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Dr Simon Mitchell, MB ChB, Dip DHM, who is the Vice-President of the Underwater and Hyperbaric Medical Society, was Director of the Slark Hyperbaric Unit at the Royal New Zealand Navy Hospital, Naval Base, Auckland, New Zealand. This work was part of his PhD research program. He is now Medical Director of the Wesley Centre for Hyperbaric Medicine, Sandford Jackson Building, Chasely Street, Auchenflower, Queensland 4066, Australia. Telephone + 61-7-3371-6033. Fax 61-7-3371-1566. E-mail smitchell@wesley.com.au .

Ora Pellett, MSc, is a research psychologist at the University of Auckland, New Zealand. She is completing a PhD in Psychology in which she is studying the neurocognitive effects of cardiac surgery and their modification by protective strategies.

Professor D F Gorman FAFOM, PhD, is Head, Occupational Medicine, Faculty of Medicine and Health Sciences, University of Auckland, New Zealand. He is a Past-President of SPUMS. His address is 52 Albert Road, Devonport, Auckland 9, New Zealand. Telephone + 64-9-373-7599. Fax + 64-9-308-2379. E-mail d.gorman@auckland.ac.nz .

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THE EFFECT OF BUBBLES ON THE LIVING BODY

Alf Brubakk

Key Words
Bubbles, decompression illness, physiology.

Introduction

There is general agreement that the basic problem in decompression is gas coming out of solution and forming a gas phase. However, it is also well known that a considerable number of bubbles can be formed without any acute signs or symptoms. Such bubbles have been called "silent" bubbles[1] and have, in particular, been observed in the pulmonary artery.[2] One conclusion that can be drawn from this observation is that acute clinical symptoms are critical dependent upon the location of the bubbles. Bubbles in the brain, for instance, could give few symptoms, as large areas of the brain are clinically silent. Bubbles in joints, on the other hand, would give symptoms, because of the rich innervation by pain receptors in these areas. One effect of this would be that we have to distinguish between primary and secondary effects of bubbles. The primary effects are related to the mechanical effect of the bubbles, which may be blockage of the circulation or distortion of tissue. The secondary effects are related to the numerous effects of the bubble surface, with activation of a large number of biochemical and cellular mechanisms. It seems obvious that this secondary effect can occur without any acute signs or symptoms.

When do bubbles form ?

Most, if not all, practical decompressions will lead to some degree of gas bubble formation in the organism

and the risk of decompression illness (DCI). The predominant theory about the growth of bubbles is that bubbles grow from preformed nuclei, as the resistance of "pure solutions" to supersaturation and gas phase development is considerable.[3] One likely theory is that the nuclei are composed of small (approximately 1 micron) stable gas bubbles.[4]

The exact threshold for bubble formation is not known, but it is probably in the range of 50-70 kPa in tissue[5] and even lower in the vascular system. Eckenhoff et al. demonstrated that saturation at 3.7 msw on air was sufficient to produce bubbles in the pulmonary artery in man.[6] The conclusion from this study must be that gas bubbles will form in the vascular system at any supersaturation and that the concept of a minimum tolerable limit of supersaturation, as least for the venous system, may only relate to clinical symptoms and not to bubble formation. Adding to this problem is the fact that it has been demonstrated repeatedly that large inter-and intra-individual differences in bubble forming "ability" exist. Factors like sex, age, body build, circulation, temperature, blood composition and degree of exercise seem to play a role.[7,8] Cavitation in joints, for example, has been demonstrated without any supersaturation following violent movements. Even under experimental conditions where the circulation is kept stable, a variation in vascular bubbles between individuals, often a factor of 10, following decompression is observed.[9] Furthermore, there are data indicating that there is a large difference is susceptibility to decompression sickness that is not directly related to the amount of vascular gas bubbles observed.[10]

Where do bubbles form ?

Bubbles have been observed in many tissues in the body following decompression. They are most commonly found in the vascular system, the white matter of the CNS, in abdominal fat, in synovial fluids and in muscles.[11] Following quite severe decompressions, we did not observe bubbles in the muscles themselves, but only on fascia.

Harvey et al. studied the limits for vascular bubble formation in cats, both at rest and after electrical stimulation and tissue injury.[12] The conclusion from these studies was that at marginal exposures, stimulation or injury was needed for bubble formation. At higher supersaturations, bubbles occurred at rest, the time of occurrence determined by the fat content. Essentially the same results have been obtained in frogs and rats.[13] Based on these studies, the authors concluded that gas bubbles are chiefly intravascular and that they are responsible for nearly all important phases of the syndrome of decompression sickness. Only in very severe cases did extravascular bubbles play a role and then only in lipid rich structures.

Venous bubbles

There is evidence from many studies that gas bubbles occur in the venous system during most decompressions.[14,15] Several studies have documented the relationship between the occurrence of many venous bubbles and the risk for clinical symptoms requiring treatment.[16,17] This, together with the fact that bubbles probably are present in the venous system during most decompressions, suggests that a diver complaining of pain in a joint may be suffering from two different conditions, namely tissue gas in and around the joint and pulmonary gas embolism.

Arterial bubbles

Gas bubbles in the arteries have been detected in divers after excursions,[18] during decompression from saturation dives[19] and at autopsy after fatal accidents.[20] Arterial gas bubbles have also been observed in large animals during and after decompression.[21-23] Thus, there is no doubt that arterial gas bubbles occur during or after some decompressions.

In divers, there are several possible pathways by which venous bubbles may reach the arterial circulation. First, venous gas bubbles may travel through the pulmonary circulation and enter the pulmonary veins and the left atrium, although the pulmonary circulation is usually considered to be a good filter for gas bubbles as well as for other emboli. Second, venous gas bubbles may pass through a patent foramen ovale (PFO) or other extraordinary connections in the heart to reach the left side of the heart. Third, if the lung has been overinflated during a rapid ascent, gas may escape directly into the pulmonary veins after alveolar rupture.[24]

Finally, gas bubbles may form in the arterial circulation if the decompression rate is sufficiently fast >0.3-1 fsw/sec.[25] All gas nuclei in the blood will not be destroyed at compression and supersaturation of the arterial blood may occur during the rapid decompression. However, an experimental study using goats did not succeed in demonstrating such bubbles in the arterial circulation after a short hyperbaric exposure and a rapid decompression.[26]

In as many as 20-34% of humans, dependent on age, the foramen ovale is patent after foetal life.[27] Normally it is functionally closed, since the pressure in the left atrium is higher than the pressure in the right atrium and the septum primum functions as a valve. However, a spontaneous shunt, not dependent on a Valsalva manoeuvre or other factors to change the pressure gradient between the atria, is diagnosed in 5-6% of humans using contrast echocardiography.[28,29]

Tissue bubbles

It seems reasonable to assume that tissue bubbles can occur if the gas load is high enough. This was apparently shown for the spinal cord by Francis et al., who showed tissue bubbles in the white matter following rapid decompression after a 15 minute dive to 300 fsw on air.[30] This work has, however, recently been challenged by Palmer, who claims that all changes observed could be explained by gas bubbles inside vessels.[31] This is also supported by the recent work of Sharpe and Broome, who showed that there was no relationship between the fat content of the spinal cord and the occurrence of gas bubbles.[32] Even if the exact mechanism of tissue injury is controversial, the evidence seems to indicate that vascular processes are the more important ones.

Primary bubble effects

Initially, the bubbles will lead to changes, mainly due to their direct mechanical effects. When gas bubbles form and expand they can obstruct the arterial and venous circulation, leading to tissue ischaemia, or they can damage the tissue and induce pain by direct pressure effects. Bubble formation may influence circulation by mechanical obstruction. Venous obstruction may lead to oedema and arterial obstruction may lead to tissue ischaemia, both of which have been observed after decompression.[33] This can reduce gas elimination, both by increasing diffusion distances and by reducing blood flow.

One important primary effect of the bubbles, which is often forgotten, is the reduction in gas elimination caused by these bubbles. Both theoretical[34] and experimental[35] studies have demonstrated that gas bubbles in the tissue will increase gas elimination time. In another study, it was shown that bubbles significantly increased the time constant of the slow component of the bi-exponential curve describing the nitrogen concentration in the pulmonary artery.[36] This can be seen in Figure 1.

Obstruction of flow to the tissue by bubbles may increase elimination time even more. Computer simulations have shown that a high number of bubbles can increase the time constant of gas elimination from muscles from 50 to 2,000 minutes (Flook, Personal communication 1999). This is partly taken into account by the new US Navy (USN) diving tables, where gas elimination is considered to be linear, not exponential.[37] However, in reality the problem may be even more complex as bubbles in the circulation may increase the transport of gas to the lungs.[38]

Secondary bubble effects

When gas bubbles are formed protein denaturation takes place at the blood-gas interface.[39] The gas-blood

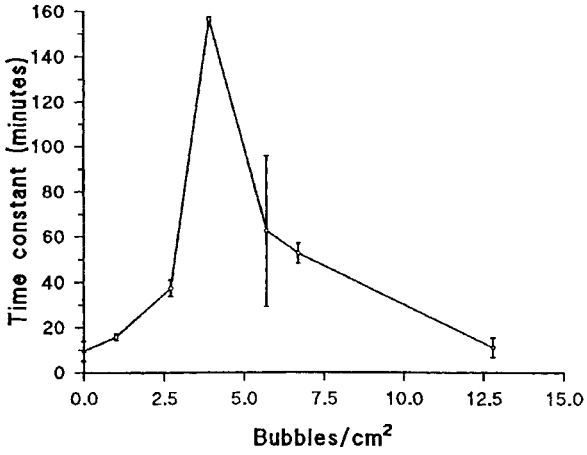

Figure 1. The relationship between the number of bubbles in the pulmonary artery and the time constant for the elimination of nitrogen from the pulmonary artery. Mean values with 95% confidence intervals.

interface is a thin layer, approximately 20 nm thick, consisting of fibrin and gamma globulin.[40,41] This layer acts as a foreign substance, activating formed elements of the blood and inducing biochemical changes such as complement activation.[42] These mechanisms play a significant role in the response of the tissue to venous gas emboli and are probably the basis for any long term effects that may occur. At present, our understanding of the importance of these mechanisms is quite fragmentary. Much further work is needed to determine the relative influence of mechanical and biochemical effects of gas embolism. An understanding of these processes is necessary for development of a rational approach to treating or preventing injury caused by vascular bubbles.

Inflammation

The response of an organism to injury is termed inflammation. In decompression sickness this inflammatory process is initiated by the surface of the bubbles, which is regarded by the organism as a foreign substance. However, this process can also be initiated by direct mechanical injury to the tissue. One important mediator of the inflammatory process is activation of the complement system. Gas bubbles activate the complement system in-vitro.[43] The degree of activation is dependent upon the amount of gas infused, varies considerably over several months in one individual and is not dependent upon the gas composition of the bubble. No relationship was seen between the degree of C5a activation in vitro and the level of C5a observed in vivo after air dives.[44]

Leucocytes are involved in many aspects of tissue injury and inflammation. Several studies have documented that leucocytes are activated by decompression. Philp et al.[45] showed that decompression led to a reduction in both

thrombocytes and leucocytes. These reductions seem to be related to the presence of gas bubbles. During decompression from a saturation dive to 440 msw, where no gas bubbles could be detected, there was no reduction in leucocyte number.[46] In another saturation dive, Benestad et al.[47] showed that decompression lead to activation of neutrophils. Contrary to what has been observed in thrombocytes, no activation of leucocytes seems to occur in vitro at pressure.

Coagulation

Aggregation of thrombocytes may lead to the formation of blood clots, thus forming solid emboli which may compound the effect of gas bubbles in the lung. Thorsen et al.[48] showed that gas bubbles lead to aggregation of thrombocytes in-vitro. Aggregation in-vitro was strongest when the bubble diameter was between 40 and 120 μm. The degree of aggregation does not seem to be dependent upon the gas content of the bubble, but only on its surface properties.[49] Aggregation of thrombocytes by gas bubbles can be considerably enhanced by adrenaline (epinephrine).[50]

Vasoconstriction/dilatation

Gas bubbles can induce vasoconstriction by direct effects on the vascular wall[51] or vasodilation or constriction by initiating the release of different vasoactive substances.[52,53]

Bubble effects on different organ systems

In the following the bubble effects will be described on some organ systems. Primary and secondary effects will be described together, as they often are quite difficult to distinguish.

Endothelial damage

Chryssanthou et al. have shown that animals exposed to decompression will show breakdown of the blood-brain barrier and the blood-lung barrier.[54] Broman et al. have demonstrated that even very short contact between gas bubbles and endothelium (1-2 minutes) will lead to such breakdown.[55] Furthermore, studies in rabbits indicate that such contact leads to endothelial damage and progressive reduction on cerebral blood flow and function. In a study by Smith et al.[56] endothelial damage could be demonstrated in pigs exposed to severe decompressions. We were able to demonstrate changes in the endothelium in pigs following exposure to gas bubbles. We found that these changes occurred at an exposure of approximately 1.5 bubbles/cm^2, equivalent to approximately Grade II-III on the Spencer scale.[57]

Even minimal endothelial injury can induce activation of both biochemical and cellular responses, which could form the basis for tissue injury following decompression.

Lung function changes

Gas emboli may block some parts of the pulmonary vascular bed, reducing or preventing blood flow through the regions of lung served by those vessels. This leads to an initial rise in the pulmonary artery pressure (PAP) and pulmonary vascular resistance (PVR), and a decrease in arterial oxygen tension (P_aO_2).[9,58,59] Following decompression, changes in diffusion capacity[60] and lung function changes similar to "small airways disease" have been seen.[61]

Central Nervous System changes.

Central nervous changes in DCI are probably caused by several mechanisms. In severe DCI, both vascular bubbles and in-vivo bubble formation probably plays a role.[33]

Exposure to vascular bubbles without clinical symptoms do not seem to have a serious effect upon the spinal cord.[62] In this group of 10 amateur and 10 professional divers, five of whom had suffered from DCI, no changes could be seen. In the brain, changes in the endothelial layer of the ventricles could be detected in a group of divers.[63] A possible explanation is that this damage is caused by gas bubbles in the spinal fluid, such bubbles will probably primarily adhere to the lining of the ventricles.

Numerous studies have shown that circulating gas bubbles change the blood-brain barrier, this is described above.

Bone

Aseptic bone necrosis is regarded as an occupational hazard for all workers under pressure.[64] There is clear indications that the incidence of bone necrosis is linked to decompression. This is perhaps best demonstrated by the fact that this disease, which is quite rare in the industrial world, is considerably more prevalent in the developing world, where diving practices produce a high incidence of decompression sickness.

Conclusions.

A recent consensus conference on long term health effects of diving[11] concluded that "changes can be seen in

lungs, CNS and bone in divers who had not had any decompression accidents", or perhaps more accurately, in divers who had not been treated for decompression illness. The mechanisms for these changes are not clear, but as bubbles in the vascular system have been observed frequently in divers, a reasonable working hypothesis would be that vascular bubbles may play a major role in this. There are still many details missing how the bubbles affect the organism. An understanding of the mechanisms involved is important, however, both for preventing injury and for treating the damage caused by these bubbles.

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Dr Alf O Brubakk was one of the guest speakers at the 1999 SPUMS Annual Scientific Meeting. His address is Department of Physiology and Biomedical Engineering, Medical Faculty, The Norwegian University of Science and Technology, Trondheim, Norway. Telephone +47-7359-1005. Fax +47-7359-8904.
E-mail Alf.O.Brubakk@medisin.ntnu.no.

37 DECOMPRESSION SICKNESS CASES TREATED IN THE DEPARTMENT OF UNDERWATER AND HYPERBARIC MEDICINE, ISTANBUL FACULTY OF MEDICINE

Akin S Toklu, Samil Aktas, Salih Aydin and Maide Çimsit

Abstract

The time interval between the onset of decompression sickness (DCS) and recompression therapy, and the first aid with medical treatments applied before reaching a recompression facility, will affect the outcome of the recompression therapy.

In this study 37 DCS cases were evaluated to find out the time interval to the onset of DCS, the type of the disease and symptoms, delay to recompression treatment, medical treatments applied during transport, recompression treatment protocols performed and outcomes.

All the patients were male. Professional divers (32 or 86.5%) outnumbered sports divers (5 or 13.5%). In 20 cases (54.1%) onset of symptoms was within the first 10 minutes after the dive. In three men (8.1%) symptoms came on underwater. Numbness, tingling and back pain were the most frequent symptoms reported by the patients with Type II DCS. Complete recovery was achieved in 32 (86.5%) of the cases by recompression therapy combined with medical treatment. Rehabilitation was needed in 12 (32.4 %) of the cases.

Omitted decompression was the most frequent cause of DCS in our cases. Additional hyperbaric oxygen therapy needed in delayed cases is evidence of the importance of immediate transport and adjunctive medical treatments.

Key Words

Decompression illness, first aid, transport, treatment.

Introduction

The hyperbaric facilities in Turkey are mostly situated in Istanbul. The only Hyperbaric and Underwater Medicine Department in civilian universities is in the Istanbul Faculty of Medicine. The Fisheries Research Institute also has a hyperbaric chamber for treating divers in Bodrum, in Aegean Sea region. The Turkish Navy has three hyperbaric units. Besides these public facilities, all with multiplace chambers, hyperbaric oxygen therapy is performed in three private hyperbaric centres, in Istanbul. The three private hyperbaric centres, which all have multiplace chambers and one also has a monoplace, are free standing and mostly use hyperbaric oxygen therapy for indications other than diving related disease. In March