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To predict altitude decompression sickness (DCS) risk with any degree of accuracy, one must weigh variables such as prebreathe time, rate of ascent/descent, time at altitude, altitude, mixed breathing gas (dependent upon altitude), and profiles with multiple ascents and descents. The length of research chamber exposures is fixed. Therefore, risk assessment is based on DCS incidence after this fixed period at simulated altitude. From an operational standpoint, variable time at altitude complicates any predictive capability, although a computer model to handle all of these variables is in development. In the interim, a retrospective study from the Armstrong Laboratory Decompression Sickness Research Database has produced risk curves which can be used to predict DCS or venous gas emboli (VGE) incidence as a function of time at various altitudes. We limited the data to: 1) zero-prebreathe exposures to less than 20,000 ft breathing 50% O_2, 50% N_2; 2) zero-prebreathe exposures to less than 20,000 ft breathing 100% O_2; and 3) 1-h prebreathe exposures to greater than 20,000 ft breathing 100% O_2. Using the curves, one can select a time/altitude of exposure and estimate the DCS and VGE percentage.

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DECOMPRESSION SICKNESS RISK VERSUS TIME AND ALTITUDE

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ABSTRACT To predict altitude decompression sickness (DCS) risk with any degree of accuracy, one must weigh variables such as prebreathe time, rate of ascent/descent, time at altitude, altitude, mixed breathing gas (dependent upon altitude), and profiles with multiple ascents and descents. The length of research chamber exposures is fixed. Therefore, risk assessment is based on DCS incidence after this fixed period at simulated altitude. From an operational standpoint, variable time at altitude complicates any predictive capability, although a computer model to handle all of these variables is in development. In the interim, a retrospective study from the Armstrong Laboratory Decompression Sickness Research Database has produced risk curves which can be used to predict DCS or venous gas emboli (VGE) incidence as a function of time at various altitudes. We limited the data to: 1) zero-prebreathe exposures to less than 20,000 ft breathing 50% O_2, 50% N_2; 2) zero-prebreathe exposures to less than 20,000 ft breathing 100% O_2; and 3) 1-h prebreathe exposures to greater than 20,000 ft breathing 100% O_2. Using the curves, one can select a time/altitude of exposure and estimate the DCS and VGE percentage.

THE PROBLEM lies with the difficulty in predicting DCS risk during hypobaric exposures which do not follow precise ascent, isobaric altitude, and descent profiles matching appropriate research data. For example, a fighter pilot who cruises to the target area at high altitude, descends to deliver ordnance, and reascends for cruise to the recovery base experiences a cockpit pressurization schedule for which there is no available research data. A tactical airlift profile with repeated drops of equipment or personnel may involve several decompression-recompression events throughout the mission or on separate sorties the same day. We are frequently asked about DCS risk associated with scenarios such as these. The Armstrong Laboratory DCS research database contains data throughout the duration of exposure, typically 4-8 h. However, response to most requests has been based on DCS incidence at the end of the exposures. It might be preferable to present information based on altitude and length of exposure.

THE LONG-TERM SOLUTION will be an Armstrong Laboratory decompression model now under development that can provide predictive and real-time feedback on DCS risk during any hypobaric profile. The model will be based on currently available research data and on new data being collected during on-going exerimental protocols. VGE data will be included in the model because further decompression is possible after VGE formation that could promote growth of the gas emboli and increase DCS risk. This model will not be fielded for several years; however, there is an interim need for some method to estimate decompression risk versus time of exposure.

AN INTERIM SOLUTION to estimate decompression risk during relatively short exposures meant extracting experimental decompression risk data from the first 2 h of longer exposures. We retrieved this data from the Armstrong Laboratory DCS Research Database which included 301 relevant experimental exposures of male subjects to altitudes between 15,000 and 30,000 ft[1,4,5,8]. The voluntary, fully informed consent of the subjects used in this research was obtained as required by AFR 169-3. The subjects had passed a USAF Flying Class II physical examination and were otherwise representative of the USAF population. The subjects were exposed in groups of 3 or less, to decompression at 5,000 ft/min from ground-level pressure at Brooks AFB, Texas (745 mmHg) to chamber pressures of 8.3-4.3 psia (simulated altitudes of 15,000 ft - 30,000 ft) for 4-8 h. The breathing mixture at simulated altitudes exceeding 20,000 ft was 100% O_2 and was preceeded by 60 min of prebreathe with 100% O_2. At altitudes below 20,000 ft, no prebreathe was performed; the breathing mixture during exposure was 50% O_2 and 50% N_2 or 100% O_2. Each of the subjects was exposed from one to three times to a given pressure depending upon subject availability and protocol requirements, but only the results of the first exposure are used to provide consistent treatment of subject data. At altitude, the subjects performed exercises as described in TABLE 1.

During the chamber exposures, two different systems were used to monitor VGE[9]. The earlier dual-probe system consisted of a System 3 echo-imaging system from IREX Medical Systems and a Doppler ultrasound system consisting of a Bidirectional Doppler Model 1053 from the Institute of Applied Physiology and Medicine, Sound Products Division. The follow-on system consisted of a single-probe Hewlett-Packard SONOS 500 or 1000 precordial Doppler ultrasound and echo-imaging system. The sounds were recorded and graded according to the Spencer scale[6]. The time between VGE recordings was

TABLE 1. Profile Exercises and Number of Subjects versus Exposure Altitude

Exposure Altitude	Number of Subjects	Exercise, Exposure Duration, and Breathing Gas during Exposure
30,000'	31	Knee bends/Arm lifts[1]; 8-hours; 100% O_2
30,000'	23	Simulated EVA[2]; 4-hours; 100% O_2
29,500'	28	Rope pull[3]; 4-hours; 100% O_2
29,500'	8	Isotonic arm[4]; 4-hours; 100% O_2
27,500'	33	Knee bends/Arm lifts[1]; 8-hours; 100% O_2
27,500'	2	Isotonic arm[4]; 4-hours; 100% O_2
25,000'	27	Knee bends/Arm lifts[1]; 8-hours; 100% O_2
25,000'	13	Knee bends/Arm lifts[1]; 6-hours; 100% O_2
22,500'	19	Knee bends/Arm lifts[1]; 8-hours; 100% O_2
19,700'	10	Simulated EVA[2]; 6-hours; 100% O_2
18,000'	10	Simulated EVA[2]; 6-hours; 100% O_2
16,500'	10	Simulated EVA[2]; 6-hours; 100% O_2
16,500'	32	Simulated EVA[2]; 6-hours; 50% O_2:50% N_2
16,000'	25	Simulated EVA[2]; 6-hours; 50% O_2:50% N_2
15,000'	10	Simulated EVA[2]; 6-hours; 100% O_2
15,000'	20	Simulated EVA[2]; 6-hours; 50% O_2:50% N_2

Note: Some studies involved identical exercises and altitudes, but had different breathing mixtures during exposure or different total exposure durations as listed. A 1-h prebreathe with 100% O_2 preceeded all exposures above 20,000 ft.

1 Five chair-height deep knee bends and five arm-lifts of 5# weights every 15 min[3];
2 Cycle ergometer hand-cranking (24 rpm; 4 Newtons resistance), torque wrench actuation (25 ft-lbs), and rope pulling (76.6 Newtons resistance), for 4-min each, 3-4 cycles/h[8];
3 Rope pulling (76.6 Newtons resistance) for 5-min, 4 cycles per h[4]; or
4 Isotonic arm exercise for 5 min, 3 cycles/h[2].

approximately 15 min. The protocol called for descent at the first report of Grade 2 DCS joint pain (mild to moderate, constant pain[10]) or any more severe symptom, e.g. neurologic manifestations.

RESULTS in Figures 1-2 show cumulative DCS and VGE incidence versus time at altitudes from 22,500 to 30,000 ft. There was, in general, an increase in VGE and DCS incidence with higher altitude and with greater time at altitudes above 20,000 ft. Although the zero-prebreathe exposures to less than 20,000 ft produced no DCS within 2 h, VGE formation was observed within 25-50 min (Fig. 3 & 4) and 25-45% of the subjects breathing 50% O_2 and 50% N_2 had VGE at the end of 2 h at 15,000-16,500 ft. While breathing 100% O_2, the incidence of VGE at 15,000 and 16,500 ft was lower. This was shown more clearly in a direct comparison of severe (Grades 3 and 4) VGE data after 6 h exposures of subjects breathing either 50% O_2 and 50% N_2 or 100% O_2[11]. Use of 100% O_2 was shown to be advantageous in reduction of VGE.

LIMITATIONS include inability to predict risk when cockpit pressurization varies within a flight. If prebreathe varies from these experimental conditions or if other breathing mixtures are used, the validity of risk assessment becomes degraded. Variation in ascent rate or environmental temperature could influence the outcome as could variation in individual susceptibility, exercise performed while decompressed, and freedom to report symptoms without consequence.

DISCUSSION AND RECOMMENDATIONS center on use of the DCS/VGE prediction curves as an interim measure until the altitude decompression model is operational. The pilot of a USAF fighter aircraft with a 5-psid cockpit flying at 36,000 ft is breathing a mixed gas at a cockpit altitude of approximately 15,000 ft. An example of applying these curves would be for the pilot to refer to the 15,000 ft curve to determine risk level. Such an exposure while breathing

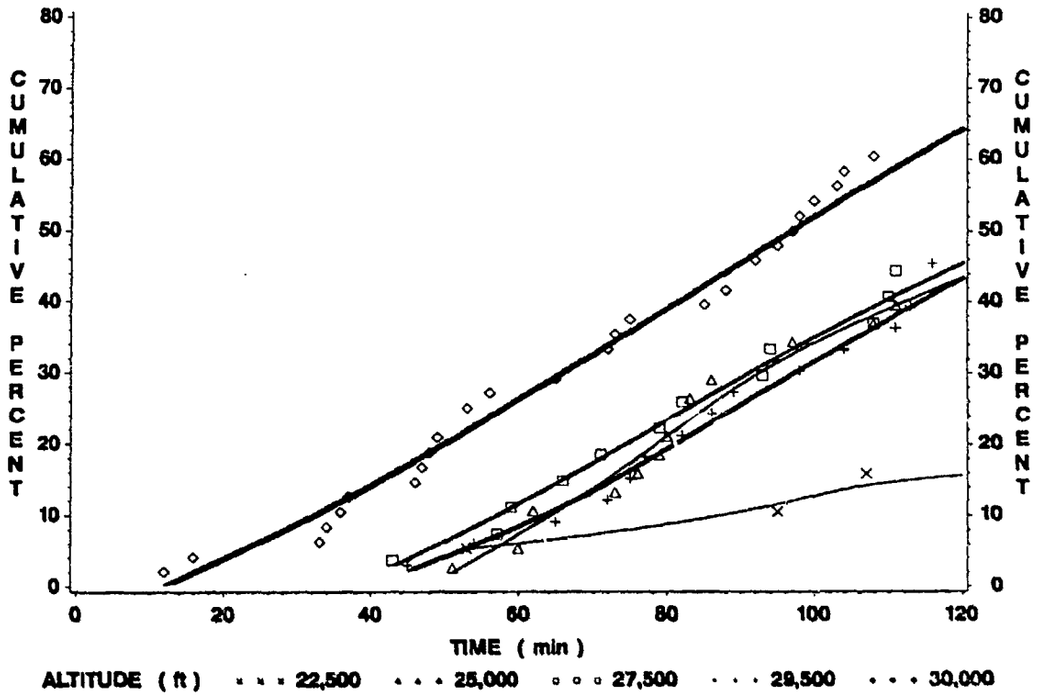

Figure 1. DCS versus Time at 22,500-30,000 ft (1-h prebreathe); 100% O_2 breathing gas during exposure

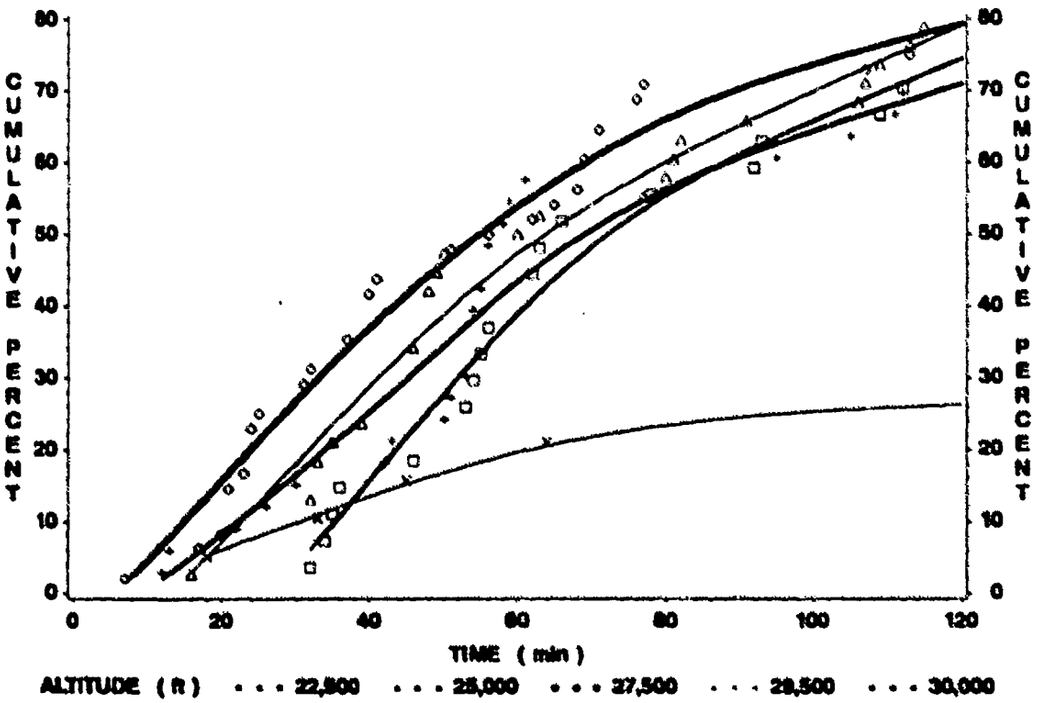

Figure 2. VGE versus Time at 22,500-30,000 ft (1-h prebreathe); 100% O_2 breathing gas during exposure

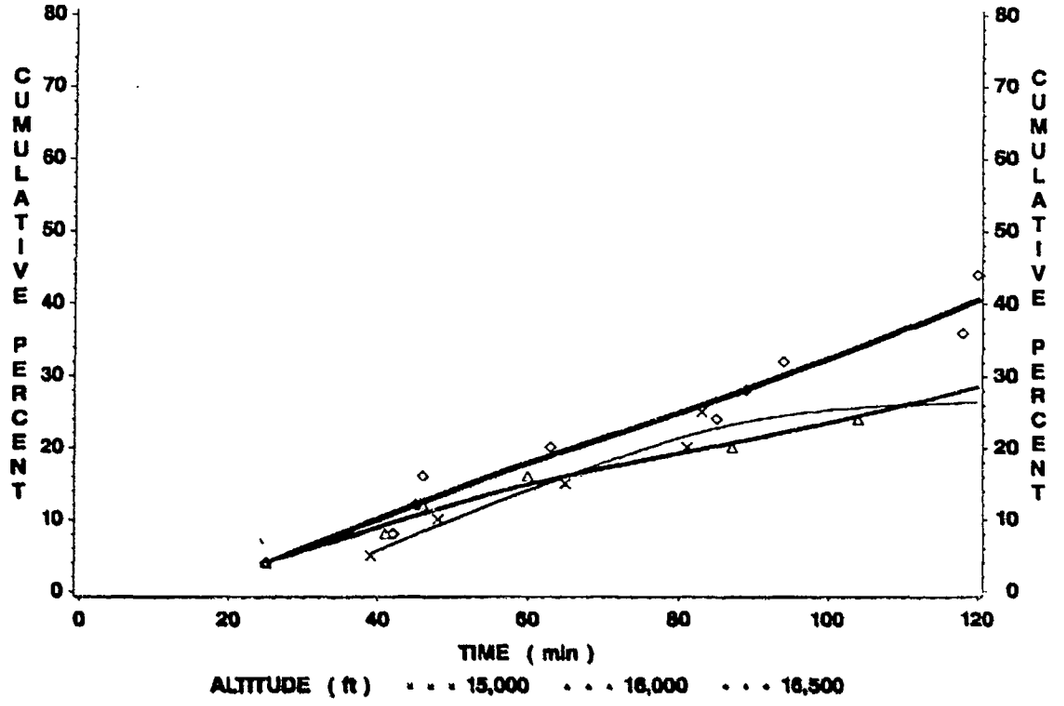

Figure 3. VGE versus Time at 15,000-16,500 ft (zero prebreathe); 50% O_2:50% N_2 breathing gas during exposure

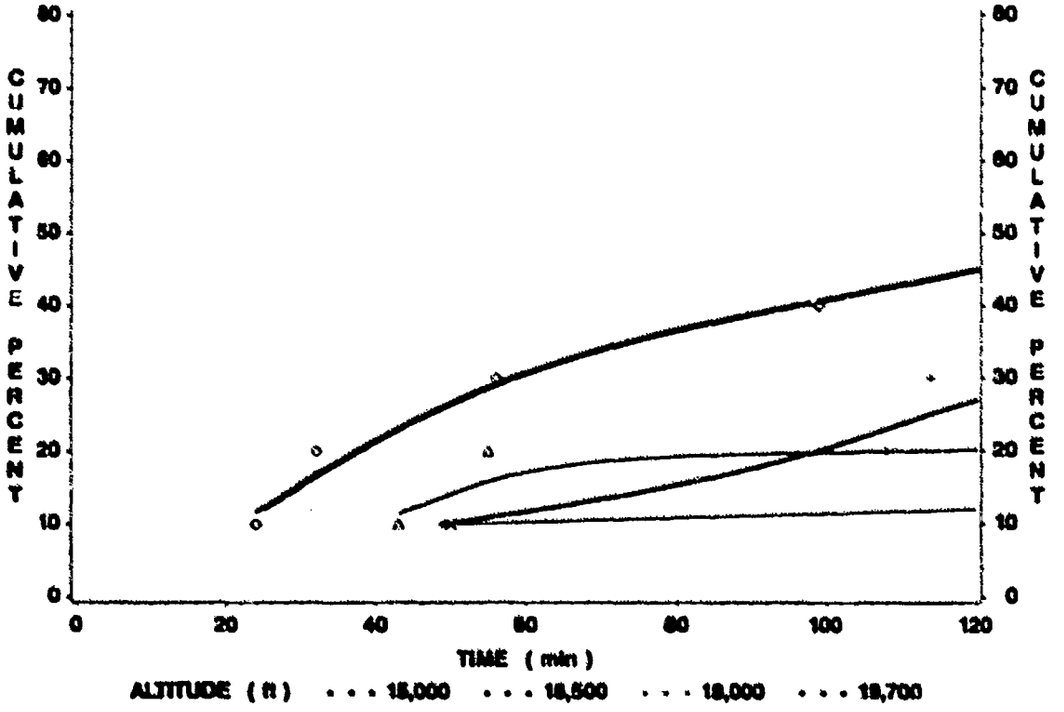

Figure 4. VGE versus Time at 15,000-19,700 ft (zero prebreathe); 100% O_2 breathing gas during exposure

50% O_2 and 50% N_2 (approximate mixture delivered to the pilot) is predicted to be DCS-free, but there is a 15% chance of VGE after 1 h (Fig 3). In the case of rapid decompression at that point from the loss of cabin pressure, the gas emboli would expand (Boyles Law). DCS symptoms may then develop with reduced or very limited latency. This risk would not be apparent to the pilot (unmonitored for VGE) without reference to the VGE curves. To reduce the risk of developing VGE at cockpit altitudes below 20,000 ft, breathing 100% O_2 during flights exceeding 30,000 ft in 5-psid cockpits is recommended as described in Webb et al.[7]

Incidence of DCS and VGE after only 1-2 h of exposure at 22,500 ft (Fig. 1-2) should raise concern since that level of decompression is consistent with future fighter operations at 60,000 ft with a 5-psid cockpit pressurization system. Flight at 60,000 ft should require, at a minimum, use of 100% O_2 from takeoff to descent[7]. An engineering solution could provide additional protection from DCS in future fighter aircraft. If such a solution is not constrained to the precedent of 40 years of fighter aircraft cockpit pressurization system design, changing the differential pressure maintained in the cockpit to 7 psid would provide considerably increased protection from DCS and VGE formation. Advanced personal equipment consisting of upper-torso counter-pressure garments may ameliorate the increased hazard due to the greater differential pressure experienced by the lungs during an explosive decompression at 60,000 ft. Further research is needed to demonstrate the adequacy of such protection and to confirm the effects of zero-prebreathe exposures to pressures simulating the full range of cockpit pressures expected in future aircraft.

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REFERENCES

1. Dixon GA, Adams JD, Harvey WT. Decompression sickness and intravenous bubble formation using a 7.6 psia simulated pressure-suit [illegible]. Aviat. Space Environ. Med. 1986;57:223-228.

2. Fischer MD, Wiegman JF, McLean SA, Olson RM. Evaluation of four different exercise types for use in altitude decompresion sickness studies. 30th Annual SAFE Symposium Proceedings. 1993;102-5.

3. Krutz RW Jr, Dixon GA. The effects of exercise on bubble formation and susceptibility at 9,100 m (30,000 ft; 4.3 psia). Aviat. Space Environ. Med. 1987;58:A97-A99.

4. Pilmanis AA, Olson RM. The effect of inflight deN2ation on altitude decompression sickness risk. [Abstract] Aviat. Space Environ. Med. 1991;62:452.

5. Smead KW. Preliminary findings: Bends screening index study. Space Life Sciences Symposium, Abstracts (unpaginated, unindexed addendum), Washington, DC, June 21-26. 1987.

6. Spencer MP. Decompression limits for compressed air determined by ultrasonically detected blood bubbles. J. Appl. Physiol. 1976;40:229-35.

7. Webb JT, Balldin UI, Pilmanis AA. Prevention of decompression sickness in current and future fighter aircraft. Aviat. Space Environ. Med. 1993a;64:1048-50.

8. Webb JT, Fischer M, Wiegman J, Pilmanis AA. Prebreathe enhancement with dual-cycle ergometry may increase decompression sickness protection. (Abstract) Aviat. Space Environ. Med. 1993b;64:420.

9. Webb JT, Olson RM, Baas CL, Hill RC. Bubble detection with an echo-image/Doppler combined probe versus separate probes: A comparison of results. (Abstract) Undersea Biomed. Res. 1989;16(supplement):89-90.

10. Webb JT, Pilmanis AA. Venous gas emboli detection and endpoints for decompression sickness research. 29th Annual SAFE Symposium Proceedings. 1991;20-3. SAFE J. 1992;22:22-5.

11. Webb JT, Pilmanis AA. Breathing gas of 100% oxygen compared with 50% oxygen:50% N2 reduces altitude-induced venous gas emboli. Aviat. Space Environ. Med. 1993;64:808-12.

BIOGRAPHIES

James T. Webb is a senior research scientist for KRUG Life Sciences Inc. in San Antonio. He has M.S. and Ph.D. degrees from the University of Washington and is board certified in Aerospace Physiology via the Aerospace Medical Association. Dr. Webb holds an Airline Transport Pilot certificate and has over 4300 flying hours including 250 combat hours in Vietnam (F-4Ds) and 2800 hours of C-141A experience. He is the 1993-1994 President of the Aerospace Physiology Society and is a principal investigator on several decompression sickness research protocols at Brooks AFB, TX.

Andrew A. Pilmanis is a research physiologist and Chief of the High Altitude Protection Function of the USAF Armstrong Laboratory's Crew Technology Division. He has M.S. and Ph.D. degrees in physiology from the University of Southern California (USC). Previously, he was on the faculty of the USC School of Medicine and director of their Hyperbaric Research and Treatment Facility on Santa Catalina Island. He was Program Director (1980-1985) for the joint NOAA/USC Undersea Research Program, responsible for the design and construction of the laboratory's saturation diving system (underwater habitat) Aquarius.