LONG TERM NEUROLOGICAL CONSEQUENCES OF DEEP DIVING

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PREFACE

The format of this publication reflects that of the meeting itself; that is, it is divided into four sections, the first three of which are introduced by keynote papers, followed by individual discussion on each paper, and concluded with a general discussion. The fourth session is a general discussion of the whole meeting.

The general discussions have been edited and summarised to concentrate on salient points. By contrast, the editing of the individual discussions has been limited to the removal of repetition and irrelevant material; in this way it is hoped that some of the flavour, as well as the content, of the meeting has been retained.

The organisers of the meeting are grateful to Norsk Hydro, Norske Shell, Saga Petroleum and Statoil for their generous financial support.

The Society's thanks are due to Dr Børge Minsaas of the Norwegian Petroleum Directorate and the local representatives of the Eurpean Underwater Biomedical Society in Stavanger for taking the largest part in organising the meeting; to Mr Philip Bell for acting as rapporteur and preparing the transcript; to Professor Ian McCallum for acting as chairman; to my fellow editors; and to Ms Kari-Ann Lian and Miss Angela Gray for secretarial assistance.

Tom Shields
for EUBS.

THE LONG TERM NEUROLOGICAL CONSEQUENCES OF DEEP DIVING.

AN INTRODUCTION TO THE PROCEEDINGS.

Prof Ian McCallum.

The form of this conference followed previous workshops organised by the EUBS by involving a limited number of invited participants in order to take advantage of the maximum amount of informed discussion between a relatively small number of people; but with full publication of the papers and discussions so that the members at large could share in the exchange of views.

The conference originated in discussions between Drs Elliott, Minsaas and Shields in 1983 when it was decided that there was a need to review the neurological effects of deep diving, particularly in relation to the compression phase and the stay at pressure. It was recognized that there were two factors which needed to be separated: the possible effects of the stay at pressure and secondly, the long-term effects of decompression from high pressure; although this might not be completely possible.

The view at the start of the conference, that a better understanding was required of the effects on the central nervous system of high ambient gas pressure, has been reinforced by the evidence or lack of it placed before the workshop. Time and again the discussion came back to the high pressure nervous syndrome and the relationship of its different elements to electroencephalographic changes and to possible, although hypothetical, permanent changes in the brain and central nervous system. There were difficulties over interpretation of single and repeated episodes of HPNS and what, if any, the permanent effects might be. There was quite marked disagreement about the significance of EEG abnormalities and what, if any, action should be

taken, particularly if the abnormalities could not be related to symptoms. If in EEG terms the changes were to be regarded as trivial, did this mean that there would be correspondingly trivial and reversible changes in the central nervous sytem or not? A number of uncontrolled variables including hypothermia, subject variations and a past history of damage to the CNS system unrelated to diving, create difficulties. What seems to be uncontrovertible is the manifestly greater danger of much shallower diving in terms of neurological decompression sickness.

In the field of neuropsychiatric testing, it became clear that there are great limitations in the use of such techniques to detect brain damage and that where brain damage is known to be present, a wide range of ability may still exist. The use of chamber dives in association with such tests has been advocated as having predictive value in the selection of subjects for operational deep diving. The widest agreement appears to have been reached over the need for standards of assessment for the central nervous system, neuropsychiatric testing, and the pathology of CNS damage and the need to seek more refined methods of investigation. In this, the EUBS must surely have an important role. In view of the lack of hard evidence of actual neurological damage from deep diving and the need for more data, is there a case for limiting deep diving now? Many feel that deep diving, when properly carried out, is safe and I think it is fair to say that there was little support for limiting such activity at the present time, but rather for intensifying the monitoring of those taking part in it.

OPENING ADDRESS

BØRGE MINSAAS, M.D., Norwegian Petroleum Directorate,
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We meet in the shadow of one of the worst disasters to the diving community killing five experienced divers by the explosive decompression from 10 ATA to 1 ATA when the chamber atmosphere escaped through the trunk connecting bell to chamber. This is a sombre reminder of the numerous hazards facing man when entering a hostile environment.

The reason behind our being present here today is the debate about what happens to the central nervous system in man when exposed to an increased ambient pressure or changes in an increased ambient pressure.

On behalf of those who have helped organise this workshop I wish you welcome. For two days we shall discuss and, if possible, decide on the state of knowledge in a restricted area of diving medicine and physiology: the long term effects on the central nervous system from pressure exposure.

The history of this relationship is as old as diving medicine and was early highlighted by the experience of giving tadpoles an anaesthetic and then reversing the effect by pressure. If anybody applied for support for such research today I think he would receive a firm "no" for an answer.

A Norwegian researcher sent a correct application form to the correct authorities in the name of a certain Charles Darwin for a 10 year grant to travel around the world to have a look at the world's biosphere because he thought mans' concept of the origin of all life was inaccurate. The application was properly handled and then turned down.

This should pinpoint the problem facing us present here today. How does one go about collecting the existing knowledge and information and then extrapolate about the future maintaining the scientific standards we are trained in? Can we arrive at the proper conclusions without the proper research? What constitutes "proper research?" Can

we point to the necessary basic research that must be done? What else should be done, what can be done?

Man under pressure is a potent source of invaluable information about human physiology and thus he is a very attractive person. The area where information can be gathered moves from the area of basic research through all kinds of experimental chamber dives to the monitoring of operational dives. Where should we concentrate our efforts?

Our conquest of the natural resources on the seabed has so far necessitated the use of manned intervention in the sea. The resources on The Norwegian Continental Shelf are found in increasingly deep waters and necessitate very careful evaluation of the future needs for human intervention capability in the water.

We are thus faced with a multifaceted problem of conflicting areas where we must try to find the way ahead. Within the limits of this workshop's definition , that is our job. Good Luck.

DEEP DIVING, A CONTROVERSIAL MEDICAL PROBLEM

Jens Smith Sivertsen, MD,
Kongsmyrveien 27 B, 5070 Mathopen, Norway.

Mr Chairman, Gentlemen,

Before I start I want to make it clear that I am not representing Mobil Exploration (Norway) here today. The statements and opinions I am to express are mine and do not necessarily reflect the policy of the company.

I have been invited to the this scientific workshop to open by presenting some rather unscientific viewpoints on diving safety and the health hazards of very deep diving. Since 1978, when I worked for the Norwegian Petroleum Directorate here in Stavanger as their advisor in diving medicine, I have quite frequently, through newspapers, lectures, discussions, expressed my scepticism on extended diving depths. Results from deep diving research for the last five years brought to my attention, especially be people promoting deep diving, have not altered this scepticism of mine.

It seems to me that a major area of discrepancy between myself and them has to do with the attitude towards diving and the diver. They look upon the diver as a pioneer, expected to push the limits of human achievements like an athlete or a sportsman. I prefer to look upon the diver as a worker with equal rights for worker protection as any other employee.

The diving activity today, and offshore deep diving in particular, violates quite a number of the Norwegian Acts related to worker protection and environment. But in the interests of the Nation, governmental authorities wink at this fact, peep through their fingers, as we say in Norway. Our national economy is dependant on the exploitation of our submarine oil and gas resources, and such exploitation is dependant on the contribution of divers. These economic forces supported by technological enthusiasm creates an enourmous motive power for

further progress ready to buy any expert willing to justify such progress.

Who is responsible for drawing the limits? Very few people concerned with diving have the necessary neutrality and independence to shout out warnings without being accused of having hidden intentions. It is this situation that places a heavy and sincere responsibility on people of the medical profession and especially on doctors engaged in occupational health. If we keep quiet, who else will raise their voices? But are there any reasons for worry? Indeed, there are. A collegue who is a participant in this workshop stated some years ago that every deep dive is a physiological experiment. As far as deep diving beyond 250 metres is concerned, I support his statement, even today. One can not just take the experience gained from shallower diving and then, so to speak, extrapolate the data to greater depths. We face a new situation, with new medical problems and health hazards.

The most obvious is the effect of compression on the central nervous system. In which other work situation other than deep diving, would one accept that the worker is suffering from reduced consciousness, loss of memory, vertigo, a pathological electroencephalogram, muscular incoordination and even blackouts and hallucinations? All these phenomena occur in the diving situation where the safety of the person is dependant on his own alertness and cooperation. I, personally, do not know of any other occupation in which this is accepted, and what is more frightening to me, we can not explain why this happens. What is the mechanisms behind these phenomena? And we certainly can not rule out long term effects on the CNS. This is why I think the word experiment is appropriate for this activity.

In addition to the HPNS problems, we face a number of other problems as we go deeper. As you know the tolerable limits for parameters like oxygen concentration, purity of gas, temperature of inhaled gas, temperature of the chamber, bell and diving suit, gas flow resistance in the diving gear and so forth, becomes narrower and narrower the deeper we go. A minor variation in the oxygen concentration, in the order of tenths of one percent, or a change in ambient or inhaled gas temperature of only a couple of degrees centigrade, harmless to divers at shallower depths, may prove fatal in deep diving. Trifling technical failures or irregularities or marginal deviations from normal procedures of no importance at 100 metres may cause the death of the

diver at 300 or 400 metres. In case of a medical emergency inside the chamber or diving complex, the access to the patient for medically trained personnel will be considerably delayed due to the mandatory slow compression rate to high pressure.

I could list many more areas of uncertainty and lack of knowledge concerning deep, prolonged and repeated exposures to high pressure, for instance, haematological effects and fluid balance disturbances. But I will conclude here. We all know that selected human beings can be exposed to, and perform work at, very high pressures and brought back to atmospheric pressure without any obvious harm or injury. We do not need more proof of this. Record dives are of limited value in this connection. What we need, in addition to improved diving technology which will make the diver superfluous, is a better understanding of the mechanisms behind the alarming symptoms we sometimes observe in deep diving, particularly the disturbance of the central nervous system. Such knowledge may enable us to prevent the immediate effect that these disturbances have on diving safety and may also give us the answer to the crucial question of long-term effects.

It is my honest conviction that operational, industrial diving deeper than 250 or 300 metres should be halted until diving physiology has caught up with diving tecnology, until it has been confirmed that a depth of 300 metres or more is a safe place to put man at work. I have a sincere hope that this workshop will baffle all I have expressed until now. Thank you, Chairman.

I: CLINICAL NEUROLOGICAL PROBLEMS IN DEEP MANDIVES AT NUTEC

Stein Tønjum, MD,
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Introduction
From 1980 to 1983 four deep dives were performed at the Norwegian Underwater Technology Center (NUTEC). Twenty-four divers were involved, six in each dive, and the depths were in the region of 300 metres of seawater (msw)(31 ATA) to 500 msw (51 ATA). During these dives a comprehensive battery of neuropsychological, and performance tests were used for monitoring effects on the central nervous system (CNS). In connection with an open sea dive to 300 msw in 1983, NUTEC was also involved in performing comprehensive medical and physiological examinations of 13 divers before and after dive.

In 1980 NUTEC (NUI) performed their first dive to the area of high pressure nervous syndrome (HPNS). In a dive called Deep Ex 1 to 300 metres of seawater (msw) (1), six divers participated, three of them professional divers and three non-professional divers (Figure 1). The subjects were subdivided into two groups of three, and one group was compressed breathing a helium-oxygen mixture (heliox), the other group breathing a mixture of helium, 10 % nitrogen and oxygen (trimix). The compression rate was to be identical for both groups, provided that no severe HPNS symptoms occurred, and was supposed to take 284 min. However, the planned holding period at 250 msw had to be extended from 3 hours to 8 hours and 41 minutes for the heliox group. One diver in this group was totally unaffected by HPNS, while the two other divers were severely incapacitated with vertigo and nausea when they moved their heads. The initial onset of these symptoms occurred on passing 210 msw, and neither of the two divers were able

to engage in other research activities for some time though they tried to do so.

After the extended holding period at 250 metres the group was compressed to 300 msw with no deterioration in the status of any of the divers following the compression.

After a night's rest, they felt much improved the next morning, and there were no further problems with HPNS for this group during the rest of the dive.

The three divers compressed on trimix had subjectively minor symptoms compared to those reported in the heliox group. However, one subject reported nausea, dizziness, euphoria and ringing in ears on reaching 250 msw.

The compression proceeded on schedule with the divers reaching 300 msw in excellent spirit, which was mainly due to the narcotic effect of the 10 % nitrogen in the breathing gas.

Conversion of the trimix group chamber atmosphere from trimix to heliox took place on the second day under pressure. A time of 1 1/4 hours was required to lower the nitrogen from 9.7 to 3.5 %. Two

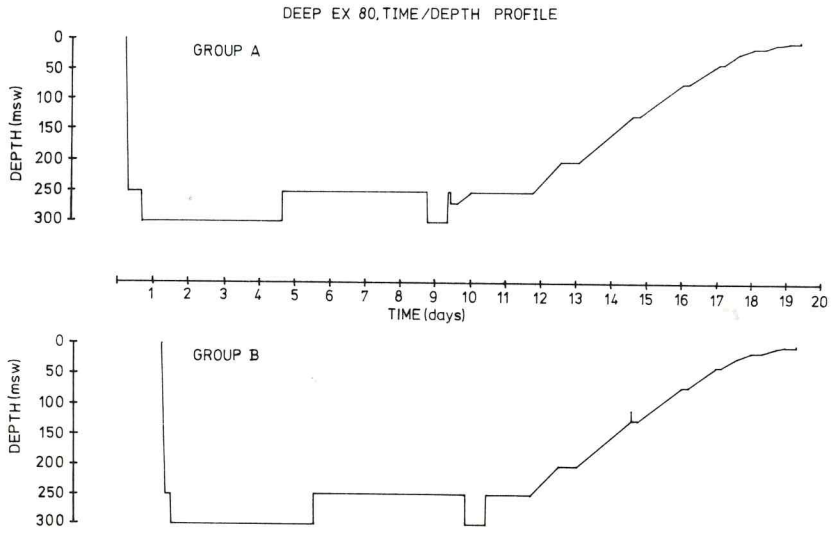

Figure 1. The dive profile for Deep Ex 80.

divers reported no symptoms following the gas change, but one diver reported flu-like symptoms starting approximately 7 hours after the reduction in chamber nitrogen, but these had disappeared the next morning.

One object of Deep Ex I was to test unlimited duration excursion ascents. The dive profile executed was fully within the realm of standard navy commercial operation procedures. Two ascents with each group were made from 300 to 250 msw. Following one of these ascents, all three divers in one group reported mild knee pains. In addition Doppler recording revealed presumed gas bubbles on the arterial side, i.e. art. femoralis and carotidis in several divers during and after excursions.

Deep Ex I was a very exhausting dive for the divers involved, because it incorporated 12 different research projects. However it did not last for more than 20 days. After the completion of the dive, two of the non- professional divers reported that they were not interested in being involved as subjects in future deep dives. One subject had psychological distress with depression lasting for months after the dive was completed.

Deep Ex II, (2), a 34 day dive with maximum depth of 500 msw was completed in the fall of 1981 (see Figure 2). It included fifteen research and development projects. Six divers took part in the dive, five of them professional divers, and one research worker with diving experience. Four of the six had participated in Deep Ex I, and Deep Ex II used again both trimix with 10 % nitrogen and heliox breathing gas during compression. The one diver very susceptible to HPNS who had been in the heliox group in Deep Ex I, was placed in the trimix group this time.

The compression time for the two groups was planned to be 32 hours for the trimix group and 25 for the heliox.

At 250 msw the very HPNS susceptible subject in the trimix group started to complain about dizziness and nausea. When reaching 300msw he got worse and stopped communicating. He was not able to fill out the status questionnaire, and reported later that he had not registered that his codivers had moved him to his bunk. At 350 msw the same diver was still not able to fill out the status questionnaire due to severe HPNS symptoms. Another diver in the trimix group reported dizziness, euphoria and vomiting attacks. The trimix group arrived at

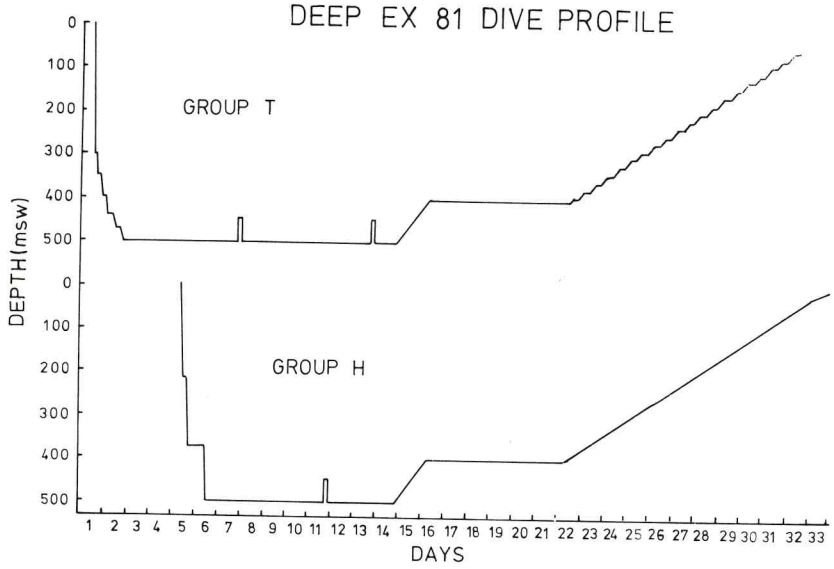

Figure 2. The dive profile for Deep Ex 81.

500 msw approximately 9 hours after schedule with two divers reporting trouble with mental concentration. All divers in this group seemed intoxicated during the period they stayed at 500 msw on trimix. They did not seem trustworthy and it was a tough time for the surface personnel responsible for the dive operations. The following is a typical example: when one diver performed the first wet dive ever on trimix, the stand-by diver was lying on his back in the dry part of the wet chamber. When he was asked if he thought that was a suitable position he confirmed with laughter that he was in total control of everything.

On their dive day 4 conversion of the trimix group chamber atmosphere from trimix to heliox started. In the evening of the following day two of the divers seemed as if they had cerebral slow motion. At this stage the nitrogen concentration had been lowered to 2 %. During that night two of the three divers became hallucinated, both visually and auditory (bird, snakes and music). They rejected the following morning that they were frightened, but admitted it, however, later when the dive was over.

In the heliox group two divers reported HPNS symptoms when reaching 216 metres, and at 376 msw, the same two divers reported dizziness and nausea, and the third reported visual disturbances in addition. When using almost the same compression profile in a 350 msw dive in 1983, other divers also reported symptoms at this early stage.

When reaching 500 msw one diver was severely dizzy and stayed on his bunk more or less continously for the following days unable to move freely around. One diver complained about some dizziness, and reported also that he felt very exhausted when executing even minor tasks during the time they stayed at 500 msw.

In both groups one diver was relatively symptom free, one diver had some symptoms, while one at periods had severe symptoms when staying at maximum depth.

After completion of the dive, the two divers who had hallucinated in connection with the gas switch from trimix to heliox explained that this incident had been a most scaring experience and had spoiled the rest of the dive for him. All the six divers who participated in the dive needed months before they had recovered both mentally and physically. One diver had tremor which lasted for months without a proper causation being established.

In 1983 two simulated heliox dives to 350 msw at NUTEC confirmed that it was possible to a great extent to avoid HPNS by using a compression profile with intermediate stops before reaching the HPNS area.

In the first dive six subjects were compressed on heliox with intermediate stops at 216 msw for 4 hours and at 296 msw for 12 hours. In total they took 24 hours from leaving surface to reaching saturation depth at 350 msw (see Figure 3).

Despite nausea and dizziness among all six divers on reaching 216 msw, only two had some dizziness on reaching 350 msw. The major problem throughout the compression was the heat. The following conclusion could be made from this dive: compared to previous deep dives at NUTEC (Deep Ex I and Deep Ex II) the divers were minimally impaired by HPNS. Although signs and symptoms of HPNS were evident, all six divers felt fit to start working in the water on the second day, 24 hours after leaving the surface.

However, a hypothesis was that the nausea and dizziness which all six divers experienced in this dive could be reduced or eliminated if

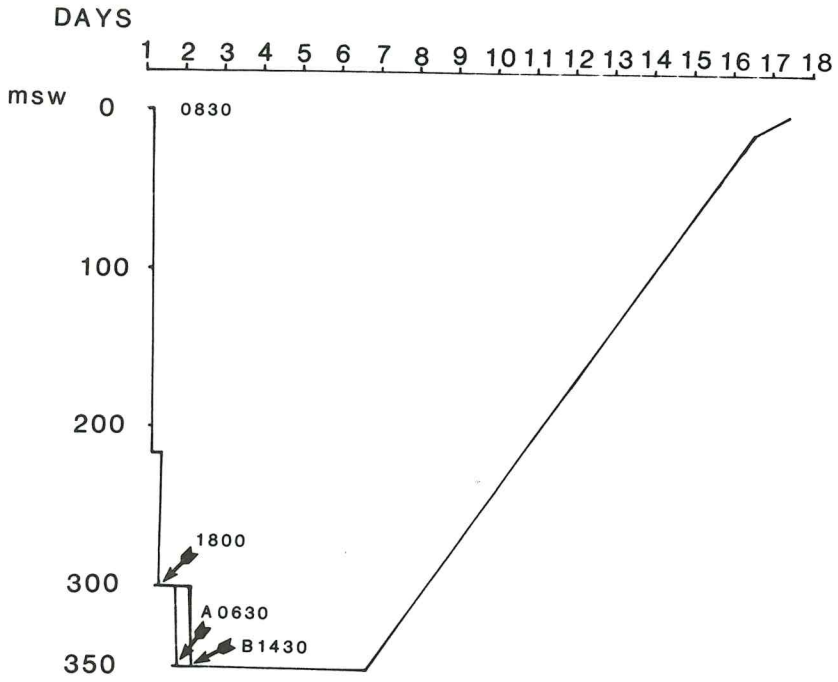

Figure 3. The dive profile for an onshore trial dive to 350 msw.

there were intermediate stops between 100 and 200 msw. In that case the divers could in some way adapt to the pressure and therefore lessen the HPNS symptoms which start to occur between 200 and 250 msw. This was confirmed in the second dive in 1983 where the divers stayed at 100 msw and 200 msw for 2 hours, and as for the previous dive at 300 msw for 12 hours (see Figure 4)

A third dive in 1983 was an open sea dive to 300 msw which took place in a Norwegian fjord. The same compression profile was used as for the second onshore trial dive in 1983 (see Figure 4), and 5 of the same divers participated. In the following papers empirical results from the 4 simulated dives and some results from the open sea dive will be discussed and the post-dive neuropsychological and neurological results presented.

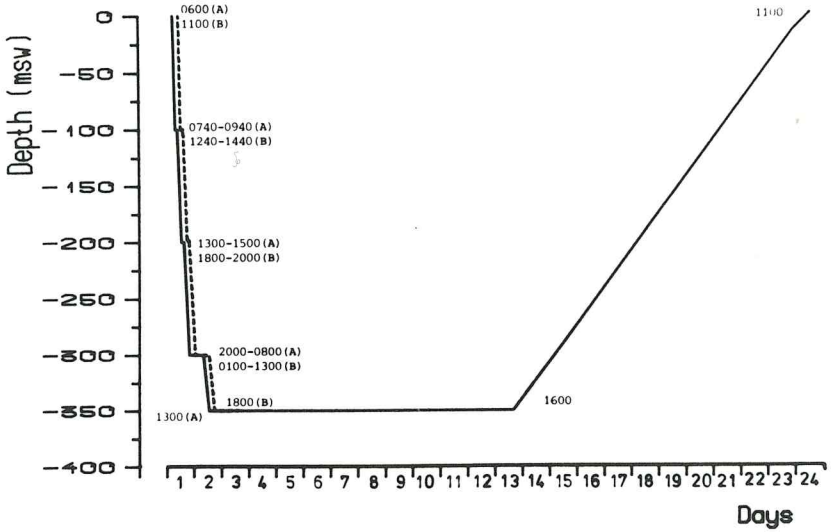

Figure 4. The dive profile for an onshore trial dive to 350 msw.

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QUESTIONS FOLLOWING DR TØNJUM'S PAPER

Dr Shields: The topic being discussed is the long-term neurological consequences. You have said that in Deep-Ex I, one of the divers had depression for several weeks afterwards and you have described the feeling of terror in the last dive. Could you elaborate on that aspect of the subject?

Dr Tønjum: The diver from the first Deep-Ex I, (we were not experienced enough exactly to know what was going on) felt that he needed help and that he was very low in mind and he finally contacted a psychologist to get help. After 2 to 3 months, he was more or less back to normal.

Dr Peterson: Did this individual have any special experiences during the dive that might be considered abnormal from the standpoint of a routine working dive?

Dr Tønjum: Yes, as I said earlier, the whole Deep-Ex I dive was very strenuous for everybody involved. We went at it very hard with long hours into the night and we had some projects in it that were very stressful. For example, one test of a survival system was very tough; we did some thermal studies where we were cooling the divers for a couple of hours. Altogether, this was a very special dive. It was not an ordinary working dive. And the same can be said for Deep-Ex II, of course.

Dr Török: Just a further point of clarification, please, about the same man. Was it his first psychiatric incident, his first incident of depression, or has he had a history of such things? What age group was he?

Dr Tønjum: He was 32 at that time and, as far as we know, it was his first episode, but the person is labile.

Prof Donald: Could I follow that question? Did this subject have any other symptoms or signs of depression as we know them, insomnia, loss of weight, appetite and so on?

Dr Tønjum: In Deep-Ex I, as I told you, we did not have enough experience of deep diving and we did not follow the divers up properly. In Deep-Ex II we almost made the same mistake, but we started weighing in the course of the dive and we had divers with up to 10 kg weight loss. I think the diver who lost the least lost 5 kg. In the two

last dives we have had, we have been watching this more carefully and there is weight loss in all divers during the dive.

Prof Bennett: A number of points here which I think we have got to separate out. We are talking about experimental dives. The nature of the experimental type of dive is highly stressful. This was your first dive in Norway. You were working the divers for excessively long hours and I was present when we asked that you try to reduce them because of fatigue and so on. Aside from that, the signs and symptoms of hallucinations and so forth, and switching gases, these again were in an experimental procedure and one which I certainly would not recommend for anybody making a deep dive in a commercial sense.

I am very interested in the man who had depression. Again, I gather from what you are saying that you did not have a pre-psychiatric screening of these individuals to know what the psychiatric status of this man was beforehand, or whether he was pre-destined for this kind of reaction in this sort of situation. The difficulty with volunteers for experimental dives is that they often volunteer for reasons that are not of the soundest quality and you do need to be very careful in selecting them. Finally, in connection with the American diver* (and I will say more in my own presentation), you should understand quite clearly that, when those EEG's were sent back, it was not only Duke EEG experts who read them. We are very conscious of potential criticism of bias, so we sent these EGG's for evaluation to other experts who had no relationship to Duke. Their considered opinion was that, though there was the occasional spike (and let us make it clear again, there were not lots of spikes) it was an occasional intermittent spike. They

* *Editorial note*
Dr Tønjums paper was submitted some time after the meeting and regrettably does not reflect precisely what was said at that time. His actual text taken from the tape of the proceedings was as follows:

"One diver who was supposed to participate Deep Ex II was an American citizen. He had earlier taken part as an experimental diver in deep dives at Duke University. Before Deep Ex II all divers had to complete a broad spectrum of neuropsychological tests, which included baseline EEG's. These later examinations were performed at the neurological department at Bergen University clinic. Two separate EEG's from the American diver revealed spikes and it was concluded that these findings could mean a pre-epileptic condition. For this reason he was not allowed to participate in Deep Ex II. Copies of the EEG were sent to Duke, but despite this he later participated in a new deep dive at Duke, evidently because he had been cleared by American neurologists."

were not convinced that this in any way resulted from a dive and had probably been there for a long period ahead of the dives. He had no pathological result from that spike and had never had any kind of convulsive activity or pre-convulsive activity. He was 100% intelligent, no problems whatsoever, and still is. He still has an occasional spike on his EGG and he has made further deep dives and feels perfectly fine. Should you stop a man's livelihood on the basis of something which appears aberrant without any signs or symptoms?

Dr Tønjum: I would like Professor Aarli to comment on that because we got in contact with him at that time and took his advice.

Prof Aarli: We were presented with his EEG, not with the diver and we were asked if it was normal. The only thing we could say was it was not normal, but I would not draw any conclusion as to the cause of this pathological EEG. I am sure everyone who read his recording would say that this is not normal, but no one would say that this is an EEG that you could see from a patient with epilepsy. If you ask me if this is a normal EEG and would you recommend a dive, my answer would be that it is not normal.

Prof Bennett: Can I just come back one minute on that. I did a lot of EEG work as well and did some six months training in a Navy hospital on EEG. I saw a lot of EEG's and I would agree with you that when you see an odd spike like that it is not something you would see in a normal EEG. On the other hand, I think the EEG is basically a soft science, it is not an objective science. In some diseases where there is a focal point then maybe it is very good but again I make the comment that one has to be careful about making dogmatic statements on such slight information when the man is perfectly healthy, and has been diving all his career and has already made two deep dives without any trouble whatsoever. In fact, he is the finest deep diver I have ever seen in my life, in my 30-year career and, again, do you destroy a man's career on the basis of one spike occurring in his EEG for which there is no other defect.

Prof Elliott: Dr Tønjum, I have heard what you said and, as you know, I have been closely associated with your Institute. I think it is becoming evident that when we are talking about the consequences of deep diving, we are looking at those things that exist after the decompression. It is also apparent to us all that what appears after the dive can be due to something that existed before the dive, be it some psychiatric disturbance or some possible pre-epileptic condition. It can be due, perhaps, to the stress of the dive. I would like to support the point that Peter Bennett made that in Deep-Ex I those outside the chamber, let alone the poor guys inside the chamber, were suffering from a certain degree of sleep deprivation. I think that is a very important factor.

Then, of course, there are the factors that we are particularly trying to pursue at this meeting. Are there any unknown aspects of the deep dive itself which should give us additional cause for worry? Would it be fair to say, from what you have described, that you have found things after a dive that would give you cause for concern? But that you are not able at this stage to say which of those possible causes they might be?

Dr Tønjum: Yes that might be right. I think after the presentation of the next two papers that this will be more apparent.

Dr James: I wonder if I could ask two things. Firstly, in the dizziness that you have mentioned, is there a rotational component, is it a vertigo or is it a non-specific dizziness? Secondly, what was the timescale over which you changed the partial pressure of nitrogen?

Dr Tønjum: The time scale, if I am not wrong, for the nitrogen was 24 hours. I think it was in the course of 24 hours brought from 10 % down to 2 %. Concerning the vertigo, perhaps, Dr Molvær is the best person to answer that.

Dr Molvær: One of the divers concerned had ENG electrodes connected in the chamber and I registered his ENG during the dizziness phase and he had no nystagmus. He was not able to describe exactly what the dizziness felt like, but it was not typically rotational. He was not spinning.

Dr Rostain: First, I am surprised at the dramatic symptoms there have been during these dives. I would like to know the duration time of your compression. Secondly, I should like to know what the ratio was of professional divers to non-professional divers. I would like to know if the professional diver had the more dramatic HPNS, or was it the non-professional diver who had the more dramatic experience.

Dr Tønjum: For the heliox compression we used what they had done at Pennsylvania earlier in the Predictive Studies. The philosophy from the start of these dives was to create HPNS, not to prevent it. So it was a very fast compression that we used for the heliox group. For

the trimix group the compression profile was co-ordinated with Dr Bennett and was probably as much as we could know about trimix compressions at the time. In answer to the second part of your question, yes it was the amateur diver who was most severely hit with HPNS, at least in the first Deep-Ex dive. In the second Deep-Ex dive the non-professional diver was semi-comatose from HPNS and he was hit the hardest, but we had professional divers as well who were severely hit by HPNS.

Prof Bennett: Let me just add that we are talking here about 1980 and 1981. The Atlantis trimix dive series, which has completed the four dives, now shows very clearly, in agreement with the French work, that the 10 % nitrogen is too high and will induce euphoria and narcosis. With the 5 % you get extremely good control of HPNS although you will get a little tremor, with very effective removal of the narcotic effects. The summary that I presented at the Royal Society meeting and which is also published in the Japan proceedings, shows graphic representations in support of that statement. I think one of the problems is that the work you are describing was at a time when we were still learning. This is experimental work and, in experimental work, you expect to see signs and symptoms that are perhaps going to be eliminated. But to go to 1000' certainly should be very easy. I don't understand the comments I have heard already that 300 metres is a dangerous dive. I don't believe it: it has been done (operationally) for six months or more on the Cognac project and we have done it repeatedly at Duke without any problems at all.

Dr Peterson: Stein, could you describe what was done for the individuals in the heliox group in Deep-Ex II to try and alleviate their symptoms? I think it will give people some insight into some of the mechanisms involved. These would be the temporary ascents from maximum depth. There were two individuals, one in particular, who was quite dizzy and the other who had more unsteadiness than actual dizziness. They remained essentially unchanged for approximately 4 days at 500 metres. There was no worsening of their condition, but no improvement either over the course of this time. On the fourth day both of these individuals were taken rather rapidly from 500 metres to 445 metres. On passing about 480 metres, the individual that had been the worst, that is the one that was quite dizzy, said that he was looking at some detail on the side of the chamber and, all of a sudden, it changed from fuzzy to very clear. Upon arrival at 445 metres, he was much improved, although he felt he was not 100 %. Over the course of that day, spending the night at 465 metres and then with the return to 500 metres over about a half-hour period before they woke up, this particular individual had no further symptoms of HPNS for the rest of the dive. The other individual had improved on the ascent but had a return of tremor on recompression to 500 metres. So, here is a situation where there is almost no change at depth over many days and then a very dramatic reversal of these symptoms and an ability to get back to 500 metres without a return of symptoms at least in one case. I think this presents some very provocative possibilities for determining mechanisms involved and the ramifications from a long term health standpoint.

Prof Bennett: One quick final comment. I understood we were going to be talking about post-diving neurological deficit, not the experimental effects of what goes on during the dive. If you are going to talk about experimental diving, let us be quite clear that if you want to dive very deep there are two parameters you want to have. You want 5 % nitrogen if you are going to 500 metres to control your HPNS, and you must have a very slow compression. The compression rates that were used on the NUTEC dives would not be acceptable to us now in the light of our subsequent knowledge from the Atlantis deep dives.

II: REVERSIBLE AND POSSIBLE IRREVERSIBLE CNS CHANGES OF DEEP DIVING. A DISCUSSION OF SOME EMPIRICAL STUDIES.

Ragnar J. Værnes,
The Norwegian Underwater Technology Center, Bergen Norway.

In Deep Ex I (1) six subjects were compressed to 300 msw, three on heliox and three on trimix with 10 % nitrogen.

During compression in Deep Ex I to 300 msw minor EEG changes were registered in the trimix group. In Figure 1 the bars at the left are slow wave activity, at the right alpha band activity, for the left and right hemisphere respectively. The EEG is expressed in percent changes from predive control level which is represented by the x-axis. There were marked changes in the heliox group with theta increase and alpha inhibition, there were, however, both individual differences and hemispheric differences. For Deep Ex I there was a close relationship between the compression EEG's and the postdive measurements as indicated in Figures 1 and 2.

For the heliox group a marked increase in tremor that lasted throughout the compression was found. For the trimix group a slight increase in this tremor was found during the first phase of compression. On reaching saturation level (31 ATA) tremor returned to predive level (see Figure 3). Despite minimal HPNS problems in the trimix group, the cognitive performance tests indicated a narcosis, especially on short and long term functions. The conclusion after Deep Ex I, however, was that trimix seemed to inhibit the HPNS.

Deep Ex I was followed by Deep Ex II, in 1981 a chamber dive to 500 msw with four of the same divers and the same examinations performed. The subjects were divided into two groups. One group (N=3)

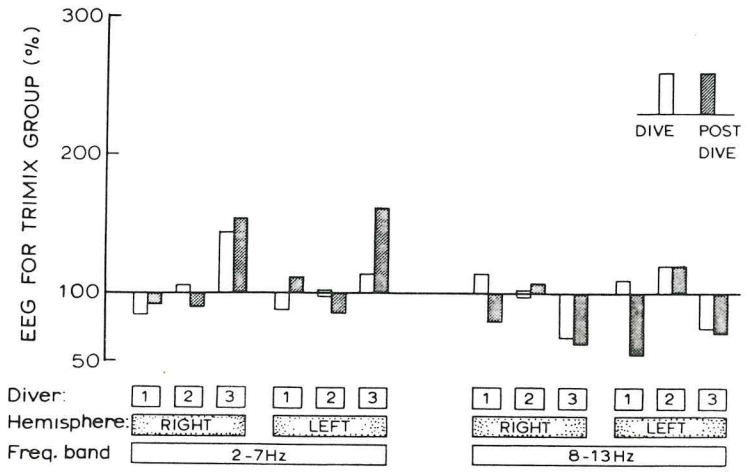

Figure 1. *Trimix Group: Percentage of change from predive level for slow wawe (2–7 Hz, left) and alpha band (8–13 Hz, right) during compression to 300 msw.*

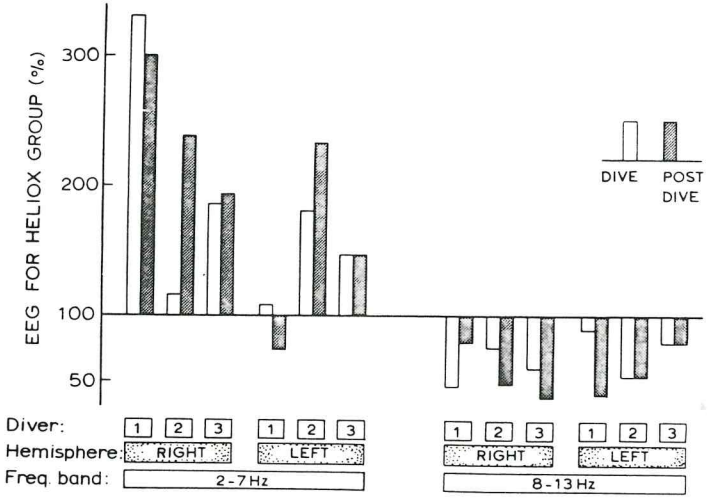

Figure 2. *Heliox Group: Percentage of change from predive level for slow wawes (2–7 Hz, left) and alpha band (8–13 Hx, right) during compression to 300 msw.*

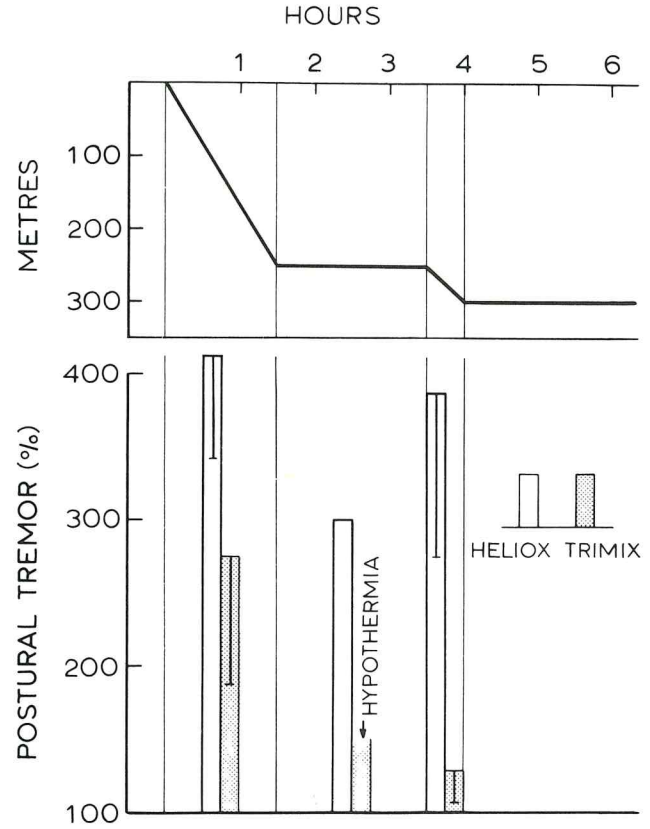

Figure 3. *Percentage of increase from predive level for postural tremor during compression for heliox and trimix, and before and after change of gas for trimix group only.*

breathed trimix with 10 % nitrogen during the compression and the other group (N=3) breathed heliox (2).

To investigate further if trimix did inhibit HPNS the most sensitive HPNS subject from Deep Ex I who had been in the heliox group was now placed in the trimix group as subject No. 3.

In Figure 8 the power spectrum EEGs from the trimix compression to 500 msw are presented. The stippled curves are the mean predive control EEGs averaged from 5 control measurements. The Fast Fou-

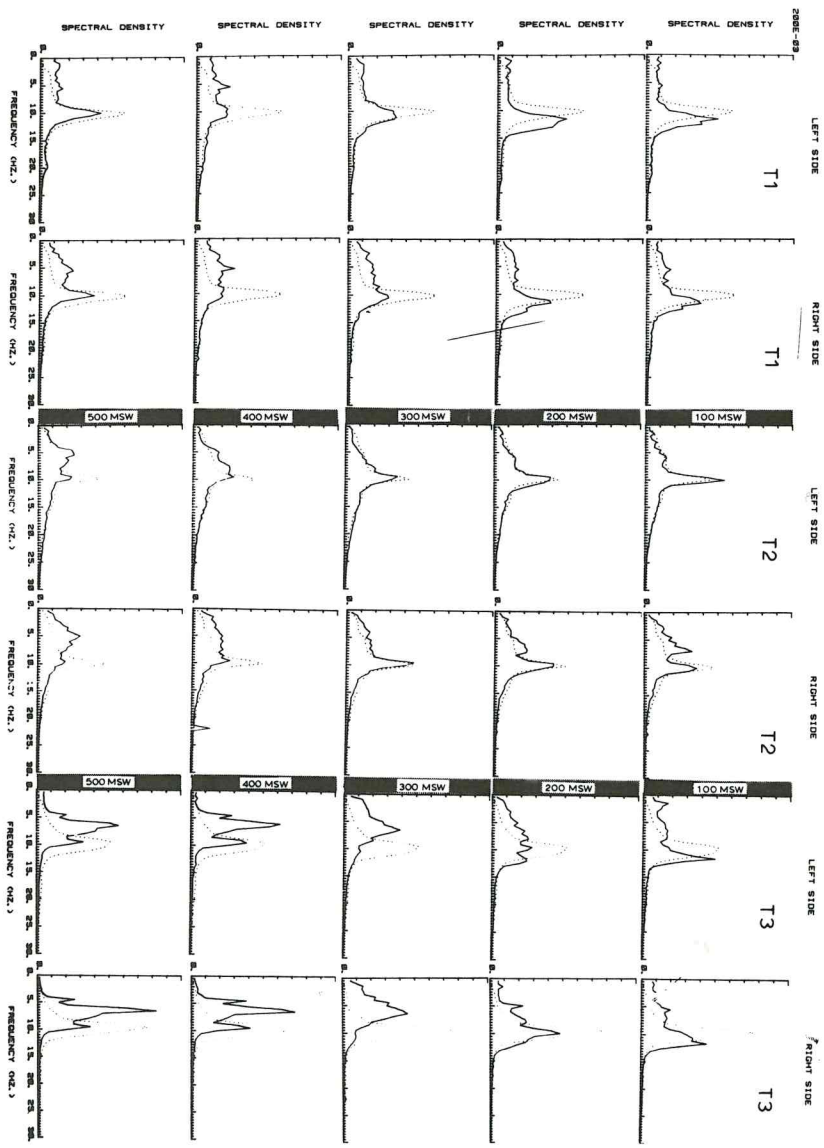

Figure 4. *Power spectrum EEG during trimix compression. Stippled curve is the mean predive power spectrum EEG.*

rier Transform (FFT) analysis of the EEG records revealed a marked increase in the power of slow waves (2-7 Hz) during compression to 500 msw for the trimix group. There was, however, considerable variance between subjects, and hemispheric differences were also found within subjects (see Figure 4). The FFT analysis also revealed a marked increase of power for slow waves during the compression on heliox, and as for the trimix group, there was considerable variance as well as hemispheric differences between the subjects (see Figure 5).

It was not possible to differentiate between the two compressions based upon the FFT-EEGs. In fact some subjects in the trimix group had more changes in the FFT-EEGs than subjects in the heliox group.

As found previously trimix inhibited the tremor, while the heliox group had a marked increase from predive control levels. However, one diver in the heliox group had no tremor change despite several other HPNS symptoms and signs.

The trimix group was markedly impaired throughout the compression on several cognitive functions especially for reasoning and long-term memory. N=2 indicate that diver no. 3 was not fit to perform the tests at those depths (see Figure 6). The heliox group was also impaired on several cognitive functions throughout the compression (see Figure 7), but not as much as the trimix group. Furthermore, there was a tendency to recovery of the cognitive functions when the heliox group reached saturation depth, while the trimix group was markedly impaired throughout the whole saturation period on the trimix up to day 4.

However, the symptoms of narcosis and HPNS were not the only problem throughout this dive. From day 4 to 5 the trimix group underwent a change of gas to heliox. Six hours after the gas change was completed severe myoclonic jerks and hallucinations occurred for two of the subjects. The hallucinations were both auditory and visual, and the symptoms lasted throughout the whole night. The symptomatology and the latency in occurrence indicated that a withdrawal syndrome had occurred. The following in-water performance trials showed that the divers who previously had been on trimix and had stayed four days longer than the heliox group under pressure were more impaired on several of the motor and cognitive in-water tests.

Based on these findings and other data from these dives not presented here, the following conclusions could be made:

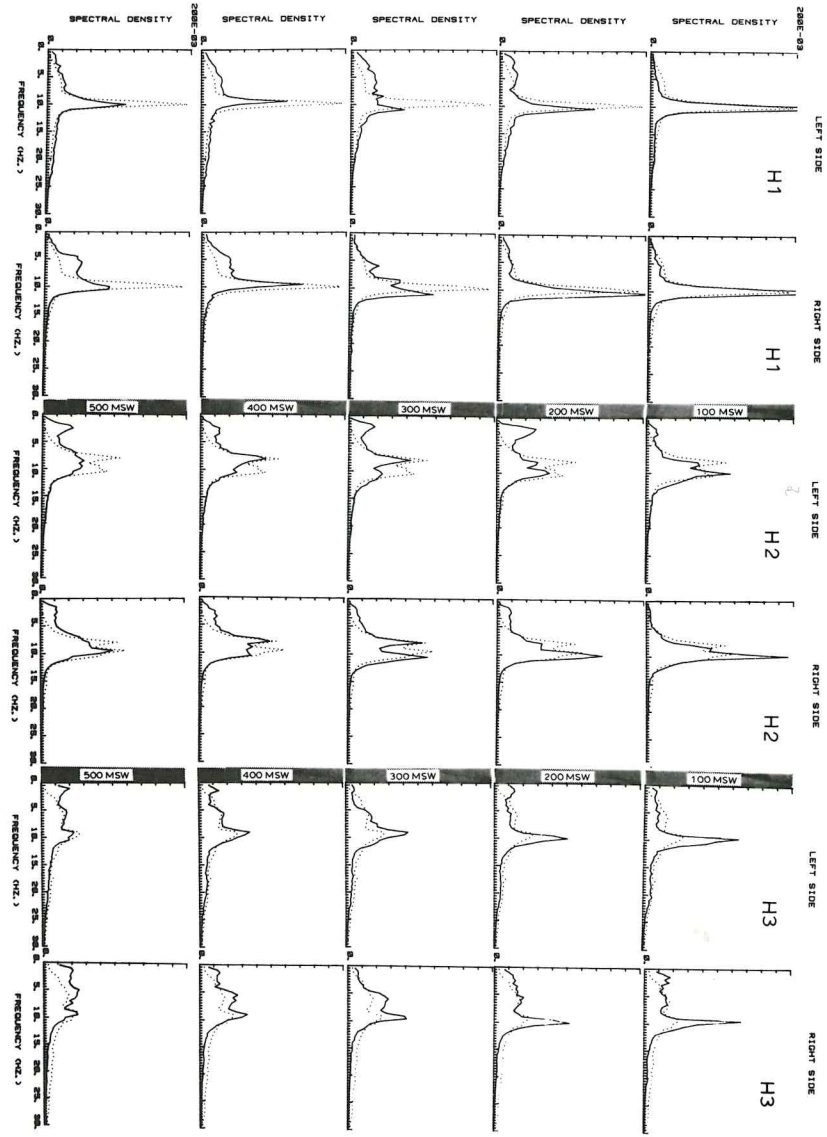

Figure 5. *Power spectrum EEG during heliox compression. Stippled curve is the mean predive power spectrum EEG.*

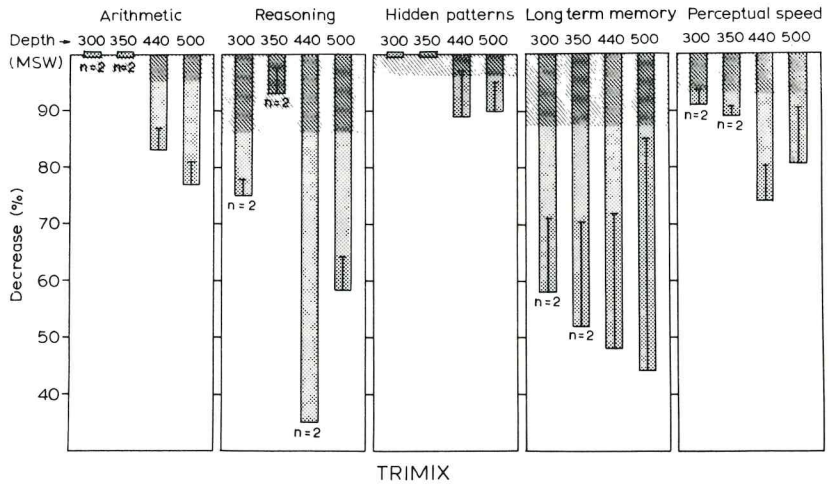

Figure 6. *Cognitive performance at stable depths during trimix compression. Line included on bars represents 1 sd of the mean; crosshatched area represents 1 sd of control values.*

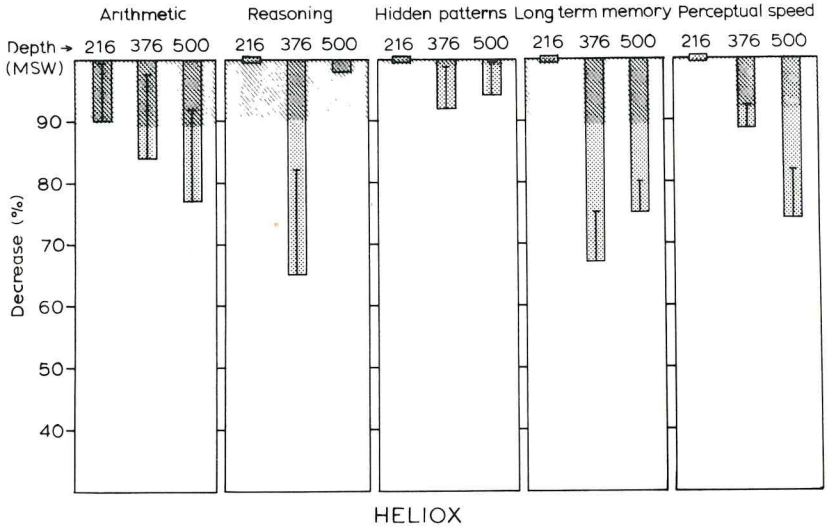

Figure 7. *Cognitive performance at stable depths depths during heliox compression. Line included on bars represents 1 sd of the mean; crosshatched area represents 1 sd of control values.*

The Compression: There were marked HPNS effects during compression for both groups. Only tremor was inhibited by the nitrogen. In addition, the trimix group was impaired due to nitrogen narcosis.

The Saturation: There was a recovery in the FFT-EEG's in both groups. There was some recovery in postural tremor and hand grip strength in the heliox group. The heliox group performed at predive level on cognitive functions whereas the trimix group was markedly impaired on the 3rd day of saturation.

The Gas-change: There were minor FFT-EEG changes during the gas-change. There was recovery on serveral cognitive functions, but there was a marked increase in postural tremor. In the 12 hour period after the completion of the gas-change severe withdrawal symptoms occurred in 2 of the subjects.

The Heliox-saturation (N=6): There was minor recovery in the FFT-EEG during saturation and the postural tremor was sustained throughout the whole saturation period. For cognitive functions the trimix group took a longer time to recover than the heliox group on heliox saturation.

It is not possible to compress divers to 350 msw on such profiles and have generally fit subjects who can enter the water with regards to safety and effectiveness of a planned task. In 1983 two simulated heliox dives to 350 msw showed that this was possible given a good compression profile with intermediate stops before reaching the HPNS-area.

In the first dive six subjects were compressed on heliox with intermediate stops at 216 msw for 4 hours and at 296 msw for 12 hours. In total they took 24 hours from leaving surface to reaching saturation depth.

As for previous heliox compressions there was an increase in postural tremor. Comparing the mean increase with Deep Ex I and Deep Ex II results, however, the tremor did not effect motor performance in this study. Furthermore, the cognitive functions were more or less within the normal variation range, except for long term memory, and comparing the performance with the results from the Deep Ex dives there was a marked recovery.

Two divers had normal FFT-EEG's throughout the whole dive. In Figure 8 an example from the compression is shown. The first plot in front is the mean predive control value averaged from 5 control meas-

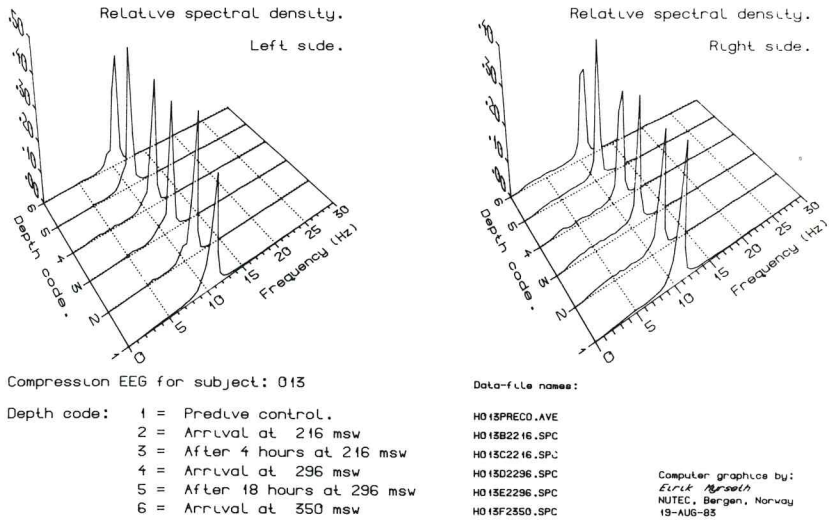

Figure 8. *Power spectrum EEG for one subject during the compression to 350 msw. The EEG in front is the mean predive control EEG.*

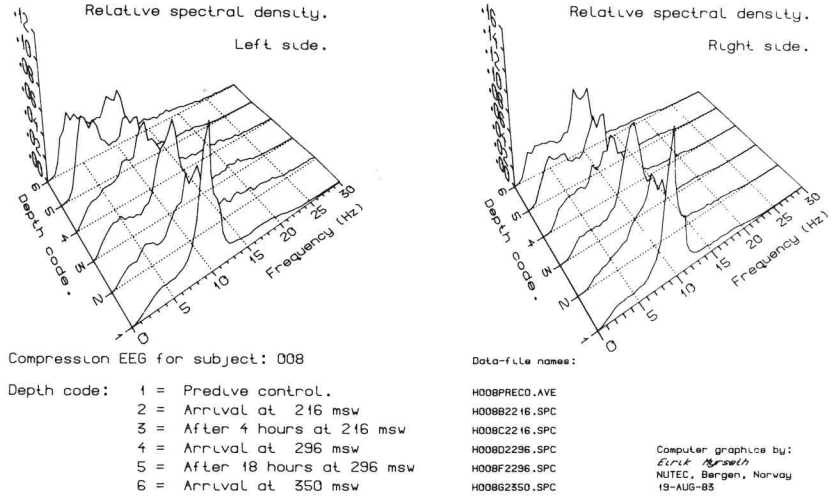

Figure 9. *Power spectrum EEG for one subject during the compression to 350 msw. The EEG in front is the mean predive control EEG.*

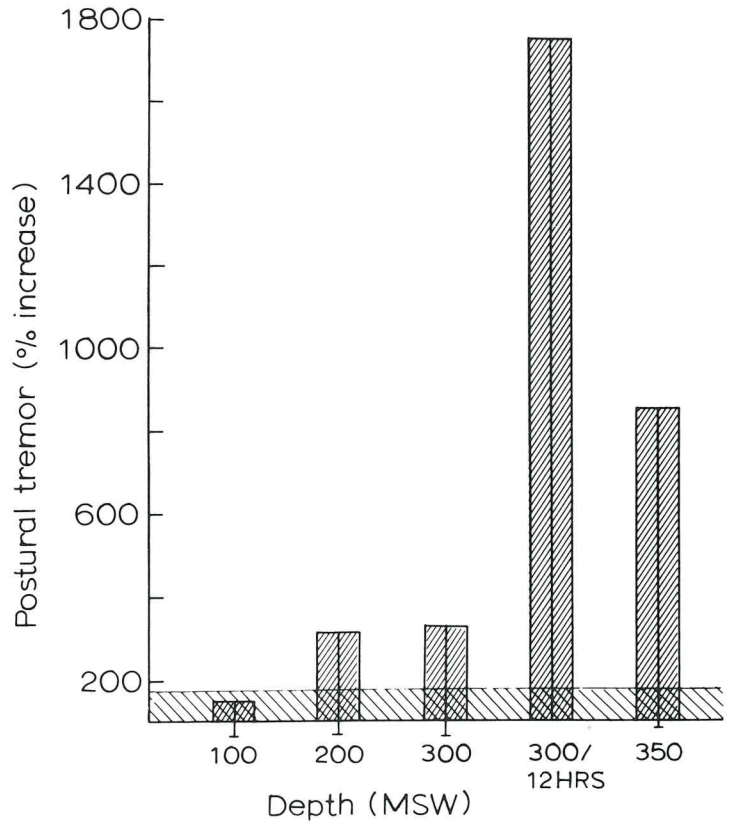

Figure 10. *Mean percent increase in postural tremor at stable intermediate depths during the compression to 350 msw.*

urements. The z-axis indicates the depth range. In this subjects had minor FFT-EEG changes and who had more marked changes. In Figure 9 a subject with marked FFT-EEG changes is shown (see Figure 9). There were both alpha band inhibition and theta increase, but this study confirmed the previous ones: there will be both individual differences and hemispheric differences. As for performance the symptoms were not very severe. Despite nausea and dizziness on reaching 350 msw, the major problem throughout the compression was the heat. The following conclusion could be made from this dive: compared to previous deep dives at NUTEC, Deep Ex I and Deep Ex II, the divers were here minimally impaired by HPNS. Although signs and symptoms of HPNS were evident, all six divers felt fit to start the "bell run" on the second day 24 hours after leaving the surface.

However, a hypothesis was proposed that the nausea and dizziness which all six divers experienced in the earlier dive could be reduced or eliminated if there were intermediate stops between 100 and 200 msw. In that case the divers could in some way adapt to the pressure and therefore lessen the HPNS symptoms which start to occur between 200 and 250 msw. This was confirmed in a second dive in 1983 where the divers stayed at 100 msw and 200 msw for 2 hours, and as for the previous dive at 300 msw for 12 hours.

As for the previous dives there was an increase in tremor (see Figure 10), but as for the previous 350 msw dive performance was mildly impaired, and only an impaired long-term memory was found (see Figure 11).

In this dive 2 subjects had a more or less normal FFT-EEG throughout the whole dive, two had some changes and two had major chan-

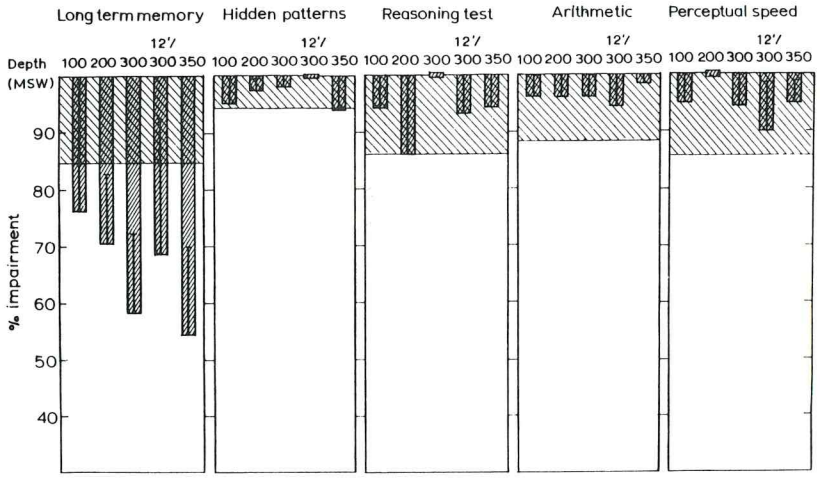

Figure 11. *Cognitive performance at stable depths during heliox compression. Line included on bars represents 1 SD of the mean; crosshatched area represents 1 SD of control values.*

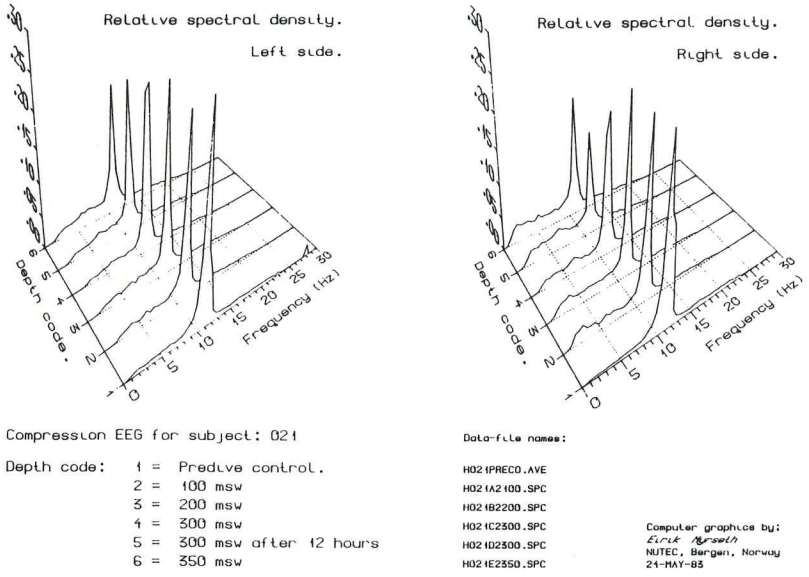

Figure 12. *Power spectrum EEG for one subject during the compression to 350 msw. The EEG is front is the mean predive control EEG.*

ges. A subject with a normal FFT-EEG throughout the compression is presented in Figure 12 and a subject with major changes is presented in Figure 13.

Symptomwise the only problem rated as such were wrist and hip pain in one of the divers, and back-pain in another. For one diver this symptom always occurred during deep diving. Compared to the previous dive no dizziness and nausea occurred from 200 msw and deeper, and the major problem at that stage of the dive was the hyperthermia. The conclusion was therefore: there were minor problems during the compression to 350 msw. The intermediate stops at 100 and 200 msw seemed to lessen the HPNS symptoms which occured between 200 and 300 msw in the previous studies.

Based on these 24 mandives to 300 msw or deeper three subjects were severely impaired during the Deep Ex dives both with regard to symptoms and far from the test results. No divers were free from CNS changes. In the two 350 msw dives performed in 1983, however, none

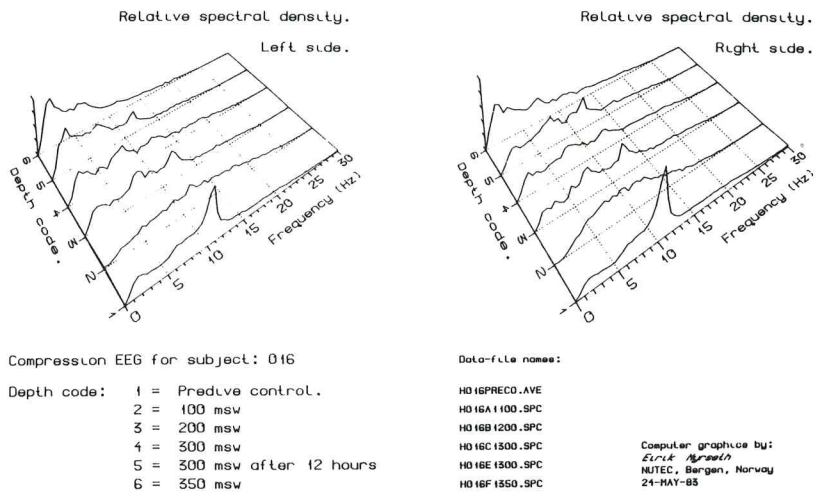

Figure 13. *Power spectrum EEG for one subject during the compression to 350 msw. The EEG is front is the mean predive control EEG.*

were severely impaired, and three had no or minor signs of HPNS. All reported some symptoms during the dive, especially problems with the high temperature during the compressions. Furthermore, it must be concluded that the staged compression profile with intermediate stops at 100 and 200 msw was the best concerning problems with the HPNS.

Pre- and postdive neuropsychological examinations

An extensive battery of tests are included in the clinical neuropsychological examinations, and are mainly based on the Halstead battery, in addition to tests from Kløve/Mathews, Kløve/Reitan and Reitan. There are 5 main examinations: several cognitive tests, (here named adaptive abilities), different steadiness/tremor tests, peripheral motor tempo and strength, imperception tests, and different memory tests. In addition autonomic reactivity, FFT-EEG and a clinical interview are included. The high validity of this battery in differentiating normal subjects and psychoneurotic patients from patients with brain pathology has been established (3) even when psychoneurotic patients show neurologic-like complaints (4). This battery shows (3,5) a high degree

of validity in distinguishing between brain lesions of different location and pathology, as well as between variables such as duration of toxic agents, e.g. alcohol (6). In a report of central nervous dysfunctions after severe near miss accidents in diving this method has also been used (7).

Post Deep Ex I, 4 divers had a reduced motor tempo, either on one or both sides. One had some memory problems and two had reduced scores on testing adaptive abilities. These results normalized within one year. Symptomwise, one diver had a depression which lasted for about half a year.

Post Deep Ex II to 500 msw, all six subjects had some reduced performance on several tests, mainly on motor tempo and strength. Two had reduced scores on adaptive abilities, and three had a more marked tremor. Symptomwise one diver reported depression and inactiveness for 2 months. For both divers who had experienced the hallucinations, it took some time to work through this traumatic episode. One diver who was relatively symptomfree during the dive, except for marked tremor increase, had both objectively and subjectively an increased tremor half a year later. This tremor, however, vanished.

For the first 350 msw dive at NUTEC in 1983 no divers reported specific or general postdive problems. However, reduced motor tempo and strength were found for the majority of divers.

Due to repeated dive activity for the divers involved in the second 350 msw dive at NUTEC, the pre- and postdive examinations for the onshore trial dive and fjord dive are presented together in Table 1. As found for the previous 350 msw dive there were relatively few changes from pre to postdive examinations, and mainly on motor tempo and tremor. However, comparing this result to the pre and postdive examinations performed in connection with the fjord dive the following was found:

Six of the 9 divers had an increased tremor, five had reduced hand grip strength and motor tempo. Four had a reduced memory score and three lowered scores on adaptive abilities. While post trial dive examinations were performed within one week, the post fjord dive examinations were performed mainly one month later due to fatigue among the divers. As for the Deep Ex dives it is important to re-examine these divers, perhaps earlier than one year from now.

Symptomwise the same tendency was found: relatively few problems

	POST OTD-DIVE								POST FIORD-DIVE								
	9	10	11	12	13	14	16	17	9	10	11	12	13	16	17	7	8
Adaptive Abilities											X		X			X	
Lateral Dominance									X								
Coordination Test																X	
Vertical Groove Stead.										X							X
Horizontal Groove Stead.	X								X				X				
Postural Tremor				X			X			X	X	X	X		X	X	
Resting Tremor			X														
Unilateral Imperception																	
Bilateral Imperception																	
Visual Imperception																	
Finger Agnosia																	
Tactile Forms																	
Grooved Pegboard														X			
Wechsler Memory Scale							X			X							
Forched Choice Memory																	X
FFT EEG																	
Autonomic Reactivity					X		X							X		X	X
Spatial Memory							X				X		X	X			
Finger Tapping Speed	X				X						X		X				
Foot Tapping Speed									X			X	X	X		X	
Hand Grip Strength	X								X		X		X	X			X
							welder	welder						welder	welder		

Table 1. *Divers with marked decrement (> 10% changes) on neuropsychological tests from pre to postdive examinations. Postdive onshore trial dive results function as predive fiord five results. (For divers no. 7 and 8 the first column is pre and postdive examinations performed in conjunction with a trial dive on heliox to 300 msw outside NUTEC).*

after the onshore dive, while 7 divers reported problems after fjord dive. The two welders had few problems, but in particular the four divers who were part of the first dive team reported several symptoms (see Table 2).

DIVER	POST COTD DIVE	POST FIORD DIVE
1	Short temperedness, "Lot of problems with memory"	Memory problems
2		Very exhausted, but no other problems
3		Tired, memory problems, 10 days with dizziness, headache and nausea, "forget trivial things", "hyperventilated for 3 weeks"
4		Very exhausted, short tempered, 6 weeks later felt OK.
5	Tired, short tempered	
6		
7		Problems initiating anything, exhausted
8		Tired
9		
10		Tired, worse memory. Difficult to find words, general memory impairment.

Table 2. *Subjective symptoms evaluated by a clinical interview one month after the onshore trial dive and the fiord dive.*

Based on these dive studies the following conclusions can be made concerning CNS signs and symptoms postdive:

1. For all deep dives at NUTEC there have been some postdive neuropsychological changes from predive control levels. These values have normalized one year later (Deep Ex I and Deep Ex II).
2. There has been a relatively high incidence of subjective problems postdive, mainly depression, inactiveness and sustained attention problems. In some cases these problems have lasted up to six months.
3. There are additional signs and symptoms comparing the fjord dive with the onshore trial dive. This can be an effect of a) repeated deep dive activity, and or b) the differences in physical and/or psychological strain/stress between the two dives.

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QUESTIONS AFTER DR VÆRNES'S PAPER

Dr Ørnhagen: It seems as if the rate of pressure change is of importance when it comes to the symptoms and, I wonder, do you know of any other environmental stress where there is an impact of rate of change, for example, cooling? Is there a possibility of an adaptation if you have a mild hypothermia that comes on very slowly, compared to a fast cooling? Or other things like exposure to toxic substances, and so on?

Dr Værnes: What we think has a significant effect for the compression is hyperthermia because this is the main problem during the compression between 100 and 300 metres. We think that the hyperthermia facilitates the early HPNS signs and symptoms. In many ways the dizziness and nausea can be a hyperthermia effect first and then we have the so called HPNS symptoms. So we think, especially for the Comex profile where we stopped at 100 and 200 metres, we lessened these problems and that the divers could adapt to the pressure and the temperature.

Dr Ørnhagen: Do you have any measurements of deep body temperature or brain temperature during the compression phase? I know it is difficult to measure but it should be important.

Dr Værnes: We had an extensive battery of temperature measurements but they were mainly performed in the in-water performance studies. Our main concern is the subjective stress of hyperthermia and therefore, when we start further studies, we shall include cortical measurements which are very valid indicators of hyperthermic stress and, of course, the "hyperthermia status questionnaire" because we have found out that this is a very significant thing to measure.

Dr Cox: Can you give us some idea about the severity of the depression in those people who were depressed. Was this a depression which was revealed as the result of direct questioning or was it, in fact, spontaneously volunteered? Did they require treatment, was it observed and corroborated by their close relatives, etc.?

Dr Værnes: They came with it themselves, especially for the Deep-Ex II dive. The subject had severe problems for several months and this was both a combination of the traumatic episode during the gas change and the subject's problems post-dive. But, as you know divers must be tough and not talk of such things, so we had, in fact, great difficulty in getting them to come out with it. I think there has been some maturation between the deep dive subjects and ourselves so they can more easily talk about those things than at that stage.

Dr Cox: Was it severe enough to require treatment?

Dr. Værnes: I don't think I can answer that. From my point of view as a psychologist it was severe enough. He would not accept treatment.

Dr Baddeley: I was intrigued by the pattern of performance decrements that you described. Whereas the overall amount of decrement across conditions was rather similar to the decrements that we observed, I was particularly interested in your memory test being the most sensitive since we found it to be very insensitive. Indeed, Carter, I think even found improved long term memory at pressure. Do you have any comments on that, perhaps you could say a little about the test?

Dr Værnes: Yes, for the Seaway dive we had, more or less, a correlation between, for example, the EEG changes and the tremor, symptoms and the cognitive tests. For the performance tests there was no correlation between memory impairment and EEG changes. It was especially so between EEG changes and the AB test from Baddeley and arithmetics. What we concluded was that memory impairment was more or less a broad stress reaction, and not as valid for the HPNS. The same thing was found for the tremor increase. There were very significant correlations between the two subjects with marked EEG changes and tremor increase, while for the minor EEG changes there were no correlations. So there is indeed a correlation between the several parameters, but not a linear one. You have to look at them and see what their relationship is in fact.

Prof Bennett: Again, I was not really prepered for a lot of HPNS discussion. I thought we were talking about post-dive neurological deficits, but if we are going to get into this, I think I will show 4 slides of the Atlantis dives later in the general discussion, if I may. These will show the comparisons between fast compressions and slow compressions and 5 % nitrogen and 10 % nitrogen, which will indicate that there is very little EEG change, there is maximum protection against the performance decrement we have been talking about, with some indication of an HPNS tremor. I think that is about the best you can do with 7-day compressions and I am talking here about 650 metres not

500 metres or 300 metres. The French have been very quiet here - I have not heard a word from them, but we do know that they have gone to 610 metres in the water with divers working for 3 to 4 hours and I think they really should say something.

Dr Rostain: Some comments on your presentation Dr Værnes. First, in relation to our own experiment, on the EEG change I would like to say that during the use of the two mixtures, helium-oxygen and helium-nitrogen mixture, we have never seen a difference on the EEG change. But for a 10 % nitrogen mixture sometimes you have an increase in slow waves, but with the use of 5 % nitrogen in an helium-oxygen mixture there is practically no difference in the EEG change. I agree with you that the nitrogen decreases the clinical symptoms, especially the tremor. I agree with you that the use of 10 % nitrogen increased the narcosis and we have said, several years ago, that the use of 5 % nitrogen is better than the use of 10 % nitrogen. The impairment in psychometric performance is due to the 10 % nitrogen. Similarly, the use of fast speeds of compression certainly provokes vertigo, nausea and so on and we said, several years ago also that the use of stages at intermediate depth is better and inhibits the HPNS. These are my comments.

Prof Bennett: I am interested in the depression. Again, I will mention very briefly in my own contribution that many of the astronauts when they came back had various signs and symptoms of depression. I wonder what quantitative measurements you made of this depression. The difficulty is when individuals in big experimental dives come in, they are exposed to a great deal of media and television attention and then this drops off. There is a lot of stress involved. I do not really understand the nature of this mild depression which I am prepared to believe is there in some cases but it seems so mild you cannot even quantify it.

Dr Værnes: I completely agree and especially the point that they were the centre of attention for several weeks pre-dive, during the dive and post-dive and then suddenly back to normal again. So, I think the depression and inactivity, the psychological syndrome you see after a dive, is a very complex one and there is very little correlation between these post-dive problems and problems during the dive. For example, the diver who had severe HPNS problems both during Deep-

Ex I and Deep-Ex II had been 100 % well post-dive, so I think there is a complex psychological thing going on, post-dive.

Prof Bennett: One case that I am going to be talking about was very vivid because it made a lot of publicity. In fact, that individual was the individual at depth who showed no symptoms whatsoever, said he was 95 % throughout the dive and was very fine, but started to complain later on, five weeks after the dive, of some kind of depression.

Dr Værnes: But, again we have these neuropsychological post-dive phenomena and Professor Aarli will talk about the neurological aspects.

III: NEUROLOGICAL CONSEQUENCES OF DEEP DIVING. SOME CASE STUDIES.

J. AArli,
Department of Neurology, University of Bergen, Norway.

In conjunction with the two onshore heliox dives in 1983 and the open sea dive a comprehensive neurological examination was performed.

Twenty-three divers were examined by EEG and neurological examination before and after the dives. Twelve had participated in two 350 msw chamber dives, six in each dive. Thirteen divers were examined before and after a 300 metres open-sea dive. Five of these divers were also included in the chamber dive studies, and two welders had participated in a welding qualification dive to 300 msw elsewhere.

One diver (A1) had a weak right abdominal reflex, a right hemihypaesthesia/hypalgesia, and EEG showed a preponderance of theta-activity over the left temporoparietal region at post-dive examination. The findings before the dive were normal. He was re-examined one month later, and the focal findings had disappeared.

This diver had complained of an unusual irritability, sleep disturbances, unusual clumsiness and tremor as the main HPNS symptoms during compression to 350 msw. In fact, out of this group of six divers, he was the one who experienced most tremor during compression. FFT-EEG recording during compression showed an impairment of alpha-rhythm and bilateral increase of 3-7 Hz activity with a maximum on the left side. This asymmetry persisted for some time after the dive.

Another diver, (B2), who also had a normal EEG and neurological status before the dive and minimum HPNS symptoms during compression, complained after 12 hours at 350 msw of acute vertigo and blurred vision. His FFT-EEG remained normal during compression, but at dive-day five, during saturation, the alpha-rhythme had disappeared and there was pronounced bilateral 2-7 Hz activity without any asymmetry.

When examined after the dive, the left plantar reflex was abolished,

the left abdominal reflex was weak in all three segments and there was an anisocoria. The tentative dignosis was a transitory brain stem lesion which must have occurred during the saturation dive.

Two divers showed focal disturbances after the open-sea dive. One of these two divers (01) had suffered CNS bends in 1976, with numbness in both legs. However, the 1983 neurological pre-dive status was normal. During compression, he complained of headache and intention tremor. He was sleepy and had a reduced concentration and memory. FFT-EEG was not performed during the dive. At the post-dive neurological examination, the left plantar reflex was abolished and the left abdominal reflex weak. The most plausible hypothesis is that an old lesion after the CNS bends in 1976 was "unmasked" during the dive.

The second diver (02) who had focal neurological findings after the open-sea dive, had had a minor head injury in childhood. The pre-dive status including EEG, was normal. During compression he complained of tremor and somnolence. At the post-dive status, the right plantar reflexes were abolished, but the status was otherwise normal. The tentative diagnosis was "unmasking" of "silent", pre-existing brain lesion, possibly related to earlier head injury.

Focal CNS dysfunction in divers has generally been explained as an effect of decompression, mainly due to gas emboli. Such focal lesions have been described in the spinal cord, but may also be seen in the cerebral hemispheres. In fact, cerebral disturbances seem to be more common than was previously appreciated.

However, in the two divers who were investigated also during the various phases of the dive, the focal disturbances seem to occur during compression and the early part of the saturation phase, respectively. None of the divers seemed to experience any subjective or objective neurological disturbances during compression and FFT-EEG markedly pathological with the maximum of slow activity over the left cerebral hemisphere, corresponding to the post-dive clinical findings from the left hemisphere.

In another diver, the HPNS symptoms and FFT-EEG findings were minimal during compression, but occurred during the first part of the saturation phase (delayed effect), and thus seem to represent a consequence of functional disturbances initiated during compression.

These focal findings may represent a direct effect of compression/saturation upon the brain. Howevr, we find it more reasonable to assume that they depict an unmasking or activation of "silent", subclinical lesions. The transitory nature of the clinical findings add further support to this hypothesis. The similar situation is met with a multiple sclerosis, where it is a well-known phenomenon that an increase in body temperature may lead to a transitory worsening of existing symptoms and may also provoke new ones (subclinical lesions). The present investigations have not revealed permanent CNS damage after deep diving, but clearly show that both subjective and objective neurological disturbances may persist for a long period after dives to 300 and 350 msw.

QUESTIONS AFTER PROF AARLI'S PAPER

Prof Elliott: Professor Aarli, I would like to ask a question of fact for elucidation. Cases A1 and B2 you said were transitory. But cases O1 and O2 were more recent: what is their current status and what is your follow-up?

Prof Aarli: No data to say anything definite because I have not seen them since this information was collected.

Dr James: I understand that some of these divers, in fact, had arterial bubbles detected. Could you comment on whether there was any relationship between some of these findings and the arterial bubbles.

Prof Aarli: I have no information on this.

Dr Tønjum: No, these divers are from the recent dives. The divers from Deep-Ex I are not included.

Prof Bennett: You made comments on 4 subjects here who were certainly showing a mild neurological deficit and you have made some case for an association between those changes and the EEG changes. However, you mentioned 23 divers and I would like to know if, in those other divers, you saw similar EEG changes without any kind of neurological deficit resulting. In other words, are you making a case which has no statistical validity at the present time, based on 4 subjects out of 23?

Prof Aarli: We have no pathological post-dive EEG from the rest of the 23 divers.

Prof Bennett: But you mentioned "pathological" which I find also difficult in the diving situation. There is a change which a neurologist would look at because there is theta activity and some is pathological, but it may be a physiological change without pathology.

Prof Aarli: Yes, but I think you will agree with me that when you have two persons under exactly the same conditions, and one of them has minimal symptoms and a normal EEG, while the other develops EEG changes which we, in clinical neurology, would regard as definitly pathological, that one would be very surprised if this is only physiological.

Prof Bennett: Here is the problem of a clinician coming in and looking at material, blind to the history of HPNS. We know that some individuals will show very little, if any, EEG change at all, whereas others will show the maximum. In the early French dive, we have a 5000 %

increase in theta activity. As far as I know, there was never any damage in those individuals. Other individuals showed no change at all. It is this biological variability that you see when exposing the human body to physiological stresses.

Prof Aarli: I would be surprised if this is an all-or- none phenomenon. The more you increase the pressure, the sooner an experimental animal will die during the exposure, will it not?

Prof Bennett: Yes, but you are talking of pressures far beyond what we are talking about at the moment. Animals have been to 8000 feet (3000 metres), and have returned to the surface without problems.

Dr Cox: Could you clarify something about the third diver? You were suggesting that this diver, who had a focal disturbance in his right hemisphere, may have had an activated old lesion, related to a bend in 1976. The "bend" in 1976 was, in fact, numbness in both legs which almost certainly was a spinal phenonmenon, whereas you are suggesting it was a right hemisphere phenomenon which was reactivated. Is that so?

Prof Aarli: I think that spinal bends are often accompanied also by brain disturbances.

Dr Rostain: I think that we must be careful about the relationship between the EEG change and some eventual lesions in the brain stem. We cannot say actually in our present state of knowledge that a diver who had EEG changes has automatically had CNS lesions. I think that this is not proved and we must take account of this; do you agree?

Prof Aarli: Yes, I totally agree with you. I do not think I have said anywhere an "organic lesion" or a "microscopic lesion". I think I would use the word "dysfunction". My main point is that these divers had a normal neurological status before the dive and, when I re-examined them after the dive, there were findings which had not been present previously.

Dr Giran: I think that one of the 4 divers in whom you found an abnormal observation after diving was allowed to go back diving with a remaining lesion.

Prof Aarli: Yes, he was.

Dr Giran: Can you explain why, if there is no risk at all in terms of commercial operation, in terms of insurance.

Prof Aarli: Well, I am not responsible for the selection of divers - I am just examining divers before and after these dives.

Prof McCallum: Perhaps somebody else could explain.

Prof Donald: Not only explain, Sir. I think there is a great deal of difference of opinion. In practice, if a man has had a definite, even small transient CNS event, some people will discount them from diving ever again, or at least to any important depths. Other will say that if the man is normal clinically with a normal EEG, it is unfair and wrong to stop him diving and he should be allowed to go back. I would be interested in your views.

De Peterson: We have heard that this man made another dive: what was the finding after that second dive, which was also a deep dive?

Prof Aarli: He was normal.

Dr Ørnhagen: Professor Aarli, could you please comment on the fact that the abdominal reflex seemed to be hit quite often here. As I remember, it was three out of four cases: how do you explain this?

Prof Aarli: Of course, the abdominal reflexes are about the most sensitive that we have and will often disappear before the plantar reflexes. They are very sensitive indicators of a lesion in the pyramidal system.

Prof Donald: Are not the cremasterics more sensitive?

Prof Aarli: In many ways you can regard the cremasteric reflex as a fourth segment of an abdominal reflex, so they will not add more information. I use them mainly when there is a question of a spinal lesion and I would like to identify the segment from a clinical point of view.

Prof Bennett: I wonder about the accuracy again in terms of that diagnostic measurement. In one subject I will be mentioning later, one neurologist (not in Duke, but elsewhere), implied that there was a decrease in the abdominal reflex on one side. We then had him looked at by a number of neurologists and none of the other neurologists could, in fact, defend that statement.

Prof Aarli: So much the worse for the American neurologists.

Dr Peterson: In 1975 a dive was done at the University of Pennsylvania to 1600 feet and I believe it is one of the very few dives in which full clinical EEGs were recorded over quite an extensive time period. The findings there showed that the periods of abnormality were transient. There was a lot of HPNS on this dive and a lot of slow wave activity but they were only at various times. This I think indicates that it might be a bit hazardous to interpret findings from spectralanalysis of

the EEGs that are taken at one time during the day and not over some extended period. You might find that a little bit later the man was normal where before he was not.

Prof Aarli: I think it is important again to stress that our point of origin was the clinical disturbance and then we would go back to data that was recorded during the dive.

Dr Török: Two points, Mr Chairman. I would like to reinforce Professor Aarli's point about the well known situation in which the bulk of our neurological evidence in a decompression situation indicates a spinal lesion. However, one or two minor signs are found which would take it to cerebral level. Of course we assume a single site for the lesion. In a decompression sickness case, the assumption of a single lesion is, I feel, sometimes not justified. There might be more than one lesion.

The second point is a question. The second subject with the reduced left plantar, the lost or reduced left abdominal reflex and the anisocoria who also had dizziness, is he the same patient whom Dr Molvær described as having a non-nystagmographically-proven type of dizziness, or is he a different man?

Prof Aarli: I think he is different.

Dr Molvær: I have also examined, with ENG, the 23 divers Professor Aarli comments on. In one of those (it is not the same person as Professor Aarli found neurological changes in) but in one of the others, I found a change in the electronystagmographic reflex on one side. He had a normal ENG after the chamber dive. In the post-dive control after the sea dive, he had a reduced reaction in the left side which was still present one month later. I do not know how much we can elaborate on this for two reasons. Firstly, we are talking about central nervous mainly and this is, as far as I have understood, a peripheral lesion in the vestibular apparatus. Secondly, it was very marginal when I first detected it. When we measure these physiological parameters we have limits, internationally, a discrepancy of 15 % between the two sites is recognised to be clinically significant. In the first examination he had only a 15.4 % discrepancy, so it was marginal. One month later it was more marked and I found a discrepancy of 21.3 %.

General Discussion on the Norwegian Experience

Prof Bennett began the main discussion period by presenting 4 slides showing the results of different tests done during the Atlantis deep dive series. These were intended to demonstrate the differing effects of rate of compression and nitrogen percentage on various aspects of diver ability.

The 5 % nitrogen mixture proved better in arithmetic and performance effects, but it was evident from the Ball-bearing Test that there was some breakthrough of tremor at greater depths, around 650 msw. This was, however, not felt to be a problem by the divers. The Hand-tool Test reveals the same effect, but again it was not felt to be debilitating. Looking at the EEG theta activity, it is most marked in fast compression with 5 % nitrogen and remarkably little change is seen if compression is slow with the nitrogen percentage at 5 %.

Dr James was concerned about the detection of arterial bubbles and asked if there was any relationship between reported symptomatology and bubbles: could they act synergistically? *Dr. Tønjum* replied that the diver with the recorded arterial bubbles was not screened using a broad clinical neurological examination before the dive, but Professor Aarli had investigated the diver afterwards. *Prof Aarli* reported that the diver had never had a recorded incident of depression although he was known to have a cyclic temperament. The present depression in the diver could have many complex causes including temperament and loss of status on the dive. He may have had a right temporal lobe lesion during the decompression, but Prof Aarli was unable to be more definite and could not comment further on possible pathology.

Prof Bennet was very concerned about the idea of arterial bubbles in relation to deep saturation diving where the rates of decompression are far slower than those used in other forms of diving and found it hard to believe that arterial bubbles could occur during such saturation decompressions when they have been reported so infrequently in air diving. *Dr Peterson* revealed that the arterial bubbles were detected during upward excursion procedures, not during the saturation decompression. The excursions were, however, within the US Navy procedures for such manoeuvres. *Dr Brubakk* reported that he detected an intense ultrasonic reflection during excursions from 300 to 250 metres at 10 metres per minute, and during the decompression from 500-400 me-

tres at 2 metres per hour, he detected similar reflections in one diver. This observation was unique but he hoped someone would do some experiments and either correct him or not. The focal lesions reported here suggested that this experiment should be done.

Dr James commented that the reported absence of bubbles in compressed air diving could be because no one is looking for them. *Dr Peterson* remarked that Dr Brubakk had the advantage of studying the bubbles at great depths, where they persist for longer times than normal. He has looked for bubbles following maximal ascent at shallower depths and found relatively few, so it could be that the bubbles at shallow depths are so brief as to be undetectable.

Prof Bennett commented that he raised the subject with the intention of outlining the risks of air diving and the associated neurological deficits that result compared with the kind of thing one sees in deep diving. He believed that air diving is more dangerous than deep diving.

Dr Cox changed the topic by asking if any comparable neurological evaluations had been made of people exposed to other environmental changes and in particular, pre- and post anaesthesia. The changes reported may not be specific to divers alone.

Dr Brubakk reported a study of two groups of old people having hip replacements under either general or epidural anaesthesia. The conclusion was that 25 % of the people who had general anaesthesia had some degree of mental retardation although it is a difficult observation to interpret. Returning to an earlier topic, he remarked that the diver with the focal symptoms was the same diver who had arterial bubbles recorded during the ascent.

Dr Ørnhagen asked if it might not be useful to invite the diver who was suffering from depression to visit the meeting and question him directly as very few of the members had dived deeper than 150 metres themselves. This was felt, however, to be ill advised due to the possible subjective embellishment of his experiences and it was agreed that the meeting be restricted to quantitative data on these marginal effects.

Dr Baddeley remarked on the difficulty in obtaining quantitative data and suggested that this could be due to the techniques available being rather blunt. For example, current techniques allow us to pick up effects on only a very small proportion of closed head injury cases, but it should be possible to pick up cumulative effects as is the case in

boxers who are regularly knocked out. He believed that if we could use more subtle tests we could pick up smaller changes, but at the present we do not know how good our techniques actually are.

Dr James pointed out that to obtain functional evidence of damage, one must encroach on the functional reserve which in some areas can be extensive. Therefore although the dysfuntion may be below the level detection it could nonetheless be permanent.

Dr Rostain relayed the subjective assessment of Dr Lamy, who has dived to 150-300 metres, that the first dive is very stressful and the recovery is hard, but later dives are easier with quicker recovery.

Dr Peterson inquired whether personality studies had been carried out on the divers before the dive. *Dr Værnes* replied that standard tests such as the Minnesota Inventory and occasionally the stress tolerance tests of the Norwegian Navy were used. No abnormal values were found. However, he did report that in a study of near-miss diving accidents involving uncontrolled ascent or hypoxia, personality changes are detected. He remarked that he was surprised to note that the symptoms detected on the deep dives, while not so severe, were similar. A study carried out in the USA by Leven and Paterson detected a pathological personality in a group of diving accident victims. *Prof Bennett* reminded the meeting that these changes are probably decompression sickness related not deep dive related.

Prof Bennett commented that sleep loss associated with deep dives might be a significant factor in producing the kind of residual effects which recover once the normal sleep-wake cycle is restored. *Prof Aarli* agreed but reminded the meeting that this would not explain the asymmetry of the reflexes detected after the dive. *Dr Værnes* reported that one of the divers who had severe HPNS problems on Deep-Ex 1 did not sleep for several days but once a good night sleep was obtained, he recovered dramatically and he had a normal EEG thereafter.

Dr Baddeley reported that he had correlated quality and amount of sleep with performance and found that sleep loss is not correlated to performance. Caging is another variable which has no observable effect. He agreed that they are both variables which contribute to performance, but not to a major degree. The quantification of sleep loss is, he thought, very difficult and the above study utilized a subjective sleep questionnaire.

Dr Rostain agreed that there is no relation between sleep loss and

performance because they have recorded a decrement on performance before there is a loss of sleep. He wished to stress that the sleep disturbance is characterised by an increase in light sleep, stages 1 and 2 and a decrease in deep sleep, stages 3 and 4. The duration of the sleep is unaltered but the organisation is changed with decreased deep sleep and a difficulty in achieving REM sleep.

Dr Baddeley reported that the subjective comments also indicate that sleep is not good during compression and Dr Rostain suggested that this deep sleep deprivation could produce fatigue and impairment if continued for a long time.

Prof Aarli agreed with Prof Bennett that it would be wrong to exaggerate the findings of focal neurological changes, but we should maintain an open mind. He hoped that they would not influence the diver in the long term, but be presented an analogy to aid understanding. In multiple sclerosis an increase in temperature can produce what appears to be reversible effects associated with a 'silent' lesion which will again be 'silent' when the temperature is lowered. This unmasking effect, he suggested, could be what happens during the compression phase of a dive where a pre-clinical lesion is unmasked. He also postulated that this might develop into a long-lasting disturbance if the diver carried out many deep dives over a short period of time and he believed that this is an area that required research.

Prof Bennett agreed that there seems to be some unmasking of psychological impairment or pre-existing pathology and indicated that this was an ideal reason for diver selection. He suggested that divers should be an elite group, highly trained and selected for deep diving on the basis of their decreased sensitivity to HPNS.

Dr James wished to return to the question of occupational risk and inquired if it was possible to predict which people were going to have their latent problems unmasked. If such problems are reversible over 3-6 months with partial incapacitation, is this acceptable ethically?

Prof Aarli commented that, in his experience, after a dive, the divers get drunk and as this is their most sensitive period they may prolong the time they are unfit for another dive, so his opinion was that the divers should refrain from alcohol for 14 days after a dive.

Prof McCallum remarked that Prof Donald had made some enquiries into the subjective views of divers' wives on the increased sensitivity of their husbands to alcohol. *Prof Donald* comments that quite a

number of wives have reported that their husbands are remarkably sensitive to quite small amounts of alcohol. Although this information was to a certain degree anecdotal, he felt that there was some basis for this report, but remarked that it would be difficult to convince the divers to abstain without very good evidence of damage. *Dr Baddeley* doubted if the increased sensitivity lasted for only 14 days as he has report of continued sensitivity in a diver who has not dived for 2 years.

Prof Donald asked if any deliberate sleep deprivation studies had been done on divers during a dive. *Prof Bennett* commented that on Atlantis IV one of the divers who could not sleep at all and had to be drugged after 5 days to induce sleep, subsequently became hypomanic at 600 metres.

Dr Becker reiterated the need for careful measurements and indicated that techniques are now being developed in the field of closed head injury, which are considerably more sensitive. These tests could be used to detect much smaller degrees of damage which might exist below the currently detectable threshold and we might consequently be able to advise certain individuals not to dive because of the unknown effects of cumulative exposure on such lesions. *Prof McCallum* remarked that the more sensitive the test, the more difficulties arise as you try to interpret them.

Dr Török commented that the unmasking of effects could cover two areas, psychological and neurological. He belived that several events in an individual's life could unmask an existing psychological aberration but the concept of unmasking a previously unknown neurological lesion, he believed to be a much more important area. The functional redundancy of neurological tissue and it's incapacity to regenerate, made it a cause for concern. The pyschological unmasking was, he felt, less of a problem.

Prof Donald inquired if there were any precautions taken to avoid the divers entering the chamber whilst suffering from withdrawal effects of psychotropic drugs or alcohol. *Prof Bennett* remarked that at Duke they try to get their subjects to refrain from drinking heavily or taking any drug for 1 month pre-dive, but this is a voluntary abstinance, so no guarantees can be given. *Dr Rostain* indicated that the sleep disturbances appear generally after 300 metres and not immediately at the beginning of the dive as would be expected for withdrawal effects and also, the sleep quality improves after 6-7 days at saturation.

Dr Becker commented that there was a marked difference between the fjord dive and the chamber dive and asked Dr Værnes if the dives were comparable in rates of compression and decompression, the length of the dive, activities performed etc. *Dr Værnes* indicated that there were several differences. Although the initial compression to 300 metres was the same, the chamber dive was then continued to 350 metres. In both dives, the bell runs lasted 8 hours and the water temperature was 6-8 degrees. The main areas of difference was the work done during the fjord dive which was very hard and the subjects were fatigued. This was the reason for postponing the post-dive neuropsychological tests for 1 month to allow recovery of the subjects, because the complete test can take up to 8 hours. Dr Værnes concluded that the difference in symptoms reported on the two dives was due to the difference in physical and/or psychological stress/strain for the different procedures. He suggested that the 1 year post-dive examination should be augmented by a 6 month examination.

Dr Baddeley asked if the performance differences reported at depth were due to the dive being carried out in the open sea which has been reported to decrease performance more than would be expected from chamber dives. *Dr Værnes* replied that the 8 hour bell runs in the chamber dive consisted of a series of psychomotor tests, finger dexterity and cognitive tests for 5 days, while the fjord dive bell runs consisted of erecting the welding habitat, not performance tests. The only information they obtained was from a status questionnaire that the divers filled out.

Prof McCallum inquired why Dr Værnes wanted to decrease the interval between follow-up tests of the divers and was told that the previous practice had been to test immediately after the dive and then one and three months later. Experience from near-miss cases of hypoxia and uncontrolled decompression was that the symptoms took 4-5 months to recover, hence the required test at 6 months.

Dr Giran reminded the meeting that there were considerable differences between chamber dives and open sea dives, even if the chamber dive had a wet component. The circumstances and the stresses are different and the jobs carried out on these dives are also different. This, he maintained, made the comparison of the two types of dive very difficult and he suggested that the chamber dives merely give an indication of possible effects; they are not a replacement for the real thing.

Dr Værnes agreed, but felt that this was also an argument for carrying out the dives in controlled conditions where severe HPNS may occur. Open sea diving, he felt, may facilitate the symptoms and hence make the dive more hazardous.

Dr Giran then went on to propose that divers should be screened before diving and graded by sensitivity to HPNS. *Dr Værnes* remarked that they would propose that all divers going deeper than 300 metres should go through a controlled chamber dive at NUTEC to allow them to be checked for HPNS sensitivity etc, but agreed that this was impractical financially. *Dr Giran* added that the selection should not be on resistance to the effects of pressure, but should include some professional selection on the ability to do the job.

Dr Baddeley remarked that if one dive session was going to be used to grade the diver, then the HPNS would have to be a reproducible phenomenon and he asked if this was the case. *Dr Værnes* replied that there appeared to be consistency among the 4 divers who did Deep-Ex 1 and Deep-Ex 2, the HPNS sensitive individual being equally sensitive during the two dives. *Prof Bennett* agreed that this was their experience as well during the Atlantis series, but he inquired what the French experience had been as he had heard reports of changed sensitivity in their divers. *Dr Rostain* replied that it was possible to reproduce the symptoms of HPNS at the same depths in the same diver and from these observations, they carried out a study of the susceptibility to HPNS after a fast compression to 180 metres. The results were that the divers could be split into 3 groups by pyschometric and neurophysiological tests carried out immediately after the compression. The three groups were:

1) unaffected at 180 metres
2) some disturbance at 180 metres
3) severe disturbance at 180 metres.

Subsequent studies revealed that the divers who were sensitive at 180 metres were the ones who were sensitive at deeper depths so that he suggested that this could be a method of selecting divers for deeper diving work. However, he stressed the professional capability of the diver is equally important, as it is useless to get a diver to depth without any disturbance if he cannot do the job once he is there.

Dr Török pointed out that susceptibility to HPNS as a single entity is probably a gross simplification as in his experience HPNS consisted

of at least four broad modalities; for example, motor-cerebellar changes, or vestibular change. The reproducibility of HPNS may be associated with one facet or subset of HPNS such as motion sickness and the vestibular system and not with another. The use of the EEG as a marker of sensitivity to HPNS could, therefore, be an equally good method.

Dr Rostain agreed that HPNS is a simplistic term for a complex entity produced by several parts of the nervous system. This is apparent from the effect of nitrogren which can diminish tremor significantly but has no effect on the EEG changes. He, therefore, felt that we should speak in terms of symptoms and not of HPNS as this was too gross, and he suggested that we should go further and grade the symptoms in terms of their importance. Tremor may not be very important to the diver but the EEG may be crucial and at present the relative importance of the symptoms is unclear.

POSSIBILITY OF RESIDUAL EFFECTS FROM SATURATION DIVES DEEPER THAN 300 M.

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Whenever a man tries to extend the limits of his environment he is going to be exposed to considerable psychological and physiological stress. In so doing there is naturally, as indeed there should be, concern for the health and well-being of the individuals concerned. At the same time, however, there is a body of opinion which, motivated by less positive attitudes such as anti-progress, anti-risk and for diverse reasons of envy, political associations, fear of the unknown, etc, unrelated to real objective sympathy for the individuals concerned, would like to stop such progress.

This leads to an unrealistic and highly motivated result that even the smallest incident or symptom, which would probably not even be noticed in the normal life style, is magnified as serious and life threatening.

Such has been the case for both the space program and deep diving. Both programs have had to suffer considerable ill-informed innuendo about post-exposure changes in the astronaut or divers after their exposures to outer space or beyond 1000 feet in ocean or simulated dives. In the space program, political expediency with the Russian competition, eliminated much of the early concern. No such competition has occurred with diving so that the post-diving medical risks have remained open to frequent rumors. This workshop is, therefore, most welcome as a real attempt to present the facts and stop the rumors.

The paper will begin by reviewing some general facts and then direct itself to more specific information regarding the Duke "Atlantis" dives.

Animal Studies

The Russians in 1977 published a 190 page volume in Russian of a study by several investigators of experiments on rats, rabbits and dogs designed to evaluate long term pressure effects by determination of pathological responses of the body. Considerable care was taken with environmental factors such as food supply, excrement removal, antibacterial and microbiological control.

In the major studies, 53 rabbits were exposed in 11 hyperbaric helium-oxygen experiments involving living in a pressure of 35 kg/cm^2 from 12 hours to 35 days. After stepwise decompression, the animals were killed and the lungs, myocardium, liver, small and large intestine, kidneys and spleen were studied by conventional histology. Their results permitted the following conclusion. "Taking into consideration that changes in the human body under hyperbaric conditions develop in the same direction as in the animal organism, favorable prognosis can be drawn concerning the possibility of man's stay and work at the depth of the oceanic shelf during a month of time. "The Japanese at JAMSTEC have carried out similar work with rats at 300 m for 120 days. This is an area which could be studied more extensively but there seems little evidence of any significant changes so far.

Human Deep Helium Diving.

Total numbers of divers exposed to increased pressures of oxygen-helium or trimix to depths equivalent to 300 m or deeper are not easy to compute but there are many, many more than the uninformed realize. So far as can be calculated there have been well over 250 experimental man-dives to depths between 300 m to 686 m involving French, American, English and Norwegian divers. Some of these dives have been detailed (Bennett 1982).

Thus the Comex company alone has over 140 such man dives to its credit, the British AMTE (Physiology Laboratory) has made over a dozen dives and the U.S. Navy have also made well over that number. Others include the University of Pennsylvania with 8 dives. NUTEC dives have also over a dozen man-dives and the Duke Atlantis experiments totalled 12 man-dives with another dozen or so at 300 m.

Let us not forget either the Shell Oil Cognac project in the Gulf of Mexico. This had very many commercial divers indeed working in

saturation for periods of up to 1 month at 31 bar over the total 6 months or so of the project.

Yet from all this deep diving exposure, no substantial evidence has surfaced for any long term functional deficits consistent with the possible pathophysiology of HPNS, decompression sickness or hypoxia or any other factor specially related to deep diving. That is not to say that some signs or symptoms, as will be discussed, have not been inferred as related to a given dive. However, these effects such as mild depression or effects on memory etc., have not interfered with the individuals ability to work and have proved difficult to separate from other life stresses, unrelated to the dive, which also can cause such symptoms including alcohol abuse and use of drugs, divorce, change in job, death of a relative etc. Such latter stresses, not uncommon in life, have been implicated as a factor in the onset of serious health consequences including emotional problems, depression, phobias, psychosomatic ailments, heart disease and even increased cancer and death rates (Singer 1982).

To my knowledge, there is presently therefore no documented case of any diver being functionally incapacitated as a result of exposure to oxygen-helium dives greater than 300 m.

Comparison with Diving to Depths Less than 1000 ft

In order to get the "hysteria" concerning post-dive risks of deep oxygen-helium or trimix diving in correct perspective, it is necessary to consider the essentially negative data provided by such dives with the much more serious dangers from shallower compressed air or mixed gas diving or from those divers who have had decompression sickness involving the central nervous system.

Thus Peters, Levin and Kelly (1977) examined 19 male commercial divers and one female for neurologic complaints related to diving. Ten of these divers had at least one episode of decompression illness involving the central nervous system and some attributed their symptoms to multiple traumatic decompressions.

The tests involved a "blinded" detailed neurological examination and neuropsychological studies including the Wechsler Adult Intelligence Scale (WAIS), Wechsler memory scale, Reitan trail making test, Reitan finger-tapping test, supraspan digit storage test of Drach-

man and Leavitl and the Minnesota Multiphasic Personality Inventory (MMPI) the details of which are given in the paper.

Eight of the divers were found to be abnormal on neurological examinations. Of the 19 divers who were examined, 7 showed neuropsychologic deficits and 8 were normal.

Of the 8 neurologically impaired, 7 were psychologically impaired. Of the 11 other divers with normal neurologic findings, 8 were unimpaired on all behavioral procedures, but 3 showed "excessive response in the pathologic direction of psychologic assessment".

The latter points to another factor, that determination of what is abnormal or normal is a far from exact science and is based often only on whether a given set of data fits within the limits established for a "normal" population seen by those workers. Nevertheless, the work does show definite evidence of significant changes in the neuropsychiatric state of divers who have had cerebral decompression sickness. Such cases have resulted in many multimillion dollar law suits in the United States.

Of even more significance is the investigation by Værnes et al (1982) of possible differences in specific CNS functions in 2 groups of divers - those with a history of diving accidents (N=9) and accident free divers (N=15) plus a reference group (N=10). Among the tests used were the Halstead neuropsychological battery with added sensorimotor tests, the WAIS, the Wechsler memory scale, the MMPI, skin conductance, power spectrum EEG and brainstem evoked potentials plus a structured interview. Eight out of the 9 accident group divers showed abnormal neuropsychological tests implicating lesions of the higher CNS.

In addition, 5 of the accident group had a syndrome of subcortical/-limbic dysfunction with specific memory defects, low autonomic recovery, sustained attention problems and emotional lability such as increased irritability.

In all the 9 cases arterial gas embolism or hypoxia were the suspected cause. All the divers had experienced one or more accidents involving CNS symptomatology requiring subsequent therapy.

Levin (1975) examined commercial divers after an average 15.7 months from when they experienced CNS decompression illness. They showed a wide range of symptomatic complaints of both a neurologic and psychiatric nature, including intensification of marital discord,

sleep disturbance, sexual dysfunction, fatigue, depression and irritability. Dizziness, buzzing in the ears, word finding difficulty and forgetfulness were the most frequent neurologic complaints. Divers with normal neuropsychological test findings, were considered to have fewer symptomatic complaints ($x = 4.0$) than divers manifesting behavioral deficits ($x = 6.0$) although the difference was not significant. Again, this shows the difficulty of interpretation of such data especially since most of these cases were involved in litigation!

However, other data were more significant such as their WAIS scores, supraspan storage and forward digit span and Wechsler memory quotient.

The MMPI was two standard deviations above the standard population for the impaired divers, with 5 of the 10 clinical scales elevated to at least 70 ± 10 from the normal 50. The control divers did not include a single such large elevation which suggests severe emotional disturbance in the impaired divers. Acute distress, depression and anxiety were apparent in the profiles, with greater body concern and disruption of cognitive efficiency.

On the basis of the above data, compared with only vague rumors as to the possiblity of post-dive neurologic deficits in deep diving, it would be more realistic to suggest that air and mixed gas diving shallow of 100 m is far more dangerous. Such diving appears to involve more neurological or CNS type decompression sickness than with the very slow decompressions of deep oxygen-helium diving that primarily seem to cause limb pains and clearly such CNS involvement can lead to post diving neurological deficits.

Duke Deep Diving Experience.

Soon after the Atlantis deep dive series started in 1979, rumors began to circulate that individuals who had undergone deep diving exposures were subject to personality changes, memory decrements and performance degradations. Although no objective evidence had been presented, routine detailed evaluations of memory and other neuropsychological performance were started with the second dive of the Atlantis series in 1980. Results of those studies gave no indication of any problem associated with such dives deeper than 300 m.

However, approximately five weeks after the 1981 Atlantis III dive to a record 2250 ft (686 m) one of the three divers reported difficulty

in concentration, inattentiveness while diving and difficulties in remembering simple things such as shopping lists and recipes. The symptoms were accompanied by feelings of acute anxiety and sadness.

Although immediate post-dive neuropsychological testing had shown nothing unusual except for a very low short-term memory score (which was present both before and after the divers exposure to 460 m in the earlier Atlantis I in 1979) the possibility that these symptoms were due to the Atlantis III dive was examined.

Over the course of ten months following the dive, numerous clinical investigations were performed at a number of different institutions and Duke Medical Center. These included the most sophisticated neurological and neuropsychological tests including CAT scans, evoked potentials, MMPI etc., for a total of over 50 tests (see Appendix 1).

During the entire time since the symptoms were first noted, the diver continued to perform successfully in difficult job and life situations. Further, the effects were mild which made quantatitive measurement difficult and highly subject to interpretation.

While it would be illuminating to divulge more fully the test data, it is not possible as the individual has understandably requested restricted release of such personal psychological and psychiatric material. However, analysis of the test data did not produce any clear relationship to the Atlantis III dive or any known process consistent with the pathophysiology of diving, decompression sickness, HPNS or hypoxia.

It may be relevant that this diver also had made over 2000 commercial dives to much lesser depths and was subject post-dive to a significantly high number of unusual serious life stresses during the post-dive period.

Thus, while it was impossible to divorce this subject totally from the deep diving exposure, after reviewing the case in detail and consulting with other centers which had no indications of similar experiences, we felt that there was insufficient reason to stop our deep diving research. We did elect, however, to institute a cautious approach with comprehensive neuropsychometric evaluations of all our divers in accordance with the tests in the Appendix, pre- and post-dive, and at regular intervals later, such as 6 to 12 months or earlier if there was any report of problems.

Some further incidents occurred of a different nature which also must be considered. During the intensive post-dive tests instituted on

all the Atlantis divers, another of the divers was noted to have abnormal changes in somatosensory evoked potentials. However, no predive data existed for this man to identify if it was always aberrant. Since, however, he had experienced joint decompression pains it was diagnosed as compatible with the spinal residua of decompression sickness, although there was no concurrent symptomatology. Since there were no other qualitative or quantitative indications of any abnormality whatsoever in attention, memory, language, perception, praxis and sensory-motor function, no further action was taken. The diver has since continued to excel at a high level of expertise in his demanding profession as an academic surgeon and researcher and demonstrates no sequalae.

Again another of the Duke divers, who had been selected to be included in the first NUTEC dive to 300 m, was noted to occasionally show single intermittent spikes on his EEG. He had made previous dives at Duke to 460 m and 686 m and complained of no problems. Norwegian EEG experts, however, were concerned that this reflected pre-convulsive indications and denied him diving approval. Further independent review by other EEG experts other than at Duke considered the activity, however, to be "within normal limits". Coupled with the fact that he had never had any kind of seizure, had already dived twice to over 460 m including a record 686 m and was otherwise in superb physical and mental health, he was allowed to dive in Atlantis IV in 1982. The small occasional spike has continued to be seen in subsequent EEG measurements, but the individual has had no other problems whatsoever and is in the peak of condition and studying for an engineering degree. This illustrates the problem of decisions made solely on such "soft" criteria as the EEG or evoked potentials without an associated symptomatology. A colleague remarked that with the enormous number of tests used plus the non-objectivity of many of them, it would be remarkable indeed if everything was absolutely normal and put these two results down to "overtest".

Finally in Atlantis IV a situation arose placing considerable question on the value of the large number of tests and the resulting considerable expense. During this dive to 650 m one of the divers became hypomanic at 650 m. Starting with inability to sleep around 500 m which was controlled with Restoril, by the second day at 650 m he was hyperactive, agitated and anxious with pressured speech, racing thoughts

and flights of ideas accompanied by deficits in short-term memory, attention and concentration, visual and auditory distortions or hallucinations, increased reflexes, obsessive writing or speaking and grandiose ideas. During decompression he was sedated with Valium until at some 400 m he was given chlorpromazine followed later by maintenance lithium. The chlorpromazine started recovery and by the time surface was reached, he was nearly back to normal. Complete recovery took several weeks of continued lithium therapy after which he fully recovered and returned to work and continues to function very well in a demanding intellectual and stressful environment as an anaesthesiologist.

Whatever the aetiology of this condition and its relationship to deep diving, it is important to realize that in spite of all the predive tests indicated in the Appendix, this man was given full approval by the psychiatrists, psychologists and neurologists to make the dive with every confidence of success.

Evaluations by Others.

Prior to this last Atlantis dive, the U.S. Navy, as a consequence of the continuing rumors about post-dive problems, also carried out extensive examinations by clinical psychlogists, psychiatrists and neurologists on the 6 USN divers who made the dive to 1800 ft in 1979. No functional deficits were found. Yet, in spite of this positive evidence, the U.S. Navy chose to limit their future diving, including research, to no more than 300 m until such diving could be considered safe.

One can with good reason ask why, since there appears to be no evidence of deep dives doing any particular lasting harm just because they are necessarily deep. Indeed as we have seen, deep diving may in fact be a lot safer than shallow diving and has no special hazards to be feared comensurate with the rumors.

Deep diving is as safe if not safer than shallower diving except for the obvious increased logistic risks. Nevertheless, as at Duke, a cautious approach is advisable and extensive neuropsychiatric and psychometric evaluations prior to a deep dive, 2 weeks following and at some delayed time thereafter - if only to prevent rumors or inappropriate litigation - are at least necessary! At Duke we do this now for nitrox saturation diving or any other non routine diving using U.S. Navy Tables, to protect against litigation.

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APPENDIX

This is a list of laboratory, radiographic and professional consultations used to evaluate divers for experimental deep diving at Duke Medical Center.

Complete blood count with differential
Serum electrolytes
Sedimentation rate
Standard test for syphilis
Urinanalysis
Electroencephalogram
Electrocardiogram
Electronystagmogram
Audiogram
Evoked potentials:
(a) Auditory
(b) Somatosensory
(c) Visual
Chest x-ray

Long bone x-rays
Bone scan
Brain C.T. scan
Dental screen
Neurological evaluation
Ophthalmological evaluation
*Psychiatric evaluation

Neuropsychiatric laboratory, to include:
(a) MMPI
*(b) Paced auditory serial addition task
(c) Wechsler adult intelligence scale
(d) Aphasia screening battery
(e) Trails
(f) Tapping
(g) Tactual performance test
*(h) Dynomometer
(i) Wechsler memory scale - revised
(j) Category test
*(k) Seashore rhythm and speech
*(l) Figural recognition test
*(m) Metamemory questionnaire
(n) Greek cross
(o) Wechsler associated learning
(p) Digit symbol
(q) Digit span
*(r) New York memory
*(s) Levin selective reminding task

Clinical Psychology evaluation which includes Bowman Gray School of Medicine's battery of memory test to diagnose mild cognitive decrements (persistent), which includes:
(a) Complex figures, Rey-Taylor test, immediate Hand delayed recall
(b) Prose narrative
(c) Rey auditory-verbal learning test
(d) Bowman Gray figural learning test
(e) Porteus Mazes

(f) Token task
(g) Hooper visual organization test
*(h) Thorough psychiatric-neuropsychologic history
Complete general medical examination.

* These studies added at the beginning of the Atlantis IV (fall 1982) workups.

QUESTIONS AFTER PROFESSOR BENNETT'S PAPER

Dr Baddeley: Just a small point about the profile of neuropsychological performance. It is commonly the case that people with very substantial brain damage may still perform above average on quite a large number of tests. It is normal to use the pattern of dysfunction, rather than the overall level. One might argue that it would be nice to have tests that did not require that, but at the moment, we do not. The ways in which your results were being viewed were actually fairly standard and you were not being particularly penalised in that respect.

Dr Værnes: The high incidence of CNS dysfunction after shallow dives, documented in my report, should not be an argument for CNS dysfunction after deep dives. The shallow dives involved uncontrolled decompressions and hypoxia accidents, but the deep dives followed the saturation and decompression profile correctly all the time.

Prof Bennett: I disagree. There has been an argument, very strongly held in the United States, that deep diving is very dangerous, that it is immoral and that people are being hurt. I am pointing out that in over 240 man dives, to my knowledge, no-one has been seriously hurt. If they are going to say that deep oxyhelium diving is dangerous and should be stopped, that the US Navy has drawn a line at 1000 feet, and diving beyond that is not safe, if they say that, then I am saying you had better look at diving to 200 feet, because that is not safe. I can give you large numbers of people whose central nervous systems really have been damaged from that exposure. So, if you want to worry about oxyhelium diving, you worry about air diving, because that is what you should stop. I am trying to make this realistic and not the witch hunt which has happened with deep oxyhelium diving. As far as I can see, it is safer than compressed air diving, and I stand on that.

Dr Værnes: We have 23 man dives and have 4 cases with mild symptomatology. That is an incidence of 20 %. I would come back to the argument that it is not that there is this incidence after 1 or 2 dives. If these divers were to carry out deep dives once or twice for several years, what then would be the incidence? This analogy to boxing again shows it to be our main concern.

Prof Bennett: I was not aware of these data of yours, and it is very important. I would like to hear more of the US Navy and French work to find out what their results were on their divers. I still say that those

are the only cases I know and that they recovered in 3 months. If that was directly related to the dive and if it was something specific to the brain in terms of disorder, that is very important. I would like to find out why, and what it means. Obviously, if this is the case, it certainly needs investigation, but let us also realise that there is nothing special about a magic line drawn at 300 metres. I am saying that compressed air diving is equally damaging and I do not hear anybody screaming to stop that. So, if you must be objective, then include air in the picture. There is more permanent damage to the brain there than I have yet seen with oxygen-helium.

Dr James: I think I would support what you are saying as far as air diving is concerned because there is a vessel moored almost outside this hotel which has been responsible for a very large number of incidents over this last season. These were almost exclusively central nervous system in origin and, knowing what we know about CNS dysfunction as result of decompression sickness, I am certain if one had the micropathology available, there would be obvious evidence of lesions within the central nervous system of these men. Going back to the rationale for using nitrogen in Trimix to alleviate HPNS symptoms, I wonder whether elevating the PO2 has a similar effect in the suppression of symptoms? Has it been tried on a short term basis?

Prof Bennett: Yes, this is very important. As far as deep diving is concerned, I like the oxygen to be as high as possible and certainly 0.5 bar wherever possible. In our very deep dives with heavy work loads there is a lactic acidosis present when the arterial blood oxygens tensions are normal. There is no real problem. So there is something about the transport of oxygen from the blood to tissue which suggests one might not be getting enough oxygen. If the oxygen is about 0.3 - 0.35 bar, I would suggest that this could happen even at the shallower depths without work. We did some work with animals a long time ago, published in the Journal of Applied Physiology, to show that, with inert gas present, the oxygen available to the brain is less than expected when giving pure oxygen without the inert gas present. So I am sure there is less oxygen getting to the brain than we think.

Dr Giran: French experience confirms Professor Bennett's opinion in terms of danger concerning air versus heliox dives. Our experience in 1981 in Comex was 16000 dives. We had only 2 CNS accidents - 1 medulla and 1 cerebral. They both occurred after an air dive. Knowing

that we are diving with no case of CNS damage for years with heliox, I would say that this is an important point.

Prof McCallum: Would your elaborate a little? You were talking about decompression sickness there, I think. In terms of long-term defects, can you confine yourself to that?

Dr Giran: Yes, I think that you can have short as well as long term effects from that kind of accident. As for the long-term effects coming from compression (or whatever event in saturation) we have no experience.

Prof Elliott: There is a point that was raised by Peter Bennett and is relevant to what we were discussing previously. Going back to the cases presented this morning, you said that 4 cases have been found. Two of these have yet to be followed up, but let us say that there is in them evidence of some medium range defect, but nevertheless transient. Could you tell us what the PO2 was in those particular dives?

Dr Tønjum: 0.45 bar. 0.5 was the highest in saturation and during decompression of one dive, it was 0.6.

Dr Becker: Where was the neuropsychological testing done (following the Atlantis dives) and who did the interpretation of the results?

Prof Bennett: They were done in a number of different areas. The neurological examinations were done at a hospital in Los Angeles and the neuropsychiatric studies were done at Bowman Grey. Dr Gillan read some of the EEG's out at Wrightsville Beach. They were also done by our own Duke people as well. Double studies were conducted as we were aware of potential criticism.

Dr Baddeley: Is there not a danger that if you do some of the neuropsychological tests yourself and then they are done somewhere else, you can get practice effects which actually distort them. Do you have sufficient parallel forms to avoid that?

Prof Bennett: I agree that this is a problem which we have to address ourselves to sooner or later if we start to do this on a routine scale. It is a good point but I think in the majority of the cases we were looking at, they had not had enough to really get used to them. But I would not like to do too many tests on them.

Dr Baddeley: I was thinking more of the standardised tests where the norms are based on the assumption that the persons are either doing it for the first time, or at the very least, have not done it for a long time.

Prof Bennett: In our own performance tests we certainly train them up to a baseline for our measurements. Then they are at baseline and all our tests are against that baseline. Those baselines do not shift once we have got them there, so they do pretty well. They come back to it when they reach decompression, but then we have been running them for 1 to 2 months beforehand, every day. They are not standard neuropsychiatric tests that we are talking about.

Dr Baddeley: Those were the ones I was actually referring to, the neuropsychological ones rather than the others.

Prof Bennett: Those tests that were done at Duke by the neuropsychological team had enough variables to have new versions every time.

Dr Smith-Sivertsen: You are comparing shallower diving with diving deeper than 300 metres as far as injury and neuropsychological effects are concerned. Of course we see very many such cases among air divers and shallow divers, but you have to compare these to the number of dives, to the number of compressions and decompressions. Furthermore, I think that the accident you have in shallow diving can be explained by sloppy organisation, poor diving and so forth. The diving that you are referring to has been done with streamlined organisation fully attended by highly qualified specialists throughout the dives. I think you have to take that into consideration. If you let deep diving loose, beyond 300 metres, you will have the same sloppy organisation in diving as you have in shallower diving. That is why I am afraid of it.

Prof Bennett: I said earlier that deep diving probably requires an elite team and any diving company has got to have a good, highly trained team who know what they are doing. You cannot stop deep diving because of the general average sloppy diving that perhaps occurs at 100 feet.

Nevertheless, the real issue is that there has been a witch hunt against diving deeper than 300 metres. That witch hunt is without foundation. There is no evidence. I have heard of 4 mild cases which have recovered. There is no evidence of long term disability. In the United States, rumours were rampant of long-term disability, of divers being injured, of brain damage, which are not true. The real problem is in air diving. So I am trying to get very clear, once and for all, that there is no valid reason for drawing a magic limiting line at 300 metres. Why not 350, or even 400 metres? Who drew the line at 300 metres? People have been trying to draw lines in deep diving since 1900. There

is a picture of Sir Leonard Hill inside a pressure chamber making his first dive to 200 feet on compressed air after which he said there was a very narcotic sensation, so very bad, that man will probably never dive much deeper than 200 feet. Well, we are a lot deeper than that.

The world and its life expands by venturing into it, not by restriction. As long as we know there is no major damage, and we proceed with caution and adequate measurements, then I think that we can proceed ethically. The statement is made that the navies of the world have drawn the line at 300 metres, therefore diving deeper than 300 metres is dangerous. Give me some evidence that it is dangerous and I will support you. I have seen no evidence.

Dr Giran: Concerning the question of accidents on air compared to heliox, I can give you the data from Comex. In 1981 a total of 16000 dives were carried out, presenting 34000 hours of bottom time. This includes 13000 dives on air with 14000 hours spent on the bottom of the sea-bed. In bell saturation, it represents 3000 dives with 19000 hours spent at bottom. When we compared this data to the occurence of accidents, it became obvious that we had more accidents versus time spent at bottom or versus number of dives when diving on air. I do confirm that, with acceptable procedures, it is more dangerous to dive on air deeper than 30 or 40 metres, than to dive to 200 or 300 metres on heliox.

Dr Smith-Sivertsen: I think the main difference is between bell diving and surface oriented diving, rather than between air or saturation.

Dr James: This difference was observed in the US Navy in 1938-39 by Behnke and Yarborough in relation to surface oriented diving vis a vis heliox versus air. They wrote it up at that time (Nav. Med. Bull 36: 542-550, 1938). It is not just bell diving versus surface oriented diving difference.

Dr Værnes: We have not proven that there are long term effects of diving but also we have not proven that there are not long-term effects of diving. It is impossible to prove that. Therefore, it is important to have longitudinal studies and I do not follow you when you say we have a hysterical reaction to our findings of neurological symptoms post-dive. As long as we cannot prove that there are no long term effects, we have to be open minded. So it is more or less a political standpoint.

Prof Bennett: I agree with you absolutely. In no way am I impuning your own work or Professor Aarli in picking up these cases. I think they are important and indeed, the purpose of the tests is to find out if anything is there.

Although the word hysteria may be wrongly placed in Europe, it is not wrongly placed in the United States. It may be better now, but rumour was rampant and the Duke team suffered. They suffered their grants being withdrawn on the basis of rumours that we were damaging divers. So, if I seem a little highly-strung about it, it is because I got burned pretty hard, personally as well as in my own laboratory, for reasons for which there was no substantive evidence: I think people had this feeling that beyond these 4 magic numbers 1000 (300 metres), something terrible will happen. Science is not like that, you go slowly and you measure and you make sure things are safe. That is what we are doing. The difficulty is that a negative attitude is stopping research in the field, let alone the practical use of this technology. I make a comparison here with the American astronauts. The astronaut programme ran into the same problem: rumours put up about memory deficits and changes in personality and so on. The only reason it proceeded was political. The Russians got into the race and the "problems" were set aside as being irrelevant. In diving this element is not there, nor should it be. So we can be objective and do the proper measurements.

US NAVY EXPERIENCE IN DEEP DIVING

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Currently, the US Navy does not do operational diving below 1000 FSW. However, in order to establish what the maximum practical operational limit for deep diving is, the US Navy Experimental Diving Unit (NEDU) has conducted four very deep experimental saturation dives. In chronological order these were to 1600, 1400, 1500 and 1800 FSW. Each of these dives involved 6 subjects and were not designed to establish the maximum depth to which humans could safely be subjected, but rather to establish what the greatest depth was at which divers could perform the complicated tasks and exercise levels which would be expected on operational dives, that is to establish the maximum practical depth limit. To accomplish this, each of the four dives involved several days at the maximum depth during which exercise was performed in the water. On each of these four dives many hours were spent doing submerged exercise using up to 6 subjects. This is in contrast to other deep dives where fewer subjects were exposed to only dry exercise. In addition to exercise studies, other studies ranging from psycho-motor performance assessment to pulmonary function testing were done. The bibliography at the end of this presentation lists the published papers describing the detailed results of studies during these dives for those who are interested. The purpose of this paper is to discuss the overall level of performance of the subjects during each of these dives, detail any long term problems which resulted from these dives, and present what NEDU feels is the practicality of deep diving. Table 1 summarizes all four US Navy deep dives. All of the dives were helium-oxygen saturation dives and used the Standard US Navy saturation decompression rates (6 FSW/hr to 200 FSW, 5 FSW/hr to 100 FSW, 4 FSW/hr to 50 FSW and 3 FSW/hr to the surface. A 6 hr stop from 0000-0600 and a 2 hour stop from 1400-1600 are taken each day).

The chamber was compressed on air to a depth of 22 FSW to establish the .35 PO_2 (38 FSW to establish a .45 PO_2 on the 1800 FSW dive) and this was the only nitrogen in the chamber. This resulted in a maximum nitrogen partial pressure of 1.32 and 1.70 ATA for these dives.

The first deep dive was done at the Taylor Diving and Salvage Facility near New Orleans, LA, in 1973 and was to a depth of 1600 FSW. Of the 6 subjects in this dive, two were physicians and two were hospital corpsmen. The remaining two were US Navy divers specially trained in MK 10 maintenance. Since this was the first dive below 1000 FSW done by the US Navy, it was by far the most intensely studied of all the four dives. Exercise studies included submerged exercise using a specially modified General Electric MK 10 closed circuit breathing apparatus and a KMB9 open circuit band mask. Testing for performance not only consisted of exercise at 1600 FSW but also measurment of vestibular function, standing steadiness, tremor, and cognitive and perceptual motor performance (1, 2, 3, 4). The compression rates for this dive were purposefully slow and stops of approximately 1 day at 400, 1000 and 1300 FSW resulted in a total compression time of just over 6 days. During compression, divers noticed no effects of pressure until below 1000 FSW. Intention tremor was the most noticeable abnormality increasing as depth approached 1600 FSW, then decreasing slightly at 1600 FSW and returning to normal during decompression (4). Vestibular studies showed no abnormalities but standing steadiness as measured on a balance rail decreased with increasing depth below 1000 FSW (3). While these changes were of experimental significance, they offered only minor impediments to the divers' ability to perform their tasks. At 1600 FSW, divers were required to insert radial arterial cannulae for blood gas studies, set up and manipulate tiny I.V. tubing valves and connections, do precise calibrations on a blood gas machine and set up and maintain the General Electric MK 10 underwater breathing apparatus. The divers were able to perform all of these tasks although they had to be slower and more deliberate than at 1 ATA. Some weight loss was observed in the divers but this could not be ascribed to a direct effect of pressure on metabolism; rather, this was probably due to the loss of taste of the food at depth and to the fact most of the divers were closely watching their weight. The most stressful situation which the divers encountered at 1600 FSW was the intense shortness of breath with only mild exertion (1.5-2.0 l/min oxy-

TABLE 1

U.S. NAVY DIVES BELOW 1000 FSW

DEPTH (PO_2) Date	SUBJECTS	DAYS AT MAX DEPTH	COMP. RATE (FPH)	PROBLEMS DIVE	LONG TERM
1600 FSW (.35-.40) APR 73	6	7	to 400 FSW 300 400-1000 FSW 40 1000-1300 FSW 30 1300-1600 FSW 20	Tremor Weight Loss Dyspnea w/exertion Knee pain during decompression.	Recurrence of knee pain 1 week post dive on plane.
1400 FSW (.35-.40) AUG 77 Knee pain in 2 divers	6	4	to 620 FSW 180 620-1000 FSW 120 (1 hr hold at 1000 FSW) 1000-1208 FSW 90 1208-1400 FSW 60	Nausea, Anorexia, weight loss below 1000 FSW. Dyspnea w/exertion Tremor Knee pain during decompression.	Malaise in 3 divers lasting 3 months. Knee pain in 2 divers lasting years.
1500 FSW (.35-.40) NOV 77	6	4	to 200 FSW 180 200-600 FSW 120 600-1000 FSW 60 1000-1152 FSW 30 1152-1500 FSW 20	Vertigo, tremor. Mild anorexia Dyspnea w/exertion Knee pain during decompression.	NONE
1800 FSW OCT 79	6	4	to 650 FSW 120 650-1000 FSW 40 1000-1600 FSW 30 1600-1800 FSW 15	Nausea, vertigo, tremor, severe anorexia, weight loss, Dyspnea at rest. No DCS.	Malaise, absent mindedness in some subjects for several months. Permanent knee injury 1 diver. Dive related?

gen consumption). This dyspnea was observed on subsequent dives and has also been observed at depths as shallow as 1000 FSW. It seems to be a function of increased gas density and static lung loading and not an effect of pressure per se (11). Mood evaluation showed increased anxiety and hostility at 1600 FSW although this was not overtly evident in diver interactions during the dive (1). Frustration at not being able to talk directly to fellow divers led to a drastic reduction in diver to diver communication and a feeling of total dependence on the outside crew; this undoubtedly contributed to this anxiety and hostility. During decompression there were several bouts of knee pain which required recompression but all divers were normal and felt well upon surfacing. Approximately 1 week after diving some divers had knee pain during their flight back to Washington, D.C., but this remitted upon landing. No long-term problems or complaints were observed in any of the 6 divers.

The ability of the diver to adapt to the increased pressure at 1600 FSW was in marked contrast to the divers' performance during the 1400 FSW dive done at the new NEDU facility in Panama City, FL in August of 1976. In this dive compression was done from the surface to 1208 FSW in just 9 hours 48 min. Below 1000 FSW all of the divers had marked gastro-intestinal distress resulting in loss of appetite. While the 6 % body weight loss during the 1400 FSW was similar to that observed during the 1600 FSW dive, the gastrointestinal symptoms observed during the 1400 FSW had been absent on the 1600 FSW dive. Tremor was severe. During this dive, two excursions were performed upward from the initial storage depth of 1208 FSW to 1020 and 1000 FSW respectively. Compression directly to 1400 FSW was made from 1000 FSW at rates 3 and 4 times greater than done during the 1600 FSW dive. This rapid compression increased the gastro-intestinal distress and tremor to the point where the divers' ability to perform their assigned tasks was affected much more than during the 1600 FSW dive. At 1400 FSW, submerged exercise was done using a specially designed breathing system (8) and the divers maximum work capacity was hampered by severe dyspnea at moderate exercise (1.8-2.4 l/min).

During decompression there were multiple bouts of knee pain which began at about 100-50 FSW. Several bouts of recompression were done and the saturation decomprssion could not be continued until the

oxygen partial pressure was raised to .42 ATA for the last 40 FSW or so. Upon surfacing, 3 divers had marked malaise which lasted several months and two divers had residual knee aches and limitation of function lasting up to 2 years. Outside of the marked malaise there were no other psycho-physiological sequelae and with the resolution of the knee pain all divers returned to normal. The marked gastro-intestinal problems and limitation of performance during the 1400 FSW dive was prescribed to the rapid compression rates used below 1000 FSW.

On the 1500 FSW dive done in November of 1977, compression rates were similar to those used during the 1600 FSW dive. There was a 4 day hold at 200 FSW and three day holds at approximately 600, 1000 and 1200 FSW where multiple upward excursions were done. Divers arrived at 1500 FSW on dive day 16. Diver perfomance was almost the same or probably better than that observed during the 1600 FSW dive. Submerged exercise was done at work loads approaching 2.5-3.0 l/min oxygen consumption on a specially built low resistance breathing system. Vertigo and anorexia were the most notable problems but the divers were able to partially adapt to these effects of pressure and perform adequately as they had during the 1600 FSW dive. Tremor was not a significant problem on this dive. There was no significant weight loss on this dive.

The same compression rates as used during the 1600 and 1500 FSW dives were used during the 1800 FSW dive down to 1600 FSW and then slowed down to 15 feet per hour from 1600 to 1800 FSW. The 6 divers were initially compressed to 640 FSW where they remained for 8 days performing manned exercise testing of a US Navy semi-closed underwater breathing apparatus. Over the next 4 days, compression was done to 1800 FSW with holds at 1000 FSW and 1400 FSW where thermal balance experiments were done. Below 1000 FSW, the divers noted a decrease in their feeling of well being and this was accompanied by weight loss. Below 1400 FSW, divers noted dyspnoea at rest and by the time they reached 1800 FSW it was so severe that divers had difficulty eating and breathing at the same time. At 1800 FSW, all divers experienced symptoms of orthostatic light-headedness and nausea and imbalance throughout the time spent below 1500 FSW. All divers had mild tremor at rest and very gross distressing tremor following exercise. Muscle fasciculations and gross myoclonic limb jerks were observed both awake and asleep. For this dive, a special low re-

sistance underwater breathing system similar to the one developed at the State University of New York at Buffalo, was built to allow accurate assessment of cardiorespiratory function during exercise at depth (12). Dyspnoea with exertion was severe and underwater exercise was limited to about 2.0 l/min. Exercise tolerance improved during decompression and was noticeably improved by 1500 FSW and back to normal at 1000 FSW (10) where sustained efforts at 3.0 l/min were possible.

Gastro-intestinal symptoms during the 1800 FSW dive were severe compared to the other three deep dives and two of the divers had frequent bouts of vomiting throughout the 4 day stay at 1800 FSW. While all 6 subjects suffered varying symptoms of the increased hydrostatic pressure, not all subjects were equally affected. Two subjects had very mild gastro-intestinal symptoms and suffered only an increased irritability. These subjects had their performance at 1800 FSW only minimally impaired. Two other subjects had gastro-intestinal symptoms which proved a nuisance but did not prevent their exercising at 1800 FSW. The other two subjects were almost completely incapacitated mainly from gastro-intestinal symptoms and one was unable to perform any exercise at all.

Psychomotor performance at 1800 FSW was severely impaired in all divers and simple tasks such as tracing out sample lines or replacing gas fittings which would take only a few minutes at 1 ATA, might take up to an hour. One diver spent 20 minutes searching for a sample line which he was holding in his hand all the time.

Decompression was accomplished without incident and there was no decompression sickness observed. This was probably the result of having raised the chamber oxygen partial pressure from the .35 - .40 ATA level used during the first 3 deep dives to .40 - .45 ATA. In the decompression schedule used for the 1800 FSW dive, the mandatory 8 hours of stop time was taken all at one time from 0800 to 1600 to allow experiments to be done at a constant depth during decompression without lengthening the decompression. (Normally, a 6 hour stop is taken from 0000 to 0600 and a 2 hour stop is taken from 1400 to 1600 each day).

After surfacing from the 1800 FSW dive, all divers noticed malaise and some divers complained of increased absent-mindedness for several months after surfacing. These symtoms were accompanied by

knee aching with prolonged standing and one diver had a marked permanent impairment in knee function. This particular diver noted some knee problems during the intensive running program instituted during the 3 month period before the dive. He had compression knee pains and noticed exacerbation of this knee pain with exercise. His knee pain did not change during decompression but increased over a several months period following the surfacing of the dive. This diver has been seen by an orthopaedic surgeon who diagnosed bilateral meniscus degeneration in both knees. Surgery was performed on one knee with some abatement of symptoms but this diver still requires knee braces, the occasional assistance of a cane and cannot stand for long periods of time. He is still able to dive and performs his tasks of maintaining the NEDU SCUBA Locker and the small boats without impairmant. It appears that this diver's knee problems were due to his marked increase in physical activity (especially running) during the pre-dive workup exacerbated by the bicycle ergometer exercise done during the dive. How much this problem was exacerbated by the increased hydrostatic exposure per se is unknown.

Approximately 3 years after surfacing from the dive, all divers who participated in the 1800 FSW dive underwent an intense series of neuropsychiatric testing at the Naval Medical Reseach Institute in Bethesda, MD and no abnormalities were found.

Based on the experience gained during these four deep saturation dives, it appears that the compression rates used during the 1400 FSW dive below 1000 FSW were too fast and this directly contributed to markedly decreased diver performance. If the compression rates for the 1600 FSW dive below 1000 FSW were too fast and this directly contributed to markedly decreased diver performance. If the compression rates for the 1600 FSW dive are used, operational dives to depths of 1500 to 1600 FSW seem practicable. At these depths, divers can still perform a reasonable amount of underwater exercise although dyspnoea with exertion is pronounced. Currently available underwater breathing apparatus could be modified and made to function adequately down to this depth. Overall performance is impaired but the divers seem to be able to compensate adequately. Below 1600 FSW, an operational capability is at present impracticable using current US Navy diving techniques. The severe impairment of diver exercise tolerance and psychomotor performance as well as the overall decrement in

diver well-being means that operational tasks could probably not be adequately performed. Also, the adequacy of currently available underwater breathing apparatus at this depth is unknown. However, the apparent absence of long term effects on divers to depths of 1800 FSW certainly make human research down to this depth practicable provided compression is slow. Thus, with adequate research and development of new techniques in breathing apparatus, diving to 1800 FSW may be possible.

Currently, the US Navy is limiting the scope of its diving to depths of 1000 FSW or less, since no operational requirement below that depth exists. At NEDU, approximately 6 man-dives every other year to 1000 FSW are done with no observable decrement in performance or long term effects. However, a battery of neuro-psychological tests is being developed which will be given before and after all dives below 600 FSW, not only to establish a data base, but to rule out subtle long term effects from deep diving which may not be obvious. These standardized tests could then be used to quantitate performance and long term effects from dives deeper than 1000 FSW more accurately, when the need for doing these dives arises.

Finally, it must be pointed out that inadvertent diver selection can make deep diving seem better than it is. On all four of these dives, subjects were randomly selected and none had dived below 1000 FSW. On the 1800 FSW dive in particular, if 3 only of the 6 subjects' performance were analyzed, diving to 1800 FSW may seem more practical than we observed. Whether diver selection is a viable method of doing these very deep dives is debatable. However, from a scientific standpoint conclusions based on the performance of only 2 or 3 subjects may be misleading.

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QUESTIONS AFTER DR THALMANN'S PAPER

Prof Donald: Could we know a little more about the increased absent-mindedness for several months after the dives? It seems rather vague. Was it detected by tests, or just reported?

Dr Thalmann: That is correct, it is rather vague. It was purely from the history of individuals who said they could not remember things like where they put their pay cheque or their car keys. This absent-mindedness still persists in one of the subjects in his judgement. From an objective standpoint, we are unable to find out whether there is an abnormality or not. Whether it would be an effect of the deep dive, or some other effect, is unknown since we did not have any good pre-dive baseline data on this particular individual.

Dr Shields: As regards pre-dive baseline data, you said that the 6 divers in the l800 feet were subjected to a battery of neuropsychological tests at NMRI and were normal. Was that normal in comparison with pre-dive data, or was that an isolated finding?

Dr Thalmann: There was no extensive neuropsychological testing done predive. The experience of the 1600, 1400 and 1500 feet dives, where there was more extensive pre and post-dive testing, indicated no changes. On this particular 1800 feet dive, we did not do a big battery of pre and postdive neuropsychological testing, only post-dive.

Dr Tønjum: Did you have new divers in each dive?

Dr Thalmann: Yes, that is correct. No one diver made more than one of those four dives. Most had not made a deep dive before. We went out of our way not to select deep divers.

Prof Bennett: I would agree. We did not select our divers either. They were, in fact, novices: scuba divers, except for two who were commercial divers and they had never been to anything like 1000 feet. The severe HPNS, you say, may be related to the profile of compression, which was linear without stages. Certainly the 1800 feet dive did not have a good compression profile because it needs to be exponential with stages.

Dr Thalmann: That is not true. We stopped 8 days at 600 feet and we had 1-day stops at three other depths. It was not exponential, but the rate did decrease on the way down. The total compression I recall was 6 days; we spent 8 days at 650 feet and then went to 1000 feet, at 40 feet per hour. We then stopped at 1000 feet for a day, then we went

at 30 feet per hour to 1400 feet where we stopped for a day. From 1400 feet we went at 30 feet per hour and then from 1600 to 1800 at 15 feet per hour. So this was, as far as we could say, about the most conservative compression that anyone was using.

The 1400 feet dive was the dive that probably had the most influence in turning the Navy off deep diving since it was such an awful dive. The 1500 feet dive, which occurred only six months later, had absolutely no problems at all. I mean, it was remarkable in that it was unremarkable. However, we did take 16 days to get to 1500 ft because there were so many studies on the way down.

Dr Becker: What about the 1800 feet dive? You described the 1400 feet dive as an awful dive, filled with problems. I presume, or dissatisfaction. I have heard anecdotes that the divers themselves were very unhappy with the 1800 feet dive. They felt they had failed, somehow, to do well on that dive and were dissatisfied with themselves. Is there anything to that?

Dr Thalmann: Yes, that is true. There was a lot of political upheaval over the 1800 feet dive and the divers themselves probably felt dejected, not from the dive, but from the political aftermath. One diver, however, did feel he personally failed. It was an individual who, from all accounts, is the hairiest-chested diver in the navy who was unable to get out of his bed at 1800 feet. How much this affected his postdive performance is unknown, but he took this as a personal failure. However, it is notable that during decompression, the divers noticed they went through some kind of a "barrier" at about 1500 feet and felt a lot better. By the time they got to 1000 feet they felt as if they were on surface. This seems unusual in the sense that that did not happen on compression, so it may have been a comparison of how awful they felt at 1800 ft.

Dr Mortensen: When selecting divers, would you select a diver for a deep dive with a past history of spinal or cerebral decompression sickness, but without residual symptoms?

Dr Thalmann: It depends what you mean by history. I am sure that some of these divers had histories of spinal decompression sickness. The reason is that the current classification of decompression sickness requires that almost any kind of neurological abnormality is called spinal cord decompression sickness. The question is, would we take a diver who had perfomed poorly on one of these dives, or would we take

a diver who had done one of these dives and dive him again? Currently, we probably would not subject divers to more than one of these dives below 1000 ft until we were sure that we could do it on a more or less routine basis. For now dives below 1000 feet are considered experimental as far as the navy is concerned.

Prof Elliott: We had heard this morning about 4 people who have had transient findings after dives to a shallower depth. Of your 24 men, could you say positively that their abdominal and plantar reflexes were normal post-dive or, if not, would it have bothered you too much anyway?

Dr Thalmann: I can say unequivocally I do not know. We normally do not do abdominal and plantar reflexes on divers for a different reason. These are divers who are subjects in helium oxygen scuba bounce dive table testing. The reason we do that is that if we have a spinal hit it is nice to know if the abdominal reflex was present before, when it is not after. We just look to see if it is there or not. From a clinical standpoint, I would not know what to do if the only thing I could find in an otherwise normal diver was a decreased abdominal reflex. If that was the only abnormality, I do not think we would do anything. The navy divers were not subjected to the kind of scrutiny that the Duke or NUTEC divers have had. This was mainly because some people involved in this dive had been around the navy for a while. After any big diving operation, there are always any number of complaints or abnormalities. You really do not know what to do with them. So we let the divers talk among themselves. After the 1800 feet dive, there was no doubt that the divers had been adversely and significantly affected by this dive because they came back and told us that they were having problems. We did not go out and ask them, they came to us saying "I have trouble remembering" or "I am grouchier than I am normally", or "my knees hurt". The same after the 1400 feet dive: the post-dive experience was again the divers themselves complaining that they had problems. We did not go out and actively seek this. By the same token, after the 1600 feet and 1500 feet dives, none of the divers had any complaints.

General Discussion on the US Experience

Prof Bennett speculated as to the cause of the problems seen after the US Navy dives and suggested that they may be connected with the severity of the HPNS symptoms experienced during the compression and bottom phase. He cited the experience at Duke where there were no indications of similar problems and suggested that this was due to the very much slower compression rates used compared to the US Navy. He felt that the occurrence of post-dive effects might be related to the compression profile and the conduct of the dive. He indicated that this should be borne in mind when considering possible causes, because with a slow compression and the right technique there should be no problem. He felt that this was analagous to decompression sickness where failure to follow the correct procedures produces the bends.

Dr Tønjum disagreed that this was the cause and stressed the importance of diver selection. His experience was that the degree of HPNS experienced was very much an individual phenomenon.

Dr Peterson outlined an example in favour of Professor Bennett's proposal. A dive was carried out to around 1200 ft in which two groups were compressed at different rates; one, a slow staged compression and the other a more direct compression. The opinion of the crew outside the chamber was that the group on the faster compression appeared anti-social and less relaxed. He suggested that, as the population from which the two groups of divers were drawn was assumed to be identical, this would support the views presented by Professor Bennett.

Prof Donald enquired about the "gastro-intestinal symptoms" experienced by the US Navy divers because they appeared quite unusual.

Dr Thalmann indicated that extensive examinations of the divers' blood levels of possible endotoxins proved negative and therefore the cause is as yet unknown. He remarked that the symptoms were not very unusual as they have occurred on two different dives.

Dr Török commented that, on the basis of his studies of the nausea and vomiting that occurred on the Alverstoke dives, he felt it might be due to some disturbance of the autonomic or vestibular systems. It may be the result, therefore, of two different mechanisms. He also indicated that he felt that the instantaneous rate of compression was as

important a parameter in assessing what he called the daily dose of compression, as the overall rate.

Dr Skogland said that the meeting had indicated that there are problems of methodology, particularly in deep diving. The number of divers involved was so small that attempts to drawn conclusions were very difficult if not impossible. He asked if it was possible to come to some agreement on a protocol that would allow mapping, in some consistent manner, of the various experiences of deep diving. He felt that the most constructive way forward was to aggregate the experiences of mixtures, compression rates and other parameters to allow the best method to be developed in the longer term. He believed it would be a sad thing if the "wait-and-see" attitude continued and no common pattern was developed to allow accumulation of all experience. He felt that this was the responsibility of all of the industry from the institutions to the diving companies.

Dr Værnes inquired about the longer term follow-ups of the US Navy divers and was told that a programme had begun with some of the divers who would be tested by a battery of tests at 1000 ft in February, 1984. The frequency of such testing was unknown as yet. As to the question of a common protocol, *Dr Thalmann* believed that the small number of dives necessitates that only one or two methods would be able to be investigated. The US Navy were, therefore, committed to continuing to use the standard Navy tables including the rates of compression and decompression. They will try and maintain their research and carry out pre- and post-dive testing which, ultimately, they hope will be portable enough to take out to sea, but he felt that it would be several years before this was complete.

Dr Becker informed the meeting of a pilot series of neuropsychological tests that are being done on a group of divers involved in a shallow diving study. The battery will be described in his paper. The study is being carried out in the spare time of the dive series, as at present it is not a required project. This pilot study will be used to test if it is realistic and worthwhile to use these tests.

Prof Bennett inquired of Prof Elliott the finding of the large number of man-dives and the six-month project on the Gulf of Mexico. He was told that the information belonged to the diving contractors and was not available for release.

Prof McCallum concluded the discussion by reminding the meeting

that the point made by Dr Skogland about pooling of data was one which they may have to return to later. There was enough information around, but no method of bringing it together.

RECENT BRITISH SIMULATED DEEP DIVING EXPERIENCE

Zoltan Török, AMTE (PL), Alverstoke, England.

The AMTE/PL Deep Dives Series

A year ago almost to the day the AMTE Physiological Laboratory performed a 6 m heliox saturation dive lasting 10 days. This almost normo-baric experiment was called "Dive 15" and marked the end of the British Deep Dive series that started as it finished, with shallow heliox exposures in 1975, intended to provide physiological control data. The objective of the series was to define a range of physiological changes, with long post-dive follow up being permitted by the fact that most dive subjects volunteered from among the Laboratory's own scientific staff. An up to date overview is currently in the press. (Ref 1)

Emphasis was placed on the compression phase and bottom time of the exposures. It was realised at the outset that considering the Laboratory's resources, the Deep Dive Series would have to consist of very few experiments, 18 in all as it turned out. Given the varying maximum depths, decompression research in the sense of testing tables was explicitly ruled out. Indeed the decompression procedures used were experimental only in the sense of their virtual uniqueness, and any decompression sickness caused was very much contrary to intent and predictions.

Eight dry chamber dives were completed to simulated depths at between 300 m and 660 m. All of these, but one, used heliox with 0.4 bar oxygen. Dive 12 b to 660 m employed 10 % nitrogen added to the chamber gas. Altogether 10 subjects took part in the experiments at 300 m and deeper, four took part in two dives, one in three dives. Only two were professional divers, and every member of this small selected group is today continuing successfully in their chosen professions. All are technologists at various levels of attainment and qualification. Two have Ph.D.s, one having obtained it after his experience of the 660 m and 540 m dives.

These dive subjects were deliberately not selected beyond the usual

essential criteria. They had to be medically fit for diving according to the normal Naval and Health and Safety Executive criteria, but beyond this, they would not be at the end of the normal distribution curve for any physiological parameter. Care was taken to ensure that each pair was temperamentally compatible, and that each subject knew what he was volunteering for, preferably from having experienced a short 180 m saturation dive in the same very small chamber. The dive coordinator never had more than four men from whom to choose two for any one exposure. This policy regarding dive subjects was quite deliberate, in order not to distort results by selection more than was unavoidable.

Regarding the subject's nervous system, past medical history was explored carefully. All aspects of neurological function were examined pre- and post-dive, where possible with techniques of clinical examination modified to yield quantitative or semiquantitative results. EEG was routinely used, however, typically only from a single occipito-parietal channel. When a multi-lead clinical EEG was obtained post-dive by hospital, the report was invariably normal.

Relating to one of the 540 m dives the subjects were examined before and after by clinical psychologists. The hospital department concerned was independent of AMTE PL, and was well practiced in supplementing neurological diagnostic information about focal brain damage by appropriate behavioural tests. Results so obtained about 3 months after the hyperbaric exposure indicated an unequivocal "No change from control values" in the two subjects so examined. This was a particularly welcome result, as one of the subjects concerned had suffered what might have been a cerebral decompression episode at 11 m in that dive. It may be appropriate to sketch salient features of this event.

The depth independent decompression from 540 m continued almost uneventfully (slight knee pain in one man at 258 m, resolving without recompression) at 28 m per day on 0.4 bar oxygen, balance helium. At 11 m one subject reported blurring of vision, headache, some loss of touch sense, and paraesthesiae in one hand. The visual symptoms consisted of loss of detail and colour vision in the peripheral fields only, of both eyes. He could read with each eye separately using his central retina only. These symptoms and an accompanying frontal headache developed insidiously, and resolved on 7 m recompression

whilst breathing on oxygen enriched gas mixture for 20 minutes. The numbness and paraesthesiae affecting one hand were first reported at this point. A further 7 m stop of compression was carried out, followed by two more 20 minute periods of high oxygen breathing (PO_2 = 1.68 bar). Recovery was prompt and complete, and decompression was resumed after a 36 hours stay at the treatment depth of 25 m.

Post-dive neurological examination revealed symmetrically increased myotatic reflexes in both legs, slight weakness in the right shoulder girdle but was otherwise normal, including a multi-lead clinical EEG. An isotope brain scan showed decreased perfusion in an area of the right middle cerebral artery, which turned into increased flow two days later in the same region.

Thus on the history, therapeutic response and post-dive findings the diagnosis of a cerebral dysbaric event may safely be made. The origin of the offending gas is thrown into doubt by a preceeding and concurrent chest pain, a somewhat similar event at 177 m in a previous dive in the same subject, and by the extreme rarity of cerebral decompression sickness (as opposed to embolism) in heliox saturation diving. The significant point here, however, is that an independant specialist assessment by neuropsychological means performed 3 months after the end of a 540 m exposure and a cerebral dysbaric event, outlined no change from pre-dive test results.

The neuropsychological test procedure used consisted essentially of well known performance and other techniques, such as selected subtasks from the Wechsler. The key factor here is that the various procedures have been well calibrated in use on patients having a relevant range of systemic or localisable space occupying intracranial lesions. Thus the testing clinical psychologist and his team were well practised in correlating various features of their findings, often minor in nature, with different kinds of brain damage.

In general, for an absence of positive finding to be meaningful, the measurement techniques must be sensitive and well controlled. For this reason alone, I would like to register a plea for post-dive neuropsychological tests to be performed by expert clinical psychologists actively practising in a hospital environment alongside a department of diagnostic neurology.

Discussion

All important physiological parameters were shown to return to normal levels within two weeks after deep diving. For example the gain of the horizontal vestibulo-ocular reflex, (a quantitative measure of vestibular function), and some EEG components may require about this length of time (Ref 2, 3).

The need for this present workshop goes back to at last 15 years, specifically to Physalie III. The abundant EEG delta activity seen in that experiment provoked fears of metabolic (hypoxic?) brain damage in analogy with some frequently seen pathological processes (Ref 4). The maximum depth reached by Physalie III has since been almost doubled, and no evidence has emerged to substantiate this concern. Vigilance must, however, be maintained in future deep exposures. Under this heading of vigilance in the future, I would like to point at some topics possibly worthy of discussion.

1. Post-dive fatigue.

For a few days after some of the simulated dives of the series a general all pervading fatigue in the sense of lassitude, lack of motivation and general sparkle in the subjects was so strong as to almost amount to lethargy. Increased slow activity in the EEG reflecting episodes of decreased vigilance and drowsiness is often seen. A loss of haemoglobin after a 3-4 week long hyperoxic (PO_2= 0.4 bar) exposure, averaging 2.5 g / 100 ml when present, is not surprising. (Ref 1).

The above EEG finding is not more than a descriptor, the loss of haemoglobin may be one of of a number of causative mechanisms. Marked degree of lassitude is now widely accepted as a possible manifestation of decompression sickness, even though direct evidence is lacking. Thus even if it is an important post-dive effect, lassitude may be due to decompression and only possibly due to high pressure per se.

2. In-dive performance decrements.

A recent review (Ref 5) called attention to the fact that in spite of recent progress with compression techniques, there may be a residue of performance decrement refractory to further improvement. This impression was gained on surveying results obtained at a variety of research centres, compression profiles, depths and chamber gases. Such

"obligatory" decrement, if it exists, may be of the order of 10 %. If this concept is accepted, a deliberate decision process is needed to accept or reject such state of events for working dives.

The next step is to consider in relation to the human central nervous system whether a factor capable of lowering its functional efficiency is like alcohol, having effects completely reversible when the agent is withdrawn, or like hypoxia, is essentially undesirable. Again, the point made here begs vigilance, nothing more.

3. Cellular hypoxia.

The concept here is that by some mechanism the availability of oxygen (or a substrate, or the activity of a respiratory enzyme) at cellular level might be less than adequate under certain hyperbaric conditions. Thus the respiratory cycle would be broken at the point of oxygen utilization. McKenzie's work (Ref 6) with the adductor pollicis in the Deep Dive Series indicated that for muscle at least this does not happen. The time course of recovery from ischemic fatigue was unimpaired at 43 bar compared to pre-dive normal control values in the same individual. There are conflicting reports on a number of different preparations, however, and once again, continued vigilance is called for.

4. Neuronal functional redundancy.

This is greatest in non specific cerebro-cortical areas, where well over half the existing neuronal population is spare i.e. standing by. In the sensory system in general redundancy is thought to be about 50 %, meaning that the character of touch or the treshold value for two point discrimination would not be affected unless more than half of the available neuronal apparatus is blocked. At the level of spinal anterior grey matter in the motor system, redundancy is zero, as each motor neuron has its own exclusive set of muscle fibres to activate. In the muscle redundancy is represented by spare power, and a capacity for "orphaned" motor units to be "adopted" by neighbouring ones.

The concept of redundancy is crucial in the interpretation of any negative finding obtained on the CNS under circumstances when hostile environmental factors may be at work. The conclusion is yet again that continued vigilance is needed in spite of all the reassuring negative findings we have to date.

CONCLUSION

In summary recent (since 1976) British experience of 8 simulated dives 300-660 m, with 10 subjects indicates no physiological, neurological or psychological changes persisting beyond two weeks post-exposure. This is also true in a well documented case of cerebral dysbaric lesion sustained during decompression. Even though some early (Physalie III) fears were not borne out during the last 15 years, constant vigilance must be maintained by all concerned by deep diving.

A range of physiological, including neuro-physiological investigations should be routinely carried out on a before-and-after basis. A neuro-psychlogical test procedure performed by clinical psychologists should not be omitted, at least not until there is enough evidence that it is in fact superfluous.

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QUESTIONS AFTER DR TÖRÖK'S PAPER

Prof Aarli: One question for the record. The post-dive examination, was that three months after the dive?

Dr Török; In the case of the 540 metre dive subjects that I referred to specifically, it was after three months. Our usual routine is to do a physical examination, with some attention to neurological signs, immediately on emerging from the chamber and about one week later.

Dr Rostain: The comment on the Physalie III experiment. You said that probably the diver on Physalie III was hypoxic, is that correct?

Dr Török: I did not. I said that from the suddenly perceived abundant slow activity seen on the EEG, Ralph Brauer was quoted by, I think, Dr Fructus as having said that "cerebral asphyxia", (his words) was suspected on Physalie III. Now, my information is from this publication and, of course you can check that.

Dr Rostain: The partial pressure of oxygen during this dive was 600 m/bar. The interpretation done after this dive and the other dives carried out afterwards is that it was not hypoxia or asphyxia, but a phenomenon due to the fast speed of compression used in these dives. The divers descend 355 m in 2 hours. We have seen afterwards that the fast compressions without stages induces slow waves and microsleep, whereas with slower speeds of compression and stages at an intermediate depth, the microsleep and the slow waves were less.

FRENCH EXPERIENCE IN DEEP DIVING.

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Y. Giran, D.Lamy, J.C. Rostain.

INTRODUCTION

Our purpose is to present the French experience in deep diving as well as the pathological neurological observations related to deep diving. The C.E.M.A. (Captain Cousteau) and C.G. DORIS experience are not included here. Therefore, only the French Navy and the COMEX experience are presented.

We shall only consider the saturation diving experience deeper than the usual operational depth performed on a routine basis, which, in our opinion, represents the lowest limit of the so called deep diving in terms of depth limit.

In 1982, COMEX divers had performed 35 saturation bell runs during offshore operations in the 200-220 msw depth range (1.5 % of the total bell runs).

The unusual operational depth can therefore, be set at approximately 250 msw. This depth represents the lower limit of deep diving, for the time being, in our opinion. On the other hand, physiological changes usually appear in that depth range.

1 - MEDICAL INVESTIGATIONS OF FRENCH DIVERS:

1.1 Routine Basis

French diving regulations (1974) require for all divers deeper than 50 msw.:

- EEG with oculo-cardiac reflex stimulation
- Basic psychological evaluation.
- Dry chamber dive test on air at 70 msw with basic psychometric tests.
- Medical examination for divers every year by an approved physician.
- In the French navy, the EEG is performed on a four year basis.
- ENG is performed in dry divers in French Navy when they join deep diving unit.

1.2 Particular Usual Medical Investigations in Deep Dives

Most of the experimental or unusual deep dives performed in France and presented here below included a particular medico-physiological investigation including:

- Neurophysiological evaluation:
  EEG, tremor, reflexological study, ENG as well as stabilometry.
- Psychometric evaluation:
  On the other hand, many other studies were performed at the same time (pulmonary function tests, thermal studies, biological study . . .)

2 – CLINICAL NEUROLOGICAL OBSERVATIONS RELATED TO FRENCH EXPERIENCE IN DEEP DIVES

Only the post-dive observations are presented hereafter.

2.1 Comex Experience

165 divers have particiapted in COMEX deep dives.

- Two vestibular bends occured during these dives. Only one diver was still presenting, one year later, a slight residual peripheral dysfunction in his ENG. These two divers do not present any residual clinical symptoms and have resumed their professional activities. One is still working in COMEX.
- One diver had suffered from a vestibular bend before participating in a deep dive, without any clinical residual symptom. The predive ENG presented a residual aspect of peripheral nature. After the deep dive, a slight loss of equilibrium was observed. The ENG showed a probable slight central dysfunction which was spontaneously improved three weeks later as well as the clinical symptoms.
- After a 350 m dive, two divers experienced a transient neurological dysfunction:
  1. One diver had a reduced feeling in the right part of the body associated with a reduction of the abdominal reflex on the same side and an increase in slow waves on the left side EEG). Three weeks later, these changes had disappeared. The proposed diagnosis was transient brain dysfunction in the left hemisphere . . .
  2. In the second diver the following were observed:
     - slight difference in pupillary size,
     - abdominal reflex slightly reduced on the left side,
     - pathological plantar reflex on the left side,
     - EEG unchanged.

EXPOSURE / DEPTH	DRY CHAMBER EXPOSURES (MEN)	WET CHAMBER EXPOSURES (MEN)	OPEN SEA EXPOSURES	TOTAL EXPOSURES	TOTAL CHAMBER TIME (MEN x HOURS)
≥ 250 MSW	70	59	61	190	ABOUT 70 000 (> 2900 days)
≥ 300 MSW	67	46	32	145	
≥ 350 MSW	24	25	6	55	
≥ 400 MSW	20	14	6	40	
≥ 450 MSW	20	6	6	32	
≥ 500 MSW	8	2	4	14	
≥ 600 MSW	4	2	0	6	

Table 1. French experience in deep diving, deeper than 250 msw.

A second post-dive neurological examination was performed three weeks later and revealed that the reflex changes were still present, but the brainstem evoked potentials were normal. The proposed diagnosis was a probable small lesion in the brainstem.

A third neurological examination four weeks later was performed and no residual symptom was found.

The two divers were then declared fit to dive and particiapted in another deep dive. They were investigated after that deep dive and nothing particular was observed post dive by the neurological specialist.

2.2 French Navy Experience

25 divers have participated in deep dives.

One vestibular bend was observed during a deep dive and no residual effect observed. No neurological abnormality was found in the Navy deep divers. No EEG change was observed in these divers.

3 – NEUROLOGICAL AND PSYCHOMETRIC COMPLEMENTARY INVESTIGATION RESULTS

3.1. Neurophysiological Study

3.1.1 Introduction

The High Pressure Nervous Syndrome has been studied on 63 subjects during dives to depths from 300 msw to 610 msw (table 2). Among these subjects, seven have performed several dives (table 3).

These dives have been carried out with helium oxygen mixture (12 dives with 32 divers) and helium-nitrogen-oxygen mixture (7 dives with 31 divers) (table 4).

3.1.2 HPNS Symptons During Dives.

The symptoms of HPNS which appeared generally between 200 and 300 msw were consequently presented in these subjects at different levels according to the individual susceptibility, the compression methods and the breathing mixture used.

With helium-oxygen mixture, from 300 msw up to 610 msw, every subject had tremor and changes in electroencephalographic activities:
– increase of slow EEG activities in frontal area area of the skull;
– decrease in fast EEG activities in all the skull.

In several subjects, for depths deeper than 400 msw, there were microsleep, dysmetria, myoclonia.

With helium-nitrogen-oxygen mixture, from 300 msw up to 450 msw, the subjects had never significant tremor or other clinical symptoms of HPNS except microsleep.

Generally, these symptoms increased with depth, did not disappear during long stays at 300 msw, 400 msw, 450 msw, 500 msw or at stays at 610 msw and presented different evolution according to the subjects. These symptoms decreased during decompression and often disappeared from 200 msw onward. Sometimes, they disappeared after the end of decompression and rarely several days after (2 cases between 2 and 3 weeks after). The persistance of the symptoms was seen generally during decompression from long saturation dives.

Consequently, with the methods of analysis used in our neurophysiological studies, we have never seen a persistence of any EEG changes or nervous disturbances such as tremor, dysmetria or myoclonia.

HPNS STUDIES FOR DIVES $\geq$ 300 msw

DEPTHS	NUMBER OF DIVERS
300 msw	63
400 msw	40
450 msw	26
500 msw	10
610 msw	6

Table 2: *Number of divers who performed dives between 300 msw and 610 msw with HPNS studies.*

DIVES FROM 300 msw to 600 msw

	$He-O_2$	$He-N_2-O_2$
Number of dives	12	7
Number of divers	32	31

Table 3: *Number of dives and divers in Heliumoxygen and Helium-nitrogen-oxygen mixtures.*

3.1.3. HPNS Symptons During Repetitive Dives.
Seven subjects have performed 2 dives at least at depths of 300 msw up to 610 msw (table 4).

Six of them have carried out dives to 610 m in helium-oxygen mixture (Physalie VI, Sagittaire V, Entex 9) (Rostain et Naquet 1974, 1978; Fructus et al 1976) and other deep dives between 300 msw and 500 msw in helium-oxygen or in helium-nitrogen-oxygen mixtures. The delay between two dives was at least 1 month to 3 years.

The results obtained from these repetitive dives showed:
- no enhancement of HPNS between two analog dives;
- a HPNS less intense in a second dive deeper than the first but carried out with methods including slower speeds of compression than the first;
- a disappearance of clinical symptoms of HPNS during a second dive performed with helium-nitrogen-oxygen mixture compared to the first one carried out with helium-oxygen mixture.

Consequently the methods of analysis used in our neurophysiological studies during repetitive dives did not indicate an enhancement of HPNS symptoms from one dive to another.

	REPETITIVE DIVES FOR DEPTHS ≥ 300 msw				
DIVERS	300m	400m	450m	500m	610m
CHE				PHY V	PHY VI
GAU	SAG III COR III			CEMA	PHY VI
BOU	COR I et II				SAG IV
JOU	COR I et II				SAG IV
RAU		JAN IV			ENT IX
OHR			ENT V		ENT IX
LEN		JAN IV	ENT VIII		

Table 4: *Number of dives performed between 300 and 610 msw by the same divers.*

3.1.4 Longitudinal Studies of the Divers.
A longitudinal study of health, physical, psychometric and intellectual capacities and EEG was performed during several years on 21 divers (LeMaire et al. 1979) and 10 divers were examined several times for 180 metre dives (Rostain et al. 1981, 1983).

3.2. Psychometric Study in Deep Dives.
The psycometric tests performed in 46 deep divers show that:
- transient changes are observed during the dive period and go back to surface values at the end of decompression. Reversibility appears to be complete.
- only four subjects were investigated several times and no long term effects were observed.

3.3 Electronystagmography
ENG was performed in eight subjects exposed to 46 ATA, pre, during, and post-dive (Renon et al 1983).

None of the eight divers suffered from DCS during decompression. No residual abnormality was observed post-dive despite some changes observed at depth.

4 – PSYCHOLOGICAL ENQUIRY IN DEEP DIVERS

25 Navy deep divers who were involved in dives deeper than 250 msw received an anonymous psychological questionnaire and 23 out of 25 filled in that questionnaire.

According to this study no particular psychological residual effect related to their deep exposures could be found.

5 – DISCUSSION

Despite various changes commonly observed during deep dives performed in France, the French experience in potential neurological effect from deep diving is extremely modest despite the number of exposures to great depth.

It can be summarized as follows:

1. Direct neurological consequences of deep decompression sickness: one subject out of three cases observed in deep diving still presents

a residual dysfunction of his inner ear. Another had his ENG test deteriorated during a deep dive (vestibular bend five years ago)

2. Direct neurological consequences of deep diving: No permanent residual neurological effect was observed in 190 men who particiapated in diving operations deeper than 250 msw up to 610 msw. Two transient residual effects were observed during the neurological examinations performed. These consequences spontaneously disappeared six weeks after the dive.

Our experience indicates that the changes observed in deep diving are apparently 100 % post-dive reversible in the following investigations:

- Neurophysiological study: EEG, tremor, reflexology.
- Psychometric study.
- ENG.
- Clinical neurological investigation.

On the other hand it appears that only 6 out of the 190 deep divers (3.1 %) stopped diving after their experience in deep diving:

- 3 medical doctors
- 2 engineers
- 1 diver who had already got a new job in the company before the dive.

None of the divers who participated in deep dives stopped diving due to neurological consequences of these deep dives.

6 – CONCLUSION

Medical records of 190 Frency navy and COMEX divers who participated in saturation diving deeper than 250 msw have been analysed in order to study the eventual long term neurological consequences of deep diving.

Apart from two cases of slight residual vestibular dysfunction without clinical symtomatology, no long term neurological defect due to deep diving has been observed so far. If deep diving might be not really anodyne, it has, however, never been demonstrated that deep diving could be responsible for permanent residual neurological damage.

Nevertheless, it is our opinion that a particular attention should still be paid on the eventual long term consequences of deep diving.

To be efficiently and safely performed, particular attention should be paid to the following human factor aspects in deep diving:

1) Appropriate selection essentially based on:
   - professional experience in deep diving (progressivity in depth)
   - high standard of medical fitness
   - strong psychological motivation
2) Adapted medico-physiological follow up, pre, during and post-dive. This could be quite easy to control during the onshore dive training which is probably necessary for great depth operation.
3) Medical supervision onboard. This supervision is performed on a routine basis in the French Navy.
4) Adapted time schedule of deep dive (total chamber time, in water lock-out time procedures).
5) Distribution of deep dive/year/man.
6) Necessary rest period after deep diving.

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QUESTIONS AFTER DR GIRAN'S/DR ROSTAIN'S PAPER.

Dr Molvær: What was the time difference between recognising these vestibular bends and the treatment? What kind of treatment did they get?

Dr Giran: There were two cases which occured during decompression some years ago and probably the treatment was applied immediately by recompression. One was during Physalie VI and the other one I cannot remember, but apparently the treatment was started immediately.

Dr Molvær: In spite of this, they had residual effects?

Dr Giran: Yes.

Prof Bennett: I think the labyrinthine problem has virtually been eliminated today. This was a period, in the 1960's, when we were just getting involved in deep diving, when we were making too rapid a decompression, too deep, and ending up with labyrinthine decompression sickness. We now decompress very slowly and I do not think anybody would get labyrinthine bends today in deep oxyhelium and trimix diving.

Dr Török: Could I please make the point that something that gives the clinical picture of a vestibular decompression sickness might well not be labyrinthine. You might easily find 4 or 5 small but nevertheless, obvious mid-brain signs which clearly makes it a mid-brain type of vestibular lesion.

Dr Giran: Yes, that is our opinion. We have now changed our treatment concerning vestibular and neurological bends and we consider that vestibular bends are to be regarded as neurological bends, and treated as such.

Dr Nome: Is the medico-physiological follow-up examination performed in accordance with a standardized protocol? Secondly, do plans exist for continuous longitudinal study of the deep divers you have been telling us about?

Dr Giran: On the first question, I would say that on a twenty year history it is quite difficult to have a standard protocol. The knowledge gained since the beginning has introduced changes. Secondly, no, no plans.

Dr Nome: We have been fairly consistent over the last four or five

years on what examination we find value in doing, but no such plans for a standardised protocol exist in France, so far as you know?

Dr Giran: Yes, it does more or less, when a deep dive is to be performed. The divers are examined and advice is asked from different specialists such as neurologists, ENT specialists and lung function specialists.

Dr Brubakk: You found 2 transient neurological changes in 190 divers. How many of these 190 divers were investigated by a neurologist within one week of coming out of the chamber?

Dr Giran: Very few. They have been examined on a routine basis by a physician who performed a medical and basic neurological examination. If there is something which is apparently abnormal, the physician can seek specialist advice.

Prof Bennett: That is all very well in a country with free medicine, but in some countries like in the United States, for example, it costs between M 9000 and M 12000 for testing three divers. You need to be pretty sure you are looking for something if you are spending this kind of money on a regular basis. So, we tend to wait for people to say they have something wrong and all divers in the Duke series know they must come and tell us of the slightest thing they think aberrant. Nevertheless, we still do a certain battery of tests on them at regular intervals.

Dr Brubakk: What you are doing now is throwing up some sort of smokescreen. The point, of course, is that we have investigated 23 divers and, out of them, 4 have focal neurological signs. This is a very high percentage. If Dr Giran's data was right that 190 divers were investigated and only 2 had focal signs, then that would mean that we were just unlucky.

Dr Giran: We did not ask a gynaecologist's advice too.

Dr Brubakk: I think this is a rather flippant remark.

Dr Giran: He probably would have found a gynaecomastia, something like that, in those subjects eventually, why not. It depends on what we are looking at.

Dr Brubakk: Your organisation probably has more experience of deep diving than anyone else in this room. The point I am trying to make is that, by more or less chance, we found a very high percentage of focal changes which suggest probably some vascular effect. We have

to establish if this is something that is common, or is it something that is extremely rare.

Dr Giran: Most of our divers are still divers, still healthy, for 10 or 15 years some of them, still working in COMEX. They did not disappear, they did not claim, or put COMEX in the court of law. This makes a quite good record, an observation which is respectable and probably reasonable.

Prof Aarli: Did I understand you correctly? Did you mean that if there had been positive neurological findings, then either the doctor who examined the patient or the patient (i.e. the person who dived himself) would have detected it? If so, I think you underestimate the French neurologists who are among the best in Europe.

Dr Giran: My basic qualification as MD seems to allow me to make a basic neurological examination in a diver and, if there is an abnormal plantar reflex, be sure, I am probably able to find it. If there is an abnormal abdominal reflex, I can probably find it also and look at the cranial nerves as well. I think that it is my responsiblity as an MD in France to do this basic neurological examination, and then, and only then to ask a specialist his advice. It is the way we practice in France.

Dr Shields: Surely, we have already heard an admission that in the early dives in the US Navy series, the neurological tests were not carried out as rigorously as would be carried out now. I do not think Dr Giran could make firm comment on the whole of the 200 or more dives COMEX have performed. Dr Brubakk is suggesting that, in a very recent series in Norway where rigorous testing has been carried out, they have found a high incidence of problems. The rest of us, who have been involved in diving for a rather longer period, cannot make the contrary claim, as in the earlier days the examinations were not carried out to the same standard.

Prof Bennett: Well, I hear great stores of "rigorous". What we are talking about is finding some very basic neurological changes and those examinations have been performed on a lot of the dives I am familiar with. A physician is supposed to have checked the divers when they come out of the chambers, including a full neurological. Certainly, in all Duke dives, the physician takes the diver into an examination room, and examines him. I believe that was done at RNPL, or certainly should have been done. So, I do not see any issue here. I think it is clear that something has been found which those of

us in deep diving over the last twenty years or more, have not seen in any quantity. Now, whether this is aberrant because of the nature of the profile, the mixture, or the men, we do not know. That means that we certainly need to go on making these tests on anybody who is doing deep diving.

*Dr Giran:*That was the second point of my conclusion: medico-physiological follow-up, pre, during and post-dive.

General Discussion on the British and French Experience

Prof Aarli opened the discussion by commenting upon the different attitudes: The first being that deep diving, when properly performed is probably relatively safe and the second, which is rather more cautious, that we do not know the possible consequences of deep diving over 10 to 15 years. He felt that there appears to be some agreement that transitory brain dysfunction does occur, but we disagree on how frequently it occurs. There also appears to be general agreement that one single dive does not lead to irreversible effects on the nervous system. He commented on the analogy to boxing, which can also produce transitory brain dysfunction. The cumulative nature of the damage in boxing was the question we were addressing in diving, and were the compression and decompression experienced by a diver analagous to the fights of a boxer? He also felt the importance of the diving protocols used over the years and he concluded that he is unsure how safe deep diving is after 10-15 years.

Prof Bennett replied that we could only wait and see as at present the information upon which to make any decision is not available. He wished that people would maintain an open mind on the subject and allow deep diving to continue. He also believed that if repeated exposure is a factor, then shallow air diving must be taken into account as well, as recorded cases of "punch-drunk" divers do occur after shallow diving. He firmly believed that it is impossible to be selective about deep diving alone and all diving, including shallow air diving, must be considered together in any causative mechanism.

Prof McCallum asked if the "punch-drunk" divers have had neurological decompression sickness and was told that certain sponge divers may have accumulated a neurological insult from ignoring the normal diving tables. He, therefore, reminded the meeting that this was the result of bad diving practice provoking neurological decompression sickness, and not a compression or bottom time phenomenon.

Dr James explained that his reservations about very deep diving in the commercial situation arose from the unlikelihood of being able to control the conditions in an open sea situation. He felt that if it is possible to demonstrate neurological dysfunction actually at depth, then it

is questionable whether this procedure could ever be used outside the laboratory in commercial situations.

Prof Bennett wished to remind the meeting that what we have been discussing is a series of experimental dives which have been consistently improving the performance of the divers at progressively deeper depths, for example to greater than 600 metres, with only a 10 % decrement in performance and no EEG changes. The French have carried out deep water dives to 600 metres and if you choose a depth of 450-500 metres, he felt that you would still be well within safe limits. He also believed that with a well trained crew and proper procedures, these dives could be carried out at sea. He believed that proper training and equipment were crucial to the success of open water dives. He wished to comment on the fatigue and malaise that occurs in some dives and not in others. He speculated that it may be due to the confinement of young, fit men in a small chamber, where the possibilities to keep in shape are few. He, therefore, felt that it was no surprise that the divers were fatigued and their muscles were out of condition. The loss of cardiac efficiency during the dive is apparent when they resume their running after the dive. This gradually returns to normal. This is a problem with any confinement and could be alleviated by giving artifical sunlight and treadmill working.

Prof McCallum remarked that fatigue appears in compressed air workers, particularly in the engineers and agents on compressed air sites, but did not know what the cause may be.

Dr Thalmann asked Prof Aarli if the abnormal EEG's seen in the divers reported earlier had returned to normal and was informed that two of the four divers remained to be examined, but the other two now had normal EEG's and reflexes. He, therefore, felt that the implication was that the post-dive neurological examinations appear to be inadequate because a weak reflex may not be noted.

Prof Aarli agreed that this was a well known phenomenon in clinical neurology and cited the case of patients with multiple sclerosis who developed symptoms that may later disappear, but he reminded the meeting that the physical damage remains.

Dr Cox asked what advice was given to the divers who exhibited neurological changes after the NUTEC dives. *Dr Molvær* replied that they had been advised to continue diving as before. *Dr Giran* added that once they had been cleared by NUTEC to carry on diving,

COMEX required another neurological examination before they would clear them as well. This was done and the neurological examination produced nothing abnormal, so they were allowed to go back to diving. They considered that they were responsible for the divers welfare and therefore took the opportunity for a further examination. He remarked that it is possible that the two divers who were not examined on the follow-up were missed because the company was not informed of the need for a further examiniation.

Prof Elliott asked Dr Giran which of the 2 divers out of the 4 he was referring to, as he felt that there was a potential ambiguity. Was he to understand that COMEX were unaware that it was 2 more of their divers who had neurological symptoms? *Dr Giran* agreed that it was 2 divers on the open sea dives that COMEX did not know about.

Prof Bennett informed the meeting that to carry out experimental dives in the USA requires that the experiments be reviewed by a 20-man peer committee of people from all disciplines within the Medical Centre, including lawyers and administrators. This committee reviews the protocol and decides if it is acceptable. He felt it was important to note this, as all the Duke dives and Pennsylvania dives had been through such a procedure successfully. This is a rigorous review which turns down a good deal of proposed human experimentation.

Dr Brubakk believed that any discussion of the normality of divers was premature as the long-term consequences of deep diving are still unknown. He posed the question of how the diver is treated if he has to retire due to a diving-related illness. Is it his problem or is it an industry's problem? Is it right to single out one group of divers and leave out the majority?

Prof Bennett believed that, if the findings that have been found to date are transient the only real question was are they cumulative in the long run? He felt that this was a difficult question to answer and would be reluctant to stop anyone diving on the basis of such information. *Dr Török* suggested that the diver should be stopped until the symptoms have disappeared in order to ensure that the findings were transient. *Prof McCallum* reiterated that these transient symptoms may be associated with permanent damage.

Dr Thalmann commented that there seems to be a very extensive discussion on relatively little information on 4 divers who have now returned to normal. He pointed out that the US Navy allows divers who

have had overt neurological decompression sickness, but who have been treated successfully, to return to diving in a month. He believed that the same kind of tests carried out on bounce divers would reveal a much greater incidence of gross abnormalities. The current belief is that, if you treat the divers' symptoms successfully and a month later the neurological examination is clear, the diver is allowed to return to diving. He felt that the neurological findings detected in deep dives were very minor compared with the effects of decompression sickness on shallow divers who are allowed to continue diving.

Prof McCallum thought we were moving into another area, but felt it could be usefully discussed. *Dr James* wished it to be noted that there is evidence that relatively minor dysfunction which is associated with decompression sickness can lead to irreversible changes. *Prof Aarli* was surprised by the reaction as the findings he reported were minor and, as far as he knew, were transitory. His interest was what happens in the brain during the dive and how long will the changes last within the dive, and he did not believe it was anything to get too excited about.

Prof Elliott was concerned that the aetiology of the transient symptoms in the 4 divers had not been addressed, be it an unmasking phenomenon or decompression sickness. He wished to ask if there was any evidence to disprove decompression sickness as the possible cause. *Prof Aarli* admitted that Prof Elliott had a point, but the symptomatology appeared to start during the compression phase according to Dr Værnes's data. It is impossible to dismiss such a possibility although the EEG did not change on decompression.

Prof Bennett asked Prof Aarli to be more specific about the EEG changes. Were the theta activity similar to those detected on compression, or something new? He replied that the findings were of interhemispheral differences, with more slow waves in the left hemisphere than the right. *Prof Bennett* then asked Dr Værnes if he saw the same sort of difference on compression or at depth? He was told that the FFT-EEG was normal among all 6 subjects post-dive. A change in the EEG was recorded for one diver during the compression and the second diver developed an abnormal EEG during saturation. These correlated with the later symptomatology that Prof Aarli detected.

Prof Donald asked the meeting's opinion on whether enough animal work has been done on this subject? Would this be a useful adjunct to

human experimentation? He felt that more animal work might help in increasing the safety of deep diving by revealing new dangers before they become apparent in men. *Prof Bennett* replied that the Japanese were carrying out extensive animal work using the cat as a model to great depths, although the pathology might not be well covered. *Prof Donald* was concerned that the cat was a rather volatile animal on which to carry out HPNS research but this was countered by *Prof Bennett* who said that the cat is a very good model because it exhibits most of the symptoms such as tremor, vomiting and EEG changes seen in humans.

Dr Værnes wished to return to the problem of a common protocol and reminded the meeting that most of the researchers in the USA, Norway and France have been doing the same sort of things pre- and post-dive. So, he believed we were talking about the same neurological parameters. He felt that we should decide what are the valid CNS parameters which should be looked at in long term studies, because at present we do not know what each laboratory is doing.

Dr Rostain commented that he believed the monkey to be the best model because the monkey produces almost identical HPNS to man. If the cat can produce symptoms analogous to man, then perhaps it has its uses, but the monkey, although expensive, is the best.

Dr Brubakk believed that the tiredness symptom present in many divers could be due to some change in the CO2 sensitivity, which is manifest as shallow, rapid breathing. He recounted the experiences of one of the diver who had been running all his life, who found that, after the dive, he was unable to run 500 metres without becoming severely out of breath. He felt it was not just a product of incarceration, because another diver who had very little HPNS took almost 5 months to return to his previous running form. He believed there are other problems that we do not understand. *Prof Bennett* remarked that the suggestion of some unknown cause is premature. We should repeat the dive exactly, but without pressure, and see what the effects were. He felt that the long decompression where the subjects are sitting around could be the cause. *Dr Nome* agreed with Prof Bennett, but believed that there were other factors involved as he had experience of examining divers who dive to 250-300 metres and the degree of fatigue appears to be unrelated to the work carried out on the dive. He, therefore,

believed that maintaining one's cardiac output does not prevent the feeling of tiredness on reaching surface.

Dr Török believed that each man was variable in his response. A subject diving in the same chamber on similar dives would feel fatigued on one and not on another for no obvious reason. He asked if it could be a decompression effect. *Dr Peterson* reminded the meeting that any number of factors will produce fatigue but suggested that possibly the high pO2 level, with its associated decrease in red cell mass, could be a factor as this also occurs in shallow nitrox saturation diving. It may, therefore, not be a neurological problem.

Dr Baddeley remarked that he felt overly fatigued after a 30 metre dive when the level of exercise had not been sufficient to cause this.

Dr Ørnhagen speculated that possibly the lung may be responsible because of the positive or negative pressure breathing promoting undue fatigue, or possibly some chemical component within the body. He enquired if anyone had a method of measuring such fatigue and he speculated that perhaps it could be due to decompression induced bubbles interfering with lung function. He concluded that the probable cause would be mysterious until the techniques of measuring and investigating the fatigue were available.

Prof McCallum commented that boredom produces fatigue, but *Dr Baddeley* replied that in the case he mentioned boredom was not a problem. *Prof Donald* reported that in the older dives, decompression fatigue could be attributed to bubbles brought about by a decompression that was close to inducing decompression sickness.

Dr Shields believed that we were talking about two different things. In a study carried out at Aberdeen concerning the diver's lifestyle, the findings indicated that the diver becomes more unfit offshore than onshore, the reverse of what was expected. This could be due to the long enforced inactivity. The undue fatigue associated with a short dive has been taken by many to be a warning of incipient decompression sickness. *Prof Bennett* commented that during a series of 200–300 ft air dives, he found that he experienced undue fatigue and associated it with the tables that they were testing which were producing silent bubbles and occasional decompression sickness, but he believed that this was a different phenomenon from the fatigue experienced on a long saturation decompression.

Dr Ørnhagen suggested that the undue fatigue was a warning signal

to the body to avoid further work because of the increased respiratory load experienced during deep dives. The body may react to the extra load by inducing a fatigue or reduce further activity. *Dr Thalmann* reported on a series of 60 ft 5-day bottom dives. In 7 out of 8 dives, the divers complained of fatigue which would last 1-2 days. Although the divers worked very hard during the dives, they did not report being tired immediately following the decompression, but after an overnight sleep they reported being "wiped out" the next day. He commented that the divers who took part in experiments done at Buffalo, in which maximal work was carried out at 200 ft did not feel overly fatigued. He, therefore, believed that the fatigue seen post-dive was a subtle form of decompression sickness, and his view was reinforced by the experiences on the 8th dive where, because one of the divers had decompression sickness, the profile was slowed by 8 hours and the divers did not feel fatigued at all.

PATHOLOGICAL FINDINGS IN THE CENTRAL NERVOUS SYSTEM AFTER DECOMPRESSION SICKNESS

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Introduction

The central nervous system with especial reference to the brain is particularly sensitive to a wide range of insults which range from toxic to physical agents. Such toxic substances range from heavy metals giving encephalopathies to solvents which may provoke psychological changes. The physical agents may be considered those which give rise to vascular damage by obstruction of the lumen by embolism or endothelial injury with subsequent platelet aggregation, and damage throughout the vessel wall. The changes resulting from toxic agents may usually be reversed when such substances are removed from the environment, and some return to normality achieved. However, physical agents usually induce some permanent and irreversible change.

Possible factors implicated

Within the complex of physical insults lies the effect of decompression sickness. The major investigation of the effects of decompression sickness since the work of Boycott et al in 1908 has been devoted to the effects on the spinal cord, but the clinical effects of decompression sickness have not been fully appreciated until recently. This is undoubtedly due to the clinically more apparant manifestations shown by the cord than the brain. In addition different mechanisms are involved as the mechanism in the cord is essentially that of venous infarction as described by Elliott et al in 1974. The effects on the brain are often major and result from the ingress of gas following barotrauma. It is nevertheless surprising that there is not some multi-organ involvement following an episode of decompression sickness. In this context, it is, therefore, necessary for the purposes of considering the long term

effects of decompression sickness to examine the effects on the body as a whole, with some tissues acting as specific target organs. Of such tissues, bone is recognised as one particularily susceptible to dysbarism. For this it is relevant to consider the observations of Elliott who in 1971, who found that there was a 5 % incidence of bone infarction in Royal Navy clearance divers subject to inadequate decompression schedules. Whatever mechanism is considered as causing such lesions, there must have been at the time a circulation in which there was some intravascular component to give an insult to areas of bone and subsequent infarction, although in these cases the bubble may be of prime importance. The relevance of this has to be related to the data obtained by Evans et al in 1970 who identified bubbles within the circulation of divers undergoing well recognized decompression schedules, by Doppler techniques. This could lend support to a view that there are cumulative effects of small intravascular bubbles, giving some vascular damage.

Elliott et al in 1973 suggested a vascular component in decompression sickness which involved platelet aggregation, and on this, Bove in 1980 and 1982, based a rationale of drug therapy for treatment of decompression sickness. Haymaker in 1950 implicated fat as a part of the spectrum of decompression sickness agents, and this is readily recognized in autopsy material in which fat embolism is identified in end-arteries or arterioles such as those of the kidney (fig 1). This was reviewed by Pauley et al in 1970 who considered lipids to be important in the pathogenesis of decompression sickness.

From the teleological argument any destabilising agent within the circulation can give rise to intimal damage. The result is well recognized as being non-specific and may be related to some change in the electrostatic polarity of endothelial cells by minute gas bubbles or lipids, and this is of especial relevance to the lesions recognized and provoked by the hyperbaric environment. This may be considered as analogus to the development of atheroma described by Adams in 1973 and later by Woolf in 1978 who found increased endothelial turnover in areas with foreign material in cases of decompression sickness (fig 2). In the long term this is followed by transport of lipids through the endothelium into the artery wall.

Irrefutable evidence that such mechanism occurs as a result of a diving environment is at present lacking, although Calder in 1980 in a

series of 8 divers identified some changes in the walls of small arterioles of the white matter of the brain (fig 3). In addition, corpora amylacia were identified adjacent to some of these vessels. Of these only 5 showed changes. Three were in helium saturation and two on air at the time of death. Three showed no changes and two of these were from a saturation environment, and one was an air diver who died as a result of a surface accident. It is not yet possible to confirm or refute the possible significance of this observation, until age/sex matched cases from a normal population have been examined. Best in 1983 stated that such changes may be seen in autopsy material, but the age is higher. This may be tentatively extrapolated as to suggest an advancement of the normal degenerative processes of blood vessels.

However, Rozsahegyi in 1959 suggested that there could be changes in cerebral function resulting from decompression sickness, and several cases were subsequently analysed by Elinskij in 1968 relating diving histories to degradation of cerebral function. Værnes et al in 1982 found by the use of discriminating psychometric tests, changes in the nervous system function after "near miss" accidents. The fact that this is little recognized may, in part, be explained by the large volume of redundant brain and the ability to compensate for areas of functional loss.

Damage to the spinal cord, however, is better recognized, with especial reference to animal experiments by Palmer et al in 1975. These obviously suffered from the lack of clinical correlation. Cases of neurological damage to spinal cord in man which have been successfully evaluated are few, and those subject to histological examination sparse. Therefore, to achieve a meaningful pattern of changes, it is necessary to interrelate experimental and human autopsy material to achieve a reasoned sequence of events.

Nevertheless, the observation remains that good clinical recovery can be made following severe neurological damage to the spinal cord, as illustrated by Palmer et al 1981, (fig 4). In this there was a loss of the facilus gracilis and lateral cortico-spinal tracts with little residual clinical evidence of damage.

Patho-physiology.

Much of the material which becomes available as the result of operational diving accidents is unsuitable for detailed histological examina-

tion. This is due to gross artefact created by explosive or non-therapeutic decompression.

Within 24 hours of the last decompression, in which symptoms are induced infarction occurs with oedema and early degeneration of myelin, which surrounds small blood vessels which do not display sufficient characteristics to be identified as arterioles or venules. The vessel walls are frequently necrotic surrounded by a margin of eosinophilic proteinaceous material. Myelin sheaths are balooned and some contain swollen axons. There is always a definite boundary separating the infarcted area from the normal myelin. The oedema also affects the grey matter of the dorsal horn, where the nerve cells become basophilic and shrunken, with eosinophilic cytoplasm. The vessels adjacent to the oedema in both white and grey matter show congestion. In addition included in the adjacent oedema accumulations of PAS positive spheroidal bodies accumulate which are up to 25 microns in greatest dimension (fig 5), and similar to those found adjacent to the cerebral vessels.

In the period from 1 to 4 weeks there is invasion of lipid phagocytes, and adjacent nerve fibres undergo Wallerian degeneration. Within the areas of necrosis blood vessels become hyperplastic with hyalinisation. Occasional lymphocytes are present adjacent to the blood vessels.

Further organisation of infarcts results in the disappearance of lipid phagocytes, with a microglial and astrocytic reaction becoming prominent. Axons may be identified in longitudinal sections of old lesions, some of which are myelinated, but oligodendroglia are not a feature.

In addition where there are small old focal lesions there may be secondary tract degeneration above and below, together with small gliotic scars which are present adjacent to the small vessels which have undergone hyaline change.

Discussion

In the most controlled decompressions it is possible to induce and identify bubbles in the white matter of the cord (fig 6). When the margins of such bubbles are closely examined using the Masson Trichrome stain, a small rim of connective tissue is identified (fig 7). Dispersal of a bubble by recompression would therefore suggest that some remnant of connective tissue would remain, and cumulation may give rise to local pressure effects.

In the more developed stage of the lesions, the appearances are consistent with the arrest of blood flow, with perivascular oedema, hemorrhage and foci of tissue necrosis. Although this may affect grey matter, with the development of histological neuronal changes, this appears to resolve. These changes are in accord with the experimental work of Wright et al in 1974, who found such changes were reversed within 14 days.

The presence of the spheroidal bodies adjacent to blood vessels is characteristic of vascular damage and Olsson et al in 1970 regarded them as a delicate indicator of vascular damage when other changes are not apparent.

On present available human data it is not possible to categorically relate the distribution of cord lesions to anatomical relationships of the arterial blood supply, but experimental work by Palmer et al in 1975 on goats shows that this is closely associated. This is pointed out by Zulch et al in 1970 in that there are three watershed zones of the spinal cord of man. The watershed areas are those most likely to suffer circulatory depletion because the tissue is at the end of the vascular supply from a major artery supply, and at a frontier zone against a region of cord supplied by another major artery. As these vessels are end arteries, there are no intramedullary anastomoses. The outer zone of the cord is supplied by the coronal arteries, of which the ventral two thirds come from the anterior spinal artery, and the dorsal third by two posterior arteries. An anterior spinal artery supplies the anterior horns, the area of the middle horn and the ventral zone of the posterior horn, together with parts of the surrounding white matter. Thus infarction of the cord, both in human and experimental materials, would appear to be in a closely related distribution to the arterial supply.

There is an opposing view of Hallenbeck et al in 1973 and Elliott et al in 1974 who suggest that the lesions are the result of venous stasis. However, the lesions in human material are similar to those described by Wolman et al in 1967 which were due to arterial embolism and which were believed to arise from the aorta. Thus, the features described by Elliott et al of venous stasis may contribute to, but are not sufficient to account for, the distribution of the pathological changes. Personal observations of divers, who die within half an hour of decompression show, the presence of bubbles both in arteries and veins and this could well account for the pathogenesis of the infarcts.

Conclusions.

There is undoubted ability of the cord to functionally recover, but there remains the fact that there is little, if any, regeneration of the cellular components which in the main are replaced by gliosis. Swash in 1983, by the use of electromyographic studies has shown that in recovered bends there is residual damage, not detectable by the routine clinical techniques. This must be regarded as an "iceberg phenomenon" in which there remains considerable underlying and undetected damage to tissues.

Extrapolating the local effects on the cord to the brain, there is every reason to suspect that in the long term there is a probability that both vessel and white matter damage may occur.

The fact that gliosis is the end result of infarction is demonstrated in figure 8, where the posterior columns are entirely replaced in a long term "recovery"

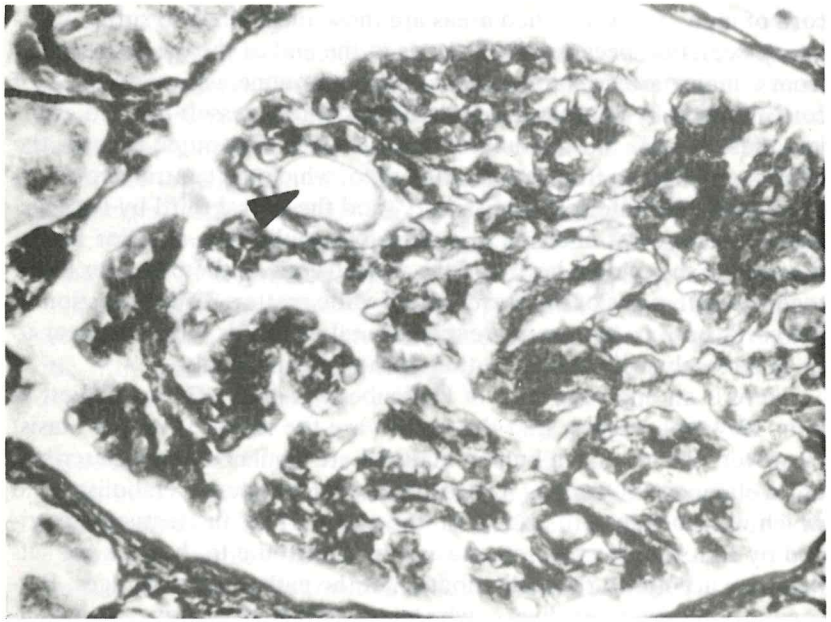

Fig 1. Fat embolism in afferent arteriole of kidney following acute decompression sickness. H&E x 120.

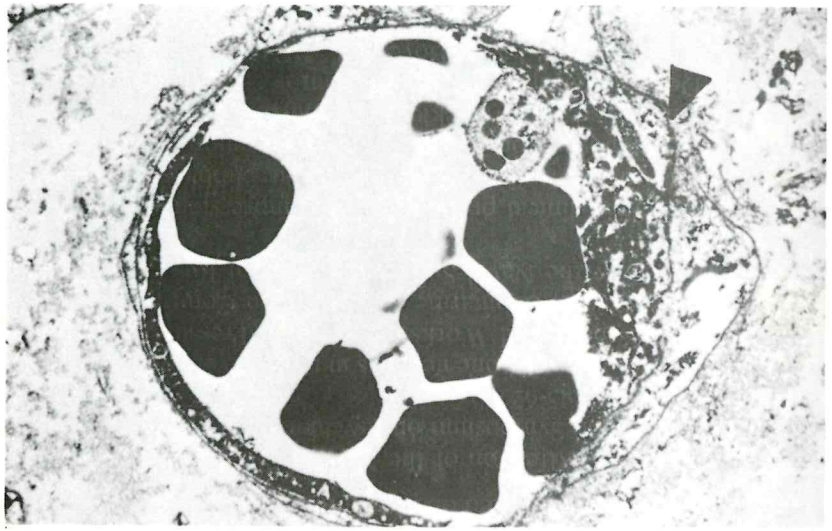

Fig 2. Electron microphotograph of vessel wall showing platelet aggregation and early destruction of endothelium x 2400.

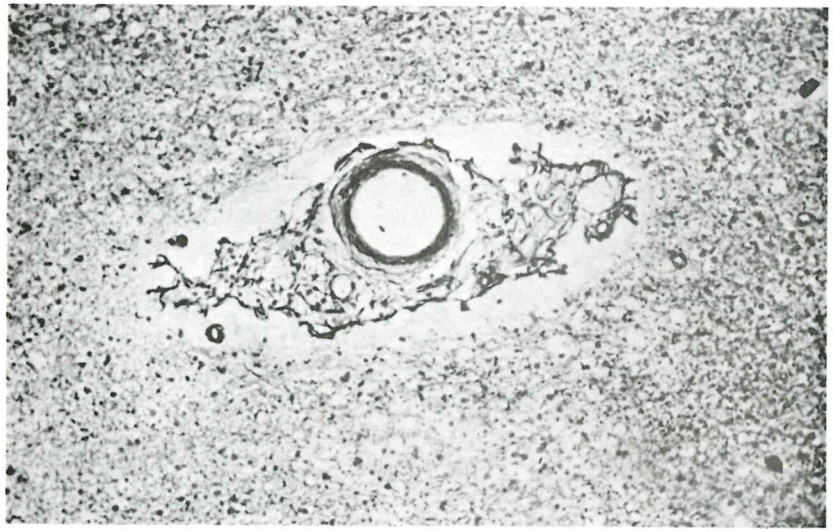

Fig 3. Hyalinised arteriole of white matter of brain H&E x 320.

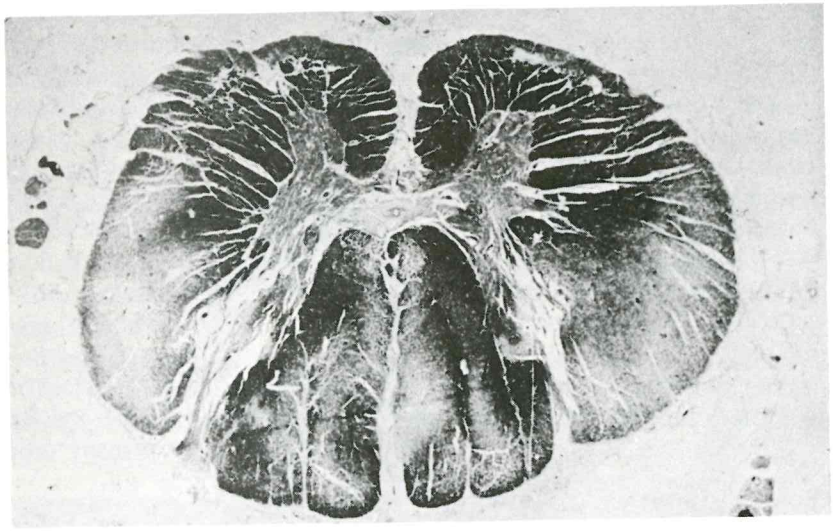

Fig 4. Transverse section of cord showing degeneration of lateral and dorsal columns. Methasol fast blue x 12.

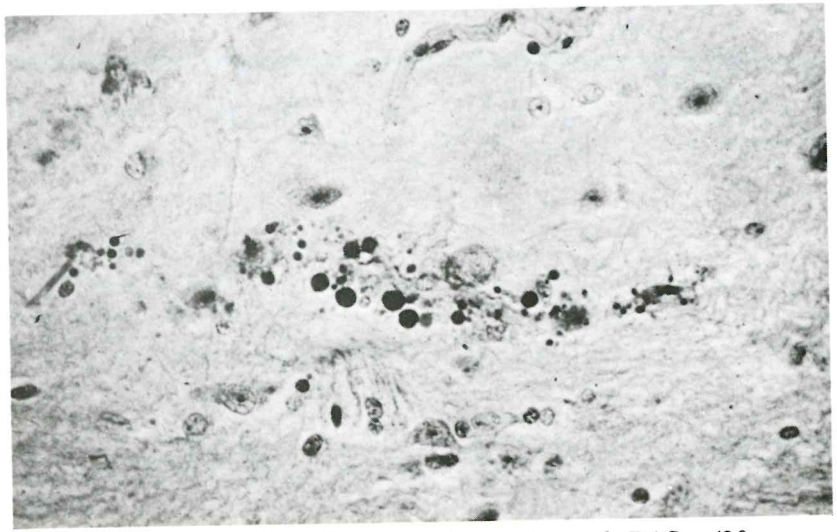

Fig 5. Spheroidal bodies adjacent to damaged vessel. PAS x 420.

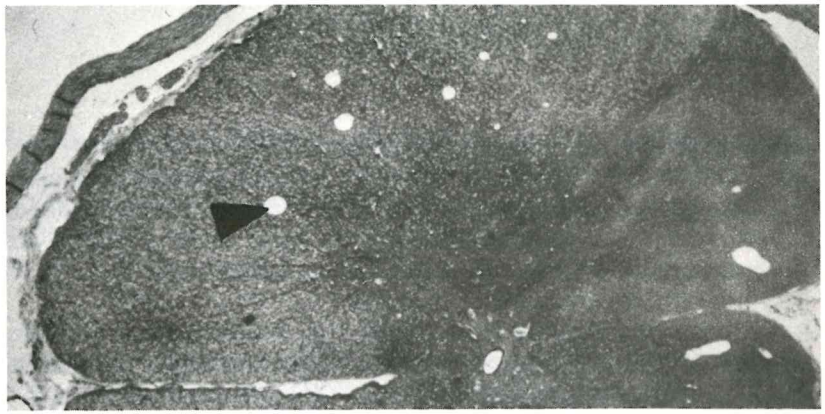

Fig 6. Gas bubbles in white matter of cord H&E x 15.

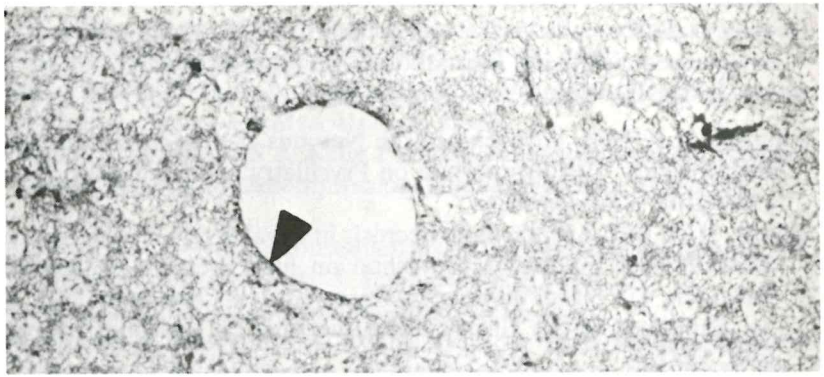

Fig 7. Bubble in white matter showing rim of young connective tissue. Masson Trichrome x 65.

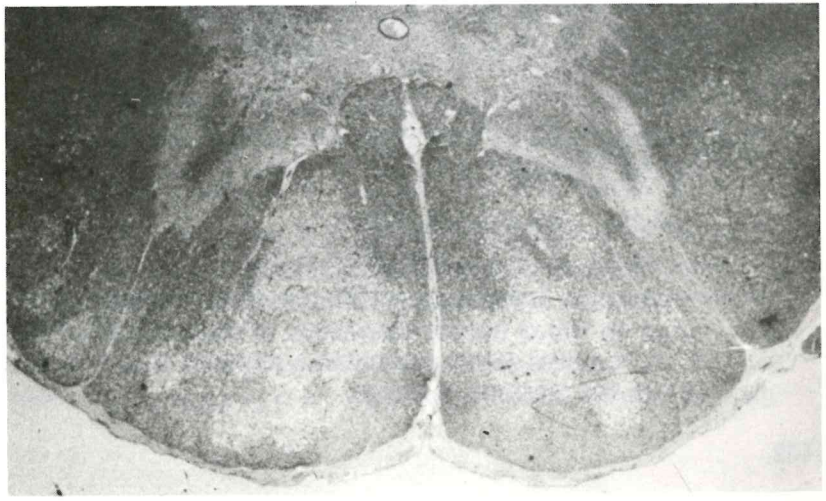

Fig 8. Gliosis of posterior columns following long term "recovery". H& E x 18.

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QUESTIONS AFTER Dr CALDER'S PAPER

Dr James: I wonder if Prof Aarli would comment on this section of the same spinal cord as we have already been shown, at the level of T 9.

Prof Aarli: I would like to know more of the former history of the diver, of minor incidents during decompression.

Prof McCallum: He was an amateur diver who had some very minor symptoms. He was sent to our compression chamber, but when he arrived there, he had no complaints and no signs. He came in with a very sketchy history as to what he had been doing. This is often a problem. He had just the one dive, to about 100'. He surfaced and within minutes of surfacing, he had a pain round his back and felt sick, then some mild ache in his left leg which cleared up very rapidly. He was given a very short pressurisation on air on the grounds that he might have some bubbles that might be improved, but again, there were no signs found, except that he limped a little, his left leg I think it was. So he was observed for a short time and then allowed to go. He came back later the same day with a rather more pronounced limp and some rather fleeting signs, I think he had some changes in the reflexes in his left leg. He was then given a longer oxygen treatment and at the end of that, he appeared to be very well, but he had a very slight limp. At the time, it was thought that it was possibly a long-standing abnormality. He was allowed to go home where he went back to work until we called him back again for a rather better neurological assessment by the neurologists at Newcastle. They did find evidence of cord defects and they thought some cerebral defect as well, but still without much in the way of clinical signs.

Prof Aarli: I think in many ways there is a similarity to what we have seen in multiple sclerosis, where you may find more pronounced changes in the spinal cord and the patient has had small episodes with recovery. So I think it says something about the possibilities of recovery.

Prof McCallum: The rest of the story is that one morning he was going to work and somebody shot him. Dr Calder examined the post-mortem material.

Prof Aarli: I have two reports, based on cases of mulitple sclerosis who had some minor disturbances and then recovered and on final autopsy, they had similar cords.

Prof McCallum: This relates to what was said yesterday. We find a minor abnormality of the EEG and we really have no idea as to what is behind it.

Prof Elliott: I think it is necessary to quote, for the record, that had this individual been a commercial diver, he would not have been permitted to continue to dive because he had got some small detectable physical signs. Although there is general relevance of this to all air diving, this particular case is a sports diver who, therefore, had the freedom to continue diving.

Prof McCallum: He didn't, in fact. There was no question of him diving again.

Prof Donald: It would be helpful if Dr Calder would clarify because I am extremely confused about what were old lesions in cases who died, were murdered or dropped dead in pubs or whatever, and the lesions in people or animals dying of decompression sickness. I am not quite sure whether we are seeing fortuitous residua of decompression sickness or the active lesions of acute decompression sickness.

Dr Calder: From the point of view of the long term effects, I think the four characteristics are here. There is loss of the myelinated fibres, loss of axons, some preservation of the subpial fibres, but the most important observation is that, where any damage occurs, there is replacement gliosis. That is the primary damage. The rest, from the acute point of view, is that of an acute inflammatory reaction which is a result of a "vascular insult" with all the attendant risk of oedema, thrombosis and then carrying-away of the degenerate myelin by the lipid phagocytes. I think it is really an extension of the acute non-pyogenic inflammatory reaction in the brain.

Prof McCallum: Dr Calder, I think if I interpret Professor Donald's request, it is to get clear the distinction between damage which might be ascribed to normal diving activity and damage following a decompression accident.

Dr Calder: From the point of view of the first part of the talk, what we were seeing was in the white matter of the brain itself. I think we are now establishing some evidence that there is vascular damage which may reflect an early degenerative process, because there is certainly some degree of hyalinisation of the small arterioles. There is, in fact, or has been, leakage in the past from these small arterioles because we have evidence of foreign bodies, for want of a better expres-

sion, this curious Periodic-Acid-Schiff positive material, which are little globules within the white matter adjacent to the blood vessels. So, I think there is no doubt that there is some evidence that there has been insult, but how one interprets this at this moment, I would hesitate to make a guess.

Prof McCallum: Do the globules appear in other situations? Can you expand a litte on them?

Dr Calder: The literature suggests that this is an interesting reflection of any vascular leakage in the brain, especially in the white matter. Certainly it occurs in people who have had little haemhorrages due to thrombocytopenia and in people who are known to have had small emboli from sub-acute endobacteriocarditis. So, it is a reflection of increased vascular permeability, therefore vascular damage. In fact, some go so far as to say that if you find these, and no evidence of anything else, it is prima facie evidence that there has been a vascular leakage. This is certainly what one finds. One would not expect to find this in a fit population but as I explained earlier, it is difficult to get controls of fit healthy population. Until we have done some forty brains and looked in the same areas, it remains unproven.

Prof Donald: Have you been able to examine brains of divers who have had a successful or uneventful diving career and then moved on and done something else? One would be most interested as to whether these people showed any particular effects.

Dr Calder: We have not yet managed to do this, but any diver is welcome to subject himself to investigation!

Dr Cox: You say that you have examined eight brains, of which five have got vascular changes. Now, as I have understood it, you only gave account of one spinal cord. How many spinal cords have you examined in which you have found evidence of old damage?

Dr Calder: We have examined seven cords so far. This includes the one with the major damage and then six others in which one has found various odd foci of gliosis or, in fact, acute changes. This is a mosaic of several cases and these are taken from the various cases. There are three in which one has found long-term changes because the diver died of something else, and the other four have had various acute changes, because they have died during the course of recovery.

Dr Cox: Have you been able to relate those changes to either a presence or absence of clinical features?

Dr Calder: Not yet because the data are difficult to obtain. We have only been doing this in the past 6 or 8 months and we are still trying to get the appropriate clinical data. It is a very long and time-consuming exercise.

Prof Aarli: It is quite amazing to see how big a lesion can be and still represent a "silent" lesion. I think that many of our divers have been sports divers for many years and may have had small accidents. Many of the transitory findings we have made may, in fact, represent unmasking of those "silent" lesions.

Dr Calder: It always amazes me that, with the cord we deal with an organ which is 40 times smaller than the brain and we find so much, and yet in the brain, where the vascular supply and everything else is 40 times bigger, we have not found anything of any great significance. Maybe we have not looked, of course. This is one of the problems. We are doing them from set blocks at the moment. I think we have got to modify our techniques and concentrate on different areas.

Dr Bennett: Do I understand that you don't have any deep helium saturation tissue at all? Have you ever looked at such tissue?

Dr Calder: I have looked at some. Five of these cases had died in helium saturation.

Prof Donald: How did they die?

Dr Calder: Most of those in helium saturation died from a variety of traumatic causes. Certainly some of them died from pure helium, and some from mechanical failures.

Dr Skullerud: I think that the vessel changes that you have described in the white matter, the surroundings and macrophages, may be hypertensive lesions. That is the most common cause of such lesions. Maybe there is some hypertension during the diving. Does the blood pressure rise during diving?

Dr Calder: I require advice on that, but as far as I know these divers all had recent medicals and were fit. Whether you get hypertension during these excursions I would ask Professor Bennett?

Prof Bennett: There is no record of hypertensive syndromes occurring. I don't think that is a regular accompaniment to diving as such unless the individuals were working very hard.

Dr Calder: The primary vessels which suffer from hypertension are the ones in the internal capsule, the pons and the medulla and not these vessels in this particular area.

Prof Aarli: Have you detected lesions where the only vascular finding is an endothelium damage?

Dr Calder: Yes, some of them. I think that it depends at which stage you get the tissue. If you get it within the first 12-16 hours, I think endothelial damage is the only lesion. A little longer and one is going to get into the stage of more damage within the media, rather like the development of atheroma.

Prof Aarli: Does that mean that you think that the primary lesion is an endothelial lesion?

Dr Calder: I have given quite a lot of thought to this and I am wondering whether it is rather like the endothelial lesion which one gets in the early stages of atheroma. There is an increased vascular permeability and then it all goes on from there. Whether the original insult is fat or gas, I am not prepared to say.

Dr Shields: You suggested that one of your slides showed young collagen round the bubble, but then you said there was no cellular infiltration. I find it difficult to understand that. Could I ask what the stain was and whether, in fact, you were not staining fibrin? Secondly, could you please remind me of the histochemistry of the PAS reaction? What was the material that you were staining in your granules?

Dr Calder: The fact was that this early bubble was within half an hour of the insult and I would not expect to get a cellular reaction at that stage. I think the collagen comes from the tissues. It is collagen-like; I am not prepared to say what it is, but it stains with a histochemical stain. So it is alien and foreign to that particular area. The next step, of course, is to do electronmicroscopy on it, to see actually what it consists of. The next point, the PAS stain essentially stains glycogen and glycogen-like substances, it becomes incredibly non-specific. There are two stages to this, because you can dissolve out the glycogen with diastase. If you have got a diastase-resistant body you have got something different.

Dr Cox: Is there any way of dating these insults to the brain or spinal cord from their histological appearance. Can you get any idea of when the insult might have occurred?

Dr Calder: Within the first day or so, we can estimate a span of a few hours. Once we are beyond 3 or 4 days, it is a very difficult thing to time things histologically.

Prof McCallum: It seems to me abundantly clear, restricting ourselves to CNS changes in divers who have been diving normally, that we badly need more material. Would you agree?

Dr Calder: Yes, that's right. I think the only answer lies in getting this disease pattern built up for human material. It is the same as the pattern of atheroma which was established in the 1950's by looking at Vietnamese casualties. It is rather sad but it is the only way.

Prof McCallum: There seems to be here, perhaps, an obligation on countries to make sure that when there are possibilities of getting material in people who have been diving for variable periods of time that it should be properly looked at.

Dr Cox: I think one might conclude, Mr Chairman, that this work is rather like a bikini, it reveals enough to be very interesting, but it conceals all that is vital. I think that a lot more is needed before we can comment.

Prof McCallum: It seems to me to be a fundamental problem in relation to Professor Aarli's findings that we should have more histological material.

Prof Donald: I would like to endorse that very strongly. I think that it should be noted and highlighted in the minutes.

Dr Shields: We are in danger of confusing the issue here. You say we have to collect material to illustrate the sort of thing that Dr Calder is showing and that it is of vital importance to what Professor Aarli is pointing out. But, Dr Calder was describing a decompression effect, and Professor Aarli (we think) the effect of a stay at pressure.

Prof McCallum: No, I was trying to make this distinction. I am talking about normal diving without decompression accidents. I am talking about divers who have had experience of saturation work, not just the need to get tissues from people that come to grief in a diving accident. As Dr Calder has pointed out, we have benefited from men who have died from quite extraneous causes, they have been murdered or maybe some other cause. But, for example, if a diver whom we know has done a lot of saturation diving had a coronary, it might be important to be able to look at his brain, even although the coronary might, in fact, create difficulties.

Dr Shields: The five cases which Dr Calder described as having died in helium saturation have all had extensive air diving experience and as far as I am aware, have all been treated for neurological decompres-

sion sickness in the past. So, we still do not know what we are looking at.

Prof McCallum: It will be impossible to get any tissue from a diver who has not done air diving.

Dr Ørnhagen: Whatever publication comes out of this workshop, may be used as a guideline for ethical committees. I therefore stress the importance of what comes out of here. We have to try and come to some good conclusion. We have to clearly separate deep dives in an experimental mode from the routine offshore diving.

Prof McCallum: It has been the intention not to make ex-cathedra announcements or recommendations. Problems could arise from appearing to set up criteria that could be quoted in the absence of sufficient background material.

Dr James: I think that the crucial thing as far as we are concerned is that we have evidence of dysfunction in men at pressure. That dysfunction involves some of the systems that we have been discussing this morning, particularly the vestibular pathways and even some of the cord tracts. The dysfunction at pressure in a deep helium oxygen saturation dive can have several explanations, some of them clearly reversible like membrane effects whereas the effects that Dr Calder has described in the spinal cord have been known for over a hundred years. Extensive cord demyelination and microinfarcts, compatible with considerable function, has also been recognised for a long time because in 1936 Lichtenstein and Zeitling wrote up an almost identical case.

Dr Skogland: Autopsy material from divers seems to be scarce. I was just wondering how pathologists cooperate internationally in this field. Can that cooperation be improved?

Dr Calder: When a diver leaves his diving occupation, very often he starts a job as a cafe proprieter or such like and that is all the coroner knows, so we are not aware of his past diving. That is the real problem. However, we do get enormous cooperation after a diving fatality everywhere except, for religious reasons, the Middle East.

VARIOUS APPROACHES TO NEUROPSYCHOLOGICAL EVALUATION OF DEEP DIVERS

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The neurological and psychological manifestations of decompression sickness and diving accidents have been documented using standard neuropsychological tests and neurological examinations. Peters, Levin and Kelly (1) studied ten divers with a history of decompression sickness involving the central nervous system, and found that eight had unequivocal neurological deficits implying multiple supraspinal lesions. The investigators concluded that cerebral disturbance following decompression sickness in divers is more common than they previously believed. In recent years, anecdotal reports have been received regarding neuro-psychological symptoms following deep dives in which no accident or decompression sickness occurred. These reports have prompted some members of the diving community to express concern about the possibility of subtle long-term effects of such hyperbaric exposure, and to acknowledge the need for careful studies involving neuropsychological testing before diving and at various intervals after deep-saturation dives. Such anecdotal reports of post-dive symptoms had been received from participants in a 40-days, 1800-feet helium-oxygen saturation dive conducted at the Navy Experimental Diving Unit, Panama City, Florida, in November, 1979. Although these divers had no previous neuropsychological testing, it was decided early in 1982 to interview the six participants carefully, and to administer neurological, electroencephalogical and neuropsychological examinations to assess possible sequelae at that point.

No accidents or decompression sickness were reported as a result of the experimental dive, but reports and anecdotal accounts do include vertigo and nausea associated with High Pressure Nervous Syndrome, as well as weight loss and difficulty performing tasks at depth.

Several of the divers reported that in the days, weeks and months following the dive they experienced a variety of behavioral symptoms of neuropsychological disorder. They reported that simple tasks required more time and more effort to accomplish, and that fatigue set in earlier and more often. Cognitive operations such as mental arithmetic calculations were more difficult or actually impossible, requiring use of paper and pencil. Attention span was reduced and reaction time was lengthened. One subject described actual failure to react at all when traffic lights changed from green to red.

Divers experienced problems in finding words, putting thoughts together and expressing thoughts easily and articulately. They also experienced a variety of memory problems. Altered states of consciousness were described: feeling "odd", sluggish, confused or depressed. Inconsistency was reported: feeling bright, sharp and capable one day, but slow and ineffective the next.

Not all the symptoms were described by every subject, and one subject reported no symptoms at all. Improvement in the months following the dive was reported consistently, and almost all felt they were back to normal or nearly so by the time this study was conducted, some three years after the dive. The residual symptoms reported included mild concentration and memory difficulties, and the inability to do calculations as well as before.

In this 1982 study, the six subjects were given physical neurological exams and waking electroencephalograms. They then were administered the Halstead-Reitan battery of neuropsychological tests (2), which includes the Wechsler Adult Intelligence Scale (WAIS) and the Minnesota Multiphasic Personality Inventory (MMPI) in addition to specialized motor, sensory, memory, attention, perceptual, and higher cognitive function tasks. It is regarded as the most thoroughly standardized and documented battery of its type, and its application in the diving industry has been strongly advocated (3).

In that study, which has already been reported elsewhere (4), the results of the physical neurologic examination were within normal limits on all but one subject. The mild sensory dysfunction in this subject was thought to be due to earlier trauma, unrelated to the dive. All the EEGs were read as normal by the neurologist/electroencephalographer.

The results of the neuropsychological tests were generally within

normal limits, as well. Only one subject's Impairment Index was in a range suggesting mild cognitive impairment (due almost entirely to problems with concentration, attention and incidental memory). All the others showed no evidence of significant impairment at this point. The modal Impairment Index (0.1) was well within normal limits. Of the 42 Halstead-Reitan subtest scores obtained on the six subjects, only nine fell beyond the standard cutoff score used to signify difficulty. Seven of these were on tasks requiring sustained attention and concentration. Essentially no sensory/perceptual, motor or language problems were seen. Memory as sampled on the Wechsler Memory Scale was in the superior range (mean MQ:129).

The mean Full Scale IQ as measured on the WAIS (110) showed no decline for the enlisted divers from the IQ estimated by their original classification test scores (112): No prior score was available for the physician, but his present IQ fell within the very-superior range.

The MMPI profiles were within normal limits, with only a few scattered elevations and no common personality pattern.

In summary, these test results failed to document significant impairment of cognitive function in the six subjects some three years after an 1,800-foot dive of 40 days duration. Only one of the six had measurable difficulty at this point, showing mild problems with sustained attention and concentration. Despite these reassuring findings, however, it should be remembered that all but one had reported significant cognitive symptoms during the weeks and months following the dive, symptoms which might have been reflected in test results gathered at that time. Such verbal and anecdotal reports underscore the need for more careful study of more subtle changes, with testing before, during, and at various intervals after each dive. They also provide some of the impetus for concern about long-term consequences even in the current absence of test evidence.

An issue related closely to this refers to the effects, not of a single deep dive such as this one, but of repeated dives to varying depths over the years of a divers career. Just as in this study, anecdotes from professional divers and others in the industry have described neuropsychological symptoms of varying severity. To my knowledge no scientific data have been collected to prove or disprove most of the claims, and of course there are serious barriers to the accumulation of such data.

As in any long-term study of behavior, it is extremely difficult to control the critical variables. During the period of study all divers are subject to other factors influencing cognitive function: all will become older, some will have diving accidents, some will have other types of accidents resulting in head injury, in autos, fights, etc, some will abuse substances such as drugs and alcohol, some will suffer from unrelated physical illnesses, some will die, or be otherwise unavailable for followup, some will undergo psychological, physical, or other life changes affecting their efficiency or motivation.

Some of these same difficulties also influence the selection and measurement of control subjects, of course. Methodology for research of this type has been developed, for projects studying intelligence changes in ageing, for example, and the task is regarded as difficult and expensive, but not impossible.

Once these methodological problems are resolved satisfactorily the question arises as to what measures are most suitable. The Halstead-Reitan battery used for the study described above, and in other settings, to identify and measure significant neuropsychological dysfunction may not be the best for these long-term studies. Some other more sensitive tests may be more appropriate, tests which measure sustained attention and information processing rate, vigilance, continuous performance, memory, visuo/spatial/perceptual abilities, motor speed, simple and complex reaction time, as well as higher cognitive reasoning tasks.

Such tests are being used in various settings for the evaluation of mild head injury for example, and some have been adopted for use in our own and other ongoing studies of Navy divers. They can be grouped into some of the general content areas just mentioned, and some specific suggestions can be made. There is some overlap also with the Performance Measuring System (5) already used in diving studies.

1. Sustained attention and information processing rate.
Gronwall and Sampson (6) have suggested that reduced channel capacity for information processing underlies residual impairment of vocational performance and social functioning in patients who exhibit no focal deficits on conventional neurological evaluation. The Paced Auditory Serial Addition Test (PASAT) employs a series of numbers which are presented at a progressively more rapid rate across several

trials. The patients task is to add each number to the immediately preceding number (e.g., given the number series 1,4,7, the patients response should be "5" after the number 4 and "11" after 7). Gronwall's studies have convincingly demonstrated a reduced rate of information processing which recovers in parallel with improvement in "post-concussional" symptoms after mild head injury (7) and the same pattern may well prevail among divers. PASAT performance is not closely tied to educational background and practice effects are minimal. The tape-recorded format ensures a standardized procedure. The Seashore Rhythm Test from the Halstead-Reitan battery is another tape-recorded test which requires sustained attention and it does not require verbal response. It may belong in this category.

2. Vigilance and continuous performance.
Mirsky and Rosvold (8) developed a test of vigilance and continuous performance which has been used in many settings. It consists of visual presentation of a sequence of letters with instructions to respond to certain combinations. It has been shown to be sensitive to mild disturbance and is unlikely to show significant practice effect over lengthy intervals, making it a possible candidate for this battery. The Trail-Making Test from the Halstead-Reitan battery may also belong in this category, having proved useful in similar studies. It also requires some symbolic processing and memory as well as sustained performance.

3. Memory.
One of the common complaints of our divers, "poor memory", is a complex concept, subject to a wide variety of definitions and measures. Several tasks can be selected from the Wechsler Memory Scale to be included here: Digit Span, Logical Memory, Word-Association learning, and Visual (figural) Recall. Another candidate, the Benton Visual Retention Test (9) consists of a series of ten displays of geometric designs which are presented for 10 seconds each. Immediately after withdrawal of a display the patient is asked to draw the design. There are three parallel forms of which one can be used as a measure of visuoconstructive ability by copying designs while they are held in view (with no demand on memory). A more involved and lengthy test in this category is the Selective Reminding Test (10). This test of verbal learning and memory has been shown to disclose defects in retrieval of

information from long term storage when administered a year or more after severe closed head injury (11). Residual impairment of memory on the Selective Reminding Test was common even in patients considered to have achieved a good recovery with average-level intellectual functioning. Four parallel forms of the test have been developed, which minimizes practice effects across sessions. Normative data in young adults have also been gathered. The major drawback with this test is its length and difficulty level.

4. Visuo/spatial/perceptive abilities.
A variety of tests are already in use and rather widely known, in addition to the ones already mentioned. Simple copying tasks may be the best to include.

5. Motor speed and reaction-time.
These types of tasks are also well-known and already widely measured.

6. Higher cognitive reasoning.
This area is difficult to assess with short, easily administered tests. Some promise is seen in the paper/pencil logical reasoning tests being standardized in various laboratories and used in the Performance Measuring System. Another possible candidate is the shortened form of the Token Test described by DeRenzi (12).

Selection of a battery from among these and other available tests requires careful attention to time and cost considerations. The dedicated neuropsychological researcher is aware of so many possible areas of deficit that no battery seems comprehensive enough. On the other hand we are all conscious of cost considerations and of the tolerance level of the divers themselves. One possible compromise solution has been suggested: a relatively long and comprehensive battery be performed initially on all divers, followed by a much shorter battery to be readministered at regular intervals. The longer and more comprehensive baseline measures would always be available.

Our own shorter, repeatable battery is quickly administered, easily scored, and already standardized. It includes parts of familiar tests and batteries:

1. Trail-Making Test, Parts A and B (5 min)

2. Digit Span Test (7 min)
3. PASAT (15 min)
4. WMS Figural Memory (3 min)
5. WMS Word-Associate Learning (5 min)

We are well aware of the limitations of this battery, but in about one-half hour we do sample a number of critical and sensitive dimensions in a battery which is accepted and tolerated by the divers and administered by one of their own corpsmen. We plan to repeat it at regular intervals and do not expect problems. Of course there are many other suitable batteries, but this is offered as a beginning in the hope that this area of research will be pursued with the vigor it deserves.

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QUESTIONS AFTER DR BECKER'S PAPER

Prof McCallum: When do you expect to have enough information to judge whether these tests are adequate or not?

Dr Becker: Perhaps by the end of 1984 we will have several dives on which we will have done pre and post testing. We will know if this battery is practical and whether there would be any results. We would try to assess at that point whether the results are appropriate, or whether we should make changes. This is not a massive research effort.

Parallel efforts are going on at the NEDU. Dr Mike Curley is beginning some studies in February when they are doing some dives. We may include this battery, or an adaptation of it. He is already using the performance measurement system, I understand.

A neuropsychologist like myself, who is at the hospital in San Diego, is interested to do a similar study. I have sent him all the information and urged him to include it. We have not agreed that this is exactly the selection that we should have. For example, some have felt that this battery does not really include any measure of language and that there should be. There are word fluency tests which may be very important, and may actually be very sensitive. They are a little bit lengthier and perhaps a little bit more difficult to score. I would not be averse to including word fluency and I know some of my colleagues want to add it.

Dr Thalmann: Mike Curley has been talking to Dr Becker and we are, right now, trying to come up with some sandardised pre and post-dive test for the 1000' dive in February.

Dr Baddeley: It is a nice idea to try to have a brief test that you have data on before the dive. I wonder a little bit about the need to check it out for sensitivity and so forth. Is there not something to be said for perhaps trying it out on another group? A mild head injury group first of all would give some indication as to whether the measures are sensitive or not. Digit span, I would have thought is, on the whole a relatively insensitive test.

Secondly, to what extent do you get fairly heavy intercorrelations between PASAT and span and trail making. I certainly feel it is worth while including some measure of semantic memory, either item fluency or possibly semantic processing. There are tests whereby the subject verifies sentences, half of which are obviously true, like "Can-

aries are yellow" or "Sharks can swim". In about 2 or 3 minutes you can get quite a lot of responses. It is very sensitive to other factors like alcohol, time of day, performance under pressure. It correlates to some extent with vocabulary, but is much more sensitive because it is essentially a speed test. I wonder if it might be worth including something of that kind?

Dr Becker: Or even the AB statements, they might also be useful?

Dr Baddeley: Yes, they are more of a reasoning test but, again, you get a lot of information in 2 minutes.

Dr Becker: I think you may be right. I am stuck with the present battery in that I decided some time ago that this is one that we gave to our group as a start. I think you may be right and it would not be appropriate to add either or both of those.

Dr Peterson: Dr Værnes, I believe in your research on the deep dives that have been discussed here, you used a number of these tests pre-dive, during the dive and also post-dive. Perhaps you could comment on how sensitive you feel the tests are that you have experience with?

Dr Værnes: We have the comprehensive battery when the diver starts the deep diving school and then we have part of this battery during deep dives. We use part of the Halstead battery and try to build up valid sub-tests which we find very sensitive. We have examined about 150-160 divers who have been at the deep diving school and we will follow them up every second or third year. We try to collect the same parameters during the deep dives at NUTEC. I agree with what you have said, I understand that you will use the PMS on the dive in February and I would suggest that as this is the part of the old SINBAD battery, the PMS is fairly good but some of the cognitive tests are missing. So I would suggest that you do some of the paper and pencil tests which are not on the PMS. I have had this as part of ours for two years, and we are now taking a re-examination of the subjects who started at the deep diving school 2-3 years ago. We shall follow them up continously.

Dr Peterson: I was thinking specifically of the trail-making test and the digit-span test, which I know you have been doing on the deep dives. Have you found any significant changes in performance on those tests following any of the dives that you have been working on?

Dr Værnes: During the dives the trail making has been noticeably

sensitive to narcosis, both in air diving to 60 metres and the trimix dives down to 300 and 500 metres. The same was found for pair-associative recall. The digit span is not that sensitive. We should decide on a protocol concerning, for example, these neuropsychological tests, because there are a lot of tests and the validity varies a lot.

Dr Ørnhagen: Dr Becker, I have a question related to the training effect of these tests here. We have been using the PASAT in Sweden to establish nitrogen narcosis to which it is very sensitive and we found that it takes a couple of test runs to establish a baseline. This will cause problems if we are going to use this test battery for more than experimental dives, if we were to investigate divers in offshore operations. It takes more than 35 minutes and you have to do it over and over again, over 3 or 4 days before you can get a baseline that is fairly stable. Could you please comment on that?

Dr Becker: I think you may be right. We have adapted it, shortened it and that is one aspect that we don't do according to Grunwald's original instructions. This may be responsible for the somewhat shorter time but, frankly, we have not given it often enough to know. I suspect what you say may be true - in other words, we are not sure our scores are stable in our people.

Dr Ørnhagen: Yes, otherwise you may come out with a better score after a dive than before a dive, so I think your baseline is very important to establish.

Dr Værnes: At NUTEC we have training before the deep dives, for several weeks and at least five control measurements are calculated of every value. This is also done for the EEG. For performance tests and for physiological tests there will always be some variation, but for several of the neuropsychological tests there is not that much learning effect.

Dr Becker: Based on our limited experience, there is considerable variation. That may be responsible for what Dr Baddeley says has been his experience, that it has not been as sensitive in the evaluation of mild head injury as Dr Grunwald, who first reported on the test, claims. Some people quickly become frustrated and give up on the task and then you are not sure what they might have done, if they had employed a more effective strategy with it. That is to drop out and return as quickly as they can. One of the reasons we have shortened the test is that as it gets faster it gets more difficult. We felt that to leave

out the last two trials would improve it. Simply do a very shortened version slower so that people can keep up.

Prof Bennett: I enjoyed your presentation very much. We have 50 tests which was just enormous in cost and time. Dr Værnes has taken most of these groups of tests right through the dive and post-dive to see if there is any continuation of any decrements that were present at depth. Yet, we heard yesterday from Professor Aarli that the only thing that shows a difference is the EEG changes. The performance tests show no change. Therefore, I wonder what we are looking at. Are we able to show something with these tests at all, or is the EEG picking up something that these tests cannot?

Dr Værnes: My conclusion yesterday was that the neuropsychological examinations were normal, post-dive, for the Deep Ex I and Deep Ex II. I would stress what Dr Becker said that one thing is the examinations done during a dive, experimentally. Another thing is the pre- and post examinations, they must be performed at a clinic where they are used to people with neurological disease. What we are doing during the deep dive is to pick out some of these neuropsychological tests as a lot of them cannot be performed in the dive. The post-dive EEG changes don't necessarily correlate with the neuropsychological post-dive impairment.

Dr Becker: We have a saying in neuropsychology that "absence of evidence" is not evidence of absence. We don't have evidence of reduced performance on these tasks in the week following the dive. This does not mean that there would not be a reduction. One of the reasons for selecting these tests was that they seem to test some of the functions reported in the anecdotal accounts.

Prof Bennett: The only measurement we have found a decrement in was verbal memory in one individual, who reported the same kinds of things that your individuals have reported. All of the other tests showed no change. If it is that mild, what are we dealing with?

Dr Becker: There is a kind of value judgement in my statement in that we must have more subtle tests to detect more subtle deficits. Then it becomes a decision as to how important in the practical world, those subtle changes are. We are all aware that people take risks, we know the dangers of boxing and yet there are a number of people who box, nevertheless. We know that there are dangers in smoking, and yet there are a number of people who smoke. Those are value judg-

ements. The gross neuropsychological batteries so far do not seem to have found evidence of damage. Therefore, if we are going to find the subtle damage, we are going to have to use more subtle tests.

Dr Værnes: Concerning the sensitivity to these tests, people with subtle symptoms caused by 'near miss accidents' showed significant changes in these neuropsychological tests, especially the trail-making and the spatial memory on a T by T.

We use several control groups. One group that we are starting on now are aviation pilots in Norway, because they have been selected, in many ways, through the same procedures as the deep diver. They have gone through very much the same performance tests. So we will be sampling F-16 and F-15 pilots in Norway, next year. The control group is a very controversial thing. For offshore divers, is it an offshore effect? Is it a shift worker effect? There are a lot of control groups you should consider.

Dr Török: We have focused attention on the sensitivity of these tests but I would like to emphasise the calibration of the tests that we might chose that is, the control data. Although the result of a psychological test may be quantitative, the meaning of that result will be derived from its calibration in a population. Unless one is careful one might choose a very sensitive test with its own vocabulary. If such a test is used in a diving situation where the numbers are statistically very small, its use will result in a descriptive exercise. You will describe the test and you will describe the dive, giving the context in which it was used.

Dr Baddeley: Can I amplify that point slightly. I worry quite a lot about the validity of neuropsychological tests and how they relate to the performance of the person in everyday situations. Partly in connection with this, we have been carrying out studies over a number of years now, using closed head injury or ageing as our source. We have been trying to relate performance on standard memory tests to reports of memory problems by the patient, by the relatives and diaries kept by both. What we find is that there are some tests that are very sensitive to the effect of head injury but don't correlate at all with the complaints of every day problems. Probably the one that comes through most strongly is logical memory; remembering a passage of prose. It is an old standard psychological test, but it really does seem to predict those people that will, and whose spouses will, complain that they

have a lot of problems in coping. So I think maybe we need to worry a little bit more about the validity of the test. On that basis I would suggest including that, rather than paired associate learning, in your battery.

Dr Giran: We performed some psychological tests during the deep dives involving forty divers. The decrements observed were quite reasonable and acceptable and went back to surface values after the decompression. Is the diver able to do the common task he is asked for? At 613 metres in water he has been able to do the task asked.

Dr Becker: What we are really pointing toward is to see if there would be decrements over a ten year period, for example, in a diving career, rather than simply as the result of one or more dives.

Dr Baddeley: The question of whether he can or cannot do the task is a rather separate issue from the long term after effects, but I think it is unfortunately more complicated than stated. It is related to, I think, a point that was made yesterday that "well, decrements are only 10 % or only 20 % and if it takes him 10 % or 20 % longer, fine, no problem". I think the difficulty stems from the possiblity that what will happen is that he will operate as though he were normal, and therefore make mistakes and a crucial mistake made, perhaps only 1 dive in 5 may be totally unacceptable. The difficulty is that we don't have a good method for translating percentage decrement in performance into the likelihood that he will do something stupid underwater. My own rule of thumb is to be much more worried by errors than by slower time, that is simply common sense. I think it is dangerous to say that he can do the job, therefore we don't need to worry.

Dr Giran: I meant only that if the neuropsychological test is said not to be sensitive enough to demonstrate abnormal behaviour, we can also observe that apparently the diver is able to perform most of the tasks he is asked to do.

Dr Baddeley: I think it is important to do both.

Prof Bennett: I would just confirm that with reference to my 10 % figure, these data show that they were merely working more slowly and the accuracy was as good, which is encouraging at least.

Dr Baddeley: I want to make two points that might seem to be inconsistent. There are data from a number of the AMTE (PL) dives. The point that I want to illustrate is that one can use the performance during pre-dive and post-dive, and use that to get some indication of

whether subjects return to pre-dive performance or not. This illustration just happens to be the first to come to hand, and is rated sleep quality. There is an impairment in rated sleep quality. Performance actually starts to improve (at about this point) even although the sleep is still pretty terrible and the sleep debt is building up. That is convenient in that it allows us to interpret our performance data as indicating a genuine pressure effect and not simply a sleep debt effect. Broadly speaking, in some 7 or 8 two-man dives the subjects do revert to their pre-dive performance. Furthermore, we have one pair of subjects that did 3 successive dives and, looking at their performance across those 3 successive dives, they simply get a little bit better with practice. So, as far as the data from the AMTE (PL) dives is concerned, we don't have any evidence of impairment. The small amount of data taken from neuropsychological tests in more recent dives also confirms that. So, at least in that respect, our data look reassuring rather than otherwise.

In contrast, however, I would like to show something rather different and would be interested in comments and thoughts on what one should do in such a situation. I got a telephone call from a diver's wife who had originally written to Val Hempleman. She wrote because she was concerned about the characteristics of her husband who was a saturation diver. She was a very intelligent woman who had been in contact with a number of wives of other divers with the same company and had done a mini-survey of seven diver's wives about symptoms that were reported. Her letter is as follows:

"With reference to our telephone conversation, I have listed below some of the physical and mental defects that seem to occur in divers after periods of saturation. This information represents the opinions of myself and six other diver's wives who were kind enough to respond to my inquiries. Not all of the ailments listed affected each diver. The number affected by the defect is shown alongside.

1. Loss of memory 4
2. Aggressive behaviour 5
3. Short tempered 5
4. Secretive 3
5. Anti-social 1
6. Slow reactions 3
7. Tiredness 4
8. Anxiety 3

9. Inability to communicate 2
10. Inability to concentrate 4
11. Problems with eyesight, rubbing, poor vision 4
12. Disinterest in appearance 4
13. Derogatory effect of alcohol 6

The latter seems to be by far the most general complaint, the seventh diver is tee-total. We all felt that it is probably the most frightening aspect of all and one of the men involved has not dived for 10 months, and yet according to his wife he was still unable to cope with the effects of alcohol".

How one interprets these, I don't know. The way she described things, both she and her husband are very sociable and one of the pleasures of life was to go out and have a drink whenever they could. She reports that, in fact, very small amounts of alcohol had really quite dramatic effects on him. Now, how one interprets this sort of data is very difficult. Clearly, these were not divers who had been taking part in PL-type or Duke-type deep dives: they were commercial divers who had been through a wide range of diving experience. It is also interesting to think of the complaints in terms of the sort of things that we might pick up on something like the Halstead-Reitan battery. They tend to be things that are not easy to detect like loss of memory. When people talk about loss of memory, typically they don't mean that somebody is doing badly on a paired associate task, they mean they forget things, they are absent-minded. What evidence we have suggests that this does not correlate all that well with performance of paired associate learning. Indeed, in one study that I carried out, we found that a measure of remembering to do something at the right time was actually negatively correlated with ability to learn a list of words. So, clearly, loss of memory might not be picked up by such a battery. Things like aggressiveness, obviously, I think, would not. Short-temperedness, secretiveness, slow reaction might do so (though this was, in fact, shown by a minority), but tiredness? One of the characteristics of mild head injury is tiredness, inability to concentrate, attentional difficulties. I know of no neuropsycological test, however, which picks this up. Similarily, disinterest in appearance and the derogatory effect of alcohol. One does not normally give a subject alcohol before testing him on a neuro-psychological test, but it may well be actually a way of potentiating some real or potential neuropsychological problems. I

feel that the data that we have got so far from the well controlled experimental deep dives is not too worrying. I am less conviced that there are not really problems out there based, perhaps, on much less spectacular but much more frequent deep dives. How does one discover if indeed that is the case and, if it is, what does one do about it?

Dr Elliot: I am very concerned about extrapolating from that letter to what you have just said. I would have thought that you could get half a dozen wives of other types of offshore personnel, or half a dozen wives of office workers currently under the stress of the recession who would say almost exactly the same as those divers' wives. If you were to include my wife, I am damned sure she would!

Dr Baddeley: As Head of the Applied Psychology Unit, I get quite a lot of odd letters. I have never had a letter like that before. I have not drawn a conclusion, I have asked a question, and I have asked "How can we answer that question"? I have indicated that, maybe, the techniques that we are describing would not answer that question. I am not saying that there is anything there, I don't know, but if there were, I think it is not clear that we would know.

Prof Bennett: I take up the point too. We are all worried about the physiological effects of pressure, as such, but there are other factors operating in the saturation diver. He is away from his wife, he is isolated, he is impaired from normal social communication and so on. All these effects could possibly induce these mild signs and symptoms that are very hard to pin down.

Dr Baddeley: They didn't sound terribly mild from her viewpoint, but then . . .

Prof Bennett: The wives never do make them sound mild. The wives of the divers involved in these interpretations of something going wrong are the most verbal, that something is wrong with my husband. He is not the same, please do something about it; you made him this way.

Dr Baddeley: They may be the most sensitive source we have, when they are treated properly. Obviously, it is necessary to get appropriate controls if one does look at these sort of data. I am not saying that one should take the letter at face value.

Prof Bennett: The danger with a list of very soft factors such as you have just shown is that if you ask a group of people to put something down, word will get around that this is it. You would pick that up ad

nauseam and you may convince yourself that you have got onto something when you have not.

Dr Baddeley: Yes, sure, there would be real problems. Obviously it is enormously complex and difficult. You can hardly go up to a chap and say your wife says that you are aggressive and can't hold your booze any more, would you like to do my tests? Do we need to worry whether there is something to this; I would have thought probably we should.

Dr Værnes: We have started a project which records psycho-social factors. We are very concerned about the control group. The parameter is offshore workers in general, chamber operators and shift workers, because is it a specific offshore deep diver effect or is it a general offshore effect? It might be both, but we have to check first, before we draw conclusions.

Dr Baddeley: As your source of psycho-social evidence, are you using just what the divers say themselves or are you getting comments from wives?

Dr Værnes: We have standard psychological, psychometric and sociological tests plus a structured interview. It has just started and is very complex. We have been very much concerned about the control groups.

Dr Baddeley: Are you asking the diver's wives as well as the diver?

Dr Værnes: We have planned to go out on field studies eventually asking their wives. We have not started yet, but it is not an easy thing to do.

Dr Baddeley: The wife's comments on the problems encountered by a closed head injury patient prove to be a more reliable and valid indicator of his problems than his own. That may be peculiar to memory, in the sense that people with memory problems forget that they have got memory problems. There may also be an overlay of bias: if you are someone who worries you exaggerate, if you are someone who wants to be macho about it then you cover it up. You do need another source of evidence.

Dr McIver: I once made a list of the symptoms presenting themselves during two weeks in a normal general practitioners surgery. They were; tiredness, lassitude or fatigue, loss of drive and energy, tendency to fall asleep in a chair in the evenings, inability to discuss, failure to concentrate, diminished mental agility, lapses in scored and recent

memory, absent-mindedness, thought block, irritability, bad-temper, insomnia, anorexia, non-specific changes in behaviour pattern, bowel and bladder symptoms, thirst and dryness of the mouth, dizzy spells, numbness, tingling or pins and needles of limbs, headaches and backaches. The factors in those patients which appeared to be present were ageing, stress, marital problems, injury to head neck or spine, organic and metabolic disorders, travel, lack of sleep, and sea-sickness. None of them had ever been diving.

Prof McCallum: Thank you. I hope nobody here recognises too many of these symptoms as their own.

Dr Ørnhagen: The blue graph Dr Baddeley showed here on sleeping quality; these were subjective estimates by those that were sleeping. Do you have any correlation to the EEG estimate of the sleep?

Dr Baddeley: We did collect some actual sleep data on some of the earlier dives. I think there was reasonably good objective evidence that they were sleeping pretty badly.

Dr Ørnhagen: Good correlation?

Dr Baddeley: I think so.

Dr Török: I would like to reinforce that question mark because correlation implies a certain tonnage of data and I wonder if we ever had this. Yes, there were some sleeping EEG's taken, but when one talks of incidence and correlation I think we just did not have the data.

Dr Værnes: Two months ago we had a survey among 100 shift workers on the north part of Norway and we found very much the same symptoms.

Prof McCallum: I would like to see similar questions addressed to wives of coal miners because you have got what is often a stressful activity with obvious dangers and I don't know whether this has ever been done.

Final Discussion

Prof Elliott wished to pick out key areas for further discussion. The meeting had begun with a presentation on the ethics of deep diving. Prof Elliott felt that the EUBS was not in a position to discuss this issue as every member has his own equally valid opinion. The purpose of the society was to try and achieve the scientific truth. Any judgements of that truth, and any subsequent guidelines that may be produced by Government bodies, are not constitutional. He felt this was a very important point.

The definition of deep diving as opposed to shallow diving was considered and, if such a boundary is necessary, 250 metres was felt by Prof Elliott to be acceptable. The industry, in its diving research initiatives, use the same figure because of the extensive experience of diving shallower than 250 metres. Diving beyond 250 metres is less frequent and therefore, though operational, is not yet routine.

Prof Elliott felt it was important to remember what has been said by the bodies who have no commercial interest, i.e. the Navies. The British, French and US Navies had indicated to us yesterday that diving to depths around 540 metres was feasible. He felt that as a group we must agree that diving deeper than 250 metres is practical and likely to continue.

The long-term neurological consequences of deep diving were then considered. Prof Elliott felt that the concern was justified, but felt the discussion had become too diffuse. He suggested we should concentrate more on the central theme of long-term neurological consequences. He was convinced that no long-term effects of deep saturation had been demonstrated, but cautioned against complacency.

Prof Elliott outlined two areas where work should be done. These included post-dive depression, undue fatigue and feelings of failure, as described by NEDU. Sleep deprivation seemed to be a possible factor and the analogy with the American space programme was useful.

The highlight of the meeting has been the meticulous presentation by Professor Aarli in which he described some physical signs in 4 divers post-dive. The cause was unknown. The minor and transient nature of these signs was reassuring. However, the sample of divers was small and there is the hypothesis of cumulative effects as in

"punch drunk" boxers. It is relevant that some of the divers in the deep dive series have been in several dives without detected effects.

Dr Calder's presentation on decompression-related long-term effects put the effects of saturation below 250 metres in perspective, and showed in particular that compressed air diving can be hazardous.

Prof Elliott then went on to discuss the possible causes of the "manifestations", as he felt the discussion of this had been inadequate. He suggested 4 topics for consideration:

1. *The in-dive phenomena*, which may be an unmasking of some pre-existing psychological state.
2. *The effect of the oxygen level*. This level seems to be similar from laboratory to laboratory but he felt that it should be remembered in any discussion.
3. *EEG changes*. One diver who had had EEG changes, subsequently was shown by Professor Aarli to have neurological changes. The relation of one to the other was as yet unknown.
4. *The influence of some pre-existing brain damage*, possibly due to decompression sickness, could be a factor in 2 of the 4 cases presented. The physical signs found were compatible with a bubble aetiology. However unlikely this may be, Prof Elliott believed that it should not yet be dismissed.

Another important area for discussion was the acquisition of more data. It is necessary to standardize procedures in some way. The method of arriving at such an agreement should be discussed first, rather than which test should be used. Possibly most data could be gathered by the use of the relatively simple physical neurological examinations during and after deep dives.

Prof Elliott summarized by commenting that he felt the meeting had been worthwhile. It had revealed no long-term health effects from deep saturation but he indicated that the remaining discussion should be directed towards two main areas:

1. What should we do to collect more data on divers in order to define the nature of a possible problem, transient though it seems.
2. How do we investigate its possible causes?

Professor McCallum then threw the meeting open and asked for any comments.

Dr James wished first to clarify one point with respect to Professor Aarli's presentation concerning the transient loss of the abdominal and plantar reflexes in one of the divers. Normally, when these are lost due to physical disease, they do not return. This suggests that the loss of these reflexes for one month might be due to a reversible, almost non-pathological insult. He suggested that the meeting should begin by looking at the "in dive" phenomena in an attempt to find mechanisms which can cause the kind of symptoms seen.

Dr Tønjum felt that a full examination of the "in dive" situation must start with the dive log. This should be searched for any special events that would not have been recorded elsewhere. He felt that it could be something that happened during the course of that dive, e.g. the PO2 or the pressure. We should not start with speculation.

Dr James replied that the evidence of neurological dysfunction is detected almost immediately after compression, before anything else can be considered. Dr Tønjum reiterated that, although these changes occur early in the dive, there is normally no after-effect and there may be some operational error responsible for this event.

Professor Bennett felt that we should try to attribute causes of the phenomena known as HPNS. There appeared to be a growing body of data as to the cause, and its relation to anaesthetic mechanisms. He believed it to be a fundamental effect on the neurotransmitters. This could explain the memory effects because of the possible serotonin changes. Whether these changes persist for as long as a month is unknown, but this should be an area for research. He informed the meeting that there is an intensive programme under way at Duke, primarily because of their findings on the Atlantis series. He believed that they would find that HPNS, which has been associated with a general increase in brain excitability, may well be the sum of specific changes in parts of the brain. He thought that some of the changes might not be as rapid as we had previously believed, and may take time to return to the baseline. If these are pressure-related effects, he suggested that they might be reversed by anaesthetics, but the problem was how much narcotic is required for this reversal?

Professor McCallum suggested that HPNS and hypomania represented two distinct observations. One was repeatable and well documented in other dives, whereas the hypomania was an isolated observation. He felt that the best method of advancing our knowledge was

to study a recurring pattern to prevent us being led astray by events which might not be relevant.

Professor Bennett agreed, but pointed out that, for example, treatment with lithium was related to earlier studies of the effect of lithium on nitrogen narcosis and HPNS where it was found to work in opposite directions. The similarity to this example was interesting and he believed that there was mounting evidence from animal studies of changes in neurotransmitters at high pressures. These kind of measurements were very important in interpreting the effects both during and after the dive.

Dr Rostain agreed with Professor Bennett that it was difficult to speak on the cause of HPNS as he believed that it was a complex disturbance at the level of some neurotransmitter. If it is a functional disturbance, he would hope that it would be reversible. If the action of pressure is at the cellular level, would the repetition of the dive cause permanent dysfunction in the cell? He thought that HPNS was a disturbance of the release or use of the neurotransmitter. He remarked on the difficulty in correlating impairment of function with the EEG changes, and gave as an example studies they have carried out in France where they found no such correlation. He cited the sleep study where they showed disagreement about the quality of sleep as judged subjectively and by EEG. He believed the cause of HPNS could not be seen at this stage and felt that it would be necessary to observe the effects of repetitive diving. He thought that all the facts he had seen did not prove that they were irreversible.

Dr Török suggested that neurotransmitter research could ideally be performed in animal experiments because of the ability to localise exactly the origin of neurotransmitters or their products to a specific site. Human experiments were necessarily limited to the detection of blood or urine-based metabolites of the neurotransmitters. He believed that the difficulty of that approach was considerable.

Dr Tønjum thought that the only way to proceed was to carry on monitoring as at present to evaluate the individual susceptibility and performance, and ultimately any long-term health effect. However, he agreed with Dr Török that we would not find the answer to HPNS without experimentation.

Professor McCallum asked Dr Calder if any of his projected research covered this area. He replied that the proposed primate research will involve deep exposures and hoped that the data would substantiate his existing findings, but he was entering into the study with an open mind. He also stressed the necessity for control groups because, if the vascular changes expected were common in the general population, the hypothesis would fall apart. Dr Török remarked that the opportunity should not be missed of inserting a neurotransmitter component into this research programme. Professor McCallum agreed and Dr Calder said he was at that moment talking with Dr Halsey at the CRC on this subject.

Dr Ørnhagen asked Dr Rostain if he believed HPNS to be a transmitter and synapse effect alone, and not a cellular one. The question was prompted by some studies that he had been carrying out using cardiac cells which show a marked sensitivity to hydrostatic pressure, in slowed metabolism and conduction. There are no synapses or transmitters involved at all in these effects, and he believed this to be a good example of a cellular effect.

Dr Rostain presented 3 possible causes of HPNS:

1. Release of neurotransmitter is disturbed at the level of the excitatory neurone.
2. Disturbance of the receptor site, i.e. post-synaptic effects.
3. Disturbance of the cell structure which may cause an effect in the contraction of a muscle cell.

He felt that all 3 possibilities might play a part. Dopamine is increased during dives and it seems to play some part in the appearance of HPNS. However, aspartic acid seems also to have an effect on the tremor. He remarked that there are a lot of things happening during compression and at depth, and it is difficult to trace the path of all the phenomena.

Professor Bennett agreed with Dr Ørnhagen that non-synaptic areas are affected, apart from the brain. He believed that there was a body of data which suggests that ion exchange is affected and perhaps the role of calcium in this area should be studied.

Dr Peterson inquired if there was a correlation between morphological or functional changes, and the occurrence of HPNS in a dive. In dives prior to those where the divers were examined by Professor Aarli, there were minor signs of HPNS which were detected by questionnaires. The analogy with boxing seems to falter here because a boxer, if hit hard enough and often enough in one fight, will have residual da-

mage, whereas a diver suffering severe HPNS for longer than 3-4 days during a dive seems to return to normal post-dive. He wondered if a correlation between HPNS and the long-term effects afterwards really existed.

Dr James asked if pathological studies had been carried out on animals which had been subjected to HPNS-inducing pressures. Professor Bennett indicated that both the Russians and the Japanese had carried out such tests using dogs and cats in which they sectioned organs and made biochemical studies of the animals whilst at pressure and found ostensibly negative results.

Dr Ørnhagen informed the meeting of some of the results of his studies on the effects of hydrogen on animals. He had done histology on rabbits exposed to 30 atm of hydrogen and found no changes. The organs involved were brain, liver, kidney, muscle and bone marrow. These animals had not in fact had HPNS so if HPNS is responsible it would not have been seen; only pressure effects would have been present. Dr Rostain had carried out routine histology on monkeys exposed to 700-800 metres and had never found any changes in the brain or spinal cord. Dr Calder inquired if these were acute or chronic experiments and was informed that the series was a short one involving 2-3 dives over 1-2 years.

Professor Bennett reminded the meeting that they had been discussing effects from one dive or at most two dives. If these effects, which may or may not be related to pathology in the brain can occur after single exposures, then cumulative effects may be less important.

Dr Ørnhagen suggested that one of the differences between animals and man was that the former often had no prior air diving experience and this might be highly significant.

Dr Calder indicated that his study would involve multiple air dives using baboons before commencing heliox dives. Dr Bennett hoped that the groups would be separated into air dives and heliox, or just heliox, or just air, otherwise we might reproduce the same confusing situation as at present seen in man. Dr Calder indicated that this would be done.

Professor Donald remarked that Professor Aarli's abnormal physical signs had been recorded post-dive making the cause open to question. Had anyone done careful neurological examination while actually at great pressures? Professor Bennett replied that this is done post-dive,

but not during the dive. Dr Török said he had carried out a series of routine clinical tests at pressure which involved the vestibular system, tremor power spectra, two-point discrimination, and certain EEG examinations. Professor Donald asked how many tests involving the plantar and abdominal reflexes were done under pressure. Dr Török replied that the studies did not involve the abdominal reflex but the plantar reflex, which was done at pressure in 5 or 6 subjects, was normal.

Dr Værnes commented that they routinely carried out Fast-Fourier-Transform-EEG, and they had now built an 8-channel EEG for focal analysis. Tremor tests involving postural and intentional tremor were also carried out and they planned to carry out several neurological examinations during saturation. He summarized that they had used an extensive battery of tests and had found that the changes recorded at depth were normalised during decompression.

Professor Bennett commented on the possible importance of oxygen effects. The Duke experience suggested that the recorded effects might be due to oxygen, or decompression, or possibly the change from trimix back to heliox. He quoted the work of Dr Brauer who carried out studies of variable oxygen from 2.4 bar to 0.21 bar and recorded no effect on HPNS in animals. In humans the range of experience is considerably narrower, from 0.4 to 0.8 bar at 300 metres and no effect on HPNS had been observed. The post-dive effects were not studied.

Dr Værnes said that he had carried out a study using hippocampal slices in supersaturated oxygen which demonstrated that minor changes in PO2 produce major changes in intra- and extra-cellular potential.

Dr Rostain informed the meeting of a study which varied the partial pressure of oxygen once HPNS had been produced in monkeys. He had normally seen an improvement of HPNS when the partial pressure of oxygen is increased. During the 180 metre dive however, they reduced the oxygen level to 120 mm Hg with men and found no increase in HPNS.

Dr Tønjum remarked that they had always been slightly afraid of playing around with the oxygen because of its toxic effects on the lung possibly affecting any treatment required during the decompression. They have consequently maintained it at about 0.5 bar.

Professor Donald asked if the meeting felt that Professor Aarli's findings were due to decompression and not due to any effect of saturation. Professor Bennett believed that was quite unproved and, until there was a better understanding of neurotransmitters which are more likely to be the cause, he felt it was impossible to distinguish between the two.

Dr Giran gave the French experience that between 8 and 10 physicians took part in their dives and, even if they did not pick up effects on the other subjects, they would have detected it in themselves if there had been anything wrong with their reflexes or neurology.

Dr Calder asked that, if neurological signs were minimal when performance was reduced, does that mean that performance is a more sensitive measure than neurological signs? How is the sensitivity of the neurological signs and symptoms decided upon? Dr Török commented that some of the neurological signs are quite gross in HPNS and gave the example of ataxia, the finger to nose test, which can commonly be grossly abnormal, and tremor which can be several times normal values. During dives, if these symptoms occur, they return to normal after the dive is completed.

Dr Peterson drew attention back to the decompression phase as the cause of these effects. The NUTEC dives involved dives by two commercial companies; in one case there were no findings, while in the other, a total of 4 in 24. If these effects are due to compression, or being on the bottom, they should have been seen in the first dive. The first dive had a greater incidence of HPNS so he suggested that this indicated the effects to be due to some decompression event rather than pressure. He also suggested that oxygen differences might indeed be relevant as one of the many possibilities could be some dive-related change in brain blood flow due to the high oxygen.

Dr Tønjum informed the meeting that diving companies used different compression and decompression profiles and all of the effects occurred with one company which used high oxygen during parts of the decompression.

Dr Brubakk wished to point out that what is seen during the compression and bottom phases is a global change in the total functioning of the CNS, whereas the reflex changes recorded after the dive are focal changes which may indicate a different genesis. He wished to remind us that the first diver mentioned in the meeting who exhibited

severe depression after a 300 metre dive has now, 3 years later, got reflex changes and these appear permanent. The diver was not investigated prior to the dive, but had no previous record of any such problems. He informed the meeting that the diver did have a high recording of bubbles during one of the ascents during the dive. It was reported afterwards that the divers had concealed symptoms of aches and pains to facilitate the speed of the decompression.

Dr Tønjum wished to remind the meeting that the diver with reflex changes had also carried out commercial air diving. Professor Bennett commented that the reports of aches and pains are typical of saturation decompression from deep dives where the divers ignore minor pains until they become persistent, before they report. The cause of these pains is not fully understood. He questioned whether the phenomena of reflexes, and memory and personality changes were related; and if so, were they the product of the same mechanism?

Dr Værnes commented on the complexity of HPNS symptoms, and Professor McCallum invited Dr Török to comment on the different elements of HPNS. Dr Török described 4 major categories of symptoms based on case history material from the AMTE (PL) series, but would not be surprised if there were more.

Professor Donald remarked that many of the HPNS symptoms are a malfunction, but not an interruption, of a neuronal pathway. He felt this was totally different from impaired reflexes and felt this was an important distinction.

Dr Baddeley wished to amplify Professor Bennett's question of whether the after-effects are more general than particular. He felt that the only way to resolve this problem is to carry out tests. The possibly transient nature of these changes necessitate immediate post-dive testing and he speculated that the findings might not be specifically dive-related, but may be a general stress reaction. Measurements were the only way to distinguish. He suggested that some of the questionnaires, such as Broadbent's Cognitive Failures test, which are specifically designed to pick up more subtle symptoms of memory lapses, should be used routinely. They are well known and easy to use.

Professor McCallum wondered if this was a good time to raise the question of cooperation as one of the problems is the relatively small number of subjects in different countries. Should we try and establish

some common pathway for neuropsychological tests and pre-dive examinations?

Professor Bennett mentioned that they used up to 50 tests but felt that the 5 or 6 tests presented by Dr Becker provided a good core to which other tests could be added. This would give a good basis for collaboration and pooling of data between Institutes.

Professor McCallum wished to distinguish some areas where a measure of cooperation could be arranged. He felt slightly worried about the air diving history of the deep divers and asked how far back in the subject's career should we be looking.

Professor Bennett stated that he would very carefully screen all deep dive subjects to uncover any problems that may arise afterwards. It is uncertain whether any company would take such measures due to the expense of putting every diver through a neuropsychiatric screen.

Dr Peterson questioned Professor Bennett on the psychological profiles carried out before Atlantis IV, and asked if anything had been detected which might have forewarned of the problems which arose later. Professor Bennett replied that the divers were passed fit to dive and unlikely to present any problem. Dr Peterson asked about evidence of introspection or tendencies that might have produced the events, and was again told that there was no such evidence. Dr Peterson then recounted a story of a professional diver who had similar effects, although over a shorter time, indicating that Professor Bennett's experience was not unique. The question was asked to ascertain if there were any general personality traits that could be used as an indicator of potential problems.

Professor McCallum inquired if submariners are subjected to this kind of test before they are allowed to go to sea and was told they were not. Being largely volunteers, they represented a self-selected group. Dr Becker informed the meeting that the US Navy does screen submariners as part of its personnel reliability and nuclear weapons programme. Professor Donald stated that there was no formal screening of Royal Naval submariners other than the normal personnel selection procedures.

Dr Sunde from the Norwegian Directorate of Health wished to ask the meeting's advice on two of his problems. Firstly, at what stage should more extensive tests be carried out on divers? Secondly, what

qualifications should be expected from the doctors who carry out such examinations?

Dr Nome questioned the approach to problem areas. As he saw it, we had agreed to carry on collecting data until we can reconvene in 15-20 years to decide whether there is or is not a problem. He felt that we should try and answer the question of common protocols and examinations, or at least start such a process. He reminded the meeting that the normal yearly diving medical does include a neurological examination. If it was not sufficient, then should we change the examination, or employ a specialist neurologist. Should there be a cut-off depth above which the present examination is satisfactory, but below which a more extensive one is required? The question of neuro-psychological examinations is at present only addressed by highly specialised groups and Dr Nome felt that some simple battery of tests such as those proposed by Dr Becker could be carried out on all divers, perhaps in cooperation with clinics or institutes.

Professor Donald remarked that Dr Værnes had already commented that relatively simple psychological tests could be evolved. Dr Baddeley agreed that the tests were available; it was more a question of which tests to choose. As we had more questions than answers, it would be prudent for the investigators to use some core of tests to allow compatibility.

Professor Donald then proposed that, if these relatively simple tests existed, perhaps the routine diving medical should include them.

Prof Elliott reminded the meeting that they were concerned with divers who dive deeper than 250 metres, and was Professor Donald suggesting that approved doctors carry out these tests on all divers? He felt that the use of such a screening battery on all divers was going too far, as to date no gross changes have been detected. He did, however, feel that diving companies or institutes using divers deeper than 250 metres should ensure that such screening, including EEG, is carried out. He then turned to the other topic of discussion, namely, the focal deficits seen in certain divers. He believed these to be much simpler to detect and that their significance should be brought to the attention of all approved doctors. The vigilance of the approved doctor is a crucial component of this neurological screening. He summarised by saying that he believed that a physical neurological examination, including

the abdominal and plantar reflexes should be performed pre- and post-dive, as we need to know if and when the reflexes can be lost.

Dr Værnes agreed that the neuropsychological examinations should be performed initially at a clinic using clinical psychologists. Professor Donald believed that if the doctor were able to use elementary psychological tests they would gain confidence in appreciating the more subtle changes. Dr Baddeley expressed doubts about encouraging widespread use of such tests by untrained personnel and believed the long-term solution to be the use of automated test systems with the specialist unit carrying out an ongoing assessment.

Professor McCallum informed the meeting that, as one who has to look at the reports from approved doctors, he found that certain tests, (e.g. lung function tests) posed problems of reproducability and possibly should be done at specialised clinics. Prof Elliott believed that the examination carried out by approved doctors at present was extensive and to add more tests would overload the already busy doctor with tests with which he may be unfamiliar. Dr Thalmann carried out physical examinations of Navy divers at NEDU and these can take up to 2 hours. He believed that if the diver was normal before and after the dive, it is unnecessary to carry out repeated neurological examinations during the dive. The point of the discussion is, he believed, that if there are no obvious signs present in the diving community of long term damage, then why carry out tests every 6 months between dives?

Dr Nome presented a parallel case of the psychiatric follow-up to the Alexander Kielland disaster which had recently been completed. Each of the 75 survivors living in Norway was visited by a psychiatrist immediately after the disaster and one year later. The report contained one chapter on the evaluation of any possible severe long term mental disorders following a catastrophic situation. It appears that the use of a 20-question questionnaire, involving weighting of the answers, applied as early as four weeks after the disaster can predict with a 90 % probability which subjects will have severe problems 1 year later. He felt this was a tool that could be easily and simply used during routine examinations. He was aware that the experts in the field have innumerable tests they could use, but he put in a plea for a simple batch of questions which could be used by the non-specialist.

Dr Tønjum agreed with Drs Elliott and Værnes that approved doctors had enough work already because of the large seasonal influx of divers in the high season.

Dr Brubakk wished to remind the meeting of Professor Aarli's comments that, even if the signs of neurological damage disappeared, that did not mean the organic damage had resolved. The absence of symptoms does not mean the absence of damage. Dr Brubakk commented on the discrepancy in the results in which 2 out of 190 divers had changes in one survey but 4 out of 23 in another. He felt we should try to decide who is right and who is wrong.

Dr Giran commented that: the neurological examination is part of the French diving medical; special examination may produce another group of elite divers deeper than 250 metres, and these would be treated as another class of diver; the diving companies dive in many areas of the world other than the North Sea, and there were great differences in the quality of divers in different areas.

Dr Thalmann commented that his point may have been misunderstood. It was his intention to bring the attention of the meeting to the high frequency of neuropsychological testing that would be required to pick up the transient symptoms that might occur. His point was that, if tests are done at some predetermined interval, there is nothing to show that something did not happen between the tests. The subject must be classed as normal. If the subject has complaints in the intervening period, then that is a good reason for most tests, otherwise it seemed to be impractical. He then commented that hidden neurological signs were a contentious issue as we all may have hidden damage and, until the data-base of spinal cord analysis was greatly increased, most people would agree that divers do not appear clinically different from any other group.

Dr Brubakk thought that what should be done now is to start a project to investigate a group of deep divers frequently, by more than one neurologist if necessary, in order to decide on the frequency of examination necessary. He felt that the deep diving proposed in the Norwegian Trench in 1984 would be a good opportunity to investigate the divers before, during and after the dives and would produce some useful data by planning in advance.

Dr Giran asked if the divers involved in the Statpipe (SDP) dive were drunk when they arrived at the hospital. How many were there and were any examined drunk and changes detected? Dr Tønjum re-

plied that 2 of the divers had a hangover. Dr Brubakk indicated that one had vomited during the caloric stimulation, causing the test to be aborted. He was not one of the two with a hangover.

Dr Cox wished to make a firm proposal as there seemed to be enough prima facie evidence that some long-term neurological investigation was necessary. He proposed a pilot scheme, limited to 2 or 3 centres where there was proper neurological control and supervision, and a small number of divers who could be examined in considerable detail. The approach would be to go for quality rather than quantity of information so that it could be used as the basis for any decision regarding further neurological examination generally. He then discussed Dr Calder's work and stressed that the pathological signs must be related to the subject's clinical history, therefore, there must be a careful record of the examinations and documentation of all commercial divers in some permanent repository.

Professor McCallum commented from experience of running such a repository for 20-30 years that there are problems of organization and funding. He also suggested that the data collected should include the divers' actual dives. He believed this to be possible from the divers' log and felt this was a useful method of obtaining data on the kind of diving carried out, as well as it's clinical consequences.

Dr Skogland was very pleased at the positive nature of the discussion. He believed that every diver diving deeper than 250 metres should undergo a broader battery of tests than normal because we know least about this area of diving. He felt that as only about 150 divers might be exposed to depths greater than 250 metres, their medical examination should be carried out only in specialist Institutes in each country. These divers should be followed up regularly once a baseline is established to allow accumulation of data over many years. He suggested that the EUBS sets up a working group to devise the least common denominator, i.e. the core examination, which would be carried out by diving companies and Institutes. His firm would continue to carry out their existing procedures because they know much more than they did before. He was concerned at Professor McCallum's wish to include the actual dive data because he believed the deep and shallow dives have to be converted to some index and such a method does not exist at present, and he felt this could be another area of study for the working group.

Professor McCallum agreed that, to his knowledge, no-one has attempted to develop an index of a deep diver's career including the likely extensive shallow air diving early in his diving life.

Dr McIver reported to the meeting that of 22 air diving decompression accidents, 12 had had Type II neurological decompression sickness. He stated also that a group met two years ago in Bethesda to discuss a baseline for physical examination. They agreed to adopt the CIRIA test examination with an extra battery of tests for deeper divers, and controls to check for more subtle signs. He asked Professor Aarli if the CIRIA examination would pick up most neurological abnormalities, and was told that it would. It should, however, include the cremasteric and perianal reflexes to give a more complete neurological examination.

Prof Elliott wondered if we were beginning to go over old ground again, and wished to steer the discussion on to a more positive approach. He expressed disagreement with Dr Brubakk's earlier comment that there was a difference between the two groups of divers studied. The results from the experiments in France and Norway could be compatible as they were done at different times after surfacing. The transient changes as detected in Norway may well have been over when the French divers were examined. He felt that the way ahead was in two main directions. Firstly, the CIRIA-based neurological examination should be used to give a good sound baseline. Some of the tests should be done during, as well as post-dive, to try to detect the time of onset of any deficit. He thought that this should be done in all divers involved in deep dives. The second direction, which seems to have less agreement, is the performance of neuropsychological and EEG studies. These have previously been done meticulously in a number of centres, but nothing has been found. Therefore he felt that it should remain in the research category. He also felt that it was inappropriate for the EUBS to sponsor working groups as he felt that would be going beyond the Society's terms of reference. It is up to the neuropsychologists to get together and decide on their methods.

Dr Peterson reiterated that the diving due to take place in the Norwegian sector in 1984, will have some form of follow-up, as a requirement is written into the contract for divers likely to dive deeper than 250 metres to have a comprehensive neurological examination in addition to the standard diving medical.

Dr Thalmann informed the meeting that he had a series of dives involving neuropsychological examinations planned for next year, and by the end of 1984 he should have data on 12 divers. As no-one was prepared to come up with a firm protocol, perhaps we should set up another meeting to which we could come back with the data. The only way ahead was to investigate the divers as thoroughly as possible and increase the database for discussion.

Professor McCallum inquired if Dr Smith-Sivertsen wished to comment on the ground covered by the meeting. Dr Smith-Sivertsen replied that he believed that the meeting had produced a fruitful discussion, although not touching on some of the aspects of his opening address. The ultimate value of the conference would only become apparent with time.

Professor McCallum asked if there were any further aspects that this group should underline.

Dr Baddeley believed that at some stage a group of those intending to use the neuropsychological test should get together and decide on a core of tests which should be used in all cases. Professor McCallum believed that this could equally well apply to EEG interpretation. It required a small group with detailed knowledge to discuss this.

Dr Shields commented that several speakers had pointed out the difficulty in amassing data due to the small number of experimental dives available, but pointed out that divers working deeper than 250 metres in the British sector are passing regularly through Aberdeen. If it were possible to screen these divers before and after going offshore, a large volume of data would be gathered.

Professor McCallum believed that special centres in the UK could be used for screening as necessary and he felt that it was important that everyone should know what is going on in other centres. Dr Shields remarked that the Royal Navy carries out routine dives to 300 metres at sea and suggested that perhaps Dr Török could obtain these data.

Professor McCallum then gave the closing address and in this he wished to re-emphasise one or two points and not summarise the whole meeting. He felt we had managed to concentrate on the neurological effects of deep diving, particularly during the compression and stay at pressure. He belived we had been able to separate this from the

neurological decompression sickness effects, although the input from this portion was interesting and useful.

The workshop had not made authoritative pronouncements, nor should it, but he felt it was an ethical problem we had been discussing first and foremost. He sensed the feeling in the meeting was that diving should continue, but there should be increased monitoring of the divers to provide information which can be used as the base for future discussion.

The EEG and neuropsychological test are felt to be deficient because they are still in a research phase and this necessitates that they be carried out by particular centres. He believed that there had been a very good exchange of information and suggested that some summary of the differing views expressed at the meeting will be available in the proceedings, but without any authoritative pronouncements. This it was felt would be particularly useful for people who were unable to attend the meeting.

LIST OF PARTICIPANTS.

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