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DOPPLER BUBBLE DETECTION AFTER HYPERBARIC EXPOSURE

Margaret Walker

Abstract

A review of the literature on the use of transcutaneous Doppler to detect circulating venous bubbles occurring after hyperbaric exposure, with emphasis on the detection of bubbles occurring after relatively small decrements in pressure, is presented. The correlation between circulating bubbles and the occurrence of decompression illness is examined.

Key Words

Bubbles, decompression illness, investigations.

Introduction

Decompression illness (DCI) may occur in many different organisms following a reduction in ambient pressure exposure. The illness is thought to develop as a result of the formation of an endogenous gas phase taking the form of small inert gas bubbles which are widespread throughout the blood and body tissues.[1] The symptoms produced by these bubbles will depend on their size, number and location. Gas bubbles in the microcirculation or moving in the venous circulation may apparently produce no clinical symptoms, whereas a bubble of similar size in the tissues may produce symptoms due to tissue distortion or damage, especially in the nervous system.[2,3] Circulating venous bubbles indicate that gas phase separation has occurred, and that bubbles may exist elsewhere in the body tissues.[4] The growth of gas bubbles in the tissues by gaseous diffusion may produce symptoms some time after the initial decompression has occurred.[2,3] Gas bubbles normally appear and grow after decompression. Rapid decompression or a large gas load, or both, leads to the earlier appearance of bubbles.

The magnitude of the decrement in pressure exposure which can be safely tolerated by humans is of fundamental importance in the field of hyperbaric medicine, where patients and attendants alike are exposed to elevated ambient pressure during routine treatment profiles. The treatment regimes currently in use throughout the world are considered to be safe in that the incidence of DCI in the attendants is negligible, though not zero. However, it is not known if small asymptomatic bubbles may be occurring during these exposures which may cause morbidity in the long term, especially where the attendants have repeated hyperbaric exposure (in some centres more than once daily).

At present, there is no reliable way to detect stationary bubbles located in the body tissues, and although pulse-echo ultrasonic imaging has been proposed for this purpose based on in-vitro studies, it has not gained widespread use in-vivo as it is only possible to examine one region of the body (eg. one limb), the subject must be totally still, and the equipment is large and bulky.[5,6] However, circulating bubbles can be relatively easily detected in the venous and arterial circulation using Doppler ultrasound. This provides a means for assessing whether gas-phase separation has occurred during decompression.

This paper examines the current understanding regarding the use of Doppler ultrasound to detect circulating bubbles and considers their relevance to DCI.

Classification of decompression illness

Interpretation of the studies carried out to date attempting to correlate the incidence of decompression illness to Doppler-detected bubble grade is made difficult by the different classifications of decompression illness used in different establishments. For the purpose of this review, the recent classification developed by the Royal Navy Institute of Naval Medicine workshop at Alverstoke, United Kingdom, in October 1990, will be used.[7] This classification is based on a clinical description of the illness. The term "decompression illness" is used to include both the previous "decompression sickness" and "cerebral arterial gas embolism". The term is prefaced by an evolutionary term (static, resolving, relapsing, progressive) and secondly by the organ system involved, with no attempt to grade the symptoms into a severity hierarchy. For example, a diver who collapsed on surfacing, was initially unconscious and then recovered, would have " resolving neurological decompression illness" and a diver with unchanging shortness of breath and paraplegia would have "static pulmonary and neurological decompression illness".

This new system does not try to classify the decompression illnesses into arbitrary grades of severity as with the previous unsatisfactory "decompression sickness" (DCS) Types I and II.[8] By describing the illness in clinical terms it is less open to variation in interpretation between observers.

Much of the published literature predates this new classification, and previous investigators have used the old Type I and II classification of DCS, with Type I being "mild", Type II being "serious" and many cases not fitting into either Type described as "unclassified".[2,8] This makes interpretation of the available data more difficult, as many symptoms and signs which would now be recognised as manifestations of decompression illness (such as neuropsychiatric changes) were not sought because they did not fit easily into the existing classification. Although more subtle deficits in higher mental function are formally included in the category of "Type II DCS" as manifestations of neurological DCS,[8] they have not been specifically sought in most studies prior to the reclassification.[9-12] This may indicate that cases of DCI were not diagnosed as such unless there were obvious symptoms, such as joint pain, and that many cases of a more subtle nature were overlooked.[4,13,14]

Thus in interpreting the results of studies to date, this possibility should be borne in mind. There is no retrospective way to ascertain if cases of DCI were overlooked. The reported incidence of DCI may therefore be an underestimate.

Doppler ultrasound in hyperbaric medicine

Behnke, in 1942, was the first to propose that silent (asymptomatic) bubble formation occurred during rapid ascent to altitudes of 6,000-8,000 m.[15] The development of medical ultrasound led to the availability of Doppler ultrasound detectors. Spencer and colleagues first detected decompression gas emboli in the arterial and venous blood of sheep following decompression from a 65 m 60 minute exposure to air and venous bubbles were shown to occur before the first stop recommended by the US Navy Exceptional Exposure Tables.[16]

At first it was difficult to detect bubbles in human subjects because the peripheral Doppler detectors could only be located over peripheral veins, and hence only a portion of the venous return was being monitored. A catheter-tip Doppler was developed for implantation into the vessels of humans, but did not gain wide acceptance due to its invasive nature.[16]

The first unequivocal bubble signals were detected by Spencer in 1969 by an external sensor placed over the brachial vein of a subject with DCI involving symptoms in the same arm. These were enhanced by local tissue manipulation, and disappeared after treatment of the DCI with recompression.[16] This finding established that Doppler ultrasound could be used during recompression treatment to optimise both the necessary extent of recompression and subsequent decompression schedule on the basis of the frequency of vascular gas bubbles detected.

It was realised at this time that improved bubble detection could be achieved if a sensor could be developed to detect bubbles flowing in the right ventricular outflow tract and pulmonary artery. The first precordial ultrasonic bubble detector was developed by Spencer et al. in 1971.[17] It incorporated a transducer which focused deeply on the large vessels and heart, thus eliminating confusion from gas emboli signals in the blood vessels of the chest wall itself.

The Doppler ultrasound probes now in common use for the detection of circulating bubbles emit 2.5 MHz continuous-wave signals using two probes of different focal length designed for use on the precordium and the peripheral vessels. The signals obtained can be recorded directly onto audio tape for later analysis.

Clinical significance of Doppler-detected bubbles

Although the ideal decompression would result in no bubble formation, it appears that some degree of gas phase formation occurs in virtually any decompression, as Doppler-detectable bubbles are found in all decompression profiles from short bounce dives to slow, saturation-dive decompressions.[1,13,15] Many of these cases have no symptoms of DCI despite the presence of significant bubbling, which throws doubt on the premise that the presence of detectable venous bubbles equals decompression illness.

Bubble formation not only alters gas elimination rates, but also may lead to DCI.[12] However, any gas remaining in solution following decompression will not of itself produce damaging effects.[15]

Following decompression, bubbles will form early in those tissues most saturated with nitrogen, and as they enlarge and exert greater pressure on the blood vessel walls, these bubbles may enter small veins and capillaries. Spontaneous intravascular bubbles tend to form preferentially in the venous circulation as the blood here is quickly saturated with gas released from the tissues at the capillary level, and the intravascular pressure is relatively low.[2] Bubbles also form early in the microcirculation following a reduction in ambient pressure due to the high gas tension and low intravascular pressure.[1,14] Bubbles located in the microcirculation are initially stationary and may cause tissue hypoxia due to obstruction of blood flow.[2,10] They may eventually embolise to the central venous circulation due to pressure or distortion of the tissue. Bubbles entering the systemic venous circulation are filtered by the lungs up to a maximal bubble load, after which they may completely obstruct pulmonary flow.[1,15,18] It is rare to find bubbles circulating in the arterial circulation, unless there is a right to left shunt present in the pulmonary bed or heart; but if the delivery rate of bubbles to the lungs exceeds a threshold level (0.35 ml/kg/min in dogs),[19,20] some may pass across the pulmonary bed and enter the arterial circulation.[2,3,16] Turbulent flow in the ventricles of the heart may also predispose to cavitation and bubble formation on both the right and left sides of the heart.[21]

The pathophysiology of DCI is thought to be complex and to involve both intravascular and extravascular bubbles, complement activation, histochemical and haematological changes,[3,22,23] so detection of intravascular bubbles is only an indicator that a decompression stress has occurred, not necessarily that clinical DCI will occur.[23] Susceptibility to DCI appears to be an individual trait, as some people will develop DCI with low-grade or no detectable bubbles, whereas others have higher-grade bubbles with no clinical evidence of DCI.[11,12,24,25]

Doppler-detected bubbles are not normally believed to be the cause of DCI, but their presence in the circulation indicates that asymptomatic bubbles may be present in other tissues of the body.[10,11,25] These asymptomatic tissue bubbles may cause subclinical damage which may have long-term effects, for example in the central nervous system.[4,25-28]

Doppler ultrasound mechanisms

Ultrasonic waves have the advantages of being highly directional, easily focused, useful for examining small structures due to their short wavelengths, and they show a large reflection at gas-liquid interfaces, making them very good gas phase detectors.[17] These properties make ultrasound waves ideal for detection of circulating (but not stationary) gas bubbles.[4,29]

The presence of gas bubbles in a liquid causes marked reflection of an ultrasound beam. In addition, bubbles of a given size are found to pulsate under the influence of periodic oscillations of the surrounding medium. This has been proposed as the mechanism for the sound generated by running water in streams.[15]

The transducer is the most important element in the Doppler ultrasound system, determining the operating frequency, the depth of penetration, the size of the ultrasonic beam and the frequency of the Doppler shift recorded.[17] The central element of the transducer is a piezo-electric crystal which changes its dimensions when an electric field is applied to it, or conversely will generate an electric field when it is deformed by vibration. The most sensitive frequency for operation of a transducer is the fundamental resonance frequency of the crystal element. Lower frequencies penetrate deeper into tissues, but result in longer wavelengths and low Doppler shift frequencies. Conversely, too high a frequency will not penetrate deeply enough and will produce Doppler shift frequencies too high to hear. Most Doppler systems use frequencies between 2.5 and 5 MHz, the lower frequencies being used to penetrate to the heart and great vessels, and the higher frequencies being used for more peripheral vessels.[15,17,30]

The Doppler effect is a change in the frequency of an ultrasonic wave when the transmitter, the receiver, or the scatterer are moving with respect to each other. Therefore, reflections from moving gas bubbles have a different frequency from the transmitted frequency. Ultrasonic

Doppler flowmeters only respond to reflections which have experienced a Doppler shift (eg, from moving bubbles in blood) and not to the reflections from stationary structures which have no Doppler shift. The Doppler shift frequency produced by moving gas bubbles is within the normal range for human hearing. The receiver has two inputs, the transmitted wave and the reflected wave. The output frequency of the receiver is the difference between the two inputs, so that there is only an output for the reflections from moving structures.[15]

Doppler signals coming from the receiver can be analysed aurally or displayed graphically in the form of flow velocity waveforms.[31,32] There are several schemes for aural processing in current usage, the most common being those of Spencer and Johanson[9,15] and Kisman and Masurel.[33,34]

The Doppler signal produced by a gas bubble in blood is a sinusoidal narrow band sound wave which sounds to the human ear like a chirp or whistle.[17] The amplitude of the bubble signal is nearly proportional to the radius of the bubble.[30] Due to their strong acoustic interface, bubbles in the blood and other liquids tend to scatter ultrasound more than do solid particles of the same size. The human ear is currently the most accurate signal processor for recognition of the bubble sounds, because the hearing mechanism can distinguish the chirping quality signals at low volumes superimposed on the background noise of Doppler blood flow, and can recognise the extra noises which occur sporadically as a break in the pattern of normal cardiac signals.[15,35]

Doppler bubble detectors can be either continuous wave (CW) or pulsed systems. The CW system is technologically simpler and produces an output in the audio frequency range which conveys both amplitude and frequency information without the need for quantitative calibrations. The pulsed Doppler allows “range gating” to select the penetration depth of the transmitted and received signal which narrows the sample volume and reduces background signals. However, the electronics required are currently complex and expensive and therefore most work has been done using CW Doppler.[6]

Gas bubbles present in the circulation are detectable as long as they are above the resonant size for the ultrasound wave. The minimum detectable bubble size is a function of blood velocity.[15] A bubble of 20 micrometers radius will be detected if the mean velocity carrying it is 55 cm/sec, but the minimum detectable size increases to 90 micrometers if the velocity is only 20 cm/sec. The smallest bubble detectable in the heart and large vessels should be approximately 80 micrometers in diameter, and in the subclavian veins, 10 micrometers.[11,15,30] Hence, bubbles can be easily detected in peripheral veins when they are not detected at the same time by precordial devices.

Nishi determined that small bubbles may not scatter enough ultrasound energy to be detectable above the background noise.[30] He maintained that although it is not possible to deduce the size of the bubbles in vivo, acoustical studies suggest that they must be about 50 micrometers in radius or larger for detection. Hills and Butler[36] measured the size distribution of intravascular bubbles produced by decompression and found diameters of 19-700 micrometers, so the smaller of these bubbles may not be detected by Doppler. Therefore, it is not possible to prove the absence of bubbles, as there may be smaller bubbles circulating which are not detected by the current Dopplers.

Safety considerations

High energy sound waves may affect tissues by several mechanisms. However, no observable effects have been seen to date in intact mammalian tissues from the amounts of power used in medical diagnostic and bubble detection equipment.[15] The intensity of most diagnostic ultrasound equipment ranges from 10 to 100 mWatts/cm^2. Tissue damage does not occur until the intensity reaches 1-100 W/cm^2. Thermal burns may be produced at energy levels higher than 100 W/cm^2.[15]

Also, low pressure areas in the sound wave may produce tearing in liquids, a process which leads to the formation of small cavities in the liquid.[15] This also may result in free radical production and a variety of chemical and biological effects. Theoretical predictions indicate that clinical ultrasound energy levels cannot produce cavitation in the megahertz frequency range. Cavitation has not been demonstrated at frequencies much over 5 MHz. In liquids of low viscosity (eg blood), operation of higher frequency ultrasound in short bursts prevents such cavitation.[15,17] Attenuation of the ultrasonic energy as it penetrates deeper into the tissues provides an additional margin of safety. No untoward effects have been observed in the monitoring of animals or humans by application of the Doppler detector.

Precordial monitoring

The precordial probe is placed at the left midsternal edge, where it overlies the right ventricle and pulmonary artery, and should be well placed to detect any bubbles in the venous circulation.[34] The heart sounds are very loud and clear at this point. Gas bubbles are heard as a “click”, “chirp”, “whistle”, or as a harsher, longer sound depending on their velocity and the angle at which each bubble crosses the ultrasound beam. These sounds are superimposed on the regular heart sounds as sporadic, irregular sounds of different frequency. The bubbles are graded according to the method of Kisman and Masurel (K-M).[33,34]

Monitoring is carried out for 60 seconds at rest (with the patient standing upright) and then for 30 seconds after each of three deep knee bends, the aim being to mobilise bubbles from the tissues with muscle contraction and increase the detection rate.

Monitoring the heart can be difficult as the valvular movement and any turbulent blood flow produces a loud background noise that may mask bubble signals. For this reason, monitoring is also carried out at peripheral sites where the level of background noise is minimal.

Peripheral monitoring

Veins such as the internal jugular, subclavian, inferior vena cava or femoral can be monitored, as bubble signals at these sites are unambiguous. However, they are monitored only as a supplement and not as an alternative to the precordial site, because they do not provide information from the whole body, only the region which they drain.[17]

The probe can be placed over a more peripheral vein such as the subclavian vein at the midpoint of the clavicle, or the femoral vein in the inguinal crease. The aim at these sites is to monitor the soft blowing sound of the venous flow, with the regular arterial pulse superimposed to facilitate bubble grading. A clench of the fist, or contraction of the calf muscles may release a shower of bubbles. The bubble noises are more easily heard at these sites, where there are no cardiac sounds to interfere with detection.[34]

The K-M classification is based on three parameters used to describe the bubble signal; frequency, percentage or duration, and amplitude.[34] Each parameter is assigned a value from 0-4. The frequency represents the number of bubbles per cardiac period, the percentage represents the percentage of cardiac periods at rest having a specified bubble frequency. After movement this is replaced with a duration parameter, which describes the number of cardiac periods following the movement which have a given bubble frequency. The amplitude is graded compared with the amplitude of the cardiac signal. The code is then written as a three digit number, and this is converted to an overall bubble grade I-IV using a table. The correlation of bubble grade between different observers is dependent on the experience of the respective observers.[35]

Despite numerous technical refinements over the last 15 years, the process of Doppler bubble scoring has remained essentially unchanged, with auditory processing by trained observers remaining the mainstay of coding. Butler et al. reported the use of computer-assisted digitisation to provide real-time and replay recordings of Doppler-detected bubbles in both dogs and human subjects, using both the precordial and subclavian sites.[31] They found that the production of visual representation of the Doppler signals by computer digitisation was a valuable aid to the audio-interpretation of the signals, especially when the observers are inexperienced or fatigued. It may also allow cardiac background signals to be more easily distinguished from bubble sounds.

Since it is not practical to monitor divers continuously, Doppler recordings are taken at intervals after the return to the surface. Current information suggests that observations should begin soon after return to the surface and be repeated at half-hour intervals for at least 2 hours, as the peak incidence of bubble formation appears to be between 1-2 hours after the decompression stress.[4,17,37]

Clinical correlation with bubble grade

Many studies have been performed in recent times in an attempt to determine the relationship between circulating bubbles and the clinical onset of DCI. To date, attempts to use Doppler detection methods to guide decompression in human divers have been sporadic and the scattered results difficult to analyse.

Initially, it was hoped that Doppler could be used to control the decompression profiles to prevent DCI, but the studies performed by Eatock and Nishi showed that bubbles tend to form after the decompression has been completed, first appearing up to 1 hour after the decompression stress, with the peak incidence at 2 hours after decompression.[37]

In general, the bubbles are detected before the onset of symptoms of DCI and it is almost certain that decompression-produced bubbles are produced after almost any period of compression, even in dives which produce no symptoms of DCI.

Attention is now focused on trying to predict the probability of development of DCI from bubble frequency.

Spencer studied a group of divers over a range of pressure-time air exposures with Doppler ultrasound in an attempt to determine the direct decompression (decompression to 1 ATA at 60 ft/minute, a rate of 0.3 m/sec) limits for man.[11] He found that there appeared to be a strong individual propensity to form circulating bubbles or venous gas emboli (VGE), which correlated with susceptibility to DCI. No DCI developed without prior detection of precordial VGE. VGE were even found to occur after exposures to pressures of as low as 2 ATA (10 m, 33 ft). After a 60 minute exposure at 2.8 ATA, 4 of 12 divers developed detectable VGE of grade II or greater on direct decompression, although 9 of 12 divers reported skin itching. Only one diver developed limb pains. Spencer found a trend for bubbles to be detected in the venous circulation sooner after a deep dive than after a shallow dive.

For example, after a dive to 1.9 ATA, bubbles were detected in the circulation 15-30 minutes after decompression, whereas after a 7.5 ATA dive, the bubbles were detected after 6-12 minutes.

Neuman et al. studied a group of US Navy personnel during routine hyperbaric chamber operations.[12] Thirty one dives were performed to either 65 msw or 40 msw. There were 5 cases of DCI, all associated with Doppler bubble counts of grade IV. However, Doppler bubble counts of grade IV were also recorded in 12 of the other 27 divers, none of whom had symptoms of DCI.

Powell et al., in 150 man-dives, found that precordially detected bubbles were predictive for limb pain in mixed-gas divers only 50% of the time, but 70% of the divers who developed clinical DCI had no precordially detectable bubbles.[24] They concluded that the presence of venous gas bubbles can be associated with an increased risk of DCI, but that this technique lacks the specificity required for personal dive monitoring.

Eatock collected data from Defence and Civil Institute of Environmental Medicine (DCIEM) and US Navy dives and correlated Doppler bubble grade with probability of developing DCI.[25] Many different dive profiles were used, including mixed-gas diving. In general, bubbles of grade I or less were associated with clinical symptoms of DCI in less than 2% of cases, with an increasing incidence of DCI for grades II-IV. Bubbles of grade IV were associated with a DCI incidence of 50%. However, the author concluded that although the number of intravascular bubbles is a good indicator of decompression stress, the correlation between bubble count and DCI is subject-dependent. Though the presence of bubbles does not necessarily produce symptoms, the possibility of long-term effects cannot be ruled out.

Bayne et al. carried out a double-blind, prospective clinical trial of Doppler bubble detection in simulated diving involving 83 men, of whom 8 developed DCI.[10] They found that diagnosis based only on the Doppler signals had no correlation with clinical diagnosis, but that bubble scores were slightly higher in the DCI group. However, 3 of the 8 divers with clinical DCI had no detectable precordial bubbles, constituting a false negative rate of 38%.

Eckenhoff et al. studied 34 healthy human subjects exposed to shallow air saturation for 48 hours at 1.77 ATA and 1.89 ATA, and then decompressed to 1 ATA in about 2 minutes.[13] Almost all subjects had Doppler-detectable VGE developing up to 4 hours and lasting for up to 12 hours after decompression. The reported incidence of DCI was 27% in the 1.89 ATA group, this being manifested by joint pains only. The incidence of other cases of questionable DCI was approximately 20% in each group, although the symptoms experienced by the individuals in this group were not described. It is interesting to note that the incidence of other symptoms (apart from joint pain) were higher than the reported DCI incidence. Fatigue was reported by 53% of divers, malaise by 26%, myalgias by 15%, headache by 15% and pruritus by 9%. All of these would be classified as symptoms of decompression illness by the new classification, so it is possible that the true incidence of DCI in this study may have been as high as 53%. It is also interesting that those subjects treated for DCI in this study had rapid and complete disappearance of VGE during treatment (USN Table 5), but in an average of 4 hours later, low-grade (grade I) VGE recurred in 3 out of 4 treated subjects. The recurrence of bubbles after a treatment table may indicate that the table is inadequate for treatment, and may correspond with the recurrence of symptoms often seen after the initial treatment of a diver with DCI.

Dunford et al. showed that intravascular bubbles can be detected with Doppler ultrasonic bubble detectors in up to 18% of recreational divers after dive profiles said not to require decompression stops.[38]

Eckenhoff et al. in a study of divers in an underwater habitat determined that 50% of humans can be expected to generate endogenous bubbles after decompression from a steady-state pressure exposure of only 1.35 ATA.[1] VGE were first detected within 1 hour of the decompression and continued on average for 4 hours. None of the subjects developed symptoms of DCI, not even the fatigue or pruritus seen in his earlier study.[13] He comments that substantial formation of an endogenous gas phase can occur without symptoms, although it remains to be proven that asymptomatic VGE after a decompression stress are indeed benign. His data suggested that the incidence of DCI after exposures of 1.38-1.64 ATA was <5%. He concluded that symptoms of DCI will be reported mostly in subjects with Doppler bubble grades greater than grade 3. He also comments that the reduction in absolute pressure of 26% (1.38 ATA to 1 ATA) experienced by the subjects in this study is equivalent to that experienced in ascending from 1ATA to an altitude of 2,500m, which is a pressure commonly attained in commercial pressurised aircraft, and that circulating bubbles may well be a common occurrence in people subjected to this decompression stress.

Sawatzky and Nishi examined the relationship between Doppler detected intravascular bubbles and the subsequent development of DCI in 1,726 dives.[14] They found a definite association between increasing Doppler grade and risk of DCI. Grade II or less bubbles were associated with only a 1-2% incidence of DCI, and Grades III and IV with a 6-11 % incidence of DCI. The maximum recorded Doppler grade was the best indicator for the risk of developing DCI, 90% of cases of DCI having detectable bubbles of grades III or IV. These results indicate that Doppler grade is not a good predictor of which individual subject will develop DCI, but allows an assessment of the risk of DCI to be made, based on Doppler bubble grade. They

concluded that it is necessary for intravascular bubbles to be present for DCI to develop.

The studies performed to date indicate that bubbles of less than grade II are generally not associated with articular pain, but with increasing bubble grade, the incidence of clinical DCI increases. It appears that bubbles may be a precursor of DCI and can therefore be used as an indicator end-point rather than relying on pain or other systemic disturbances for assessing the efficacy of decompression procedures. The Defence and Civil Institute of Environmental Medicine in Canada, where much Doppler research has been carried out on decompression tables, have adopted the policy that dive profiles which produce peak Doppler-detectable bubbles of Grade II or greater in 50% or more of the subjects, probably constitute an unacceptable decompression stress.[4] Unfortunately, cases of DCI still occur with no detectable circulating bubbles, so the policy is only a guide. Also there is great individual variation in the number of bubbles produced between different divers carrying out the same decompression profiles, and in the same divers carrying out the same decompression profiles on different days. Divers who are fatigued before a dive produce more bubbles and have a greater risk of DCI.[6] Other factors known to predispose to DCI include state of hydration, obesity, age, level of stress and infection,[3] and it is likely that these factors may be associated with the production of more circulating bubbles.

In saturation diving on helium breathing mixtures, the correlation between DCI and Doppler-detected bubbles appears to be different from that seen in non-saturation dives. There is a greater incidence of DCI at all bubble grades to the extent that some corrective action has been recommended to modify the decompression schedule in helium dives if the detected bubble grade is greater than grade II.[6]

It must be remembered that in the studies quoted here, which were performed largely in the USA and Canada, the definition of DCI differs from that now in use in Australia.[7,8,14] Joint pain has been required for the clinical diagnosis of DCI until recent times. It is now widely recognised that more subtle effects such as neuropsychological changes and concentration deficits are also symptoms of DCI. One wonders whether the investigators involved missed an appreciable number of subjects with significant DCI in the studies carried out to date, because the subjects had no joint pain. It could be that bubbles of the lower K-M grades may be pathogenetically significant and cause more subtle changes than previously thought.

Summary

The hope that Doppler-detected bubbles could be used as a diagnostic aid for DCI arose from the belief that circulating bubbles caused symptoms of DCI. The current evidence suggests that this is not true. However, circulating bubbles indicate a decompression stress has occurred and may indirectly point to the presence of bubbles elsewhere in the body tissues.[6] Intravascular bubbles may also cause subclinical damage which may have long term effects.[13]

It is certainly clear that circulating bubbles are formed even after decompression from very shallow depths and, although these low-grade bubbles are dismissed as non-significant by the North American authors, it is not known what the long term effect of repeated exposure to such circulating bubbles is. They may be implicated in the neuropsychological changes now being recognised in long-term divers.[4,25-28] Aggressive behaviour, inability to concentrate, poor memory and tiredness now form a symptom complex being increasingly recognised in long term divers, many of whom have never suffered an episode of clinical DCI. Also, dysbaric osteonecrosis occurs in many divers who have never had an episode of clinical DCI and may also be related to chronic exposure to VGE.[28]

The studies performed to date conclude that circulating venous bubbles of less than grade II are probably of little clinical significance, as they are associated with a very low incidence of DCI (<2%), but bubbles of grades III-IV are more significant, being associated with a higher incidence of clinical DCI. Although authorities such as the DCIEM accept bubbles of grade II or less as an acceptable risk in their dive tables and decompression schedules, on current evidence the validity of this assumption should be questioned.

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DIVING DOCTOR'S DIARY

SCUBA KIDS

Carl Edmonds

Case summary

(The full and excellent description of this case report, by the child/patient/diver herself, can be read in Scuba Diver)[1]

At the age of 12 years her father gave her a birthday present, a scuba diving course.

The family were very conscientious and ensured that she was examined by a diving doctor, who also performed lung function and provocation tests at the local hospital. I have not been able to track them down.

The qualification, which she received without difficulty, allowed her to dive in association with another certified diver. This she achieved by her father driving her many hours to the coast, every few months.

During one of these visits she did a dive to 6 m for 37 minutes, totally uneventful. Some 8 hours later, while travelling over the mountain range that surrounds Sydney, she became aware of a tingling in one knee. The usual plea from her father to tell him of anything that could possibly go wrong with a pain in the joint or something like that, was initially ignored but she finally did disclose the tingling sensation.

She was then starting to become a little apprehensive, made worse by reviewing a diving manual which described the symptoms of decompression sickness. At this stage both she and her father were panicking, she in tears and her father driving wildly to return to Sydney.

They contacted the Divers Emergency Service number on their mobile phone, and talked for some time to the doctor. By this stage, with the patient on the phone talking to DES, the whole situation deteriorated. The patient burst into tears and her father took the phone. She was shaking all over and very apprehensive. As she stated "I am going to die here in this car, on some ******* mountain".

The symptoms developed further and she complained of numbness and tingling in all extremities and an increased numbness in the leg.

They then came down from the mountain, but at this stage she was in a bad state with chest pains, difficulty breathing, blurred vision and tiredness etc. She was crying, dad was swearing.

By the time they got to a local teaching hospital, the whole family was close to tears. By then it was all a very big emergency. Whatever else was happening, she was hyperventilating and confused.

The ambulance, the paramedics and the intravenous drips all combined to deliver a live, but very distressed, patient to the Naval base.

Hyperbaric treatment was then given, on the very reasonable presumption and diagnosis of a cerebral arterial gas embolism from pulmonary barotrauma. The patient responded well to this, and there were few, in any, remaining symptoms during the subsequent hospitalisation.

The professional diving physician who saw her did strongly suggest that, because of her asthma (did I not mention that she took Pulmicort regularly?) that she should not continue scuba diving.

She was seen by the "best diving doctors in the state", who gave her and her parents a variety of advice, including references to "wild cat bends" and suggestions to wait until she is a little older before she resumes scuba diving. (This certainly does not say much for the standard of diving medicine in New South Wales.)

It was on the basis of the above report, which has been much abbreviated by me, but is in all its colour and glamour in the Scuba Diver article, that I prepared the following response for the magazine.