either during the immediate hour of close observation after emergence from the chamber, or within the next several days of "on call" observation.

Subject I, who ingested 1.5 g/kg body weight of ethanol as pure grain ethyl alcohol diluted in iced ginger ale over a 30-minute period, showed blood ethanol values which rose to a peak of 180 mg/100 ml at 3 hours after ingestion began, or 2-1/2 hours after it was completed. There was some nausea and emesis at about 3-1/2 and 5 hours after beginning ingestion, while the subject was under 3 ATA pressure, and it is estimated that about 20% of the ingested ethanol dose was lost. The emesis appeared to be associated with some erratic swings of the blood ethanol values, perhaps due to fluid shifts, and the truly linear portion of the curve was not reached until the terminal portion of the compression period and the decompression period (Figure 7). It may be noted that no impressive or significant increase in the downward slope could be seen during the periods of 100% O_2 breathing, nor any flattening during the 4% O_2 breathing.

Subject II received 1.0 g/kg body weight of pure grain ethyl alcohol in 1 liter of PSS over a 40-minute period, and showed a generally more predictable curve (Figure 8). It should be noted that he experienced no nausea, discomfort or unpleasant effect. His peak ethanol value of 195 mg/100 ml was reached at the end of the infusion period, and then very rapidly fell as the ethanol equilibrated in the larger volume of the body water. Thereafter, the decline was linear, with no substantial effects of changing either pressure or oxygen concentration in the inspired air.

While there could conceivably be minor alterations in the overall rate of ethanol metabolism masked by the biological and chemical fluctuations affect-

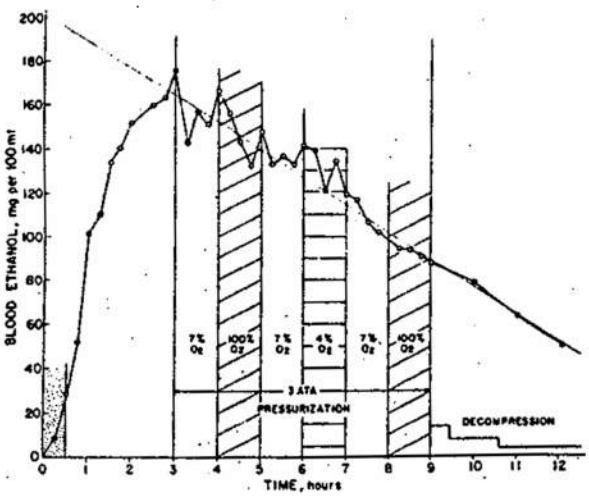


Fig. 7. Ethanol disappearance curve for subject I, after oral load of 1.5 g/kg body weight.

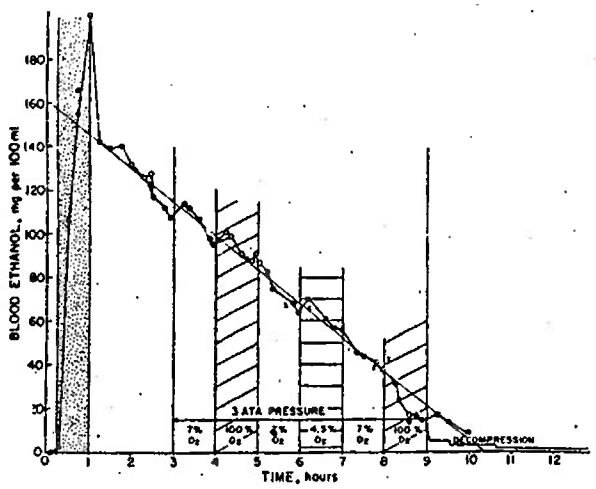


Fig. 8. Ethanol disappearance curve for subject II, after intravenous load of 1.0 g/kg body weight.

ing the determinations, it appears that no major degree of acceleration of ethanol disappearance can be produced even by pushing oxygenation to the limit of safe tolerance. No central nervous system hyper-irritability, no pulmonary difficulties, or other evidences of oxygen toxicity were observed during these studies. Further studies to confirm and extend these observations are in progress.

DISCUSSION

Ethanol oxidation appears to proceed at a fairly constant rate in a given individual, ranging from about 40 to 230 mg/kg body weight/hour[1], and in the subjects used, at 74 and 96 mg/kg/hr. No significant increase is effected by exercise, hyperthyroidism, or cold[12], only slightly by pyruvate, fructose, amino acids or insulin[13], but retarded by fasting or fatty acids[14]. It has recently been learned that accelerated ethanol metabolism may be induced by prolonged ingestion of large amounts of alcohol, and suggested that an hepatic microsomal ethanol oxidizing system[15,16] may be responsible, although some dispute exists as to the quantitative role of this system and possible contamination by the catalase system[17].

No previous work on hyperbaric oxygenation as a possible means of modifying ethanol metabolic rates is known to exist, but the question seemed reasonable, for all of the known mechanisms for ethanol oxidation depend ultimately on availability of O_2 to receive electrons and protons to form water from the hydrogen derived from ethanol. It is of interest, therefore, that providing oxygen in excess, or at least in amounts not ordinarily available to the hepatic sites of ethanol oxidation,

does not appear to cause enough acceleration in any of the processes to be reflected significantly in the overall disappearance rate of ethanol. This observation does not exclude the possibility that one of the systems for ethanol oxidation might be enhanced by hyperoxygenation, but shows only that the effect on the whole man is not detectable within the biological and chemical limitations of this test system.

It was interesting that the directly-observed NADH/NAD^+ in the liver and kidney of intact, whole rats exposed to 10 ATA of oxygen did in fact fall[8], and this ratio was known to be elevated by ethanol metabolism[18]. Although there is no previous data on the tissue oxygen partial pressure in the liver cells under conditions of hyperbaric oxygenation, it seemed likely that it could only increase, and probably significantly, upon exposure to 100% O_2 at 3 ATA for as long as 60 minutes, even though the normal liver is unique in receiving the bulk of its blood flow from the portal vein rather than from its hepatic artery. Direct measurement of hepatic venous and arterial pO_2 under conditions similar to those used in these studies is being carried out, along with estimation of transhepatic lactate/pyruvate and β-hydroxybutyrate/acetoacetate ratios, and will be the subject of a subsequent report from this laboratory.

These findings would seem to indicate that metabolic intermediates may play a more important role in transfer of reducing equivalents from hydrogen to oxygen in the various intracellular compartments of the hepatocyte than does oxygen itself. The known high affinity of cytochrome oxidase for oxygen, which allows it to catalyze transfer of electrons to oxygen at near maximal rates even when intramitochondrial pO_2 levels are quite low, is consistent with our experimental findings. Further work on the critical rate-limiting steps, and possible accumulations of metabolites, electrons, or protons proximal to those sites may shed light on the possible pathogenesis of damage to hepatic mitochondrial and endoplasmic reticular membrane damage leading to cell death and tissue damage in susceptible individuals during ethanol metabolism.

CONCLUSIONS

The rate of ethanol metabolism, under conditions which cause it to be maximal for an individual at surface-room air conditions, is independent of wide changes in the inspired oxygen tension to the limits of human tolerance in the hypoxic and hyperoxic ranges. It is considered likely that metabolic intermediates within the hepatic cell cytosol and mitochondria play a more important role that oxygen itself in transferring hydrogen from ethanol to the oxygen in the formation of resultant water.

ACKNOWLEDGEMENTS

These studies could not have been performed without the personnel and facilities of the Institute for Environmental Medicine of the University of Pennsylvania. The help of Drs. C.J. Lambertsen and J. G. Dickson, Mrs. J. Amand, Miss N. Struble and Dr. B.

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APPENDIX A

Alcohol Study Decompression Table
(Designed November, 1970)

The decompression schedule used in this study was based on the following considerations:

1. 93% N_2 at 66 fswg has the same partial pressure as air at 84 fswg. Consequently, a 90 fswg air table was used.

2. Since the U.S. Navy Standard Table for Exceptional Exposures has 80 and 100 fswg tables, but none at 90 fswg, a 90 fswg table was generated using the Institute for Environmental Medicine's PADUA computer program (Pennsylvania Analysis of Decompression for Undersea and Aerospace). This program uses the Workman Method, and was given the following N_2 half-times and M-values:

Compartment :	1	2	3	4	5	6	7	8	9
Half-time (min) :	10	40	80	120	160	200	240	360	500
Surfacing M-value: (fswa)	88	56	54	52	51	51	50	50	49
ΔM/10 fsw :	16	14	13	12	11.5	11	11	10	10

3. A 240-minute bottom time was chosen, based on the four hours of nitrogen breathing by the subject. This was clearly conservative for the subject because of the two hours of 100% oxygen breathing. The attendant's exposure was analyzed using PADUA based on alternate hour-periods of 93% and 50% nitrogen during the six hours, and was found to have lower tensions in each compartment than would exist after 240 minutes on air at 90 fswg. Hence, the schedule was conservative for the attendant as well.

4. Therefore, an exceptional exposure table was developed as shown on the accompanying computer print-out (see below) for a 240-minute exposure to 7% O_2-93% N_2 at 3 ATA. From an assumed equivalent air depth of 90 feet, a 1-minute ascent was made on air to 30 feet, 28 minutes were allowed on air at that depth, 70 minutes at 20 feet, and 130 minutes at 10 feet before surfacing. Further safety factors were provided by the two half-hour periods of O_2 breathing at the 20-foot and 10-foot levels by the attendant, who breathed air for the balance of the time at those stops.

GMPADUA1 12:54EST 11/2Ø/7Ø

LIST OUTPUT-FILE NAME.?HBOUT

LIST DATA-FILE NAME.?HBALCOHO

ACCTIME	DEPTHG	TIME		
24Ø.ØØ L	8Ø.ØØ T	1.ØØ		
241.ØØ R	3Ø.ØØ S	7.ØØ	6	
248.ØØ R	2Ø.ØØ S	57.ØØ	52	
3Ø5.ØØ R	1Ø.ØØ S	1Ø8.ØØ	120	
413.ØØ R	Ø.	172	179:20	
24Ø.ØØ L	9Ø.ØØ T	1.ØØ		
241.ØØ R	3Ø.ØØ S	28.ØØ		90 ft/240 min PADUA interpolation
269.ØØ R	2Ø.ØØ S	7Ø.ØØ		of U.S.N. tables for exceptional
339.ØØ R	1Ø.ØØ S	13Ø.ØØ		exposure
469.ØØ R	Ø.	229		
24Ø.ØØ L	1ØØ.ØØ T	1.ØØ		
241.ØØ R	4Ø.ØØ S	8.ØØ	14	
249.ØØ R	3Ø.ØØ S	47.ØØ	42	
296.ØØ R	2Ø.ØØ S	85.ØØ	84	
381.ØØ R	1Ø.ØØ S	151.ØØ	142	
532.ØØ R	Ø.	292	283:40	

END OF JOB.

```
PROGRAM STOP AT 715

USED      1.38 UNITS
LIST HBALCOHO


HBALCOHO      12:56EST      11/2Ø/7Ø

1ØØ  PAR  9  1  33  1Ø  YES
11Ø  HAL  1Ø  4Ø  8Ø  12Ø  16Ø  2ØØ  24Ø  36Ø  5ØØ
12Ø  MVA  88  56  54  52  51  51  5Ø  5Ø  49
13Ø  DEL  16  14  13  12  11.5  11  11  1Ø  1Ø
14Ø  SAT  Ø  Ø  .21  .79
15Ø  LEV  8Ø  24Ø  .21  .79
21Ø  DEC  Ø  6Ø  .21  .79
22Ø  SAT  Ø  Ø  .21  .79
23Ø  LEV  9Ø  24Ø  .21  .79
24Ø  DEC  Ø  6Ø  .21  .79
245  SAT  Ø  Ø  .21  .79
25Ø  LEV  1ØØ  24Ø  .21  .79
26Ø  DEC  Ø  6Ø  .21  .79
4ØØ  FIN  Ø  Ø  Ø  Ø


READY
LIST HBOUT


HBOUT      12:57EST      11/2Ø/7Ø

1ØØØ  TIME  Ø.  DEPTHG  Ø.
1ØØ1  26.1  26.1  26.1  26.1  26.1  26.1  26.1  26.1  26.1
1ØØ2  TIME  24Ø.ØØ  DEPTHG  8Ø.ØØ
1ØØ3  89.3  88.3  81.4  73.5  66.9  61.8  57.7  49.5  44.Ø
1ØØ4  TIME  241.ØØ  DEPTHG  3Ø.ØØ
1ØØ5  87.9  88.Ø  81.3  73.4  66.9  61.8  57.7  49.5  44.Ø
1ØØ6  TIME  248.ØØ  DEPTHG  3Ø.ØØ
1ØØ7  73.3  83.6  79.4  72.5  66.4  61.5  57.5  49.5  44.Ø
1ØØ8  TIME  3Ø5.ØØ  DEPTHG  2Ø.ØØ
1ØØ9  42.5  57.4  64.8  63.9  61.1  58.Ø  55.2  48.7  43.9
1Ø1Ø  TIME  413.ØØ  DEPTHG  1Ø.ØØ
1Ø11  34.Ø  37.6  46.1  5Ø.Ø  5Ø.9  5Ø.5  49.5  45.9  42.5
1Ø12  TIME  Ø.  DEPTHG  Ø.
1Ø13  26.1  26.1  26.1  26.1  26.1  26.1  26.1  26.1  26.1
```

READY
LIST HBOUT (Continued)

HBOUT 12:57EST 11/2Ø/7Ø

```
1Ø14  TIME  24Ø.ØØ  DEPTHG   9Ø.ØØ
1Ø15   97.2   96.1   88.3    79.4   72.Ø  66.2  61.6  52.4  46.2
1Ø16  TIME  241.ØØ  DEPTHG   3Ø.ØØ
1Ø17   95.6   95.7   88.2    79.4   72.Ø  66.2  61.7  52.4  46.2
1Ø18  TIME  269.ØØ  DEPTHG   3Ø.ØØ
1Ø19   56.3   78.Ø   79.9    74.9   69.5  64.7  6Ø.7  52.3  46.4
1Ø2Ø  TIME  339.ØØ  DEPTHG   2Ø.ØØ
1Ø21   42.Ø   52.6   62.6    63.9   62.3  59.8  57.3  51.Ø  46.Ø
1Ø22  TIME  469.ØØ  DEPTHG   1Ø.ØØ
1Ø23   34.Ø   35.9   43.3    48.1   5Ø.1  5Ø.4  5Ø.Ø  47.2  44.Ø
1Ø24  TIME    Ø.    DEPTHG     Ø.
1Ø25   26.1   26.1   26.1    26.1   26.1  26.1  26.1  26.1  26.1
1Ø26  TIME  24Ø.ØØ  DEPTHG  1ØØ.ØØ
1Ø27  1Ø5.1  1Ø3.8   95.2    85.3   77.1  7Ø.7  65.6  55.3  48.4
1Ø28  TIME  241.ØØ  DEPTHG   4Ø.ØØ
1Ø29  1Ø3.5  1Ø3.4   95.1    85.3   77.2  7Ø.7  65.6  55.4  48.5
1Ø3Ø  TIME  249.ØØ  DEPTHG   4Ø.ØØ
1Ø31   84.Ø   97.5   92.6    84.Ø   76.5  7Ø.4  65.4  55.4  48.6
1Ø32  TIME  296.ØØ  DEPTHG   3Ø.ØØ
1Ø33   51.1   7Ø.9   78.3    75.9   71.6  67.3  63.4  54.9  48.7
1Ø34  TIME  381.ØØ  DEPTHG   2Ø.ØØ
1Ø35   41.9   48.5   59.3    62.7   62.4  6Ø.8  58.7  52.9  47.9
1Ø36  TIME  532.ØØ  DEPTHG   1Ø.ØØ
1Ø37   34.Ø   35.Ø   4Ø.8    46.Ø   48.8  49.9  5Ø.Ø  48.2  45.3
```

READY
BYE
ØØØ3.Ø8 CRU ØØØØ.19 TCH ØØØ4.3Ø KC

OFF AT 13:ØØEST 11/2Ø/7Ø