HOW SAFE IS PEARL DIVING?

Robert M Wong

Abstract

The pearl divers of Broome, in Western Australia, perform repetitive and multi-day diving and yet they have an overall incidence of decompression illness of 0.01%. This paper discusses the reasons why.

Introduction

On initial inspection of the pearl divers dive profiles,[1] one could be excused for thinking that the profiles are unworkable, because the mode of diving have **inadequate decompressions** and **inadequate surface intervals** when compared with the conventional decompression tables.

Moreover, they appear to violate all the current recognised risk factors as they require more than 3 repetitive dives per day, multi-day diving for more than 4 consecutive days, deep dives to more than 30 m and diving from deep to shallow on occasions.

Notwithstanding the above, their overall incidence of decompression illness (DCI) among the pearl divers is less than 0.01% and, for the past four years, the clinical manifestation has been confined to musculo-skeletal symptoms only.

Current data on repetitive and multi-day diving

It is well known that repetitive dives and multi-day diving cause problems and run a high risk of DCI. This has been demonstrated both clinically and in experiments. A number of papers have presented theoretical reasons for such risks. The "critical diameter model" or "arterial emboli model" suggests that bubbles are trapped in the lungs during normal decompression and with repetitive diving the recompression of the next dive may allow bubbles to pass across to the arterial side causing serious neurological DCI.[2,3]

The existence of this transmission has been demonstrated in mice and in guinea pigs.[4] It has also been shown that there was central movement of bubbles formed in the periphery by the compression phase of a repeat dive.[5] Showers of bubbles could be seen ascending the inferior vena cava during the compression phase of the second dive and bubbles were observed in the aorta during the second decompression. Therefore, an ill judged repeat dive not only could give rise to an increased likelihood of DCI but may also result in far more severe symptoms.[6]

While it is convenient to accept the arterial emboli model, it has to be pointed out that Francis et al.[7] injected air into spinal arteries in dogs and found the emboli to be distributed in the grey matter rather than the white matter where spinal lesions are typically seen.

Other experimental evidence also showed that repetitive diving was a reliable means of producing spinal DCS in goats and dogs.[8,9]

Imbert et al.[10] indicated that the currently available data suggest that the risk of Type II decompression sickness (DCS) decreases as the surface intervals increase and also that the arterial emboli model successfully permitted correlation of Type II DCS occurrences with depth changes or recompressions. Nevertheless, after 6 hours, it appears that recompression at the beginning of the second dive no longer produces arterial bubbles.

It has long been recognised that sports divers who perform repetitive and multi-day diving have a high incidence of neurological DCI.[11] Information on risk and incidence is scarce, however, by applying the method of maximum likelihood to repetitive air dives using the DCIEM Sport Diving Tables for a two-dive profile, it is estimated that the probability to be around 1.1% to 3.2%.[12]

Notwithstanding the above theoretical and experimental evidence of harm caused by repetitive diving, the pearl divers appear to do this with no incidence of neurological DCI.

Since the pearl divers continuously "break the rules" of conventional diving, why then are they not prone to DCI just like everyone else? Why is it that they have an incidence of less than 0.01% and are confined to musculo-skeletal symptoms?

In practice, the acceptable risk of DCI has been quoted as US Navy divers 3-4%, Caisson workers 2%, Commercial divers 0.1-0.5% and the space shuttle as 6%.[13]

Pearl divers of Broome

Despite their unconventional way of diving, it appears that the pearl divers of Broome dive reasonably safely.

The success of the profiles could be due to a number of factors which are different from the conventional decompression tables.

These are a slow rate of ascent, an appropriate depth of the decompression stop, the use of oxygen in decompression, suitable interdive intervals and perhaps acclimatisation.

Is there an optimum rate of ascent?

Dr M Foley wrote at the time of construction of the bridge at Argenteuil over the Seine in 1861 that he considered the rate of decompression to be unimportant. He thought that one minute per atmosphere was enough.[14] However, most scientists at the time believed that a slower decompression should be followed.

In diving, unlike caisson work, there appears to be some very different opinions. From the historical perspective, Haldanian practice used an ascent rate of 25 ft/min (7.6 m/min).[15] Haldanian "staged decompression" was challenged by Sir Leonard Hill,[16] who advocated a slow bleed approach of uniform decompression, a technique in use by the pearl divers. A study of the pearl divers in Torres Strait led to the thermodynamic approach advocated by Brian Hills.[17]

Although it has generally been accepted in the diving community that the "standard ascent rate" of air diving ought to be 60 ft/min or 18 msw/min., supporting evidence is wanting. The 1952 issue of Bureau of Ships Diving Manual NAVSHIPS 250-880 stated that the ascent rate was not to exceed 25 ft/min. Commander Doug Fane USN, wanted his "frogmen" to ascent at 100 ft/min or greater. As a compromise between the "frogmen" and the standard hard hat divers that 60 ft/min came into being. (US Navy Air decompression tables 1958).[18] The new USN decompression tables (1993) have been modified to a 30 ft/min ascent rate.

Most air decompression tables concentrate on the depth and duration of the decompression stops as well as the duration of the surface intervals. The ascent rate is given less attention.

A survey of various dive tables shows that the recommended ascent rates vary. The USN and PADI use 60 ft/min (18 m/min). The Swiss (Bühlmann/Hahn) tables use 10-15 m/min to 6 m, then 6 m/min to the surface. Huggins uses 40 ft/min (12 m/min) to 20 ft (6 m) then 20 ft/min (6 m/min) to the surface, in addition a three minute stop at 20 ft (6 m) is required for dives deeper than 60 fsw (18 m). The DCIEM tables use 18 m/min (± 3 m/min). The Royal Navy uses 20 m/min while the Bassett tables have 10 m/min as the ascent rate.

If the ascent rate is too slow, there is a penalty of added bottom time. For instance, the DCIEM table states that if the ascent rate is less than 15 m/min and if delay starts deeper than half the maximum depth of the dive the time delay is added to the bottom time and the diver is required to decompress in accordance with the new bottom time; if however, the delay starts shallower than half the maximum depth of the dive, then the delay is added to stop time of the next stop. If no stop is required, then the diver has to stop at 3 m for the time of the delay. Similar rules are applied to the USN decompression tables where the rate is less than 60 ft/min (18 m/min) and the depth of 50 ft (15 m).

Is slower better?

The size of bubbles is determined by degree of saturation and the rate of pressure reduction (ascent). Therefore, a slow ascent has the advantage of maintaining the micronuclei under pressure but has the disadvantage of slowing diffusion from the tissue.

Lewis, using the US Navy's 6 compartment model as a basis of calculation, showed that by halving the ascent rate from 60 ft/min (18 m/min) to 30 ft/min (9 m/min) is the equivalent of 0.8 minutes of decompression stop at 15 ft (4.5 m) when diving to the USN's no-decompression limits.[19] Wienke considers safety stops of 2-4 minutes are easier and more efficient than slowing the ascent rate.[20]

Hamilton while agreeing that slowing the ascent rate decreases the incidence of DCI, points out that a slower ascent rate results in a penalty of bottom time.[21] He considers that a short stop is slightly more beneficial than a slow ascent rate and does not increase the dive time as much.

Yount, using the varying permeability (VP) model as a basis for calculation, considers that there is no minimum rate of ascent.[22] Van Liew considers that the slower the ascent the better.[23]

Smith implanted Doppler cuffs around the inferior vena cava in 2 goats.[24] He chamber dived the goats to 220 fsw (66 m or 660 kPa) for 20 minutes and compared the results of using the USN Decompression Procedure (Exceptional Exposure Table) and a 2 phase linear decompression with 30 ft/min (9 m/min) ascent rate to 80 fsw (24 m), then ascent at 2 ft/min (0.6 m/min) to the surface. With the latter incidence of precordial bubbles was greatly reduced.

It was stated by Vann that in 60 dives using a variety of decompression profiles, venous bubbles were nearly abolished by reduced ascent rates and deeper initial decompression.[25]

Mano, using gelatine gel experiments, demonstrated that as the ascent rate decreased, the number of bubbles also decreased. He further demonstrated that the optimum rate of decompression was 9 msw/min (30 ft/m).[26]

Rapid ascent to the surface may generate bubbles of a diameter small enough to cross the lungs,[27] and that repetitive diving with short surface intervals and yo-yo diving could be expected to produce arterial bubbles.

However, it has also been stated that a carefully planned sequence of repeat dives may have a lower than expected incidence of symptoms because the nuclei are consumed and the number of bubbles decreases and that dives must be conducted in decreasing severity.[19]

Daniels has calculated that if decompression in a saturation dive was slowed by 25 times, then bubble formation could be avoided.[28]

Evans and Walder demonstrated that hydrostatic treatment of *Cragnon cragnon* could reduce the extent of bubble formation on subsequent decompression and postulated the existence of micronuclei.[29] Other workers using other models indicated that bubble formation within tissue is initiated as micronuclei and that they are pressure sensitive.[30] Daniels et al. have investigated and confirmed the effect of hydrostatic pressure on the common shrimp *Cragnon cragnon* and that regeneration of micronuclei does occur.[31]

Brubbak noted that, in saturation dives, there were more bubbles formed during the first than the second ascent. This was postulated to be due to consumption of the micronuclei during the first ascent and inadequate time for subsequent regeneration.[32]

From the practical viewpoint, Zannini reported that the Italian commercial divers now routinely use 10 m/min ascent rate and reported no incidence of DCS in over 24,000 dives at depths ranging from 10 to 50 m.[33]

Koch modified USN treatment table 6 by slowing the ascent from 18 msw to 9 msw and reported a dramatic reduction of recurrence of symptoms (personal communication 1992).

The ascent rates of therapeutic tables vary greatly.[34] USN 6 uses 1 fsw/min (0.3 m/min) while USN 1 uses 20 fsw/min, 10 f/min and 6 f/min (6, 3 and 1.8 m/min). RN 71 uses 60 m/h at depth, then 6 m/min to 0.5 m/min towards the surface, while COMEX Cx 30 requires 5 mins/m to 2 mins/m.

Since we assume that, in DCI, bubbles are the offending agent the rationale for treatment, in simplistic terms, is to recompress the bubbles and use a breathing gas that would cause the bubbles to shrink by creating a large diffusion gradient, then use an ascent rate(s) that does not encourage bubbles to grow on decompression. As it is generally accepted that after a compressed air dive bubbles are formed on most decompressions,[35] especially with repetitive dives and multi-day diving, one should logically consider a slow rate of ascent to avoid bubble formation. Perhaps, this was the rationale and the lesson learnt by the pearl divers in developing their profiles from their personal experience of trial and error over the past 100 years.

A slow rate of ascent, apart from decreasing the risks of pulmonary barotrauma and its sequelae, does appear to shift the type of DCI from neurological to musculo-skeletal symptoms, (in the same manner as in saturation diving where it is uncommon to have symptoms affecting the spinal cord) and therefore confer a degree of safety for repetitive diving.

Analysis of DCI incidents at the Royal Australian Navy (RAN) Submarine Escape Training Facility, HMAS STIRLING, revealed that 7 (54%) of the 13 instructors suffered DCI when exposed to multiple and rapid ascents (buoyant and hooded ascents) from depths of between 18 and 22 msw. (Loxton personal communication).

Depth of decompression stops

Pilmanis showed the effectiveness of a decompression stop in reducing bubble numbers. A 2 minute stop at 10 ft (6 m) after a 100 ft (30 m) dive for 25 minutes (USN no-decompression limit) produced a dramatic drop, by more than 5 fold in the first 15 minutes, in bubble numbers when compared with a direct ascent to the surface (Fig. 1). It was suggested that short “safety stops” could be beneficial in reducing the occurrence of “silent bubbles” in divers using the limits of the USN no-decompression Tables.[36]

While there is consensus regarding the benefits of a decompression stop, there are varying opinions about the optimum depth of decompression.

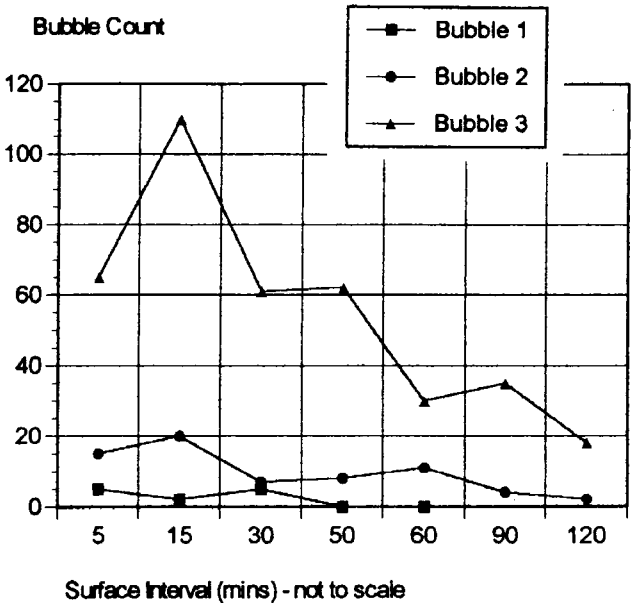
The decompression tables of the USN and RN have decompression stops at 3 m, 6 m, 9 m and 12 m, whereas RNPL/BSAC have stops at 5 m and 10 m.

Le Messurier and Hills studied hard hat divers in Torres Strait and found that they surfaced directly from 33 -44 ft (10-13 m) and decompressed in about 2/3 of the time required by a USN table.[37] The pearl divers from Broome also chose to decompress at a greater depth than the standard decompression tables, they staged at 7-8 fathoms (13-14 m or 42-48 ft) on air. Nowadays, they use 9 m on oxygen.

Kindwall reported that Behnke felt that the USN decompression stops were too shallow.[38] It has been demonstrated by Kindwall et al. that more gas can be eliminated at 50 fsw (15 m) than at 10 fsw (3 m) in a given amount of time.[39] Hills has also shown that goats can be decompressed in a shorter time period without DCI symptoms if the 20 fsw (6 m) and 30 fsw (9 m) stops were prolonged instead of coming to 10 fsw (3 m) for the final stop.[40] As stated above, Mano has demonstrated, with his gelatine model ,that the most effective first stop was at 9 msw.[26]

FIGURE 1

PILMANIS EXPERIMENTS ON ASCENT PROCEDURES



All dives were to 100 ft (30 m) for 15 minutes.
Bubble 1 had decompression for 1 minute at 20ft (6 m) and 4 minutes at 10 ft (3 m).
Bubble 2 had decompression for 2 minutes at 10 ft (3 m).
Bubble 3 was a direct ascent to the surface.

Trials in the UK indicated that when decompression stops were done at 6 msw rather than 3 msw, the incidence of DCI dropped by some 40%.[41] It appears that deeper in-water stops are beneficial for elimination of inert gas. Further, inert gas elimination is more effective without bubble formation, because the effective half time for elimination of gas in a bubble is greater than for the elimination of dissolved gas.[25] It is known that dissolved and free gases within tissues do not behave in the same manner.

From this evidence the slow rate of ascent and the 9 msw decompression stop chosen by the pearl divers might well be the optimal way for their mode of diving.

Oxygen decompression

Oxygen decompression was used as early as 1917 in the recovery of gold from the sunken "Laurentic" when oxygen was given immediately after surfacing from the sea.[42]

The investigations by the Admiralty Committee on Deep Diving in 1930 subjected divers to partial pressures of oxygen of 1.7 to 2.27 ATA, and it was noted that the divers showed confusion and amnesia as well as unreliable and unpredictable behaviour. They attributed these symptoms to raised tension of oxygen; psychological factors or indeed increased tension of nitrogen. The "Damant Tables" using oxygen for decompression from 60 fsw (18 m) upwards (in the dry) were adopted by the Royal Navy in 1932. Despite the experiment in 1933 in which 2 RN Officers (Damant and Phillips) breathed oxygen at 4 ATA in a RCC and convulsed in 18 and 13 minutes respectively, the lesson of oxygen toxicity did not register. The USN introduced in-water oxygen decompression from 60 fsw (9 m) in 1942 and the Royal Navy followed suit.[42]

It is well known that hyperbaric oxygen could give rise to CNS oxygen toxicity. The toxic effects of hyperbaric oxygenation had been amply demonstrated by Bert using dogs, rats and other animals.[14]

It was known also that breathing oxygen deeper than 33 fsw (10 m) underwater was dangerous and that any oxygen stops should be in a chamber and with an attendant. But Behnke in 1946 felt that it was safe to decompress on oxygen at 40-60 fsw (12-18 m) provided the divers were at *complete rest.* This practice finally ceased when there were a number of cases of acute oxygen poisoning at 60 fsw (18 m).[42]

In spite of the above, there are some well-known and respected decompression tables that make use of oxygen in decompression such as DCIEM, COMEX, French Navy, Duke University etc. In the UK sector of the North Sea, oxygen is not administered at depths greater than 40 fsw, and some Dutch companies limit oxygen breathing to 9 msw, in a wet bell.[42] Imbert indicated that COMEX has 2 types of oxygen decompression tables, one at 6 msw for a surface demand regulator, and a 12 msw table for use in a wet bell.[43] He indicated further that from the COMEX data bank, there were some 5,600 dives using 12 msw oxygen decompression with more than 30 minutes of oxygen breathing, suggesting that it is a "safe" procedure. However a few cases of toxicity were recorded, but these were found to be due to errors in procedure such as sending the wrong gas to the diver.

It must be remembered that the navies of the world use oxygen diving with rebreathing sets for clandestine operations to avoid the tell-tale bubbles. The Royal Navy employs a 8 m (26 ft) limit for **swimming** oxygen divers.[44] The USN allows oxygen dives to 30 ft (9 m) for up to 80 minutes.[45] The RAN allows a maximum depth of dive to 10 m (33 ft) and underwater swimming to a maximum depth of 8 m (26 ft).[46] Nonetheless, central nervous system (CNS) oxygen toxicity with convulsions have been reported at 25 fsw (7.5 msw) after 72 minutes.[47] These were "working dives". A second series[48] conducted by the same authors, with no convulsions at 25 ft (7.5 m), gave rise to the official USN depth time limits of oxygen diving

of 4 hours maximum at 25 ft (7.5 m), 80 minutes at 30 ft (9 m), 25 minutes at 35 ft (10.5 m), 15 minutes at 40 ft (12 m) and 10 minutes at 50 ft (15 m).

It has been stated by Donald[42] that rest does not give the remarkable protection from oxygen poisoning underwater as has been claimed, but the comparative tolerance at rest and on exercise at 30-50 ft underwater has not been fully studied since the Royal Navy series in 1945.

The Draft Standard of AS2299-1992[49] specifically prohibits the breathing of oxygen under water on the basis of risks of "fatal underwater toxicity reaction". It did not specify the depth nor the duration of oxygen breathing.

Notwithstanding the above, there are obvious advantages in the use of oxygen. In the Haldanian model, it was assumed that inert gas was dissolved. It is known that on decompression, bubbles are formed and that the faster the rate of ascent, the more bubbles are formed. The elimination of dissolved gas and bubbles have totally different gas kinetics. Oxygen creates a large diffusion gradient for elimination of bubbles. Hence, one could hypothesise that the pearl divers "slow rate of ascent" would eliminate a large proportion of inert gas and the bubbles which are formed are either removed or reduce in size because of the large diffusion gradient generated by the breathing of oxygen.

It has been claimed that oxygen decreases decompression time by 30% to 50% depending on the depths of the dives.[50] The French Navy considers the use of oxygen reduces decompression time by 30% at all depth ranges.[51] Imbert and Bontoux,[50] using the French air decompression tables with in-water oxygen decompression, indicated that oxygen decompression not only saves decompression time but also has the effect of decreasing the incidence of DCI to 2-3 times lower than with air decompression for dives of the same depths and bottom times.

Fife et al.[52] reported the successful use of oxygen decompression in 7,500 dives, ranging in depths of 50-60 m (166-200 ft), in the excavation of a Bronze Age Shipwreck. There were 3 cases of DCI with no incidence of oxygen toxicity. The decompression tables used were modified USN tables as well as Duke University tables with oxygen introduced at 20 and 10 ft (6 and 3 m).

Furthermore, oxygen might confer other safety benefits. Dysbaric osteonecrosis (DON) is a well known occupational hazard for divers and caisson workers. Kindwall reported that ever since the Germans and the French introduced the use of oxygen in decompression for caisson workers, there have not been any cases of DON.[38] It is not known whether oxygen decompression confers the same degree of safety to the divers.

It has to be borne in mind however, that experimental data has demonstrated that animal fat cells enlarge when exposed to increased partial pressures of oxygen and this could play an important role in the cause of DON.[53] However, it has not been reported that the divers of the armed forces of the world who use oxygen rebreathing sets have a high incidence of DON.

Although CNS oxygen toxicity is a recognised hazard, since the introduction of oxygen in decompression in the pearling industry in 1981, there has not been a single incident of CNS oxygen toxicity.

Long term use of oxygen also raises doubt of pulmonary toxicity. Sterk and Schrier suggested that long term exposure in the order of 400-500 UPTD (Units of Pulmonary Toxic Dose) each day could be a risk.[54] Nonetheless, Donald, with his extensive experience with the RN divers using oxygen, considers that cumulative effects are most unlikely.[42] The experience with the pearl divers so far indicate that Donald was right.

Suitable interdive interval (surface interval)

It is assumed that on surfacing after compressed air diving inert gas is eliminated, it is also known that elimination of inert gas is not the mirror image of uptake, it takes longer. Based on this assumption, for repetitive diving, increasingly longer surface intervals are required after each dive. However, the exact time for elimination of inert gas is unknown. Various figures have been quoted,[13] Rogers (PADI Recreational Dive Planner) assumes 6 hours,[55] the French Navy requires 8 hours, the USN says 12 hours and the DCIEM tables need 18 hours.

When the various mathematical models are used to calculate the decompression requirements for various dives, the results are quite different for different tables. For instance the no-decompression limits for a dive to 18 m (60 ft) and to 40 m (120 ft) are shown in Table 1.

Similarly, different results are obtained for repetitive dives, for instance after a dive to 30 m for 10 mins and a surface interval of 2 hours 30 minutes, a repetitive dive to 15 msw will give widely differing results (Table 2).

The calculations for repetitive diving exposures, which rely on the concept of compartments and half times for elimination of inert gas, are based, at best, on a premise which needs proof. The Haldanian model employs a number of compartments in parallel, varying from 6 for the USN to 16 for the Bühlmann tables, the DCIEM model uses compartments in series and the diffusion model uses a slab concept. Furthermore, elimination of inert gas is not the mirror image of uptake, it is slower. It may not be exponential, it could be linear or could be a combination of the two.

TABLE 1

NO-DECOMPRESSION LIMITS FOR DIVES TO 18 AND 40 M

Table	18 m	40 m
RN	60 mins	9 mins
USN	60 mins	10 mins
RNPL	57 mins	11 mins
Bassett	50 mins	5 mins
DCIEM	50 mins	5 mins

TABLE 2

REPLETITIVE DIVE AVAILABLE AFTER A DIVE TO 30 M FOR 10 MINS AND A SURFACE INTERVAL OF 2 HOURS 30 MINUTES

Table	Dive
RN	10 min
RNPL	15 min
USN	79 min
Bassett	49 min
DCIEM	55 min

An optimum surface interval for repetitive diving?

Western Australian pearl divers of bygone days had their surface intervals dictated by the time needed for the lugger to turn around, sail up current and get into position for the next dive, this was 10 to 40 minutes and a natural occurrence of their daily work. Nevertheless, there are examples where surface intervals do play a part in the safety of the divers.

In the experiments with *Cragnon cragnon* ,[31] it was found that pressure pre-treatment can eliminate bubble formation after subatmospheric decompression. It was also found that regeneration of bubbles does occur after a half-time of 8-10 hours and the effect of pre-treatment was not evident after 24 hours. If dives were performed in rapid succession, then micronuclei could have been consumed and therefore not cause any problems.

In Taravana,[56] a condition akin to DCI, that affected breath-hold pearl divers of the Tuamoto Archipelago in the South Pacific, it was found that prolonging the surface interval from their usual 3-4 minutes to 10 minutes made the phenomenon of Taravana rare. These were not compressed air dives, so the inert gas load would be much lower, and 10 minutes might be all that was required to eliminate all the extra inert gas.

Yo-yo diving has long been considered a risk factor, however, a study of the Scottish fish farm divers by Shields et al.[57] has refuted this allegation, but the number of surface excursions was less than 10. Another study by Parker et al.[58] using the USN Probabilistic Decompression Model showed that the risk of DCI in yo-yo diving does increase especially if a large number of descents (more than 10) are made. A surface interval of approximately 5 minutes resulted in the highest estimated probability of DCS [P(DCS)] and longer surface intervals (10-120 min) provided intermediate estimates. A surface interval of 0 min (immediate return to depth) provides the lowest estimate of P(DCS).

For the technique of surface decompression, where the diver ascends rapidly to the surface, from either the bottom or a stop, and is then recompressed to 3 m deeper in the RCC, the DCIEM surface decompression tables allow a maximum time of 7 minutes to reach the required pressure. The pearl divers' technique could be viewed in a similar light in that they surface and are recompressed within 20 minutes to a depth of the previous dive. The difference is that they ascend slowly (producing fewer bubbles) to a 9 m stop using oxygen, so eliminating bubbles which are formed, and use a fixed surface interval of 20 minutes. This method does not explain the success of diving in deeper depths than 25 m where the schedules are more akin to the conventional profiles, with a longer surface interval after each dive.

The safe surface interval before flying after diving varies depending on the type of dive, which can range from no-decompression dives though decompression dives, repetitive and multi-day dives to saturation dives. Opinion also varies regarding flying after treatment of DCI. Currently, there is no consensus. Essentially, the relative safety of the surface interval depends very much on the adequacy of the decompression. If the decompression is inadequate, then theoretically, large number of asymptomatic bubbles could be generated which could lead to symptoms on altitude exposure.[59]

Reflecting on the old hard hat diving days, the pearl divers used to ignore surface intervals. The time spent between drift dives on deck was dependent on the time it took the old luggers to sail against the tide and to swing around to drift again. That short surface interval appeared to be adequate in shallower waters, however, when they dived in deep waters, the incidence of DCI rose.

With the current PPA Profiles, the Non-rotational profiles all have 20 minute (11 msw has only 15 minutes) surface intervals. This applies to depths from 13 m to 23 m. However it is not possible to dive the 23 msw profile to the number of dives allowed by the original Code of Practice of the PPA. The 19 msw and 21 msw profiles are also high risk.

When one views the hyperbaric stress of each dive expressed as PrT (P=absolute pressure, rT= square root of

time in minutes of bottom time), it appears that the product PrT should not exceed the value of 17 for repetitive diving when the surface interval is limited to 20 minutes (Fig 2)

In simplistic terms, it seems as if the body can tolerate a certain amount of supersaturation after a dive (perhaps this is the limit of supersaturation of the fast compartments, but the slow compartments dictate how much surface interval is required) and provided that some elimination of inert gas is permitted between dives to buffer the additive effect of the next dive's increase in inert gas load, if this is not too high; the situation could be tolerated. However, once the limit is exceeded, such as when PrT is greater than 17, the short surface interval of 20 minutes becomes inadequate, the slow compartments are gradually being filled up with each repetitive dive and become super-saturated on surfacing which would lead to symptoms of DCI. This simplistic view has not taken into consideration that short surface intervals do limit the growth in size of bubbles. The risk of DCI could perhaps be avoided if the subsequent slow ascent rate is very, very slow and that the decompression stop is very, very long. It is interesting that the pearl divers have learned by trial and error that longer surface intervals were required when diving to depths of over 23 m. Preliminary testing of the high stress profiles appear to be promising when the PrT value is reduced. By reducing the bottom time of the 23 msw profile from 40 minutes to 25 minutes, the bubble grades are reduced to an acceptable level.

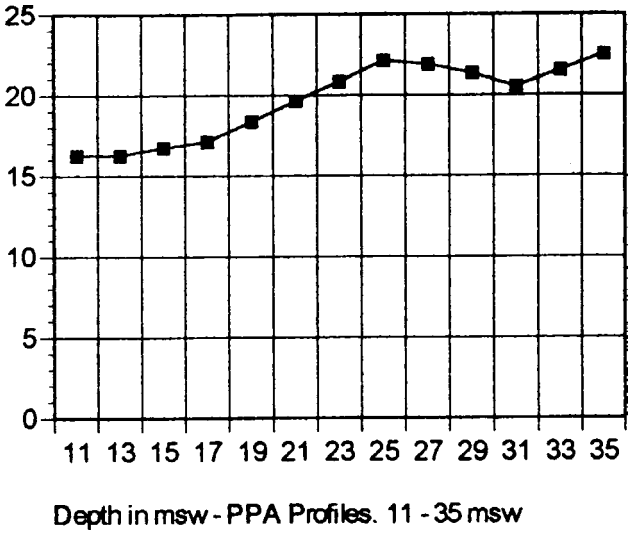
This phenomenon of high PrT with short surface interval is also observable in conventional diving. Among the DCI cases who presented to the RAN for treatment, there is a group who appeared to be refractory to treatment and had dive profiles with the repetitive group (RG) designated as E and F in the DCIEM Standard Air Tables (Loxton personal communication).

On examination of the repetitive factors/surface intervals table, table 4 of the DCIEM Standard Air Tables, it is allowable to do repetitive dives with short surface intervals. In RGs A to F, one could have short surface intervals of 15 to 30 minutes. From group G onwards, longer surface intervals are required, with a minimum of 30 minutes for group G and a minimum of 3 hours for group M. The bottom times required for dives to fall into the E and F groups need to be 50 minutes for 15 m, 40 minutes for 18 msw etc. These will give PrT values of 17 to 18.5 for the group E dives (from depths of 18 to 36 m). For dives that fall into repetitive dive group F, the PrT values range from 18.62 to 20.57 (Table 3). For a 21 msw dive, the PrT value ranges from 9.8 for Group A to 31 for Group N. Group E and F have values of 18.34 and 19.61 respectively, with permissible surface intervals of 15 to 30 minutes (Table 4).

From the foregoing, it appears that there is a minimum tolerable surface interval for different modes of

FIGURE 2

PrT OF PPA PROFILES DIVED AS WRITTEN



Bottom times of PPA Profiles

Depth in m	Bottom time of each dive	Number of dives
11	60	10
13	50	10
15	45	10
17	40	10
19	40	10
21	40	9
23	40	8
25	40	5
27	35	5
29	30	5
31	25	5
33	25	5
35	25	4

TABLE 3

REPETITIVE DIVE GROUPS E AND F WITH PrT VALUES

Depth	Bottom Time		PrT value	
in msw	Group E	Group F	Group E	Group F
15	50	60	17. 68	19.34
18	40	50	17. 7	19.8
21	35	40	18.34	19.61
24	25	30	17	18.62
27	25	30	18. 5	20.27
33	0	20	0	19.23
36	15	20	17.82	20.57

TABLE 4

REPETITIVE DIVE GROUPS FOR 21 msw DIVES AND PrT VALUES

Repetitive Group	Bottom Time	PrT value
A	10	9.8
B	20	13.86
C	25	15.5
D	30	16.98
E	35	18.34
F	40	19.61
G	50	21.92
H	60	24.01
J	70	25.94
K	80	27.72
M	90	29.41
N	100	31.0

diving, the period depends on the hyperbaric stress of the dive(s) and the subsequent adequacy of decompression. Nevertheless, there appears to be a "safe period of grace" after surfacing before sufficient number of bubbles are formed and grow in size to cause symptoms.

If the recompression is rapid enough, little harm would befall the diver provided that the subsequent decompression is adequate, as illustrated by the practice of surface decompression.

The effects of surface interval on the pearl divers profiles are under investigation. Preliminary testing shows that when a stressful profile is modified by reducing the PrT, the short 20 minute surface interval is feasible. Unfortunately, the PrT values are not additive, and no prediction could be made.

From the RCC trials of the Pearl Diving Profiles, the pre-dive bubble grades were invariably Grade I- to Grade I+ after a 12 hour break),[60] indicating the presence of moving bubbles, suggesting that inert gas elimination takes longer than 12 hours with their mode of diving. After about 17 to 19 hours, the Doppler score was generally 0. The testing is continuing.

Acclimatisation

It has been suggested that acclimatisation is a factor in preventing DCI. This is highly unlikely because the divers dive daily for the duration of the neap tide then return to shore for a week. When they dive again, the depth could be deeper. Also it is known that acclimatisation is depth specific and that caisson workers lose this advantage after even a week-end off.[61] The pearl divers do not dive during the spring tides.

If acclimatisation is a factor, then one would expect the bubble grades to decrease at the end of the diving trip. If however, multi-day diving is a high risk factor, then one would expect to see rising bubble grades towards the end of the trip. In reality, bubble grades stay reasonably constant.

Long term health

The long term health effects of this mode of diving are not known. The divers have an annual medical examination, in accordance with AS2299. So far these have not demonstrated any overt impairment. No formal psychological assessment has yet been instituted. The common medical problems such as salt water aspiration, marine animal stings and hearing problems will be the subject of another report.

From the diving records, 43% of the divers commenced diving before 1990, 33% between 1990 and 1993 and 24% started diving in 1994.

Dysbaric osteonecrosis

Dysbaric osteonecrosis was recognised in tunnel workers in 1912, but only in 1942 was it accepted as a condition that affected divers.[62]

Studies on naval divers and commercial divers have shown a lesion in 5 to 7% in Navy divers and 4.8% (in 1979) in commercial divers.[63-65] These divers dived to conventional and experimental profiles.

Ohta and Matsunaga reported that 50.5% of Japanese diving fishermen had radiological lesions and juxta-articular lesions were seen only in those who had dived deeper than 20 m and had over 5 years of experience.[66] The age range was from 16 to over 50. The incidence increased with age, particularly over the age of 30 and in those with over 10 years of diving experience.

Kawashima also demonstrated a high incidence of DON in Japanese divers who dived to unconventional profiles, and particularly to depths in excess of 30 m.[67] 54.4% of divers with more than 5 years of experience were affected. The divers were aged between 16 and 64.

It is known in the past that there are a number of pearl divers who have DON. However, since the introduction of AS2299 diving medical in 1990, no new cases have been detected in divers with less than 5 years' experience in the industry. This covers the period since the adoption of uniform diving profiles by the PPA and the four years of diving the modified profiles. Modification of profiles is on going. All radiographs are performed at Royal Perth Hospital or the Broome District Hospital. Any

suspicious lesions are investigated and followed up. A medical registry was started in 1990 which includes a survey of all long bone X-rays. It is interesting that despite the unconventional profiles of the pearl divers, there has not been an overwhelming incidence of DON seen in the Japanese divers. Perhaps, oxygen breathing decompression provides a protective effect or perhaps the onset of DON takes much longer.

At the end of 1995, there have been 2 cases of dysbaric osteonecrosis reported. These 2 divers had started diving in the pearling industry in the 1980s when the use of profiles were not uniform and the usage of oxygen for decompression was haphazard.

The pearl divers dive profiles are being tested continuously and their mode of diving is being documented for further study.

Summary

The dive schedules of the Western Australian pearl divers are based on years of experience and are all "dived" profiles. Despite the fact that their mode of diving does not conform to the current decompression models and mathematical calculations, they nevertheless "invented" a mode of diving which is "safe" or at least with an acceptable DCI rate. They have manipulated all the diving variables, bottom time, surface interval, ascent rate, depth of decompression, to their advantage and to the limit. It is however, not recommended that non-pearl divers adopt such mode of diving.

In common with previous investigation, the profiles share the slow ascent rate and the deep decompression stop of other pearl divers.

One can hypothesise that the mode of diving which afforded relatively safe diving for the pearl divers appears to be due to a combination of the factors discussed. The slow ascent rate might assist in minimising bubble formation; the depth of decompression in combination with oxygen breathing assist in off-gassing and the elimination of bubbles that are formed.

The surface interval which seems inadequate for repetitive diving, appears to have a depth limit in usage and the pearl divers appeared to have explored that use to the limit.

"It is not what we don't know that delays progress, but what we think we know that is not actually so!"

It has to be stressed that this report is confined to the mode of diving in Western Australia in the 1990s. Not all pearl diving in Australia is done in the manner described.

There is diving in the Northern Territory and in Queensland, but the mode of diving is not the same as described here, which is peculiar to Western Australia.

It is also known that prior to the 1990s, there were a number of cases of DCI with neurological symptoms. However, most of these were from pearl farm diving and not from drift diving. A lot of the minor cases, the niggles, were treated with in-water recompression, on air or oxygen or both, and only those cases which required recompression, as judged by the Head Divers, were transferred to either to the Royal Darwin Hospital or to Perth, to HMAS LEEUWIN until 1984 and then to HMAS STIRLING. The RAN treated all diving accidents in Western Australia until late 1989 when Fremantle Hospital opened its Hyperbaric Medicine Unit. The recompression chamber in Broome District Hospital became operational in 1991. It is worth noting that prior to 1987 there was no medical practitioner in Broome with any knowledge of Underwater Medicine. Since 1989 it is one of the pre-requisites for appointment to the Broome District Hospital. Currently, there are 3 Medical Practitioners with Underwater Medicine training in Broome District Hospital, one in private practice and one with the Broome Aboriginal Medical Services.

Acknowledgments

I would like to thank all those who have provided information used in this paper, especially members of the pearling industry, the Pearl Producers Association Inc. WA, pearl divers past and present and the R&D Corporation, Fisheries Department, Western Australia.

Key Words

Occuptional diving, osteonecrosis, oxygen, safety, tables.

References

1 Wong RM. Western Australian pearl divers drift diving. *SPUMS J* 1996; 26 (1) Suppl: 30-35

2 James PB. The size distribution of gas emboli arising during decompression. A review of the concept of critical diameter of gas emboli. *Proceedings of the XIIIth Annual Congress of the EUBS, Lubeck, G ermany, October 1982.* Lubeck: EUBS, 1982

3 Hills BA and James PB. Spinal Decompression sickness: Mechanism studies and a model. *Undersea Biomed Res* 1982; 9 (3): 185-201

4 Gait DJ, Miller KW, Paton WDM, Smith, EB and Welch B The redistribution of vascular bubbles in multiple dives. *Undersea Biomed Res* 1975; 2: 42-49

5 Griffiths HB, Miller KW, Paton WDM, and Smith EB On the role of separated gas in decompression procedures. *Proc Roy Soc London* 1971; B 178:

389-406

6 Daniels S. Bubble formation in animals during decompression. in diving in animals and man. In *The Kongsvoll Symposium 1985*. Brubaak AO, Kanwisher JW and Sundnes G. Eds. Trondheim: Tapir Publishers 1986; 229-264

7 Francis TJR, Pezeshkpour GH and Dutka AJ. Arterial gas embolism as a pathophysiologic mechanism for spinal decompression sickness. *Undersea Biomed Res* 1989; 16 (6): 439-451

8 Hills BA Decompression Sickness: a fundamental study of "surface excursion" diving and the selection of limb bends versus CNS symptoms. *Aerospace Med* 1971; 42 (8): 833-836

9 Sykes JJW and Yaffe LJ. Light and electron microscope alterations in spinal cord myelin sheaths after decompression sickness *Undersea Biomed Res* 1985; 12 (3): 251-258

10 Imbert JP, Fructus X and Montbardon S. Short and repetitive decompression in air diving procedures: The commercial diving experience. In *Proceedings of Repetitive Diving Workshop*. Lang MA and Vann RD. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1991; 63-72

11 Bennett PB. Recreational diving. In *Proceedings of Repetitive Diving Workshop*. Lang MA and Vann RD. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1991; 1.

12 Tikuisis P and Nishi RY. Application of maximum likelihood analysis to repetitive air diving. In *Proceedings of Repetitive Diving Workshop*. Lang MA and Vann RD. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1991; 263-268

13 Vann RD and Thalmann ED. Decompression physiology and practice. In *The Physiology and Medicine of Diving. 4th Ed.* Bennett PB and Elliott DH. Eds. ISBN 0 7020-1589-X. London: W B Saunders Co Ltd.,1993; 376-432

14 Bert P. *Barometetric Pressure* 1878. Translated into English in 1943 by Hitchcock MA and Hitchcock FA. Bethesda, Maryland: Undersea Medical Society, 1978

15 Boycott AE, Damant GCC and Haldane JS. The prevention of compressed air illness. *J Hyg Camb* 1908; 8: 342-443

16 Hill L. *Caisson sickness and physiology of work in compressed air*. London: Edward Arnold, 1912

17 Hills BA. *A Thermodynamic and Kinetic Approach to Decompression Sickness: a 400 page monograph*. Adelaide: Libraries Board of South Australia, 1966

18 Lanphier EH. A historical look at ascent. In *Proceedings of Biomechanics of Safe Ascents Workshop*. Lang MA and Egstrom G. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1989; 5-8.

19 Lewis JE. A review of ascent procedures for scientific and recreational diving. In *Proceedings of Biomechanics of Safe Ascents Workshop*. Lang MA and Egstrom G. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1989; 153-162

20 Wienke BR. *Basic Decompression Theory and Application*. ISBN 0-941332-17-9. Flagstaff, Arizona: Best Publishing Company, 1991

21 Hamilton RW Slow ascents rates: Beneficial but a tradeoff. In *Proceedings of Biomechanics of Safe Ascents Workshop*. Lang MA and Egstrom G. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1989; 79-82.

22 Yount D The physics of bubble formation. In *Proceedings of Biomechanics of Safe Ascents Workshop*. Lang MA and Egstrom G. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1989; 31-44.

23 Van Liew HD. Growth of pre-existing bubbles and gas nuclei in the body during ascent from depth. In *Proceedings of Biomechanics of Safe Ascents Workshop*. Lang MA and Egstrom G. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1989; 47-53.

24 Smith KH. Decompression studies with ultrasound. In *Decompression procedures for depths in excess of 400 feet. 9th Undersea Medical Society Workshop. UMS Report No. WS: 2-28-76*. Hamilton RW Jr. Ed. Bethesda, Maryland: Undersea Medical Society, 1976; 229-236

25 Vann RD. Decompression physiology. In *Proceedings of Repetitive Diving Workshop*. Lang MA and Vann RD. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1991; 187-201.

26 Mano Y. Bubble formation and DCS. In *The Physiological Basis of Decompression. 38th Undersea and Hyperbaric Medical Society Workshop.* Vann RD. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1989; 235-239

27 Hennessy TR. On the site of origin, evolution and effects of decompression microbubbles. In *Proceedings of the International Symposium on Supersaturation and Bubble Formation in Fluids and Organisms*. Trondheim, Norway: *Publisher*, 1989

28 Daniels S. The effects of pressure profile on bubble formation. In *The Physiological Basis of Decomnpression. 38th Undersea and Hyperbaric Medical Society Workshop*. Vann RD. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1989; 197-221

29 Evans A and Walder DN. Significance of gas micronuclei in the aetiology of decompression sickness. *Nature* 1969; 222: 251-252

30 Vann RD, Grimstead J and Nielsen CH Evidence for Gas Nuclei in Decompressed Rats. Undersea Biomed Res 1980; (7): 107-112.

31 Daniels S, Eastaugh KC, Paton WDM and Smith EB.

Micronuclei and bubble formation: A quantitive study using the common shrimp, *Cragnon cragnon*. In *The Proceedings of the Eighth Symposium on Underwater Physiology*. Bachrach AJ and Matzenm MM. Eds. Bethesda, Maryland: Undersea Medical Society, 1984; 147-159

32 Brubbak AO, Peterson R, Grip A, Holand B, Onarheim J, Segal K et al. Gas bubbles in the circulation of divers after ascending excursion from 300 to 250 msw. *J App Physiol Respirat Environ Exercise Physiol* 1986

33 Zannini D. Discussion on Lehner CE and Lanphier EH. Influence of pressure profile on DCS symptoms. In *The Physiological Basis of Decompression. 38th Undersea and Hyperbaric Medical Society Workshop*. Vann RD. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1989; 323.

34 Berhage TE, Vorosmarti J Jr and Bernard EEP. *An Evaluation of Recompression Treatment Tables used throughout the World by Government and Industry*. Bethesda, Maryland: Naval Medical Research Institute, US Department of the Navy. 1978

35 Evans A, Barnard EEP and Walder DN. Detection of gas bubbles in man at decompression. *Aerospace Med* 1972; 43: 1095-1096

36 Pilmanis A. Ascent and silent bubbles. In *Proceedings of Biomechanics of Safe Ascents Workshop*. Lang MA and Egstrom G. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1989; 65-71

37 Le Messurier DH and Hills BA. Decompression sickness: A thermodynamic approach arising from a study of Torres Strait diving techniques. *Hvalradeks Skrifter* 1965; 48: 54-84

38 Kindwall E. Caisson decompression. In *The Physiological Basis of Decomnpression. 38th Undersea and Hyperbaric Medical Society Workshop*. Vann RD. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1989; 375-395

39 Kindwall EP, Baz A, Lanphier EH, Lightfoot E and Seireg A. Nitrogen elimination in man during decompression. *Undersea Biomed Res* 1975; 2 (4): 285-297

40 Hills BA. Limited supersaturation versus phase equilibration in predicting the occurrence of decompression sickness. *Clin Sci* 1970; 38 (Feb): 251-267

41 Gordon G. Staged decompression following no-decompression diving. In *Safe Limits: an International Dive Symposium*. Brisbane, Queensland: Division of Workplace Health and Safety, 1994; 101-106

42 Donald K. *Oxygen and the Diver* ISBN 1 85421 176 5. Hanley Swan, Worcestershire: The SPA Ltd., 1992.

43 Imbert G. Discussion on Shields TG. In *The Physiological Basis of Decomnpression. 38th Undersea and Hyperbaric Medical Society Workshop*. Vann RD. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1989; 399-410

44 BR 2806. Royal Navy Diving Manual. London: HMSO, 1987

45 US Navy Diving Manual. San Pedro, California: Best Publishing Co., 1988

46 ABR 155. Volume 2. Canberra: Australian Government Publishing Service, 1990

47 Butler FK. and Thalmann ED. CNS oxygen toxicity in closed circuit scuba divers 1. In *The Proceedings of the Eighth Symposium on Underwater Physiology*. Bachrach AJ and Matzen MM. Eds. Bethesda, Maryland: Undersea Medical Society, 1984; 15-30.

48 Butler FK and Thalmann ED. CNS oxygen toxicity in scuba divers 2. *Undersea Biomed Res* 1986; 13: 193-223

49 *Joint Draft National Regulatory Model and Draft National Code of Practice for Occupational Diving (revision of AS 2299-1992)*. Sydney: Standards Association of Australia, 1993

50 Imbert JP, and Bontoux M. Production of procedures. COMEX. In *Decompression in Surface-based Diving. 36th Undersea and Hyperbaric Medical Society Workshop*. Hashimoto XX and Lanphier EH. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987; 90-100

51 Edmonds, C, Lowry C and Pennefather J. Diving and Subaquatic Medicine. 3rd Ed. ISBN 0 7506 0259 7. Oxford: Butterworth-Heineman Ltd., 1992: 523-533

52 Fife CE, Pollard GW, Mebane AE, Boso AE and Vann RD. A database of open water, compressed air, multi-day repetitive dives to depths between 100 and 190 fsw. In *Proceedings of Repetitive Diving Workshop*. Lang MA and Vann RD. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1991; 45-54

53 Walder DN and Holloway I Role of oxygen in dysbaric osteonecrosis. In *9th International Symposium on Underwater and Hyperbaric Physiology*. Bove AA, Bachrach AJ and Greenbaum LJ. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987; 613-626

54 Sterk W and Schrier LM. Effects of intermittent exposure hyperoxia in operational diving. In *Proceedings of XIIth Annual Meeting, European Undersea Biomedical Society held at Gotenberg*. 1985; 123-131

55 Hamilton RW, Rogers RE, Powell MR, Vann RD, Dunford R, Spencer MP et al. *The DSAT Recreational Dive Planner. Developement and validity of no-stop decompression procedures for recreational diving*. Tarrytown, New York: Hamilton Research Ltd. and Diving Science and

Technology Inc., 1994
56 Cross ER. Taravana. Diving syndrome in the Tuamotu diver. Kn Physiology of Breath-hold Diving and the Ama of Japan. Rahn H and Yokoyama T. Eds. Washington, DC: National Academy of Science, 1965; 207-219
57 Shields TG, Cattanach S, Duff P, Evans SA and Wilcock SE. *Decompression Sickness arising from Diving at Fish Farms. Final Report*. Aberdeen: Hyperbaric Research Unit, Robert Gordon University, 1993
58 Parker EC, Survanshi SS, Thalmann ED and Weathersby PK. Analysis of the risk of decompression sickness to yo-yo diving using the USN probablistic decompression model. *Undersea and Hyperbaric Med* 1994; 21 (Suppl): 62
59 Sheffield PJ. *Flying after Diving. 39th Undersea and Hyperbaric Medical Society Workshop. UHMS Publication No 77 (Flydiv) 12/1/89.* Kensington, Maryland: Undersea and Hyperbaric Medical Society, 1989
60 Wong RM. *Pearl Divers Diving Safety. Project 91/15.* Perth: Fisheries Research and Developement Corporation, Fisheries Department of Western Australia, Pearl Producers Association Inc., 1994.
61 Walder DN. Adapatation to decompression sickness in caisson work. In *Proceedings of 3rd International Biometerology Congress, Oxford, 1966.* Tromp SW and Weihe WM. Eds. Pau, France: Pergamon Press, 1967; 350-359
62 Kawashima M. Asceptic bone necrosis in Japanese divers. *Bull Tokyo Med Dent Univ* 1976; 23: 71-92
63 Harrison JAB, Elliott DH. *Asceptic bone necrosis in clearnance divers. CRWP 1/74 UPS 4/76. Report prepared for the Underwater Physiology Subcommittee of the Royal Naval Personnel Research Committee.* London: HMSO, 1974.
64 McCallum RI. Bone necrosis due to decompression. *Phil Trans R Soc Lond* 1984; B 304: 185-191
65 Evans A, King JD, McCallum RI, Thickett VB, Trowbridge WP, Walder DN et al. Asceptic bone necrosis in commercial divers. A report from the Decompression Sickness Central Registry and Radiological Panel. *Lancet* 1981; ii: 384-388
66 Ohta Y and Matsunaga H. Bone lesions in divers. *J Bone and Joint Surg* 1974; 56B (1): II-8-204-II-8-217.
67 Kawashima M, Torisu T, Hayashi K and Kaymo Y Avascular bone necrosis in Japanese diving fishermen. In *Proceedings of the 5th International Hyperbaric Congress.* Trapp WG, Bannister EW, Davison AJ and Trapp PA. Eds. Burnaby, British Columbia: Simon Fraser University, 1974; 855-862

Dr R M Wong, FANZCA, Dip D&HM, is a Consultant Anaesthetist at the Royal Perth Hospital and is a Consultant Anaesthetist and Consultant in Underwater Medicine to the Royal Australian Navy. His address is Department of Anaesthesia, Royal Perth Hospital, PO Box X2213, GPO Perth, Western Australia 6001.

DIVER COMMUNICATION SYSTEM

Robert M Wong

The Head Diver can signal to the skipper to tell him to change speed of the vessel, change course etc. by the number of beeps he sends. "Buoy means that there a lot of oysters around and is a request to mark the spot with a buoy, but nowadays withe accuracy of GPS, it is just marked on the chart. To increase speed generally means that the divers are going over a pretty bare patch; whereas, to slow means either the vessel is going too fast or that they are onto good grounds as well. The speed is adjusted by pulling in or letting out the drogue. Adjust weights means that the weights have to be lifted off the sea bed. The divers like to have the weights about a metre off the ground. Each lead weight is 50 kg. Diver up means it is the end of the drift. The bottom time is kept by the Head Diver, when he sends the signal those on board usually just bang on the side of the vessel three times on each side, the sound can be heard by all the divers. The head diver is usually on the inside of the starboard side of the drift line.

The illustration shows the signals used on board the ODIN II.

Key Words
Communication, occupational diving.