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South Pacific Underwater Medicine Society

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EDITORIAL

There are probably too many people about who believe in restricting their interest to matters which directly concern them, discarding or ignoring information which is not obviously relevant to their immediate problems. The tunnel vision approach can be readily observed in Diving, as in other disciplines. Good recent examples have been the conspicuous failure of those involved in debating the problems of Deep Rescue Practice (BS-AC) and Emergency Ascent Training (US diving organisations and UMS) simultaneously (or previously) to discuss the possible reasons for the diver being in need of the proposed procedure. The limitation of interest to that immediate concern is apparent in many reports of treatment centres concerning the cases of Air Embolism and Decompression Sickness they receive, little curiosity being shown in most cases as to the why of their occurrence. While admittedly their immediate and urgent need is to reach a working diagnosis and institute treatment in a patient only too ready to give an "edited" version of events and then escape from attention and questioning, nonetheless there has been too great a willingness to apportion blame rather than seek predisposing factors such as the victim's experience of good luck on previous similar dives, the existence of fatigue or cold, liking for risk, faulty or unused depth and time gauges, etc. etc. At the Diving Officers' Conference of the BS-AC in 1978 Surgeon Commander Ramsay Pearson RN, took the line that divers should now be aware of the fallibility of the Tables, bends occurring on occasion even when care is taken over the dive profile. Possibly trouble results from the frequent lack of significant symptoms despite violation of advised diving times, and the lack of personalisation of the reports. Nobody is afraid of "Type II Bends" but most would cry shy of the symptoms justifying such a diagnostic label. Too few reports are published by those treating DCS victims. This is not a plea for horror reports but for better communication.

An excellent review of present views on DCS is given by Dr Charles Brown, and the lesson is rounded out by the case discussion paper he also presents. Although his papers were prepared for a lay audience they are highly appropriate to our pages also, for he evidently subscribes to the belief that an educated diver has a right (need?) to be well informed concerning diving medicine. One of the New Zealand cases reported in this issue may seem to confirm this viewpoint! Readers may notice the difficulty which is often present in deciding between DCS Type I and II, or Air Embolism, in the actual clinical situation. The New Zealand cases, for which credit in reporting is due to the New Zealand Underwater Association and Dr Adair, could serve as a basis for a useful seminar on diving problems, and illustrate the value of case reports. The fatalities remind us that the diving risks continue until safely in the boat or ashore, and that cardiac problems can strike at 20 years as well as in the older age group (the actual autopsy reports have not been seen). The DCS cases possibly indicate the existence of a general diving pattern of disregard for The Tables, a serious educational problem. In the UK the RN advice has been (rather surprisingly) that divers with possible symptoms which are "non urgent" should contact their own doctor rather than the RN experts. Surgeon Commander Pearson has no doubt good reasons for this advice, but it could lead to problems should the victim be only too happy to be reassured as to his condition by a physician unversed in diving medicine. It is better to be growled at for being neurotic about an acne than commiserated with when delayed treatment is incompletely effective. Case 12 from New Zealand is a beautiful cameo of a report, highly relevant to diving safety concepts.

The paper by Dr Otto Molvaer has an importance far beyond the conventional boundaries of Diving Medicine and illustrates the importance of giving consideration to the "off-cuts" which inevitably occur when considering available information for publication or discussion. He presents evidence that hearing loss occurs with spring board diving and such water activities as free diving and underwater polo. The unilateral and limited nature of the loss of hearing explains the (probable) under-reporting of this damage among "divers" of the various disciplines. The paper can be regarded as a tocsin, a warning to those responsible for the health of sports people that there may be a hitherto unsuspected danger to the long term health of their charges.

It is a pleasure to present Commander Warner's annual paper to the International Diving Symposium, given earlier this year in the USA. He is able to report that the fatality rates for North Sea divers have been reduced to the level experienced by many other industrial workers. Study of morbidity reports will hopefully better define the lesser dangers and ensure that they are similarly reduced: it should be possible to become safer than coal-face working.

In the next issue we hops to present reports from the Singapore meeting, which promises to be an exciting and informative convention.

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ACUTE HEARING LOSS FOLLOWING DIVING INTO AND IN WATER

OI MOLVÆR

Norwegian Underwater Institute

SUMMARY

Acute and often permanent inner ear injury from water-related activities involving rapid changes of pressure in the middle ears may be more common than is generally believed.

25 cases are reported to illustrate the relatively minor trauma experienced by victim, which however results in permanent inner ear damage.

Cases are described resulting from diving into water (3), free diving (2), playing underwater polo (free diving) (1) , riding an underwater sledge (SCUBA diving) (1), ordinary SCUBA diving (17) and hard hat diving (1).

Decompression sickness was not implicated in these cases. The value of a baseline audiogram is stressed, and a base-line vestibular test is also desirable.

INTRODUCTION

The occurrence of inner ear barotrauma during diving with transient or permanent injury to the cochlea and/or the vestibular apparatus has become widely known during the last decade[13].

At the invitation of Dr Douglas Walker, The Editor of the SPUMS Journal, I have collected the case histories and audiograms of the 25 patients with diving induced hearing loss which appears to be due to inner ear damage that I have treated over the past ten years. As a result many of the cases have been reported elsewhere, though mostly only in summary. The details of the previously reported cases are as follows.

Cases 1-12 have been published in summary previously in Norwegian (The paper has an English summary).[6] Cases 13-14 have been described in detail in English elsewhere.[5] Cases 15-23 also have been published in English previously, case 15 in detail and cases 16-23 summarily in a table.[7] Cases 1 and 15 also have been briefly mentioned in a short survey article in English.[8] Case 1 also was described in English in another paper.[4] Cases 24 and 25 have not been published before.

In the audiograms which accompany the case reports the following conventions apply.

The first audiogram is indicated with crosses, the second with circles, the third with dots, and the fourth with squares. Arrows pointing downwards below a symbol mean that the hearing loss was greater than the loss indicated by the symbol, ie. beyond the capabilities of the audiometer.

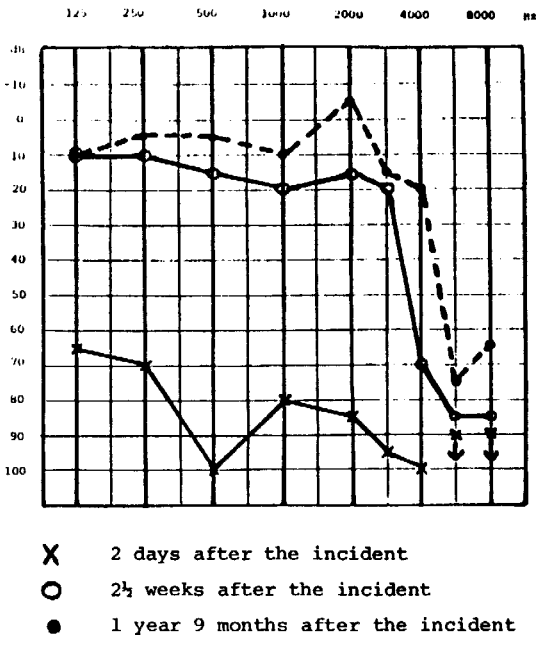
Post operative audiograms have dashed lines.

CASE REPORTS

Case 1

A 21 year old man made a free dive to the bottom of a 4 metre deep swimming pool. At the bottom he experienced fullness and tinnitus in his right ear, but subjectively he became free of symptoms after a while. Before this incident his audiogram was normal and after he had a notch in the high frequency range. In 1974 he did the same dive in the same place and the same thing happened. This time he was examined by an audiologist and his audiogram is shown in Figure 1. After a week he still had a large hearing loss and was admitted to our ward where we kept him in bed for a week. As he still had the bad loss in the high frequency range, which could influence his career as a jet pilot, we explored his right middle ear and found a perilymphatic fistula in the oval window. The fistula was closed surgically, and his hearing became normal through all frequencies up to and including 4 kHz.

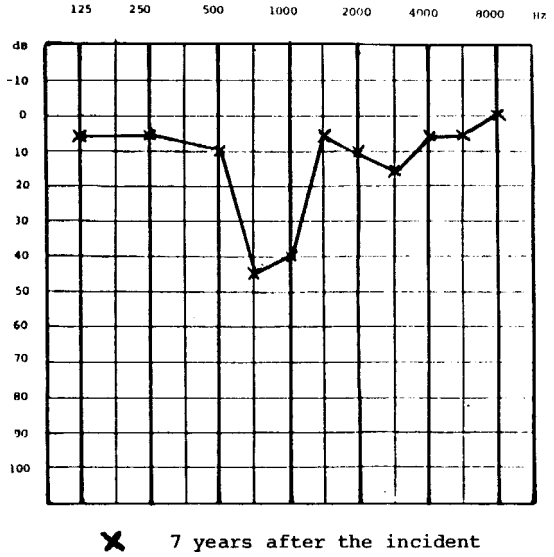
Figure 1 RIGHT EAR CASE 1



Case 2

A 19 year old man experienced left sided tinnitus during ascent from a free dive to about 10 metres depth. Tinnitus lasted for 3 months. Before the dive his audiogram was normal. After the dive he had a sensorineural notch at 1 kHz. His last audiogram, 7 years after the incident had a sensorineural notch between 500 and 1500 Hz (Figure 2).

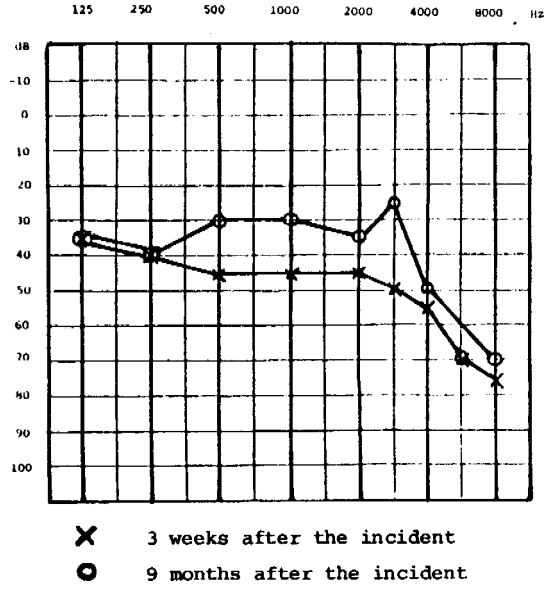
Figure 2 Left EAR CASE 2



Case 3

A 15 year old boy plunged head foremost into the water from 1 metre height. The depth was about 2 metres. He experienced fullness in his right ear, felt unsteady, unwell and nauseated so he took to his bed for a day. After one and a half days he felt well except for the sensation of fullness in his right ear. This still was the case when we saw him three weeks after the incident. His audiogram then showed a sensorineural loss (see Figure 3). Nine months after the incident his audiogram showed some improvement, but still with a sensorineural loss.

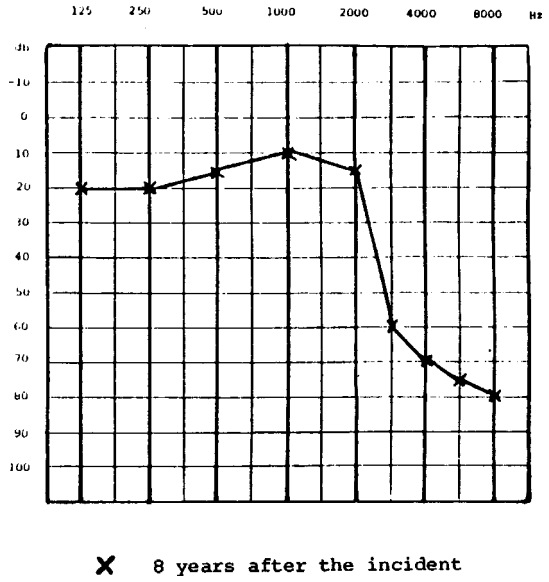
FIGURE 3 RIGHT EAR CASE 3



Case 4

A 28 year old man plunged head foremost from 5 to 6 metres height and free dived to the bottom. He does not know how deep he went, but he had difficulties in reaching the bottom. Afterwards he had a sensation of water in his ears and experienced vertigo. Next day he woke up with left sided tinnitus which persisted. Three months later he saw a doctor who gave him vasodilating tablets without any effect. Ten years later he still has the bothersome tinnitus. Before the dive he judged his hearing as normal and he had no ear symptoms. Now he can no longer hear high frequency sounds in his left ear and audiometry 5 years after the incident showed a sensorineural left sided loss in the high frequency range, unchanged three years later (Figure 4).

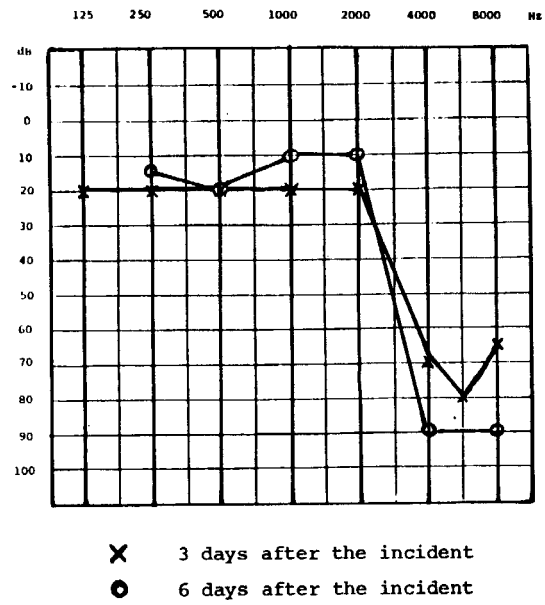
FIGURE 4 LEFT EAR CASE 4



Case 5

A 34 year old man, a sports diver, performed 2 open sea SCUBA dives. The first to 28 metres with a bottom time of 8 minutes. After a surface interval of 10 minutes he went down to 20 metres for 21 minutes. He had difficulties with pressure equilibration to his middle ears, and had to do forceful Valsalva manoeuvres several times. Immediately after surfacing after the last dive he experienced reduced hearing and tinnitus of the right side. Later in the day he became increasingly nauseated, experienced vertigo and vomited. He had to stay in bed the rest of the day. The symptoms ameliorated gradually. When he saw a doctor a couple of days later he still had reduced hearing, tinnitus, nausea and spinning vertigo which became worse when he changed position and closed his eyes. He was unsteady during Romberg's test and the 'finger-nose' test was not completely normal. His eardrums were slightly congested and his audiogram showed a large hearing loss in the high frequency range on the right side (Figure 5). He was treated with hyperbaric oxygen and after 10 minutes the nausea and vertigo became less bothersome and the tinnitus changed character. After the treatment he felt better and Romberg's test was normal as well as the 'finger-nose' test. Three days later he was seen by an audiologist who found a large sensorineural high tone loss on the right side (Figure 5).

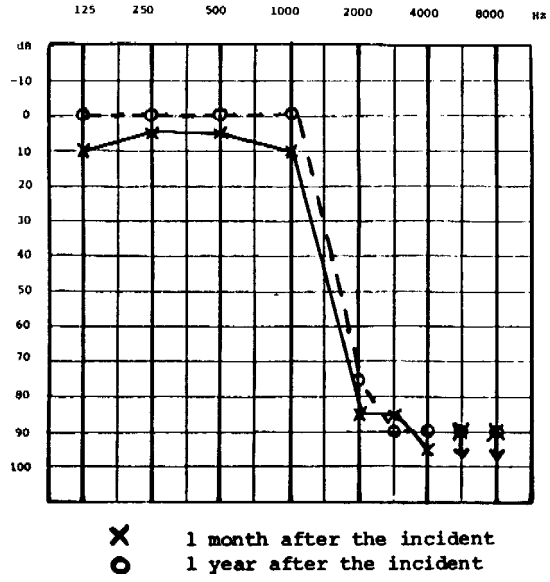
FIGURE 5 RIGHT EAR CASE 5



Case 6

A 25 year old man, a sports diver, did a 20 metre open sea SCUBA dive. At a depth of 2 metres during the descent he experienced pressure in his right ear and could not equalize the pressure in the middle ear. Therefore he returned to the surface and made another attempt. He then reached 20 metres where he remained for less than half an hour before he returned to the surface. When he came ashore he experienced fullness in his right ear and later tinnitus. The next day he saw a doctor who found his right eardrum slightly retracted and some fluid in the middle ear. As the tinnitus and reduced hearing still was present one month later he saw a doctor again and an audiogram showed a big sensorineural high tone loss on the right side (Figure 6). One month later the hearing had not improved and he was admitted to an ENT ward where his right middle ear was explored surgically. No perilymphatic fistula was found, and an audiogram one year after the incident showed no improvement (Figure 6).

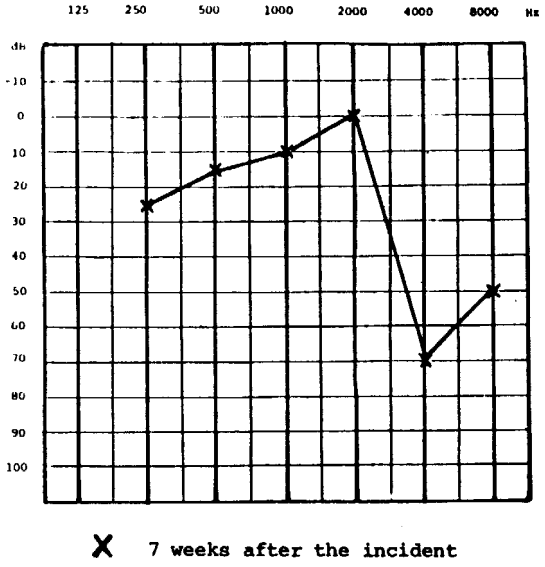
FIGURE 6 RIGHT EAR CASE 6



Case 7

A 36 year old man dived with SCUBA gear to 15 metres in the sea for 60 minutes. He went up and down between the bottom and the surface about 6 times and all the time had difficulties with the pressure equalization to the middle ears. When he reached the surface after the dive he experienced pressure in his right ear, reduced hearing and tinnitus. As the symptoms lasted he saw a doctor a little less than a week later and a sensorineural hearing loss in the high frequency range on the right side to 80 dB at 4kHz was found. He was treated with hyperbaric oxygen and 500 ml of Dextran iv without any effect. Afterwards he was checked by an audiologist who found a sensorineural high tone loss on the right side (Figure 7).

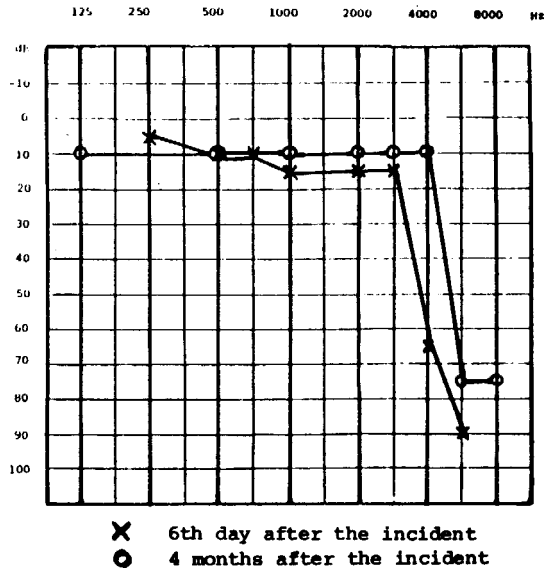
FIGURE 7 RIGHT EAR CASE 7



Case 8

A 29 year old man performed an open sea SCUBA dive to 25 metres. During the dive he got a sensation of fullness and tinnitus in his right ear and experienced transient vertigo.

FIGURE 8 RIGHT EAR CASE 8



As his ear symptoms continued he saw a doctor 6 days later and a large sensorineural hearing loss in the high tone range was found on the right side (Figure 8). One month previously a screening audiogram at 20 dB level had shown normal hearing. Because his trouble did not clear up he was seen by an audiologist a week later and his audiogram was unchanged. Four months after the incident hearing had improved remarkably at 4 kHz, but still he had a big loss for 6 and 8 kHz (Figure 8).

Case 9

A 27 year old man performed an open sea SCUBA dive to 40 metres depth. The bottom time did not require decompression stops according to the diving tables he was using. He had trouble with pressure equalization to his middle ears during the descent and made forceful Valsalva manoeuvres. Before the dive his hearing was normal but afterwards he had a feeling of fullness in his ears, tinnitus and subjectively reduced hearing. A week later he saw a doctor who found a bilateral hearing loss (Figure 9a and 9b).

FIGURE 9a RIGHT EAR CASE 9

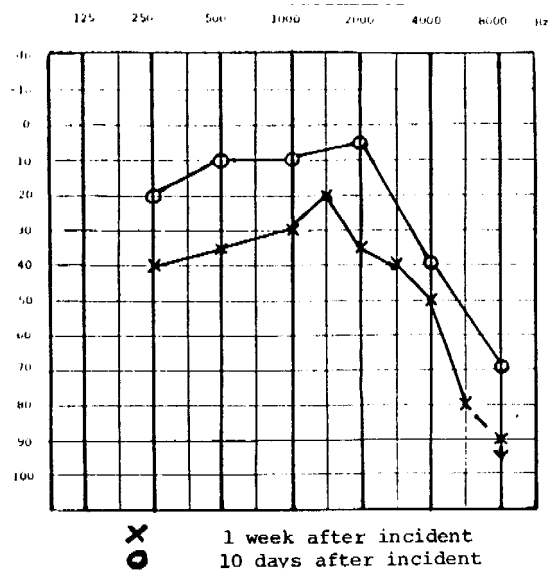
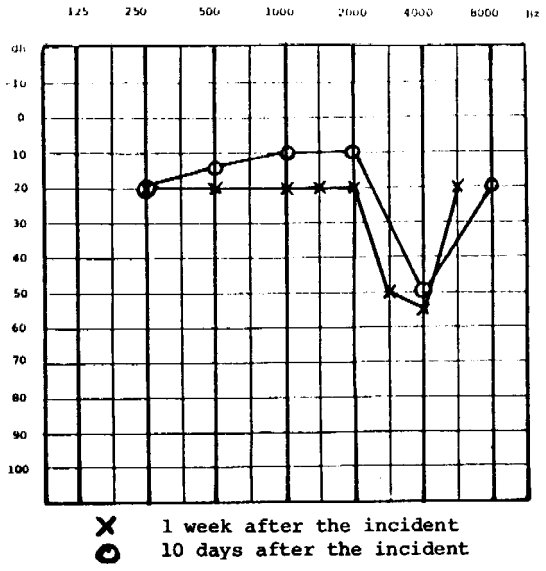


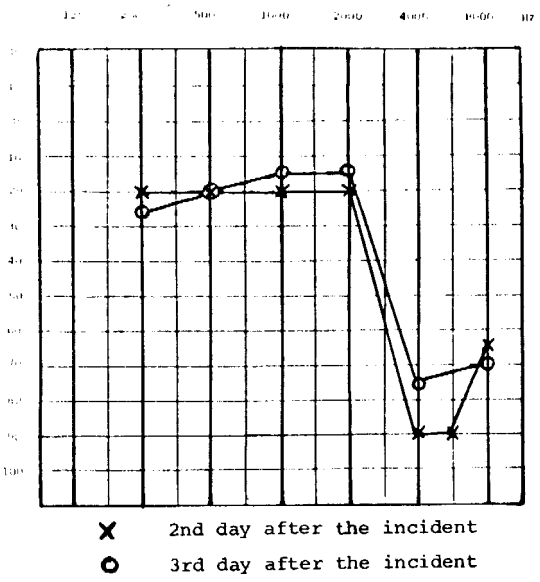
FIGURE 9b LEFT EAR CASE 9



Case 10

A 26 year old professional diver performed 2 sea dives in standard hardhat equipment. The first dive was to 46 metres depth with a bottom time of 9 minutes and surface decompression. Four minutes after surfacing he got a feeling of fullness in his left ear and experienced vertigo. Nevertheless, he dived again one and a half hours later to 9 metres depth for 40 minutes. His symptoms continued. A couple of hours after the last dive he complained of reduced hearing. The next day he experienced transient nausea, variable vertigo and a constant feeling of fullness in his ear. He was seen by a doctor, and 2 days later fullness in the ear was his only symptom and his audiogram showed a big sensorineural high tone loss in the left ear (Figure 10). The loss at the highest frequencies was too large to be measured by the available equipment. He was given hyperbaric oxygen therapy plus 500 ml of Dextran iv. After that his feeling of fullness in the ear was less and his high tone hearing had become measurable (Figure 10). The third day after the dive he was seen by the audiologist who verified the sensorineural high tone loss on the left side. His hearing had been normal before the dive.

Figure 10 LEFT EAR CASE 10



Case 11

A 28 year old man made an open sea SCUBA dive to 30 metres. During descent he experienced difficulties with pressure equalization to the middle ears.

Afterwards he had a feeling of fullness and tinnitus in his ears and performing a Valsalva manoeuvre triggered slight vertigo. When examined 10 days later his pure tone, SRT and Bekesy audiogram were normal, but caloric testing showed right sided canal dysfunction. Three weeks later this had normalised, but even 7 weeks after the incident he still experienced fullness in his ear and tinnitus.

Case 12

A 19 year old diving student in the Navy experienced increasing difficulties with pressure equalisation to the middle ears during a ride on an underwater sledge. Before the dive he had a slight discharge from his nose. The sledge went much up and down between 10 and 15 metres and the greatest depth reached was 20 metres. The bottom time was 26 minutes. No decompression was necessary. Immediately after surfacing he experienced fullness in his right ear and a quarter of an hour later vertigo, slight nausea and increasing tinnitus with high frequency. He thought he had become seasick and went to bed. When his right-sided hearing loss and tinnitus persisted the next morning he saw a doctor and the audiogram showed reduced hearing on both ears (Figure 11 and 12), worse on the right side. He was immediately recompressed with hyperbaric oxygen. During the treatment the hearing loss was subjectively reduced and so was the tinnitus. An audiogram taken shortly after the treatment showed reduction of the hearing loss, which was confirmed by an audiologist the next day (Figures 11 and 12).

FIGURE 11 RIGHT EAR CASE 12

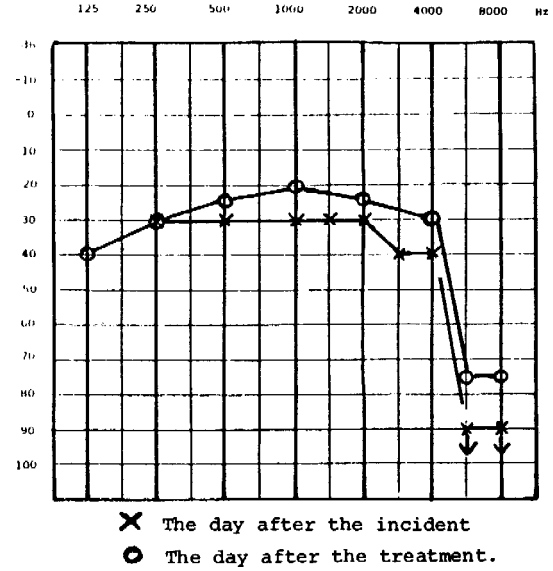
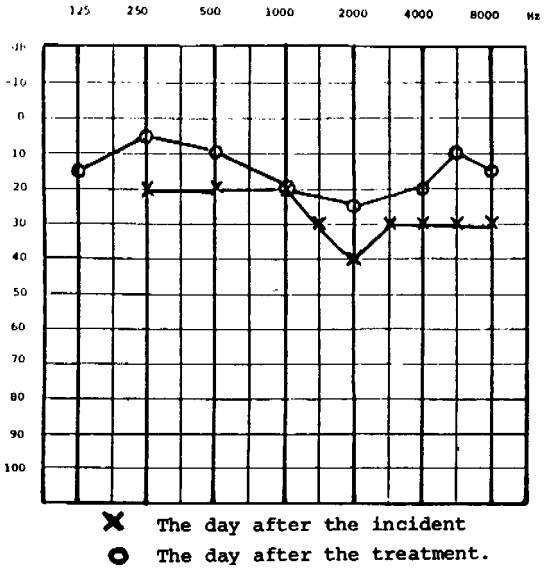


FIGURE 12 LEFT EAR CASE 12

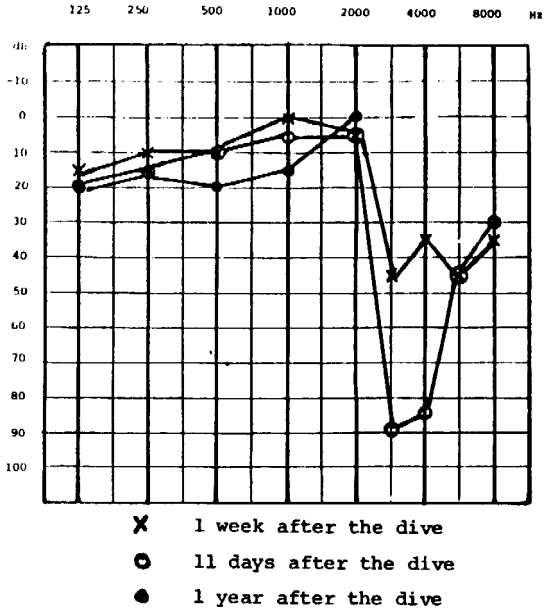


Before the dive a screening audiogram at 20 dB level was normal except for a dip to 25 dB at 8 kHz on both sides.

Case 13 (previously reported as diver 1 in reference 5)

A 21 year old female nurse with 1 year of diving experience performed an open sea, no decompression, SCUBA dive to 37 metres depth. She had trouble with pressure equilibration to the middle ears with pain in the right ear. She made several forceful Valsalva manoeuvres. On coming ashore she experienced spinning vertigo, tinnitus and reduced hearing in her right ear. The vertigo subsided gradually, but the tinnitus and hearing loss persisted. A week later she saw a physician who found a sensorineural high tone loss in her right ear (Figure 13). Four days later the loss was greater (Figure 13) and after a year she had no detectable hearing in the high tone range in that ear (Figure 13). Caloric responses were normal. She has stopped diving.

FIGURE 13 RIGHT EAR CASE 13



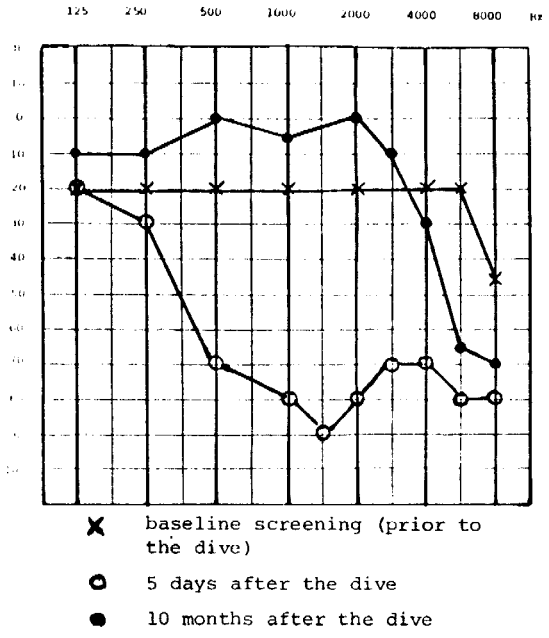
Comment: In this case the hearing loss progressed, while usually it will ameliorate to a certain extent shortly after the acute phase.

Case 14 (previously reported as diver 2 in reference 5)

A 23 year old male Navy SCUBA diver performed an open sea, no decompression, dive to 15 metres. During the dive he suddenly experienced vertigo. His right ear felt plugged combined with tinnitus and reduced hearing. The symptom subsided in the course of the day, but the following night he woke up suddenly with spinning vertigo, retching and a cold sweat. He had horizontal nystagmus grade III to the left, fluid in the right middle ear and a big sensorineural hearing loss (Figure 14). Good caloric responses were obtained from both labyrinths.

His symptoms subsided gradually, but even 10 months after the dive he had tinnitus and hearing loss (Figure 14). He stopped diving.

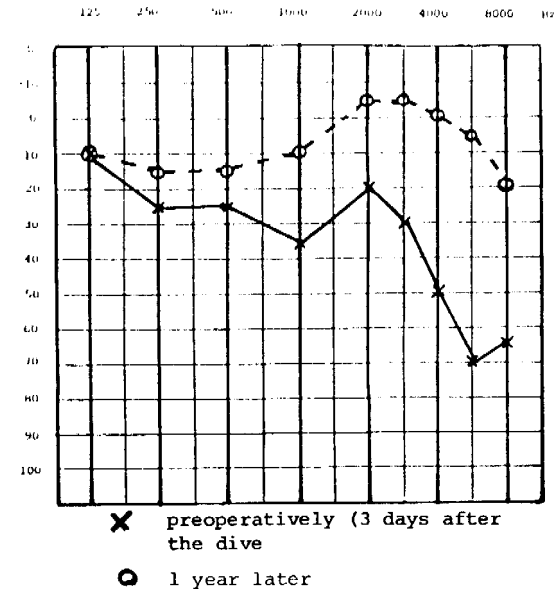
FIGURE 14 RIGHT EAR CASE 14



Case 15 (previously reported as diver 1 in reference 7)

A 21 year old SCUBA student experienced severe difficulty with pressure equilibration to the middle ears during descent during his first open sea dive. He suffered haematotympanon on one side, and rupture of the tympanic membrane and an oval window fistula on the other. Both were successfully repaired surgically with an excellent result (Figure 15).

FIGURE 15 LEFT EAR CASE 15

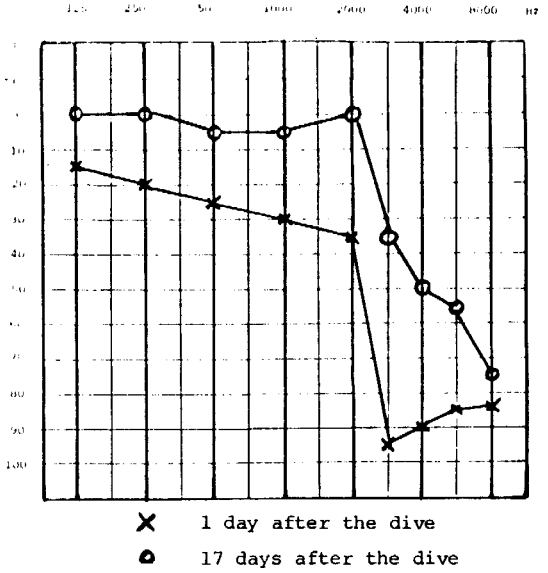


Case 16 (diver 2 in reference 7)

A 23 year old male medical student who suffered from hay fever, had six months experience as a SCUBA diver. He made a series of 3 poorly planned and performed repetitive dives to a maximum depth of 25 metres. There was some trouble with pressure equilibration to the ears during descent. On coming ashore he experienced reduced hearing and tinnitus in his left ear, spinning vertigo, nausea and vomiting. His sensorineural high tone loss improved during bed rest, but the lasting loss was significant (Figure 16). The response to caloric stimulation was slower on the affected side than on the right.

As he used neither a watch nor a depth gauge it is difficult to figure out his exact dive profiles. It is likely that he should have carried out an 8 minute decompression stop at 3 metres on his last dive. This calculation was not made until 5 days after the incident. He had no other symptoms indicating decompression sickness, and symptoms from the inner ear only are very rare (even unheard of?) in decompression sickness from shallow air dives. Consequently we concluded that this was not a case of decompression sickness, and recompression therapy was not tried.

FIGURE 16 LEFT EAR CASE 16

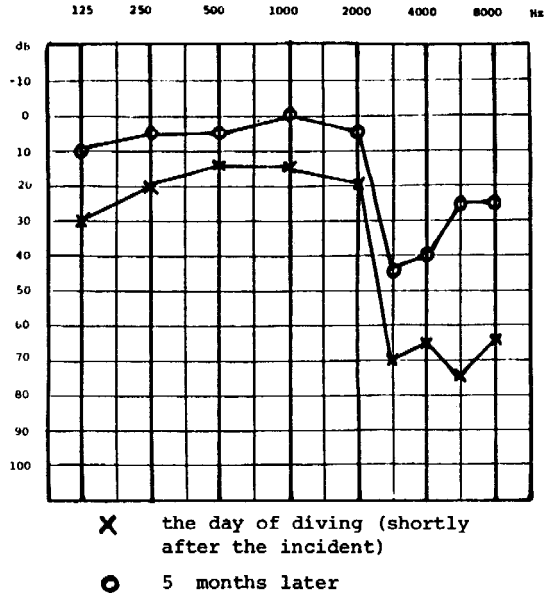


Case 17 (diver 3 in reference 7)

A 33 year old male SCUBA diver who, 3 weeks before the incident that brought him to our notice, experienced marked vertigo with a tendency to fall to the left after diving. This subsided in the course of a couple of days. On the second occasion he experienced acute spinning vertigo after repetitive dives to 18 metres. He needed assistance to get ashore, where he was unable to stand without support. There was no spontaneous nystagmus. There was no sign of barotrauma to the middle ears. However there was sensorineural hearing loss in the right ear (Figure 17). After 1 day of bed rest in hospital ENG showed normal vestibular responses on both

sides, and there was no Rombergism. When discharged after 4 days he still had tinnitus, and even 5 months later an audiogram showed evidence of permanent cochlear injury (Figure 17).

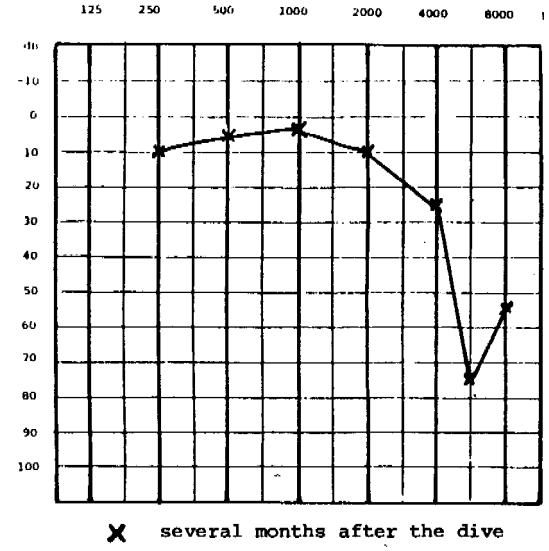
FIGURE 17 EIGHT EAR CASE 17



Case 18 (diver 4 in reference 7)

A fire brigade diver without previous ear symptoms had difficulties with pressure equilibration to the middle ears during a shallow SCUBA descent and performed several forceful Valsalva manoeuvres. He then experienced reduced hearing, vertigo, ringing and pain in his right ear. The tinnitus lasted for 3 months, and his sensorineural high tone loss persisted (Figure 18).

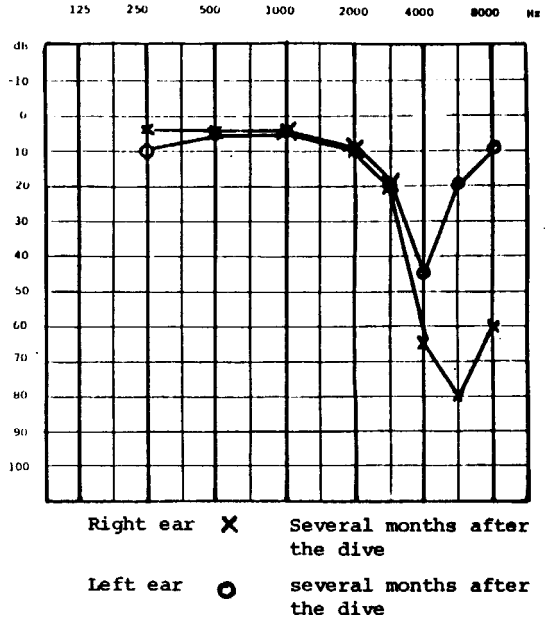
FIGURE 18. RIGHT EAR CASE 18



Case 19 (diver 5 in reference 7)

A fire brigade diver without previous ear symptoms had trouble with pressure equilibration to the middle ears during a shallow SCUBA descent, and performed several forceful Valsalva manceuvres. He experienced lasting tinnitus and reduced hearing for high frequency tones in both ears. His sensorineural high tone losses are shown in Figure 19.

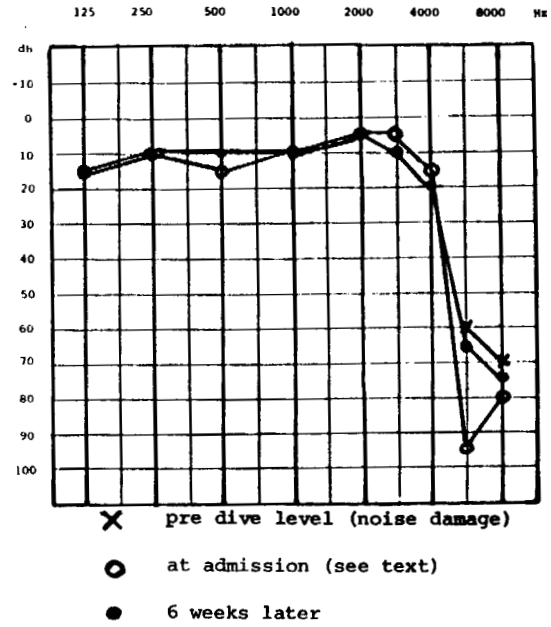
FIGURE 19 EIGHT AND LEFT EARS CASE 19



Case 20 (diver 6 in reference 7)

A professional diver with 10 years experience who already had a “noise notch” in the audiograms for both ears. Five weeks before the hospital admission he experienced acute spinning vertigo shortly after leaving the surface. He continued to dive daily in spite of the same experience each dive. Sometimes the vertigo subsided so that he could perform his task, but sometimes it forced him to abort the dive. The vertigo would now and then last for several minutes after coming ashore. He had the same experience during dry, simulated test dives in a hyperbaric chamber. After one week in hospital he was symptom free and managed well in a shallow test dive.

FIGURE 20 LEFT EAR CASE 20

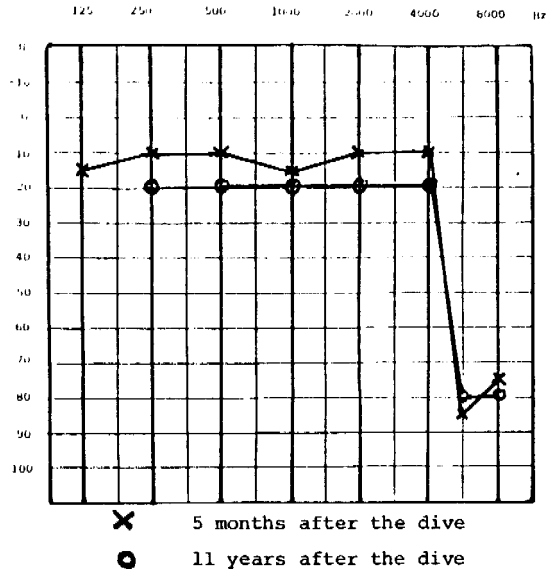


Comment: He had no tinnitus and no Rombergism on admission, and no explanation to his trouble was found. His ears were not explored surgically, so we do not know whether he had a perilymphatic fistula. His main complaints were vestibular, but his hearing deteriorated on the left side during the actual episode, and did not quite recover to the previous level (Figure 20).

Case 21 (diver 7 in reference 7)

A 21 year old Navy diver with a normal screening audiogram at 20dB level when he entered the Navy. He performed a no decompression sea dive with nitrox (20 metres for 20 minutes) in spite of having a cold and had great difficulties with pressure equilibration to his middle ears during descent, wherefore he performed several forceful Valsalva manoeuvres. Suddenly he got a feeling that "something" happened to his right ear. Afterwards he experienced tinnitus and felt as if the ear was plugged. He was not dizzy. He did not see a physician at this time. His next audiogram 5 months later showed a dramatic change (Figure 21) with a severe loss at 6 and 8 kHtz. Since then he has experienced tinnitus periodically, and even 11 years after the incident he still had a considerable sensorineural high tone loss in that ear (Figure 21).

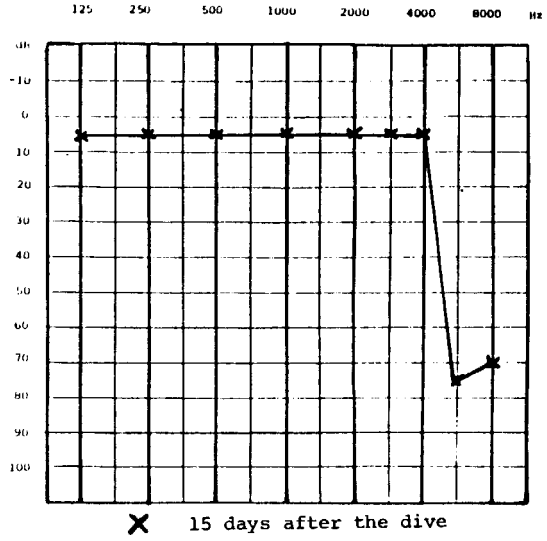
FIGURE 21 RIGHT EAR CASE 21



Case 22 (diver 8 in reference 7)

A 31 year old male underwater polo player who during a game experienced pain and vertigo and felt as if his right ear became plugged. He was forced to surface and abandon the game. The spinning vertigo lasted for the rest of the day, but the plugged sensation, high pitched tinnitus and distortion of hearing continued. He therefore saw a physician 15 days later, and a marked sensorineural high tone loss was found in his right ear (Figure 22).

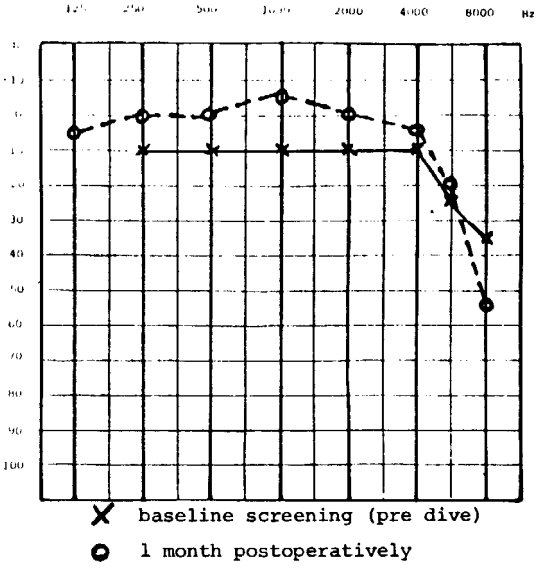
FIGURE 22 RIGHT EAR CASE 22



Case 23 (diver 9 in reference 7)

A 20 year old male dived into the water from a 5 metre high spring-board and experienced acute hearing loss, tinnitus and vertigo which was made worse by Valsalva manoeuvres. An exploratory tympanotomy revealed a round window fistula in his left ear, which was closed with fat. Postoperatively his vertigo disappeared and nystagmus could not even be elicited by the Valsalva manoeuvre. One month later his hearing had still not reached pre-incident level (Figure 23), but his main trouble, the vertigo, had completely disappeared.

FIGURE 23 LEFT EAR CASE 23

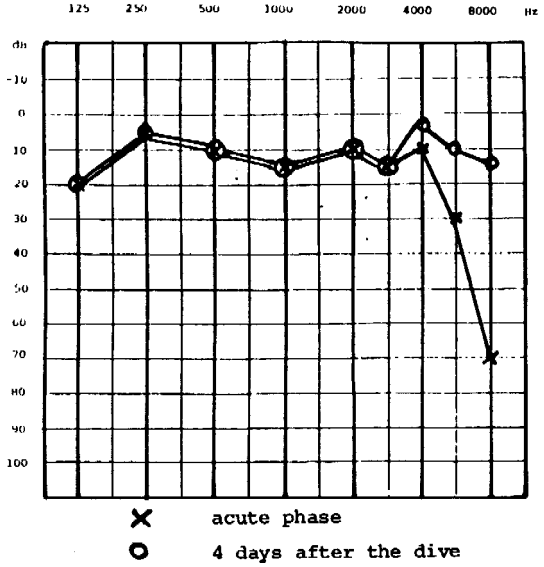


Case 24

A 24 year old male professional diver had, because of a cold, severe trouble with pressure equalization to the ears during a 10 minute dive to 30 metres. He experienced vertigo and nausea and coming ashore he could not stand unaided because of spinning vertigo, vomiting and head-

ache. He fell toward the right and had nystagmus of third degree to the left. He also complained of right-sided tinnitus, and had a sensorineural high tone loss in that ear (Figure 24). Although the dive profile did not indicate decompression sickness as likely he received a therapeutic recompression in the nearest hyperbaric chamber, and was then admitted to the local ENT-ward. After 4 days of bed rest he was discharged symptom-free, with normal pure tone hearing (Figure 24) and no loss of speech discrimination. Caloric vestibular tests were also normal.

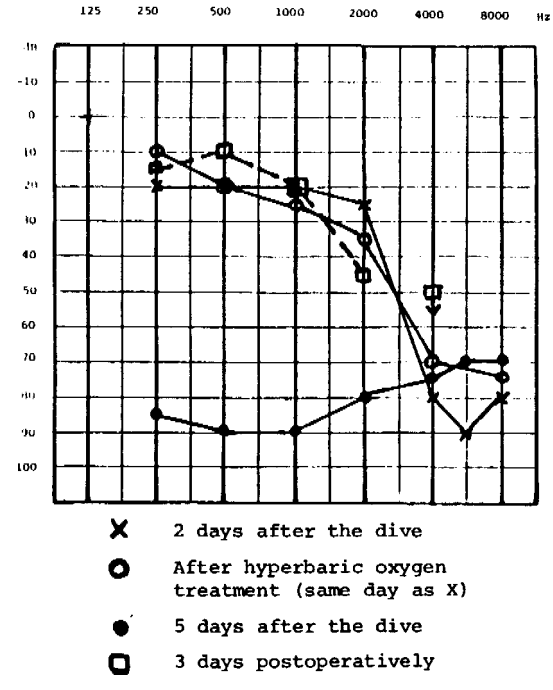
FIGURE 24 RIGHT EAR CASE 24



Case 25

A 26 year old sport SCUBA diver with a couple of years of experience. He had been exposed to noise as an aircraft mechanic. He always had to perform Valsalva's manoeuvre during descent and would always experience a feeling of plugs in his ears for a short while after a dive. This specific dive to 15 metres for half an hour was uneventful. The plugged feeling in his right ear did not clear after the dive, and he also experienced tinnitus periodically. Therefore he saw a physician 2 days later, and a marked sensorineural high tone loss was found in his right ear (Figure 25). Hyperbaric oxygenation was tried, but the hearing was little affected by this (Figure 25). After 3 days of bed rest his hearing had deteriorated dramatically in the middle and lower frequencies (Figure 25). He experienced diplacusis, his speech discrimination score was poor and Fowler's ABLB test showed positive recruitment. An exploratory tympanotomy revealed a round window fistula that was closed with lyo-dura. Three days postoperatively his bone conduction hearing (with surgical packing still in his ear canal) showed normal hearing in the low frequencies, but still a considerable sensorineural high tone loss (Figure 25). His speech discrimination score however was 100. (This case is completely "fresh" and will be followed up further).

FIGURE 25 RIGHT EAR CASE 25



DISCUSSION

Many of the cases did not see a physician until a long time after the incident, a fact that affects the prognosis unfavourably. The chances are that many others do not see a doctor at all. To evaluate a case of hearing loss properly a baseline audiogram is essential, and a baseline vestibular test is highly desirable.

All these patients will probably benefit from bed rest, and some will need surgical treatment. The place of hyperbaric oxygen treatment in these cases is disputed. In most instances the victim will manage pretty well after a while because both ears seldom are affected, but a residual sensorineural high tone loss is the rule.

An injured inner ear seems to be more vulnerable than a healthy one, and thus a person with for instance a noise damage may be more susceptible than others to diving related inner ear injury. Therefore at least sport divers experiencing diving injuries to their inner ears should consider giving up diving.

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SPUMS COMMITTEE MEETING
Held at 25 Hastings Road, Frankston, 27th March, 1980 at 1930 hours.

ATTENDANCE: John Knight, Bill Hurst, Victor Brand, Chris Lourey.

APOLOGIES: Bill Rehfisch, Douglas Walker, Daryl Wallner.

MINUTES OF PREVIOUS MEETING:
Read and accepted as correct.

BUSINESS ARISING:
Col. Jimmy How (Singapore Armed Forces) coming to Australia in May 1980. Decided to host the visitor at dinner with the committee members.

CORRESPONDENCE: Letters from:

Dr Paul Webb	-	President-elect UMS
Dr Ken Kizer	-	President - HUMA
Dr C Schilling	-	Secretary - UMS

Plus general correspondence between the Secretary and participants in the 1980 AGM.

TREASURERS' REPORT:
Current balance as at 21st March, 1980 - $3,792.27.
The Savings Interest and Investment accounts the same as previous and both with the addition of accrued interest.

PRESIDENT'S REPORT:
Reported correspondence from Vice-Admiral, Sir John Rawlins re attendance at 1980 AGM.

SECRETARY'S REPORT:

1. 1980 AGM plans and attendance progressing satisfactorily.
2. All committee members to give recommendations as to a suitable site for the 1981 AGM. With this in mind Dr A Slark (NZ) to be approached re the consideration of NZ members.
3. Discussion to continue with regard to future conference organization. Suggested that at least one person (not necessarily the secretary) be responsible for organizing the AGM.

NEWSLETTER:

1. The next newsletter is about to launch into print.

2. Dr John Knight to post the previous newsletter to new members.

3. Reported that the current arrangement between the Editor and the President is working well. Decided to continue this arrangement.

4. Dr D Walker (Editor) to be advised to submit application for petty cash to reimburse general expenses as well as postage etc.

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE:

1. Letter from CO HMAS Penguin advising that the course will in future be limited to five civilian members only.

2. Letter from Dr I Unsworth advising that the 1980 one week course in Hyperbaric medicine will not occur.

3. The Secretary reported a phone call from Captain R Grey - Navy Office Canberra, where it was stated that some discussion is being undertaken to consider deleting the Hyperbaric medicine section from the Diploma. No action taken - awaiting documented evidence and discussion from the Navy.

4. Letter from Dr Bruce Logan outlining his experience and enquiring whether this met the requirements of 6 months full time or equivalent part time experience required for the Diploma. Referred to the Diploma Committee.

GENERAL BUSINESS:
The discussion and plans to have a National Network to be raised at the AGM.

FUTURE MEETING:
Date to be decided.

Meeting closed 2200 hours.

DR CHRISTOPHER J LOUREY
SECRETARY - SPUMS

* * * * *

FREE ASCENT AND "DUMB" ANIMALS

In the UMS hosted Workshop on Emergency Ascent Training (1977), to he discussed in a later issue, Dr Eric Kindwall included the following information:-

"To demonstrate this point (that the key to successful emergency ascent was to be sufficiently relaxed in the water), Dr Charles Shilling, who was supervisor of Submarine Escape Training at the New London Escape Tower in the 1940s, took a mongrel dog to 100 feet in the roving bell and threw him out into the water. Dr Shilling reports that the dog swam towards the surface, exhaling all the way, as he followed behind in the roving bell. The dog performed beautifully with no previous escape training, did not embolise, and remained Dr Shilling's pet for many months. (Admittedly the dog had been supplied by Harvard University, so he may have been smarter than some)."

THE CAUSATION OF PERILYMPH FISTULAE IN DIVERS

Paul Donoghue and John Knight

On 23 December 1979 a 23 year old man went swimming in a lake. He had no snorkel or SCUBA equipment. He duck-dived to the bottom of the lake (10 feet - 3 metres). He stayed on the bottom by holding on to a rock. Suddenly he noticed a low pitched tinnitus in his left ear. He was not trying to Valsalva at the time. When he left the water the tinnitus disappeared.

Soon after this he had attacks of left sided tinnitus whenever he stooped down or turned his head to the right. On 28 December 1979 he developed, in addition, attacks of vertigo lasting a few minutes whenever he stooped down. At no stage did he notice any hearing loss.

He attended the casualty department at the Royal Victorian Eye and Ear Hospital on 28 February 1980 complaining of postural tinnitus and postural vertigo for 2 months. On clinical examination he had a hearing deficit on the left side. An audiogram revealed a 25 Db sensorineural hearing loss at 400 Hz. Otherwise he was normal, and in particular was fistula negative and Romberg negative.

Thirteen days later (12 March 1980) his audiogram was unchanged when he was operated upon. A perilymph fistula was discovered at the anterior-superior margin of the oval window. The surrounding musoca was scarified and the fistula plugged with ear lobe fat.

Post-operatively the positional vertigo disappeared. The original low pitched positional tinnitus diminished.

His first post-operative audiogram (17 March 1980) showed a slightly increased sensorineural deafness at 3000 and 4000 Hz. At this examination his ear was noted to be bleeding. A repeat audiogram the next day showed a large air-bone gap, and increased sensorineural deafness. Until this episode of bleeding from his ear there was little change in his hearing. Presumably the haemorrhage dislodged the fat graft and inflicted damage on his inner ear.

Follow up audiograms have shown a steady reduction in the air bone gap (conductive element) and some improvement in the sensorineural deafness.

DISCUSSION

a. Diagnosis.

This case confirms the points made by Molvaer.[1] Especially it is necessary to take a careful history when the patient presents with aural symptoms such as tinnitus. Tinnitus is a common presenting complaint of perilymph fistula. Vertigo often follows by several days and hearing loss is often understated.

b. Mechanism of Injury

In order to rupture a membrane the pressure must be markedly different on the two sides. A raised pressure in the fluid in the inner ear with a normal middle ear air pressure fulfils this condition. This pressure rise is achieved by:

a. raising the CSF pressure and transmitting the pressure rise to the perilymph.
b. by compressing the CSF (so raising its pressure)by implosion of the stapes into the oval window

In either case there can now occur a true explosive fistula, one due to a higher inside pressure.

The likely explanation for the perilymph fistulae of breathhold divers is that there is excessive inward movement of the stapes due to excessive inward movement of the ear drum as a result of the rapid rise in ambient pressures in a diver who does not clear his ears (equalise middle ear pressures with ambient) as he descends. This could either rupture the membrane round the foot plate or stress the round window beyond its elastic limits. In either case creating a fistula. In some divers the ear-drum ruptures before damage is done to the inner ear windows.

Goodhill[2,3] postulates explosive and implosive routes for the causation of perilymph fistulae. The explosive force being CSF transfer through the cochlear aqueduct, and perhaps through the internal auditory meatus and lamina cribrosa. This explains the perilymph fistulae occurring when effort and hypertension cause a CSF pressure rise. His implosive route (which includes Valsalva manoeuvres) is less clear as he implies that the fistulae are due to the sudden rise in middle ear pressure, without specifying the mechanical effects in the middle ear which damage the windows.

A membrane supported on one side by fluid is unlikely to be deformed by any rise in air pressure on its outer side since water is virtually incompressible at physiological pressures. Any rise in middle ear pressure must press equally on oval and round windows unless the ear drum moves in response to the rise in pressure. Goodhill's diagram 37-8 3 showing implosive routes is mechanically wrong. Normal stapes movement produces normal round window movement. Excessive compression of the perilymph by stapes movement at the oval window will result in a pressure rise at the round window and at the perilymph - CSF transfer points. If the cochlear aqueduct, lamina cribrosa and internal auditory meatus can transfer some fluid away from the inner ear the pressure rise in the inner ear will remain below that required to deform the round window beyond its limits. However the mechanical movement of the stapes may overstress the footplate membrane, so rupturing it and relieving the pressure in the inner ear before the round window membrane fails.

It has been postulated[4] that with a forceful Valsalva manoeuvre occurring suddenly the ear-drum is blown outwards pulling the ossicles suddenly outwards. The stapes is then exerting a negative pressure on the inner ear and as a result the round window is sucked in beyond its elastic limits. Alternatively the oval window might crack first so causing an oval window fistula. It is difficult to imagine that there would be enough movement of the ear-drum, which

has been forced in by unopposed water pressure, in a diver under water for this to happen. It seems quite possible in air and could explain the hearing loss associated with an oval window fistula reported by Friedman and Sasaki[5] during positive pressure resuscitation. However, this mechanism is not implosive. The only implosive injury is that of the diver who does not clear his ears (equalise middle ear pressures with ambient) as he descends rapidly.

The reason now advanced for the Valsalva induced fistulae in divers is that they are explosive due to the accompanying CSF pressure rise.

A further mechanism occurs in SCUBA divers during the ascent phase of the dive. A case was described by Mannerheim[6] where the diver sustained both a perilymph fistula and a ruptured ear drum during ascent. Not satisfied with a perfect recovery of his hearing following gelfoam packing of his round window niche he repeated the performance some months later. During the second operation photographs were taken showing a steady drip through a split in the round window membrane. Again packing with gelfoam resulted in a restoration of normal hearing.

The likely mechanism on both occasions was that he failed to clear his ears adequately on descent. The pressure imbalance resulted in transudation of fluid,[7] which probably clotted and blocked the Eustachian tube. Ascent resulted in pain, as reduction of ambient pressure resulted in the middle ear having a higher pressure than its surroundings. As a result the ear-drum was forced outwards. Depending on the relative strengths of the membranes the drum or one of the windows ruptures first. If a window goes first the drum will follow. However if the drum goes first the inner ear membranes are protected by the return of the stapes to its normal position.

Goodhill2,3 reviewed 76 cases of presumed perilymph fistulae including 17 who were not explored so cannot be confirmed as having had a fistula. In 59 explorations, 47 fistulae were found. 24 of the patients had fistulae of the oval window alone, 19 had fistulae both of oval and round windows and only 4 had fistulae of the round window alone. He did not give a breakdown into implosive and explosive aetiologies, nor did he mention how many suffered their damage while diving. The left ear was affected twice as often as the right.

c. Operative Technique.

Goodhill recommends the use of tragal perichondium. The fistula was patched with fat in our case. The initial results were good but the events following the bleed in the 5th post operative day suggest that the fistula was still unhealed at that time. In our experience two cases where the leaks were patched with crushed gelfoam had prompt cessation of the leak, and steady improvement in hearing from the time of the operation. As these fistulae are linear tears (which heal from side to side once the edges are approximated) the important thing is to provide a firm splint to hold the edges together and so allow healing by stopping the leak.

CLASSIFICATION

In view of the above we offer a more physiological classification.

Type 1

Due to raised CSF pressure transmitted to the inner ear (Goodhill's Explosive). Acting by bulging the window membranes beyond their elastic limits.

Type 2

Due to an excessive outward movement of the eardrum and ossicles creating a negative pressure in the inner ear (wrongly called implosive by Goodhill). Found in SCUBA divers during ascent and with positive pressure ventilation.

Type 3

Due to an excessive inward excursion of the ear drum and ossicles creating a positive pressure in the inner ear. (Correctly called implosive by Goodhill). This mechanism requires:

a. a rapidly rising ambient pressure,

b. failure to equalise the middle ear pressure with ambient,

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SITUATION REPORT COVERING DIVING IN THE OFF-SHORE INDUSTRY IN THE NORTH SEA IN 1979

by Commander SA Warner OBE, DSC, Chief Inspector of Diving, UK.

Mr Chairman, Ladies and Gentlemen.

Thank you once again for asking me to talk at your annual Symposium. I have no doubt in my mind that the International Diving Symposium is the premier forum in the world for discussion of all diving matters.

Activity in the North Sea during 1979 continued at much the same exposure level as 1978. The main activities involved construction, inspection and maintenance, repair operations and limited pipeline operations. Exploration activity was at a low level, however, this is expected to increase in the very near future which in turn, one hopes, would indicate a general increase in diving activity in 1981 and onwards. It is estimated that the diver involvement in the whole of the North Sea peaked at about 2000 plus last year.

I propose to start as I did last year with a very brief run down on last year's accident record. There were a total of 3 fatalities in 2 diving accidents in the North Sea last year, both of which occurred in the UK sector.

The first one involved a diver engaged in grit blasting operations at about 120 feet. The diver was breathing air from a surface supply and either pulled or blew off his helmet. He was wearing a dry suit with suit inflation and it is possible that he "blew up" and that the air in the suit blew past his neck seal into his personal neoprene hood which in turn lifted off his head and blocked the vent hole in his main hood.

In August a diving bell was lost from the diving support vessel "Wildrake" whilst operating on a SALM base at just under 500 feet.

Rescue operations failed to recover the bell and the divers before they both died. I am unable to give you further details on this particular accident until a public Fatal Accident Inquiry has been held except that I can say that the diving operation was not taking place from a dynamically positioned vessel and almost certainly cold played a major part in their deaths.

Dangerous occurrences, minor incidents and near misses continued to occur in the UK sector at a level not unlike that of last year.

You may remember, that I told you last year about an accident in which 2 divers lost their lives in a diving bell as a result of a dynamically positioned vessel being blown off station, dragging the bell's wires, life support system and the bottom guide weight wires across the anchor cables of another vessel. In one incident 3 methods of the bell recovery were lost.

In spite of the fact that dynamically positioned vessels have been in operation for many years I am still worried about the various danger aspects of divers operating from them.

As I told you last year the UK initiated a project to form a risk analysis study of diving from dynamically positioned vessels. The original study has been completed and a draft guidance note was issued to the industry as a consultative document. Many remarks have been received and many proposed amendments have been submitted. These are being studied and sorted out. It is hoped that agreed "guidance notes" will be issued in the very near future. The final part of the project is being carried out as a co-operative effort between the United Kingdom and Norway. I will ensure that interested parties in the United States receive copies of the final document as soon as it is issued.

We continue to analyse the figures we have on accidents and an up to date broad breakdown of the percentage involvement of various factors in the fatal accidents. I would like to point out that I am quoting the involvement of those factors and not necessarily the major cause of the accident:-

Human error	50%
Medical unfitness	6%
Inadequate training	16%
Equipment failure	30%
Lack of equipment	9%
Inadequate medical supervision	4%
Poor diving supervision	25%
Poor equipment maintenance	7%
Surface interface weather conditions	12%
Inadequate decompression schedules	Nil

for fatal accidents

Just before I left UK I received a copy of the report on Occupational Mortality in North Sea Divers by Dr Maurice Cross.

In the sight of the media, commercial oilfield diving is dangerous and a fatal accident usually receives considerable coverage. This tends to create a disproportionate amount of emphasis on the risks of diving and reflects adversely on the industry. In fact, little objective information exists as to the true fatality incidence in relationship to other activities such as coal-mining or surface on oil installations which may carry as high an incidence.

Using as a population substrata the numbers of certificates of fitness to dive issued by UK diving physicians and examining the numbers of deaths in individuals who would have been in possession of such certificates, a formal analysis has been made of the Standardised Mortality Ratio for commercial diving. It would appear that the safety standard have improved to the point where, from being one of the most dangerous activities in 1925, commercial divers now have the same fatality rates as other industrial workers such as engineers and coal-face workers.

However, minor cases of decompression sickness and the very occasional serious case continues to arise, but, in general, the therapeutic tables available appear to be adequate when used correctly.

At one period I was getting worried about the number of minor decompression sickness incidents being reported when using the United States Navy Surface Decompression Schedules. On investigation it was found that the incidents were probably less than 2%. I am sure that you will appreciate there is considerable difficulty in getting hard facts and exact figures on all these incidents and my knowledge is only as good as the information that is passed to me by the diving companies.

I hate the idea of generating "paperwork" but there is no doubt that something like a quarterly return covering such factors as total diver exposure time, depth, number of cases of decompression sickness, tables employed, etc, etc, would be of considerable help in progressing diving safety and certainly would help those who are carrying out research.

It is perhaps of interest to note that in the UK there was one fatality in inshore diving operations and 13 amongst sport divers in 1979.

The design of diving bells and the technique of employment them is very topical at the moment. The Association of Offshore Diving Contractors have produced guidance of diving bell operations recently. Tom Hollobone, the secretary of the AODC is here at the Symposium and would be pleased to provide further information to anybody interested.

I still believe that prevention of accidents must be the foremost approach to diving bell safety techniques. However, there is no doubt that serious consideration must be given to the provision of autonomous life support for bells. The biggest problem of course is to provide enough heat to maintain safe thermal balance for, say, 24 hours. As you know this provides a very difficult objective in deep helium atmospheres.

In addition, history has shown that there is a very real need for "bell" relocation systems which can be used in the event of a bell or submersible being lost. Probably the best technique for relocation is the employment of sonar beacons and as I told you on Tuesday, investigation into this subject is currently taking place in the UK and Norway. I regret unfortunately there is no international organisation or register which allocates sonar frequencies. To a certain extent one is restricted to a comparatively short band by the law of physics but I believe that it is necessary to have an internationally accepted emergency frequency which provides the necessary range, free from interference and compatible with sonar equipment fitted to diving support ships or, with portable location equipment. It is envisaged that diving bells be fitted with a pinger which can be activated in an emergency, that diving support ships be either fitted with or have available location equipment for that frequency and, that the diving industry have available diver held final location sets.

The AODC in conjunction with the UK and Norwegian government intend to hold a meeting in Aberdeen on 21 February to finalise the requirements for a sonar beacon relocation system and in particular the frequency. The decisions made at this meeting will be publicised immediately. Tom Hollobone is the contact man for anybody wishing to attend.

Since the introduction of the first transfer under pressure diving system there has been discussions and arguments about bell design and operation techniques. I think the time has come for the industry and governments to take a very close look at this problem and attempt to answer a few of the main questions in an agreed and unified voice.

I would like to use this Symposium to generate thought on the particular subjects of diving bell operations. I suggest this, because, other industries and indeed some people within the diving industry produce statements that "divers are still flying by the seats of their pants". In many ways this is true but tremendous progress has been made within a short period of time. Much of this progress has been initiated by the pioneering spirit backed up by a tremendous amount of medical and physiological research.

In spite of all this I would like to generate thought on the following subjects:

- Should a diving bell be heavy or should a bell be light with added weights that can be slipped in an emergency?

- Should the weights, if fitted, be underslung, or secured around the bell?

- Should there be an interlock between the bottom door of the bell and the slipping device to prevent accidental slipping of the weights with the bottom door open?

- Should there be an interlock between the slipping device and any cutting device for the lifting wire and umbilical to prevent the bell turning upside down or even obtaining an upside down position half way to the surface, anchored by a piece of lifting cable or umbilical, when the weights are slipped?

- Would the introduction of such a system of interlock increase safety or would they increase the areas for error thereby introducing more danger?

- Should the bell be top mated, side mated, or is a rolling technique acceptable? In answering this particular question one has to remember that history shows that divers becoming unconscious in the bell have in the past fallen onto the bottom door and thereby, in top mating bells, have produced a tremendous problem for rescuers.
- What is the best method of securing the bell to the transfer under pressure chamber?

- Should the hatches be round or elliptical in shape?

- What is the best size for hatches on which to standardise?

Bell, through water, communications present another area which requires a much closer study. What is the best frequency for through water communications?

I believe that we should aim to standardise on basic bell design and mating techniques in the long term. It is probably because such things as diving bells take a very long time to wear out that little impetus is given to standardisation.

We continue to place considerable emphasis on good diving training and the need for all new divers to achieve minimum standards. The UK standards have been available for several years now and there is a legal requirement on diving companies to ensure that these standards are met. You may have heard that I have written to some American Diving schools and pointed out that their curriculum does not achieve the terminal objectives required in the UK standards. This was not a sudden, without warning action. The basic air standard was published in 1975 and the mixed gas standard in 1976. These standards are designed on the achievement of certain terminal objectives. In January 1977 and again in January 1978 diving memorandums were issued drawing attention to the need to conform to these standards. I would suggest that our introduction time scale has been very generous.

There is no doubt in my mind that in general the United States diving training centres produce extremely knowledgeable graduates but, most of them sadly lack exposure to open water diving, exposure to depth and pressure.

I think it is fair to say that as a general rule diving graduates in the United States continue their diving career with several years' apprenticeship. In the North Sea they are often literally thrown into the deep end. This is one of the reasons that we require them to have had exposure to depth and pressure.

The United Kingdom continues to support research projects in diving safety matters and work continues on:-

Carbon dioxide retention in divers.
Central body rewarming device.
Excursion diving tables for oxy-nitrogen.
Investigation of unexplained unconsciousness in divers.
Investigation of anaesthesia in high ambient pressure.
Safe thermal conditions for divers.
Long term environmental effects on divers.
Effect of a muscle relaxant at hyperbaric pressure.
Production of a "black box" dive recorder.

And of course, research continues into the safety aspects of divers operating from dynamically positioned vessels.

CONFEDERATION MONDIALE DES ACTIVITIES SUB-AQUATIQUES

World Underwater Federation
34, rue du Colisee, 75008 PARIS, FRANCE

Paris, March 1, 1980.

CIRCULAR LETTER TO ALL FEDERAL DOCTORS

SUBJECT: NEXT MEDICAL COLLOQUY IN CANCUN (MEXICO) IN DECEMBER 1980

Dear Colleague,

The Medical and Accident Prevention Commission, convened in Barcelona in January 1980, has decided to hold during the CMAS World Congress:

- a General Assembly with elections
- a Medical Colloquy

The organisation of the medical colloquy has been confided to Dr Spinola from the Mexican Federation and to me.

It is the first time that the Medical Colloquy is held in Central America and know that a great number of American doctors are interested by this manifestation which will allow them to establish closer contacts with their European colleagues.

So this Medical Colloquy will take place in Cancun (Yucatan) from the 1st to the 5th of December, 1980.

The main themes of this colloquy are as follows:

- "Aptitude and inaptitude for diving" with presentation of the new CMAS international form of aptitude, prepared by Dr Ehm (RFA).
- "Diving accidents and their treatments" and presentation by Dr Tailleur and Dr Holmberg, of the form for diving accidents.
- Free communications.

The persons who wish to present papers must send us:
- before the 30th Jane, 1980: the title, the abstract, the duration and
- before the 31st August, 1980 the complete text of the paper.

We are preparing the programme of this colloquy in order to let a large place for touring and diving, for Mexico is a country particularly favoured in these fields.

We hope that you will be interested by our proposition and that you will come numerous to Mexico.

Yours sincerely,

Dr Marcel BIBAS
Responsible for Public Relations for CMP CMAS.

DEPARTMENT OF ENERGY
Petroleum Engineering Division
London.
November, 1979

DIVING SAFETY MEMORANDUM NO. 12
DIVING EMERGENCIES

1. Medical Emergency Call Services

During several recent diving emergencies associated with UK offshore operations, a pattern has emerged in which initial engineering problems have developed rapidly into accelerating human factor problems.

A tendency has been noted for diving contractors to delay alerting medical backup services until this late stage.

Although around the clock diving medical emergency call services are well established; see Diving Safety Memo No 1-6 March 1979; it should be remembered that the lapsed time involving mobilisation prior to the medical expertise being available on site, is an important factor which must be taken into consideration.

In such cases, even if medical emergency call services are not required immediately, they should be alerted as early as possible. This will allow appropriate arrangements to be made in respect of personnel and equipment, thereby minimising the lead time should mobilisation be required.

2. Communication

Diving contractors are reminded that communication of accurate information is essential in cases of diving medical emergency, since this provides the basis for initial medical assessment and subsequent advice.

Details of such information are contained in the AODC/DMAC Form 1 “Aide Memoire for Recording and Transmission of Medical Data to Shore” copies of which have been distributed throughout the diving industry.

* * * * *

DIVING SAFETY MEMORANDUM NO. 13
POTENTIAL DANGER TO DIVERS

A diver was recently put at risk when cutting off a pile guide from a production platform. Some three years prior to the “near miss” two grouting lines approximately 2 inches in diameter and 45 feet in length had been cut and were only secured to the support by clamps.

Having cut the pile guide the diver remained under water to assist in the operation while it was recovered. He had been warned of possible danger by the supervisor.

As the pile guide was being lifted to the surface the grouting lines became detached and descended to the bottom and only prompt action by the diver avoided him being taken to the sea bed.

Since this type of operation is likely to be on the increase, diving companies, diving supervisors and divers should beware of the possible hazard involved.

Diving supervisors should also ensure that divers are not put at risk from insecure equipment above water such as scaffolding which could fall and trap or injure the diver.

Sea Bed Debris

All reports by divers of debris on the seabed or offshore structures which could cause a hazard to diving operations should be reported to the Offshore Installation Manager and recorded in the log.

The Diving Medical Advisory Committee,
London.
November, 1979.

* * *

A NOTE REGARDING SAFE DIVING DISTANCE FROM SEISMIC SURVEYING OPERATIONS

Consideration has been given to the safety of divers in the vicinity of seismic surveying operations. This note sets down guidelines which it is suggested will be of help in relevant cases.

When seismic surveys are to be conducted in the vicinity of diving activities, it is considered that a safe distance between the seismic source and the diver in the water should be 1,500 metres where the seismic source is of the airgun or gas gun type and its capacity is not greater than 4,400 cubic inches or equivalent.

When conventional explosive (for example TNT) is used, safe distances may be considerably less than those advised for air and gas guns but, unless there are compelling reasons for not doing so, it is advised that the limiting distance of 1,500 metres should still be observed.

It is unlikely that seismic work will ever be conducted in shallow water but it should be pointed out that the calculations used to determine the figure of 1,500 metres are not applicable in extremely shallow water depths, eg. less than 10 metres.

Finally, it should be pointed out that the psychological effect of seismic surveying upon a diver in the water may be significant and will certainly distract his attention from the work he is engaged upon, long before the sound and shock waves from the explosion have any physical effect upon him or reach levels of intensity which even begin to approach danger.

ILLUSTRATIVE DIVING INCIDENTS FROM NEW ZEALAND

These twelve cases have been taken from the reports prepared by Dr Allan Adair for the New Zealand Underwater Association Inc and circulated by them. The purpose of such reports is to personify misadventures such that the dry words of an article or text book achieve a relevance to each reader, who hopefully applies the conclusion he (or she) draws from the reports to improve subsequent diving practices.

Case Notes

There were three fatalities, five cases of decompression sickness, two cases where diver entrapment occurred and two where excessively rapid ascent occurred. Buddies played a highly significant and useful part in the vain attempt to save Case 11.

Case 1

This experienced diver was making his second dive that day, the first having been aborted due to turbulence. This dive was similarly terminated after a short time at 40 feet and the pair of divers surfaced, the victim partly inflating his compensator and rolling onto his back with mask removed while his buddy climbed into the boat. He was seen near the boat's stern before a large wave hit the boat, and was seen shortly afterwards 20 - 30 feet behind the boat. Then another large wave came and he was not seen for a few minutes. He was seen near rocks, rescued and brought to the boat but failed to respond to cardio-pulmonary resuscitation. The medical evidence supported the Coroner's verdict of drowning.

There is no direct evidence as to the reason for the drowning, but a reasonable reconstruction might well be that he was pushed under water by the boat when it was hit by the first wave. At this time he was not wearing his mask nor using either his snorkel or demand valve.

It is recommended that all divers retain and use all gear, including mask and snorkel, until right into the boat.

Case 2

He dived for 30 minutes at 80 - 100 feet, there being no previous dives in the preceding 36 hours. A surface swim of 300-400 yards back to the boat in choppy seas left him very tired when he reached it. The DCP needle was between 'A' and 'C' at this time. On reaching the boat he complained of pins and needles in his legs and difficulty on standing. He was put on oxygen for 20 minutes and taken to the Naval Base, arriving there 6 and a half hours after surfacing. He was treated for decompression sickness, receiving recompression and oxygen on three occasions. Long term follow-up is not known. Age 32.

This dive required decompression stop of 3 minutes at 10 feet if calculated, as is advised, by conventional method of total dive time ascribed to greatest depth. Although he spent time at lesser depth he experienced severe exertion during his surface swim back to the boat, possibly the critical factor that precipitated the onset of symptoms. The DCP was still indicating safe time, but he contracted decompression sickness.

Case 3

This 42 year old diver was hunting crayfish. His first dive was for 30 minutes at 60 feet, the second for 15-20 minutes at 100 feet. Surface interval was 7 and a half hours. On reaching the surface he felt nauseated and unsteady, and 5 - 10 minutes later developed numbness and tingling in his body and legs from the waist down. He was taken to the Devonport Naval Base and treated with recompression and oxygen with good results.

If times and depths are calculated using the USN tables he was just inside the no-decompression times, but the tables have a percentage 'hit rate'. The accuracy of the time and depth measurement is not known. It would have been safer to have made the deeper dive first: calculation shows that this would have left him 25 minutes into safe time instead of the theoretical 2 minutes. It is unwise to go up to or even close to the 'no stop' time.

Case 4

In this incident the diver was low on air when his contents gauge became caught in a crevice between rocks in such a position that he was unable to reach round and free it. He was, however, able to cut it with his knife and so became free to ascent to the surface.

He was fortunate to have a good quality knife but it might have been better to take off the tank and back pack and free the gauge. Of course a buddy could have set him free, if he'd had one.

Case 5

The diving was from a boat in the open sea, and dive times are approximate because the victim was not wearing a watch. First dive was approximately 60 feet for 49 minutes, with a return to the surface for a new tank and a further 45 minutes approximately at 60 feet. Soon after returning to the surface he became 'jovial' and complained of dizziness. He was taken back into the water and spent 3 minutes at 20 feet and 2 minutes at 10 feet. He still felt unwell and had a pain in one elbow following this 'treatment' so was taken to the recompression chamber on the 'Pacific Installer'. Recompression was carried out with successful results.

Analysis:

1. Total time was 60 feet for 93 minutes, which is 33 minutes into decompression time.
2. Victim was not wearing watch (or DCP).
3. An attempt was made to recompress in water. Little air was available.
4. No information is available concerning possibility of administration of oxygen to the victim.

5. Diver had ‘several years’ experience’.

The diver was either unaware of no-stop times for his dive depth, or was unable to time his dives. Recompression in water was an inadvisable attempt and could have compromised his chances of recovery. Oxygen is a basic first aid

Case 6

This 42 year old diver had a history of a bend the previous year. He wore a highlift compensator, which he inflated when at 60 feet and was unable to deflate during his very rapid ascent. Some time after getting into the boat he felt a pain in his chest, and was given oxygen for 15 minutes with some relief but was left with some ache in the ribs which persisted. Next day he returned to Auckland and attended the Devonport Naval Base for a check. Apparently he had some residual damage, for he noticed himself that there is a heaviness in his legs and a mild burning sensation.

The victim says that he was not aware of the correct procedure of venting the compensator. This is a major safety factor with modern high-lift compensators and there is NO DOUBT that instruction should be given by the dive shop in the use before they are sold. The dive times are not known but the ‘bend?’ was probably due to the extremely high ascent rate, which can be as fast as 200 feet/min with such compensators.

Case 7

The dive profile was 15 metres for 45 minutes, surface interval of 30 minutes, then second dive to 25+ metres (no depth gauge this dive) for 30 minutes. On surfacing he complained of a stiffness in the right shoulder, right elbow and right hip joint. He reported to the Accident and Emergency Department at Christchurch Hospital 12 hours later and was examined by an experienced diving doctor. Diagnosis: mild type 1 decompression sickness. Treatment was oxygen, intravenous fluids, and aspirin. He was symptom free on discharge from hospital and did not report for follow-up.

He was 22 minutes into decompression and failed to observe no-stop times. There was negligence in omitting to wear his depth gauge for the second dive and delaying attendance at hospital for 12 hours. In addition, the dive profile is incorrect in that the deepest dive was the second dive.

Case 8

This case is markedly similar to the previous one and occurred the same day. The dive pattern was first dive 15 metres for 45 minutes, surface interval of 90 minutes then a second dive, to 25+ metres for 30 minutes (no depth gauge). On surfacing he suffered headache and nausea, later pins and needles occurred in his feet and weakness in the knees. Pins and needles were experienced in the fingers 8 - 9 hours later. He presented at the Accident and Emergency Department of Christchurch Hospital 11-12 hours after the dive. Diagnosed as Type 2 DCS. Treatment was initially oxygen at atmospheric pressure and intravenous fluids, later recompression USN Navy Table 5 with relief of symptoms.

The dive profile shows that he would have been 7 mins into decompression time. He failed to observe no-stop times for his dives, omitted a depth gauge, delayed seeking medical aid for 11-12 hours, failed to attend follow-up medical check, and chose to make the deeper dive the second one.

Case 9

Diving with a buddy when he ran out of aid at 50-60 feet. He buddy breathed till about 15 feet from surface, then broke off and ascended alone. He was found to be in distress and soon became unconscious. He was taken to a local hospital where he was observed for five hours, after which he was transferred to Devonport. He was recompressed on three occasions and ended with very little residual damage.

It is unwise to run out of air at depth (have a contents gauge), and unwise to break off buddy breathing to ‘run for the surface” (as are starting ascent with full lungs and a panic feeling). The delay in transfer from the local hospital to the Navy Base illustrates the value of a buddy with diving knowledge to supply information to medical attendants in certain circumstances. Oxygen should have been used in first aid.

Case 10

The victim was aged 20 years and had just completed a course of instruction, passing all tests. He entered the sea with his buddy and they snorkelled together approximately 100 metres offshore through broken reef water. The victim became very puffed and so both inflated their compensators, then continued a little further on snorkel. As victim was still ‘puffed’ they decided to change to SCUBA and descend. Victim had a little difficulty deflating his compensator, attempting to use the LP inflator button (not connected by hose at this time; oral inflation only). Buddy used the damp valve for him. Neither was conversant with this type of compensator. They descended to 20 feet and slowly worked their way out to some bigger rocks, taking time to feed fish with Kina on the way. As the visibility was not good and they seemed to be getting in each other’s way continually they agreed to part company around a rock, victim going to the left and the buddy to the right. Buddy found that the rock spread further than expected underwater and became worried as the two would not meet up as easily as planned, so surfaced. By this time there was a 3 foot swell so, after calling out his friend’s name without getting any reply, he climbed onto a rock and continued to call out and look for his companion. He thought that he saw someone on the beach and swam ashore to give his mate a ‘talking to’ but found nobody there. However his calls had been heard and the alarm raised. The victim was found the following day in about 20 feet of water. Autopsy revealed the presence of coronary artery disease.

The victim was unwell (excessively breathless while snorkelling) but proceeded with the dive. He was unfamiliar with the use of his equipment (compensator) and the divers separated. Water conditions were possibly beyond the victim's ability to cope with, he being newly trained.

Case 11

The victim was aged 53 years and undergoing his second familiarisation dive of his training course. It was noticed that he had some difficulty with breathing and complained of a pressure sensation in his chest. He became cyanosed and unconscious. At this time the instructor was with him and had inflated his buoyancy vest sufficient to give buoyancy without causing a constrictive feeling around his chest. Symptom onset was after surfacing and practicing vest inflation. When unconsciousness occurred the instructor dropped the victim's weight belt and commenced mouth to mouth resuscitation and summoned help by whistle. The victim was still making breathing efforts for himself while being towed ashore (150-200 yards). Sea conditions were calm and the dive conducted without rush. The victim failed to respond to Emergency Unit care ashore.

This was stated to be a bad heart attack. The attention received from those present was excellent, but unavailing. Statistically there is an increased risk of heart attacks in older persons while diving and such fatalities are probably unavoidable on occasion despite every precaution.

Case 12

On a night dive at 80 feet with an inexperienced buddy, this experienced diver became trapped in a small cave due to the tank getting caught under a ledge. After a mild initial panic the diver managed to get free without assistance. The buddy offered no help because he though that the buckling legs were due to efforts to catch a crayfish. During this short time of entrapment the victim used up 1000psi of air!

It was unwise to enter a cave at 80 feet on a night dive, especially when with an inexperienced buddy. Situation was saved by lack of panic, due to experience.

DISCUSSION

These reports underline the correctness of the usual guide lines for safe diving. These include adequate training, abort the dive if unfit to continue in complete safety, wear correct equipment (compensator, contents gauge, knife, etc.) and be familiar with its use, dive within the skills of yourself and your buddy, and avoid separation from the buddy. Avoidance of dives which require decompression stops is advised, as also any reliance on decompression meters to allow marginally permissible dive profiles. As always, do not run out of air, and remember that the potential dangers of diving still operate until you have left the water and are safely ashore. If such advice saves your life give thanks to those who prepared the incident reports for the NZUA.

AN UNUSUAL DIVING PROBLEM

The following report of what could have been a fatal diving accident is taken from a safety note of the US Naval Safety Centre and contains a lesson for us all.

The diver checked out a set of twin 72 cu ft open circuit scuba bottles for a recreational dive from a military activity diving locker. He gauged the bottles and found that they were fully charged (2250psi). He then placed them in the back of his car. He did not gauge them again before making the dive, though he did check the regulator on the set to ensure that it was functioning properly.

The dive took place in 8 to 20 feet of water near an old pier. When low on air, after about 1 hour, the diver attempted to surface but was unable to leave the bottom, a problem which persisted despite releasing his weight belt. He then proceeded to a nearby pier piling and pulled himself up the piling to the surface. The extra buoyancy of his life jacket, while not enough to keep him on the surface, was enough to enable him to kick and bob his way to shore. The diving locker disassembled the bottles used on the dive and found that one of the bottles in the twin 72 manifold was completely filled with water. Even at the shallow depths of this dive the water pressure was sufficient to force water into the bottle through the blow-out plug.

Comment: The diver had placed the bottles in the back of his car and the hot July weather resulted in the blow-out plug rupturing due to pressure increase of the contained air after several hours of sun heating. The blow-out plug opening allowed water to enter the tank during the dive.

Recommendations

1. Always gauge bottles before entering the water.
2. Do not stow bottles in direct sunlight for prolonged periods.
3. Put reserve in the down position to equalise the scuba bottles and to alert of escaping air from the blow-out plug.
4. Do not hesitate to ditch bottles in an emergency.
5. Have a buddy.

THAT'S SCIENCE FOR YOU[

Unconfirmed newspaper reports tell the sad tale of obstruction to the march of progress by ill-informed environmentalists and others. In late 1978 it was suggested that some Canadian Polar Bears be fed crude oil to show that there would be no ill effects should an oil leak occur. The resultant public reaction was followed by an official statement of disassociation from Imperial Oil, Canada's biggest oil company. But the Experts eventually won permission to demonstrate the safety of crude oil to polar bears. A recent report states that they are now trying to find out why the bears died when Scientific Experts knew they wouldn't.

THE PHYSIOLOGY OF DECOMPRESSION SICKNESS

Charles Brown MD

THIS PAPER WAS ORIGINALLY PRESENTED AT A PADI WORKSHOP AND WE ACKNOWLEDGE WITH PLEASURE PERMISSION TO PRINT IT IN OUR JOURNAL.

Decompression is reduction of ambient pressure, and decompression sickness is any ailment resulting thereby, except for some. We don't count reverse squeeze, lung overpressure, or hypoxia of altitude. DS (decompression sickness or bends) is essentially bubble trouble. Innumerable other factors enter in, but bubble formation is the primary event, and bubble behaviour is the prime source of grief. Therefore this talk is concerned mostly with how and where bubbles form, how they make mischief, and how we get rid of them.

The answers seemed reasonably clear after the excellent work of Bert and Haldane many years ago. Bubbles formed because of a 2:1 supersaturation ratio was exceeded. They distorted tissues and blocked blood vessels. They were banished by recompression. Experience has shown those answers overly simplistic. There are too many things they don't explain. Divers get hit while obeying Navy rules: others don't in spite of flagrant violations. Many divers with proven bubbles have no symptoms, while others are bent in the absence of detectable bubbles. Some serious DS cases recover without treatment; others do not even if recompressed. Bends is more likely after two 40 minute dives three hours apart than after one 80 minute dive, in spite of outgassing during the surface interval. Yet frequent diving confers partial immunity. It's plain that we're talking about a very complex disorder. Today, after thousands of studies by hundreds of investigators, we're more confused than ever, but we have learned a few things.

How Bubbles Form

First, you've got to have micronuclei, or so it seems. Put a glass of water into a chamber and run it to 20 ATM's. If you then decompress it rapidly, it bubbles. But if while still saturated at 20 ATM the water is poured into a cylinder that has no air space, and is further compressed by a piston to 2,000 ATM, when decompressed to the surface it won't bubble at all. We infer that water contains micro-bubbles that are normally stable, but can be dissolved by immense pressure. We call them gas micronuclei. Without them, water tolerates huge supersaturation stress without bubbling.

How do people get micronuclei? The stuff we drink is one source. Scientists say that cosmic rays from space, and radioactive decay of trace elements in our diets cause micro-explosions that make more. And statistical analysis of the random movement of dissolved gas molecules shows that frequently some of them come close enough together to crowd out other molecules and so find themselves out of solution. What stabilizes micronuclei is not known. Surface tension forces, huge at small diameters, would inhibit their growth, but would also tend to squeeze them back into solution. Maybe they escape surface tension by hiding in tiny tissue crevices. Or maybe they're really something other than microbubbles.

Anyway, real bubbles can form in the body in a number of ways even without reduction of ambient pressure. Pull a finger till the knuckle pops. The pop announces sudden gas in the joint, caused by the vacuum you made in pulling. Let go, and the gas goes away in 20 minutes.

Exercise generates cavitation forces along muscles and tendons that produce bubbles. And bubbles can form in the skin by a process known as counter-diffusion. If you breathe nitrogen while surrounded by helium (as commercial divers often do) the two gases diffusing through the skin in opposite directions cause local super-saturation and bubbling - a common form of skin bends. A similar mechanism might help explain the high incidence of vestibular hits in deep diving. Theoretically bubbles could form in tissue saturated with gas while cold, and then quickly warmed, because gas solubility falls as temperature rises. Strong sound waves can also cause bubbles.

Any or all of the above may contribute to DS, but there's no doubt that the most effective way to get a lot of bubbles in a hurry, short of boiling a diver, is to rapidly decompress him. How much supersaturation a diver will tolerate before bubbles form is not known. Haldane thought the total tension of dissolved gases would have to be more than double the ambient pressure before bubbles would form - hence his exhortation not to exceed a 2:1 supersaturation ratio during ascent, and his stipulation of 33 feet as the no-decompression limit.

He was wrong. How do we know? Weighing a diver underwater before and after decompression shows that he gains buoyancy, implying a gas phase separation. Also, dives to 33 feet or even less have on occasion produced DS. The connection is confirmed by Doppler monitors which, placed over the vena cava, sound off upon decompression.

Since Haldane, others have held that supersaturation tolerance is properly expressed not as a ratio, but as a pressure differential - so many feet sea water less than tissue gas tension. Both methods for building tables share a common fault - they really aim at defining how much supersaturation stress can be tolerated without producing symptoms, not bubbles. More recently, Brian Hills came up with the astounding notion that divers can count on little or no supersaturation tolerance. A glass of 7-Up sitting still tolerates considerable super-saturation, but stir it and bubbles burst forth. A diver is more like 7-Up being stirred than sitting still. What he tolerates is not so much a specific supersaturation stress as a certain silent bubble load.

Where Bubbles Form

In practice, bubbles are commonly detected in veins and in fat. The electron microscope reveals bubbles in fat breaking through capillary walls to reach the blood, and some think that may be a main source of venous bubbles. Another source is lymph, which drains tissue fluid into veins. With increasing provocation, we'd expect bubbling in other tissues. Last to bubble would be arterial blood. It's fresh from the lungs, which swept out excess nitrogen, and it's at higher hydrostatic pressure than obtains elsewhere in the body. This is fortunate, since bubbles in arteries are particularly dangerous.

How Bubbles Make Mischief

Bubbles do indeed distort and disrupt tissues and block blood vessels. But this doesn't necessarily hurt. If you place your hand into a little chamber that seals around the wrist, and pump the air out, your hand swells with the bubbles, yet you feel no pain. To feel pain, you need pain nerves, and most tissues don't have them.

Painful DS is mostly limb bends. The site of the bubble in a limb bend has long been debated. Recently, bubbles have been seen in tendons. Maybe they arise from small fat inclusions, which tendons have. Other factors fit.

Tendons have poor circulation, so they lose gas slowly. They have a structural pattern which a growing bubble would deform, and they have pain nerves to complain about it. Exercise, known to favour bends, creates a relative vacuum in a tendon by stretching it, and generates shear forces by sliding it; both favour bubbling. And finally, injecting saline into a tendon produces bends-like pain. Of course other tissues such as ligament, cartilage, and periosteum, have not been ruled out as sites of limb bends pain.

Pain can also be a feature of serious DS. When bubbles bother pain fibres in the spinal cord, pain is felt wherever the fibres originate - usually the lower limbs, and often the back and abdomen.

Bubbles can cause pain with or without doing much damage, and they can cause damage with or without pain. Bubbles in the spinal cord or a coronary artery cause both pain and damage. Bubbles in the inner ear can cause ringing noise, hearing loss, dizziness, staggering, and nausea, all without pain. Bubbles in the brain can cause stroke symptoms - no pain. Bubbles blocking vessels in various large organs cause no pain. They don't even cause damage if collateral circulation is good enough that blood flow bypasses the block. If it doesn't, damage must result. Evidence for it is a feeling of great fatigue several hours after diving. A blood sample taken then will show elevation of enzymes released from damaged cells. In this latter case, healing seems to be prompt and complete else a lot of us here today wouldn't be functional.

Finally, bubbles (or other emboli) in bone cortex may cause silent damage that erupts in painful disability months or years later. We're talking about aseptic bone necrosis, or as it is now termed, dysbaric osteonecrosis. When such areas of bone death occur near a major joint, and eventually the joint surface collapses into them, it's sudden crippling. Dysbaric osteonecrosis correlates poorly with limb bends, and not at all with serious DS. Nobody knows why. An intriguing hypothesis is that uranium^{238}, which tends to concentrate near the ends of long bones, insures a plentiful supply of gas micronuclei to initiate bubble formation in those areas.

The Pulmonary Bubble Trap

Since ordinary dives produce bubbles capable of blocking blood vessels, why isn't DS far more common? Recall that most bubbles either arise in veins or are delivered to venous blood. Venous blood goes to the right heart, which pumps it through the lungs. A lung happens to be a superb bubble trap. Bubbles big enough to block vessels are filtered out, and remain stuck till their gas diffuses out to the alveoli.

Isn't this hard on the lungs? It appears that a diver won't notice symptoms until 25-60% of his pulmonary circulation is blocked, depending on how active he is, and on whose figures you read. Near the limit, there are warnings. Greek sponge divers learned that when a drag on a cigarette sets off a fit of coughing, it's time to pack in the day's diving and take an underwater decompression stop. Even a deep breath may provoke the coughing.

Chokes

If the lung's capacity to trap and scrub bubbles is exceeded, a lot of bad things can happen - like the chokes, spinal hits, and arterial gas emboli. The chokes is a syndrome of pain, shortness of breath, and coughing. The lungs get water-logged and still - it's harder to breathe. Reflex or chemically induced bronchospasm further reduces air delivery. Alveoli without blood flow can't exchange gas, so oxygen uptake goes down. Yet these alveoli are still ventilated, so part of the work of breathing is wasted. All this blockage causes high resistance to flow of blood through the lungs. Back pressure builds up in the pulmonary artery, and the right ventricle must work harder to pump the blood it gets from the systemic circulation. When it can't maintain the pace, circulation slows and back pressure builds up in the systemic veins. Nitrogen delivery from tissues to lungs falls off, and this slows outgassing and favours more bubbling. When the capillary beds feel the back pressure, there is a shift of water from blood to tissue, reducing the blood volume - a prelude to shock.

Spinal Hits

Serious DS can occur well before the bubble load in the lungs is large enough to cause chokes. Spinal bends is a special case. It is often due to blockage of the veins that drain the spinal cord - the vertebral venous plexus. It is most apt to occur when circulation is slow, and it's aided by an anatomic peculiarity. Most veins have one way valves to prevent back flow. The veins of the azygous system, which drains the vertebral plexus, do not. If anything increases the pressure in the chest, like say a cough, or exhaling against airway obstruction, there could be a temporary back flow of blood through the azygous system pushing bubbles just emerged from the vertebral plexus back into it, where they hang up. Indeed, venous blockage isn't unique to the spinal cord. Studies of other tissues have shown bubbles first apparent in venules to grow back into capillaries. And spinal hits can also result from arterial emboli.

Arterial Embolism

Bubbles gain access to the arteries when allowed to by-pass the pulmonary trap, or when it is somehow released. An unborn baby has a hole in the wall between the upper heart chambers, so that blood can flow directly from the right to the left side, by-passing the non-functioning lungs. This opening (the foramen ovale) is supposed to close after birth, but doesn't always. Up to 50% of adults retain at least a pin-hole defect, and the vast majority don't know it. Normally it doesn't matter. But when lung circulation is blocked and back pressure in the right atrium

builds up, a significant amount of blood and bubbles will take the short cut to the arterial side of the circulation.

Lungs themselves have shunts that bypass their own capillary beds. These seem to be closed most of the time, but they open as the bubble load increases. In one experiment, tiny beads of various sizes were injected intravenously. A few beads with diameters larger than lung capillaries always got through. As the rate of injection was increased, more beads of larger sizes got through. What opens the shunts is uncertain - maybe increased pressure, hypoxia, elevated CO_2, or all three. Or conceivably, bubbles could rupture pulmonary arterioles and break directly into venules, to by-pass the trap.

Finally, a repetitive dive could spring the bubble-trap by squeezing some of the bubbles down small enough to pass through. More nitrogen is taken aboard during the dive, and upon ascent it diffuses into those pre-existing bubbles and enlarges them to more than original diameter. Any that happen to be in the arteries at the moment they become too big to transit the micro-circulation become arterial emboli. As mentioned before, the harm they cause depends upon where they happen to lodge. This helps explain the random incidence of DS for a given provocation - the fact that if you plot the incidence on a graph, you get the bellshaped curve that reflects pure chance.

Since some bubbles do reach the arteries, the question arises why brain hits are so rare in DS, while so common in the air embolism of lung over-pressure accidents. There is disturbing news. Maybe brain hits are not so rare. After lung tear, a lot of air enters the carotid artery all at once. DS bubbles are much smaller, but they keep coming. They would tend to block much tinier vessels in more scattered areas. It has been reported that very careful neurological examination of DS patients often does show evidence of subtle brain damage. It has also been casually observed that some commercial divers of long experience seem to undergo personality changes. Finally, autopsies of goats exposed to frequent decompression stress showed unequivocal brain damage. The pathologist opined that one of the brains couldn't have been more than fifty per cent functional. Yet none of the goats had demonstrated abnormal behaviour.

Biochemical Disorders

So far, we've been looking at DS as a mechanical disorder - the gas laws in action. There's another side - biochemistry in action and it's a whole new ball game. It's tempting to say that blood recognizes bubbles as foreign invaders and counter-attacks. That's probably nonsense, since the counter attack does more harm than the bubbles would if left alone. More likely, bubbles trigger the damage control system for plugging up leaks in vessels, but at the wrong time and in the wrong places.

It all starts with blood globulins - large, complex, biologically potent molecules. These have one pole that attracts to water, and another that attracts to fat, and typically arrange themselves in groups with the fat loving poles inward. Bubbles change this. For reasons best known to biophysicists, the blood-bubble interface generates an electro-kinetic force that makes the molecules flip. They re-align with their fat loving poles at the interface. This somehow activates them to do strange things. Nearby blood platelets become sticky. They adhere to bubbles and to each other, whereupon they too become activated. The result is the release or the induction of a witch's brew of highly bioactive substances with odd names like serotonin, bradykinin, kallikrein, histamin, SMAF, prostaglandins, etc. Then things really liven up.

The blood clotting mechanism is cranked up. Lipids split off lipoproteins and coalesce into globules of fat. Capillary permeability is increased, and fluid leaks from blood into tissue spaces, decreasing blood volume. Arterioles constrict intensely, reducing and sometimes shutting off circulation to capillary beds. By the time waste accumulation and hypoxia make the arterioles relax, the capillaries have been damaged. Some of their lining cells peel off and are swept into the blood stream. Plasma leaks through the damaged capillaries into tissue spaces, so blood loses more volume, and becomes further concentrated. This increases its viscosity, which slows circulation, favours more clotting, and makes red cells sludge together in clumps.

How far all this goes depends upon the bubble load. When the body's compensatory mechanisms are overwhelmed, the victim slides into shock. This is one reason why it's so important to get potentially serious DS into a chamber fast. Wait till the victim's in bad shape, and the results of recompression will be disappointing. You can't squeeze out the platelet masses, fibrin clots, fat globules, red cell clumps, and other cellular debris clogging up his circulation. He needs a lot of medical intervention - intravenous hydration, anticoagulants, etc.

In practice of course the mechanical and biochemical effects of bubbles interact. Bubbles might hang up where tiny arteries branch into two smaller ones because surface tension opposes deforming a sphere into two wieners. But why should bubbles hang up in small veins? Doubtless because they attract platelets, which become sticky.

Predisposing Factors

Some things predispose to DS, and we may now ask how. The most important seem to be obesity, age, exertion, and illness. After a long dive, fat holds a lot of nitrogen which, because of poor circulation, it can't unload. So it Bubbles. Also, overweight people tend to have elevated blood lipid levels, which favours the biochemical derangement in DS. Incidentally, fatty meals temporarily raise blood lipids and so favour DS.

Estimates of the importance of age vary, up to an 11% annual increase in bends liability for young men. All agree that the risk increases sharply at middle age and beyond. The simplest explanation is increase in body fat and, more important, in arteriosclerosis, which reduces circulatory efficiency.

Exercise during a dive speeds up circulation and, therefore, nitrogen uptake. It also generates lots of CO_2 which, because ventilation is impaired at depth, results in elevation of the CO_2 tension. We don't know why, but CO_2 seems quicker than nitrogen to promote early bubble growth. Once bubbles form, any excess CO_2 enlarges them faster. Upon ascent, exercise

promotes bubbles by cavitation and shearing forces, and by generally shaking up the body.

A few years ago, aspirin got an undeserved reputation for causing bends. The real culprit turned out to be the condition for which the diver had taken the aspirin. Though the mechanism is not understood, illness seems to predispose especially to serious DS. In this context, illness includes alcohol abuse and other conditions that lower metabolic efficiency. Dehydration predisposes at least in part because it's a head start to haemoconcentration. Divers shouldn't be thirsty.

De-disposing Factors

Caisson workers and people who dive a lot acquire partial immunity to bends. This is said to peak in two weeks at a level of 75% protection. We think it results from subclinical bubbling, since intravenous injections of air in animals also confers immunity. A favoured explanation is that silent bubble showers from repeated decompressions consume micronuclei faster than they can be replaced. The curious fact that immunity for one depth does not extend to deeper depths is consistent, if we assume that some micronuclei are more stable than others and require more supersaturation stress before they will commence expansion. Consumption of blood clotting factors faster then replacement might further add to the protection. To avoid unpleasant surprises, it is most important for a diver to remember that immunity is lost in 2-3 weeks of not diving.

Out-gassing

We'll now address our final question. How do all these divers running around with silent bubbles ever get rid of them? Say for example you've just completed a nice legal dive - 50 minutes at 60 feet. You have silent bubbles. When all of your excess dissolved nitrogen has diffused either into the bubbles or into the alveoli, and equilibrium is reached, what's to keep those bubbles from becoming permanent guests? They're at ambient pressure, so why should any gas leave them? One reason is that a bubble is never really at ambient pressure. Surface tension and tissue elastic recoil exert at least a little compressive force upon it. Another and far more important reason is the inherent unsaturation of living tissue.

Inherent Unsaturation

Full equilibration between tissue and alveolar air cannot be reached, but rather a steady state in which tissue gas tension is less than ambient, and therefore less than bubble gas tension. This is tricky to grasp, but it's very important. Here's how it goes. We say that, by the laws of Henry and Dalton, dissolved gas tensions in the body are in equilibrium with gas tensions in alveolar air, which must add up to ambient pressure. But it's not quite true. Actually, only arterial blood equilibrates with alveolar air. When arterial blood gets into capillaries, oxygen is lost and CO_2 is picked up. Oxygen is poorly soluble in blood plasma, and the small amount dissolved is rapidly soaked up by the tissues, so oxygen tension drops way down. True, haemoglobin unloads more to meet tissue needs, but only at this low tension. Conversely, the CO picked up is very soluble, and most of it is found in chemical combinations anyway, so while oxygen tension drops a lot, CO_2 tension rises very little. Therefore, the sum of all dissolved gas tensions is lower in venous blood than in arterial blood. Since tissues are in equilibrium with venous blood, their total gas tension is also less than ambient pressure, and therefore less than gas tension in bubbles. Thus, a bubble is always super-saturated relative to the tissue it occupies, and the tissue is unsaturated relative to the bubble. So Bubble gas must slowly dissolve into tissue fluid until there is no more bubble.

The magnitude of inherent saturation has been measured in rabbits at the surface as 80-94 mm of mercury. At depth, breathing compressed air, arterial oxygen tension is very much greater, but venous oxygen tension is not. Inherent unsaturation, determined by the difference between them, therefore becomes very much greater. This has surprising implications for decompression practice. Take a commercial diver who has put in four hours at 150 feet and wants up. Haldane would bring him up half way to achieve maximal tolerable super-saturation, and thus maximal out-gassing gradient. Hills says that's wrong. People don't tolerate much super-saturation, so you'll just produce a lot of bubbles. Once bubbles appear, excess dissolved nitrogen will diffuse into them. Gas tension in tissues and blood will therefore drop toward ambient, and the out-gassing gradient in the lungs will become low. The diver must then wait till inherent unsaturation and slow alveolar diffusion remove a lot of gas from the bubbles before he can safely ascend to the next stop. The more shallow he gets, the smaller is the inherent unsaturation, so the stops have to be longer. What a drag!

The right way, by Hills, is the opposite. Start the ascent slowly, with much deeper stops, so any bubbles starting to form will be promptly banished by the high inherent unsaturation at those depths. This is greatly facilitated if the bubbles are never permitted to grow, for with tiny bubbles, you get much more help from surface tension, and the surface to volume ratio is very great, permitting rapid re-solution of gas.

This approach has good experimental support, and is now being used in some commercial operations. The Navy tables are based on the Haldane model, but extensively modified through experience. Let us emphasize that, as of now, they are the only ones thoroughly tested and currently recommended for sport diving.

Oxygen in DS Treatment

Since inherent unsaturation results from oxygen metabolism, it is sometimes called the oxygen window for out-gassing. To open the window wider for faster safe ascents (in commercial diving) or for treatment of DS, you need simply replace all or part of the diver's air supply with oxygen. This trick further improves the out-gassing gradient for nitrogen because it lowers inspired nitrogen. Another advantage of oxygen for treatment is that at three atmospheres absolute, enough dissolves in plasma to supply basal tissue needs without help from red blood cells. At such high tensions, oxygen will diffuse through tissue fluid around blocked vessels enough to keep some of the deprived cells alive.

Other Aspects

We're done with bubbles, but before we have done with DS, we must at least consider the

possibility that other factors associated with decompression may play a role. Notable is osmotic pressure - the tendency of water to flow (along its own concentration gradient) from areas where the concentration of dissolved materials is low to where it is high. During ascent, fast tissues lose gas more rapidly than slow tissues, and the resulting osmotic gradients would tend to pull water from the fast into the slow tissues. A special case is blood, which is thought to equilibrate with tissues by the time it traverses capillaries. In the arterial portion of the capillary, up to where equilibrium is reached, blood would lose water to the tissue. As we've seen, haemoconcentration favours DS.

Osmotic gradients created by the descent phase of the dive could also be significant. Dissolved gas in working muscle and in bone marrow would rise much faster than in the adjacent bone cortex, tending to dehydrate it. Haemoconcentration in the cortex capillaries would favour red cell sludging and blood clotting, and thus osteonecrosis. If this is significant, as animal studies suggest it may be, dysbaric osteonecrosis becomes, to that extent, compression sickness.

UNDERWATER RECOMPRESSION OF BENDS

Charles Brown MD

(This article is reprinted by kind permission of NAUI and is from NAUI News October 1979).

People dive everywhere. Charles Howell dived in Thailand, which is fine except that the nearest recompression facility is far away in the Philippine Islands. When he got bent, he opted for recompression underwater instead of the 15-plus hour journey to Subic Bay. Here's how it went.

The dive was the first of the weekend. It was mostly at 60 to 70 feet, with a maximum depth of 85 feet and a bottom time of 23 minutes. Soon after surfacing he noticed loss of sensation in his right hand, and then complete paralysis of the right arm and leg. What to do? He could go to hospital and hope for the best, or set out for Subic Bay, or put on a tank and go back down. None of the options was attractive, so it became a matter of deciding which was the least bad.

A quick inventory of resources showed that: 1. Seven full tanks of air were available; 2. The victim was mentally clear and felt confident that he could manage his own air supply; 3. The water temperature would permit several hours of exposure without hypothermia becoming a problem; and 4. There was present a very experienced divemaster who could accompany and observe the victim. All things considered, underwater recompression seemed the best bet.

The profile elected was 20 minutes at 60 feet, 10 minutes at 50 feet, 10 minutes at 40 feet, 20 minutes at 30 feet, 30 minutes at 20 feet, and 60 minutes at 10 feet. It worked - Mr Howell regained the use of his limbs at 40 feet on the way down, and felt quite normal after decompression. The following day, however, the right limbs felt a little weaker than the left, and ever since they tire more easily with exercise.

We learnt of this case from Arthur Rhodes, NAUI, in Thailand. He is disturbed. He takes the US Navy view that underwater treatment of bends is dangerous and should be condemned. He fears that its success in the present case will entice others to try it, with disastrous results, and he asks for our comments.

First, we congratulate Mr Howell and the people who advised and assisted him. He had a very serious problem that needed a quick solution, and it was provided. It's hard to argue against success. Yet looking through our retrospectroscope (the medical equivalent of Monday morning quarterbacking) we can see possibilities for improving the management. First, use of the reverse slant position at the first sign of trouble might or might not have been beneficial. Second, newer theories of bubble resolution suggest that some of the time spent at 10 feet might have been better spent at the 30 and 20 foot stops. Still, the schedule used was more in keeping with conventional practice and cannot be faulted. Third, the 2 and a half hour treatment that was possible falls far short of any approved schedule. If oxygen could have been obtained, its use for some hours after surfacing might have improved the outcome. Fourth, a neurological exam after the apparent cure might have revealed subtle residual defects and resulted in advice to get to a chamber. Finally, since central nervous system symptoms can occur after apparently successful treatment, and since a recurrence is best treated with hyperbaric oxygen, it would have been well to get near a chamber for a day or two of observation - assuming a flight with cabin pressure near sea level and oxygen available.

And now that we've gone on record as agreeing with Howell's underwater recompression - a controversial opinion - we hasten to add that we also agree fully with Arthur Rhodes. A decision to treat bends underwater is almost always wrong. Rarely is anyone present with the expertise to analyse the situation and invent the best possible treatment schedule. For many reasons, safe treatment of adequate duration would usually be impossible anyway. The victim might be or become unable to help himself, or the air supply inadequate, or the water too cold, or darkness would set in, or the weather deteriorate. The victim's nitrogen burden would usually be large enough that any feasible underwater schedule would hardly dent it. The depth required to relieve symptoms would usually cause further ingassing, adding to the nitrogen burden. Unless the ascent could be stretched over a great many hours, it would produce larger bubbles and worse symptoms than before treatment. Finally, valuable time would be lost. The victim needs hyperbaric oxygen, and the longer he is deprived of it, the less will be his chances of recovery. There are horror stories of victims and rescuers dying during prolonged underwater treatment attempts. In most cases, the outcome will be better if all efforts are directed towards reaching a chamber

even if far away.

What is different about the Howell case is the combination of an unusually favourable resource inventory and a relatively low nitrogen burden. The dive was well within the no-decompression limit and the ascent rate was 60 feet per minute. The diver was a healthy young man in good condition with no discernible predisposing factors for bends. He should not have been bent, and indeed we believe he probably was not. The symptoms were those of arterial gas embolism in a relatively localised area of the brain. It would be most unusual for decompression bubbles to get past the lungs and reach the arteries in dangerous quantity in absence of a larger nitrogen burden. If they did, we'd expect them to block smaller arteries in more localities, causing more diffuse symptomatology.

Howell probably had a lung over-pressure accident. Although he didn't hold his breath or admit to a recent cold or wheezing, he did have a medical history consistent with this diagnosis. One lung had been punctured during surgical removal of a non-malignant but huge tumour from the pleural space. Scar tissue perhaps blocked a small airway. Why not sooner? The dive that hurt him was only his eighth.

The tendency of lung over-pressure to cause embolism has nothing to do with nitrogen supersaturation, but the outlook for success of treatment certainly does. Here the embolism was small enough that 40 feet afforded relief, and a rather short decompression sufficed to prevent a major recurrence. And the nitrogen load already aboard was not enough for the treatment schedule (actually a repetitive dive) to bend the diver. If it had been, the higher nitrogen tension and reduced efficiency of circulation would have interfered with resolution of the air embolism as well. Since the success of recompression in treating air embolism depends heavily on speed, we feel that when the chamber is distant, a trial of on scene water recompression is justified if a careful analysis of all pertinent facts leads to a reasonable expectation of success. If relief is not prompt, the trial should be aborted.

There is another alternative, provided it's planned for ahead of time. It was worked out for Australian divers who might find themselves several thousand miles and several days from a chamber, and has been described by that stalwart innovator and individualist, Dr Carl Edmonds. You carry along emergency oxygen bottles, and enough lines, floats, and anchors to permit accurate depth staging. Thermal protection and night lights are also needed. Underwater recompression on oxygen has the huge advantage that there is no additional uptake of nitrogen. Bubble resolution and outgassing are far more efficient than when air is used. It would seem to be a valuable technique for Thailand until such time as a chamber is available. If you're bound to dive in the boonies anywhere in the world, be superconservative, carry oxygen at least for surface use, and have your contingency plans worked out in advance.

A final suggestion. US Air Force planes frequent many parts of the world. The hyperbaric unit duty officer at Brookes AFB, San Antonio, Texas knows where they all are. Call 512-536-3278 (that's 512 RED FAST). He can give expert advice for managing your emergency, and can sometimes divert a plane, possibly even one with cabin pressurised to sea level, to take the victim to the nearest functional chamber - he knows where those are too.

An alternative source of advice is the RAN School of Underwater Medicine, HMAS PENGUIN, telephone (02) 960 4444 extension 333 during working hours (8 - 4). At other times the duty Medical Officer at the School can be contacted through the RAN Diving School at HMAS PENGUIN (02) 960 0321 where the telephone is manned 24 hours a day. The duty hand can then "beep" the doctor. When contacting HMAS PENGUIN make it clear you want medical advice about a diving emergency and remember to give your name and telephone number (so that you can be contacted) and the patient's name.

For these divers visiting Thailand the nearest American Recompression facility is at Subic Bay in the Philippines. However both the Malaysian and Singaporean Armed Forces have Recompression facilities and do treat civilians.

GOING DEEP WITH THE LATERAL THINKERS

Vickers-Intertek reported last year that successful trials had been completed using a new concept of dive procedure which enables direct work on well-heads, etc. without the usual risks of exposure to ambient pressure of the divers, and reduced contamination and fire hazard. Like all great ideas it is based on an obvious and simple thought, but one which is such only in retrospect.

It is pressure changes which result in the complicated and sometimes dangerous physiological changes in the diver, yet the 1 ATA suits presently available are somewhat cumbersome. However, to provide a gaseous 1 ATA environment for the diver introduces problems of strong pressure hulls and danger from explosive or toxic fumes during welding and other work procedures. The answer was to combine the advantages of a water environment with those of 1 ATA element.

The Neutrobaric chamber is lowered onto the seabed installation and sealed.

The vessel is depressurised to 1 ATA but left full of water, worker divers being now able to enter from a submersible mated to it, wearing scuba or hose supply.

Equalisation between the diver chamber of the submersible and the work system will be easy as the pressure reduction will be accompanied by little change in the volume of the water as it is decompressed.

The only problem posed by this system will be the dismay caused among those who have an interest in the solving of problems of adapting to great ambient pressures or whose self image (and pay rates!) are tied to depth measurements of the work site.

SUBSCRIPTIONS

Members pay $20.00 yearly and Associate Members $15.00. Associate membership is available to those neither medically qualified nor engaged in hyperbaric or underwater related research. Membership entitles attendance at meetings and the Annual Scientific Conference and receipt of the Journal/Newsletter. Anyone interested in joining SPUMS should write to the Secretary of SPUMS, Dr Christopher J Lourey, 43 Canadian Bay Road, Mount Eliza, Victoria, 3930.

COURSES IN UNDERWATER MEDICINE

The Diving Medical Centre will be running courses in Underwater Medicine in 1980 and 1981.

1. November 22nd to 29th, 1980 Bay of Islands (New Zealand) with an optional week 29th November to 6th December 1980 in Tonga.
2. Honolulu, June 2nd to 16th, 1981. First week in Honolulu, second week in the outer islands.

For further details contact:
Diving Medical Centre,
6 Hale Road, Mosman, NSW 2088.

NOTES TO CORRESPONDENCE AND AUTHORS

Please type all correspondence, in double spacing and on only one side of the paper, and be certain to give your name and address even though they may not be for publication. Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide these in a presentation suitable for photo-reduction direct. Books, journals, notices of symposia etc., will be given consideration for notice in this journal.

CONSTITUTION

As only one member wrote to the Secretary before 31 March 1980 the constitution circulated with the October to December 1979 issue is deemed to have been accepted by the members and becomes the Constitution of the South Pacific Underwater Medicine Society.

JOHN KNIGHT
President SPUMS

OIL RIGS PATROLLED BY SEA STARS

Any clean, hard object placed into shallow, oceanic waters will, in a short time, normally acquire a thick encrustation of organisms coating its surface. This is a significant problem on the legs of giant offshore rigs, where the added weight and increased surface area caused by such fouling make the rigs more vulnerable to waveshock during severe storms.

Of all the encrusting organisms, marine bivalves are the greatest offenders on the rigs. These molluscs rapidly form huge masses of heavy shells, which previously could only be removed at great cost by divers using hammers, chisels, and high-pressure water jets. Now several major oil companies are experimenting with a type of biological control of the encrusting bivalves, using the common sea star.

Chevron USA, whose oil rigs dot the California coastal waters, has been plagued with massive fouling by the Californian mussel *(Mytilus californianus),* the clear jewel box *(Chama pellucida).* and the giant rock scallop *(Hinnites multirugosus).* To counter these dangerous build-ups, Chevron researchers released large numbers of the common West Coast sea star *(Pisaster ochraceus)*, a known predator of bivalves, onto the platform legs. To ensure a continuous invasion of more sea stars from the surrounding sea floor, the workers also removed from the bases of the legs a thick barrier of stinging sea anemones (*Metridium* spp.), which had previously impeded the movement of the sea stars onto the rig.

The sea-star predators soon devoured most of the bi-valves, and the experiment was deemed a success.

Only the dead mussel shells, however, were eventually removed by wave action from the rig, due to their loose attachment to the substrate by byssal threads. The firmly cemented jewelbox and scallop shells continued to present a problem, and these empty cemented shells additionally acted as a protective substrate onto which more jewel-box larvae could settle out.

Fouling on Atlantic oil platforms, especially those of the North Sea, may be more easily controlled using sea stars. Since no species of Chama live in the North Sea, the encrustation by bivalves is mainly due to the activities of the blue mussel *(Mytilus edulis).* once the sea-anemone barrier has been removed, the mussels may be quickly brought under control by releasing large numbers of the highly predaceous, common European sea star *(Asterias rubens)*.

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