SESSION VI
VENTILATION/GAS EXCHANGE/ CARDIOVASCULAR

49 RELATIONS AMONG GAS-PHASE DIFFUSIVITY, DENSITY AND PRESSURE IN GAS MIXTURES. H.D. Van Liew and C.V. Paganelli. Department of Physiology, State University of New York at Buffalo, Buffalo, NY 14214.

Diffusion of a particular gas in a mixture of three or more gases depends on diffusivities and concentrations of the other gases and also on environmental pressure. a) Estimates of effective gas-phase diffusivities in hyperbaric environments can be calculated from binary diffusivities by the Wilke equation. Sample calculations show that addition of CO_2 and H_2O to inspired gas has very little effect on diffusivity of O_2 but that neglect of lesser components of a mixture, such as the N_2 in "trimix" or the He in crude neon, would lead to errors of 10 to 20%. b) It is not possible to match a compressed air environment with a He-O_2 or a He-O_2-N_2 environment for both density and diffusivity. At the same environmental pressure, diffusivities of O_2 and CO_2 are greater in a mixture containing He than in a mixture containing N_2. However, at the same environmental density, diffusivities of O_2 and CO_2 in a He mixture can be less than half their values in air. Consider a He-O_2 environment that matches a certain air environment as far as density-dependent functions, such as pulmonary airflow resistance, are concerned. The He-O_2 environment would give rise to more hindrance to diffusive gas mixing in the lung than the density-matched air environment. A good overview of the effect of hyperbaric environments on gas-phase diffusivities can be obtained by a plot of diffusivity vs gas density. Most useful mixtures are included in a hyperbolic-shaped band; diffusivity falls to below 25% of the room air value when density is 5 times normal. (Supported in part by NHLBI Grant PO1-HL-14414.)

50 DENSITY DEPENDENCE OF HIGH FREQUENCY VENTILATION. J.R. Clarke and L.D. Homer. Naval Medical Research Institute, Bethesda, MD 20814.

High frequency ventilation (HFV) can in concept assist diver breathing during periods of high respiratory demand. However, the density dependence of HFV is poorly understood. We used a loudspeaker to assist mixing and diffusion between a mechanical compartment containing either O2 or SF6 (driving compartment) and another compartment (mixing compartment) containing air. The rate of nitrogen washout from the latter compartment was used as an index of mixing efficiency. The speaker was driven with either a sinusoidal or random signal and the spectral characteristics and power of the pressures generated in each compartment were determined by a Fast Fourier Transform Analyzer and PDP11/34 computer. For matched powers in the driving compartment, the rates of nitrogen washout were highly frequency dependent. The frequency for optimal N2 washout was much lower when the denser gas was used for washin. When powers were matched in the mixing compartment, however, the rates of N2 washout were independent of gas density. Density dependence of HFV, therefore, appears to be due to impedances between the driving and mixing compartments. Driving the system with random noise proved as effective as sinusoidal excitation, and minimized the changing of optimal driving frequencies under conditions of changing gas density.

51 GAS INERTIA MAY BIAS VENTILATORY MEASUREMENTS UNDER PRESSURE. D.D. Hickey, D.C. Marky,* and R.J. Smith†. Hyperbaric Res.

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A bag-in-box apparatus with a spirometer was used to measure the ventilatory minute volume in subjects exercising at air pressures up to 6.76 atm. During rest there was good agreement between minute volumes derived from the expired gas in the bag and the sum of tidal volumes from the spirometer, while during exercise the bag volume exceeded the spirometer volume by up to 15%. This was found to be due to the inertia of high density gas in the breathing hoses. Given a sufficient flow rate, the gas would continue to flow from the box to the bag following end-expiration and end-inspiration. The spirometer would not record this because it only sees changes in the sum of box and bag volumes, while emptying the bag through a gas meter will record the volume of gas actually moved. A model was constructed to investigate the phenomenon. It was concluded that many different conventional setups for respiratory measurements may be subject to this type of error. Solutions to the problem include a collapsible tube section downstream from the subject, a pneumotachograph or chest-mounted magnetometers or impedance devices.

This work was supported by ONR contract N00014-78-C-0205 with funds provided by the Naval Medical Research and Development Command.

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52 ARTERIAL PO2 AND GAS EXCHANGE AT 46.7 AND 65.6 ATA. E.M. Camporesi, J.V. Salzano, R.E. Moon and B. Stolp. F.G. Hall Environmental Laboratory, Duke University Medical Center, Durham, N.C. 27710, USA.

Arterial blood gases (ABG) were measured at rest and during the 6th min of graded steady state exercise in 3 subjects, during 2 dry chamber simulated dives at 47 ATA (gas density 8 to 12 g/L), and during one at 66 ATA (17 g/L). Breathing gas contained 0%, 5% or 10% N2 added to Heliox (.5 ATA O2). 1 ATA controls were obtained while breathing 50-50 Nitrox. VD/VT was calculated using the substitution PACO2 = PaCO2. Results at rest (r) and during the 6th min of work (w) at 720 kpm/min are summarized in the following table (PO2 in mm Hg; PB in ATA, gas density in g/L; VT in L BTPS and $\dot{V}E$ in L/min BTPS; each value mean of 3 subjects).

PB	Dens	PaO_2r	VTr	$\dot{V}Er$	VD/VTr	PaO_2w	VTw	$\dot{V}Ew$	VD/VTw
1	1.2	278	1.2	11	.34	269	1.8	49	.16
46.7	8	258	1.0	14	.49	262	2.3	59	.32
46.7	10	268	0.9	10	.45	247	2.4	55	.32
46.7	10	271	1.2	16	.51	267	2.5	55	.39
46.7	12	258	1.3	14	.51	255	3.0	48	.39
65.6	17	292	1.3	14	.50	295	3.2	42	.30

Variations in PaO2 at rest reflect slight variations in PIO2 with no systematic influence of inspired gas density or hydrostatic pressure. The PaO2 during an exercise which required approximately 60% of the surface $\dot{V}O2$ max was essentially unchanged from rest during each dive. Therefore a three- to five-fold increase in VE from rest to exercise, with gas densities ranging from 1.2 to 17 g/L, did not significantly alter arterial PO2. However, an increase in CO2 dead space (larger VD/VT ratios), not linearly related to gas density, developed at pressure, both at rest and during exercise.

Supported in part by NIH Grant #HL 07896.

53 ARTERIAL ACID-BASE PARAMETERS AT DEPTHS TO 650 METERS. J.V. Salzano, E.M. Camporesi, R.E. Moon and B. Stolp. F.G. Hall Environmental Laboratory, Duke University Medical Center, Durham, N.C. 27710, USA.

Arterial PCO2 and arterial pH (pHa) were measured at rest and during graded steady-state exercise at 1 ATA, during two simulated dives in the dry to pressures of 46.7 ATA and during one dive to 65.6 ATA. The inspired gas densities at 46.7 ATA varied from 8 to 12 g/L depending on whether 0%, 5% or 10% N2 was added to Heliox (0.5 ATA O2); at 65.6 ATA the inspired gas contained 10% N2 in Heliox and had a density of 17 g/L. A control period preceded each dive; three males served as subjects in each dive. The usual exercise responses were observed at 1 ATA: alveolar ventilation ($\dot{V}A$) increased in proportion to work rate up to 50-60% of $\dot{V}O2$ max; at higher work rates $\dot{V}A$ increased out of proportion to the increase in work rate. The onset of the alinear response of $\dot{V}A$ signifies the attainment of the anaerobic threshold (AT). Work rates above AT were accompanied, at 1 ATA, by hypocapnia and metabolic acidosis. The respiratory alkalosis partially compensated the metabolic acidosis, and the pHa at a work rate above AT (1080 kpm/min, pHa = 7.39) was only slightly less than at work rates at AT (720 kpm/min, pHa = 7.42). Results at pressure were different from controls but were not correlated with depth or gas density. Therefore data from each of the dives were pooled for comparison with pooled 1 ATA values. At depth: $\dot{V}A$ increased linearly with work rate presenting no ventilatory evidence of AT. Arterial pH, however, indicated the development of a metabolic acidosis and a "depth-AT" at work rates less than "surface-AT". In addition, hypercapnia was present at the work rates which resulted in metabolic acidosis. These results indicate that the inspired gas density and/or hydrostatic pressure blunt or prevent the ventilatory compensation for metabolic acidosis which normally occurs above AT. Therefore, at pressure an acidemia due both to hypercapnia and to metabolic acidosis will occur at work rates which are above "depth-AT".

Supported in part by NIH Grant #HL 07896.

54 EXERCISE CAPACITY OF DIVERS AT 41 BAR. K. Segadal*, R. E. Peterson, G. Bolstad* and J. E. Jacobsen*. Norwegian Underwater Technology Center, P.O. Box 6, N-5034 YTRE LAKSEVÅG, Norway.

An important aspect of practical diving operations at great depth is the exercise capacity of divers while breathing from an underwater breathing apparatus (UBA). Exercise studies conducted in the dry at high pressure with low resistance breathing assemblies (LR) have demonstrated the capability of men to perform hard work even when breathing extremely dense gases. In-water exercise studies at high pressure with relatively low density helium-oxygen mixtures have produced conclusions of severly restricted exercise capacity, however. To gain further insight into the work capacity of divers breathing from an UBA at high pressure, experiments were conducted at 400 msw during Deep Ex II. Six subjects each exercised 4 or 5 times on a bicycle ergomenter. Most of the exercise profiles were step-type starting at 50 watts and increasing by 25 watts each 6 minutes until exhaustion. Measurements included ventilation, lung volume variations, esophageal and mouth pressures, heart rates, mixed expired and breath-by-breath O_2 and CO_2 concentrations. The subjects breathed from a modified GSD-400 mask (open circuit demand UBA), a LR or were totally unrestricted. One run was conducted with the subject "semisubmerged". The other runs were done dry. Exercise was ventilation limited. Though some of the subjects experienced dyspnea even during their routine daily activities, all were able to work to loads of about 200 watts. Performances were not significantly different whether the UBA, LR or no breathing assembly was used. Exercise at levels above 225 watts could only be done for 1-4 minutes even by warmed-up fresh subjects who performed at those levels for 40 minutes or longer at sea level.

55 HIGH OXYGEN PRESSURES RETARD OXYGEN CONSUMPTION ON-RESPONSE TO ARM EXERCISE. D.D. Hickey, C.E.G. Lundgren, D.R. Pendergast*, and H.T. Swanson*†. Hyperbaric Res. Lab., Department of Physiology, State University of N.Y. at Buffalo, N.Y. 14214.

Divers frequently perform strenuous arm exercise. Compared to leg work, arm work is associated with a slower rise of O_2 uptake when work is begun. This study was undertaken to see if the O_2 on-response half time ($t1/2\ \dot{V}O_2$) in arm work could be reduced by increasing arterial O_2 content and pressure by hyperbaric O_2. The $t1/2\ \dot{V}O_2$ during a square wave load of 50 W arm exercise on an ergometer was studied in 6 subjects. Performance in hyperbaric O_2 (PO_2 2.24 atm and PN_2 0.76 atm) was compared to results in normoxia (PO_2 0.21 atm and PN_2 2.79 atm) at the same total pressure (3.0 atm). Oxygen uptake, CO_2 elimination, and minute ventilation were measured breath-by-breath during rest and 5 min of exercise. During hyperbaric O_2 inhalation there was a marked and significant increase in the $t1/2\ \dot{V}O_2$ by 38% compared to the normoxic situation. Likewise, the t1/2 for reaching a steady state CO_2 elimination increased by 53%. By contrast, there was no change in the rate at which minute ventilation increased. The retardation of the on-responses for O_2 and CO_2 exchange indicates an impediment to O_2 delivery, e.g. by vasoconstriction, and/or hindered O_2 utilization in the periphery. It may be speculated that this mechanism could induce early fatigue in divers performing arm work.

This work was supported by ONR contract N00014-78-C-0205 with funds provided by the Naval Medical Research and Development Command. †Authors' names in alphabetical order.

56 CARDIAC EXCITATION-CONTRACTION COUPLING EFFICIENCY DURING HYPERBARIC OXYGEN EXPOSURE AT 1000 FSW. T.J. Doubt and D.E. Evans. Hyperbaric Medicine Program Center, Naval Medical Research Institute, Bethesda, MD 20814.

Our previous work, in anesthetized cats with paced heart rates of 190-260 beats/min, has shown that heliox dives (pO_2 = 0.35 atm) to 1000 fsw lessen ventricular excitation-contraction (E-C) coupling efficiency. The present experiments examined E-C coupling relationships after a 30 min exposure to pO_2 = 2 atm (HBO) at 1000 fsw. The predive slope of Q-T interval of the ECG versus diastolic interval (0.56 ± 0.09, $p < 0.001$) was reduced to 0.35 ± 0.05 ($p < 0.001$) at 1000 fsw on heliox (pO_2 = 0.35 atm). After HBO exposure at 1000 fsw, Q-T was less at all paced heart rates, and the interval-duration slope increased to 0.40 ± 0.06 ($p < 0.001$). Predive levels of contractility (ratio of maximum +dP/dt to ventricular pressure at +dP/dt) were inversely related to Q-T (-0.19 ± 0.05, $p < 0.01$); with 1000 fsw heliox the slope was not significantly different from zero. The HBO exposure increased contractility values, but did not appreciably change the slope relation to Q-T. Time-to-peak ventricular pressure versus Q-T (0.79 ± 0.14, $p < 0.001$) was reduced by 1000 fsw heliox (0.27 ± 0.27), with partial restoration of the slope after HBO exposure (0.51 ± 0.13, $p < 0.01$). Developed left ventricular pressure (LVP) versus Q-T (0.61 ± 0.05, $p < 0.001$) was reduced at 1000 fsw heliox (0.52 ± 0.04, $p < 0.001$). The HBO increased LVP values and slightly increased the LVP vs. Q-T slope (0.55 ± 0.05, $p < 0.001$). Duration of ventricular systole versus Q-T (1.17 ± 0.17, $p < 0.001$) was reduced at 1000 fsw heliox (0.89 ± 0.19, $p < 0.001$, with no appreciable change after HBO. These results indicate that HBO may increase E-C coupling efficiency at 1000 fsw, possibly through steady-state metabolic effects. (Supported by NMRDC Work Unit MR0001.001.1269).