A comparison of acute Hypoxia induced by low concentrations of oxygen at ground level, 10,000 feet and by air at 25,000 feet. Implications for military Aircrew training.

George Evetts, Alice Hartley, Simon Keane, James Keegan,
Andrew Simpson, Alison Taylor, Alec Hurley and **John Ernsting.**

Kings College Aerospace Medicine Group, London
Royal Air Force Centre of Aviation Medicine, Henlow, UK

ABSRACT

Introduction. Acute hypoxia is a danger to all aviators. All military aircrew are given personal hypoxia experience in a hypobaric chamber as part of ground crew training, which is refreshed every 3-5 years. Current concerns with this training are the risks of developing decompression sickness (DCS). Other methods of training employing the use of reduced concentrations of oxygen at ground level and at 10,000ft in a decompression chamber have been considered. The latter was proposed by Cable and is used for hypoxia training in the Royal Australian Air Force. It has the advantage over ground level hypoxia in that it allows aircrew to experience the effects of low environmental pressure. Both methods have been investigated only once and require further validation.

Methods. Eleven subjects underwent three episodes of hypoxia breathing an inspired (tracheal) PO_2 of 49.1mmHg, equivalent to breathing air at 25,000 feet. Subjects breathed 6.9% oxygen at ground level, 10.3% oxygen at 10,000 ft, and 20.9% (room air) at 25,000ft. in a hypobaric chamber. End-tidal partial pressures of carbon dioxide and oxygen were measured together with pulmonary ventilation and arterial oxygen saturations. Heart rate and arterial blood pressure were also monitored. Hypoxic symptoms were recorded using a questionnaire which asked subjects to grade different symptoms in severity 1 to 5. Psychomotor performance was assessed using the 'manikin' computer task throughout the periods of hypoxia.

Results. Pulmonary ventilation was significantly higher during the hypoxia episodes at altitude. This was reflected in the end-tidal PCO_2 values, which became significantly lower at altitude. No other differences were found between the ground and 25,000ft hypoxia. The hypoxia episode at 10,000ft had statistically higher end-tidal PO_2 values, but only by 1-2mmHg. Heart rate and ventilatory response, along with arterial oxygen saturations were also different, but only by a small percentage. Symptoms were concordant across the three methods, in number, incidence, and severity. ‘Manikin’ performance data showed no difference in mean reaction time or mean percentage of tasks answered correct.

Discussion. The differences in pulmonary ventilation across the three methods were thought to be due to decreased air density and resistance at the lower barometric pressure. The differences at 10,000ft simulated altitude were operationally insignificant, with concordant symptoms, psychomotor performance and very similar physiological responses.

Conclusions. For the purposes of hypoxia training, inducing hypoxia using low concentrations of oxygen at ground level and at 10,000ft produces virtually the same physiological and performance responses and identical symptoms as breathing air at 25,000 ft.. Inducing hypoxia at 10,000ft has the advantage in that it provides the trainee with experience of reduced environmental pressure.

HYPOBARIC HYPOXIA is accepted as the most serious potential danger during flight. Harding and Mills (1) stated 'the fall in total barometric pressure and the consequent reduction in partial pressure of oxygen, poses the greatest single threat to anyone who flies.' The three main causes in aviation are ascent to altitude breathing air, failure of oxygen equipment, and rapid cabin decompression (2). Each of these results in a reduction of inspired oxygen partial pressure, either sudden or progressive in time course, depending on the rate of decompression, ascent, or extent of failure of the oxygen equipment. The rate of alveolar PO_2 reduction and end alveolar PO_2 determines the time course and severity of symptoms and physiological affects of hypoxia, which include dyspnoea, hyperventilation, parathesiae, sweating, light-headedness, visual disturbances, mental incapacitation, impaired learning, dizziness, euphoria, and unconsciousness (2). The most important effects in aviation are mental incapacitation, and unconsciousness. A decrement in behavioural processes such as short-term memory, selective attention, logical reasoning, and spatial orientation can have drastic affects on aircrew performance. Unconsciousness ensues if arterial oxygen is reduced to less than 30mmHg for an sustained length of time, depending on the degree of hypocapnia.

Hypoxia training is now provided to all military aircrew. Lectures on the effects of pressure change, hypoxia and hyperventilation are followed up by personal experience of hypoxia in a hypobaric chamber. 'Refresher' training is received every three to five years depending on the Air Force policy.

A typical hypoxia experience profile is as follows: (3)

1. Climb to 25,000ft at 4,000ft per minute breathing from oxygen regulator.
2. Removal of oxygen mask for hypoxia experience under direction and supervision of the medical officer.
3. Replace mask.
4. Descend to ground level at 4,000ft per minute.

The continuation of this training is supported by the incidence of decompression and hypoxia in aviation. Over the period of 1970-1980 there were 298 reported incidents in the United States Air Force (USAF) (4) with a significant number of aircrew recognising their symptoms due to recalling decompression chamber experience. Canadian Forces Ejection seat aircraft between 1962 and 1982 encountered 47 cases of cabin decompression at various altitudes from 15,000ft to 40,000ft (5). Several of these were also initially acknowledged by the pilot due to the presence of hypoxic symptoms. Canadian Forces Transport aircraft had 47 cases of cabin decompression over the period of 1963-1984, of which 85% were slow and 11% intermediate. In one case, hypoxia symptoms felt by the pilot were the only indication of a loss of cabin pressure (6). This data represents 20, and 22 years of peacetime. In the event of war this incidence has been shown to increase with 540 incidences recorded within a six-year period in operational aircraft (4). More recent data from the Australian Air force (7) supports that from USAF and Canadian Forces. All crew had undergone physiological training and out of 29 cases of hypoxia, only four cases went unrecognised. 20 members recognised their own symptoms, whilst 3 cases were recognised by another crewmember.

Cable concluded his report with a recommendation for continuation of hypoxia training since 75.8% recognised their own symptoms.

Hypobaric chamber training is clearly desirable, with a significant number of hypoxic incidences occurring, and a large number of potential accidents prevented through aircrew sign and symptoms recognition due to chamber hypoxia experience. However hypobaric chamber training comes with a risk of developing decompression sickness.

Decompression sickness is rarely reported below 18,000ft (8). Out of 58 cases of decompression sickness in United States Air Force (USAF), between 1970 and 1980, 53 were above 18,000ft, and none were reported below 10,000ft (9). The highest incidence reported in USAF during the years between 1970 and 1980 was at 25,000ft (9).

Incidence of DCS with hypobaric training is reported to be low, the incidence in US Navy Chambers being 0.029% 1972-1975 (11), and 0.07% 1981-1988 (12) in trainees. However the same authors report incidence amongst inside observers to be relatively high, 0.38%, and 0.25% respectively. All incidences occurred on exposure to 25,000ft.

This increased risk is supported by data from US Army hypobaric chamber use, which had a significantly higher incidence of DCS in observers compared to trainees ($p<0.01$), (10). There has been speculation as to the reason why a higher incidence amongst observers has been reported. Piwinski et al (10) suggested that this was due to the physical differences to the trainees. It was found that the age range of trainees 21-25, whilst the inside observers age range was 31-35. He continues, “In this approximate 10-year age difference, there is the probability of a higher body fat percentage and less strenuous physical activity in our technicians.” The exposure rates between trainees and observers are also incomparable, observers having multiple exposures in a short time span, a factor known to increase risk of DCS. However low, the risk of DCS is still present, and can ground an experienced airman or medical officer. A method proposed by Cable for hypoxia induction is the use of a reduced oxygen fraction at 10,000ft in a decompression chamber, equivalent to breathing air at 25,000ft. The method is in use by the Australian Air Force.

Reduced oxygen fraction has been shown to produce affects consistent with that of hypobaric hypoxia (12). Baumgardner et al (13) compared hypoxia induced at 18,000ft, and 25,000ft with equivalent reduced oxygen fraction at ground level. Cardio respiratory affects, mental performance, and hypoxic symptoms were recorded. Reduced oxygen fractions were 10.0% for 18,000ft comparison, and 7.1% for 25,000ft comparison. These produced inspired (tracheal) PO_2 within 2mmHg of that at corresponding altitude. The abstract concluded that for the purpose of demonstrating hypoxia to students, the symptoms, performance, and physiological changes were similar.

The mixed method proposed by Cable has yet to be validated This study re-evaluates the use of reduced oxygen fraction as an alternative to hypobaric exposure at 25,000ft, and compares the mixed method of hypoxia induction to that of pure hypobaric hypoxia.

METHODS

Subjects

Subjects were recruited form the postgraduate student population of Kings College London who provided signed informed consent to participate in the study. Health Screening was conducted by standard Royal Air force Health Questionnaires and examination by the Chamber Medical Officer. Ethical approval to conduct the study was provided by the King's College and Royal Air Force Centre research ethics committees.

Phases

<u>Ground level Phase</u>

- Inhalation of 6.9% Oxygen at the barometric pressure of ground level giving an inspired tracheal PO_2 of 49.1mmHg

<u>10,000ft Phase</u>

- Inhalation of 10.3% oxygen, at a barometric pressure of 523mmHg giving an inspired tracheal PO_2 of 49.1mmHg.

<u>25,000ft Phase</u>

- Inhalation of 20.9% oxygen, at a barometric pressure of 282mmHg, giving an inspired tracheal PO_2 of 49.1mmHg

Protocol

All phases were conducted in Number 1 hypobaric chamber at RAF Centre for Aviation Medicine, RAF Henlow, Hertfordshire. Sequence of altitude exposure was randomised. This minimised bias towards difference in symptoms and physiological change that could occur upon familiarization from repeated exposure to hypoxia. Subjects were also blinded as to which exposure they were receiving. Mimic of decompression at ground level was accomplished by passing of air through the open valves creating the noise of decompression. Subjects were also unaware as to when the hypoxic gas was administered. (A sign was held to the window behind of the subjects for the information of the medical officer.)

All subjects were administered a hyperoxic gas mixture during decompression, before hypoxia, based on what the regulator would deliver at that altitude.

Each phase comprises of the following general form

1. Control period breathing room air
2. Breathing of hyperoxic gas mixture
3. Decompression
4. Exposure to hypoxic gas mixture
5. Recovery with hyperoxic mixture
6. Recompression to ground level

All gases were delivered through the Type 517 pressure demand oxygen regulator attached to a RAF P/Q oronasal mask. Oxygen and carbon dioxide partial pressures at the mouth for end-tidal partial pressures ($P_{ET}\ O_2$ and $P_{ET}CO_2$) were measured using an AirSpec QP 9000 Spectrometer. Pulmonary ventilation was measured with a Fleisch capillary flow meter with a P.K. Morgan Ltd. pressure transducer indicator and digital integrator. Respiratory rate was recorded from the frequency of the carbon dioxide tension wave using the computerised chart recorder v4.3 capability. Arterial oxygen saturations and blood pressure were measured by means of a standard oximeter (Kontron) and digital artery pressure recorder (Finapres^{TM}) respectively. A modified lead II electrocardiogram was displayed on a cardiac monitor (Morgan 509) for safety purposes. All variables were recorded using a power lab interface with Chart v4.3 for windows software, and also Graphtec Linearcorder WR 3320 for hard copy.

Psychomotor performance (complex reaction time) was measured using a computer based spatial orientation task, "Manikin" which involved the determination of a flag holding hand of the 'Manikin' in one of four orientations. The analysis program calculated mean reaction time, and mean percentage correct answers for each minute of the exercise. The programme has been demonstrated to show decrements in psychomotor performance at altitude of 5,000ft (15)

A subjective Symptoms Questionnaire was employed in order to compare symptoms experienced during each hypoxic exposure.

Safety

A Royal Air Force physician, experienced in hypoxia training, supervised all subjects. Hypoxia was terminated after 5 minutes or upon any of the following.

- The subject requested cessation of hypoxia
- The subject failed to respond to the manikin task for a continuous period of 15 seconds.
- End-tidal PO_2 fell to 30mmg
- Arterial saturations fell to 60%
- A Sudden fall in heart rate of greater than 20 beats/min
- A decision by the physician to end the hypoxia.

ANALYSIS

To standardise for differing durations of hypoxia, time scales were normalised to 100%, allowing comparison mean values for each tenth of hypoxia. Variables were compared by single analysis of variance (ANOVA) with replication. Where normal distribution was found, simple one-way ANOVA was used. Where normal distribution was not found, non-parametric Friedman repeated measures analysis of variance on ranks was used for comparison. Symptoms data was compared on symptoms score, incidence of separate symptoms and severity of each symptom using single analysis of variance (ANOVAR) with replication. Where significance was found with ANOVA, post-hoc analysis of means was used to determine the difference. A confidence interval of $p<0.05$ was assumed.

RESULTS

Twelve subjects were recruited onto the study, of which eleven successfully underwent all three hypoxia phases. The times for P_{ET} O2 to decrease from 49.1mmHg to 30mmHg showed no significant difference at the three barometric pressures ($p=0.275$) (Table I). $P_{ET}O2$ values at 10,000ft were consecutively

~1mmHg higher than at ground and 25,000ft barometric pressures at all time points of hypoxia (p=<0.05) (figure 1).

ALTITUDE (feet)	MEAN HYPOXIA TIME (Sec ± SE) N=11
GROUND	**140.73** ± 14.56
10,000	**118.82** ± 11.17
25,000	**142.91** ± 15.21

Table I. Mean duration of hypoxia at the three barometric pressures

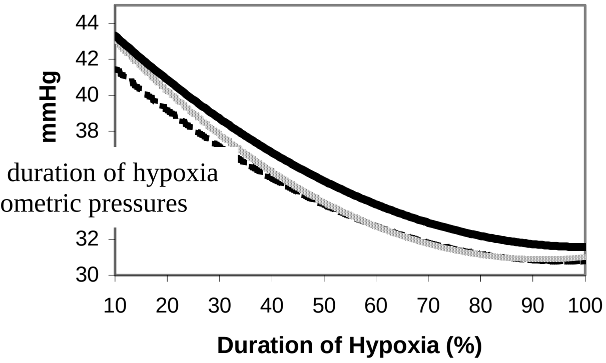

Fig 1. Mean PETO2 of subjects during hypoxia
Ground (broken),
25,000ft (grey)
10,000ft (black)

S_aO_2 was highly correlated with $P_{ET}O_2$ at all barometric pressures, regression giving equal gradients and intersects. S_aO_2 followed the same trend as $P_{ET}O_2$, 10,000ft being significantly higher during hypoxia than ground and 25,000ft ($p<0.05$) (fig 2). There was no change in respiratory rates at the three barometric pressures.

A decrease in barometric pressure induced significantly higher pulmonary ventilation rates ($p<0.05$), although baseline values were not significantly different ($p>0.05$). The pulmonary ventilation change during hypoxia was less at 10,000ft than 25,000ft and ground level, the latter being significant ($p<0.05$). End point mean pulmonary ventilation (l/min BTPS) increase from baseline during ground and 25,000ft simulated altitude was >20% higher than at 10,000ft simulated altitude (table II).

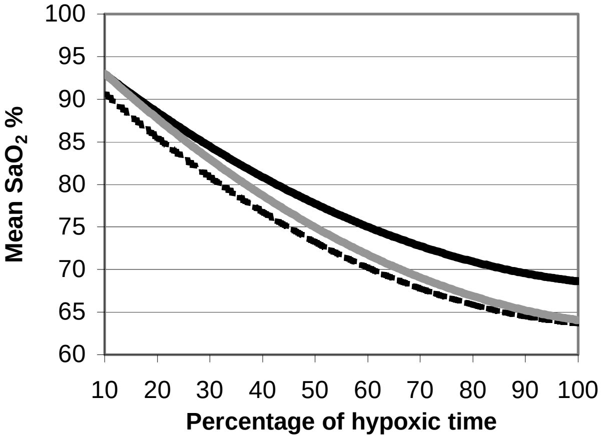

Fig 2. Mean SaO2 of subjects during hypoxia
Ground (broken line)
25,000ft (grey), and
10,000ft (black)

Table II. Total increase in Pulmonary ventilation from baseline at ground, 10,000ft and 25,000ft

ALTITUDE (feet)	TOTAL % INCREASE FROM BASELINE(mean ± SE)
GROUND	**59 ±** 12
10,000ft	**28 ±** 16
25,000ft	**56 ±** 20

Baseline $P_{ET}CO_2$ followed pulmonary ventilation, being significantly higher at ground level than at 10,000ft and 25,000ft simulated altitudes ($p<0.05$) (table III), and decreasing less at the 10,000ft simulated altitude than 25,000ft and ground level studies. However this was not significant. Change in heart rate during hypoxia was significantly less at 10,000ft simulated altitude than ground and 25,000ft simulated altitudes (fig 3).

ALTITUDE (feet)	MEAN±SE OF $P_{ET}CO_2$ mmHg (n=11)
GROUND	**38.25** ± 0.69
10,000	**34.92** ± 1.50
25,000ft	**35.07** ± 1.28

Table III. Mean baseline $P_{ET}CO_2$ ± SE (mmHg) of subjects at ground, 10,000ft and 25,000ft.

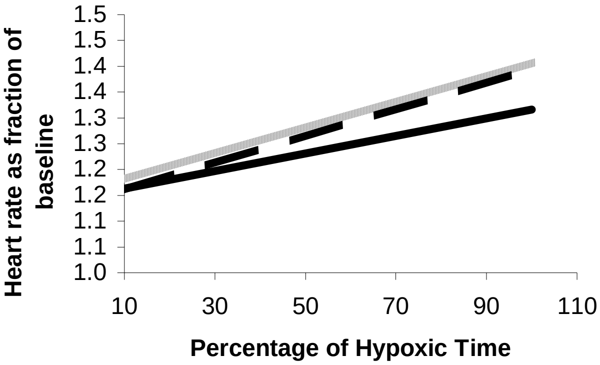

Fig 3. Mean change in heart rate of subjects during hypoxia.
Ground (broken line),
25,000ft (grey)
10,000ft (black)

Systolic, diastolic and mean arterial blood pressure changes with hypoxia were not significantly different between the three barometric pressures. (p=0.921, 0.676, and 0.849 respectively). Symptoms recorded by the symptoms questionnaire were not significantly different in number, score, frequency, or severity during hypoxia at the three barometric pressures. (p=0.425, 0.458, 0.296, and 0.154 respectively).

Tables IV and V show mean symptom number and score respectively. Analysis of the ‘manikin’ data revealed no significance in mean reaction time, and accuracy across the three hypoxic episodes. ($p>0.05$).

ALTITUDE (feet)	MEAN SYMPTOMS NUMBER (±SE) N=11
GROUND	**5.27** ± 0.81
10,000	**5.64** ± 1.14
25,000ft	**6.36** ± 0.85

Table IV. Mean symptoms number at each barometric pressure.

ALTITUDE (feet)	MEAN SYMPTOMS SCORE (±SE) N=11
GROUND	**15.00** ± 3.61
10,000	**16.55** ± 4.96
25,000ft	**17.91** ± 3.28

Table V. Mean symptoms score at each barometric pressure.

DISCUSSION

The results did highlight some subtle differences in cardio respiratory changes in response to hypoxia. $P_{ET}O_2$ was consistently higher during hypoxia at 10,000ft. This was reflected in arterial oxygen saturations, and heart rate change. Pulmonary ventilation also differed, which was thought to be due to decreased air density at the higher altitudes. $P_{ET}O_2$ differences amounted to only 1-2 mmHg, which would be physiologically and operationally insignificant, particularly in respect to providing experience of hypoxia to aircrew. More importantly the symptoms and psychomotor performance data was consistent across the three methods. The previous study by Baumgardner et al, (15) had similar findings, in that the 18,000ft method of hypoxia gave higher $P_{ET}O_2$ and arterial saturations and less heart rate increase. Symptoms and psychomotor performance also showed no difference.

CONCLUSIONS

Using a reduced oxygen fraction at ground level and at 10,000ft simulated altitude is valid as a technique for hypoxia experience training for military aircrew. The mixed method using a gas mixture at 10,000ft has the advantage over ground level experience in that it allows aircrew to experience the physiological affects of a reduced pressure environment. Reported decompression sickness risk is zero, thus it allows medical personnel to undergo several training sessions each day, in contrast to training at 25,000ft which has safety limitations.

References

1 Harding, R.M., & Mills, J.F., 1988. Aviation Medicine 2nd Edition. British Medical Association Publications.

2 Harding R.M., revised by Gradwell, D.P., 1999b. Hypoxia and Hyperventilation. Aviation Medicine, Ernsting, J., Nicholson, A., Rainford, D. 3rd Ed. Butterworth Heinemann. Ch2:p43- 58.

3 Royal Air Force, 2002. Experience of Hypoxia at 25,000ft Feet. Official document TM33. Ch 2:p2. Unpublished.

4 Rayman, R.B., & McNaughton, G.B., 1983a. Hypoxia: USAF Experience 1970-1980. Aviat. Space Environ. Med. 54(4):357-359.

5 Brooks, C.J., 1984. Loss of cabin Pressure in Canadian Forces Ejection Seat Aircraft, 1962-1982. Aviat. Space Environ. Med. 55:1154-63.

6 Brooks, C.J., 1987. Loss of Cabin Pressure in Canadian Forces Transport Aircraft, 1963-1984. Aviat. Space Environ. Med. 58:268-75

7 Cable, G.G., 2003. In-Flight Hypoxia Incidents in Military Aircraft: Causes and Implications for Training. Aviat Space and Environ. Med. 74:169-172

8 Piwinski, S., Cassingham, R., Mills, J., Sippo, A., Mitchell, R., Jenkins, E., 1986. Decompression Sickness incidence over 63 months of hypobaric chamber operation. Aviat. Space Environ. Med. 56:1097-101.

9 Rayman, R.B., & McNaughton, G.B., 1983b. Decompression Sickness: USAF Experience 1970-80. Aviat. Space Environ. Med. 54(3):258-260.

10 Bason, R., Pheeny, H., & Dully Jr., F.E., 1976. Incidence of decompression sickness in Navy low-pressure chambers. Aviat. Space Environ. Med. 47(9):995-997

11 Bason, R. & Yacavone, D., 1991. Decompression Sickness: U.S. Navy Altitude Chamber Experience 1 October 1981 to September 1988. Aviat. Space Environ. Med. 62:1180-4

12 Sausen, K.P., Wallick, M.T., Slobodnik, B., Chimiak, J.M., Bower, E.A., Stiney, M.E., Clark, J.B., 2001. The reduced oxygen breathing paradigm for hypoxia training: physiological, cognitive, and subjective effects. Aviat. Space Environ. Med. 72:539-45

13 Baumgardner, F.W., Ernsting, J., Holden, R., & Storm, W.F., 1980 Responses to hypoxia imposed by two method. Abstract of papers given to Aerospace Medical Association Scientific Meeting.