

Abstract

There is currently a renewed interest in the design of supersonic aircraft and other subsonic aircraft that will fly at higher altitudes (approximately 50,000 to 60,000 feet). These relatively higher altitudes hold the possibility of exposing passengers to greater degrees of hypoxia than in the current generation aircraft and therefore the Federal Aviation Administration (FAA) is interested in quantifying these risks. Primarily they are interested in creating a model with which they can predict permanent neurological damage in the event of a rapid decompression. There is a lack of data in this area and thus the current study was proposed.

Decompressions from 8,000 feet to 25,000 feet were performed in two time periods: 20 seconds and 90 seconds. 13 Subjects were used and 22 decompressions were performed, 3 decompressions were aborted and out of the remaining runs nine were 90s decompressions and ten were 20s decompressions. Certain physiological parameters were measured continuously throughout the decompressions, these were: pulmonary ventilation, blood pressure, heart rate, oxygen saturation of haemoglobin and end-tidal partial pressures of oxygen and carbon dioxide.

It was found that pulmonary ventilation rates did not change significantly, systolic blood pressure increased during the slow decompressions, heart rate increased in both decompression profiles, oxygen saturations dropped in both profiles, end-tidal partial pressure of oxygen reduced in a linear fashion with decreasing barometric pressure and end-tidal partial pressure of carbon dioxide decreased slightly.

Comparison between the two rates of decompression found that there was only one area where a difference occurred. This was in blood pressure, where systolic blood pressure increased in the slow decompression but did not do so in the fast decompression. All other measurements were not significantly different thus it was concluded that as far as this study was concerned the rate of decompression had next to no influence upon the measured variables.

In conclusion further studies have to be carried out using higher final altitudes, as this is more realistic, a greater number of subjects and with a few more different rates of decompression. Ideally all the subjects should perform an equal number of each decompression profile. This study is set to become the first in a series of studies into this area.

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Introduction

Aviation medicine is a new and developing field of research. As the face of aviation changes with modern demands and better technology, medical research into areas that were previously not viable in this industry now becomes necessary and vital. The following report is concerned with one such area. We look forward to addressing some of the problems that may result from the new generation aircraft.

The Need for this Research

In the current day and age, aviation has become convenient and efficient. Flights over greater distances are possible. The one variable that has not changed very much over the past few decades is the speed of travel. Passenger aircraft, except for the Concorde service (which has now been discontinued), are all subsonic. Supersonic and next-generation aircraft are likely to fly at higher altitudes, probably in the range of 50,000 to 60,000 feet. In anticipation of this, the Federal Aviation Administration (FAA) is interested in creating a model to quantify the risks of neurological damage in the event of an accidental decompression whilst at these relatively high cruising altitudes. Specifically, they would like to determine the degree of hypoxia that would cause permanent brain damage or prove to be fatal. There are only a few studies that have been conducted that provide data for such a model (Nicholson & Ernsting - 1967), (Brierley & Nicholson – 1969).

The fact that the final altitude is higher than the current levels, 50,000 ft as compared to 35,000ft, means two things; firstly, that decompression will occur at a faster rate and secondly, that the maximum cabin altitude reached will be higher (assuming that the other variables such as cabin space and defect size are the same in both models). A faster decompression and exposure to higher cabin altitudes equates to a more acute and greater degree of hypoxia than in the current generation aircraft. It is in this area that this study concentrates.

Physiological Aspects of Rapid Decompressions

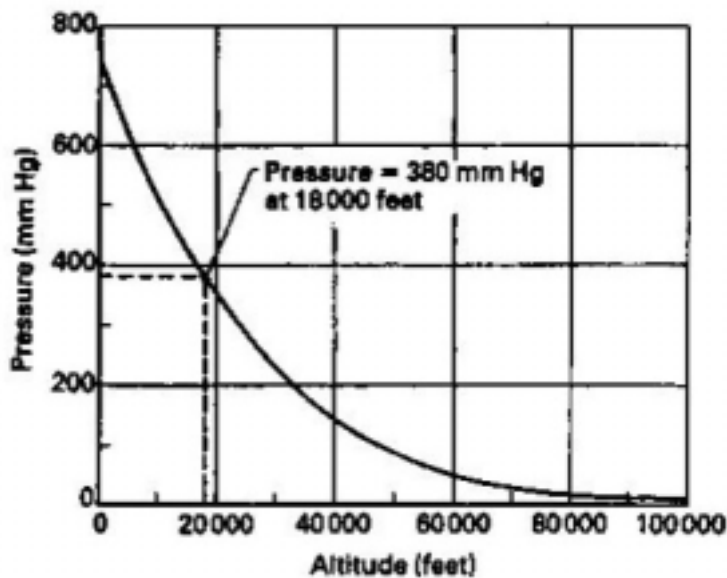
It is now necessary to provide a brief outline of the physiology related to this experiment.

Reduction in Barometric Pressure with Increasing Altitude

The reduction in the partial pressure of oxygen that occurs with increasing altitude poses the most important physiological danger in high altitude flying (RM Harding & Gradwell). The collection of gases that compose the atmosphere are exposed to two opposing factors: the heat from the sun and the force of gravity. The thermal energy from the sun acts to expand the gases and allow the molecules to escape the forces of attraction between them, thus resulting in molecules escaping into the vacuum of space at the outer edge of the atmosphere. The force of gravity on the other hand, pulls the gases towards the earth and causes increased density closer to the surface of the earth, where gravity can

have its greatest effect and the thermal energy from the sun its least effect. Therefore, barometric pressure decreases as we ascend from ground level. This occurs in exponential fashion, as is shown by Figure 1.

Figure 1 : The decay of barometric pressure with altitude



(Figure taken from: Figure 1.2 P6 Ernsting – Aviation Medicine)

The fractional concentrations of the two main gases (oxygen and nitrogen) and the other minor gases that constitute the atmosphere, however, stay fairly constant with ascent between sea level and approximately 300,000 ft (Chapter 1 – RM Harding). Taking these

two factors into consideration, it can be derived that the partial pressures of each gas will fall as they form a constant fraction of a decreasing barometric pressure.

*N.B. Alveolar gas is representative of the gaseous environment in the body (Boothby - Chapter 3 - 1954) whereas the inspired gas is not. Therefore, alveolar gas fractions are the vital measurement used throughout this study.

Outline of Gas Exchange – Alveolar Ventilation

Gas exchange occurs across the alveolar and pulmonary capillary membranes via passive diffusion. The diffusion gradient is largest at the pulmonary artery end. As gas passes from the alveoli across the membrane into the blood, this gradient decreases until about one third of the way along the pulmonary capillaries and blood partial pressures of oxygen become nearly equal to the alveolar partial pressures. Oxygen enters the blood and carbon dioxide is removed from it.

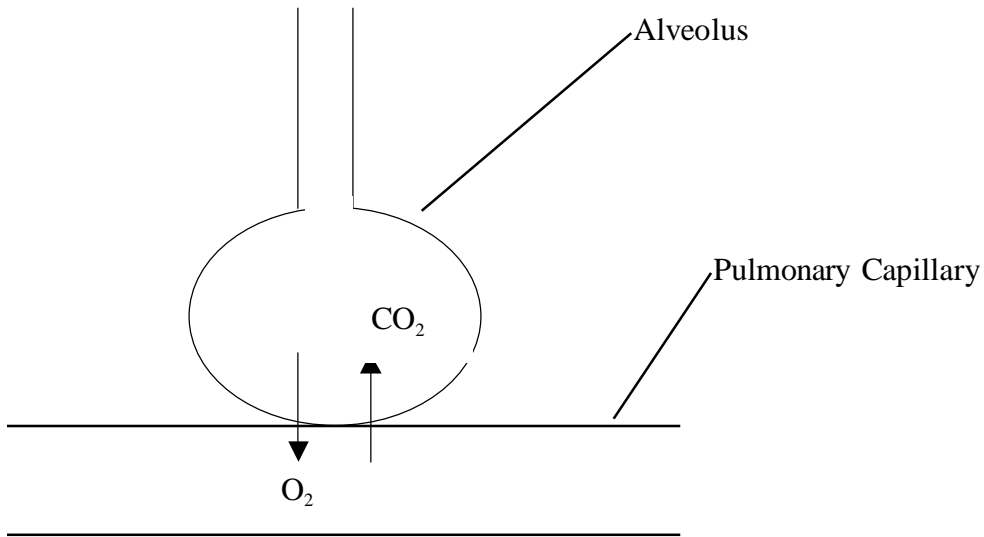


Figure – Simple diagram of gas exchange

The two main factors that vary and thus modulate this diffusion from the alveolar gas to the blood are the diffusion gradient and the rate of blood flow. The diffusion gradient for oxygen is decreased in hypoxia as the alveolar tension of oxygen is reduced and therefore transit time may not be enough for equalization of partial pressures to occur.

It is important to understand that partial pressure in the mixed pulmonary venous blood is about 4 mmHg less than that of the mixed alveolar gas (Chapter 4 – Ernsting) because of the ventilation-perfusion inequality of the upright lung. However, sampling the alveolar gas tensions allow us to estimate the arterial blood gas tensions (Boothby - Chapter 3 – 1954). Expired gas consists of alveolar gas and the inspired gas that remained in the functional dead space during gas exchange in the alveoli. Therefore, end tidal gas would be approximately the same as alveolar gas, except for slight dilution with previously

inspired gas.

Partial Pressure of Oxygen on Acute Exposure to Hypobaric Conditions

The difference between the inspired partial pressure of oxygen and the alveolar partial pressure of oxygen is a function involving the fractional concentration of oxygen being breathed, the alveolar PCO₂, and the respiratory exchange ratio. This is shown by the equation below – (The Alveolar Air Equation).

$$P_{I}O_2 - P_{A}O_2 = P_{A}CO_2 \left[F_{I}O_2 + \frac{1 - F_{I}O_2}{R} \right]$$

Where R = respiratory gas exchange ratio.

The equation above shows that a decrease in the P_ACO₂ will result in a decrease in the difference between inspired and alveolar partial pressures of oxygen. This is noteworthy when exposed to altitudes above approximately 10,000 ft when a hypoxic ventilatory drive occurs and alveolar PCO₂ is reduced. This will be explained under the carbon dioxide section.

This reduction in partial pressure of oxygen in the ambient air means that there is a decreased PO₂ in the inspired gas, which results in a lower alveolar partial pressure of oxygen (P_AO₂). This is important in that the reduced alveolar PO₂ will lead to a decreased

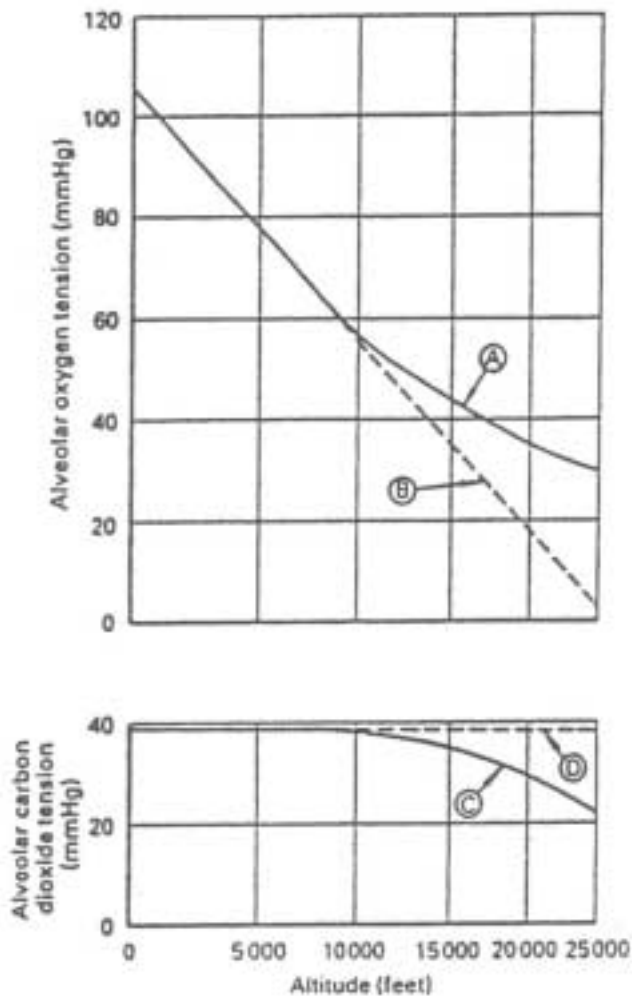
oxygenation of the pulmonary blood, which can then lead to impairment of function by reducing the level of oxygen available to cerebral tissues. Mild hypoxia, akin to ascending to a level of 8,000ft, is too small a level of hypoxia to affect oxidative phosphorylation, but probably acts to cause impairment via slowing the activity of oxygenases which are involved with synthesis of neurotransmitters (Ernsting - Patterson Lecture 1995), (Ernsting - Chapter 5 (RM Harding)).

Partial Pressure of Carbon Dioxide on Acute Exposure to Hypobaric Conditions

In contrast to $P_{A}O_2$, the alveolar partial pressure of Carbon dioxide ($P_{A}CO_2$) is independent of the environmental pressure and unlike the reduction of $P_{A}O_2$ that occurs with ascent, $P_{A}CO_2$ does not decrease. This is true so long as the ratio of the production of CO_2 in the body to the alveolar ventilation stays constant. $P_{A}CO_2$ is dependent on this ratio.

Therefore, with a constant ventilatory rate, $P_{A}CO_2$ does not change. However, hyperventilation occurs as a result of the PO_2 level falling below a level of 55mmHg to 45mmHg. Below this level, a hypoxic drive to ventilation is induced and alveolar ventilation is increased. As stated above, PCO_2 is dependent on the ratio of CO_2 production to alveolar ventilation and thus as alveolar ventilation is increased and 'blows off' CO_2 at a faster rate, a resultant decrease in the level of CO_2 occurs. This then results in a decreased difference between inspired partial pressure of oxygen and the alveolar partial pressure of oxygen.

Effect of Altitude on alveolar partial pressure of oxygen and carbon dioxide



(Figure taken from : Figure 5.1 P46 Ernsting – Aviation Medicine)

This hypocapnia can also further limit (in addition to the lowered oxygen tension in the cerebral blood) the amount of oxygen the cerebral tissues receive through the contraction of the cerebral vessels that this would cause and by increasing the alkalinity of the tissues. Beyond 15,000 ft, however, vasodilation occurs and this blood flow limiting effect is lost.

Impairment Levels

The degree of impairment that is found with reduced alveolar oxygen tensions is well documented. This allows us to devise a safe model within the design of an aircraft that should limit attempt to limit accidental exposure. However, one must keep in mind that there is a high degree of variability between individuals in the level of impairment at a specific inspired PO_2 .

It has generally been found that 8,000 ft is the maximum cabin altitude for passenger craft, where breathing air is the norm (Ernsting – 1984). Even at this altitude, however, complex tasks and the ability to learn novel tasks is impaired, as is "light sensitivity of the dark adapted eye" (Ernsting - Chapter 5 – (RM Harding)). Below a level of 10,000 ft (approx 55mmHg P_{AO_2}), the performance of well-learned tasks is not significantly impaired. Muscular in-coordination can occur at levels of above 15,000ft. Memory is impaired above the levels of 8,000 to 10,000ft. Cognitive task performance starts decreasing above a level of 10,000ft, and beyond this starts to rapidly decay.

Thus, it can be seen that in order for operating aircrew to safely handle the aircraft, adequate oxygenation has to be maintained. This level of oxygenation requires a cabin altitude of 8,000 ft or less whilst breathing air (Ernsting - Patterson lecture 1995). Other researchers (Marotte et al – 1990) have suggested that this maximum altitude be even lower, between 5,000 to 6,000ft, as there is a level of impairment in certain functions

even between 6,000 and 8,000ft. Other researchers who have found no detriment in function between these altitudes contest this information, however (Paul & Fraser – 1994). This is less important in the case of a healthy passenger at rest for whom a slightly higher cabin altitude may be acceptable, as the passenger is not actively involved with the operation of the aircraft and also is most likely to be at rest, whereas the crew may be in a state of activity (exercise exacerbates the effects of hypoxia). Even though studies have not found any significant deterioration in performance of well learnt tasks at an altitude of 8,000 ft, the fact that novel learning is impaired means there is a reduced ability to cope with an emergency situation which can occur in the air.

It has been established that a reduction of the alveolar PO_2 below 30mmHg is the critical level beyond which unconsciousness and brain damage/death can occur (Ernsting – 1978). Spatial orientation tasks and the ability to recall a sequence of newly learnt operations is lost once the arterial oxygen partial pressure has dropped below 30mmHg (Ernsting & Billings - 1974). An area, bounded above by the 30mmHg oxygen tension line and below by the alveolar oxygen tension, of or exceeding about 140mmHg will result in the loss of consciousness. For these reasons it is vital to prevent an exposure that would cause such a profound drop in oxygen partial pressures.

Cabin Pressurization and its Failure

The section above demonstrates the need for cabin pressurization, as most passenger aircraft, for reasons of economy and performance, cruise at altitudes higher than 8,000 ft.

Ideally, in terms of purely physiological considerations, the pressure should be maintained at 1 atmosphere throughout the flight. This, however, would mean that a very high pressure-differential would be present when the aircraft is at altitude and therefore would require greater strength of the structure, thus weighing more and reducing performance. The greater force on the structure would also reduce durability. The compression systems required to maintain such pressurization would be power-hungry and thus reduce economy. Therefore, as is often the case, a compromise between engineering, economic, and medical factors has to be reached. As a result, cabin altitude in passenger aircraft is maintained between 6,000 and 8,000 ft when the aircraft is at its cruising altitude, which is normally in the range of 30,000 to 40,000ft.

With the safety brought on by the pressurization of the cabin in relation to hypoxia, there also comes a price: accidental decompression of the cabin. This can occur via a breach in the structural integrity or a failure of the compression system whilst at altitude. It is a small but ever present possibility. A fast decompression, such as would occur under the circumstances, introduces the threat of a fairly rapid exposure to a substantial degree of hypoxia.

This study is concerned with the decompression of relatively large passenger aircraft and in this scenario the time of decompression is likely to be no less than 20 to 30 seconds (Ernsting - Patterson lecture 1995). Shorter time scales for decompression may occur in incidents of a catastrophic nature in which the survival of the aircraft itself is unlikely. As such, this scenario is not one where survival is likely for the crew and passengers aboard

even if adequate oxygenation is achieved.

Behaviour of Alveolar Gases on Rapid Decompression

Upon a rapid decompression, alveolar gases will almost mirror the rapid fall in environmental gas tensions. There is thus an acute fall. This fall is dependent on the inspired gas being breathed before the decompression (Marotte et al – 1990) and the final altitude in relation to the initial altitude. Marotte et al – 1990 found that a 40% oxygen concentration was required prior to decompression from 8,000 to 40,000 ft to prevent 'significant hypoxia'. However, normal healthy passengers in the current generation of passenger craft will be breathing ambient air and the fall of alveolar PO_2 in this case would be much greater than would occur if the passenger were breathing pure or a high concentration oxygen mixture prior to the incident (Ernsting - Chapter 5 (RM Harding)).

The alveolar partial pressure of carbon dioxide also falls as venting of the expanded gas in the lungs leads to the removal of carbon dioxide at a faster rate. This effect becomes more pronounced in a faster decompression and when the final altitude to initial altitude ratio is larger, due to a greater expansion of the gas. The body, however, recovers from this to an extent through the release of carbon dioxide from stores. In very rapid decompressions (1-2 seconds), and from 8,000 to 40,000ft, the P_{ACO_2} value may drop to 10mmHg and then recover back up to 25-30mmHg (Ernsting - Chapter 5 – (RM Harding)).

Note that with a very rapid decompression to high altitudes, oxygen donation from the blood to the environment can occur if the partial pressure of oxygen in the alveolar air is less than that in the blood.

This section will not describe the recovery of alveolar gas partial pressures once oxygen is supplied, as it is outside the scope of this particular study. However in simple outline, the earlier 100% oxygen is administered after a rapid decompression the better the recovery. Another important factor is the gas being breathed before decompression; higher concentrations of oxygen in this gas are protective.

Decompression Sickness

Decompression sickness is a multi-symptom condition. Skin irritation (creeps), respiratory disturbances (chokes), and limb pain (bends) are some of the more common manifestations. The basic causal mechanism is the supersaturation of nitrogen gas, leading to formation of bubbles of gas in the blood.

Nitrogen is dissolved in the body fluids and tissues of humans and this dissolved gas exerts a partial pressure similar to that of the partial pressure of nitrogen in the inspired air. When humans ascend to altitude, barometric pressure and thus inspired nitrogen partial pressure, decreases. Nitrogen is removed from the body via the lungs. However, if the rate of this removal is slower than the rate of fall of barometric pressure, the tissues and body fluids can become supersaturated with nitrogen. The partial pressure of nitrogen

in the body fluids will become greater than that of the hydrostatic pressure and bubbles will form.

The risk of decompression sickness in the event of a rapid decompression rises to a level of significance only when there is prolonged exposure (longer than 5-10 minutes) to altitudes above 22,000 – 25,000 ft (Ernsting - Chapter 8 – (A Macmillan)). It is noted that decompression sickness has occurred on exposure to altitudes of 18,000 ft, however, this is very rare (Ernsting - Chapter 8 – (A Macmillan)). This was a potential hazard in our study but the risk of it occurring was not significant as appropriate measures were taken to reduce this possibility. In an actual decompression it would most likely not occur unless the exposures were of the characteristics described above and these are unlikely if an immediate descent is made, which would normally be the case.

Oxygen Carriage in Blood

Oxygen is carried in the blood in two forms, dissolved and combined with haemoglobin. The amount that is found dissolved is very small compared to that which is chemically combined to haemoglobin: approximately 1.5% of the concentration of oxygen combined with haemoglobin (Ernsting - Chapter 4 – (J Ernsting)) in a person breathing air with an alveolar oxygen partial pressure of 13.3kPa (approximately that at sea level).

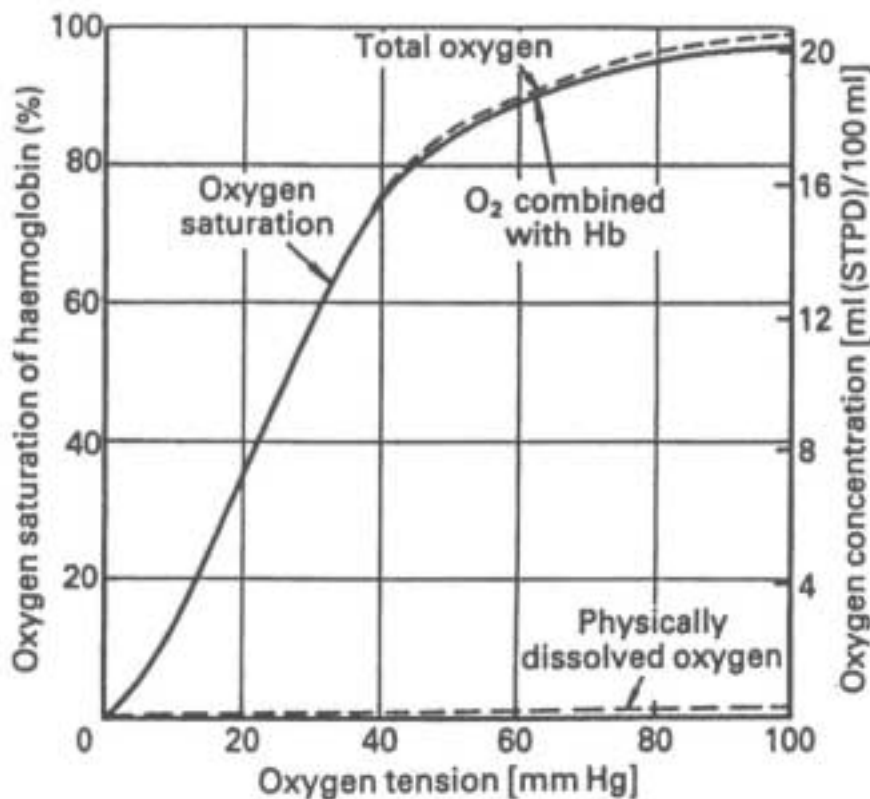
Oxy-haemoglobin is the reversible combination of oxygen and haemoglobin. Haemoglobin is found normally at about a concentration of 15g/ 100ml of blood. Each

gramme can combine with 1.39ml STPD (Standard temperature and pressure, dry) of oxygen (Ernsting - Chapter 4 – (J Ernsting)). The total amount of oxygen that can be carried in combination with haemoglobin can thus be calculated: $1.39 \times 15 = 20.8$ ml/100ml of blood. This is known as the oxygen capacity of blood.

The amount of oxygen in the blood is commonly expressed as oxygen saturation of haemoglobin –

$$\frac{\text{Concentration of oxygen combined with haemoglobin}}{\text{Oxygen capacity of blood}} \times 100\%$$

Oxygen Haemoglobin Saturation Curve



(Figure taken from : Figure 4.4 P31 Ernsting – Aviation Medicine)

The above figure illustrates how the oxygen saturation of haemoglobin varies with oxygen partial pressure. As can be seen, a drop in alveolar oxygen tension to 30mmHg would result in an oxygen saturation of approximately 50-60 %. The shape of this curve means that such a large drop in oxygen tension from 100mmHg (Sea level) to 30mmHg (approximately 25,000 ft) only results to about a 40 % reduction in saturation. However, this decrease is still significant and causes the large degree of impairment discussed earlier.

Previous Studies

The basic physiology of fast decompressions has been outlined above. Here follows a description of two studies that were conducted in the past which relate directly to this topic of research. There is very little data on this particular area and the two studies that are discussed below are two of the most important and valid.

Nicholson & Ernsting in 1967, using baboons, simulated hypothetical decompression profiles for varying defect sizes in a model with an aircraft altitude of 60,000ft. With a 4-inch diameter defect, the hypothesized maximum cabin altitude reached was 30,000 ft from an initial cabin altitude of 6,000 ft. In this run, unconsciousness occurred at 30,000 ft and the respiratory rate was increased and gradually went back to normal upon recompression. No brain damage was seen in this profile. With the hypothesized profile

of a defect size of 6 inches however (with a maximum cabin altitude of 42,500 ft and time above 40,000 ft of 1.5 minutes), permanent brain damage and death occurred in 2 out of a total of 3 baboons. The researchers in this study (Nicholson & Ernsting) concluded that a prolonged period of hypoxia caused permanent brain damage but could not determine what extent of oxygen deprivation was necessary for this to occur.

In a subsequent study by Brierley & Nicholson – 1969 using monkeys, the hypothesized decompression profiles were revised with newly available data that showed that maximum cabin altitudes would be lower and exposure times shorter. Electrocortical and neuropathological studies were carried out to determine whether brain damage occurred after exposure to the different decompressions. The study found no evidence of brain damage with a 6, 7, and 8-inch diameter defect. They concluded that in the case of a decompression in an aircraft at such high altitudes (60,000 ft) with a decompression profile not exceeding 8 minutes above an altitude of 10,000 ft and a maximum cabin altitude of 42,700 ft, no permanent brain damage would occur. However, they also noted that progressively increasing peak altitude had an increasingly deleterious effect on cerebral function.

Decompression Profile of a Passenger Aircraft

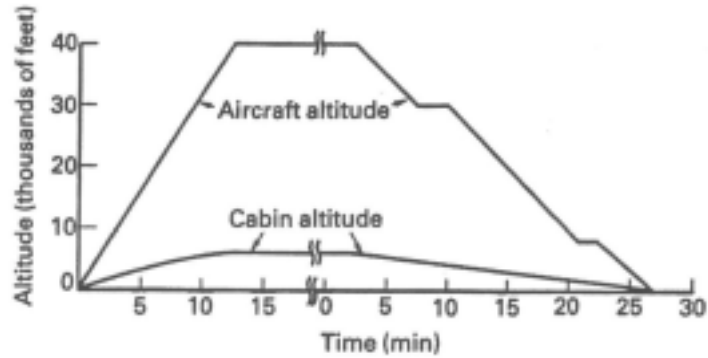


Figure 8.2 The time course of the cabin altitude (lower curve) of a high-differential passenger aircraft during a flight (aircraft altitude, upper curve) up to 40 000 feet and back to ground level.

(Figure taken from : Figure 8.2 P116 Ernsting – Aviation Medicine)

These studies were all carried out on the assumption that a rapid descent is initiated after the decompression occurs, and thus reducing the maximum cabin altitude to which passengers are exposed.

Due to the renewed interest in supersonic airliners and the lack of data on high altitude flying as shown, our study was proposed to be a preliminary study into this area that would lead to further studies into the physiological events of a rapid decompression at the estimated altitudes of these ‘high flyers’.

Current Study

In our study we will use two decompression profiles: 20 seconds which, as explained above, is the sort of time of decompression one would expect in a large passenger aircraft

at such altitudes, and 90 seconds, which is also within such a model but represents a slower decompression caused by a smaller defect or failure. As it is a preliminary study, the maximum cabin altitude exposure is limited to 25,000 ft. In a future study this may possibly be elevated to between 30,000 and 40,000 ft.

Hypothesis & Objectives

This study was performed to investigate the effects of decompression from 8,000 to 25,000 ft upon end-tidal partial pressures of oxygen and carbon dioxide, pulmonary ventilation, arterial oxygen saturation, heart rate, and arterial blood pressure in seated subjects at rest breathing air.

One of the main objectives of this study was to determine whether the time of decompression, i.e. the two different rates used (20 seconds and 90 seconds), gave rise to a difference in the effects observed in terms of end tidal oxygen and other variables. Therefore the null hypothesis is: in the population of healthy adults there will be no difference in the physiological changes observed between the two different rates of decompression.

This study was designed to be a preliminary investigation for a study incorporating a higher final altitude. It was carried out to determine the changes that occur in physiological parameters and to aid in the design of a more extensive and complete study in the future.

Methods

Research Ethics Committee Approval for Study

The study was approved by the King's College London Research Ethics Committee and the Royal Air Force Experimental Medicine Ethics Committee. A copy of the application to the King's College Research ethics committee can be found in Appendix 1.

Subjects

Subjects were initially recruited via an email invitation sent out to King's College Students. They were informed as to what exactly was going to take place and the possible risks. They were also informed that they could withdraw at any time and for whatever reason without such a decision having any effect on the standard of care they would receive. This information was provided on a subject information sheet, which is attached in Appendix 1 and an investigator was available to answer any questions that the potential subjects had. If the a potential subject wished to go through with the study, full consent was obtained. These records are held by Professor John Ernsting at 4.1 Shepherd's House, Guy's Campus, King's College London.

A medical check-up of the subjects was carried out once they had agreed to take part in the study. This included a medical history and an examination of the ears. Medically unsuitable subjects were screened out at this point.

Initially, a total of 10 subjects were recruited in this manner (6 male and 4 female). However, due to various problems, which are detailed in the section titled 'Problems/Errors Encountered', recruitment of 3 further subjects (all male) was carried out at the RAF Centre of Aviation Medicine (CAM). The subjects were all healthy fit individuals and were free of upper respiratory infections and/or ear infections at the time of recruitment, as required by the safety protocol. A total of 22 decompressions (runs) were performed with the use of a total of 13 different subjects.

Subjects had to undergo another examination of their ears on the day of their experimental decompression to determine that they would be able to 'clear them' (by introducing air into the middle ear cavity via the eustachian tube) during the descent. The ear drums were checked for any signs of inflammation and the subject was also asked to perform a valsalva maneuver to determine whether the eustachian tubes were patent and allowed air to enter the middle ear cavity. This was performed on all the subjects and observers that would be inside the hypobaric chamber on that day. An available medical officer carried out the examination. Those that failed the examination would not be allowed to be either an observer or subject on that particular day.

Measured Variables & Equipment

The study was designed to measure certain physiological indicators that were relevant to the exposures to hypobaric conditions. These factors were measured to allow the

comparison of the two rates of decompression and also to firmly establish what occurred during the decompressions.

The decompressions were carried out in a hypobaric chamber manufactured by Aeroform Limited (1955) - SIEBEGIORAN & CO LTD. A continuous measure of the barometric pressure inside the chamber was achieved using a pressure differential transducer (CELESCO DP315-0020-111). Due to various instrumentation limitations, a steady rate of decompression could not be achieved in our experiments. However, the decompression profiles were reproducible throughout the experiment (i.e. they were very similar). A RAF type P/Q mask was worn by subjects. This was connected via a fleisch capillary flowmeter and a Douglas two-way tap to a demand oxygen regulatory (Mk-517). Inspiratory flow rate was calculated continuously using the fleisch capillary flowmeter. The flow rate was determined by a transducer, which measured the pressure drop across the resistance element of the fleisch. Flow rate was integrated by a digital integrator to provide a recording of inspired volume. The partial pressures of oxygen and carbon dioxide in the mask orifice were also measured continuously using a mass spectrometer (Airspec Qp9000). This allowed us to establish the end tidal partial pressures of each gas. The lag time of the mass spectrometer was calculated for analysis. These values were used to correlate the end-tidal gas tensions to real time barometric pressure change.

Electrodes were attached to each subject's chest and allowed those conducting the experiment to obtain a single lead electrocardiogram (ECG) trace using a Charter

Kontron Micromom N-X1. The device had a display on which the trace was displayed. It also produced a measure of heart rate, which was recorded on the chart recorder trace. The subject's digital artery blood pressure was yet another physiological characteristic that was measured. This was done non-invasively via a finger cuff sensor connected to a device called a Finometer, made by Finapres Medical Systems (FMS). Using the Finometer, systolic and diastolic blood pressure were measured. Pulse oximetry was carried out to measure arterial oxygen saturation of haemoglobin at both the ear and the ring finger using two pulse oximeters and their respective probes. The pulse oximeter connected to the ear was an Ohmeda Biox 3740 Pulse Ox and the pulse oximeter connected to the finger was a Kontron vitalstat.

All the data mentioned above was simultaneously sent to two chart recorders (Graph Linearcorder WR3320) and to a personal computer where the data was stored digitally in a program called 'Chart'. The variables mentioned above were recorded on the chart recorders before, during and after the decompression. There were two chart recorders: a four channel recorder and an eight channel recorder. Thus a 'hard' paper copy as well as a disk copy was produced. The respective channels and what exactly they recorded follows:

Chart Recorder Traces:

4-Channel Recorder : Barometric Pressure – 7,000 to 26,000 feet

Fractional concentration of Oxygen – 0 to 22 % (FSD)

Fractional concentration of Carbon dioxide – 0 to 20% (FSD) *

SpO₂ – ear pulse oximetry – 50 to 100 %

8 Channel Recorder : Barometric Pressure – 8,000 to 26,000 feet

Integrated flow volume – maximum gain

ECG trace to detect any anomalies in heart rhythm (later switched from 4th run on to FCO₂)

Heart Rate - 0 to 100 bpm

Arterial blood pressure – 0 to 200mmHg

Fractional concentration of Oxygen – 0 to 100 % (FSD)

SpO₂ – ear pulse oximetry

SpO₂ – finger pulse oximetry

* The FSD was set to 20%, as at 25,000 ft 40mmHg of CO₂ composes a fraction of 17%. Barometric pressure is approximately 282. Subtract from this the water vapour pressure of 47mmHg and 40mmHg becomes approximately 17% of the fraction. Remember that the mass spectrometer measures dry gas concentrations.

The photograph that follows shows the instrumentation outside the chamber:



- Finometer
- Kontron Vitalstat
- Ohmed Biox 3740
- Kontron ECG
- Chart Recorder

Mass Spectrometer

All leads, wires, and tubes communicating from the inside of the chamber to the equipment outside the chamber, were sent through the chamber wall such that no air would leak. Good seals were maintained. The tap allowing calibration gases into a socket in the hypobaric chamber was closed before decompression. This tap, when open, allowed the sampling line of the mass spectrometer (which was located inside the chamber) to be used to detect the calibration gases.

Calibrations - Pre & Post

Most of the calibrations were performed before and after each experimental run. The

mass spectrometer, however, was calibrated once at the start of the day and again at the end of the day. The recording area on the chart recorder for the Kontron Vitalstat was calibrated using a separate power source to generate the 0.915V that the device used as its 100% saturation output. 0V was used to calibrate the 0% saturation line. This finger pulse oximeter was also only calibrated once a day. The rest, as follows, were calibrated before and after each run.

A volume calibration was performed using a 3 Litre syringe. With the digital volume integrator 'zeroed' and the chart recorder running, 3 litres of air was pumped through the fleisch capillary flowmeter, recording a step of 3 litres on the chart recorder trace.

Following this, gas calibrations were carried out using certified concentration gas containers. The concentrations of the gases used are as follows:

BOC Gases – Setting up - 14.21% Oxygen, 9.22% Carbon dioxide *
 Gas A – 5.19 % Oxygen, 2.48% Carbon dioxide *
 Gas B – 9.92% oxygen, 1.01% Carbon dioxide *
 Gas C – 79.68% Oxygen, 20.32% Carbon dioxide
 Gas D – 100% Nitrogen
 100% Oxygen tank

* The remaining fraction in these containers was Nitrogen.

Since the mass spectrometer sampling line was located inside the chamber, gas sampling had to be carried out by inserting this sampling tube into a socket on the inside. This socket was connected to the sampling gases outside the chamber one at a time. The chart recorders were started and gases A, B, 'Setting up', C, and also 100% oxygen were put through the mass spectrometer sampling line separately. The values of each gas were recorded on the chart recorder trace once the reading was stable. Any adjustments required, i.e. adjusting the zero line and the full-scale deflection to the correct point on the chart, were done during the pre-calibrations and no such adjustments were made during post-calibrations. The post calibrations were done to determine whether any change in the calibrations had occurred during the experiment. If a change had occurred between the post and the pre-calibration deflections, it had been determined previously that half of this change was to be taken as having occurred halfway through the recording.

The Finometer, which estimated digital artery blood pressure (BP), produced a continuous calibration signal while it was not operational. This calibration signal had a FSD that represented 200 mmHg. It also produced a signal for 100mmHg and 0mmHg, in sequence. This signal was used to calibrate the trace channel for BP. The ohmeda pulse oximeter (response time of 3 seconds) was switched to produce a calibration signal for 100% saturation, 50% saturation, and 0% saturation and this signal was used to calibrate the trace for this channel.

At the start of each day, the instrumentation that measured the pressure differential

between the inside of the chamber and the outside was calibrated by an RAF electronics technician. This pressure differential was used to calculate the barometric pressure inside the chamber. Prior to each decompression, the atmospheric pressure and temperature were recorded.

Experimental Preparation

Subject Preparation

Subjects were fitted with RAF type G cloth helmets and an adapted RAF type P/Q oronasal mask. Different sizes were available for appropriate fitting. They were then asked to rub a rubifacient cream for approximately two to three minutes on the lobe of their left ear (where the pulse oximeter would be placed). This cream vasodilates the vessels, ensuring a large blood flow in the area. This increased blood flow aided the pulse oximeter probe to measure saturation levels. After cleaning the area of skin with an alcohol swab, three electrodes were then attached to the subjects body in the appropriate position to enable an electrocardiogram (ECG) trace to be obtained during decompressions. The procedures and a short briefing on what was to happen in the chamber and during the run was given once more to the subject and an opportunity was provided them to ask any questions they may have had. This was done in order to calm any last moment anxiety or apprehension that may have been present and so that the subjects would be clear on the procedures about to take place.

Setting up Subjects Inside Hypobaric Chamber

While the subjects were being prepared as described above, the pre-calibrations for the experimental run were being carried out. After these were completed, the subject was seated inside the hypobaric chamber and the ECG wires were then attached to the adhesive electrodes on the subject's chest to provide a single lead ECG. The ECG allowed the measurement of heart rate and also to detect any abnormalities in the trace in terms of cardiac arrhythmias. Following this the subject was secured in place comfortably on the seat using a four-point seat harness. The ear pulse oximeter sensor was positioned on the left ear lobe and the cloth helmet was pulled over the sensor carefully so as not to dislodge the sensor from its position.

Next, the Finometer sensor was placed on the index or ring finger of the left hand, ensuring appropriate fitting. The sensor was a rubber cuff that wrapped around the finger. By varying the air pressure inside the cuff the instrument was able to determine the blood pressure at that point. Once the cuff was securely in place on the finger, a second sensor of the finometer system that measured height was placed at the same level as the finger cuff while the hand was resting on the subject's leg (as it would be throughout the experimental run). At this point the finometer was reset and the position of the height sensor was marked by the machine. The sensor was then moved and attached to the seat harness at the subject's heart level (where it would remain for the rest of the experimental run). The Finometer was then started. This calibration allowed the Finometer to assess the height difference between where blood pressure was being measured by the finger

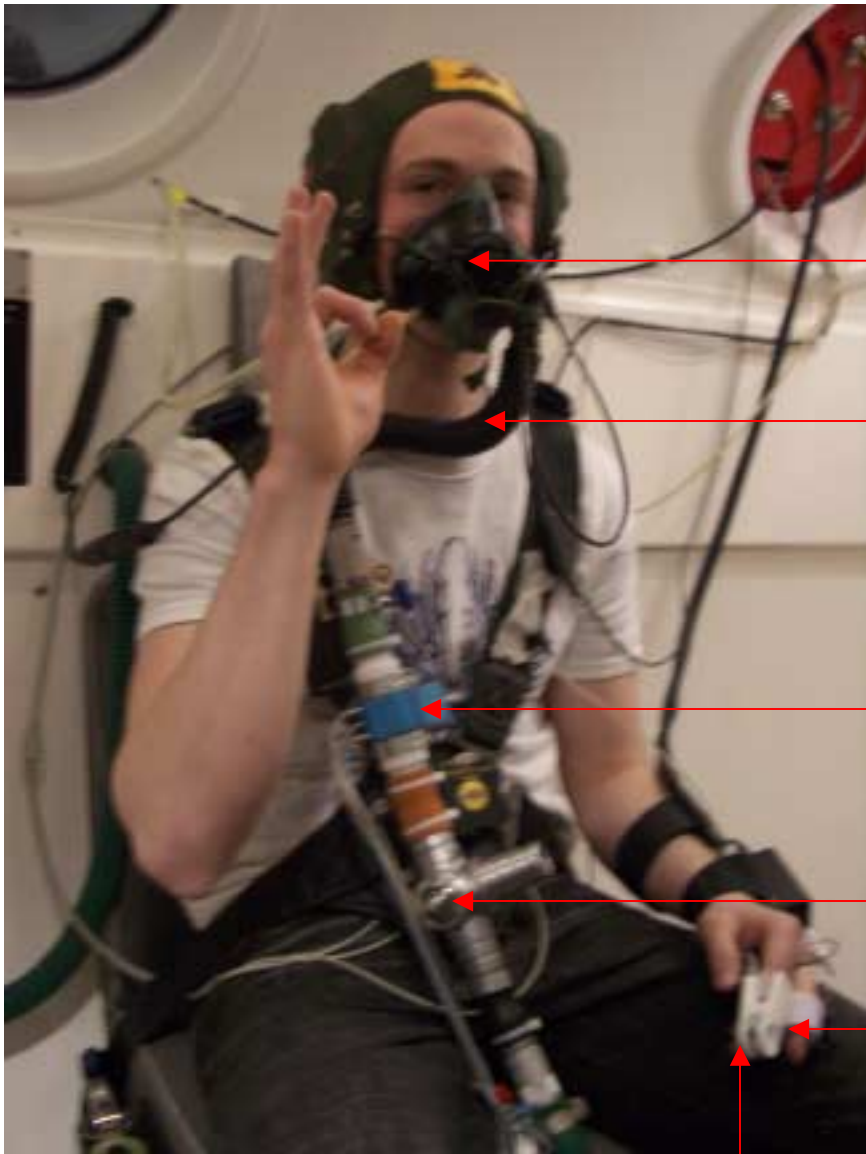
cuff and the level of the heart. This height difference was used in an internal algorithm to estimate the systolic and diastolic blood pressure at heart level.

A finger pulse oximeter was also appropriately placed on the middle or ring finger. This finger pulse oximeter was in place as a backup to the ear oximeter, in case the ear oximeter failed.

The next step was to fit the RAF type P/Q oronasal mask on the subject attached via the cloth helmet. Once this was done the inlet hosing from the mask was connected to the breathing equipment hosing with the tap turned to ambient air. The breathing equipment is described below:

Breathing Equipment

This consisted of a pressure demand oxygen regulator (Mk517) attached to hosing that was connected to a two-way tap, in this case, a 2.0cm bore Douglas tap. This tap allowed the gas available to the subject to be switched, either to allow demand oxygen regulator (100% oxygen) gas or ambient chamber air to be breathed. The inlet hose of the RAF type P/Q oronasal mask worn by the subject was connected to the tap and the rest of the breathing equipment via a fleisch capillary flowmeter. Below is a photograph of the assembly including some other equipment.



RAF type P/Q oronasal mask

Mask Inlet Hose

Fleisch capillary flowmeter

Two-way Douglas Tap

Finometer Finger Cuff

Finger Pulse oximeter probe

Once the breathing equipment had been connected up to the mask, it was secured in place by attaching it to the subject's harness making sure that the tap or inlet for ambient air would not be occluded in any way.

Mask Leak Test

A mask leak test was performed in order to ensure that the subject would receive adequate oxygenation once the tap was switched to 100 % oxygen at 25,000 feet. It was carried out by switching the two-way Douglas tap to allow 100% oxygen to pass through to the RAF oronasal mask and thus to the subject via the fleisch capillary flowmeter. The subject was asked to then take a breath and hold, whilst outside an observer would observe the flow rate trace produced on the chart recorder. It is important to note at this point that as gas flows through the fleisch capillary flowmeter in the 'inspiratory direction' i.e. towards the subject, it produces an electronic signal that then produces a deflection on the chart recorder. If, however, there is no flow, the recorder will not show any deflection. This was the basis of determining whether a leak was present. When the subject held their breath, a leak would be evident if the recorder showed a deflection from the zero line. If no leak were present, then no deflection would be seen on the chart recorder trace. In the event of a mask leak, gas has a route of escape even while the subject is holding breath, and therefore flow is maintained and a deflection is seen.

If a leak was found to be present, adjustments were made to the fitting of the mask so as to reduce this leak as much as possible, until it was at least at a satisfactory level if not abolished all together.

Oxygen Purge

A purge of the breathing apparatus with oxygen was performed finally after all other adjustments. For this, the inlet hose of the RAF oronasal mask was disconnected from the rest of the breathing apparatus. The Mk517 demand oxygen regulator was switched to the 100% oxygen position and the two way tap determining whether ambient or regulator air passed through the fleisch was switched to regulator gas. This allowed oxygen to pass through the hosing and out at the end of fleisch capillary flowmeter. After 1 or 2 seconds the tap was then switched to ambient air. This effectively purged the system of ambient air up to the point of the tap and filled the section behind the tap to the regulator with 100% oxygen. This was done to reduce the functional dead space in the breathing apparatus to enable quick delivery of oxygen to the subject once the tap was turned to regulator gas (100% oxygen) during the run.

After this was done the inlet hose of the RAF mask was reconnected to the fleisch capillary flow meter and the rest of the breathing apparatus.

Other Connections to Mask

The external safety 100% oxygen supply was connected to the subject's oronasal mask by inserting its supply tube onto the mask in such fashion as to be 'air-tight' and so that if switched on would introduce 100% oxygen into the mask orifice for the subject to breathe. This safety oxygen was from a 100% oxygen gas canister outside the chamber and was a safety precaution to be used in the event that 100% oxygen was not delivered to the subject upon reaching 25,000 ft, whether this be because the observer fails to do so,

or because of a failure of the oxygen demand regulator or any other cause.

The mass spectrometer sampling line was sealed onto a small tube that was continuous between the inside of the mask orifice and the sampling line. This would allow sampling of the gases being breathed by the subject at all times. All these tubes going into and out of the mask were sealed such that no air could escape.

A two-way communications channel was connected to the communications system to allow the subject to talk to and hear the researchers outside as well as the medical officer and the observer.

Observer

Observers were prepared simultaneously with subjects. The observer was either the medical officer for that run or a colleague briefed by the medical officer. The observer was secured comfortably in their seat by a four-point seat harness and was fitted with a standard RAF type P/Q oronasal mask. The inlet hose to the observer's mask was connected via hosing to a separate oxygen demand regulator of the same type as the subject (Mk517), with it switched to provide 100% oxygen. In the observer's case, he/she would be breathing 100% oxygen throughout the run. A mask leak test and appropriate fitting was carried out for the observer by RAF staff in their mask/helmet fitting room. The observer was also connected into the communications loop so that they could communicate freely with those involved in the run, including the subject.

A final check of all the 'in-chamber' connections was carried out by a medical officer before proceeding.

Experimental Protocol

Twenty second and ninety second profiles were chosen because they represent the normal expected decompression profile of future generation aircraft.

Once all the instrumentation was ready and the subject was in place, with the observer ready inside the chamber, a medical officer performed a quick check of all the vital in-chamber connections. Following this, the chamber was sealed and depressurized at a rate of approximately 4000 ft/min to 8,000ft, with the subject breathing chamber air. The chamber was held at 8,000 ft for approximately 6 to 8 minutes to allow the subject's heart rate, blood pressure, and ventilation to become steady. At the end of this period on the instructions of the medical officer the rapid decompression was initiated from 8,000 ft to 25,000 ft in either 20 seconds or 90 seconds. The subject breathed normally throughout the decompression as these rates of decompression wouldn't pose a risk in terms of pulmonary barotrauma. Immediately upon reaching 25,000 ft, the observer switched the Douglas two-way tap on the subject's breathing apparatus to allow breathing of the 100% oxygen from the oxygen regulator. Thirty to sixty seconds (approximate time for recovery of pulse oximetry saturation) after reaching 25,000 ft, descent is initiated back down to ground level pressure at 4,000 ft per minute. Once back at ground level any

instrumentation was removed from the subject and he/she was helped out, given a symptom sheet, and asked to fill it in.

Data analysis/Statistics

All statistics including t-tests were performed using Microsoft Excel Analysis Toolpak. Statistical significance was determined by a P value of less than 0.05. The error bars in the graphs produced are of standard error.

Results

The results obtained in the study are outlined below, comparisons were also made between the two rates of decompression. In brief pulmonary ventilation was not found to increase, systolic blood pressure increased in the slow decompression, heart rate increased in both decompressions, end-tidal oxygen partial pressures fell linearly with decreasing barometric pressure and end-tidal carbon dioxide partial pressures decreased but not by a great deal.

A sample trace with annotations can be found in the appendix.

Problems / Errors Encountered

During the study one subject had to withdraw from participating in the hypobaric chamber exposures as the subject had inflamed ear drums at that time and it was thought the subject would not be able to 'clear their ears' effectively during descent and thus ran the risk of otitic barotrauma. Another subject developed the same problem before the subject's second decompression and therefore did not proceed with it.

Some of the decompressions (runs) were aborted and in some runs the data was unusable for various reasons. Due to a failure of the mass spectrometer, three runs were aborted after they had been started. The sampling line of the mass spectrometer had become clogged with various substances from the expirations of the subjects. This sampling

cannula had to be replaced. The replacement sampling line, however, was not of the ideal type for the situation and allowed a leak of air from outside the chamber to the inside where pressure is below atmospheric. This leak was observed as a mask leak (i.e. dilution with ambient air was taking place), whereby oxygen concentrations did not reach 100% when the tap was turned to allow 100% regulator air into the mask. The sampling line was surrounded on the outside by a second tube and this tube was not completely sealed onto the inner sampling line, thus allowing air to enter via this area. This problem was resolved by sealing the leak. The pulse oximeter probe on the ear failed several times, once decompression from 8,000 to 25,000 feet had been started. The delay time on the finger pulse oximeter was larger – 34.3 ± 2.89 seconds – as compared to the ear pulse oximeter readings which had a delay time of approximately 10.8 ± 0.92 seconds. As a quicker response was seen at the ear, this was considered our primary source of data for oxygen saturation of haemoglobin. The finger probe, however, proved to be more reliable and failed only twice to provide us with data whereas the ear probe failed seven times. Minor problems were also encountered when fitting the finometer finger cuff on small fingers. Additionally, a mask leak occurred for one individual as we did not have an appropriate size.

The ventilation values calculated were not converted to body temperature pressure and saturation (BTPS), as we had no recordings of the conditions inside the cabin. In hindsight, this is data that should have been collected to allow the conversion. Pressure was also obviously changing constantly during the decompression and therefore would have made the conversion even more complicated. In order to standardize the results, this

conversion would have been useful. However, the trends seen will not be corrupted in any way and thus the conversion in this context is not as important as it first appears.

Pulmonary Ventilation

Possible anxiety in some subjects had caused a varying degree of hyperventilation to be present even at 8,000 ft. Because ventilation rates were high initially, any change that occurred or was to occur during decompression may have been masked. There was no statistically significant difference found between ventilation at 8,000 feet and during the last time period of decompression, in either the slow or the fast decompressions.

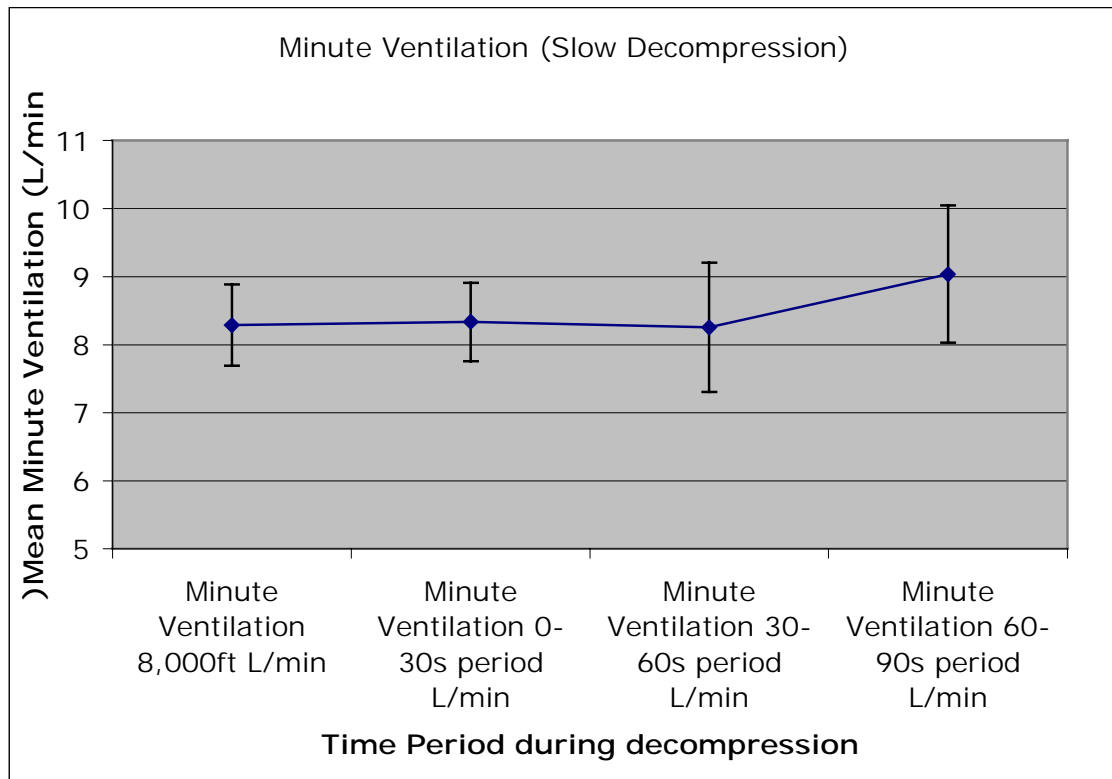
Slow Runs (90 Second decompression)

Ventilation during three 30-second periods were calculated during the time the subject was at 8,000 feet. Minute ventilation was calculated from this and a mean across all the slow runs for this was 8.29 ± 0.6 L/min.

The same was calculated for three 30-second periods from the point of the start of decompression until 25,000 feet was reached (90 seconds). A mean minute ventilation for the last time period (60s to 90s after the start of decompression) was calculated from all the runs and this was 9.04 ± 1.01 L/min.

A paired student's t-test for two sample means was then carried out to determine whether

there was a statistically significant difference between the minute ventilation at 8,000 ft and that in the time period approaching 25,000 ft. The two-tail P-value was calculated to be 0.326, indicating that there was no statistically significant difference.



The Figure above shows the minute ventilation during the various time periods during the slow decompression.

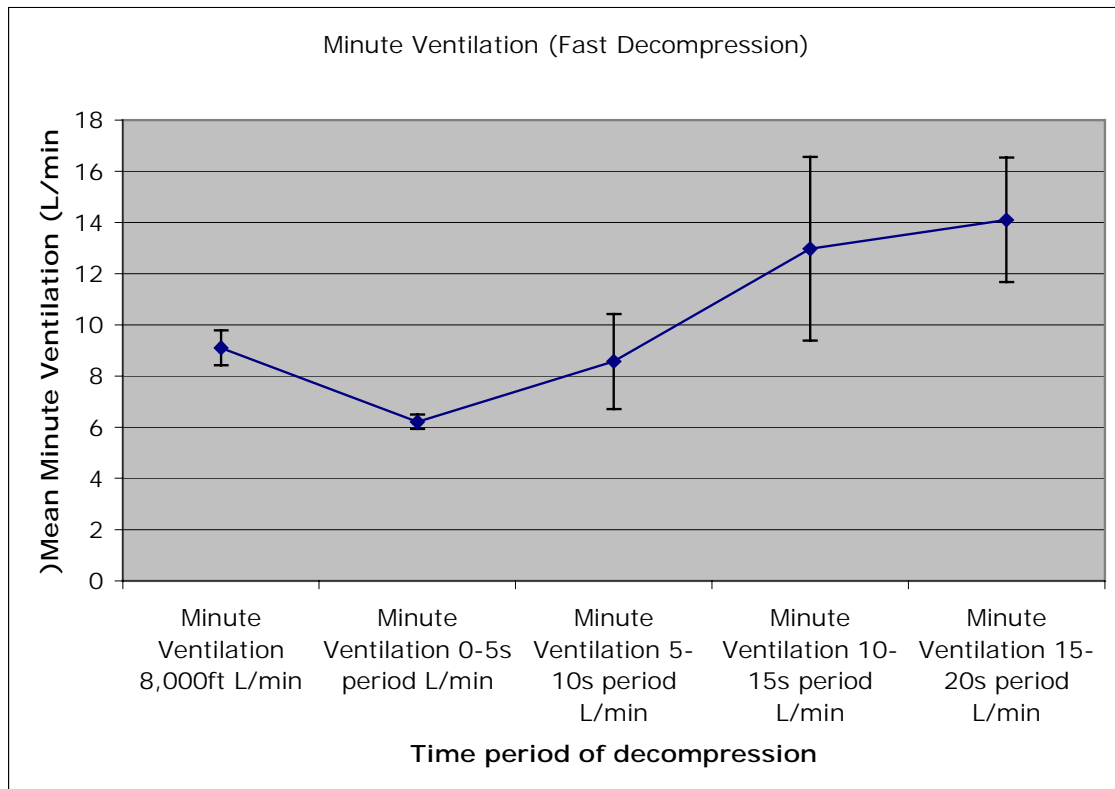
Fast Runs (20 Second decompression)

In the fast runs ventilation during three 30-second periods whilst at 8,000 ft were calculated and minute ventilation was calculated from this for each period. The mean

minute ventilation across all the runs at 8,000 ft was then calculated to be 9.10 ± 0.60 L/min.

Ventilation was then calculated during four 5-second periods, from the start of decompression to the point when 25,000 ft was reached. Minute ventilation for each period was then calculated. The minute ventilation during the last 5-second period was used and a mean for this value across all the runs was found to be 14.10 ± 2.43 L/min.

A t-test of the same type as before was carried out between the minute ventilation at 8,000ft and in the 15-20s period. The calculated two-tail P-value was 0.095. Thus, there was no statistically significant difference.

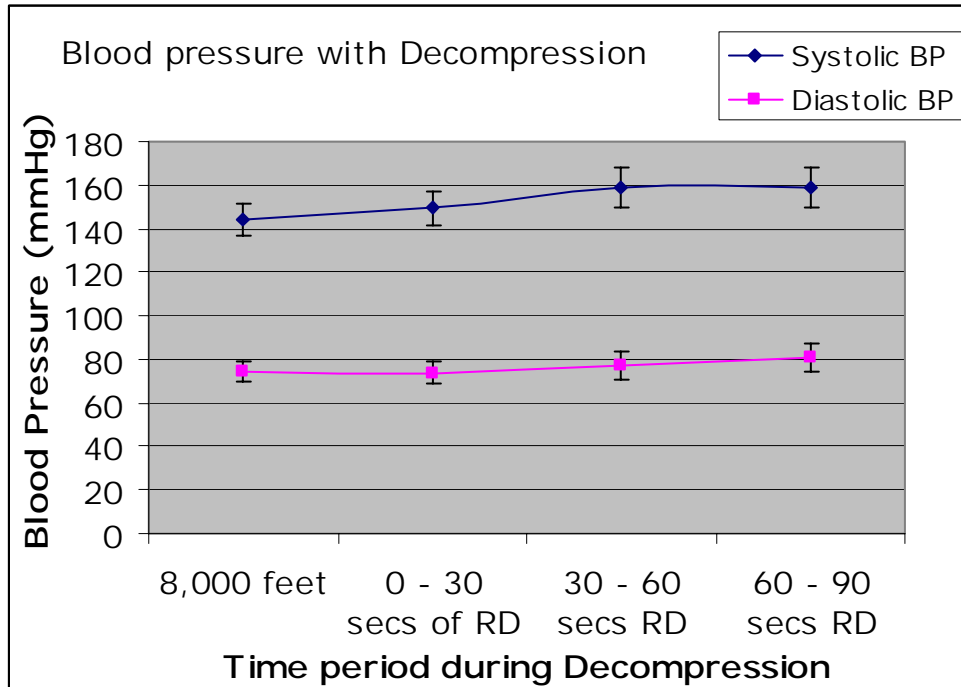


Blood Pressure

Systolic Blood pressure was found to have increased during the slow decompression. This increase was statistically significant. No such change was found in the fast decompression.

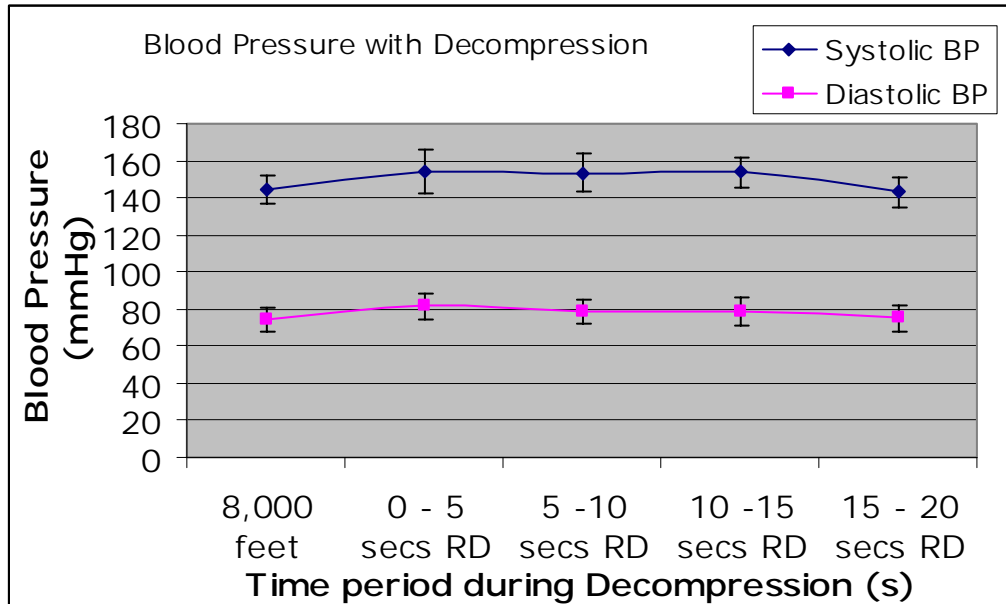
Slow Runs (90 Second decompression)

Average systolic and diastolic blood pressure (BP) was calculated at 8,000 ft and then for every 30-second period after the start of decompression. The average BP at 8,000 ft was found to be 144 ± 7 / 74 ± 4.3 mmHg and this was compared to the average BP in the period 60 to 90 seconds, which was 159 ± 9.1 / 81 ± 6.4 mmHg. A paired t-test was carried out and the two systolic means were found to be statistically significant, $P=0.023$. A t-test performed on the diastolic means at the start and end, found not statistically significant difference, $P = 0.053$. The trend can be seen in the graph below.



Fast Decompression (20 seconds)

The same was performed for the fast decompression, with a resulting average BP at 8,000 ft of 145 ± 7.3 / 74 ± 6.4 mmHg and in the last time period (15-20s in this case) of 143 ± 8.3 / 75 ± 6.8 mmHg. No statistical significance was found between these two periods in either systolic or diastolic blood pressure.

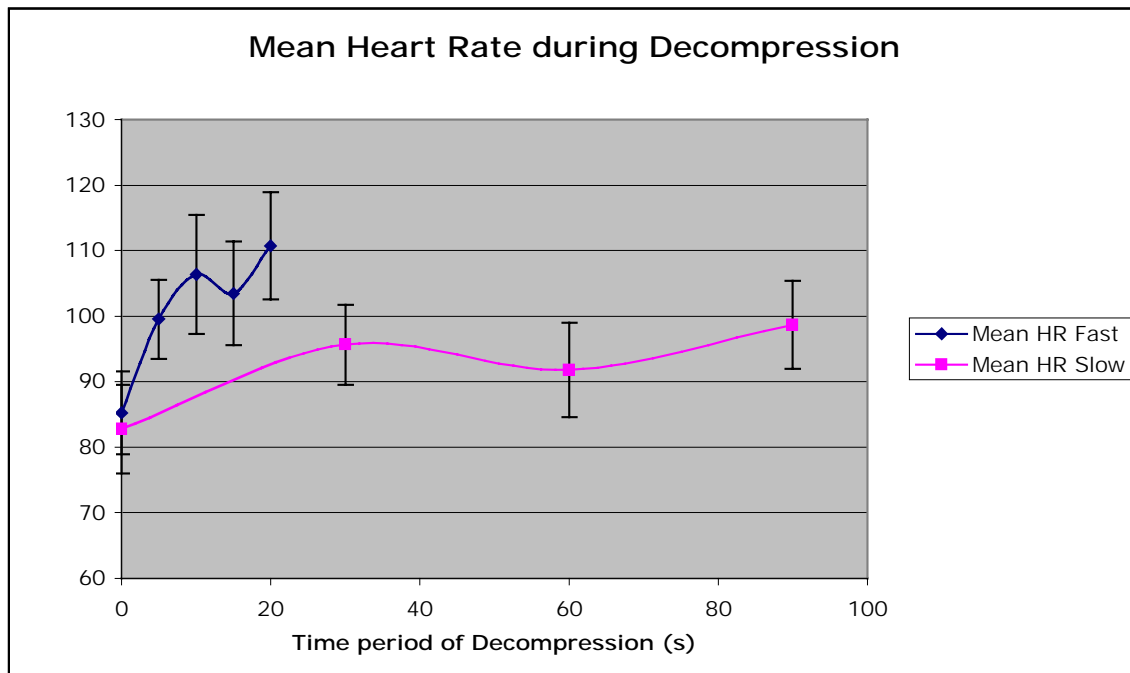


Comparison between the two rates

Further t-tests were carried out to determine whether there was a difference in systolic or diastolic between the two rates of decompression. There was no significant difference found.

Heart Rate

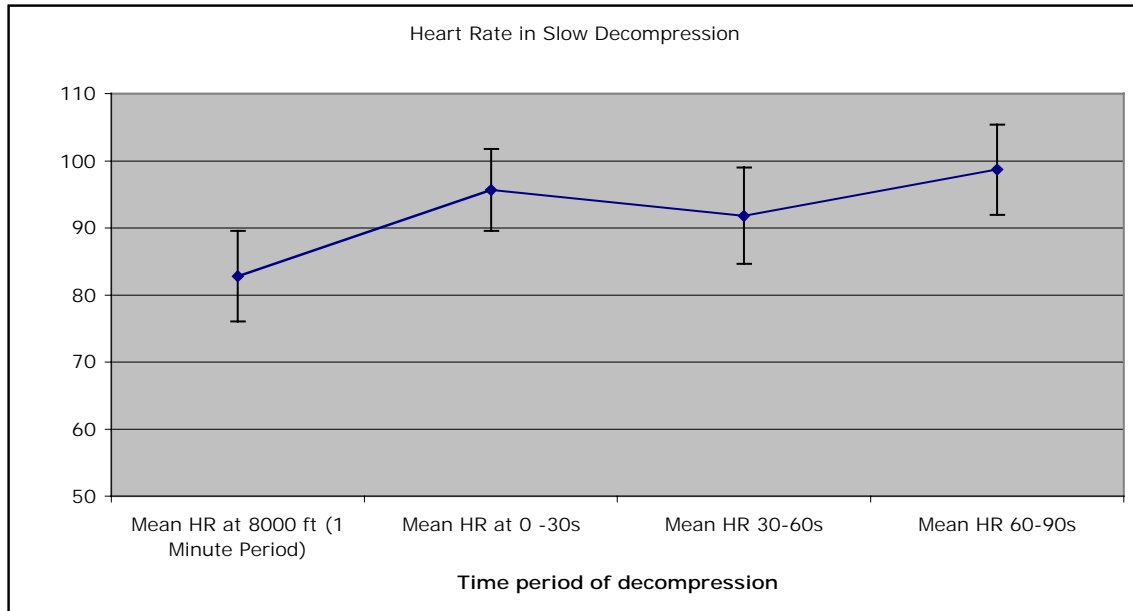
Heart rate (HR) was found to increase in both decompression profiles. It can be seen from the figure below that heart rate increased at a more rapid rate in the faster decompression. While this increase may appear to be greater than that in the slower decompression, the difference between the two ‘last-period’ mean heart rates was found not to be statistically significant.



Slow Decompression (90 second Runs)

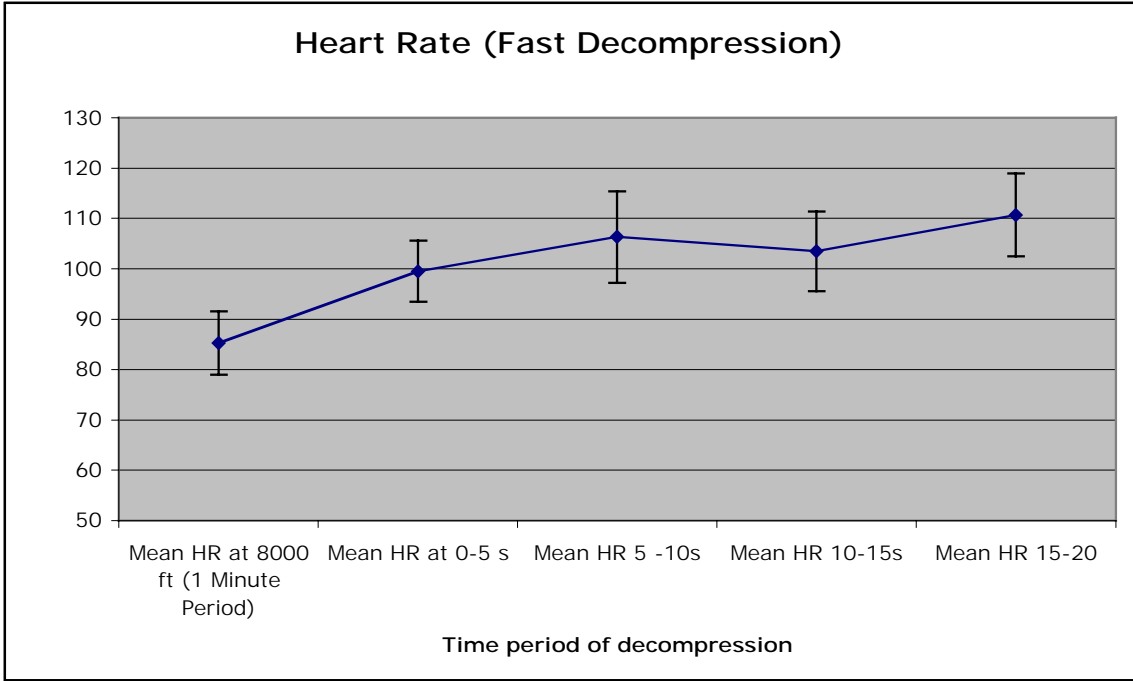
In the slow decompressions, the mean HR at 8,000 ft was 82.8 ± 6.74 bpm and in the last time period (60 to 90s) the HR was 98.7 ± 6.74 bpm. There was a very strong statistically significant difference between these two periods, $P < 0.01$.

The increase in heart rate during the decompression is shown in the figure below.



Fast Decompression (20 Second Runs)

In the fast decompressions, a mean HR of 85.3 ± 6.28 bpm at 8,000 ft and 110.7 ± 8.18 bpm in the last time period (15-20s) was found. There was very strongly statistically significant difference between the two means, $P < 0.01$, confirming an increase in heart rate in the fast decompression.



Oxygen Saturation of Haemoglobin

Our data in the area of oxygen saturation of haemoglobin was incomplete as several times the pulse oximeter probe failed to provide a reading. In certain runs the probe failed to detect the saturation once decompression had been initiated. Therefore, a thorough analysis of these results was not carried out. However, here follows the minimum pulse oximetry saturations that were recorded with each different subject:

Fast Decompression

	Ear SPO2 (%)		Finger SPO2 (%)	
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subject	8,000 ft	minimum at 25,000 ft	8,000 ft	minimum at 25,000 ft
B			98	87
C	94	80	97	77
D	94	85	97	83
E	97	80	97	67
F	91	60	94	87
G	94	74	96	74
H			94	85
I	94	84	95	93
M	94	70	92	51
Mean	94.00	76.14	95.56	78.22
standard deviation	1.73	8.88	1.94	12.88
standard error	0.58	2.96	0.65	4.29

Slow Decompression

	Ear SPO2 (%)		Finger SPO2 (%)	
Subject	8,000 ft	minimum at 25,000 ft	8,000 ft	minimum at 25,000 ft
B			97	73
C			96	64
D	91	83	92	67
E	93	69	96	72

F	93	72	95	65
G	95	70	94	59
H			97	84
J	89	46	94	61
L	95	77	94	69
Mean	92.67	69.50	95.00	68.22
standard deviation	2.34	12.63	1.66	7.53
standard error	0.78	4.21	0.55	2.51

The mean percentage saturation decreased during decompression as measured by both pulse oximeters. T-tests were performed on the finger pulse oximeter data and the reduction in percentage saturation that occurred on decompression to 25,000ft from 8,000ft was found to be statistically significant. The mean percentage saturation at 25,000ft was compared between the two runs and no statistically significant difference was found.

End-Tidal Oxygen Partial Pressures

The end-tidal oxygen partial pressures ($P_{ET}O_2$) are very close in value to alveolar partial pressures. These were measured in order to determine whether rate of decompression has an influence on the alveolar oxygen partial pressure. It was found that there was no difference in the end-tidals at the end of decompression (25,000 ft) in the two different decompression profiles. The decrease in end-tidal partial pressure of oxygen with

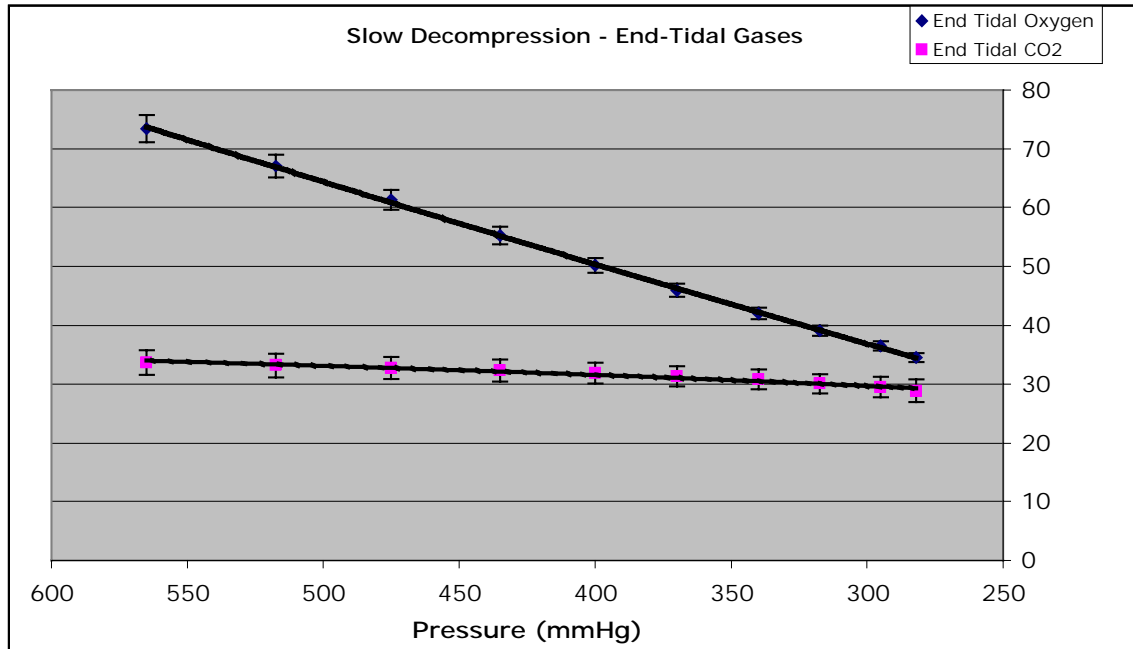
dropping barometric pressure (increasing altitude) occurred as expected.

Slow Decompression (90 second Runs)

End-tidal partial pressure of oxygen dropped from an average of 73.4 ± 2.32 mmHg at 8,000ft (Barometric pressure = 565mmHg) to an average of 34.5 ± 0.78 mmHg at 25,000 ft (Barometric pressure = 282mmHg). This difference was statistically significant, $P < 0.01$.

The end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) also dropped from an initial average at 8,000 ft of 33.7 ± 2.09 mmHg to 28.9 ± 1.90 mmHg. This reduction was strongly significant, $P < 0.01$. The initial average shows that a certain degree of hyperventilation must have been occurring, as normal P_ACO_2 is expected to be approximately 40mmHg, and the value obtained is notably lower than this.

The figure below shows the trends in reduction of end-tidal gases during the slow decompressions

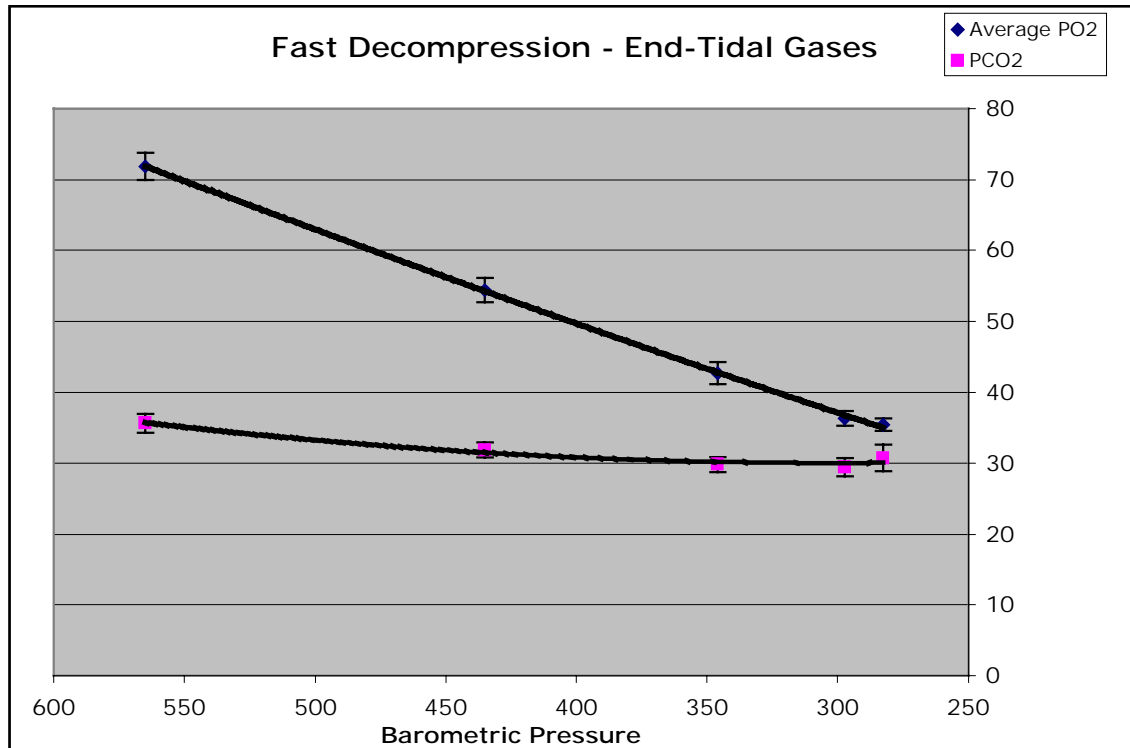


Fast Decompression (20 second Runs)

A fall in the $P_{ET}O_2$ with decreasing barometric pressure was seen as in the slow decompressions. The mean $P_{ET}O_2$ at 8,000 ft was 71.9 ± 1.93 mmHg and this fell to 35.4 ± 0.89 mmHg at 25,000 ft. The difference between the two means was statistically significant, $P < 0.01$.

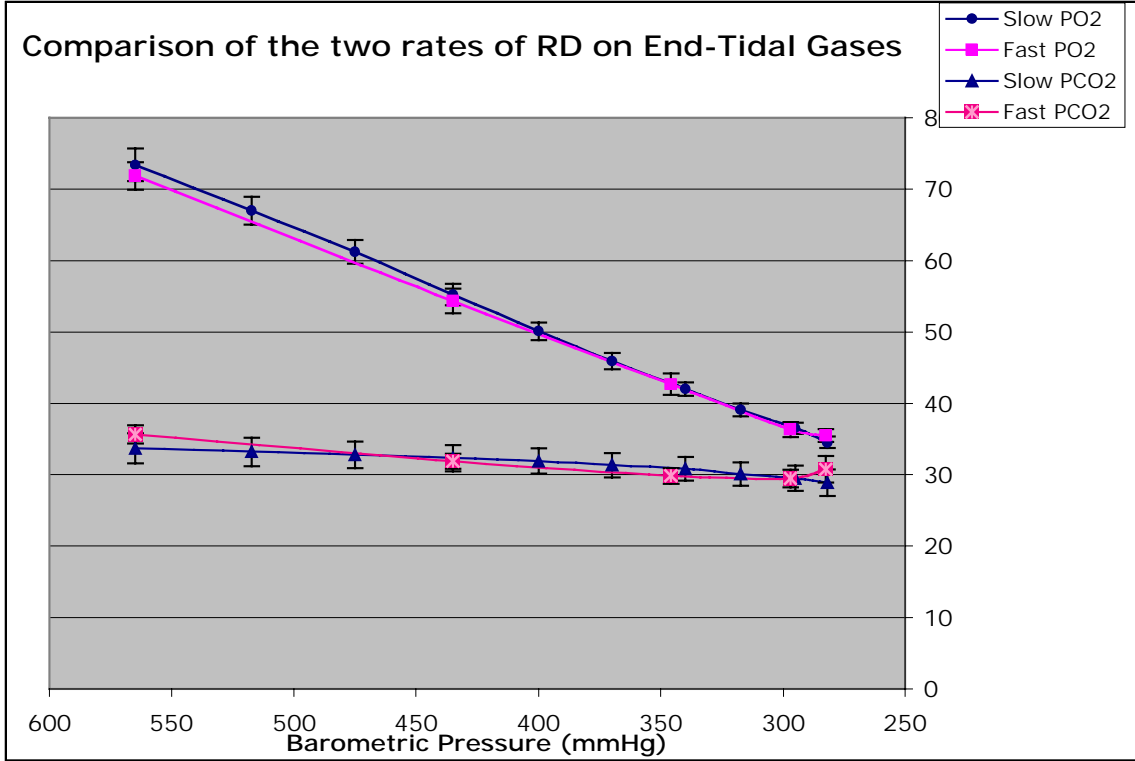
$P_{ET}CO_2$ also decreased from a mean at 8,000ft of 35.6 ± 1.29 mmHg to 30.8 ± 1.87 mmHg at 25,000ft. This difference was also statistically significant, $P < 0.01$.

The trends of the end-tidal gases during the fast decompressions can be seen in the figure below:



There was no significant difference between the end-tidal oxygen and carbon dioxide partial pressures at 8,000 ft and at 25,000 ft. Therefore the rate of decompression seems to have had no influence on the final end-tidals at 25,000ft.

The figure below illustrates how similar the fall in end-tidal gas partial pressures are when plotted against decreasing barometric pressure. A larger graph is available in the Appendix.



Discussion

In summation, the current study found no statistically significant differences between the two rates of decompression. The only difference found was that blood pressure increased during the slow decompression. Yet this trend was not observed during the fast decompression. There were, however, trends to be noted within the decompressions.

Ventilation was found not to have changed significantly during either the fast or slow decompression. As explained in the introduction, a hypoxic drive to ventilation is induced above altitudes of approximately 10,000 feet. Therefore, an increased rate of ventilation was expected. There are a few simple reasons why this increase may not have been observed or did not occur. The hyperventilation already present amongst the subjects at 8,000 feet, as indicated by a low end-tidal partial pressure of carbon dioxide, may have reduced the degree of any change that would have occurred under non-hyperventilating conditions. To the naked eye an increasing trend is visible, however, the change was not statistically significant. The reason a statistically significant difference was not obtained may simply be that there were not enough subjects to cope with the high variability that was obtained. The expected increase in ventilation rate may also take a longer time to develop and there simply may have not been enough time for the full effect of the change to take place. A collusion of all these factors may be the reason that no significant increase was observed during decompression.

The trends in blood pressure (BP) observed were slightly strange in that a statistically significant systolic BP increase seen during the slow decompression was not present in the fast decompression. This increase in blood pressure during the slow decompression may possibly be attributed to the subject's anxiety about the decompression and an increasing sense of this as decompression progresses with the sounds of hissing air, etc. However, this proposition cannot be reconciled with the fact that such a trend was not observed during the rapid decompression, where subjects are likely to be even more anxious and there are louder sounds and greater effects to be sensed during decompression. It has been found that altitude hypoxia induces an increase in cardiac output (Mortazavi et al. – 2003). The hypoxia perhaps stimulates a sympathetic response in order to increase cardiac output and thus provide hypoxic cells with more oxygen. This sympathetic response may only have had time to set in and be observed during the slow decompression. This is the most likely answer. Another possibility, as was previously mentioned, is that the variability was affected by the number of participants. Thus, a trend could have become apparent had more subjects been used in this experiment.

Heart rate was shown to increase in both the decompression profiles. This is probably due to a mixture of anxiety and the adrenergic response discussed above. However, as this trend was seen in both the decompression profiles, it is strange that blood pressure did not also increase during the fast decompression. The increase was more acute in the faster decompression and this could possibly be explained by greater anxiety during the fast decompression as well as the fact that exposure to hypoxia at a more rapid rate may have induced a faster adrenergic response.

The pulse oximetry data was patchy and in some cases inaccurate. Further analysis of this data is not warranted considering the number of recordings obtained and their quality. It can only be noted that, as was obviously expected, percentage saturation fell in both profiles. The reasons for this are explained in the introduction.

The main focus of this study was on the end-tidal oxygen partial pressures, as this is the front line cause of many of the physiological effects observed during a decompression or exposure to altitude. One key and interesting observation to note is that the data obtained did not match the classic Mayo Clinic compiled data of alveolar partial pressures of oxygen and carbon dioxide at various altitudes (Boothby – 1943 Mayo Clinic Aeromedical unit). This Mayo Clinic data shows a departure from the linear trends of both oxygen and carbon dioxide due to the hypoxic induced ventilation that occurs (this effect is described in detail in the introduction). The data found in the current study does not show this departure from the linear trend. The carbon dioxide partial pressure decreased slightly with decreasing barometric pressure, yet not as much as the Mayo Clinic data would suggest. However, the partial pressure of oxygen decreased in almost perfectly linear fashion. A simple and probable explanation for this result is that there was not enough time in the current study for a significant change in ventilation to occur and to cause the effect referred to above. In the Mayo Clinic compiled data, however, alveolar samples were taken 10 minutes after reaching the altitude and thus sufficient time was allowed to acclimatize to the altitude and thus allow the ventilatory response to occur.

The main goal of the current study was to determine whether the rate of decompression altered the end-tidal partial pressures in any way. It was found that there was no difference observed between the two rates of decompression. The values obtained are in fact remarkably similar to each other, as can be seen in the graph showing end-tidal partial pressures against barometric pressure. The similarity between the two rates leaves almost no room for the possibility of suggesting that a difference may be found in the future if several different rates are used. This, however, is still a possibility and therefore a study with several different rates of decompression may be warranted.

Conclusion

The current study failed to find evidence against the null hypothesis. Except for the different results of blood pressure, there were no other differences in the physiological variables measured between the two rates of decompression. Therefore this study can conclude that the rate of decompression has next to no influence upon the physiological results of a decompression. Intuitively one would tend to assume that the faster the decompression the greater the detriment to the normal physiology, but this was not the case.

During the discussion it was suggested that some of the expected changes such as adrenergic response and hypoxic drive to ventilation had not occurred due to inadequate time for these responses to have set in. It would be interesting to find out whether all these changes occur in a longer but practical rate of decompression. If it does so then there is a difference between that rate and the rates at which these effects cannot occur, in that the body in which the adaptations take place will be better able to cope with the hypoxia than the body without these adaptations.

This study was designed to be a preliminary study and a future study at higher altitudes, incorporating different rates of decompression would be the next step forward. These higher altitudes would provide a more practical and realistic decompression profile. Good data was obtained in the study and will no doubt help in the design of a future study.

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