THE CONTRIBUTION OF ELEVATED PERIPHERAL TISSUE TEMPERATURE TO VENOUS GAS EMBOLI (VGE) FORMATION

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Abstract

This purpose of this study was to evaluate the contribution of post-dive peripheral tissue warming to the production of venous gas emboli (VGE) in divers.

Inert gas elimination from the tissues is limited by both perfusion and diffusion. If changes in diffusion are matched by corresponding perfusion (vasoactive) changes, decompression should be asymptomatic (within allowable exposure limits). Under conditions when the diffusion of inert gas from the tissues is not matched by blood perfusion, VGE will ensue. Increasing tissue temperature will decrease inert gas solubility and thus diffusion into the blood. It has been demonstrated that problems may arise during rapid changes in peripheral temperature, as often occurs post-dive, when divers previously exposed to cold water actively rewarm themselves in showers or baths. The effect of moderate rewarming, however, may be to increase the rate of inert gas elimination without the formation of VGE since increased perfusion is encouraged. The effect of mild post-dive warming was investigated.

Ten male subjects, between the ages of 21 and 29 years

completed two dry chamber dives to 70 feet for 35 minutes (no decompression limit of the Canadian Forces Air Diving tables). Each dive was followed by a 30 minute head-out immersion in either a thermoneutral (28°C) or warm (38°C) bath. Non-invasive Doppler ultrasonic monitoring was then carried out at 30 minute intervals for the next 150 minutes to assess measurable VGE. Subjects did not display VGE formation in either the control or experimental conditions.

Our findings suggest that: 1) the Canadian Forces table limits (for the profile employed) provide safe no-decompression limits not compromised by mild post-dive warming, and 2) mild peripheral warming, since not bubble generating, may be a useful adjunctive therapy in the management of decompression sickness by increasing the rate of inert gas elimination.

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I. Review of Literature

Decompression Sickness

The effect of hyperbaric exposure is complex. A debilitating condition that can arise from the decompression following exposure is decompression sickness (DCS). In fact, it is not a single condition, but a diverse syndrome, the expression of which is variable and capricious. To understand the action of DCS, physical properties of aquatic and hyperbaric environments and human physiology and biophysical parameters must be understood as they interact with each other. The amount of attention given to this is great, our understanding somewhat less so. A number of excellent reviews have been completed on the physiology of diving (Lin, 1988), decompression theory (Vann, 1982; Hempleman, 1984), decompression procedures (Hempleman, 1982), pathogenesis and manifestation of decompression disorders (Hills, 1977; Hallenbeck and Andersen, 1982; Elliott and Kindwall, 1982), and the treatment of DCS (Davis and Elliott, 1982; Farmer et al., 1984). The goal of the present introduction is not to supersede these works but to provide background pertinent to the thesis project undertaken.

Historical Perspectives

The first clinical observation of decompression induced gas bubbles is attributed to Robert Boyle (1670) and the first experimental evidence implicating gas bubbles in the etiology of

DCS to Paul Bert (1878).

Generally attributed to the efforts of J. S. Haldane, Boycott et al. (1908) suggested that tissues may be supersaturated to a limited degree before symptomatic DCS would develop. Experimenting with goats, they observed that if the ambient pressure was reduced to no less than one half the saturation pressure, symptoms would not arise. They identified a "critical supersaturation ratio" of 1.58 to 1. Ostensibly, the body fluids and tissues could tolerate 1.58 times the nitrogen pressure within them than the absolute pressure on them without complication. This finding formed the foundation upon which Haldane's decompression schedules (tables) were constructed.

Haldane's decompression schedules (Boycott et al., 1908) portray a somewhat arbitrary attempt to produce an nitrogen uptake-elimination model representative of the full range of bodily tissues. Each of five 'compartments' - representing different tissue types - has a corresponding 'half-time'. The half-times indicate the period of time required for a tissue to gain or lose 50% of the available gas pressure. The compartments are thus identified as 5, 10, 20, 40 and 75 minute tissues. Experience has shown, however, that safe supersaturation ratios can not be so easily calculated. Success of Haldane's model has proven to be limited. In fact, significantly less pressure reduction than allowed by these decompression schedules have been shown to result

in DCS (Rivera, 1964; McLeod, 1986).

Another limitation of early decompression theory involves the relationship of developing bubbles to symptoms of DCS. The presumption that the onset of DCS correlates with the development of bubbles has not been born out. Behnke (1942) first suggested that asymptomatic, or 'silent bubbles', could result from decompression events. Although present, they would not necessarily precipitate symptomatic DCS. This was confirmed with the application of Doppler ultrasonic technology to diving (Gillis et al., 1968; Spencer and Campbell, 1968). Circulating bubbles were regularly detected prior to the onset and even in the absence of DCS.

Etiology

It has been generally accepted that physical bubbling is responsible for the development of symptomatic DCS. The physiological effects of a mechanical obstruction in the vascular and/or lymphatic systems lead to tissue ischemia. Long term ischemia can result in permanent injury. This is a reasonable assumption in view of the circumstantial evidence. Nitrogen is five times more soluble in fat than other tissues (Vernon, 1907; Buhlmann, 1984). This results in localized high concentrations of nitrogen in fatty tissues that can present extensive bubble

formation upon decompression. An example of such a site is bone marrow, which becomes progressively more fatty with maturity (Ganong, 1985). Regions of high marrow concentration (such as the long bones) commonly display symptomatic problems.

Recent work has improved the appreciation of biophysical and biological effects associated with bubble formation and DCS. Ethical considerations, however, will continue to restrict the experimental data collected.

Manifestation

A number of classification schemes for the signs and symptoms of DCS can be found in the literature. A bipartite system has been used most extensively (Erde, 1975; Edmonds et al., 1981; Arthur and Margulies, 1987). Signs and symptoms are divided into Type I ('pain only', mild) and Type II ('serious'). Providing more detail, signs and symptoms can be grouped into five categories - four primarily acute and one recognized as chronic:

1) Cutaneous Manifestations. Cutaneous manifestations (or 'skin bends') represent the least severe form of DCS. Included are pruritis (intense itching), lividity (abnormal coloration), mottling, blotching, a variety of rashes, localized swelling, and subcutaneous emphysema (Rivera, 1964). These are considered Type

I manifestations under the bipartite system, although some of the signs (for example, pruritis) are not considered to represent true DCS (Elliott and Hallenbeck, 1975).

2) Pain Only. Pain only manifestations are generally referred to as Type I DCS under all classification schemes. These usually involve pain located intra- and peri-articular (in and around joints) (Kizer, 1984). The pain is typically described as a deep, dull ache, and is often associated with vague numbness (this is not to be confused with neurological paresthesia). Movement usually aggravates the discomfort, and recompression causes rapid resolution. Shoulders, elbows and knees are the joints most commonly affected although the discomfort can be more diffuse.

3) Neurological Disorders. Neurological manifestations are more serious examples of DCS. They are usually referred to as Type II problems, but as such are grouped with other distinct manifestations under the bipartite system. A vast array of signs and symptoms are possible, ranging from the very minor to completely debilitating. The lower thoracic, lumbar, or sacral spinal cord can be affected, producing some degree of paralysis and/or paresthesia (Dick and Massey, 1985). A common complication is urinary retention, in fact, this is a strong diagnostic sign (Kizer, 1983). Although the pathophysiology remains unclear (Douglas and Robinson, 1988), the spinal cord is recognized as remarkably susceptible to acute DCS (Hughes and Eckenhoff, 1986).

Problems are often seen following dives within the conventional exposure limits (Dick and Massey, 1985). The spinal cord may also be predisposed to chronic damage arising from repeated, asymptomatic exposure (Palmer et al., 1987).

Any of the sensory systems also may be affected (Dick and Massey, 1985; Arthur and Margulies, 1987). Signs and symptoms involving the visual system may include blurred vision or nystagmus (uncontrollable, jerky eye movement). Vestibulocochlear compromise can result in dizziness and/or vertigo (extreme dizziness and disorientation), tinnitus (ringing in the ears), or deafness.

Some neurological symptoms may be recognized as global. The best example is extreme fatigue (Strauss and Prockop, 1973). It may or may not be associated with other signs and symptoms of DCS.

4) Pulmonary Manifestations. Pulmonary manifestations (commonly called the 'chokes') result from failing efforts by the pulmonary system to filter circulating bubbles out of the bloodstream. While this filtering is efficient under normal conditions (Emerson et al., 1967; Spencer and Oyama, 1971; Hills and Butler, 1981) edema (fluid accumulation) may develop when more than 10% of the pulmonary vascular bed becomes obstructed by bubbles (Kizer, 1983). Responses includes tachypnea (accelerated heart rate), chest pain, non-productive cough, dyspnea (difficulty breathing), and ultimately signs of circulatory compromise (eg.

distention of neck veins) (Greenstein et al., 1981). Pulmonary signs and symptoms have been considered Type II manifestations under the bipartite system (Arthur and Margulies, 1987). They have also been labelled independently as Type III signs and symptoms (Strauss and Samson, 1986).

5) Dysbaric Osteonecrosis. Dysbaric osteonecrosis represents a chronic DCS disorder, occasionally referred to as a Type IV manifestation (Strauss and Samson, 1986). First identified in caisson workers in 1911 (Bassoe, 1911), it was finally reported in divers in 1941 (Calder, 1982). The problem is one of bone lesions, either on the shaft (medullary, Type B), or associated with joints (juxta-articular, Type A) developing some time after decompression insult. While shaft lesions can remain relatively benign, juxta-articular lesions will progress to a debilitating state. Affliction rates, primarily estimated for the commercial diving population, display a remarkable range, from 1.7% (Sphar et al., 1977) to 76.6% (Ohta and Matsunaga, 1974), with reports citing virtually every point in between (Kawashima, 1976 - 59.5%; Biersner and Hunter, 1983 - 33%; Asahi et al., 1968 - 19%; McCallum et al., 1976 - 2.7%). Detailed reviews can be found elsewhere (McCallum and Harrison, 1982; Walder, 1984).

Factors Affecting Susceptibility

Undoubtedly, the greatest limitation of any set of decompression tables is the inability to assess individual variability in susceptibility to DCS. This is exemplified by the number of cases where only one diver in a pair becomes symptomatic when both were together for the entire dive, or when an individual becomes symptomatic following a profile that he or she had completed any number of times previously without complications. An extensive range of factors, both physiological and environmental, have been found to affect individuals on a day to day and a long term basis. Included are: adiposity, age, dehydration, smoking, restricted local circulation (possibly caused by previous tissue injuries or limb positioning), exceptional exertion, and exceptional thermal exposures. Many other factors have also been proposed as contributing to increased susceptibility. These include gender and the use of birth control pills. Conversely, some factors have been reported to increase resistance to DCS, such as acclimatization, prophylactic use of aspirin, race, and the use of non-standard decompression schedules.

Increased adiposity, usually assessed in terms of percent body fat has been correlated to an increased risk of developing DCS (Gray, 1951; Dembert et al., 1984). Since fatty tissues have a greater capacity to absorb nitrogen and are generally more poorly perfused than other tissues, they are susceptible to the formation

of bubbles and ultimately decompression 'hits', due to their greater loading and relative inability to off load gas during and following decompression. Obesity, defined as being more than 20% above recommended weight, has been suggested as a guideline for restricting participation in commercial diving activity (Linaweaver, 1984; McCallum and Petrie, 1984).

The majority of subjects involved in experiments to determine safe decompression limits usually have optimal health and physiologic function. From a researchers point of view, this reduces the number of potentially confounding variables to be considered. It is often reported that the U.S. Navy decompression schedules are based on the responses of 'young, healthy, male subjects'. Perhaps the critical component of this 'optimality' involves circulatory performance. As individuals move further afield from this optimum the uptake and elimination of gases can be impaired.

Increasing age is correlated with impaired circulatory patterns. This may be due to progressive arteriosclerosis. Regardless of the mechanism, advancing age does increase individual predisposition to DCS (Szasz, 1982; Becker and Parell, 1983). Gray (1951) demonstrated a roughly linear increase between age and susceptibility. Few experimental data are available to confirm this relationship, but it is generally recommended that with increasing age, decompression safety will be improved if additional

safety factors are calculated into all dive planning, and that this should contraindicate involvement in commercial types of exposure (Linaweaver, 1984). A recommendation often promoted within the recreational diving community is that an additional 10 minutes be cut off the no-decompression limits for every decade of life beyond 20 years of age.

Age is only one of several conditions affecting circulation. Over the short term, dehydration results in hemoconcentration which effects the quality of circulation. Insufficient fluid intake can result from inadequate thirst perception (Manjarrez and Birrer, 1983) or from the concern over discomfort arising from increased urination associated with immersion in water (Epstein, 1978; Shiraki, 1987). Alcohol ingestion will also aggravate body fluid loss. Any degree of hemoconcentration will aggravate and be aggravated by the bubble development concomitant with DCS (Hallenbeck et al., 1973; Bove, 1982), thus aggressive replacement of fluids should be an important concern of all divers.

Limb positioning can also have important circulatory affects. While not well documented, disruptions in local circulation may precipitate DCS. Divers wearing wet or dry suits during decompressed in a bell often display pruritis at areas where excessive pressure has been applied by the suit (Igor Mekjavic, personal communication). Similar problems may be expected in individuals suffering local circulatory insufficiencies caused by

scar tissue from previous injuries. It is a common practice during chamber exposures to encourage divers to move around at regular intervals and to avoid positions that may compromise circulation (eg. cross-legged) (Don Hedges, personal communication).

Extreme dives, in terms of excessive exertion or cold can also increase the risk of developing DCS. Exertion will increase circulation, thus enhancing the uptake of inert gas during the compression or bottom phases of a dive. The greater the uptake, the greater the likelihood that decompression will result in supersaturation ratios high enough to cause bubble formation. Moderate exercise during decompression may actually protect against the development of DCS since increased perfusion will enhance inert gas elimination (Vann, unpublished). However, aggressive exertion during decompression or post-decompression exercise may facilitate the onset of symptomatic DCS through cavitation effects of stretching tendons/muscles or simple agitation encouraging bubble evolution (Brown, 1979). A final concern associated with exertion involves the role of increased CO_2 levels. Exercise will enhance the CO_2 retention already associated with hyperbaric exposure (Anthonisen et al., 1976; Dwyer et al., 1977; Van Liew and Sponholtz, 1981). Although the role, and certainly the mechanism, remain unclear (Hickey et al., 1983), CO_2 retention would appear to encourage DCS (Bell et al., 1986).

Extremely cold diving exposures complicate pressure effects

as well. Dunford and Hayward (1981) suggest that cold exposures result in a rapid decrease in peripheral circulation which will lead to decreased inert gas uptake. They compared subjects wearing well insulating dry-suits with those wearing poorly insulating 1/8 inch wet suits in 10°C water. Results from their study confirmed this effect under the conditions they created. However, it is expected in reality that most divers will wear reasonably appropriate exposure equipment for the situations they expect to encounter and will therefore start their dives warm and in a near optimal circulatory state. This ensures a fairly rapid uptake of inert gas through at least the early compressive phase, which is traditionally the deepest part of the dive. As the cold starts to take affect, however, peripheral circulation is decreased, which subsequently reduces the peripheral tissue's ability to eliminate gas during and following compression until rewarming is complete. If this post-dive rewarming is carried out too rapidly, the result can be the manifestation of signs and symptoms of DCS in the peripheral tissues. This is demonstrated by post-dive, cold exposed subjects that suffered skin bends following hot showers taken at the end of their experimental trials (Mekjavic and Kakitsuba, unpublished). Unfortunately, despite general recommendation to avoid hot post-dive showers (Mebane and Dick, 1985), experimental evidence has not appeared to refute or confirm these postulations.

Smoking is implicated in predisposing, and perhaps most

importantly, in complicating the effects of DCS. It has been suggested to increase platelet adhesiveness and will increase the likelihood of developing gas trapping diseases like emphysema and bronchitis (Shilling, 1984). This may result in increased clotting activity in response to a given decompression insult. While the standard treatment of recompression will mitigate the effects of physical bubbles, it will not effect existing blood clots. Subsequently, possibly complicated adjunctive treatment becomes necessary.

Prior incidence with DCS is another consideration in determining predisposition. Many of the individuals having suffered from DCS figure in repeat cases (Golding et al., 1960). This would appear to indicate a pattern for personal susceptibility.

Many authors have reported gender as a predisposing factor, with females the more susceptible (Bassett, 1973; Bangasser, 1979). A preliminary report based on female involvement in military diving has recently challenged this position (Zwingelberg et al., 1987). Statistical review of this report, however, has indicated that the data accumulated to date are inadequate to allow valid comparison between male and female counterparts (Robinson, 1988).

Use of birth control pills has also been suggested to predispose to or aggravate DCS. They have been shown to increase

platelet adhesiveness and potentiate blood clot development (Kizer, 1981a). As with smoking, this could complicate any case of DCS. However, because of the number of women employing them with apparent impunity and the lack of clinical evidence, their use is not contraindicated at this time (Fife, 1984).

Countering the predisposing factors are those suggested to confer an increased resistance against DCS. Acclimatization is one reported to have a dramatic effect. Golding et al. (1960) found that the rate of DCS could fall to 0.1% of the first day incidence after two to three weeks of daily exposure in caisson workers. This benefit was rapidly lost when daily exposure ceased. They estimated that only half of the full protection was present after seven days. The differences in exposures between caisson workers and divers may make this effect inapplicable to the diver. In fact, recent analysis of the Divers Alert Network (DAN) accident files for North America found that multi-day and repetitive diving is involved in 55% and 64%, respectively of the reported cases of DCS (DAN, unpublished).

Aspirin has been proposed to provide a prophylactic benefit (when administered in combination with other antiplatelet drugs) by decreasing platelet activity and blood viscosity (Catron and Flynn, 1982). No clinical or experimental trials have confirmed the benefits of aspirin alone. Reliance on this type of proposed safeguard has obviously not been encouraged.

Racial trends have been observed in relation to DCS susceptibility (Kawashima, 1976; Wade et al., 1978). While unconfirmed at present, these likely represent acclimatization (Hills, 1977), rather than a race effect.

The use of alternative decompression schedules (as compared to the standard U.S. Navy tables) may increase safety. Significant controversy exists as to optimal decompression stop depths. British procedures employ significantly deeper stop depths than the U.S. (Edmonds et al., 1981). Supporting the British approach it has been demonstrated that adding deeper stops to those outlined in the U.S. tables will decrease bubble formation in divers (Neuman et al., 1976). The Canadian Forces Air Diving Tables and Procedures (1986), developed at the Defense and Civil Institute of Environmental Medicine (DCIEM) rely on the same decompression stop depths as the U.S. Navy, but are significantly more conservative in terms of allowable exposure time. Either approach is expected to confer a greater degree of protection on the divers.

The factors discussed above, predisposing or protective, can not be easily controlled. Therefore, it is clear that a single set of tables will not be satisfactory for all individuals. For this reason, procedures focus on minimizing apparent risk, and managing the result when problems develop.

Decompression Sickness Therapy

Recompression

Definitive management of DCS relies on recompression therapy. Although open water recompression has been proposed to meet this end in the absence of recompression facilities (Edmonds et al., 1981; Canadian Forces Air Diving Tables, 1986), chamber treatments are generally considered to be the only satisfactory alternative due to the treatment time required and the potential for complications that can be ill handled under less controlled underwater conditions (Boettger, 1983; Mebane and Dick, 1985).

Specific treatment will vary based on the facility, equipment and personnel available. Recompression can be approached in one of three ways: 1) to a pressure (depth) dependent upon the depth and duration of the original dive, or 2) to a predetermined fixed depth (ie. according to standard tables of recompression therapy), or 3) to a depth which produces a clinically acceptable result.

While the third option is often employed in Australia (Edmonds et al., 1981) the classical approach in North America is to follow (and often modify) the established U.S. Navy derived procedures that rely on the second option (U.S. Navy Handbook, 1979). U.S. Navy Table 6 (employing oxygen) calls for an initial treatment depth of 2.8 ATA (60 fsw), After a given period of time at this

depth, the subject is decompressed to 1.4 ATA (30 fsw) and spends a similar period of time prior to returning to surface ambient pressure. A review of treatments has demonstrated that while rare cases displaying dramatic deterioration at the maximum depth of this protocol may profit from exposure to 6.0 ATA (165 fsw), the majority of cases would not benefit (Leitch and Green, 1985).

There are three benefits of recompression. First, existing bubbles are physically decreased in size. Further, bubble reabsorption is promoted. Finally, further bubble evolution is prevented (Kunkle and Beckman, 1983). Even if recompression must be delayed, benefit from treatment is gained (Rivera, 1964; Kizer, 1982).

Adjunctive Therapies

While recompression will remain the mainstay treatment for DCS, adjunctive therapies can be crucial. Secondary bubble effects, with a general theme of inflammatory responses, can complicate cases by aggravating the already significant hematologic effects resulting from hyperbaric exposure (Philp et al., 1975; Diercks and Eisman, 1977; Andersen et al., 1981; Neuman et al., 1981). These will most likely to be seen when recompression is delayed more than 8 to 10 minutes (Bove, 1982).

A number of adjunctive therapies have been proposed to accompany recompression. Oxygen therapy is the most accepted of these. It is used as an interim measure to mitigate or completely relieve signs and symptoms during transport and prior to recompression. In this capacity it is proving to have long range benefits. While data are limited, it is suggested that aggressive oxygen therapy prior to recompression can lead to faster and more complete resolution of the condition with less permanent damage (DAN, unpublished). Use of oxygen breathing in conjunction with recompression therapy is also beneficial. In fact, it is the norm of present treatments (Berghage and McCracken, 1979; Myers and Schnitzer, 1984). The U.S. Navy Table 6 recompression protocol previously outlined relies on oxygen breathing. The following four benefits are gained from oxygen breathing: 1) the gradient for nitrogen elimination at the alveolar-capillary interface is maximized; 2) the potential for adequate oxygen diffusion in lungs with reduced function resulting from edema is increased; 3) the dissolved oxygen content in the plasma is increased resulting in increased tissue oxygenation (Boerema et al. (1960) demonstrated that with all their hemoglobin removed, swine still received adequate oxygenation through dissolved transport to survive when breathing 100% oxygen at three atmospheres pressure); and 4) high partial pressures of oxygen will cause a reflexive vasoconstriction (Bird and Tefler, 1965), this resulting in reduced swelling and edema in insulted tissues. While a toxic response to oxygen breathing is possible (Butler and Thalmann, 1986), this can usually

be controlled with periodic 'air breaks'. Table 6 calls for five minutes of air breathing following every 20 minutes of oxygen breathing. Confirming expectations, recent investigation has demonstrated that oxygen should not create a risk in encouraging DCS through its application (Weathersby et al., 1987).

Different breathing media are presently being assessed for their effectiveness in treating DCS. A preliminary animal study has shown that the addition of perfluorocarbon emulsions (to enhance the solubilities of both oxygen and nitrogen) to a 100% oxygen medium will increase both hemodynamic and neurologic protection from DCS (Spiess et al., 1988). Further data must be collected regarding this technique. Exemplifying the complicated nature of treatment, reports reviewing helium-oxygen combinations in treating DCS have ranged from very positive (Douglas and Robinson, 1988) to very negative (Catron et al., 1987; Lillo et al., 1988).

Other adjunctive aids recommended include: blood volume expanders and fluid administration (Cockett and Nakamura, 1964; Wells et al., 1978; Merton et al., 1983), antiplatelet drugs (Whitcraft and Karas, 1976; Philp et al., 1979), antiedematous drugs such as corticosteroids (Pauley and Cockett, 1970; Kizer, 1981b; Jaffe, 1986), hypothermia (Erde, 1963; Simmons et al., 1982), anticonvulsants (Cox, 1980), antiarrhythmics (Kizer, 1980), and anticoagulants (Cockett et al., 1970; Reeves and Workman,

1971). The limited data available provide generally inconclusive or conflicting results (Catron and Flynn, 1982). Although warranting further investigation, the general approach to adjunctive therapies other than oxygen (U.S. Navy Handbook, 1979; Mebane and Dick, 1985) and fluid administration (Cockett et al., 1965; Heimbecker et al., 1968; Strauss and Samson, 1986) is very cautious (Bayne, 1978; Greene and Lambertsen, 1980).

Conclusions

Exposure to the stress of decompression is not benign. The interaction of a number of factors may result in development of DCS. Predisposing factors exist which alter individual risk, and the limitations of present understanding demands that a cautious approach be taken at all levels of involvement. Further investigation is required to overcome these limitations.

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Doppler Ultrasound

Doppler ultrasonic assessment has become a common component of diving research. This is not to imply that the methodology is universally accepted. Controversy remains regarding the techniques. This review will present background on the development of ultrasonic technology and techniques pertinent to the diving researcher. Technical reviews of ultrasonic theory and equipment are not included in this paper. They have been completed by Kalmus (1954), Wells (1969, 1977{a & b}), Rubissow and MacKay (1971), Nishi (1972), Spencer and Clarke (1972), Spencer and Johanson (1974), Kisman (1977), and Eatock et al. (1985).

Referring to the diving literature related to ultrasonic evaluation one sees a variety of nomenclature. 'Bubbles' are equivalent to 'vascular gas emboli' or VGE, and bubble formation to 'gas phase separation'. The terminology used in this review will be standardized, unless the introduction of secondary descriptors benefits the discussion.

The Doppler ultrasonic flowmeter was first reviewed by Kalmus (1954). Baldes et al. (1957) used this technology to measure blood flow velocity. Subsequently, Franklin et al. (1961) observed that circulating bubbles could be detected during the evaluation of blood flow. In fact, bubbles will produce a much stronger reflected signal than similar sized blood cells.

The first report that ultrasound could be used to identify bubbles caused by decompression sickness was made by Mackay (1963). He detected bubbles in a rat that had been rapidly decompressed following a severe hyperbaric exposure. Gillis et al. (1968a) demonstrated that surgically implanted Doppler ultrasonic blood flowmeters could detect circulating bubbles in decompressing swine before they reached the first decompression stop recommended by the U.S. Navy exposure tables. Spencer and Campbell (1968) reported similar results with sheep.

Both Gillis et al. (1968a) and Spencer and Campbell (1968) suggested that further efforts with ultrasonic technology would allow it to be used successfully with human subjects. This was encouraged by the development of transcutaneous transducers that allowed for uncomplicated and non-invasive monitoring of subjects (Gillis et al., 1968b).

As a result of the early success and enthusiasm apparent in the literature and the new ability to apply it non-invasively, Doppler ultrasonic analysis received extensive use and review. A more dramatic range of results were found through the subsequent investigations.

Risks Associated With Doppler Ultrasound

The risk factors of human exposure to ultrasound have long been recognized. The energy levels employed by some ultrasonic systems have been shown to cause cavitation and production of physical bubbles. In fact, the intensities used in physiotherapy are reportedly above cavitation levels (Smith and Spencer, 1970). Most of the early investigators applying ultrasonic technology to the evaluation of decompression derived bubbles expressed their awareness of this concern (Gillis et al., 1969; Spencer et al., 1969; Smith and Spencer, 1970; Spencer and Clarke, 1972; Rubissow and Mackay, 1974; Hills and Grulke, 1975; Spencer, 1976; Daniels et al., 1979).

Gillis et al. (1969), recognizing the hazards of ultrasonic cavitation, recommended that investigators limit the power of the units to be employed. Smith and Spencer (1970) then reported that intensity levels required to produce cavitation were 100 $W\text{-}cm^{-2}$ in blood and variable in tissue with the minimum intensity being 1.3 to 1.6 $W\text{-}cm^{-2}$ in brain tissue. Their ultrasound system applied power levels of approximately 0.01 $W\text{-}cm^{-2}$, as they express it, "two magnitudes below reported cavitation thresholds for tissue and four magnitudes below water or blood". Supported by the lack of complications, they concluded that the working equipment and techniques did not present a significant risk to subjects.

Spencer et al. (1969) experimentally tested the impact during the first trials using human subjects. They alternately started and stopped an upstream detector to evaluate any changes noted at a downstream detector. They found no changes resulting from this action.

Subsequent investigators have generally accepted that negligible risk is associated with the power levels required by these ultrasonic techniques (Rubissow and Mackay, 1974; Spencer, 1976; Daniels et al., 1979).

Doppler Protocol Development

Early investigations did not lend themselves to cross-correlation or replication. Researchers generally used subjective and often undescribable bubble evaluation schemes. Gillis et al. (1968b) reported that characteristic bubble sounds could be best described as "chirps". Evans and Walder (1970) categorized them as "plops" - these being "not so high pitched as the chirps". Powell's (1974) scoring procedure was described as follows: 0 = normal heart sounds free of bubbles; 1 = "running water" sounds; 2 = "popping" sounds; 3 = "roaring" sounds. Hills and Grulke (1975) classified bubbles as those sounds best described as "blups" or "chirps". To this point, the evaluation of recorded signals was not highly satisfactory.

Dramatic improvements in standardization of signal interpretation were realized when Spencer and Johanson (1974) developed a simple but more quantifiable grading system. They classified bubble sounds as follows:

Grade 0 = No bubble signals.

Grade I = An occasional bubble signal. The great majority of cardiac cycles are free of bubble signals.

Grade II = Many but less than half of the cardiac cycles contain bubble signals.

Grade III= All of the cardiac cycles contain bubble signals, but not obscuring signals of cardiac motion.

Grade IV = Bubble signals sounding continuously throughout systole and diastole and obscuring normal cardiac signals.

Spencer and Johanson's (1974) system was then adopted as the standard for many subsequent investigations (Spencer, 1976; Neuman et al., 1976; Spencer, 1978; Powell et al., 1983; Daniels, 1984; Huggins, 1984; Bayne et al., 1985; Eckenhoff et al., 1986).

While the reliability of Spencer and Johanson's (1974) grading system was applauded, further factors had to be considered. Manley

(1969) first observed that muscular movement resulted in a marked increase in sensible bubbles. Spencer and Clarke (1972) noted that bodily movements considerably augmented the signals measured, at both precordial and basilic vein measuring sites. These movements included arm raising, fist clenching and passive compression of the forearm. There was then no doubt that bubble grades during movement were always higher than bubble grades while at rest (Gardette, 1979; Eckenhoff et al., 1986).

Refinements to the evaluation procedures included comparison of 'at rest' and 'movement' (ie. limb flexing) conditions (Kisman et al., 1978). This was felt to enhance the detection of early bubble development.

Further experience with the Kisman-Masurel scale led to the adapted procedures presented in detail by Eatock and Nishi (1986). They maintained the Grade 0-IV scale, evaluated during rest and movement cases, and quantified bubbles based on three parameters: 1) frequency - number of bubbles per cardiac period, 2) percentage/duration - percentage of cardiac periods with specified bubble frequency, and 3) amplitude - bubble sounds in comparison to normal cardiac sounds. This methodology provides the most current approach available in the literature.

Signal Interpretation

The Spencer and Johanson (1974) scale discussed previously provides the common reference point for Doppler signal interpretation. The most straightforward aspect of the interpretation involves bubble grades. It is generally accepted that the bubble grades of III and IV are more strongly correlated with the development of clinical signs and symptoms of decompression sickness (DCS) than the lower grades (Gardette, 1979; Daniels, 1984; Strauss and Samson, 1986; Webb et al., 1988).

VGE development is affected by inter- and intra-individual variability. Both aspects demand further investigation. Inter-individual susceptibility has been considered by Spencer (1976). He concluded that individuals who were "bubble prone" would be "bends prone". If so, Doppler assessments could be used to pre-screen divers prior to regular participation. This was subsequently attempted by Spencer and Aggenbach (1978). They completed 31 chamber exposures to 165 feet for 10 minutes followed by direct ascent to the surface (ascent rate standard 60 feet per minute). Six subjects then completed equivalent dives in open water. VGE scores were notably higher following the open water exposures, and most importantly, trends towards individual susceptibility following open water dives could be estimated from chamber scores, this supporting the findings of Spencer and Johanson (1974).

The reliability of correlating individual scores to susceptibility has not been verified by other investigators. Powell et al. (1983) reviewed 150 man-dives with exposures ranging from 100-220 meters in depth and between 15-60 minutes in bottom time. They contradict Spencer's (1976) conclusion regarding proneness and report extreme inter-individual variation with some "bubble prone" individuals being "not bends prone" and some "bends prone" individuals being "not bubble prone". This seems a doubtful challenge since measurable bubble presence appears to be a prerequisite of decompression sickness (Daniels, 1984) and that increasing bubble grades, as reported earlier, correlate most strongly.

Intra-individual variability has been considered by Eckenhoff et al. (1986). They used Doppler ultrasound to quantify bubbles in 34 subjects completing air saturation dives for 48 hours at 1.77 atmospheres absolute (ATA) (25.5 fsw, n = 19), and at 1.89 ATA (29.5 fsw, n = 15). Decompression back to 1 ATA following these exposures was completed in approximately two minutes. They found four cases of DCS (27%) and three "possible DCS" (20%) following 29.5 fsw exposure, and five "possible" (26%) at 25.5 fsw. Their data support the findings of Spencer (1976), that there is significant inter-individual variability in susceptibility to decompression sickness. They suggest that the length of time venous bubbles are present may be more representative of decompression stress than the peak score. Once more, no

corroborating reports have been found in the literature.

Experimental Results Using Doppler Techniques

The use of ultrasonic bubble detection to evaluate human subjects after diving was first reported by Gillis et al. (1969) and Spencer et al. (1969). Neither of these studies reported major success with the techniques. Gillis et al. (1969) dived one individual to 175 feet for 60 minutes (decompression according to U.S. Navy tables). No bubbles were identified despite the fact that this subject became symptomatic. This was felt to be a result of inadequate training of the subject/technician in positioning the transducer and a subsequent poor quality signal. They dived two other subjects to 60 feet for 45 minutes. Again, no bubbles were identified. The mildness of this exposure made the lack of identifiable bubble signals understandable.

Spencer et al. (1969) reported on seven exposures ranging from 100 feet for 80 minutes to 200 feet for 60 minutes (all decompressions according to U.S. Navy tables). Although three of their subjects became symptomatic, they observed only "possible" bubble signals in two of them (none of the other displayed measurable bubbles). This was suggested to result from some unsuccessful experimentation with evaluation sites. Lack of experience in interpreting the signals was also reported to be a

factor (Spencer and Clarke, 1972).

Human experiments that were more successful in terms of assessment were completed by Spencer and Clarke (1972). A single male subject was dived to 200 fsw for 30 minutes (decompression according to the U.S. Navy Tables). The resultant bubble signals were clear and extensive. Counting individual bubbles was often impossible. Despite the bubble severity, however, in this case the subject did not become symptomatic.

Spencer (1976) exposed human subjects to a wide range of dry chamber schedules (from 233 fsw for 7 minutes to 25 fsw for 720 minutes) and open water dives to 165 fsw for 10 minutes. Although his statements remain conjectural about specific exposures, he generalized that: 1) there was notable inter- and intra-individual variation in the formation of VGE, correlating with susceptibility to developing DCS; 2) DCS did not develop without the prior detection of precordial VGE; and 3) open ocean exposures increased the percentage of VGE and DCS when compared to chamber exposures.

Neuman et al. (1976) completed two series of dives. The first, to 210 fsw for a 50 minute bottom time. This involved 18 man-dives. The second series involved 16 subjects exposed to 132 fsw for a 30 minute total bottom time (TBT). Again, they achieved encouraging ultrasonic results. They observed: 1) a significant linear increase in bubble scores during the decompression stages

of the dives; 2) a statistically significant bubble score reduction for those divers being exposed to an additional deep decompression stop (not called for in the U.S. Navy exposure tables) that was not solely related to the additional decompression time involved; 3) that there was a statistically significant relationship between bubble scores and decompression sickness. All seven of the divers who experienced decompression sickness had Grade IV bubble scores during their last three recordings, whereas only 12 of the other 27 divers had similar bubble scores; and 4) there were no significant correlations between age, weight, or body composition and the occurrence of either decompression sickness or mean bubble score.

The investigations reported above reflect an early trend of increasing confidence in Doppler ultrasonic results. Unfortunately, the exposures employed in each of the successful examples are the more severe ones. Less conclusive but still supportive results can be seen from the more moderate studies. They confirm the efficacy of Doppler ultrasonic procedures with some qualification. These involve a wide variety of exposures.

Gardette (1979) reviewed Doppler assessments completed during 67 helium-oxygen chamber dives, evaluating 232 subjects with over 2000 observations. These included bounce, excursion and saturation dives. Depths ranged from 35 to 480 meters, with bottom times from 30 minutes to eight days. Gardette found that bubbles identified

during movement generally indicated less risk than bubbles found while at rest. He reported a 1% incidence of DCS in subjects with no detectable bubbles at rest for bounce and excursion dives. Saturation dives yielded different results, though. Absence of bubbles at rest was associated with a 14% incidence of pain, and no bubbles during movement corresponded to a pain incidence of 10%. Gardette concluded that a higher incidence of DCS related problems would be found with higher bubble grades in general and that 'movement' case bubbles would be less indicative of decompression sickness risk. However, it would not be possible to predict pain. Further, it appeared that while Doppler evaluation could be acceptable for bounce and excursion diving, it was not suitable for saturation exposures.

Gardette's findings are supported by other investigators. DCS incidence, similar to his 1%, have been reported for air dives by Spencer and Johanson (1974) at 1.3% and Nashimoto and Gotoh (1978) at 0%. Likewise, his 14% becoming symptomatic in the absence of bubbles at rest is similar to Powell et al.'s (1983) finding of 16%. Brubakk et al. (1986) confirmed the difficulty in assessing saturation dives. They followed six saturation divers executing ascending excursions from 300 to 250 msw. They found no correlation between circulating bubbles and the development of decompression sickness.

Bayne et al. (1985) used Doppler to follow 83 subjects during

chamber dives to 285 fsw for bottom times ranging from 6 to 11 minutes (decompression according to standard U.S. Navy tables). Based on Doppler scores alone, 36 of the subjects had decompression sickness. Only five of these individuals had clinically demonstrable signs or symptoms. False positive scoring was acceptable in that Doppler assessments are supposed to be more sensitive than subjective reporting. Their concern was with three cases that required treatment without Doppler signs. This represented a 38% false negative rate. Using a Chi-square test of independence they report a probability between 25% and 50% that the clinical and Doppler diagnoses refer to independent processes. They concluded that bubble scores may be weakly related to clinical symptoms, but far too weakly to be of diagnostic value in individual cases.

Bayne et al.'s concerns present few difficulties to those interested in ultrasonic assessment. Doppler ultrasonic detection has generally been promoted as an early warning system to encourage closer observation of individuals following the recognition of bubbles. It has not been promoted as a replacement for clinical diagnosis. The majority of investigators use Doppler derived information in a more restricted manner. For instance, Masurel et al. (1978) followed six divers over a six day saturation dive to 430 msw with daily excursions (five to 460 msw and one to 501 msw for durations between 2.5 and five hours). They reported that while bubble scores did not necessarily indicate immediate danger

for divers, they were useful in adjusting decompression schedules to minimize the flow of bubbles. This dynamic implementation of Doppler results is probably the most significant benefit to be derived from the technology. In fact, Bayne et al.'s (1985) concerns simply act to reinforce this distinction and the importance of developing alternative methodologies as discussed below.

Alternative Methodologies

Doppler ultrasonic systems are limited in that they are only sensitive to bubbles moving within the narrow focal range of the transducer. Individual bubble size and progression can not be monitored and tissue bubbles or stationary vascular bubbles can not be identified. Problematically, it has been reported that tissue bubbles appear prior to circulating bubbles. To meet this concern, alternative methodologies that are sensitive to both moving and stationary bubbles have been proposed and employed (Christman et al., 1986). Pulsed-echo ultrasound is the most commonly reported (Walder et al., 1968; Manley, 1969; Rubissow and MacKay, 1974; Horton and Wells, 1976; Daniels et al., 1979; Hills et al., 1983; Daniels, 1984; Brubakk et al., 1986). Acoustical-optical imaging techniques have also been proposed (Buckles and Knox, 1969).

Rubissow and MacKay (1974) employed a non-invasive pulsed-echo (scanning) ultrasonic system. Using a frequency of 7.5 MHz, bubbles with diameters within the range of 2-5 microns were "routinely" imaged, with some as small as 0.5-1.0 microns in diameter were detected under optimal conditions. A variety of subject species were used in their work. Guinea pigs to verify the direct effect of pressure (Boyle's Law) on bubble size during recompression. Goldfish for their ability to repeatedly survive severe bubble formation, and Transparent Glass Fish (Ambassis lala) for simultaneous ultrasonic and optical bubble growth studies. Human subjects were used (12 man-dives, 400 fsw saturation dive using helium as the breathing medium) to compare ultrasonic imaging with a commercially available Doppler system. The pulsed-echo imaging picked up bubbles at the site of bends pain (the knees) in two diver and "pre-clinical" bubbles in the knee of another. The Doppler system picked up no bubble signals during the trials. Since saturation dives seem to yield the worst Doppler results, it is possible that this exposure was not optimal for comparing the two systems.

Horton and Wells (1976) introduced their own "pulsed, ultrasonic echo-ranging system". Backed up by a limited number of trials with dogs, they primarily based their work on simulated 'bubble' assessment. They were able to locate bubbles to a minimum size of 26 microns.

Daniels et al (1979) presented a pulse-echo ultrasonic imaging system operating on a frequency of 8 MHz. Using guinea pigs they were able to detect bubbles down to 10 microns. As they expected, they could establish no uniform relationship between bubble radius and image size. Semi-quantitative assessment of relationship was possible. Precise measurements of rates of growth of bubbles less than 300 microns in diameter could not be made.

Hills et al (1983) reported on a pulsed-echoing system that overcame instabilities of the earliest models. The problem, however, is that the apparatus is too complex and not robust enough for routine use outside of the laboratory. They used a lower frequency system than employed by previous investigators (1.5 MHz) to evaluate potential change in the velocity of sound with change in bubble size. They found that the sensitivity of the velocity of sound to bubble content was great enough that they felt they could detect a 2:1 reduction in the velocity of sound with a much simpler and less-expensive apparatus than they employed. They felt that monitoring the velocity of sound would allow for the detection of all tissue gas, whether stationary or moving, and could form the basis for an inexpensive device sufficiently acceptable for routine use offshore.

Daniels (1984) expressed concern over the difficulties of previous investigators in achieving strong correlations between the presence of circulating bubbles and the development of

symptomatic decompression sickness. He completed a series of studies employing both animal and human subjects using pulse-echoing imaging that allowed the evaluation of circulating as well as stationary bubbles. He was able to conclude that: 1) initial bubbles are intravascular; 2) both the number of bubbles and the number of sites of formation are dependent on the magnitude of the decompression; and 3) before symptoms of DCS occur, an accumulation of stationary bubbles will be observed.

Daniels' (1984) finding that stationary bubbles accumulate prior to the onset of symptoms of DCS emphasizes the benefits of the pulse-echo analysis. However, the fact that intravascular bubbles will be detected first promotes the idea that more sensitive Doppler systems may hold the ultimate appeal.

Buckles and Knox (1969) proposed a second alternative to Doppler systems with acoustic-optical imaging. They attained direct optical images from acoustic wave forms. They exposed hamsters to 13.6 ATA for 30 minutes followed by explosive decompression completed within five seconds. They were then able to identify bubbles to less than 700 microns in diameter. Buckles and Knox concluded that these findings warranted further investigation. Possibly because of the increased complexity of their system over other elucidative equipment, no follow-up has been found in the literature.

Conclusions

Despite inherent weaknesses in the techniques and numerous proposals for alternative approaches, the basic Doppler techniques continue to be used. While more comprehensive or controlled analysis might be preferable, Doppler techniques remain significantly easier and less expensive to employ. It must be emphasized, however, that the sensitivity of Doppler techniques is limited. At best, they permit only gross analysis of subjects and rough cross-correlation of bubble "grades" with the risk of developing decompression sickness. Nevertheless, they represent techniques that are worthwhile within the scope of their design.

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II. Laboratory Study

Statement of the Problem

Literature concerned with the effect of thermal variation on compression/decompression is sparse. In related work, nitrogen washout studies employing human subjects have demonstrated that significantly greater inert gas elimination is seen when ambient temperatures are warm as opposed to thermoneutral (Balldin and Lundgren, 1972; Balldin, 1973). This increase, correlating with temperature rise, can be seen to result from vasodilation increasing circulation to the periphery thereby encouraging nitrogen outflow. This response to hyperthermia has been supported by the work of Bove et al. (1978) with rabbits. Measuring ^{133}Xenon washout, they found elimination rates to be higher during heat stress.

Dunford and Hayward (1981) demonstrated that divers cooled during compression displayed post-decompression venous gas emboli (VGE or bubble) scores one-third as high as those seen in subjects kept warm during the compression phase. They postulated that cooling the body would decrease both the uptake and the elimination of inert gas. Thermoregulatory vasoconstriction would cause a decrease in blood flow to the peripheral tissues, thereby decreasing the rate of nitrogen exchange. Conversely, elevating the ambient temperature should result in vasodilation, increased perfusion and subsequently an increase in the rate of uptake or elimination of inert gas (depending on the phase being considered).

Indeed, Dunford and Hayward (1981) reported that subjects demonstrate a more rapid decline in post-dive bubble scores following active warming compared to passive warming. Unfortunately, they were inconsistent in that active rewarming employed water immersion while passive rewarming took place in sleeping bags. Without providing a control, Dunford and Hayward indicated that the increased hydrostatic pressure resulting from the water immersion of their rewarming procedure was responsible for the accelerated decline in bubble scores. This could be indirectly supported by Balldin and Lundgren (1972), who found that immersion in water to the neck increased nitrogen elimination during oxygen breathing. Studies comparing thermally different post-dive immersion responses have not been found.

The complex, often paradoxical, nature of temperature effects on gas uptake and elimination and the practical implications should make thermal factors an important point of research concern. Reduced thresholds for bubble formation have been reported to accompany both hypo- and hyperthermic exposure (Shida et al., 1982; Lin et al., 1984). In fact, opposing mechanisms may be responsible for the similar response to either form of thermal stress.

The paucity of literature makes it difficult to prescribe optimal dive and post-dive thermal practices or treatment protocols. Further investigation seemed warranted. In this study, the effect of post-dive peripheral warming on inert gas elimination

is investigated.

The Problem

This study evaluates the effect of post-dive thermal status on inert gas elimination.

Hypotheses

Three major hypotheses are evaluated:

1) Identical dive profiles will result in similar VGE scores immediately post-dive in subjects with similar physical characteristics.

2) Post-dive warming will result in higher immediate post-immersion VGE scores than thermoneutral post-dive exposure.

3) Post-dive warming will result in faster elimination of nitrogen, hence VGE scores will fall more rapidly following warm post-dive conditions compared to thermoneutral post-dive conditions.

Significance of the Study

To date, most concern regarding thermal conditions and diving has been limited to cold instead of heat stress. While this may not be completely inappropriate, a balance must be struck in light of modern diving equipment and operating procedures. The prevalent use of drysuits by divers (recreation, scientific and commercial) makes cold stress less of a problem that it has been in the past. Further, given the present emphasis on diver comfort, it is not uncommon to find hot showers, saunas, and even whirlpools readily available for post-dive comfort. This suggests a need for a clear understanding of the effects of heat stress on divers.

It is generally recognized that increasing the rate of nitrogen elimination can be beneficial. Shorter obligatory surface intervals would be required if off-gassing rates could be increased. Such benefits will not exist, however, if localized gas phase separation exceeds perfusion rates. Perfusion rates act as the limiter for bodily elimination of inert gas. The gas must reach the alveolar-capillary interface in manageable volumes and sizes to pass into the alveoli without coalescing into large bubbles capable of disrupting any aspect of transport, circulation and/or alveolar-capillary membrane exchange.

A postulation on the possible effects of thermal factors may provide a good example of the potential complications. An

individual who is warm on initial exposure to hyperbaric conditions may demonstrate rapid uptake of inert gas throughout the body since perfusion rates will be relatively high throughout the core and periphery. Subsequent cooling over the course of the exposure may result in decreased perfusion rates in some of the now nitrogen loaded tissues. Active rewarming following decompression may result in the peripheral tissue being supersaturated with nitrogen since gas solubility decreases with increasing temperature (Mack and Lin, 1986). This may result in the formation of bubbles in peripheral tissues before perfusion rates can be restored or increased to the point that the gas can be eliminated in a controlled manner. As suggested by the preliminary work of Mekjavic and Kakitsuba (unpublished), this could result in an increased incidence of cutaneous bends and therefore is contraindicated.

This study was conceived as a step towards quantifying the effects of post-dive thermal factors on nitrogen elimination in divers. It is anticipated that the findings will contribute to the clarification of optimal post-dive behaviour for divers and possibly of management procedures for diving accidents that have been complicated by thermal factors.

Delimitations

Dry chamber dives were employed to enhance the reliability of this experimentation. It is understood that this approach may affect the validity of the results. Caution is advised in applying these findings to less controlled situations.

In the interest of minimizing potentially confounding factors, intersubject variation was restricted. Participation was limited to males to remove possible gender effects. An age range of 18 to 35 years was established to minimize effects of related physical changes, such as changes in circulatory performance. In addition, the minimum age of 18 years simplified consent procedures.

Limitations

While the clinical patterns of decompression sickness are well recognized, controversy exists regarding the dominant mechanisms involved and their etiology. This study considers peripheral areas without dwelling on these issues.

The appropriateness of Doppler ultrasonic techniques has been questioned by some investigators and are therefore not universally accepted. Reservations notwithstanding, they continue to be used. While alternative procedures might exist, they are generally

significantly more difficult and expensive to employ. It must be emphasized, though, that the sensitivity of Doppler ultrasound is limited. At best, only gross analysis of circulating VGE within subjects and rough cross-correlation of bubble "grades" with the risk of developing decompression sickness is permitted (Gardette, 1979; Powell et al., 1983; Dunford, 1988).

A further limitation of this study is the hyperbaric exposure selected. The protocol employed a dive profile (70 feet for 35 minutes) typically more conservative than many regularly experienced by recreational, scientific or commercial divers. It was markedly less severe than those selected by previous investigators that demonstrated good Doppler results (Spencer and Clarke, 1972; Dunford and Hayward, 1981). Spencer and Clarke completed a chamber dive to 200 feet for 30 minutes (decompression according to U.S. Navy tables). Dunford and Hayward (1981) exposed their subjects to open water (10°C) dives to 78 feet for 38 minutes. A recent shift in appreciation of the potential hazards of hyperbaric exposure has necessitated a more conservative approach. Like many Canadian institutions / organizations involved in diving experimentation, Simon Fraser University has adopted a more conservative set of exposure tables than have been employed in the past. These were developed at the Defense and Civil Institute of Environmental Medicine (DCIEM) in Ontario with the intent of limiting dives to minimal grade or bubble free exposures (Lauckner et al., 1984(a&b), 1985). Further concerns of the

University regarding the ethics of exposing humans to situations expected to produce VGE made obtaining clearance for the experimentation difficult. To allay these concerns, the very moderate exposure was used. It was recognized, therefore, that the combination of a mild hyperbaric exposure and the relative insensitivity of the Doppler technique might result in indistinguishable differences between the conditions. Despite this, the lower risk to the subjects dictated that a preliminary investigation use this protocol.

Finally, limitations of time, money and equipment necessarily restrict the focus of this study to the post-dive status of divers following warm dives. Other possible protocols would include post-dive status following cold exposures as well as warm exposures, however, these will have to await future work.

Methodology

All dives were completed in the Environmental Physiology Unit hyperbaric chamber at Simon Fraser University. Anthropometric assessments were completed in the Buchanan Fitness Laboratory at the University of British Columbia.

This project was conducted in accordance with guidelines reviewed in the 'Policy of the School of Kinesiology on the Medical Clearance and Supervision of Human Research Subjects' (Appendix I).

Subjects

Ten male subjects participated in the study. All were certified divers between the ages of 18 and 35 years. Advertisement was carried out among divers registered with the University of British Columbia Diving Operations Office, divers associated with the Simon Fraser University School of Kinesiology and through privately owned scuba stores in the greater Vancouver area. Selection was based upon voluntary availability and medical assurance of fitness for diving (Appendix II).

Testing Procedures

No subject was allowed to participate in the study without first being made aware of the purpose of the study, the testing procedures and protocols and any known or suspected problems which might result from the experimental procedure (Appendix III), or general hyperbaric exposure (Appendix IV). Written consent was provided by each subject at this point (Appendix V).

Subjects were requested to avoid diving activity seven days prior to all experimental trials to avoid possible confounding caused by residual nitrogen. They were further asked to standardize their dietary and behaviourial patterns prior to all trials (Appendix VI).

Subjects completed two trials. One with warm, and the other with thermoneutral post-dive exposure. The order of warm and thermoneutral exposures was randomized within the study group. Trials were held on separate days at least four days apart to ensure complete offgassing from prior exposure and trials for individual subjects were scheduled at the same time of day to avoid intraindividual variation due to circadian rhythms.

A questionnaire was administered to all subjects prior to both trials to identify factors potentially affecting the outcome of the trials (Appendix VII).

Testing Protocols

Physical characteristics were determined for all subjects. These included height, weight and percentage body fat. Skinfold values were measured at six sites to provide information requested by the agency assisting in analyzing the study results (Defense and Civil Institute of Environmental Medicine - DCIEM). The percentage of body weight made up of fat was estimated by a hydrostatic weighing technique outlined by Katch et al. (1967).

Upon arrival at the hyperbaric laboratory, subjects were given rectal temperature probes to be inserted 15 cm. Core temperature was then evaluated for each subject. Following this and prior to diving, a reference signal was recorded for each subject using a Doppler Ultrasonic Bubble Detector interfaced with a cassette recorder. This according to the protocol described by Eatock and Nishi (1986). The baseline allowed comparison with post-dive signals to estimate circulating bubble activity resulting from the dive and post-dive exposures. The 'dives' were dry hyperbaric chamber exposures to an equivalent ocean depth of 70 feet for an actual bottom time of 35 minutes (no staged decompression required). Subjects were instructed to remain at rest throughout. Immediately following the dive, subjects walked to four feet deep "hot tubs" set up in the laboratory. Just prior to entering the water, core temperature was evaluated. Subjects were then immersed to the neck while in a sitting position (at rest) in either

thermoneutral (28°C) or warm (38°C) water for 30 minutes. Following the water immersion, subjects moved out of the tubs and were evaluated ultrasonically in a manner similar to the baseline evaluation. Simultaneously, core temperature was assessed to determine any changes resulting from the immersion exposure. Subjects were then encouraged to remain at rest for the next 2.5 hours within the laboratory area. At 30 minute intervals ultrasonic evaluations were made. The rectal probes were removed when and if subjects demonstrated core temperature stability over the period of two evaluations.

Apparatus

The hyperbaric chamber employed in this study was a multi-person/multi-lock hypo/hyperbaric chamber constructed by Perry Oceanographics, Florida.

The Doppler Ultrasonic Bubble Detector (Model 1032 G) was produced by the Institute of Applied Physiology, Seattle. This was used in conjunction with a precordial lead and headphones. Sound transmission was increased with Aquasonic 100 ultrasound transmission gel.

Ultrasound recordings were made using a Sony Stereo Cassette Deck (Model TC-188SD) and high quality Maxell 90 minute tapes.

Data Analysis

Ultrasound recordings were evaluated in terms of bubble 'grade' (VGE score) according to a protocol described by Eatock and Nishi (1986). This was completed at DCIEM, Downsview, Ontario. This protocol is a refinement based on the original Kisman-Masurel coding procedure (Kisman et al., 1978). Evaluations were completed in a blind manner, with no reference to experimental conditions.

Resultant bubble grades were to be compared using a two-way analysis of variance (ANOVA) and trend analysis to determine differences between the two exposure conditions.

Only complete subject results were to be analyzed statistically. Incomplete results were to be included in the discussion.

Results

Physical characteristics of the subjects are presented in Table I. Mean age was 24.9 +/- 2.8 years (range 21-29 years). Mean height was 182.3 +/- 4.6 cm (range 173.4-190.1 cm). Mean weight was 76.7 +/- 5.6 kg (range 71.7-88.8 kg). Mean percent body fat was 13.4 +/- 4.6% (range 6.6-21.7%).

Core temperature was evaluated rectally for most of the subjects through both experimental trials. Subjects 01, 07, and 09 declined evaluation during their second trials for personal reasons. No changes in core temperature were observed in those monitored throughout either condition.

All ultrasonic evaluations were completed according to the planned schedule. At the time of recording, no bubbles could be identified aurally by the recording technician. Interpretation of the tape records by two independent evaluators at the Defense and Civil Institute of Environmental Medicine confirmed that measurable bubbles were not present in any subject under either condition. Subsequently, no statistical interpretation was conducted.

Table I Anthropometric Characteristics

Subject	Age (yrs)	Height (cm)	Weight (kg)	Body Fat (%)
01	22	178.2	74.5	8.2
02	23	185.7	73.2	12.0
03	29	184.9	73.4	16.0
04	25	190.1	78.8	13.0
05	21	185.0	77.1	6.6
06	23	181.3	73.1	12.2
07	27	182.9	71.7	10.6
08	25	173.4	72.2	14.7
09	29	179.9	88.8	21.7
10	25	181.9	83.7	18.6
$\bar{X}$	24.9	182.3	76.7	13.4
SD	2.8	4.6	5.6	4.6

Discussion

The human system has not specifically evolved for rapidly changing pressure, but within a narrow optimal operating range it has a remarkable capacity for protecting itself. The body can withstand repeated compression and decompression respiring gases of varying densities, and in some instances, compositions. Present appreciation of the impact of hyperbaric exposure highlights the marvel of the human system's adaptability under adverse conditions.

Decompression sickness (DCS) arises when the ability of the body to adequately manage a given decompression stress is surpassed. Assessing safe parameters is a complicated task. Actual response can diverge dramatically from physiological baselines as a result of inter- and intra-individual variability. The best general protection is probably to ensure that all physiological systems are operating within their optimal range.

Essentially, safe decompression will occur if the uptake and elimination of inert gas can be regulated so that excessive gas phase separation (bubble formation) does not occur. The extent of VGE (circulating bubbles) presence is normally used as a rough measure of relative risk.

The primary impact of thermal stress (warm or cold) is the

alteration of circulation/perfusion rates. Both diffusion (Hills, 1967; 1977) and perfusion (Ohta et al., 1978) are important in the uptake and elimination of inert gas. Perfusion rates are more variable and are therefore considered the prime governor of gas exchange under optimal conditions (Ohta et al., 1978), but the key consideration is that perfusion must balance diffusion. Unmatched diffusion will result in excessive gas phase separation - potentially leading to DCS. Therefore, factors which affect perfusion can significantly alter the perfusion/diffusion balance and potentially challenge the norms established in the existing decompression tables (Bove et al., 1978). This would act as a critical element in altering the risk of developing DCS.

Exposure to cold will cause vasoconstriction, subsequently reducing peripheral perfusion. If experienced during compression, reduced gas uptake will result. Dunford and Hayward (1981) found that this could confer added protection against DCS since lesser quantities of gas will have been absorbed during compression, thereby decreasing the risk of excessive gas phase separation upon decompression. If the cold stress is introduced during the bottom time, or during or after the decompression phase, more insidious results may be expected. Peripheral tissues will be at least partially loaded with inert gas before the vasoconstriction occurs. Excess gas (compared to surface partial pressures) would be trapped in the periphery and therefore increase the probability of bubble formation during decompression if supersaturation is not matched

by perfusion rates. Gas phase separation could then result in potentially disruptive bubble growth and coalescence before the bubbles could be removed.

Exposure to heat will cause vasodilatation and perfusion rate increases. Occurring immediately before or during compression or during bottom time, gas uptake will be increased. During non-saturation dives, this can be seen to increase the risk of developing DCS during or following decompression since an increased gas load will be borne by the body. Mild heat applied during decompression may counter this effect. Enhanced perfusion during decompression could be seen to confer protection against DCS by increasing the rate of elimination. Vann (1982) and Dick et al. (1984) demonstrated that mild exercise during a dive will increase the rate of post-dive nitrogen elimination due to increased perfusion and reduced hypothermic vasoconstriction. The important point to observe is that hypothermic vasoconstriction was reduced, not reversed. The energy expenditure kept the divers adequately perfused throughout the exposure and enhanced the decompression gas elimination. Applying external sources of heat to an already cold diver will not provide the same response. Gas solubility is strongly affected by temperature. Most gases decrease in solubility by one to six percent for every 1°C rise in temperature (Weathersby and Homer, 1980). Warming peripheral tissue may then result in problematic gas phase separation before the perfusion rates increase sufficiently to remove the gas. Indeed, this was

seen by Mekjavic and Kakitsuba (unpublished). They observed that while six hours post-decompression were uneventful, hot showers then precipitated symptoms of cutaneous DCS in three out of four subjects who had come from a three hour post-decompression cold exposure. Symptoms were not observed in subjects coming from a similar post-decompression hot air exposure. They concluded that should post-dive warming be carried out, it should not be applied rapidly at the risk of precipitating DCS. Indirectly supporting this recommendation, Simmons et al. (1982) observed a higher incidence of DCS associated with summer caisson work compared to winter operations. Since the pressurized area had relatively uniform temperatures throughout the year they suggested that the higher incidence may be a result of the rapid post-decompression heating.

A series of studies have been completed evaluating the impact of various aspects of thermal stress. Balldin (1973) demonstrated that increased ambient temperatures cause significant increases in the rate of nitrogen elimination during oxygen breathing. Balldin and Liner (1978) demonstrated that chemically induced vasodilatation enhanced elimination and provided dramatic protection against DCS in rabbits. Shida et al. (1982) found that rats exposed to hyperthermic conditions throughout compression and decompression phases of a dive to 10 ATA were able to withstand significantly greater pressure reductions before detectable bubble formation ensued. Unfortunately, work has not been reported to

consider external post-dive warming and the potentially complicated interactions between solubility, gas phase separation and perfusion. Since post-dive external warming is commonly experienced by divers through showers or hot tubs, this area demands further attention.

The present investigation was designed to evaluate the impact of post-decompression warming on divers. Employing a protocol that had subjects warm throughout the compression and decompression phases it was expected that post-dive warming would accelerate inert gas elimination rates due to the increased perfusion. Using measurable bubble formation as a guide to elimination rates, it was expected that higher bubble grades would be seen immediately following warm as compared to thermoneutral immersions. More rapid degradation of the bubble grades in the warmed group would confirm the accelerated elimination. Demonstrating this phenomenon, the next step would be to contrast these results with similar immersion protocols following dives where: 1) the subjects were warm through the compression and bottom phases of the dive and cold through the decompression phase, and 2) the subject was cold throughout all phases of the dive. Cold throughout, divers should demonstrate lower bubble grades following rewarming. Warm during compression and cold during decompression, the divers would demonstrate the greatest susceptibility to DCS with post-dive warming since peripheral tissues would have significant inert gas loads that would be trapped due to vasoconstriction through the decompression

phase. These subjects would display the highest and sustained bubble grades reflecting the greater volume of dissolved gas and the delay in elimination until VGE were allowed to form. These subjects would have the greatest risk of developing cutaneous DCS.

The present study did not produce measurable VGE following either thermoneutral and warm post-dive immersion exposures. This reflected the moderateness of the dive protocol employed. The majority of research relying on Doppler assessments have used the more stringent exposure limits allowed by the U.S. Navy tables. The 50 minute no-decompression limit of these tables is contrasted to the 35 minute limit established of the Canadian Forces Air Diving Tables (1986). While previous work has confirmed that caution must be exercised when following the U.S. Navy tables, it is concluded from the present investigation that within the limits of the Canadian tables, mild post-dive warming does not appear to pose a significant risk to the diver.

It must be stressed that only mild post-dive heating should be considered by divers. Large differentials between skin temperature and heating temperature may still precipitate VGE formation and DCS following moderate dive exposures for the reasons of solubility and perfusion/diffusion inequities. The best recommendation is that a temperate approach be maintained in all activities related to diving.

The present investigation supports the use of mild peripheral warming as an adjunctive therapy for decompression sickness. There are two separate lines of logic for this recommendation. Using the endogenous heat production of exercise to maintain subject warmth (Dunford and Hayward, 1981) has resulted in significantly greater VGE scores than the presently employed passive peripheral warming. This suggests that some combination of the mechanical irritation and cavitation associated with muscular contraction, the decreased solubility of muscle tissue resulting from localized warming, and the increased partial pressure of CO_2 associated with exertion may be responsible for the development of VGE. Passive peripheral warming may provide the safest means of increasing inert gas elimination without encouraging VGE development.

The second benefit of peripheral warming relates to one of the most accepted adjunctive therapies in the present management of DCS - oxygen therapy. The benefits of oxygen in maximizing the gradient for nitrogen elimination, improving oxygen transport in the lung, and increasing dissolved oxygen transport are well established. However, high partial pressures of oxygen have also been demonstrated to significantly reduce peripheral circulation (Bird and Tefler, 1965). While this has been reported to have only minimal effect on nitrogen elimination (Plewes and Farhi, 1983), it may exacerbate perfusion/diffusion imbalances in peripheral tissues. Passive peripheral warming would assist in reversing this development.

Conclusions

The existing literature offers scant consideration of the impact of thermal factors on decompression safety. Experimental investigation is required to elucidate the mechanisms and responses resulting from thermal stress.

The present investigation has confirmed that the Canadian Forces Air Diving Tables provide a safe limit of hyperbaric exposure for the profile tested, regardless of moderate post-dive thermal stress. For practical application, it is emphasized that only moderate heating should be considered.

Increased inert gas elimination in the absence of VGE formation caused by mild post-dive peripheral warming suggests that this could be an appropriate adjunctive therapy for decompression sickness.

Recommendations

Further investigation should consider:

1) the role of skin temperature, perfusion and peripheral warming in inert gas elimination and VGE formation - to provide recommendations for diving practices and possibly DCS therapy;

2) the importance of partial pressures of CO_2 and mechanical irritation in the generation of VGE - to provide recommendations for post-dive activity levels.

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Appendices

A. Subject Forms/Information Materials

Appendix I

Policy Of The School Of Kinesiology On The Medical Clearance And Supervision Of Human Research Subjects

1. Informed Consent

Informed consent must include evidence that the subject has agreed to participate in the experiment on the basis of a clear description of the risks and potential benefits involved as well as the understanding that withdrawal from participation is the subject's right at any time without prejudice. Informed consent must be secured by the experimenter before the subject can participate in the research.

2. Medical Clearance

Medical clearance must be obtained in advance for all subjects in research which involves significant risk of injury or disease, and in particular when research involves exposure to hypoxia, hyperbaric oxygen or other hyperbaric application of biological, chemical or physical toxins.

The examining physician must be informed of the nature of the hazards the subject will face. In hyperbaric research, the standard medical clearance form authorized by the Simon Fraser University Diving Control Board must be used.

3. Medical Supervision

a. The following areas of research require the presence of a physician on campus who is informed of the nature of the research and available to render assistance in an emergency:

   i. hypothermia
   ii. hyperthermia
   iii. hyperbaric oxygenation
   iv. hyperbaric exposure

b. The following stressors require the presence of a physician <u>in the lab</u> during experimental sessions:

   i. hypothermia with the subject's core temperature falling below 35°C during the application of cold stress;

ii. hyperthermia with core temperature rising above 40°C;
iii. hyperbaric oxygenation with P_IO_2 greater than 2 ATA;
iv. hypoxia with P_IO_2 less than 90 mm Hg, or with ambient temperature less than 19°C or greater than 24°C and P_IO_2 less than 105 mm Hg;
v. exercise stress tests when:
(a) the subject is exercised to exhaustion and has a predicted $\dot{V}O_2$ max less than 40 ml/kg/min, or
(b) the subject has an underlying disease or condition which is a significant predisposition to cardiac arrest during exercise

c. During exercise stress tests other than as described in section 3.b.v., above, a physician must be available within approximately three minutes access of the site of the tests, and must be informed in advance that such testing will be taking place.

d. During hyperbaric research, in the event that the supervising physician must enter the chamber to assess or treat a subject, a back-up physician must be available to provide medical supervision from outside the chamber.

e. During all research described in this section (3), at least one person in the lab must hold valid certification in basic cardiopulmonary resuscitation.

4. Resuscitation Supplies

The "crash cart" containing equipment and supplies for use in resuscitation of victims of cardiac arrest, decompression accidents and hypo/hyperthermic accidents shall be available on site whenever research is undertaken in which there is a significant risk of such accidents. Normally, the cart will remain in the Environmental Physiology Unit and will not be moved nor disturbed unless so ordered by a physician involved in medical supervision. The physicians on faculty in Kinesiology shall periodically check such equipment and supplies and arrange for necessary replacements or repairs, the costs being borne by the School of Kinesiology.

5. Compensation For Physician's Services

Medical clearance and medical supervision in research in Kinesiology shall normally be among the responsibilities of the physicians on faculty, with full respect given to these physicians' teaching, professional and individual research responsibilities in scheduling such clearance and supervision. Research funded by external agencies and requiring medical

clearance of subjects should have, as a budget item, compensation to the School of Kinesiology if physicians on faculty are asked to provide this service.

Contract work which requires the medical clearance and/or supervision of subjects by physicians should include budget items for direct financial compensation of such physicians, be they on faculty or not. Provision of such medical services in contract work is not one of the responsibilities of the physicians on faculty.

6. University Ethics Committee

Kinesiology ethics policy for human experimentation will naturally be subordinate to University policy in case of conflict between the two. Submissions of research proposals from Kinesiology to the University Ethics committee shall include sufficiently detailed information concerning risks and safeguards to permit that committee to judge the adequacy of such safeguards in their deliberations.

7. Application Of This Policy

Copies of this policy shall be distributed to all faculty, students, and staff involved in research on human subjects involving a significant risk of injury or disease, whenever such research is done under the aegis of the School of Kinesiology. Adherence to this policy is mandatory unless exemption is granted in advance by the School of Kinesiology and the University Ethics Committee.

Ratified 09 October 1985

Appendix II

Medical Questionnaire (Confidential)

Surname _____

Given Name _____

Address _____

Phone _____

Date of Birth _____

Weight _____

Family Physician _____

Address _____

Phone _____

Medical History

Please answer the following questions accurately since they are designed to identify subjects who should not participate within the proposed study. Please place a check-mark by any condition which applies to you. Responses will be viewed only by the principal investigator and departmental physician.

Have you suffered, or do you now suffer from any of the following?

1. asthma [ ]
2. bronchitis [ ]
3. tuberculosis, emphysema, fibrosis, pleurisy [ ]
4. other respiratory abnormality or scarring [ ]
5. pneumothorax or collapsed lung [ ]
6. nasal obstruction [ ]
7. frequent or severe nose bleeds [ ]
8. frequent cold or sore throats [ ]
9. chest pain and persistent cough [ ]

10. coughing up blood (hemoptysis) [ ]
11. heart disease [ ]
12. high or low blood pressure [ ]
13. abnormal EKG [ ]
14. claustrophobia [ ]
15. alcohol or drug abuse [ ]
16. allergies [ ]
17. communicable diseases or contact with patients with same [ ]
18. diabetes [ ]
19. dizziness, fainting spells or fits [ ]
20. do you smoke or have you smoked in the past? [ ]
21. are you under medical care now or taking medication? [ ]

Please clarify affirmative answers.

I declare the above answers are, to the best of my knowledge, a true and accurate statement of my medical history.

Signed _____

Date _____

Witness _____

Appendix III

Subject Information Package (1)

The Effects Of Post-Dive Warming On Vascular Gas Emboli Production

Item 1 Project Objectives:

The primary objective of this study is to investigate the effects of post-dive thermal exposure on the production of vascular gas emboli.

Item 2 Test Procedures:

Volunteers who participate in this study must first meet minimal standards of good health as certified by them in the subject Medical Questionnaire and after examination by a diving physician. If the physician is other than Don Hedges, MD (diving medical officer of the SFU School of Kinesiology) the completed medical records must be made available for his review.

All subjects will undergo two hyperbaric chamber dives (dry) completed seven days apart (note: all subjects shall refrain from any other hyperbaric exposure seven days prior to both test sessions). All exposures will be to an equivalent ocean depth of 70 feet (fsw) for 35 minutes. Subjects shall remain at rest throughout the exposure. Following the dive subjects will be immersed to the neck in thermoneutral (30°C) or warm (37°C) water for 30 minutes.

Monitoring of subjects for vascular gas bubbles will be done non-invasively employing a Doppler Ultrasonic Bubble Detector. Observations will be made immediately after the dive and at 30 minute intervals for the next four hours. Throughout this period subjects will remain at rest in the Environmental Physiology Laboratory.

Item 3 Risks and Discomforts:

a(i) Hyperbaric Exposure

Exposure to hyperbaric air at 70 fsw is considered to be a fairly conservative diving exposure commonly experienced by sport divers. Subjects should be aware, however, of the serious risks of hyperbaric chamber diving. These are outlined in the separate section

entitled "Risks During Exposure to Hypo/Hyperbaric Conditions" (Subject Information Package (2)). It is important that subjects READ THIS INFORMATION CAREFULLY.

a(ii) Water Immersion

The immersion of subjects to the neck in this study should offer little discomfort and no risk. Sensation of temperature will be mild and not uncomfortable.

a(iii) Safety Precautions

During each dive fully trained technical staff, including two chamber operators and a physician trained in diving medicine will be on hand. In addition, a tender trained in CPR will accompany subjects during the chamber dives. Instruments and materials needed for resuscitation and for the treatment of decompression sickness and related condition will be available in the laboratory at all times. Sessions in the chamber will be undertaken during hours when the Health Services is open and physicians there will be informed in advance of the nature of the sessions in the remote chance that their assistance would be required (eg. should the physician on site need to enter the chamber to assist a subject).

Item 4 Inquiries:

Questions regarding the procedures are welcome. If you have any doubts please ask for further explanations.

Item 5 Freedom of Consent:

Participation is on a voluntary basis. You are free to deny consent at any time during or between trials.

Item 6 Confidentiality:

All questions, answers, and results from this study will be identified in the resultant manuscript and/or publications by use of subject codes only.

Appendix IV

Subject Information Package (2)

Risks During Exposure To Hypo/Hyperbaric Conditions

Environmental Physiology Unit
School of Kinesiology
Simon Fraser University

February, 1986

Risks during open-water or hypo/hyperbaric chamber diving include the following:

1. Otic Barotrauma

Known as an "ear squeeze", this injury is caused by failure to equalize air pressure between the external environment and the middle ear, generally during pressurization. It involves ear discomfort, pain and sometimes ringing in the ears or bleeding in the ear drum; occasionally the drum is perforated. It generally resolves rapidly (days) but if severe may require medication and/or a specialist's attention. Its prevention requires gentle ear-clearing manoeuvres every second or two during changes in surrounding pressure. If such manoeuvres fail, reducing the pressure difference (eg. by partial ascent during a dive) should be tried, with repetition of the clearing manoeuvres. If this fails, the exposure should be terminated in an orderly manner. Correction is easiest if undertaken at the first sign of inability to equalize. DO NOT WAIT UNTIL YOU FEEL PAIN! Equalization will be harder if you have a cold or "flu"; under these circumstances hypo/hyperbaric exposure must be postponed. Seasonal allergies may also require postponement of an exposure, unless advised otherwise by the physician responsible for medical aspects of the dive. DO NOT TAKE over-the-counter remedies unless so advised by the physician.

2. Decompression Sickness

Generally known as "the bends", this condition develops when nitrogen bubbles form in body tissues during depressurization. Some nitrogen is dissolved in tissues even at the surface, but much more is "loaded" into the tissues during compressed air breathing, in direct proportion to the depth or pressure and to the time spent at hyperbaric pressure. On depressurization, the nitrogen is "supersaturated" in the tissues and if this depressurization occurs too rapidly to allow the offloading of the nitrogen from the blood into the

alveoli of the lungs, the nitrogen in the blood and tissues can form bubbles large enough to do damage and to cause symptoms as described below.

a. Limb Bends

If the bubbles form in tissues in and around joints, the result will be a steadily increasing deep aching pain in the involved joint(s). This pain increases with time and can become excruciating if not treated. Predispositions include previous injury or surgery in a joint or, during exposure, a cramped posture limiting circulation in the joint area. Treatment is immediate repressurization in a hyperbaric chamber using a well established protocol alternating air/oxygen breathing (U.S. Navy treatment tables are used at Simon Fraser University); this is virtually 100% effective in uncomplicated cases treated rapidly. It is thus mandatory for all divers, subjects and tenders exposed to hyperbaric conditions to maintain a one hour (or more) "bends watch", ie. to stay under supervision for that time in the chamber laboratory; to report any and all symptoms which arise immediately to the chamber operator or physician responsible, to wear a diver's medical alert bracelet for the next 24 hours during which s/he must not be left alone; to report any and all symptoms during that or the subsequent period (to the operator, physician, or nearest treatment facility - eg. Vancouver General Hospital); and during that 24 hours to abstain from flying or diving except as approved by the operator and physician, since altitude depressurization (flying) increases the rate of bubble formation and repetitive dives increases the onloading of nitrogen at a time when nitrogen from the first exposure may not have been fully off-loaded.

b. Nervous System Bends

Bubble formation during decompression can occur in the circulation of the central nervous system, producing deleterious effects by direct mechanical obstruction of blood flow or indirectly by complex interactions with blood components. Commonly these effects occur at the spinal cord level, due to the sluggish blood flow in the extra-vertebral venous system. Spinal involvement can produce a variety of symptoms including numbness, weakness or paralysis of one or more limbs, loss of coordination, and changes in bowel or bladder control. Other manifestations of nervous system bends include dizziness with or without "ringing" in the ears and hearing loss (vestibular and/or auditory system involvement) as well as decreased alertness, level of consciousness and ability to think clearly. THESE

EFFECTS INDICATE AN EMERGENCY! They must be reported immediately since any delay in treatment reduces the likelihood of full recovery. Treatment is immediate recompression, as for limb bends, but with a different treatment table plus or minus the addition of certain drugs (eg. steroids) and resuscitatory manoeuvres as needed. Although symptoms of nervous system involvement typically develop within the first few minutes after decompression, they may be subtle and/or arise later; the same bends watch and 24 hour surveillance is required as described for limb bends.

c. "The Chokes"

This condition develops when large numbers of bubbles come out of solution into the venous circulation and overwhelm the capacity of the lungs to filter them out (all venous blood passes from the right side of the heart into the pulmonary circulation, the vessels of which subdivide many times into smaller and smaller vessels where bubbles are trapped). Symptoms include a burning sort of chest pain, shortness of breath and a cough with or without hemoptysis (blood). Treatment is immediate recompression as described for limb and neurologic bends.

d. "Skin Bends"

Skin bends usually develops after short, deep dry chamber dives and involves bubble formation in the skin during depressurization. it is generally not serious although it may produce significant discomfort, including itchiness and tenderness with a reddening of the skin and/or a splotchy red rash. Although recompression is rarely required, skin bends may be associated with a higher probability of co-existent more serious forms of decompression sickness; hence, symptoms of skin bends must be reported immediately.

e. Dysbaric Osteonecrosis

This is a delayed form of decompression sickness in which cysts form in bones, usually near large joints, and commonly in people who dive frequently over several years. It is believed that this condition rarely, if ever, develops unless the diver has missed a decompression stop (see below) during a previous dive. It is slowly progressive, so that continued diving may cause the cysts to enlarge, which is particularly problematic if the cysts come to involve the cartilage within a joint. Periodic long bone x-rays are sometimes used in monitoring for cyst development among very frequent divers. There is no specific treatment for this

condition. Evidence suggesting dysbaric osteonecrosis includes bone or joint pain and/or a fracture of a long bone; cystic bone breaks more easily than normal bone. Management includes orthopaedic consultation and sometimes surgery, as well as a discontinuation of diving.

f. Miscellaneous

Lymphatic congestion (bubbles) may develop on decompression, often manifested by facial swelling. Abdominal pain may sometimes arise and has been attributed to expanding gas in the intestines during decompression. Vague or unusual symptoms may also arise. All symptoms must be reported after decompression (including hypobaric exposure); decisions about treatment and follow-up and management should be left to the physician, not the individual exposed to pressure changes.

g. Prevention of DCS

Controlled depressurization is the key to the prevention of decompression sickness. Diving tables exist (eg. U.S. Navy tables) which require a specific rate of pressure change (eg. so many feet per minute ascent from a dive) as well as "stops" for specified periods at specific depths (pressure levels) during ascent from a dive. Use of these tables is mandatory and is meant to allow a controlled off-loading of nitrogen from the tissues, into the blood and thence into the lungs, such that the risk of bends is reduced to less than 3-5%. These tables were derived empirically, that is, based on data from many dives in which various decompression profiles were used and the relative risk of decompression sickness was determined. A further decrease in risk can be obtained by over-estimating the amount of exposure to pressurization (depth and time), and by breathing 100% oxygen during one or more decompression stops (this increases the diffusion gradient for nitrogen both from the tissues to the blood and from the blood to the alveoli of the lungs). During chamber dives, the chamber operator and diving physician select the decompression table (ascent protocol) to be used.

3. Arterial Gas Embolism

This is a fairly rare, but sometimes lethal condition in which gas bubbles form in the arterial circulation; these bubbles can obstruct blood flow to the heart ("heart attack") or brain ("stroke"), producing emergency situations and/or death. The usual cause is a rapid decompression with the glottis closed

(ed. breath-holding on ascent), such that the air trapped within the lung space, increasing as it does in volume on depressurization, bursts through the lung membranes and enters the arterial circulation there. In this context it is important to remember that the volume of a unit amount of air doubles as pressure goes from twice normal to normal (atmospheric) pressure, eg., as a diver ascends from 33 feet of seawater to the surface. Evidence of gas embolism is usually dramatic, with the commonest presentation being the unconscious diver on the surface, who has lost consciousness on ascent due to brain damage or a heart attack and who is therefore also at risk of drowning. In the conscious diver, evidence includes impaired alertness or thinking, deficits in movement, speech or sensation, or a symptom complex including chest pain (left of central), shortness of breath and nausea. Treatment includes resuscitation immediately (hence the need for persons trained in cardiopulmonary resuscitation to be on the scene during any hyperbaric exposure -- including recreational dives), 100% oxygen and transport in a head-down position (reduces bubble circulation to the brain) to the nearest hyperbaric treatment facility (hence the need for divers to know in advance where such facilities are and how transportation can be acquired). At S.F.U., we are able to initiate resuscitation and to undertake treatment by recompression within the hyperbaric chamber. The best form of treatment in this and all other conditions is prevention, by proper breathing techniques on ascent and by the exclusion from hyperbaric exposure of all individuals who are known to be predisposed to gas embolism. These people include anyone with a history of surgery in which the chest wall was opened (eg. bypass grafts) as well as people with a known abnormal communication between the venous circulation by which venous gas bubbles can pass into the arteries (eg. interventricular septal defect) or with known obstructive lung disease (chronic bronchitis, emphysema, asthma, etc). People with asthma, for example, must not dive because they run a high risk of gas trapping in a pulmonary segment at depth, due to airway spasm and mucous secretions, with a consequent risk of "burst lung" and gas embolism on decompression. Although periodic chest x-rays with full inhalation and exhalation can help determine which individuals should be excluded from diving, some underlying conditions cannot be determined clinically, so that risks cannot be reduced to zero.

4. Hypoxia or Anoxia

Periods of inadequate or zero oxygen supply to bodily tissues can lead to permanent injury or even death. Equipment failure or other accidents, both underwater and in the hyperbaric/hypobaric chamber, can potentially cause hypoxia or anoxia, for which the treatment is clearly the restoration

of adequate oxygenation. Prevention requires proper maintenance and use of equipment, with special attention to equipment status before use (eg., the check-list the chamber operators use) and to the provision of adequate back-up equipment as well as an emergency protocol which is rehearsed and understood beforehand. Individuals certified in cardiopulmonary resuscitation mst be present during exposure.

5. Drowning.

This special hazard of open-water diving occurs most often as a result of equipment failure or misuse, or as a result of unconsciousness due to one of the other risks listed above. Inadequate equipment preparation and maintenance, sudden weather changes, solo diving, panic, fatigue and hypothermia all contribute to the risk of drowning. Cardiopulmonary resuscitation and transport to a treatment centre are the immediate needs.

6. Oxygen toxicity.

Oxygen is a direct toxin to tissues when present in concentrations significantly higher than normal (about 0.21 atmospheres of pressure). Nerve tissue and the lungs are particularly sensitive. Lung toxicity develops gradually, during exposure to hyperbaric oxygen for hours or days. Toxicity varies directly with the oxygen partial pressure and the duration of exposure, and manifests initially as a measurable decrease in vital capacity (reflecting in part a decreased elasticity of the lungs). This decrease is reversible once the hyperbaric oxygenation has ceased.

Oxygen toxicity to the nervous system develops much more rapidly (sometimes within minutes), producing in the extreme a grand-mal type of convulsions which may or may not be preceded by warning signs such as twitching of facial muscles, nausea, numbness and tingling sensations, dizziness, confusion or shortness of breath. The risk of convulsions varies directly with oxygen partial pressure, though there is great inter-individual variation in susceptibility and the risk is increased with exercise and with elevated temperature. Treatment includes basic resuscitation (airway, breathing, circulation and disability), discontinuation of hyperbaric oxygenation (as soon as that can safely be done) and, occasionally, the administration of medications by a physician. The convulsions stop once hyperbaric oxygenation is discontinued and there are apparently no long-term effects of the convulsions, nor is there an increased risk of epileptic seizures outside the hyperbaric environment in these individuals. Those individuals who have a history of

epilepsy, however, must not undertake hyperbaric exposure, both because of the strong likelihood of severe injury or death in the event of seizure onset "at depth" and because of a probable increased risk of oxygen convulsions in these individuals, rendering hyperbaric oxygenation treatment of diving-related accidents problematic.

It should be stressed that it is the partial pressure of oxygen rather than its percentage composition in the gas mixture that is critical in oxygen toxicity. Thus, breathing 40% oxygen in nitrogen at 6 atmospheres of pressure (partial pressure equals 2.4 atmospheres) could cause convulsions in some individuals who would not succumb breathing 100% oxygen at 2 atmospheres of pressure (partial pressure equals 2.0 atmospheres).

7. Hypothermia

Of special concern in open-water diving in B.C., hypothermia is defined as a lowering of the body core temperature. Mild hypothermia (core temperature in the range of 35-36°C) is generally well tolerated; further decreases in core temperature are associated with impairment of cognitive function (planning, making judgments, responding to emergencies), decreased psychomotor ability, decreasing level of consciousness progressing to loss of consciousness and ultimately death due directly to hypothermia (ventricular fibrillation, or cardiac muscle contraction in an asynchronous manner that fails to pump blood, occurs at temperatures below 28°C) or to drowning related to loss of consciousness. Prevention in cold waters, other than limiting the time of exposure, requires the use of a drysuit or a wet suit at least 3/8 of an inch thick. Treatment requires rewarming, which on the scene in open water diving generally starts with body-to-body heat transfer (eg. the victim and companion in skin contact in a sleeping bag), as well as basic CPR, with urgent transfer to a medical facility for further treatment and follow-up.

8. Fire Hazards

Fire may occur whenever combustible materials are brought into contact with oxygen, especially hyperbaric oxygen and especially in the presence of sparks. Work in the hyperbaric chamber requires strict adherence to the regulations banning smoking and prohibiting the use of equipment which may produce sparks. Work in the dry chamber requires the wearing of flame-resistant suits and the elimination insofar as is possible of the accumulation of flammable materials inside the chamber. The inside and outside operators must be familiar

with the fire-fighting apparatus, which must be kept in working order.

The above material describes the major risks and hazards associated with hyperbaric exposure of short duration in a chamber and with hyperbaric exposure both in the chamber and in open water. Before agreeing to undertake such exposure it is your responsibility to become informed of the risks and hazards well enough to be able to give truly informed consent to such exposure.

* *

I have read and understood this document.

Name _____

Signature _____

Date _____

Appendix V

Informed Consent

Environmental Physiology Unit
School of Kinesiology
Simon Fraser University

Note: The University and those conducting this project subscribe to the ethical conduct of research and to the protection at all times of the interests, comfort and safety of subjects. This form and the information it contains are given to you for your own protection and full understanding of the procedures, risks and benefits involved.

Your signature on this form will signify that you have received the document described below regarding this project, that you have received adequate opportunity to consider the information in the document, and that you voluntarily agree to participate in the project.

Having been asked by Dr. Igor Mekjavic of the School of Kinesiology of Simon Fraser University to participate in a research project experiment, I have read the procedures specified in the document entitled:

Subject Information Package:

1) The effects of post-dive warming on vascular gas emboli production.

2) Risks during exposure to hypo/hyperbaric conditions.

I understand the procedures to be used in this experiment and the personal risks to me in taking part.

I understand that I may withdraw my participation in this experiment at any time.

I also understand that I may register any complaint I might have about the experiment with the chief researcher named above or with Dr. J. Dickinson, Director of the School of Kinesiology, Simon Fraser University.

I may obtain a copy of the results of this study, upon its completion, by contacting Dr. Igor Mekjavic or Neal Pollock.

I agree to participate by completing the chamber dive, post-dive immersion period and subsequent observation period while remaining at rest as described in the document referred to above, during the period of 05/87 to 06/87 at the Environmental Physiology Unit

(School of Kinesiology) at Simon Fraser University.

Name (Please Print) _____
Address _____
Signature _____
Date _____
Signature of Witness _____

Once signed, a copy of this consent form and a subject feedback form should be provided to you.

Appendix VII

Dive Questionnaire

Name: _____

Profile: _____

Date Dived: _____

1. In the 24 hours prior to the dive did you participate in physical exercise? Yes No

2. If so, what did you do?

3. Is this a regular activity? Yes No

4. Did you injure or stress any part of your body? Yes No

5. If so, please describe the injury or stress.

6. What medication, if any, did you take during the 24 hours prior to diving?

7. What was your alcohol intake during the 24 hours prior to diving?

8. If you are a smoker, how many cigarettes/cigars/pipefulls did you smoke in the 24 hours prior to diving?

9. If you dove previously in the series, did you have any unusual feeling of fatigue or mood in the 24 hours following your last dive? Yes No

10. If so, please describe these feelings, and give the date of the dive.

B. Glossary

Glossary

Absolute Pressure - the sum of barometric and hydrostatic pressures

Atmospheres Absolute (ATA) - see 'Absolute Pressure'

Bounce Dive - short, non-saturation dive beginning and ending at the surface

Bubble - physical formation caused by decompression resulting in supersaturation of tissue and /or vascular nitrogen

Bubble Grade/Score - assigned rating of vascular gas emboli (bubble) severity, usually determined by some variation of the Kisman-Masurel (K-M) Code

Caisson - watertight pressure chamber used for underwater construction

Cavitation - formation of a phase separation (bubbles) due to a localized partial vacuum in a liquid. Ultrasonic energy may cause cavitation in the body by producing transient but substantial reductions in the local fluid pressure. Supersaturation, pressure, and temperature can affect the growth rate of cavitation bubbles

Decompression Sickness (DCS) - condition caused by too rapid a reduction in pressure and having a great variety of signs and symptoms. Synonyms: bends, caisson disease, compressed air illness. Forms include:

> Cutaneous - skin symptoms may include itching, rash, discoloration and/or swelling
>
> Mild ('Pain Only', Type I) - most common symptoms may involve localized pain (usually in the joints) and extreme fatigue
>
> Neurological (serious, Type II) - common symptoms may involve central nervous system (whole or partial paralysis, loss of consciousness, disorientation), or sensory systems (vertigo [extreme dizziness/disorientation], nausea, tinnitus [ringing in ears], blurred vision)
>
> Pulmonary - common symptoms include shortness of breath, non-productive cough, and rapid heart rate
>
> Dysbaric Osteonecrosis - long term complications of DCS affecting the skeletal system; results in juxta-articular (associated with joints) or medullary (shaft) bone lesions

Decompression - in diving, a phase when the pressure is being reduced

Diffusion - process in which particles of liquids, gases, or solids intermingle as the result of movement caused by thermal agitation and, in dissolved substances, move from a region of higher to one of lower concentration

Dive - exposure to hyperbaria and subsequent return to normal ambient pressure resulting in phases of compression and decompression

Doppler - process for evaluating moving reflective objects, in diving, gas bubbles in the blood

Dysbarism - term denoting any pathological condition caused by a change in pressure; includes but is not synonymous with DCS

Equivalent Ocean Depth - the depth in seawater resulting in an equivalent post-dive pressure differential to that achieved following a dive completed at ambient pressures less than sea level (ie. altitude)

Excursion Dive - movement of a diver either vertically (up or down) or horizontally from the work platform, bell, chamber, or habitat, usually during a saturation dive

FSW/fsw - depth of sea water in feet

Gas Phase Separation - formation of physical bubbles by previously dissolved gas

Half-Time - the time required to reach 50 percent of the final state; in diving, the time required for a tissue to absorb or eliminate one-half the equilibrium amount of inert gas

Kisman-Masurel (K-M) Code - a three dimensional classification system evaluating doppler detected bubbles; considers frequency, amplitude, and percentage (or duration); combination of the three parameters yields a single bubble grade/score

MSW/msw - depth of sea water in meters

Perfusion - flow of blood (or lymph) through an organ or tissue

Saturation Dive - dive of such a duration that no more gas can be absorbed into the tissues of the body. The tissues and the free gas in the environment are in equilibrium

Tissue Half-Time - see Half-Time

Total Bottom Time (TBT) - time of compression plus time at depth

Venous Gas Emboli (VGE) - see 'Bubble'