University of St Andrews

The Pathophysiology of Saturation Diving

What are the Effects on the Human Body?

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050007618

Degree Title: BSc Human Biology

1st May 2009

NUMBER OF WORDS: 19,536

INCLUDING TITLE PAGE, CONTENTS, DECLARATION OF AUTHORSHIP, ABSTRACT, FIGURE LEGENDS ACKNOWLEDGEMENTS AND REFERENCES

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ii: Declaration of Authorship

I, the undersigned, hereby declare that the content of this dissertation is all my own work, other than the usual input, discussion, support and advice from my supervisor. I have received no special training or assistance either individually or as part of a group from either other staff or other students. The names of the persons which have assisted me with additional information I was unable to find in the works I researched have been thanked in the Acknowledgements. Specifically, I would like to mention Mr. David Smith from the National Hyperbaric Centre in Aberdeen for his useful information on current and future saturation diving techniques.

Signature:. Hugh William Finlayson Date: 1st May 2009

iii: Abstract

The pathophysiology of diving is an important topic to research because of the wide variety and potentially lethal effects caused by high pressure on the body. In this study the pathophysiology is looked at in both the short and the long term to determine the overall effects of diving and in particular saturation diving on the human body. Better understanding of the physiology of these effects will allow the industry to improve the safety record even more, in addition to providing assistance in developing the next generation of techniques for both diving and the treatment of diving related disorders.

Each important area of the body will be examined in depth, looking at the well known short term effects and the lesser known long term effects associated with saturation diving as well as the relevant treatments. It is known that saturation diving has particular effects on the neurological, cardiopulmonary and musculoskeletal systems and the pathophysiology of these is the main focus of this dissertation. However, there are also effects on other body systems that will also be described.

Finally, the treatment protocols followed for saturation diving disorders have a wide range of applications outside the commercial diving industry. The rapidly growing area of recreational diving, in particular, is more frequently utilising the treatments in recent years. Greater understanding of the pathophysiology of diving will lead to new and more effective treatments, resulting in an improvement in the already high treatment success rate observed.

The conclusion reached is that saturation diving has a wide-ranging and in some instances, unknown physiological effects on the body. Further research is still required to fully complete the physiological profile of the conditions that arise from saturation and other forms of diving. A full understanding would allow for the development of more effective and safer diving techniques and treatments. However there is a lack of funding for this research in the current financial climate due the reduction in financial support from the largely oil related, commercial diving industry.

Chapter 1 – Overall Introduction

Diving under the water has always fascinated mankind and the invention of diving techniques has allowed man to venture into the underwater world. These inventions started the underwater exploration trend that still continues today and the use of this new technology has created a specialised profession in its own right. As the equipment improved so the depths attained increased along with the dangers because of the poor understanding of the effects of pressure on the body and the gases being used (Nitrogen and Oxygen (air)).

After the Second World War there was an expansion in the diving industry with the advent of mixed gas diving which allowed for greater depths than were achievable whilst breathing air. This led to the advent of saturation diving in the late 1940s. The growth of the industry was small at first until the oil industry boom in the 1960s. As a result there was a rapid increase in the research into the effects of saturation diving on the body as well as the development of new techniques. Alongside the increase in the commercial diving industry there was also a rapid growth observed in recreational diving with organisations established to train and qualify new divers such as the Profession Association of Diving Instructors (PADI) and the British Sub-Aqua Club (BSAC). Understandably the conditions observed in saturation divers are also seen, albeit to a lesser extent, in recreational divers.

Despite all the research completed on the effects of diving and pressure on the human body during the oil industry boom of the last three decades, there are still many unknowns in the understanding of the pathophysiology of saturation diving and diving in general.

Saturation diving is a form of diving that requires the divers to be living under pressure for a long period of time so as to avoid the lengthy decompression required in order to return to atmospheric pressure after each dive. The theory is that after 12 hours or more of being under

pressure the body becomes saturated with gas (equilibrium between partial pressure of gas in the environment and in the body tissues) and can no longer absorb any more gas (Vorosmarti, 1997).

Therefore, the time needed to return to atmospheric pressure during decompression does not proportionately increase the longer you dive after saturation of the body's tissues has occurred. Economically this is efficient as it allows the divers to work for long periods at a dive site without having to return to atmospheric pressure in between each excursion to the work site. With the explosion of growth seen in the oil industry in the 1960s the demand for commercial divers increased dramatically, along with the need for saturation divers, as the depths of offshore oil exploration increased. The oil industry funded much of the growth and development of the techniques and equipment observed in modern saturation diving along with some assistance from the military, in particular both the British and American Navies.

These techniques and equipment are still in use today, along with the guidelines for decompressing back to atmospheric pressure developed by the Royal Navy and the United States Navy. These guidelines comprise rules where the utmost priority is given to the safety of the diver(s). As a result the accident rate incidence and the overall risks have reduced dramatically over the last three decades.

Saturation diving has a number of effects on individual organs as well as the body as whole. The main areas affected are those that have direct contact with the main causes of injury in divers, the gases they breathe. For example, the cardiovascular and respiratory systems, which are in contact with the unnatural hyperoxic atmosphere the divers breathe whilst under pressure. Here the pressurised gases have a number of effects which can compromise the efficiency of these systems to function. These are particularly noticeable when a diver decompresses too quickly and develops what is called Decompression Sickness (DCS), more commonly known as 'the bends'. This requires urgent treatment in a recompression chamber (Brubakk *et al*, 2003).

Most of the effects of saturation diving are reversible in the short term. Long term conditions can develop and these can have permanent debilitating effects if prolonged periods of time are spent living under pressure. In-depth research into the effects of saturation diving was mainly done during the 70s, 80s and early 90s funded by the oil industry to improve the safety records of their operations. Little new research has been done recently because of lack of funding and so the underlying physiology of many of the disorders encountered in the profession are still poorly understood. The effects that saturation diving has on the body are well known though and now there are established theories for the mechanisms of action for them (Brubakk *et al*, 2003).

There are a number of risks which saturation divers face whilst they carry out their everyday work, related to the factors that they are routinely surrounded by. Danger from both the environment and the equipment are minimised through strict and standardised safety regulations that are enforced and updated regularly. The current controls have been developed over decades of research carried out by the British, French and American Navies, the commercial dive companies, the oil companies and academic institutions (Vorosmarti, 1997). Since their introduction, the number of safety related incidences has dramatically decreased. The profession is therefore a much safer environment to work in than it was at the start of the operations forty plus years ago. This is because the guidelines are based on what is now known and understood about all the potential conditions that can be encountered, whilst still leaving room for the unexpected to occur and are vehemently enforced. Naturally there are still risks present in the undertaking of a saturation dive but the chances of any one of them occurring is very small in the modern diving environment.

Chapter 2 – Overview of Saturation Diving - The Methods, History and Risks Involved

Saturation diving is used commercially (and to a lesser extent militarily) to perform work underwater at depths of up to 300m (984ft) although deeper depths are possible (Brubakk *et al*, 2003). By using this method of diving, commercial divers can achieve much greater depths, as well as much longer bottom times with dives of six hours or more common. Prolonged diving at such depths is impossible using modern recreational diving equipment. The depths achieved and the duration of time under pressure, (over 12 hours in order for saturation of body tissues to occur (Vorosmarti, 1997)) in saturation diving have necessitated commercial divers breathing a different mix of gases from recreational divers. They rely on an umbilical from the surface to supply their breathing gas. The two commonest mixtures of gas used by saturation divers are Heliox (Helium and Oxygen) and Trimix (Helium, Nitrogen and Oxygen). These mixtures allow divers to reach much greater depths with less risk of nitrogen narcosis (Brubakk *et al*, 2003) which can impair a diver's ability to function. As the depth increases the lower partial pressure of nitrogen as well as of oxygen compared to air in these mixtures ensures the partial pressure of the oxygen stays within acceptable limits (>1.4 bar for short term/>0.5 bar for long term for O_2) and that of nitrogen below the level that severe narcosis occurs (Brubakk *et al*, 2003).

Deeper than 120m (400ft) divers can begin to experience symptoms of High Pressure Nervous Syndrome (HPNS) if diving on Heliox and so Nitrogen is being added back into the mix at these depths. This induces slight nitrogen narcosis which suppresses the tremors associated with HPNS (Brubakk *et al*, 2003). In addition to this slower compression rates that are now used reduce the effects of HPNS on the body (Lounsbury *et al*, 2002).

2.2 - History

The concept of saturation has been talked about for more than a century from when Haldane, Boycott and Damant first wrote about it in their report from 1907. In this they demonstrated no problems with allowing workers to be at pressures of up to 4 bar for over 6 hours (Vorosmarti, 1997). However the practice of saturation diving never appeared to be considered in these early times but then the diving that was being done was shallow diving (˜15m (50ft) and rarely more than ˜30m (100ft) (Vorosmarti, 1997)). The main reason presumably for this was that given the shallow depths of the diving it was unnecessary although it could have been achieved as the engineering capability was available.

The first intentional saturation dive (in a chamber) was carried out in December 1938 by Max Nohl who spent 27 hours at 30m (100ft) on air (Vorosmarti, 1997). However this first saturation dive was not seen as a way to spend prolonged periods of time on the seabed but rather as proof that humans could be decompressed following a long period of time at pressure. The relevance of this was because of the tunnelling operations that were widespread at the time. Animals were used to pull carts whilst under pressure but when they were brought back to the surface they perished having been well whilst living under pressure. After the 27 hours, Nohl was decompressed for 5 hours and returned to the surface where he got decompression sickness but recovered after recompression therapy. These findings were published and it was believed that 27 hours was the maximum time anyone could live under pressure and survive (Vorosmarti, 1997).

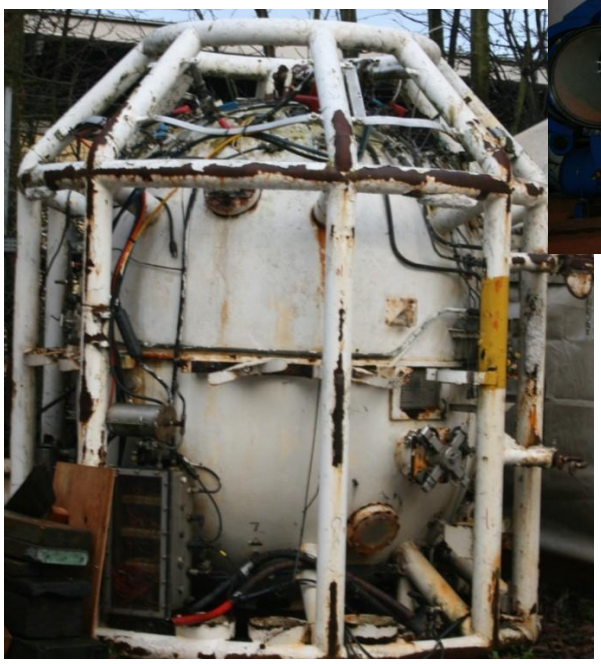
From this inauspicious beginning saturation diving progressed in the 1940s and the 50s with research into new techniques, in particular, the use of different breathing gases such as Heliox. The first operational use was on the recovery of the USS Squalus in 1939 off Portsmouth, New Hampshire at a depth of 73m (240ft) (Web Reference 1). It became more widely used following

the birth of the offshore oil industry, with the advancements in the technology being driven by commercial demand. With this, deeper and deeper dives were achieved and the techniques used perfected to make the diving more economical and safer.

2.3 - Equipment and Methods

In order to achieve the depths at which the work is undertaken the divers live in special chambers called decompression chambers. These allow the divers to stay at a pressure constant with the depth that they are working at whilst breathing the same mixture of gases as they would on a dive. Maintaining the divers at the bottom pressure prevents them having to decompress to the same pressure as the surface each time they finish a dive. The diving would be uneconomical because if done as multiple dives the total decompression time would greatly exceed that of the single decompression time required after a single saturation dive. The divers live and work at this pressure for periods of up to 28 days. To transfer the divers from the decompression chamber to the dive site, which can be at depths greater than 200m (655ft), the divers enter a closed system dive bell (Brubakk *et al*, 2003). This type of bell is similar to a bathysphere and it was first developed in the 1950s by Picard (Bathyscaphe) and Barton and Beebe (Bathysphere). Since then the design has been perfected with changes in hatch size to make access easier (A.J. Bachrach, 1998). The dive bell attaches to the decompression chamber in which the divers live by fixing on to the transfer lock. The bell is pressurised at the same level as the chamber and the dive site at which the diver will be working. Once lowered the divers can exit the bell and access the worksite. Upon completion of the work the divers return to the bell to be raised to the surface and return to the chamber where they can relax and sleep until the next dive or the end of their decompression cycle (Brubakk *et al*, 2003).

Figure 2.1→: The Saturation Diving chamber at the National Hyperbaric Centre in Aberdeen, UK. The chamber on the right is the living quarters (4 people – duplicate chamber out of sight on the left side). The large chamber in the middle is the transfer chamber where the diving bell would be attached if it was located on a Diving Support Vessel. One of the transfer locks can be observed on the front of the transfer chamber. Picture Copyright Hugh Finlayson (2008).



← Figure 2.2: A Closed Diving Bell as used on a Diving Support Vessel to transport divers to and from the Saturation Chamber to the work site. The connection point where the bell connects to the transfer chamber is not shown in this picture (it is on the opposite side). This one is no longer in use and is located at the National Hyperbaric Centre in Aberdeen, UK. If it was in use it would be kept in perfect condition to minimise any risk of malfunctions. Picture Copyright Hugh Finlayson (2008).

Commercial saturation divers normally only undertake one dive a day though these dives are usually around 6 hours in duration (Brubakk *et al*, 2003). After the completion of their series of dives (in general around 2-3 weeks) the divers can spend up to 3 weeks decompressing back to atmospheric pressure so that they can return to normal life (Brubakk *et al*, 2003). All this takes place in the decompression chamber (figure 2.1), which is a cylindrical tube that is pressurised to the depth at which the divers are working and is a crucial part of commercial saturation diving operations.

The chamber varies in size depending on the location and the number of people it is required to house. It is carefully managed to ensure that the breathing mixture and the pressure are the same as the diver would experience whilst on their dive. This requires a complex series of locks and valves to ensure that contact can be made with the divers (pass them food etc) without rapidly

decompressing the chamber which would be fatal for the divers (Brubakk *et al*, 2003).

Decompression chambers are kept on the surface to enable the management of the gas mix by the dive control team (figure 2.3/2.4). The most common location for depressurisation chambers used by saturation divers is either on an oil rig or on a Diving Support Vessel (DSV) (Brubakk *et al*, 2003).

Figure 2.3/2.4: Two pictures of the Saturation Diving Chamber Control Centre at the National Hyperbaric Centre in Aberdeen. The right picture shows the controls relating to the delivery of the breathing gases to the chamber. In the left picture the control for interaction with the divers is shown (the chamber can just be seen through the glass). Both Pictures Copyright Hugh Finlayson (2008).

Figure 2.5: One of Subsea 7's DSVs "Seven Atlantic". This is one of the biggest and most efficient DSVs currently operating (Operational Q1 2009). She has a 24 man Saturation Diving Chamber rated to 350m (1150ft) as well as two hyperbaric lifeboats capable of carrying 18 people each. Dive Bells are located on both sides of the ship (Subsea 7, 2009).

The Diving Support Vessel (figure 2.5) is a ship which is equipped with the latest in global positioning systems (GPS) combined with thrusters on the sides of the ship which can automatically and accurately hold it in position to within around 5m (15ft) (Brubakk *et al*, 2003). To raise and lower the diving bell the ship uses a crane. It may also contain a moon pool to enable the recovery of the bell to be virtually unaffected by sea state. Contained on a DSV used in heliox saturation diving (below 50m/164ft) there are gas recovery systems. These are used because of the relatively high price of helium (~$1/ft^3 - ~$15/ft^3 (~$35/m^3 - ~$525/m^3) Brubakk *et al*, 2003). Taking a consumption rate of 1.5ft^3/min (0.042m^3/min) on the surface (1 bar), a 200m (656ft) saturation dive (20 bar) would cost around $250-1800 an hour (depending on the source of helium) resulting in costs of over $1 million if the gas was not recycled on an operation of a month in duration. The gas is recovered to be used again to prevent the complicated logistics and costs involved delivering the required volume of gas to the divers each month. This involves the diver exhaling the gas (as normal), passing it through a no return valve which takes it back to the DSV (via the dive bell). The gas is then dehumidified, scrubbed of carbon dioxide and filtered before being enriched with oxygen to ensure it is breathable again. This is then compressed for use with careful monitoring of the gas mixture to ensure it is safe for consumption (Brubakk *et al*, 2003).

Figure 2.6/2.7: Pictures of the Helium Gas Recycler Sections at the National Hyperbaric Centre in Aberdeen's Saturation Diving Chamber. The left picture shows the filters that clean the Helium gas of contaminants such as Carbon Dioxide before enriching it with oxygen. On the right it shows the piping that connects the system to the dive chamber which is on the floor above. Both Pictures Copyright Hugh Finlayson (2008).

The equipment the divers wear is the key component that enables saturation divers to work safely and effectively. The helmet (figure 2.8/2.9) contains all the necessary communications equipment, breathing apparatus and any external fittings such as a light. In order to handle the cold, modern saturation divers normally wear either a dry suit or a hot water suit. Hypothermia occurs much quicker in water than in air due to the conductive properties of water compared to air. A hot water suit is similar to a wet suit but instead of the ambient water being warmed by the body, warm water is pumped around pipes in the suit to keep the diver warm. Normally the suit will be sealed to the helmet to prevent the cold water leaking into it which would result in cooling the diver down as well as problems with breathing. Attached to the helmet is the umbilical hose, which contains all the necessary cables to give the diver: breathing gas, electricity, communications and the hot water tube that provides the insulation if in a hot water suit (or another air hose if in a dry suit). In addition whilst on a deep saturation dive the breathing gas is heated so that the diver does not lose as much heat through his respiratory tract as if the gas was at the ambient water temperature. The other form of insulating suit that a diver can wear in cold water is a dry suit. A dry suit prevents any water coming into contact with the body through tight neoprene seals and the diver maintains body heat by wearing insulating clothing. Buoyancy is controlled by regulating the amount of gas inside the dry suit. For this reason another hose comes from the umbilical to provide the dry suit with gas in order to regulate buoyancy (Brubakk *et al*, 2003).

←Figure 2.8 and 2.9: The classic diving helmet: United States Navy Mark V Diving Helmet (left). Used from 1916 until 1984 in the USN and is still in use today by some commercial divers (Diving Heritage, 2005). Divex Ultrajewel 601 Helium Reclaim Helmet (right). This is the modern equivalent of the Mark V and is a light weight helmet. This type of helmet has now almost completely replaced the Mark V in modern saturation diving (Divex, 2004).

2.4 - Risks

Modern saturation diving is a dangerous occupation however it is now safer than it was even 20 years ago. (DMAC, 2006) The reasons for this are due to the increase in understanding of the physiological effects of the diving (covered in the next chapters), in addition to the improvements in the techniques and equipment used in the operations and the improvements in the regulation of the industry from within and from governments.

There is still the ever present risk from the environment and its unpredictability affecting the working conditions of the divers. These include (but are not limited to): currents, poor visibility, temperature and marine life. Divers are also at risk from the machinery they are operating and the structures on which they are working. Falling machinery or structures are an ever present threat which cannot be avoided but can be minimised with effective controls which are now in place.

The other major risk factor for saturation divers is equipment failure. Rapid decompression from depth is fatal for the diver and any malfunctions in the decompression chamber that result in an ascent can have major repercussions for the health of the diver. Whilst working underwater rapid decompression is unlikely to occur and so the malfunction of the umbilical hose which supplies the diver with all their requirements presents the greatest risk. Loss of the breathing supply is the most serious. However, most modern saturation divers carry a "pony tank" which can supply them with up to half an hour of breathing gas in the event of emergency, giving them enough time to return to the dive bell. A less serious risk is the malfunction of the supply of hot water used to keep the diver warm, which can result in the diver getting hypothermia in a short space of time. To combat this divers normally wear a thin under suit to slow the onset of hypothermia if this should occur. The least severe, but ultimately still risky, consequence of losing the umbilical hose is the loss of electricity and communications with the diver. These are

unlikely to be restored without repairing or replacing the umbilical hose (most DSVs now carry a spare umbilical hose, (DMAC, 2006)).

Being a saturation diver still exposes you to a number of potentially life threatening physiological factors that can be debilitating in the long term. The neurological and cardiopulmonary systems are particularly susceptible to damage by the working conditions associated with saturation diving.

Chapter 3 – Overview of the Physiology of Saturation Diving

The study of the physiology of saturation diving is important to understand the nature of the effects caused by the conditions which the divers work. These are related to how the properties of gases change as the pressure increases with depth and the differing interactions with body tissue. As the pressure increases the gases compress in volume but there are still the same numbers of gas molecules present, just in smaller volume. This means that the body is exposed to a much higher partial pressure of gas at depth than is experienced at atmospheric pressure. The effects of this change is minimised by the divers breathing mixtures of gases which contain less oxygen than air (hypoxic). Consequently, the partial pressure of oxygen is at a slightly higher level than is experienced in atmospheric air but is not at levels where oxygen becomes toxic to the body (partial pressure >1.4/0.5 bar (short/long term) (Brubakk *et al*, 2003)).

The physiological effects of the high partial pressure of gas in the environment the divers are working in are both varied and widespread. In addition to the increase in the number of gas molecules in a set volume of gas there is also an increase in the solubility of the gas into tissue (Henry's Law – Appendix 2). This contributes to the saturation of the body tissues with gas and accounts for many of the causes of decompression disorders that have been described. The release from solution and expansion of the dissolved gases present in the tissues (and bloodstream) during ascent (or descent) prevents/inhibits the gases from being expelled from the body through the lungs (only place to expel the gas) (Lounsbury *et al*, 2002). The bubbles of gas can then impair the normal function of the body in the area where they are trapped. Anywhere in the body can be affected and the effects are different depending on location. This is the most severe physiological effect of the gases (nitrogen, oxygen and helium). However they can also have an effect without expanding in less severe ways but which still inhibit the usual function of the body.

There are three main gases used in saturation diving that can have an effect on the body, namely: oxygen, nitrogen and helium. These have a number of differing effects on the body whilst under pressure. These are related to how the properties of the gases change under pressure. All three become narcotic under a high pressure with nitrogen being the most and helium the least narcotic (Brubakk *et al*, 2003/Web Reference 2/Appendix 3). As a result of this, helium affects the body the least whilst nitrogen has the greatest affect.

Unlike the other two gases oxygen affects the body in both high (hyperoxia) and low (hypoxia) concentrations. Hyperoxia occurs in an environment where there is greater than 1.4 bar of oxygen in the short term (>0.5 in the long term). Toxicity can occur at 6m (20ft) if breathing 100% oxygen but does not occur until deeper than 70m (230ft) if breathing air. As oxygen is required for survival the strict regulation of its partial pressure (*P*) in the breathing gas mixtures is critical so as to avoid oxygen toxicity or deficiency (Brubakk *et al*, 2003)

The impact of oxygen on the body is universal and affects its overall function. The severity increases the higher the PO_2 and the longer the exposure. The tissues of the lungs and central nervous system are particularly susceptible to damage, leading to a reduced ability to uptake oxygen and a decrease in neuronal function respectively. Both can lead to death through convulsions and the destruction of neurons (CNS) or oxygen deficiency, anoxemia (contributed to by haemolysis) and acidosis (cardiopulmonary system). Hypoxia has less severe effects than hyperoxia and the length of time between the onset of the symptoms and death is much greater, allowing time to correct the imbalance (Brubakk *et al*, 2003).

Nitrogen is not essential for survival but still affects the body through inert gas narcosis. Under high pressure nitrogen begins to have anaesthetic properties resulting in a reduction in neuronal function because the dissolving of nitrogen into the tissues of the nerves affects the way they generate and propagate action potentials (Lounsburg *et al*, 2002). Nitrogen is also the gas

bubbles most commonly implicated in Decompression Illness. This is through the gas leaving solution as the pressure is released and becoming gaseous again. It then expands in the tissue or blood vessels and causes the effects observed in Decompression Illness (Brubakk *et al*, 2003).

Helium has the least effect on the body because it is less narcotic than the other two and is also less soluble in the tissues. There is some interaction between the gas and the body tissues but many of the effects once attributed to helium are now attributed to pressure. Helium is also saturated in tissues and can leave solution and expand on ascent causing the effects of Decompression Illness in a similar manner to nitrogen (Brubakk *et al*, 2003).

The different body systems are affected by saturation diving in different ways with the nervous and cardiopulmonary systems being more affected than the others. The effects are thought to be due to the interaction between the saturated dissolved gas and the pressure that is experienced by the divers working at depth.

In the nervous system, they slow down the generation and propagation of action potentials through inhibiting the sodium channels of the nerves. This impacts on the body's ability to react quickly to environmental signals by increasing reaction time and decreasing balance. In these severe cases, convulsions and tremors are common due to the severe inhibition of nervous function caused by the gases interaction with it. In these instances the body can become unconscious in an attempt to prevent further damage to the brain and nervous tissue (Brubakk *et al*, 2003).

In the cardiopulmonary system free flowing bubbles in the bloodstream can become trapped and cause a gas embolism in any area of the vasculature leading to a variety of downstream effects. These can include effects similar to a myocardial infarction if the embolism occurs in the coronary arteries or a stroke if in the cerebral arteries. Severe tissue damage can occur in areas where a gas embolism has impaired the blood supply and tissue death is possible. Less severe

are the instances of rupture of the capillaries caused by the bubbles leading to irritable rashes and bruises on the skin around the locations of the embolism. The lungs are severely affected by the overexpansion caused by the gases saturated in the lung and surrounding tissue. This can rupture both the alveoli and the lung itself causing a pneumothorax leading to a collapsed lung or an arterial gas embolism (Brubakk *et al*, 2003).

The musculoskeletal system is poorly vascularised and is a prime location for the gases to become trapped causing joint and muscle pain. Further damage may occur through inflammation caused by an immunological response to the gaseous bubbles. This can lead to problems with necrosis of the internal bone structure due to its disruption by the bubbles of saturated gas dissolved within the bone marrow (Brubakk *et al*, 2003).

Other body systems are less affected physiologically but there are still small changes observed. In the male reproduction system a significant decrease in fertility has been observed in some saturation divers due to the saturation of the surrounding tissues (Aitken *et al*, 2000). The immune system is also affected in a similar way to the male testes with a decrease observed in the CD4:CD8 ratio and in the number of $CD4^+$ (T-Helper) cells which increases the susceptibility to infection (Matsuo *et al*, 2000).

Overall the physiological effects of the saturation of the tissues are widespread, affecting most of the body. However the mechanisms for some of the conditions are poorly understood and further research is needed to clarify the complete mechanisms of action.

The effects of the gases under pressure also allow for the treatment of the disorders caused by the expansion of the gas bubbles to be relatively simple. This requires the recompression of the diver in a chamber to 18m (60ft) at which depth the bubbles are compressed to a size that they can be expelled from the lungs. The process is helped by breathing 100% oxygen. The oxygen replaces the dissolved gas increasing the rate at which the size decrease is observed.

Chapter 4 – The Neurological Effects of Saturation Diving

The brain and the nervous system are potentially the most significant parts of the body affected by saturation diving. Damage to these systems can result in long term suffering and fatalities. The reasons why the effects observed occur and what causes them has only recently been uncovered. It is understood that after long durations of breathing a high partial pressure of oxygen, convulsions and unconsciousness can occur and similar effects can occur through inert gas narcosis (Brubakk *et al*, 2003). However why and how these affect the body in the long and short term are still poorly defined. It is known that there are dangers associated with saturation diving, which can lead to short term neurological effects such as convulsions, loss of consciousness and tunnel vision but in the long term, the neurological effects of these occurring multiple times has not been studied in depth and is still relatively unknown. Repeated exposures to high pressure as experienced in saturation diving can have detrimental effects on neurological health even without exposure to a diving accident. The deeper the dive the greater the potential damage to the body (Brubakk *et al,* 2003/Værnes *et al,* 1989).

Damage to the neurological systems (Type II DCS) is most commonly sustained after involvement in a diving related accident that has impacted the function of the nervous system and the brain through the presence of gaseous bubbles in the surrounding tissues. These have appeared as a result of the conditions relating to the accident and can cause all the described symptoms as a result of where the bubbles get trapped. A previous exposure to such an incident increases the likelihood of another exposure happening in addition to increasing the risk that permanent damage will be done (Værnes *et al,* 1989). This is because the damage caused by the previous exposure has not healed and another exposure increases the impact on the tissues further impairing their functions. .

4.2 – The Short Term Neurological Effects

The short term neurological effects of saturation diving are those that are most well known because they have been studied in more depth and can be readily related to recreational diving. During day to day operations these are the effects that are most likely to be encountered. The causes are varied but most of them are related to problems/malfunctions which are encountered rather than the dive itself. These include; hyperoxia, hypoxia, decompression sickness, high pressure nervous syndrome (HPNS), nitrogen narcosis and barotrauma. The first five conditions can induce convulsions, unconsciousness and death. All of the conditions can be very painful and dangerous for a diver to have to cope with during operations. In order for the above conditions to affect the neurological system they must be the severe form. This is because lower partial pressures of gas are not strong enough to elicit the physiological effects associated with the conditions in a short period of time, whereas saturation with high partial pressures of gas can overload the system and elicit the effects much quicker (Brubakk *et al*, 2003). The effects are explained in the individual subsections of the disorders.

The conditions outlined above are not the only changes observed during and after saturation dives. In tests conducted at the National Hyperbaric Centre in Aberdeen during the early 1990s, divers were tested on their vestibular health related to postural control. All the divers used in the experiments had been diving for a long period of time (9-22 years, mean of 12.75) and had conducted operations to depths greater than 200m (656ft) (Nordahl *et al*, 1995). Their postural control was measured during a 'dry dive' in the chamber whilst breathing Heliox to a maximum depth of 470m (1542ft), maintained for 8 days (4 days compression and isocompression) and 200m (656ft) for 4 days before 18 days of decompression to total a 32 day dive (Nordahl *et al*, 1995). Balance was measured at both depths by using a balance platform test with their eyes open and closed and the results taken and analysed. A significant difference was observed in

75% (3 out of 4 divers) in the sway distance and path length when measured at depth compared to the results observed at the surface both pre and post dive (which were similar) (Nordahl *et al*, 1995).

These effects are thought to be observed because of the effects of High Pressure Nervous Syndrome (HPNS) as the onset depth is similar to the depth at which HPNS starts although this varies between subjects. Disturbance of the vestibulo-cerebellar section of the balance system is the likeliest cause although this has not been proven (Nordahl *et al*, 1995). It is the area of vestibular control in the brain and alterations to the function of this region have dramatic consequences on the ability to maintain balance. Here it is possible that the interactions between the gas molecules (nitrogen and helium) and the nervous system and the effect this has on nerve conduction under high pressures (see Nitrogen Narcosis) may reduce balance properties of divers. The changes in balance control have similar properties to narcosis with the improvement noted as the depth decreases. There is no 'hangover' from the effects felt whilst under pressure in a similar way to narcosis (Nordahl *et al*, 1995). Hence the inhibition of the initiation and propagation of action potentials along the nerves of the vestibular system may be decreased under pressure due to the gaseous interactions within the sodium channels. As a result of the decreased conduction the body is unable to respond as quickly to the small changes in posture that occur all the time thus increasing the path length observed in the experiments.

4.3 – The Long Term Neurological Effects

Studies in Norway on saturation divers' neuropsychological health have shown that in 20% of saturation divers who had been diving for more than 3.5 years (or a single deep saturation dive of between 300 and 500m (1000-1600ft)) there was evidence of mild to moderate impairment of autonomic reaction time. This was more prominent in those divers with low autonomic reactivity before diving (Værnes *et al*, 1989). There was also an increase in tremors and impairment noted in the spatial memory and vigilance of the divers (Værnes *et al*, 1989). A positive correlation has been shown between the length of time diving and the severity of the impairments observed (Værnes *et al*, 1989). Overall this leads to a decrease in the effectiveness of a diver over time especially if they are working in deep conditions (greater than 300m (1000ft)). This can be minimised by ensuring a break between dives although a long absence from diving is not commercially viable. In the North Sea, to minimise any long term complications from living in saturation, a one month on/one month off policy (maximum of 28 continuous days in saturation) is operated. Although if required through the unavailability of another diver due to illness or another complication divers can come up from one dive and descend straight into another one (Smith, 2008/9).

Evidence of improvement in neural health was seen after a year without any saturation dives but this was only observed in the vigilance tests. No significant improvements were observed in any of the other factors (Værnes *et al*, 1989). This means that although no obvious neurological damage is observed in the short term during saturation diving, in the long term it can have some severely debilitating effects on the nervous systems which are incurable and can be harmful to the quality of life after diving. Whilst this is likely to be due to the normal pattern of neural repair there is no evidence to support this from the results seen in the experiments.

In addition significant changes were observed in all parts of the nervous system (central (CNS), peripheral (PNS) and autonomic sections (ANS)) with the different nervous systems showing different changes. In the CNS the main symptom was a significant decrease in concentration span (Todnem *et al*, 1991). In the PNS the main symptom was paraesthesia in the peripheries (hands and feet). There was no specific stand out feature in the ANS despite the significant differences observed between the controls and the saturation divers that were defined through interviews (Todnem *et al*, 1991).

Analysis of the sources has led me to believe that these changes to the nervous system in the long term are likely to be caused by similar physiological mechanisms to the short term effects. In this way the interaction between the breathing gases and the nervous tissue when the body is saturated with gas decreases the rate at which nervous transmission can occur. It may also damage the nervous structure itself through inflammation damage caused by the immune system's reaction to the gaseous bubbles or directly by the bubbles coming into contact with the neural tissue. These effects damage the ability for the nerves to generate and transmit signals, resulting in the effects described. The slight recovery seen is evidence of the body repairing this damage to attempt to improve nervous communication.

4.4 – Nitrogen Narcosis

Nitrogen narcosis is the commonest neurological consequence of diving and can be experienced by any diver (whilst breathing air) below around 27m (88ft). This depth of onset can be increased by changing the breathing mixture to contain less nitrogen. This increases the depth you can go without observing the effects of narcosis as they are dependent on the nitrogen concentration in the breathing gas. It was described by the prominent diving pioneer Jacques Cousteau as the "Rapture of the Deep" (Lounsbury *et al*, 2002) and it has a number of debilitating effects on the function of humans under pressure. The mechanism of action is still

relatively unknown although there are a number of theories and it is thought to be similar to that of anaesthetics (Lounsbury *et al*, 2002). Anaesthetics inhibit the sodium channels affecting the way action potentials are generated by neurons and their propagation along the neurons. This increases with the concentration of anaesthetic and gives very similar symptoms to those seen in narcosis (Web Reference 6). The effects increase in severity with depth.

Observing these effects in divers can be compared to intoxication with alcohol and is described in diving circles as the 'martini law'. As the depth increases the effects observed are similar to having a set number of gin martinis (increase of 1 for each 10m (33ft) increase in depth) (Lounsbury *et al*, 2002). Initially reaction time is reduced; reasoning is decreased (usually mild) and manual dexterity is reduced. As depth increases beyond 45m (150ft) they become more severe with a feeling of euphoria; decrease in mental awareness and concentration; peripheral tingling and a disregard to personal safety (particularly dangerous in a sub-aqua environment). Beyond 90m (300ft), on air, divers begin to lose consciousness and can encounter amnesia or death by drowning. The problems with narcosis are minimised in saturation diving by breathing mixtures with reduced/no nitrogen in them (Trimix/Heliox) (Lounsbury *et al*, 2002) although it can still occur, just at much greater depths than those observed whilst breathing air. However at great depths nitrogen narcosis is not the only problem as the pressure also begins to have an impact body beyond around 120m (400ft).

4.5 – High Pressure Nervous Syndrome (HPNS)

High Pressure Nervous Syndrome is the name given to the condition which is encountered by saturation divers below 120m (400ft) although most occurrences occur below 180m (600ft). Originally it was thought to be caused by Helium being narcotic but experiments with liquid breathing in animals has shown that pressure is the main factor involved (Lounsbury *et al*, 2002). The effects observed are similar to nitrogen narcosis with the main symptom observed being neurological tremors (Talpalar *et al*, 2006). As a result of this the mechanism of action is likely to be similar to narcosis but with the neural processes affected by the pressure. This is because of the general absence of nitrogen in the breathing mixtures used at the depth HPNS occurs. Despite this it is still possible for divers to work efficiently between 300 and 500m (1000ft-1600ft). Methods to reduce HPNS have been researched but it still occurs, only at greater depths (around 600m (2000ft) using Hydrogen/Helium/Oxygen gas mixtures). The occurrence of HPNS has been reduced by slowing the compression rates during the dives (Lounsbury *et al*, 2002). In addition it has been found that small additions of nitrogen to the breathing mixtures can ease the symptoms of HPNS. This is through the induction of slight nitrogen narcosis which prevents the symptoms associated with HPNS, although the mechanism is unknown (Brubakk *et al*, 2003). This limits the safe working depth range to between 450 and 600m (1500-2000ft) where HPNS effects are manageable but not too severe using commercially viable breathing gases (heliox) (Lounsbury *et al*, 2002).

4.6 – Neurological Decompression Sickness (Type II DCS)

Of all the neurological complications that can be encountered whilst diving, Type II Decompression Sickness is the most severe. Conversely of the 3 main neurological disorders, it is the least likely to occur. Prior occurrences though increase the chances of a second incidence (Brubakk *et al*, 2003).

It is caused by the migration of nitrogen gas bubbles from the tissues into the bloodstream, through the heart into the systemic circulation. This is caused either by a pulmonary barotrauma or an arterial gas embolism (AGE) (Brubakk *et al*, 2003). Once in the systemic circulation the bubbles can become trapped anywhere resulting in various severe effects. Neurologically the most severe of these is a cranial arterial gas embolism (CAGE) which is similar to a stroke in the physiological effect (blocking of blood supply to areas of the brain) (Neuman, 2002). The bubbles can lodge anywhere in the nervous system or brain with potentially fatal outcomes (Brubakk *et al* 2003). Wherever the bubbles get trapped there can be activation of the immune system, including complement, which can induce inflammation to the affected region putting pressure on the surrounding nerves and causing either severe pain or loss of function (or both) (Brubakk *et al*, 2003).

Chapter 5 – The Cardiovascular and Respiratory Effects of Saturation Diving

The Cardiovascular system may also be affected by saturation diving and diving in general because of the direct contact of the breathing gases with the lungs and the blood vessels. This primary contact results in the cardiovascular system being the first major body system to be affected by any problems encountered whilst on a dive and it shows several effects of this interaction (Brubakk *et al*, 2003). In saturation diving, the health of the lungs is paramount to ensuring an incident free dive occurs. Any problems with the lungs can allow bubbles of gas to permeate into the blood vessels where they can cause a multitude of problems most of which are serious and some fatal. The majority of these effects are observed in the short term but can have lasting long term effects as well. The lungs in particular are affected in the long term from saturation diving with the main observations being a difference in vital capacity, the flow volume of the lungs and the transfer capability of the lungs for Carbon Monoxide (CO) (Thorsen *et al*, 1994). The changes in lung function are down to the artificially high pressure, high density and high oxygen environment in which the divers work under (Thorsen *et al*, 1990). These changes make breathing much more challenging and significantly constrict the air spaces in the body, the largest of which are the lungs (Cotes *et al*, 1987).

The cardiovascular system is less severely affected than the pulmonary system because there is less contact between it and the environment. The problems related to the cardiovascular system are severe and can often be fatal (e.g. AGE). They are related to the presence of gas bubbles in the vasculature that have leaked through the alveoli or diffused from tissues on ascent.

5.2 – The Short Term Cardiovascular and Respiratory Effects

In the short term the cardiovascular system is not severely affected by saturation diving unless there are some problems on the dive which induce in descending order of severity: an arterial gas embolism (AGE), a barotrauma, or the perforation of blood capillaries caused by nitrogen gas bubbles. The volume of the system is compressed at depth and the lungs therefore have a much smaller capacity. In addition, due to the density of the breathing gases being much greater, breathing is more laboured at depth and can affect the ability of the lungs to function effectively.

As a result of the pressure, lung function is altered after a single saturation dive (14-30 days in duration) from normal although these changes do partially recover after a period without diving (˜3-6 weeks) (Thorsen *et al*, 1990). However a full recovery can take much longer. There was no significant change observed in the dynamic lung volume but significant changes have been observed in other lung functions. These include an increase in the total lung capacity (by 4.3%) and in the residual lung volume (by 14.8%) as well as a large increase in the closing volume (by 16.7%). The increase in these factors correlated with a decrease in the transfer factor for Carbon Monoxide (reduction from 13.0 ± 1.6 to 11.8 ± 1.7 mmol/min/kPa when a haemoglobin concentration of 146g/l is used) with no change in the alveolar volume observed (Thorsen *et al*, 1990). These functions are particularly altered on deep saturation dives (below 180m (590ft)) because of the greater pressure of the surrounding environment and the effect it has on the body and the breathing gases used. The reasons for these changes in the lung volume could be down to the compression of the lungs during the descent and their subsequent re-expansion as the diver resurfaces leading to some stretching of the lung tissue evidenced by the results. The effects on the other lung factors are possibly down to the irritation of the lung tissue caused by spending a long period of time in a hyperoxic and high pressure environment. Venous gas micro-embolisms (small bubbles blocking the veins) are also thought to impact the functions of the lungs through

partial asphyxiation of the tissues and slight oedema caused by the leakage of fluid from the vessels where the embolisms have occurred (Thorsen *et al*, 1994). The decrease in the Carbon Monoxide (CO) transfer factor could be due to the tissues being saturated with other gases which displace the ability of those tissues to transfer CO and thus decreasing their efficiency.

In the short term there is no lasting damage done to the blood vessels even if some of the capillaries have been perforated by bubbles of gas. These are repaired by the bodies repair mechanisms without any residual effects on the integrity of the blood vessels. However complete regression of any symptoms is advisable before undertaking any further dives to prevent any increase in the risk of long term damage (Thorsen *et al*, 1990).

5.3 – The Long Term Cardiovascular and Respiratory Effects

The effects observed after a single saturation dive are still present after a year without diving and there is little improvement seen although there was some normalisation observed after 1-2 months (Thorsen *et al*, 1990). In the longer term the cardiopulmonary health of the divers decreases with particular damage done to the conductance ability of the smaller bronchioles. This leads to a significant decrease in the forced expiration volume (1.8%), the forced mid flow expiratory rate (6.5%) and in the transfer factor of CO (4.6%) without a significant change in the maximum forced vital capacity after three years of saturation diving (Skogstad *et al*, 2000). Over a period of 6 years in the same study it was observed that there was a significant reduction in the forced expiration volume and the forced expired volume in one second of 0.91% and 0.84% per annum respectively compared to 0.24% and 0.16% for the control group of policemen (Skogstad *et al*, 2002). A similar pattern was observed in the transfer factor of CO with annual decrease of 1.33% observed in the divers and only 0.43% in the policemen. These observations correlated with the number of dives done suggesting that the greater number of dives within a set

time period, the greater the damage to the cardiopulmonary system. After the cessation of diving work there is little improvement in the results (Skogstad *et al*, 2002).

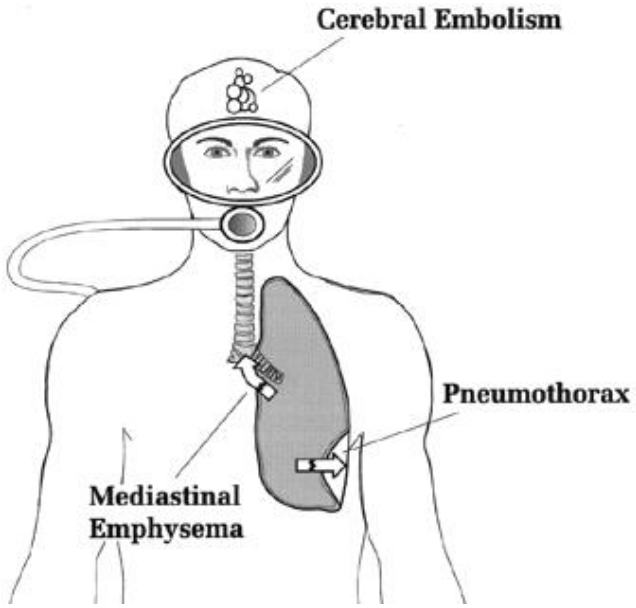
The heart and the blood vessels are less affected than the lungs and over the long term there is no confirmation of any lasting effects from diving on them (Thorsen *et al*, 1990). Any injury to the vasculature is rapidly repaired thus preventing any long term effects on the surrounding tissues. However severe long term effects can result from damage caused by a diving injury such as a barotrauma or AGE. Any tissue deprived of an adequate blood supply for too long will have suffered from permanent damage. The effects of this damage are much more debilitating than the damage itself with paralysis, brain damage and tissue damage (and possible gangrene) all possible. Any one of these long term effects of cardiopulmonary damage will indefinitely halt a diving career.

5.4 – Barotrauma

A barotrauma is an injury caused by a change in pressure (Lounsbury *et al*, 2002) and is commonly observed in diving and is rarely serious. It can occur in any gas filled cavity in the body or in the divers' equipment (mask or suit) and can either be caused by the creation of a slight vacuum (equipment) or due to the expansion of the gas in the body during a dive (body cavities). This can occur either on ascent or descent from a dive with similar symptoms observed for both. The most common form of barotrauma is ear barotrauma (in particular of the middle ear) caused by an inability to equalise the pressure between the outside pressure and the pressure behind the tympanic membrane, either on ascent (uncommon) or descent (Lounsbury *et al*, 2002). This results in haemorrhaging in the ear and a possible rupture of the ear drum prohibiting diving until it is completely resolved. Inner ear barotrauma can develop from the bursting of either the round window or the vestibular membrane, which can lead to deafness and tinnitus. Deafness can either be instantaneous (severe bursting of the round window) or onset

over a couple of days (less severe burst). If treated quickly hearing normally returns and no adverse effects are suffered but no diving is permitted whilst the injury heals (Lounsbury *et al*, 2002).

The most severe form of barotrauma is a pulmonary barotrauma which occurs in the lungs. This occurs either on the ascent (more severe) or on the descent (less common/dangerous) and is caused by an overexpansion of the air in the lungs (Lounsbury *et al*, 2002). The greater the depth that is being ascended from, the more severe the damage, although it possible for it to occur at any depth. Pulmonary barotrauma can be severe with death a possibility in rare cases. There are few symptoms from the barotrauma itself but its effects can be extensive, including: pneumothorax, arterial gas embolisms (AGEs), cerebral arterial gas embolisms (CAGEs) and tissue damage. All the effects of a pulmonary barotrauma can have severe short and long term consequences on the health of the diver. The former is the least serious of the conditions and can result in a collapsed lung which results in a cessation for diving until the healing is complete. Arterial gas embolism and CAGE are the most severe of the effects that can be caused by a pulmonary barotrauma and can result in death in severe cases (Lounsbury *et al*, 2002).



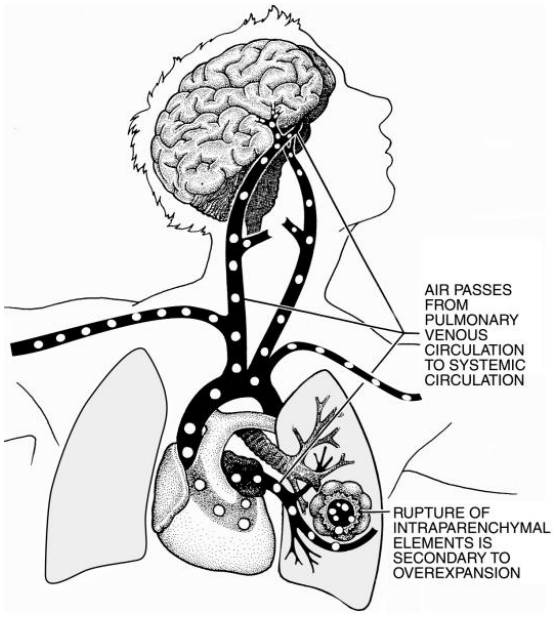


Figure 5.1 (left): Diagram showing the possible results of a pulmonary barotrauma caused by lung over expansion during ascent or decent. The one result not listed is an AGE (Web Reference 11). Figure 5.2 (right): Diagram showing the mechanism of action of an AGE caused by a pulmonary barotrauma (Neuman, 2002).

5.5 – Arterial Gas Embolism (AGE)

Arterial Gas Embolism (AGE) is one of the most serious complications that can be encountered in a diving situation. It is normally caused by a pulmonary barotrauma which allows large bubbles through the alveoli and into the vasculature (see figure 5.2). There are a number of reasons for this to occur which were described in the previous section.

The presence of bubbles of gas (usually nitrogen but other inert gases are possible) in the vasculature is not normally a problem however due to their large size they can partially or completely obstruct an artery starving the end organ of blood. Depending on where the blockage occurs the symptoms observed can be similar to a stroke (cranial (C) artery - CAGE), a myocardial infarction (coronary artery) or an obstruction in any of the other arteries in the body. As a result the effects of AGE are varied in both location and severity. In the most severe cases (˜4%) AGE results in a myocardial infarction, a loss of consciousness and a cessation of breathing (apnoea) resulting in death within minutes. The exact mechanisms behind this are unclear and have been unable to be replicated in experiments in both animals and humans. There

have been no advances into why a myocardial infarction occurs with AGE but not with other similar gaseous blockages (Neuman, 2002).

If the AGE is less severe, then the symptoms can vary depending on the location of the blockage. In a CAGE (cranial arterial gas embolism) the symptoms are similar to a stroke with loss of consciousness, confusion and lack of coordination. Some of these effects may have lasting neurological effects similar to a stroke but respond well to treatment with few residual symptoms (Neuman, 2002).

AGE is the most serious of the conditions that can be encountered during diving operations but is rarely observed in saturation diving procedures unless there is a malfunction in equipment.

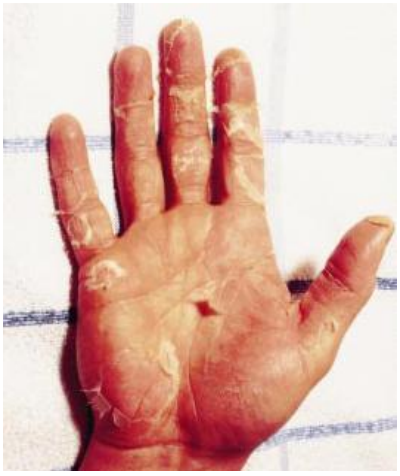
5.6 – Decompression Sickness (Type I DCS)

Type I decompression sickness is the lesser form of the illness. It is when bubbles have been trapped in the tissues of the body but have not entered the systemic circulation. These are normally in areas such as joints and muscles which have poor blood supply but can also occur in the skin (Brubakk *et al*, 2003). There are no lasting effects from this form of decompression sickness apart from a small chance of lasting joint pain/damage. If bone DCS is involved there is an increase in the risk of developing dysbaric osteonecrosis although this is more often associated with the severer form of DCS (Type II). The main effects are a 'marbling' or an itchy rash of the skin where the blood capillaries have burst and/or limb and/or joint pain. This is thought to be caused by the expansion of gaseous bubbles in these tissues where they have become trapped and initiated an immunological response from the body (pain) or have caused the rupture of a blood capillary giving rise the marbling effect (Brubakk *et al*, 2003).

5.7 – The Effects on the Skin

The effects observed on the skin from saturation diving are mainly due to problems associated with the superficial blood vessels being affected by Type I DCS (marbling) and have been described in the previous sub-chapter. However there are other forms of skin damage attributed to lengthy exposure to a hyperbaric environment.

One of the commonest skin problem noticed in saturation divers (82% - 81/98 in the North Sea) is called 'divers hand' and is characterised by peeling of the outer layers of skin on the hands and less commonly the feet (Ahlén *et al*, 1998). This can develop at any point during a dive and in some instances post dive. These symptoms last for one to two weeks before starting to heal, with some sensitivity observed for four to six weeks afterwards. It is caused by the formation of horizontal clefts in the upper sections of the *corpus striatum* (Ahlén *et al*, 1998). The reasons for the formation of these clefts are unknown but the histology shows it has the appearance of being forced apart. Unusually there is no inflammation, meaning the cause has a low antigenic reactivity, which does not activate the immune systems defences. The causes could either be abrasion on the machinery being used on the dive or the infiltration of the skin tissue by gaseous bubbles which disrupt the organisation of the cells and cause the peeling.



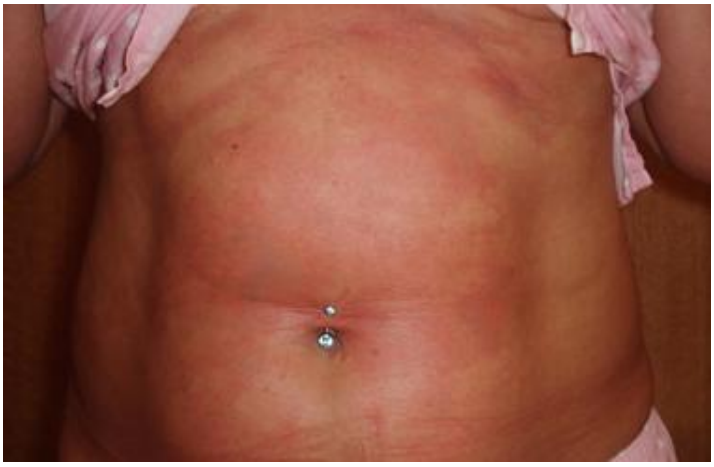


Figure 5.3 (left): A picture of an incidence of Diver's Hand showing the characteristic skin peeling on the palm of the hand caused by clefts in the corpus striatum (Ahlén *et al*, 1998). Figure 5.4 (right): A picture of the skin rash known as marbling this is often seen in cases of Type I Decompression Sickness (Web Reference 10).

The other major problem is the risk of infection from various microbes (in particular *Pseudomonas aeruginosa* and *Staphylococcus aureus*) that can flourish in the environment found within decompression chambers. It is thought that these bacteria are introduced into the systems in the seawater that is remaining on the divers' equipment when they return from a dive to the chamber (Ahlén *et al*, 2003). Once in the chamber the bacteria can cause a number of skin infections which can become persistent (Ahlén *et al*, 2000). These infections can spread quickly in the chamber and become serious, incapacitating the diver for the duration of the infection. The sites of infection are usually wounds that have been received on the dive (scratches etc.) providing an entry point for the microbes into the body. After recovery the diver can return to operations with no long term effects caused by the infection.

The treatment of the infections whilst the divers are in saturation can be problematic because of the difference in pressure between the physician and the divers, which inhibits adequate examination and diagnosis. Antibiotics can be provided but further treatment requires the recompression of the diver (or compression of the doctor). Therefore prevention of the infection is better than having to treat it. To this end the chamber and all the equipment are thoroughly cleansed to remove any microbes before the operation commences. However this has not completely prevented the infections from occurring in North Sea operations and more effective hygienic controls are required (Ahlén *et al*, 2000).

The reasons for the infections being prevalent in saturation diving systems is likely due to the favourable conditions for bacterial growth in the chamber (oxygen, humidity and warmth). The bacteria then can multiply rapidly and spread through the various systems present in the chamber, increasing the chances of them infecting one of the divers.

None of the effects on the skin are life threatening and usually heal without any adverse affects observed in the long term. The damage observed is normally superficial and recovers after a

period of time. There is no long term damage done to the hand by these effects and in most cases after healing diving can recommence (a few days to a week). In severe cases it is possible for it to end careers although this is rare.

Chapter 6 – The Musculoskeletal Effects of Saturation Diving

The musculoskeletal system is rarely affected by saturation diving in the short term. However over long periods of time there are a number of problems that develop from spending long periods of time living under pressure. These problems are thought to be a result of gaseous bubbles in the tissues that make up the musculoskeletal system, although the mechanisms behind this are still undefined (Brubakk *et al*, 2003). In the short term these problems manifest themselves as localised pain although this normally only occurs in incidences of Type I DCS (mild and severe). These can be relieved by recompressing in a decompression chamber and then decompressing slowly which removes any gaseous bubbles which are trapped in the joints. The main long term factor associated with saturation diving (and other pressurised environments although it is most common in divers) is dysbaric osteonecrosis (DON). This is the ischaemic death of the cellular components of bone and bone marrow (Brubakk, *et al*, 2002) and can lead to effects similar to osteoporosis – weakened bones with an increased likelihood of a break occurring in a fall or similar occurrence. This occurs in a small percentage of divers and the effects can be disabling in a very small percentage of cases. The likelihood of DON increases if the divers have had a prior case of DCS.

6.2 – The Short Term Musculoskeletal Effects

In the short term there is very little physiological risk to the musculoskeletal system from saturation diving because of the limited contact with the factors that cause the effects seen in both the neurological and cardiopulmonary systems. The environment in which the divers are working is main short term risk. If the work involves machinery the risk comes from falling equipment and machinery which poses the chance of fractured bones. The main physiological effect on the musculoskeletal system is the effect of type I decompression sickness. This has no long term problems associated with it and is caused in a very similar way to the more serious Type II decompression sickness that affects the neurological and cardiovascular systems. The exact mechanism as to why pain develops in the joints is unknown because large gaseous bubbles in the joints do not necessarily cause any DCS symptoms (Brubakk *et al*, 2003). Bubbles in the articular cartilage are thought to be involved in the formation of symptoms, particularly in saturation divers because of the aqueous nature of the tissue and the relative paucity of blood vessels surrounding it. This means it will absorb and release gas very slowly and could be the reason joint pain is the most commonly reported symptom in saturation divers with Type I DCS (Brubakk *et al*, 2003). This is only one of the theories of the causes of joint pain.

There are several other theories relating to how gaseous deposits in and around joints cause pain. These include those where surrounding nerves are pressurised either directly or indirectly from pressure elsewhere in the joint (e.g. bone marrow or tendons). However there are some irregularities with this in that only tendons have been reported to have this affect and there is no affect from the same issue with ligaments (Brubakk *et al*, 2003). Another explanation for joint pain comes from a problem with the nervous system within the same limb as the joint pain.

Joint pain can also be experienced during the compression phase of the dive but can be eased if a constant depth is held for 24 hours or more. This is called hyperbaric arthralgia and the mechanisms behind it are unknown although there are two theories. The first is that there is a difference in the formation of the bubbles as depth and pressure increase which irritates the joints leading to symptoms similar to a sprain as well as 'dry and grainy joints' (Lounsbury *et al*, 2002). The other theory is that there is a change in the osmolarity of the joint fluid which dehydrates the joint and causes the described symptoms. The mechanism behind this though is unclear but could be due to the dissolving of the gaseous bubbles in the joint fluid which increase the particles dissolved in the fluid. There is no link between hyperbaric arthralgia and the degeneration of joints or aseptic bone necrosis so there is no long term damage associated with it in divers. With the slow compression rates now used to combat the effects of HPNS the incidence of hyperbaric arthralgia in divers has been reduced (Lounsbury *et al*, 2002).

6.3 – The Long Term Musculoskeletal Effects

There are few long term effects of saturation diving on the muscles outside of the normal wear and tear associated with ageing. As divers are relatively active in order to maintain their fitness for the labour they perform during dives, muscle wastage due to inactivity is not a problem commonly observed, although it may be noticed for other reasons. The main long term problems are associated with the skeletal system. Necrosis of the internal bone structure is the most important, resulting in a general weakening of the bone similar to that seen in osteoporosis and is called dysbaric osteonecrosis (DON). This primarily occurs in the long bones which contain yellow (fatty) marrow such as the humerus, femur and tibia (Brubakk *et al*, 2003). They have a higher capacity for nitrogen (lipids are more soluble to nitrogen) than the shorter bones which contain the marrow that produces the blood cells (haematopoietic (red) marrow). In divers the most common site is the humeral head (head of the humerus) in the upper arm. In the majority

of cases of DON there are no symptoms that inhibit the quality of life. However, in more serious cases, which can arise from the spread of the lesions into the joints (surrounding the upper ends of the humerus and femur); the symptoms can be very debilitating (M^{c}Callum, 1984).

In the North Sea, a report by the British Medical Research Council Decompression Sickness Registry in 1981, found that 4.2% (204/4980) of commercial divers had evidence of at least one DON related lesion (Brubakk *et al*, 2003). Most of these were asymptomatic and did not cause any notable problems. There was no incidence of DON found in any divers who had not dived any deeper than 30m (100ft). A significant relationship exists between the occurrence of severe DCS and the incidence of DON, confirming the theory that inadequate decompression increases the likelihood of DON occurring in subsequent years (Brubakk *et al*, 2003). This is thought to be because DON is caused by the infiltration of the bone tissue with gaseous bubbles which if they expand whilst trapped in the bone (as seen in DCS) then they can disrupt the structure of the cells and cause the necrotic lesions observed in DON (Brubakk *et al*, 2003). In tests in the USA it was found that divers had a significantly higher risk of bone necrosis than non-diving controls (M^{c}Callum, 1984). DON is more common in saturation divers than recreational divers but most of the research into DON has been carried out on recreational or indigenous divers and not commercial saturation divers. This is because of the higher rate of DCS observed, particularly in indigenous divers, due to the dive profiles they follow and the fact a large sample size is possible. In the British Royal Navy today all divers are screened for DON as part of their routine medicals (Southerland, 2006). Any evidence of DON prevents them from continuing to dive. This is as a precaution to reduce further development of the bone lesions which can hamper the quality of life of the diver in a similar way to osteoporosis (e.g. non-traumatic factures or vertebral collapse).

Chapter 7 – The Effects of Saturation Diving on Other Body Systems

As discussed in previous chapters, the main body systems affected by saturation diving are the neurological, cardiopulmonary and musculoskeletal systems. However there are other body functions that maybe affected. Generally these are short term effects although there are some long term effects but little research has been carried out in this area.

The commonest reason for the changes documented is the repeated exposure to high pressures which have detrimental effects on the functioning of the systems and the exposure to high concentrations of gas in the tissues. There are short-term changes in the levels of certain proteins observed in saturation diving but the effects of these changes have not been experimentally defined. In the endocrine and the reproductive systems the changes are not as life threatening as those on the three major systems but can decrease the quality of life with respect to fertility.

7.2 – The Effects on Bloodstream Factors and Heat Shock Protein Expression

The factors in the bloodstream are affected by saturation diving due to changes in their levels. There are several disorders of bloodstream factors (particularly endocrine factors) which are relatively common in the population (such as diabetes mellitus) but these have no adverse affect on the health or the fitness to dive of a diver (Brubakk *et al*, 2003).

During a simulated dive, there are changes in the levels of cortisol and thyrotropin (or thyroid stimulating hormone (TSH)) in particular observed circulating in the bloodstream. This is thought to be in response to stress induced by the changes in atmospheric pressure associated with the dive. There are no long term problems associated with these changes (Popova *et al*, 2008).

Other changes observed are in the metabolic factors in the blood and the metabolism of lipids. The latter is evidenced by an increase in the levels of triglycerides, low-density lipoproteins, and polyunsaturated fatty acids in the plasma (Buravkova *et al*, 2007). The physiology behind this

occurrence has yet to be defined although it is probably also due to the altered atmosphere and the increased pressure. These also have no long term problems associated and the short term implications are minimal although adequate research is outstanding.

The concentration of erythropoietin and haemoglobin changes during the course of a dive. This though is a normal physiological change in response to increased oxygen concentrations and has no ill effects on the function of the body. After the end of a dive they rapidly return to just above normal levels (Hofsø *et al*, 2005). The changes are thought to result from a sustained change in PO_2 (partial pressure of oxygen) rather than the hyperoxic environment is the initiator these changes. However, the change in PO_2 observed is a direct result of the environmental changes encountered during a dive.

These are not the only changes that are noted in the cells of the bloodstream. There are changes noted in the immune system, particularly the lymphocyte subsets. A decrease in the CD4:CD8 ratio, decrease in the number of $CD4^+$ cells (T-helper cells) and of natural killer (NK) cells are noted, especially during the bottom phase of a dive and particularly on deep saturation dives (Matsuo *et al*, 2000). It is thought to be part of the body's stress response to the extreme environment that is present during a saturation dive. These changes increase the susceptibility of the divers to succumb to infection during a dive and skin infections in particular are common as discussed in section 5.4.

There is an increased expression of heat shock proteins (HSPs); that can also be induced by oxidative stress (Matsuo *et al*, 2000). In a hyperbaric environment the oxidative stress is low, allowing the examination of their expression without interference. In this instance HSP-27 is an effective marker for hyperbaric stress as it has increased expression in these conditions which has effects on the expression of B-lymphocytes. This is thought to contribute to the lymphocyte subset changes observed in divers during saturation dives (Matsuo *et al*, 2000). Both these changes can be used as identification factors for hyperbaric stress with the lymphocyte subset

changes being an early (decompression phase) indicator and the altered expression of HSP-27 for the later stages of a dive (recompression) and post dive. They are both short term changes returning to normal within a couple of days of recompression from a saturation dive and so having no impact on the availability of a diver for operations.

7.3 – The Effects on the Reproductive System

There is only one affect that has been observed in saturation diving on the reproductive system and it is specific to males. This is a significant decrease in the quality of semen observed directly after the decompression phase of the dive and is relative to the semen quality prior to the dive began (Aitken *et al*, 2000). During the dive there were no changes recorded in semen concentration, however three months after the dive the concentration had decreased to levels associated with low sperm count. A similar response was noted in the number of rapidly moving sperm and their overall motility with this being normal during the dive but decreasing substantially in the period post dive (Aitken *et al*, 2000). As a result the effective fertility of the subjects is dramatically reduced to the extent where due to their low fertilising potential the ability to fertilise an egg is minimal.

These changes in semen quality tend to persist and full recovery may not occur even after 9 months with even further decreases in quality observed in some cases (Aitken *et al*, 2000). Similar results have been observed in previous experiments in the 1980s using rodents and a heliox environment but the physiology of the relationship was not confirmed. Further research is now required to discover the underlying physiology for this quality decrease. It is known that spermatozoa are harmed by oxidative stress but no evidence of an increase was observed in the experiments (Aitken *et al*, 2000). Therefore the decrease is likely to be also due to the effects of the pressure (greater than 30 bar) and the breathing gases (Heliox) on sperm production, maturation and function. Due to the low number of women in the commercial saturation diving

field there have been few studies done of the effects on the female reproductive systems of saturation diving. As a result there is no data to compare the two reproductive systems.

7.4 – The Environmental Effects

The environment has many risks for a diver particularly where heavy machinery such as encountered whilst working in the oil industry is involved. On the whole these are not life threatening but minor accidents can rapidly become major incidents due to the nature of the extreme environment they occur in (Brubakk *et al*, 2003).

The main dangers from the environment are the possibility of equipment or the workings falling on the diver(s) causing a blunt force injury. The risks faced by this injury vary depending on the location of the injury and whether the equipment or the workings have trapped the diver so that they are rendered immobile. These can also lead to other serious conditions such as hypothermia if the diver's protective clothing is damaged. If the accident results in the diver becoming unconscious, then the implications can be severe. An increased risk of decompression illness occurs due to the loss of buoyancy control with the resulting possibility of an uncontrolled ascent. However usually there is a backup diver on hand to provide emergency assistance and so the chances of serious DCI are reduced but the increased risk of hypothermia is the big danger (Brubakk *et al*, 2003).

7.5: Hypothermia

Falling workings are not the only risk; the divers normally find themselves working in cold water (e.g. the North Sea). This can lead to hypothermia despite all the protective equipment provided if they remain at the dive site for too long a period of time, or if they rupture their protective suits with either their equipment or on the workings (Brubakk *et al*, 2003). Cold water enters the insulation of the suit and decreases the duration of time that it takes for the effects of hypothermia to set in. Hypothermia leads to contraction of the blood vessels in the peripheries which results in reduced circulation to those areas of tissue. This reduction in circulation reduces the capacity of the tissues to expel any dissolved gases that are present; increasing the likelihood of the bubbles becoming trapped in the tissues and causing decompression sickness (Brubakk *et al*, 2003). In addition to the increased risk of decompression sickness there is also an increased risk of a gas embolism caused by bubbles present in the bloodstream because of the constriction of the blood vessels caused by the hypothermia. The constriction also significantly increases the peripheral resistance of the blood vessels which increases the presence of bubbles (detected by Doppler ultrasonic transducer) in the bloodstream by a large margin (25% in 40°C experiments compared to 75% of subjects at 10°C). This increase in the presence of bubbles increases the chances that decompression sickness will occur especially when combined with the constriction of the blood vessels that is also present (Mekjavić *et al*, 1989). These risks are ever present when diving in cold environments and can occur in various degrees of severity despite the protective clothing that is worn by the divers to maintain body temperature.

Chapter 8 – The Treatments for Diving Related Disorders

Due to the similar nature of the causes of diving related disorders, the treatments are often much the same. The standard treatment is a period of time in a recompression chamber during which time the diver is recompressed before beginning a slow decompression phase. Recompression therapy usually follows either the procedures of the Royal Navy (RN) tables and the United States Navy (USN) tables. These tables were developed in the 1960s and are very similar except for their numbering (RN Table 62 = USN Table 6) (Web Reference 7).

There are different tables depending on the severity of the decompression illness. These are used in sequential order and always start with the same table (RN 62/USN 6) before progressing to the other tables if symptoms persist after the first round of decompression therapy. Both tables use a combination of breathing air and 100% oxygen during the recompression and attain a maximum depth of 18m (60ft). Consequently this reduces the bubble size allowing them to be expelled through the lungs. This is achieved by the oxygen replacing the nitrogen that is in the bubbles by diffusion bringing about a further reduction in size on top of the recompression of the diver that allows the expulsion of the bubbles (Brubakk *et al*, 2003/Web Reference 7).

The length of time the recompression therapy takes depends on the severity of the decompression illness and whether or not the symptoms have been relieved after the first treatment. Both the USN Table 6 and the RN Table 62 (the basic treatment programmes) last for two hundred and eighty five minutes (4h 45min) after which time most symptoms have been relieved (Brubakk *et al*, 2003/Web Reference 7).

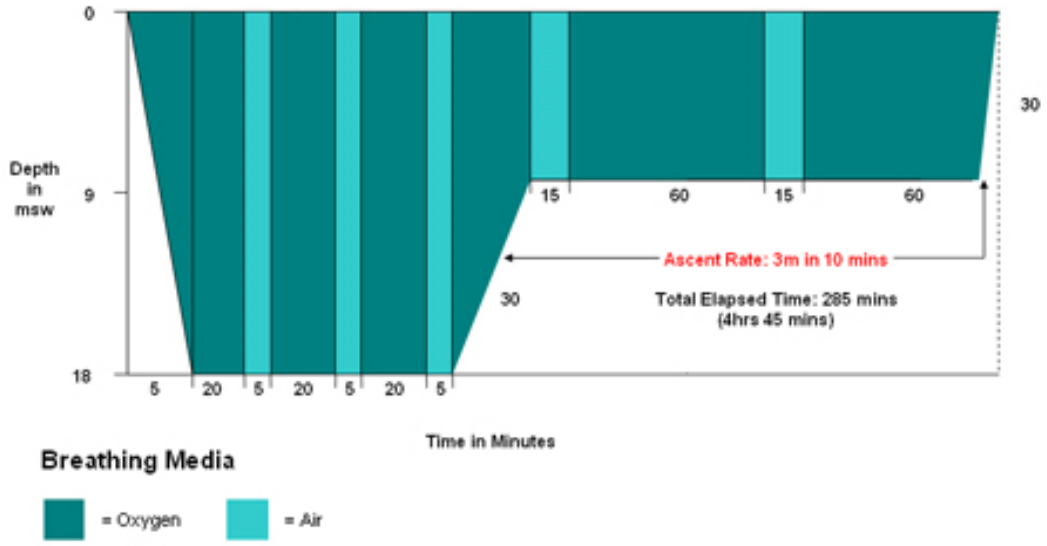


Figure 8.1: Royal Navy Table 62 (USN Table 6). This is the standard recompression table used in the treatment of diving related disorders (Web Reference 7).

The outcome following decompression therapy is very favourable and most cases result in recovery from decompression illness. An average success rate of 81% can be expected based on data calculated from a series of papers on the treatment of DCI using the USN tables (Brubakk *et al*, 2003).

Currently saturation divers rarely require decompression therapy with the improvements of the safety regulations that have occurred. However the risk is ever present. The majority of the treatments are given to recreational or indigenous divers who have developed one of the diving related disorders through making too rapid an ascent (Brubakk *et al*, 2003).

8.2 – Decompression Sickness (DCS)

The standard treatment of decompression sickness follows the normal recompression therapy procedure as laid down in RN treatment table 62 or USN treatment table 6. This involves a rapid decompression to 18m (60ft) whilst breathing air taking 5 minutes, followed by alternate breathing of 100% oxygen in 20 minute blocks, then air for 5 minutes to avoid the risks of hyperoxia. Three cycles of this last for an hour before the slow ascent (30 minutes) to 9m (30ft) whilst breathing 100% oxygen. The diver is held at this depth until the end of the treatment. At 9m (30ft) the diver breathes 100% oxygen for 60 minute periods interspaced by 15 minute periods of air breathing again to avoid the effects of hyperoxia. After two cycles at 9m (30ft) (2.5 hours) the diver undergoes a further slow ascent to the surface (30 minutes). If the symptoms have not been relieved after the first cycle of treatment at either 18m (60ft) or 9m (30ft) then the cycles can be repeated until there are no residual symptoms (Brubakk *et al*, 2003/Web Reference 7). This series of cycles should reduce the bubble size to the extent that they can be exhaled through the lungs and the symptoms of DCS are relieved.

Occasionally other breathing gases are used instead of air to increase the partial pressure of oxygen hence increasing the rate of reduction in size of the bubbles. Nitrox 50:50 (50% N_2: 50% O_2) or Heliox 50:50 (50% He_2: 50% O_2) are commonly used with tables that require depth greater than 18m (60ft) (Brubakk *et al*, 2003).

In saturation diving because of the depth at which the incidence has often occurred at the use of USN Table 6/RN Table 62 is insufficient to remove the effects of DCS. A longer time and greater depth maybe required for the adequate resolution of the symptoms. In these incidences the use of deeper tables is required; either USN Table 4 (less commonly used nowadays) or RN Table 71 (more commonly used). Both tables last over 40 hours in duration. The RN Table 71 (61m (200ft)) is recommended because it is both longer and deeper than USN Table 4 (50m

(165ft)) which has fallen out of favour because of failures to adequately remove all symptoms. In both tables air is breathed throughout the treatment (updated to include the same cycles as seen in USN table 6/RN table 62 from 18m (60ft)) although in the modern revisions of USN Table 4 there are 25 minute/5 minute O_2/air cycles to improve the effectiveness of the treatment (Brubakk *et al*, 2003).

Royal Navy table 71 is 47 hours and 44 minutes in duration and usually air is breathed throughout the treatment. The first section involves a quick descent to the treatment depth, usually between 60 and 70m (200ft-230ft) depending on the breathing gas. 61m (200ft) is the maximum depth if using air. This depth is held for 30 minutes before a rapid ascent to 50m (165ft) in 1 minute (if at 61m (200ft)). After this the diver is slowly raised towards the surface at steadily decreasing rates of ascent (6m (20ft)/h, 3m (10ft)/h, 2m (7ft)/h and 1.5m (5ft)/h) taking 17 hours to ascend to 22m (70ft). From 22m (70ft) the ascent rate is decreased further to 1m (3ft) an hour for a 10 hour decompression to 12m (35ft). From here it takes a further 20 hours to decompress the diver to the surface (Brubakk *et al*, 2003). This treatment programme generally removes most symptoms of DCI encountered in commercial and saturation diving.

8.3 – Barotrauma and Arterial Gas Embolism (AGE)

Again the treatment for barotrauma and AGE is very similar to that seen for decompression sickness with similar recompression procedures followed. However because of the more serious nature of the complaints a longer time recompressing is generally required to relieve the symptoms and a lower success rate is seen (Brubakk *et al*, 2003). In these cases the treatment programme depends on the severity of the case (same with DCS incidents) and therefore there are a variety of different ways to successfully treat the conditions with recompression therapy. The most common treatment is a variation of USN table 6 called table 6A. This is the same as table 6 except for a 30 minute period at 50m (165ft) at the start of the treatment breathing either air or Nitrox 50:50. After this excursion there is a quick ascent to 18m (60ft) from where the procedures used in USN table 6 are followed until the end of the treatment programme (Brubakk *et al*, 2003).

8.4 – Nitrogen Narcosis and High Pressure Nervous Syndrome

There is no need for recompression therapy in the treatment of Nitrogen Narcosis or HPNS because as the diver ascends towards the surface from the depth have been at the symptoms are relieved. This normally relieves the symptoms of both disorders and no further treatment is required. The incidence can be reduced by breathing gas mixtures that have less nitrogen in them to avoid Nitrogen Narcosis (e.g. Heliox, Trimix or Nitrox). To reduce the occurrence of HPNS slower compression rates (Lounsbury *et al*, 2002) and/or the addition of small amounts of nitrogen to the breathing mixture are used (Brubakk *et al*, 2003). These procedures are not totally protective but increase the depth at which they might have been encountered if breathing air (nitrogen narcosis) or Heliox (HPNS) (Brubakk *et al*, 2003).

8.5 – The Future of Diving Related Disorders Treatment

The future of the treatment of diving related disorders is likely to be the same as the procedures used currently with adaptations made where necessary as new information comes to light.

One interesting area of development is the use of perfluorocarbon emulsions, injected into the patient's veins. Their many functions include being rich in surfactants, highly soluble for both oxygen and nitrogen; increasing plasma volume and decreasing the viscosity of the blood inducing improvements in microcirculation and possibly some anticoagulant properties (Brubakk *et al*, 2003). These effects will improve the efficiency in which the bubbles caused by the disorders can be expelled from the body through the lungs and thus reduce the time spent in the decompression chamber for treatment. The possible reasons for this are the bubbles dissolve in the emulsions rapidly and due to the increased viscosity of the blood are speedily transported to the lungs where the gas is expelled. On top of the possible reduction in treatment time the emulsions may also reduce the incidence of blood clots being caused by the bubbles due to the anticoagulant properties and the fact that they make the blood less viscous.

In addition to the research into perfluorocarbon emulsions, researchers are also investigated ways of managing the immune system, in particular the complement system to attempt to prevent or reduce the effects caused by this system reacting to the perceived foreign objects. These include inflammation and irritation caused by the bubbles in the body initiated a reaction. Control of this process would reduce the immunological effects of DCI and assist in decreasing the recovery time (Brubakk *et* al, 2003).

Further research into the physiology of the conditions described is also required so that improvements in treatments can be made in response to the findings of the investigations. The combination of this and possible future treatments will lead to more effective management and an improved recovery rate particularly in severe decompression disorders.

Chapter 9 – Summary of Overall Effects on the Body of Saturation Diving

Saturation diving has many potential effects on the body both long and short term, particularly but not exclusively on the neurological, musculoskeletal and the cardiopulmonary systems. Most of the short term effects observed are reversible although they can increase the risk of long term problems occurring in various body systems. Repeated incidences of decompression disorders increase the likelihood of long term permanent damage, as does deep saturation diving (below 300m (1000ft)). The long term effects resulting from the disorders are generally untreatable. Therefore the best way to treat them is by avoiding the causative conditions in the first place.

Overall however the effects of saturation diving are now well known although the exact physiology behind the mechanisms is still relatively poorly understood and further funding and research is required to improve the understanding of the body's responses to exposure to the high pressure, hyperoxic environment that is generally associated with saturation diving. Full understanding of these mechanisms would allow for better regulation within the industry, the development of safer ways to undertake the operations and also improvements in the treatments currently available.

The treatments available and increase in the safety standards applied in saturation diving over the last two or three decades have reduced the long term effects of many of the disorders. Prevention of the causes of the diseases and their effects is naturally better than treatment and is more effective. If the increases in safety standards that have been seen in the last two decades are continued in the future then the incidences of the harmful effects of saturation diving on the body will continue to decrease.

Still further research is required into the exact physiology behind many of the conditions which would allow for further improvements in dive practice. Treatments to reduce the long term effects associated with saturation diving and diving in general are currently lacking and further

research into the risk factors are required to provide improved treatments of them. This research is crucial to the future developments in techniques for both diving and treatments but requires money to be invested to ensure the future security and continued safety in the industry. Any advances made in the treatment of diving related disorders in the saturation diving community can then be applied across the diving community worldwide. The result would be an overall decrease in the number of injuries and deaths particularly in the recreational diving community.

Chapter 10 – The Future of Saturation Diving

The future of saturation diving is in doubt because of the increase in the effectiveness of remotely operated underwater vehicles (ROV) and the need to reduce costs. However there are still jobs that require divers and will continue to do so in the future because of their intricate nature or confined spaces. As ROV technology improves the number of subsea jobs they will replace divers on will increase, as companies attempt to cut expenditure whilst still maintaining a good working efficiency and ensuring a successful completion of the task.

Saturation divers are expensive because of the equipment needed (DSVs and decompression chambers) and their wages (up to £1000/day when in saturation (Smith, 2008/9)). Further research into identifying suitable breathing gases that will be both commercially viable and allow for effective working on tasks unsuitable for ROVs both at currents depths and deeper if necessary are required.

One of the main problems with saturation diving is the decompression time and the need for large ships to carry the specialised equipment required for undertaking a safe saturation diving operation. The United States Navy is currently experimenting with chemicals which dissolve the nitrogen (and other gases) bubbles found in divers into a liquid form in an attempt and reduce the need for decompression by preventing the main cause of decompression illness (Smith, 2008/9). This method is still in the very early experimental stages and its full potential is as yet uncertain. Research is ongoing into ways of reducing the required decompression time by identifying other inert gases to replace those currently in use whilst taking into account the depths involved ensuring comparable operations to those observed today to be undertaken. The other option for reducing the decompression time is liquid breathing using one of a variety of fluorocarbons. This would remove the dangers related to the compressibility of gases because liquids are non-compressible thus removing one of the main factors contributing to the costs and risks associated

with saturation diving currently. There are many problems with this theory, primarily the risk of drowning. In addition, although the fluorocarbons have a good affinity for oxygen they are very poor carriers of carbon dioxide. This makes expulsion of the exhaled carbon dioxide difficult thus increasing the risk of hypercapnia. However this theory is still very much in its infancy (because of these problems) and a workable solution is probably many decades away (Web Reference 9).

The use of atmospheric diving suits (ADSs) is another area of development into removing the problems of decompression time for divers. These maintain the pressure at the same level as the surface no matter what the depth is. In this way they are similar to space suits and submarines. These suits have been around for over two centuries but have been too cumbersome to have any real use in the diving industry (Thornton *et al*, 2001) or to replace the work of saturation divers in the last three decades. However recently they have come a long way from their primitive beginnings and are much more versatile. The current suits being developed have the capacity to replace saturation divers in some roles as well as allowing for deeper exploration than saturation divers can safely achieve. Being essentially one man submersibles they could provide the key to getting a diver to a deep site to do work that is unsuitable for an ROV to undertake and without the need for a diver to spend weeks in a decompression chamber. However there are still problems with this development including their inability to work in currents or turbid waters (Web Reference 8) which is what the conditions generally entail at the locations where the work will be carried out. In addition to these the fine control functions are still not at human dexterity levels and there are therefore certain applications where divers are still preferred. In time these will be overcome and the prevalence of ADSs in the current saturation diving industry will increase because of the economics of using them compared to a whole team of saturation divers.

Saturation divers have the advantage over the current forms of technology being developed to replace them because of certain conditions (e.g. sea state and visibility) and actions (requiring human dexterity) that only they are capable of undertaking at depth. In the future as the capabilities of both ROV and ADS technology develop and improve the situations in which it is advantageous to use saturation divers over the cheaper alternatives will be reduced. The research into alternative methods to reduce the decompression times are therefore important to provide a stop-gap measure whilst the technology is still developing the required capabilities. Further research into the physiology behind the diving related disorders in saturation divers would also improve our overall understanding of them and give rise to new ideas for more effective treatments that would benefit the whole diving community and perhaps save many lives.

Chapter 11 – Conclusion

Saturation diving is a dangerous occupation and has a number of physiological effects which contribute to the reasons why it is such a hazardous profession. In recent years the safety and the understanding of the physiology of the disorders has improved greatly although there is still a way to go to fully understand the physiological mechanisms of action of some the conditions. As has been described the pathophysiology of saturation diving is similar across all of the body's systems because the stimulus which causes the diseases to occur is very similar. However the pathways that lead to the symptoms observed are still poorly understood and with the contraction of the main employer of saturation divers, the oil industry, the research money to look deeper into these pathways has started to dry up leaving many unanswered questions. Overall in the 60 years since the advent of saturation diving there have been many advances in both the technology used and the safety procedures developed as a result of the research that was carried out. In the last 20 years the number of new discoveries and increases in our understanding of saturation diving has decreased as the research becomes more expensive and the risks unacceptable. In a large percentage of the experiments carried out there were occurrences of fatalities or serious injuries, which would not be tolerated in the modern research environment.

The overall way in which the body is affected by saturation diving in the short term is the most understood section of the research that has been undertaken because of the duration of time required for the studies to be done. The way in which these affects work on the body is also fairly well defined and we have a good understanding of how to minimise the risks associated with these disorders. As a result the safety legislation brought in, in response to the research has greatly improved the overall safety of the industry as well as decreasing the number of incidents that have occurred.

However the long term pathophysiology of saturation divers has yet to be fully defined because the advent of commercial scale saturation diving has only been around for the last 35-40 years and so the full consequences of a career in saturation diving are yet to be fully researched because of the lack of suitable candidates. The duration since the end of their careers is too short for an accurate long term study of their health and any problems related to their career as a saturation diver to be analysed. As a result of this the pathophysiology of saturation diving and the debilitating effects that may occur in later life are still unknown. This means that in the near future more research is required to collate the data that can be gathered from the first generation of saturation divers to attempt to understand the long term pathophysiology of saturation diving.

The pathophysiology of saturation diving is known to show different effects throughout the human body (and similar ones in animals). These show similar pathologies when examined despite the differing nature of the tissues because of the similar cause of the problems – pressure and gaseous bubbles. The major effects of saturation diving have been researched in much more detail than the minor effects because of the more serious consequences encountered by divers if they have problems with these systems. There is, therefore, a good deal of understanding of the pathophysiology of saturation diving (and diving in general) on the neurological, cardiopulmonary systems. This data was created from experiments done over the last century on the mainly short term effects of pressure and different gaseous mixtures on both humans and animals. Through examining the physical manifestations of the conditions as well as the pathologies of the tissues from cadavers from divers who died from the conditions, the pathophysiology of the short term effects of saturation diving have been fairly well described. These effects are known to be reversible and improve on the cessation of saturation diving. They mainly affect the tissue directly due to the interaction between the gaseous breathing gases and the aqueous nature of the tissues which leads to the body tissues absorbing a proportion of the

gas. This absorbed gas is the cause of almost all the problems and conditions observed in both saturation divers and recreational divers. The pathophysiology in the short term of saturation diving conditions is the damage to the tissues of the lungs, blood vessels, bone and muscle (less frequent), reproductive and endocrine organs (infrequently), nerves and the brain (if very serious) along with the activation of the immune systems inflammatory response which can make the conditions more serious. The immune systems reaction to the conditions is documented with inflammation present in most of the conditions and pro-inflammatory substances known to be released to enhance the tackling of the perceived foreign body (gaseous bubbles). In the short term this results in increased damage to the tissues and if the cause of the inflammation is not removed, chronic inflammation can occur. In the long term this can lead to a decreased quality of life due to the subsequent pain caused by the inflammation although due to the limited research into the long term consequences of saturation diving there is no definite pathophysiology of the conditions in the long term.

The future for saturation diving depends a lot on the understanding of the pathophysiology of the conditions related to attempt to further reduce the chances of these occurring during a dive. Research into this field is aiming to discover a way to reduce the duration of time required to be spent in saturation. This would help operations by reducing the costs, because keeping divers in saturation is expensive and with use of cheaper alternatives on the horizon such as ROVs and ADSs. These two cheaper alternatives are receiving more funding than research into new and more effective saturation diving techniques. However there is still the requirement in some instances where there needs to be hands on attention at the work site which is where the dexterity that is available with saturation divers comes into use. For this reason a greater understanding of the pathophysiology of saturation diving than we currently have would further reduce the unknowns that still exist and reduce the possible long term consequences of saturation diving

career. This greater understanding will also further increase the safety of the environment in which the divers have to work in as well as allowing for better treatment programs if problems and illness are encountered.

In conclusion the pathophysiology of saturation diving is, at least in the short term effects of the conditions, well defined but more research is required to define the pathophysiology of the long term effects. Looking in depth at the methods of action in the short term will help to see if there are any possible methods to reduce the risks to divers and improve any possible treatments both current and future. Any future research (and the research that has already been done) also has an effect on the recreational diving industry so whilst the requirement for saturation divers is decreasing there has been a massive increase in the number of recreational divers. The requirement for up to date knowledge of the pathophysiology and effects of diving conditions is still required despite the slight decline in the commercial diving industry of which saturation diving is a part.

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Acknowledgements

I would like to thank the following people for giving me some extra information and assistance in producing this dissertation on top of what I researched from academic papers and books.

Mr David Smith, Managing Director of the National Hyperbaric Centre in Aberdeen for providing assistance on information of the projects that are being researched for future techniques and some of the details of the timescales of saturation divers in the North Sea. I would also like to thank him for allowing me to visit the facility he runs in Aberdeen to take pictures of the equipment for use in this thesis.

http://www.nationalhyperbariccentre.com

Appendix I: Glossary of Terms

• Arterial Gas Embolism (AGE)	Artery blockage caused by gas bubbles in the systemic circulation.
• Atmospheric Dive Suit (ADS)	Armoured suit similar to a single man submersible that keeps the diver at the same pressure as the surface avoiding the need for a lengthy time in a decompression chamber between dives.
• Bar	The units given to atmospheric pressure. This increases by 1 unit for every 10m descent underwater. 0m – 1 bar, 10m – 2 bar etc.
• Bottom Time	The length of time spent submerged underwater before returning to the surface from a dive.
• Carbon Monoxide	CO. Gas, highly toxic to the body. Can be present as a contaminant in breathing gas and also produced from incomplete combustion (respiration).
• Comex	French Commercial Dive Company that pioneered deep saturation diving and also produced decompression tables similar to RN/USN.
• Compressed Air	The most commonly used breathing gas in recreational diving. Made up of normal atmospheric air (21% Oxygen and 79% Nitrogen) that is compressed into an air tank carried on the divers back.
• Dive Bell	An open or closed chamber that the diver enters to be lowered to the work site.
• Decompression Chamber	The chamber saturation divers enter to compress (pressurise) to the work site depth and are ultimately recompressed to atmospheric pressure at the end of the dive. The chamber is kept at the same pressure (depth) as the work site throughout the dive and is where the divers live for the duration of the dive. The chamber normally stays on the surface support facility. It is also used in the treatment of diving related disorders such as Decompression Illness.
• Decompression Illness (DCI)	Collective name given to the two main diving related disorders: decompression sickness (DCS) and arterial gas embolism (AGE).
• Decompression Sickness (DCS)	The name given to the effects of bubbles in the body expanding due, generally to a rapid ascent and causing problems because they are unable to escape from the body through the lungs.

- Diving Support Vessel (DSV) — A ship that contains all the equipment required to operate a saturation diving operation. These include a decompression chamber, moon pool, thrusters, breathing gas tanks and a crane for raising and lowering the dive bell.
- Dysbaric Osteonecrosis — DON. Necrosis (death) of bone and bone marrow caused by exposure to a high pressure environment.
- Heliox — A mixture of Helium and Oxygen of varying percentages used in saturation diving. Allows diving to depths greater than 300m (1000ft).
- High Pressure Nervous Syndrome (HPNS) — A condition thought to be caused by pressure that occurs at depths greater than 120m and is characterised by tremors and loss of motor control.
- Hyperoxia — Excessive oxygen (oxygen poisoning). Occurs at partial pressure greater than 1.6 bar in the short term and 0.5 bar in the long term.
- Hyperoxic — A gas mixture that contains more oxygen than is present in air. Commonly used in the treatment of Decompression Illnesses → e.g. the use of 100% oxygen.
- Hypoxia — Lack of oxygen (oxygen starvation). Occurs at partial pressure lower than 0.15 bar.
- Marbling — An itchy rash or bruise that is caused by Type I DCS through the bursting of capillaries by the expansion of bubbles in the skin capillary beds.
- Moon Pool — An opening in the bottom of a ship's hull which allows for access to the water in order to raise or lower equipment such as dive bells.
- No Decompression Limits (NDL's) — Tables created by the US Navy which state the maximum length of time under pressure for a SCUBA Diver and the length of time needed in between dives to lower your nitrogen saturation levels
- Nitrogen Narcosis — A condition that affects divers as the partial pressure of nitrogen increases as the depth increases. On air the effects are first observed below ~27m (88ft). Results in similar symptoms to being inebriated. All symptoms are relieved on ascent to a shallower depth.

- Nitrox — An enriched air mix in which the percentage of oxygen can range from between 22% and 99% with the remainder being Nitrogen. Depths achieved are similar to air (depending on mix). In recreational diving the mixes used increase the amount of oxygen in the mix and allow for longer bottom times but at a lower depth.
- Paraesthesia — Burning or prickling sensation that is usually felt in the hands, arms, legs, or feet, but can also occur in other parts of the body.
- ROV — Remotely Operated Underwater Vehicle. Unmanned submersible that can perform tasks without the need for a diver to be on site.
- Partial Pressure (*P*) — The pressure of a gas at a known temperature and pressure when in a mix of gases.
- Royal Navy (RN) — Navy of the United Kingdom. Pioneers of diving and mixed gas diving. Created some of the treatment tables still used today.
- Saturation — When the body tissues can no longer dissolve any more gas within them. Occurs after around 12 hours in humans.
- SCUBA — Self Contained Underwater Breathing Apparatus. Most widely used form of breathing apparatus recreational diving. Maximum recommended safe depth whilst breathing air is 40m (130ft) according to both PADI (http://www.padi.com) and BSAC (http://www.bsac.com).
- Treatment Table — Tables designed by the Royal Navy, United States Navy and some commercial companies such as Comex. Used to treat decompression illness in a decompression chamber.
- Trimix — A mixture of Helium, Nitrogen and Oxygen of varying percentages which is becoming more prevalent in saturation diving (and recreational diving). Allows diving to similar depths as Heliox but with less chance of HPNS – offset with slight nitrogen narcosis.
- United States Navy (USN) — Navy of the United States. Pioneers of diving and mixed gas diving. Created some of the treatment tables still used today.

Appendix II – Henry's Law

This is important for saturation divers because it is possible to predict the rough amount of gas that will enter the body tissues in solution during a saturation dive. It takes around 24 hours for the body to be completely saturated with gas (normally Nitrogen, Helium has a much lower solubility constant in body tissue than Nitrogen although tissue saturation still occurs).

"When a gas is in contact with the surface of a liquid, the amount of the gas which will go into solution is proportional to the partial pressure of that gas. A simple rationale for Henry's law is that if the partial pressure of a gas is twice as high, then on the average twice as many molecules will hit the liquid surface in a given time interval, and on the average twice as many will be captured and go into solution. For a gas mixture, Henry's law helps to predict the amount of each gas which will go into solution, but different gases have different solubility's and this also affects the rate. The constant of proportionality in Henry's law must take this into account. For example, in the gas exchange processes in respiration, the solubility of carbon dioxide is about 22 times that of oxygen when they are in contact with the plasma of the human body."

http://hyperphysics.phy-astr.gsu.edu/hbase/Kinetic/henry.html Astrophysics Department, Georgia State University. [Accessed 26/11/08]

Appendix III: Table of Narcotic Potency

This table displays the effect that various inert gases have in terms of the narcotic properties under pressure. These all are relative to Nitrogen which has a narcotic potency of 1. Table adapted from Brubakk *et al*, 2003 with costs from Web Reference 2 where available in US Dollars (Finnish price in markka converted to $). The various uses come from the thesis with some additions from Web Reference 2 for the more exotic gases (Ne/Ar/Kr/Xe).

Gas	Molecular Weight	Solubility in Lipid	Relative Narcotic Potency	Cost (10 litres at 200 bar ($))	Uses
Helium (He)	4	0.015	0.2 (least)	~50-100	Heliox/Trimix Deep Diving
Neon (Ne)	20	0.019	0.3	~2000	Neox ($Ne:O_2$)/Neoquad (Ne:Trimix) Deep Diving (rare because of costs)
Hydrogen (H_2)	2	0.036	0.6	Unknown	Hydrox ($H_2:O_2$)/Hydreliox ($O_2:H_2$:He). Deep Diving (relatively rarely)
Nitrogen (N_2)	28	0.067	1	~5-15	Air/Nitrox/Enriched Air/Trimix. Shallow diving/recreational diving
Argon (Ar)	40	0.140	2.3	~10-30	Argox ($Ar:O_2$) - shallow in water decompression stops/ dry suit gas (Ar)
Krypton (Kr)	83.7	0.430	2.5	~8000	Useless for Diving (too Narcotic)
Xenon (Xe)	131.3	1.700	25.6 (most)	~4500	Useless for Diving (too Narcotic)
Oxygen (O_2)	32	0.110	1.7	~8-10	Treatments (100%) + diving (lower %). Essential for survival.
Carbon Dioxide (CO_2)	44	1.340	20.0	N/A	Expelled breathing gas.