FIRC 35

No 1984/89

- ☐ NEW PROPOSAL
- ☐ CONTINUING PROJECT
- ☒ FINAL REPORT
- ☐ PROGRESS REPORT

FISHING INDUSTRY RESEARCH TRUST ACCOUNT

TITLE OF PROPOSAL/PROJECT: Abalone diver's health & safety survey

ORGANISATION: Diving Medical Research Labs.

PERSON(S) RESPONSIBLE:

FUNDS SOUGHT/GRANTED

YEAR	SOUGHT	GRANTED
1984/85		$19,003

RELATED APPLICATIONS: N/A

RECEIVED 27/6/1986 DISTRIBUTED ...../...../19...

Secretary
Fishing Industry Research Committee

Dr. Carl Edmonds.

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No. 1 Harrison Street
Cremorne 2090
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24th June, 1986.

The Secretary,
Fishing Industry Research Committee,
C/- Department of Primary Industry,
Edmund Barton Building,
Broughton Street,
BARTON, A.C.T. 2600.

Dear Sir,

Re: FIRTA Project 84/89 Abalone Divers' Health & Safety Survey.

Thank you very mucy for your understanding and patience in waiting for the final report of the above project. It was completed early this year (February, 1986), however, it has taken a disproportionately long time to arrange for all the researchers to complete their papers and reports. This has now been successfully collated, and a copy of the report is attached.

Please feel free to use the report as you wish. I have taken the liberty of including various recommendations within the report, for your consideration. I am also in the process of obtaining a high quality publication of the report - at no expense to the Department of Primary Industry, however, this will take many months to complete, and I felt that the computer printout of the report would be more suitable for your purposes than would result if we were to delay the despatch until the final "glossy" presentation is ready.

The final presentation will take the form of a book, printed by the National Safety Council, but clearly showing the source of the work, i.e. from a grant issued by the Fishing Industry Research Committee.

I also apologise for not being able to squeeze my sort of accounting into your particular format. We don't have such things as payroll tax, compensation, etc. I have, nevertheless, given a run down of the finances of the final half of the project, for your perusal.

The comments which need to be made, as regards the overall operation, are as follows:-

1. The report could only have been completed because of the enormous assistance given by a whole army of specialists, most of whom donated their time and work, without cost. Some idea of the range of expertise that we had available

can be found in the acknowledgements section of the report.

2. Without the backing of the Fishing Industry Research Committee, none of this could have been completed.

3. Despite my initial fears, the abalone divers were enormously co-operative, very enthusiastic, and I feel sure would co-operate with any such projects in the future.

4. The estimate of the funding, as was suggested by me in the initial request, was totally inaccurate. The main reason for this was my own naivety, not realising the costs of such "incidentals" as postage, transport of equipment and x-rays, and the enormous phone bill that results when one tries to co-ordinate a large group of interstate specialists. Nevertheless, we remained within the overall budget by exploiting a great deal of the expertise, which was then given on a voluntary basis. It was also obtained by reducing the fees to various groups, so that the operating costs could be met.

As you will see, the actual expense documented was slightly in excess of the grant, however, this is explained by the fact that some interest was obtained while waiting for the final amount to be utilised. The full grant was used, with the principal investigator reducing his fees to ensure that the books balanced. A great deal of other expenses were incurred, however, these were met privately.

5. It should not be considered that either the principal investigator, or any of the consultants, lost on the overall deal. The academic value to most of the workers, would be considered of great importance, albeit not financial. The fact that much of the work is already appearing in the medical and physiological and occupational journals, gives support to the belief that most of those involved had a great personal interest, as well as an academic expertise.

Thank you again for your enormous assistance in this project, and I assure you of my full co-operation if you need any further advice or elaboration on the results.

Yours faithfully,

CARL EDMONDS
Principal Investigator

SECOND STATEMENT OF EXPENSES FIRTA. 84/89

SALARY & WAGES

McCallum & Associates - Industrial Psychology Consultants	$335.00
Psychometric assessments in field - T. Coulton	400.00
Psychological design, equipment and analysis - T. Coulton	330.00
Technical officers, on site Total	880.00
Secretarial services (admin, typing, co-ordinating, etc.)	480.00
Final preparation of manuscript - computer printout	400.00
Principal investigator	1455.00

	$4280.00

OPERATING COSTS

Including all printing, stationery, office equipment, telephone costs, hiring of equipment, rooms, psychometric test papers, x-ray packaging and transport, computer materials, etc.	886.00
Fleets Fliers and taxi deliveries	495.00
Postage	280.00

	$1661.00

TRANSPORT AND ACCOMMODATION for principal investigator, technical staff, consultants

. outstanding fees for otolaryngologist and staff, East Australian area	239.00
. Melbourne and south Victorian coast areas,	2533.00
. Tasmanian areas, both Hobart & Launceston	3195.00

	$5967.00

CAPITAL

All major items of equipment (>$100.00) were either removed or rented for the project.

TOTAL $11,908.00

TOTAL COSTING OF PROJECT FIRTA 84/89.

	Previous Report	Current	Total
Salary & Wages	2345	4280	6625
Operating Costs	680	1661	2341
Transport & Accommodation	4367	5967	10334
Capital Items	169	-	169
			19469

Fukta 84/89

THE ABALONE DIVER

CARL EDMONDS

JUNE 1986

THE ABALONE DIVER

CONTENTS

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CONTRIBUTIONS

Most of the research material from this survey has been, or is being, presented in specialised scientific publications or at academic meetings. It is included in this text as part of the original agreement amongst the contributors, and in order to give an overview of the whole survey.

The following data has been, in part or in full, presented as follows:-

Chapter 8 in Undersea Biomedical Research Volume 12, No. 3, 1985 and in the Medical Journal of Australia, Volume 255, No. 22, 1986.

Chapter 15 in Undersea Biomedical Research Volume 12, No. 3, 1985.

Chapter 16 in the Medical Journal of Australia, Vol 144, 399-401, 1986.

Chapter 18 Part 1 has been accepted for publication in the British Journal of Industrial Medicine, in press. Part 2 will be submitted separately, in the near future.

Chapters 6, 9, 13 & 19 are scheduled for presentation at the 9th International Symposium on Underwater and Hyperbaric Physiology, in Kobe, Japan, in September 1986, and will be published in the Proceedings of that Meeting and in the Undersea Biomedical Research Journal.

Mr. John Boughton
Mr. Terry Coulton
Dr. Linda Hayward.
Ms Phoebe Holt
Dr. Ann Williamson,

I cannot express my admiration sufficiently for the superb assistance given by the abalone divers, and especially there own representative from
N.S.W.,(Barry MacRoberts),
South Australia (John Kroezen),
Port Fairey (Len McCall),
Tasmania (Carl White),
Malacoota (Norm Land),
Melbourne (Henry Bishop).

Even though I feel greatly indebted to all the abalone divers for the time and effort they have spent in assisting me with this project, I must make special note of some abalone divers who were especially hospitable and helpful.
Norm Craig from Port Lincoln,
Richard Cullenward from Port Fairy
John Smythe from Eden

A variety of people involved with the abalone divers and of value to the project, should also be acknowledged for their assistance. These include
Bob Ramsay of Port Lincoln,
Dr. Wolf of Tasmania.
Frank Traugott of Sydney

Perhaps the greatest acknowledgement should be given to my wife, Cindy. She contributed as general assistant, secretary, typist, proof reader, technician, advisor and deputised for me in many family duties that conflicted with this study.

Acknowledgement must be given to the Fishing Industry Research Committee, who sponsored the survey, and who assisted with some of the financial problems which arose.

I would like to express my gratitude to the National Safety Council of Australia (Victorian Division) and its Director, John Friedrich, who have subsidised the publication of the report, and supplied copies of it to the numerous participants and organisations involved.

Carl EDMONDS
Diving Medical Centre
Sydney

ACKNOWLEDGEMENTS

This survey could never have been performed without the co-operation of a host of individuals and organisations.

First, I would like to acknowledge the enormous assistance given by Barry MacRoberts of the N.S.W. Fishermen's Co-operative and Gary Hamer, Marine Biologist of the State Fisheries Department. It was the enthusiasm and encouragement of these two fisheries officials who have the welfare of the abalone divers at heart, and who initiated our first seminar to the abalone divers of N.S.W. From this contact, the current project evolved.

A group of <u>diving physicians</u>, who have always had the interests of the abalone divers at heart, gave unstinting support throughout the project. These include

Dr. Christopher Lowry - who has worked tirelessly and without reward for divers over the last decade. He was also of enormous assistance in lecturing, diving and planning the whole survey.

Dr. Robert Thomas, Queensland, who conducted previous seminars for abalone divers.

Also, because of their interest in abalone diver safety and assistance in this work,
Dr. Tony Swain, South Australia.
Dr. David Brownbill, Victoria.
Dr. Des Gorman, Royal Australian Navy.
Dr. Peter McCartney, Tasmania.
Dr. Geoff McFarlane, Victoria.
Dr. Bart McKenzie, Queensland.
Dr. John Silver, Victoria.

<u>Otologists</u> who have been very helpful in either the planning or the initiation of the survey, include

Dr. Peter Freeman, Victoria.
Dr. Gavin Earles, Tasmania.
Dr. Phillip Moore, Tasmania.
Dr. Dean Beaumont, South Australia.

<u>Neurologists</u> who have been extremely valuable during the survey include

Dr. Vignandra, N.S.W.
Dr. Robert Hjorth, Victoria.
Dr. Roy Beran, N.S.W.

<u>Radiologists</u> who have been helpful in the planning and interpretation of the survey results, include

Dr.J.K. Davidson, United Kingdom
Dr. Peter Barnes, South Australia.
Dr. Michael Jones, N.S.W.
Professor W. O'Hare, Victoria.
Dr. Tony Beasley, Tasmania.
Dr. Burden, Tasmania,

<u>Psychiatrists and psychologists</u> who have greatly assisted the project include:
Professor Gavin Andrews
Dr. David Ball

INTRODUCTION.

Anecdotes abound concerning the exploits and attitudes of abalone divers.

Stories of deep dives and fearsome encounters are often enhanced socially to entertain the less knowledgeable listener. As a consequence the general impression one has of abalone divers is that they are rather devil-may-care individuals who regularly flaunt the safe working practices adopted elsewhere.

Those who work in the abalone industry know nothing could be further from the truth. A great many hours underwater coupled with the need to operate small boats in sometimes hazardous conditions requires a constant awareness of any threat to personal safety, and has led to occasional impatience with those who proclaimed an expertise that they were often unable to demonstrate in the work place. However, given the opportunity to learn from "real" professionals and those whose diving knowledge is respected, abalone divers prove most willing and attentive students.

The high esteem abalone divers have for Carl Edmonds, Chris Lowry and their associates was amply demonstrated by the attendance at the seminars they conducted.

This study will be of great value to the abalone industry, highlighting for divers and administrators areas of concern, whilst laying to rest some unfounded fears. It is unique in that, for the first time, Australian abalone divers have been recognised as having to contend with rather special industrial problems.

For their sympathetic understanding, genuine concern and, most of all, contribution to the available knowledge, Carl Edmonds and his associates have gained the deep gratitude of our divers, their families and colleagues.

BARRY McROBERTS
National Secretary,
Australian Abalone Producers' Association.

Chapter 1

HISTORY OF ABALONE DIVING *

CARL EDMONDS

ABALONE AS A RESOURCE

The abalone is a seagoing mollusc or sea snail, comprising only one genus "Haliotis" in the family Haliotidae.It looks somewhat like a flat rock with a row of holes drilled on its outer edge, and is often covered with barnacles, moss and other marine growths.

Abalone live on rocky reefs in shallow oceanic waters, usually where there is profuse marine growth. Because they live in the swell zone, Haliotids evolved a strong muscular foot with which they cling to their rocky homesites. It is this foot that is sought as a gastronomic delicacy.

Some species grow up to 30 cm. (12 inches) but most are somewhat smaller. Only the larger species are commercially exploited, and these are usually found on cooler, temperate coasts. Tropical species are usually small and not found in commercial densities. There are over 100 species worldwide.

Abalone breathe, spawn and defaecate through the holes around the edge of the shell. They are vegetarians and eat drifting pieces of seaweed that are broken off by the swell, hence their prevalence in cold,rough waters with heavy surge. Alternatively, if drift algae is scarce, they graze on small algae attached to rocks. They may only move a few metres in a whole lifetime, but if they do crawl, they thrust their head and eyestalks from under the wide part of the shell, and can move at a reasonable pace over a short distance.

Unlike species such as the oyster and garden snail where the same individual can be both male and female, abalone have separate sexes which do not change. Spawning usually occurs over summer and autumn but varies with species and even between locations for one species. Fertilisation occurs in the water and after 3 to 6 days a shell forms and the abalone settles on a suitable substrate, and very quickly adopts the habits of adult abalone.

ABALONE DIVING OVERSEAS

The first mention of abalone appears to have been by Aristotle in the fourth century B.C., who referred to it as the "wild limpet" also called the "sea ear". They were familiar to the Greeks who ate them fried and were given the name "otia" (little ear) by Pliny.

Asia

The oldest substantial abalone fishing was probably conducted by the Japanese; reference is made to the Ama divers in a document dated around 30 A.D. and there are many references to abalone in an anthology of Japanese poetry published in 760. The Japanese called their abalone "awabi".

There are twenty different names for abalone in the countries where they are fished in Europe, Africa, North America, and Asia and the Pacific basin. The best known is probably the New Zealand "paua" and nearly as beautiful is the South African "perlemoen".

The Koreans and Chinese, like the Japanese, had artisanal fisheries predating recorded history. Abalone is considered a delicacy and an aphrodisiac throughout Asia. The nacre of the shell is used decoratively for furniture and jewellery.

America

Abalone were fished by the American indians in California and Mexico for several thousand years as evidenced by shell midden excavations of jewellery and knives as well as whole shells. The modern fisheries in California and Mexico were started in about 1860 by Chinese immigrants who were prohibited from fishing this delicacy in their own country and so were enthusiastic about this industry in their new land. They exported most of the catch to the orient in dried form in the early years, but because of increasiang export taxes on dried abalone, canning plants were established.

The catch was initially taken by wading, but when laws were introduced prohibiting commercial harvesting in waters less than 30 feet deep, the Chinese yielded the fishing to the Japanese who were more orientated to diving. This was done with snorkels and later in "standard rig" (hard hat) until the 1960's, when hookah and SCUBA were introduced. The resource was so heavily fished that a large number of controls were introduced both on sports divers and commercial operators including a total ban on the export of abalone meat in 1945. The return of the sea otter to Californian waters has meant further difficulties because their favourite dietary item is abalone, and divers must work harder to find their catch in areas that are becoming more and more restricted.

ABALONE DIVING IN AUSTRALIA

Apparently the first abalone divers were the aboriginals. The women amazed the white explorers with their capability of remaining underwater for great periods of time. This was in constrast with the aboriginal male who, with few exceptions, could not swim. The aboriginals of western Tasmania used shellfish as a major part of their diet, together with other marine foods. This may have been related to the difficulty in obtaining the land animals, such as kangaroos, in the wet and thickly wooded mountains.

Women would bring sufficient abalone and crayfish to the tribe, staying near he campfires to warm themselves before returning to the sea. They would carry baskets around their waists, woven from white flowering sag, and these were said to be durable. They would descend, hand over hand down the kelp to reach the seabed. They shaped bits of wood into the shape of a spatula, smoothing them with a shell, and used them for separating the shells from the rock.

We do not know how long the aboriginals have been catching abalone, however, it is estimated that this society have been isolated for perhaps 20,000 years from any other human group.

A similar situation existed on the western coast of Australia, where the aboriginal women divers were renown for their aquatic feats, and were exploited by the white settlers, to obtain pearl in a similar manner to their Tasmanian relatives.

During the 19th century there was some harvesting by Chinese migrants and in 1950 a small scale abalone fishing industry was attempted, and failed, in Tasmania.

The Australian abalone fishing industry is now the world's largest. Despite this, it has really only been a significant commercial industry for approximately 20 years. There were some earlier attempts, and it is thought that exploitation of the abalone in Australia may have begun before 1960, but there is very little information regarding this. The first record of sale of abalone was in New South Wales at the sydney fish markets in 1960. In January 1961, there were three boxes of abalone consigned to the Sydney fish market, and were sold for 37 cents per kilogram live weight.

In 1961, Reid Industries Pty. Ltd., a Sydney canning firm, obtained supplies of abalone, and an order was placed by Malaysian buyers to obtain more of this food. The first batch was dispatched in 1962, and there has been a gradual increase in the industry from that time.

Jack Lucas in his boat the "Tradewinds", carried out a survey along the New South Wales coast and then into Victoria and Tasmania, identifying the abalone grounds. It was at this stage that many of the first abalone divers commenced work professionally. They were basically itinerant fishermen moving up and down and around the coastline, looking for new fishing grounds, as each became "fished out". They lived in caravans, and worked usually only 2 to 3 days per week. In the Tasmanian region, determination as to whether they would move was made more by weather, as in Tasmania the area was prolific with these shellfish, and the main determinate was the ability of the diver to reach the area.

The public image of the abalone diver was one of a very extroverted individual, with the character of a pirate and living a maverick lifestyle. He would wander from coast to coast chasing weather, women and wealth. The image was probably accurate in those earlier days, but that type of person did not survive, perhaps due to the effects of the lifestyle, the cost of the equipment, the combined effects of drugs and alcohol and the great disruption to normal social life. The gypsy lifestyle, and the absence from home for long periods, was said to have resulted in a marriage failure rate approaching 90%. The activity was not noted for its longevity. Divers would tend to be considered fortunate if they survived into their late forties, and still be productive.

Fortunes were literally made on good days, and spent in the hotels during the bad days. One newspaper branded them as "Beach bums and sea hippies, a freewheeling bunch of nonconformers who don't give a damn for their detractors, the authorities, the sharks or any other perils of their unusual calling". There were some catches in excess of 700 Kgs meat (2000Kg live weight). The average daily earnings were probably well in excess of twice the weekly average earning of other occupations.

Very rapidly, some other factors became evident. The abalone which could be so easily collected from the shallows were rapidly exploited, and there was considerable public concern regarding the possible extinction of the species. Certainly by the late 1960's, the depth from which abalone were being taken had increased to such a degree that surface supplied compressed air diving was required.

A number of anecdotal reports were made regarding the problems encountered with abalone diving.

These reports inferred a very high degree of occupational illness. A Mallacoota (northeast Victoria) report in 1976 stated that 42 cases of bends had been treated over the preceding four years. Of the 30 divers in the co-operative, 20% had incurable asceptic bone necrosis (dysbaric osteonecrosis), 30% had chronic problems involving ear damage, 10% had suffered brain damage, and 90% had reduced respiratory function. It was also stated that 25% suffered from sinus problems initiated by diving. The same report suggested that dysbaric osteonecrosis may have been as high as 60% in other areas not served by recompression chamber.

Australian Fisheries,(3) an Australian government magazine reported a survey on professional abalone divers which showed 60% suffered from partial deafness, 50% from lung damage, 12% from dysbaric osteonecrosis and 12% from "arthritis". Fishery managers in three of the four southeastern states responded to the problems of heavy fishing and the effects on divers' health by introducing restricted fisheries. New South Wales did likewise when it obtained the legislative power to do so in 1979.

These restricted fisheries permits and licences are saleable and depending on the price of abalone, enormous premiums are attached to the permits, currently up to $400,000.

The very considerable capital outlay required for the abalone diving, together with the change from the traditional gypsy lifestyle to one in which the divers would live in one area, marry and have children, and dive only around that area,resulted in a very different group of personalities developing. Instead of diving wherever the weather and the fishing grounds permitted, it changed so that divers would be limited in the amount of the catch that was available. Because they were obliged to purchase licences at considerable expense, they tended to become more respectable members of the local community. This not only meant that they would dive less extreme profiles, but also that they would become more knowledgeable and concerned about the problems that seemed to plague their predecessors.

Specific reasons for this recently expressed concern about their long term medical problems include:

- the expense of life superannuation and "loss of income" insurance policies;

- high financial commitments and the need to maintain cash flows to service substantial borrowings;

- the increasing abundance of evidence on the negative health aspects of intensive, long term underwater work that abalone divers perform (almost uniquely).

Administrators too, have recognised the problems and have incorporated divers' welfare and/or a reasonable return to labour, risk and capital in their management plans, and no longer merely manage the fish resources alone.

Local variations

There are 13 species of abalone in Australian waters. Three are harvested commercially, the blacklip (H.ruber), greenlip (H.laevigata) and Roe's abalone (H.roei).

Blacklip abalone favour colder waters beneath large boulders and clefts in cliff faces. They become smaller, on average, as latitude decreases north of Tasmania so divers in Victoria and particularly New South Wales must dive longer to take the same weights.

Western Bars Strait is the transition zone between blacklip and greenlip areas. Greenlip abalone grow more in sandy environments on patchy reefs with strong currents and slightly warmer waters. Hence diving conditions in South Australia are different to those on the east coast.

In some areas of Australia, it is still possible to obtain large catches of abalone in shallow depths, i.e. from breathhold diving in the top 30 feet. In other areas such as Tasmania, the 30 - 60ft. area has been much more heavilly fished. In less than 30 ft. there is a great deal of surge and decreased visibility, with associated enormous changes in the pressure and its effects on divers. Beyond 60ft. it is much calmer for diving, but has the inherent problems with decompression sickness and dysbaric osteonecrosis.

In areas such as Eden, although the divers are aware that they should ideally commence diving deep and gradually move into more shallow waters as the day progresses, the sea conditions often make this impossible. In fact, the seas usually increase during the day, and therefore it is very difficult to dive close to the shore in the shallow areas towards the end of the day due to the surge. They may therefore have to dive in the very opposite manner, i.e. shallow diving when the seas are calm, in the mornings, and moving deeper as the day progresses, and the sea conditions deteriorate. This increases to a considerable degree the danger of decompression sickness.

* Special acknowledgement for the preparation of this chapter must be given to Gary HAMER, of the Division of State fisheries, New South Wales Department of

Agriculture. As an ex-abalone diver, turned marine biologist, he is well respected by both former and current colleagues, and inspired the commencement of this survey.

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Chapter 2

ABALONE DIVER SURVEY

CARL EDMONDS

In Australia, abalone is found mainly in the southern half of the continental coastal areas. There are now over 300 potential licences allocated for professional abalone divers.

In many cases, the older divers were ex-fishermen who had then employed their boat handling skills to undertake abalone diving when their own industry became less profitable. In other cases, and especially with the younger divers, they had moved from having an interest in the amateur diving scene, to employ these skills as a professional diver.

Even though these licenses are available, they do not represent the number of abalone divers. In some cases the licences are held by divers who are no longer active, but who may be retaining them for a variety of reasons, including the possibility of; capital gain, the passing of the licence to a friend or relative, or the hope of resuming diving activity at some time in the future. Occasionally, the licences are simply hoarded for their own sake. In a number of states, the authorities require the newer divers to possess two licenses (attempting to maintain sustainable stock yields).

Thus the survey had a potential population of well under 300 divers, of whom 152 were able to be assessed.

GEOGRAPHICAL DISTRIBUTION

The abalone diving licences are distributed as follows:-

Tasmania	125
Victoria	90
N.S.W.	57
South Australia	35
Western Australia	14

The most populous State for abalone divers is Tasmania, possessing 125 professional abalone diving licences. This is also the state which produces the largest amount of abalone for the Australian export market.

To give an approximate geographical overview of N.S.W., approximately half the licences in New South Wales are held in or around the area of Eden.

South of Eden, when crossing over into Victoria, one finds that a small but active group of 26 divers are based in Mallacoota. Travelling further west along the Victorian coastline, one finds approximately 48 of the divers centred around Melbourne and Port Phillip Bay, whereas 16 are in western Victoria, near Warrnambool, port Fairy or Portland.

In South Australia, there are 35 licenced divers, with 20 holding licences for the area between Port Lincoln and Ceduna.

There are also some islands around the southern coastline of Australia which have only a few divers, who were not able to be examined for logistic reasons - even though they are incorporated in the above numbers.

Although 14 licences are held in Western Australia, especially around the south western coastline, these divers were not included in the survey because of the low cost effectiveness of taking staff and researchers to such a remote area. There are some licences held further north, but the fishermen there are basically surface swimmers, free diving or fishing very shallow depths with compressed air.

From the above figures it can be seen that there was a maximum population available of well under 300 abalone divers, and of this potential the survey sample encorporated 152 subjects.

TERMS OF REFERENCE

The logistics of the survey was dominated by the need to maintain the co-operation of the abalone divers. As a group they tend to have a healthy disrespect for authority, and especially when that authority could interfere with their commercial livelihood.

It was necessary to stress that the survey would be held with total confidentiality as regards the findings on any specific diver. Reassurance had to be given, repeatedly, that no specific information about any identifiable diver would be supplied to the local or state authorities responsible for the licensing of the diver, or to any other authority, be it medical or from the fishing industry. It was evident that the abalone divers did not wish for unsolicited experts to be imposed upon them, and did not wish any legislation which they themselves had not formulated.

This may have seemed a little paranoid, but it is possibly understandable, considering the widespread attitude amongst the divers that the only interest the government has in them is as a source of funding for the government treasury.

This belief is typically expressed by one of the divers (Knight, 1985, in his publication on Abalone Diving in Tasmanian Waters): "The predatory actions of the Department of State Theft must be fought and endured. Taxation has destroyed a lot of private enterprise in this country, and contributed to the mass of unemployed.... When the amount of money earned varies in direct proportion to the damage inflicted upon the body, one can be excused for having strong views. In fact, I have a deep and well nourished loathing of bureaucrats and politicians who merely occupy space and have mass.... Individuals who sit smooth bottomed and pompous, lining their own pockets, consuming food and producing excreta....Greed governs the depth an abalone diver works. Not merely his own, but also the taxation department's.

There is a widespread disquiet with the amount of money is diverted to the government for a variety of reasons (including licence fees), with the divers having an almost universal belief that they are not receiving any service or recompense for their unwilling contributions. There was also a suspicion in some areas that the survey might be a way of removing unfit divers from the industry.

In fact their fears, at least regarding this survey, were not warranted. At no stage was there any pressure to disclose information on any specific diver to any authority, and nor has there been any request for the preparation of legislation to restrict abalone divers in any way.

The contrary was the case. I have been approached on a number of times by abalone divers or their representatives in an attempt to utilise the findings of the survey to promote the divers' own interests.

Neither I nor any members of the survey team had any official medical responsibility for the divers, nor did we have any power over their fitness to dive or their ability to obtain licences. This fact was finally appreciated by the divers, who probably accepted the major researchers as fellow divers more than officials.

The one requirement placed apon our actions was that travel and accommodation expense records had to be kept to obtain reimbursement, and this was limited to a previously agreed amount.

METHODOLOGY

The field survey was conducted in eight separate sectors. In each instance it was necessary to work through the representative of the divers in each area. In most cases, this was a professional diver, but in some cases, it was a fully employed representative of the local fishing industry.

The divers were acquainted with the overall format of the survey, and were informed that they would be asked to fill in questionnaires, undergo medical history taking and examination, and be exposed to a variety of investigations. It was made clear, to us and then by us, that there would be no invasive techniques performed, and there would be no attempt at compulsion in forcing any of the various investigations pertaining to the survey.

In return, after the interviews had been performed, the divers would be given a series of lectures and presentations to extend their knowledge of recent developments in diving physiology and medicine. The lectures given to each group were designed to be be attractive to them and be applicable to their particular diving area. Ample time was left for questions and discussions during the meetings. Otherwise the task was very time critical, with all the researchers aware that we were investigating too much, too quickly.

Attempts by various other interested parties to attend the survey e.g. local doctors, paramedics. amateur divers, etc., were strongly discouraged. The reason for this was that we felt information from the abalone divers would be less forthcoming if local authorities were seen to be present. As an alternative, an offer was made and accepted in most areas, to lecture to the amateur divers, and the local medical and paramedical groups.

In each area there were between 3 and 8 survey researchers. The number and the type of researchers depended on what was being specifically investigated with that group. Although the questionnaires and some investigations were applied to all divers, some investigations were varied; some were dropped and others were added, on the basis of previous findings.

The diver was first given a questionnaire, as depicted. The questionnaire was mainly for identification and as a basis for the subsequent consultation in which the diving history, profiles and problems could be probed in greater detail.

The clinical examination included a brief neurological, respiratory and otological examination, and then a series of specific investigations were performed, including respiratory function measurements, pure tone audiometry, neuropsychological batteries, etc.

During the consultation an open-ended question was asked regarding any medical problems that might be associated with diving. The results were recorded, even though they could not fit into the standardised format.

Specific and predetermined questioning was carried out regarding otological and orthoepedic problems, so that the information could be analysed under these sections.

As can be appreciated from the methodology, it was necessary to utilise a reasonably large area, with a variety of consulting rooms and support personnel. Sometimes these were available from the local hospitals, universities or community centres. It was also necessary for divers to travel long distances (without financial recompense) and arrange accomodation for the 2 day seminar.

Following the examination of each diver a decision was made as to whether further specialist investigation was indicated, and this facility was arranged previously with a variety of consultants knowledgeable in diving medicine.

In some instances, the consultant specialists who were interested in the survey, were actually available at the time. In other situations, referrals had to be made to the closest major medical centre.

DATA HANDLING

The results were compiled independently by each of the researchers. Apart from the personal data and diving information, each of the researchers handled their own specific data and presented this within their own academic units. Some of the researchers were not completely knowledgeable regarding diving activities and terminology, however it was felt that there should be no modifications to their final presentations to increase the sophistication of their work. As an example, a physiologist expert in regression analysis, is not necessarily as competent in evaluating the diving stresses.

The diving data, and the diving physiology has been dealt with only by clinicians who are accepted as experts on diving medicine, by such a designation from the Royal Australian Navy, and by possession of a Diploma of Diving and Hyperbaric Medicine issued by the South Pacific Underwater Medicine Society.

As a general rule, I attempted to pair a diving medical expert with at least one specialist in the branch of medicine that is being assessed in its relationship to the diving.

The results of various aspects of the survey are presented both to speciality medical and physiological publications and in a collected text. The information may be reproduced by application to the various researchers.

Chapter 3

THE ABALONE DIVER, A PROFILE.

Carl EDMONDS

The divers averaged 38 years of age, and were of normal build, with a body mass index of 25 (normal for Australian male population between 35-39 years = 25.0). They averaged an alcohol intake of between 12 and 13 cans of beer (375 ml. with an alcohol content of approximately 5%) per week, and 7 cigarettes per day. The exact figures are as follows:-

	Average	Standard Deviation	range	Number
Age	38.1	7.8	23-63	152
Body Mass Index	24.9	3.0	18.9-33.4	139
Cans of beer/day	1.7	2.2	0-10	152
Cigs.per day	7.1	13.3	0-99	152

Diving Exposure

Professional abalone diving had been possible for approximately 20 years, although there were many fewer licences available in the initial part of that period. Of this group of 152 divers, the average duration of diving was over 16 years, whereas over 12 years were spent professional abalone diving. During that time as a professional abalone diver, the average diver spent over 5 hours per day diving on compressed air (Hookah), for over 105 days each year, reaching just over (15.25 metres) 50 ft. on a typical day. He claimed to have been bent over 4 times, but probably did not recognise the less dramatic types of decompression sickness.

	Average	Standard Deviation	Range	Number
Years diving	16.1	7.75	0.4-35	152
Years abalone diving	12.1	7.1	0.1-31	152
Hours diving per day	5.2	1.0	1.5-9	152
Days diving per year	105.8	30.3	28-200	149
Maximum depth on an average day(feet)	50.2	17.9	15-100	152
Decompression sickness incidence	4.1	12.2	0-100	152

When attempting to assess the safety of the dive profiles, it was appreciated that we were reliant apon the subjective reports of the divers. There was no doubt of the depths in the area, as these were available from the maritime charts. Also the divers were very experienced boatsmen, very well acquainted with the area. In some areas we were fortunate enough to have dived with the abalone divers, and our observations supported their claims.

Nevertheless the reporting was anecdotal, and had none of the precision of another survey recorded on six hardhat shellfish divers of Japan, by Nashimoto et al (1986). They utilised a dive profile recording system involving a complex underwater computer/recording system.

It is unlikely that the divers were unaware of their factual diving profile. It was possible that they may have been expansive in their claims, however this would have been counterproductive if they believed the researchers to represent officialdom. The impression most wished to leave was of their knowledge of, and compliance with, safe diving procedures. This impression did conflict with other information.

Based on the reported dive profile from the 152 professional divers, a grading was given according to the theoretical likelihood of producing decompression sickness or bubble formation. The following arbitrary grading was made:-

Grade 1 No decompression diving, less than 10 metres (33 ft.)
Grade 2 Diving within the conventional U.S. Navy Tables. No decompression.
Grade 3 A reasonable diving protocol but one which did not necessarily comply with standard decompression tables.
Grade 4 Diving with an omitted decompression of less than one hour, according to U.S. Navy Tables.
Grade 5 Diving with an omitted decompression of one hour or more according to U.S. Navy Tables.

In each case, the grading was made on the typical dive profile described by the diver. It was presumed that the grading represented the degree of "decompression stress".

The following frequencies were found with the various grades of decompression stress.

Grade 1	20	13%
Grade 2	20	13%
Grade 3	23	15%
Grade 4	67	44%
Grade 5	22	14%

It can be seen that 58% of the divers employed a dive profile which required some time for decompression, but which was omitted.

Official training in either professional organisations such as the Royal Australian Navy or amateur organisations such as the Professional Association of Diving Instructors or the Federation of Australian Underwater Instructors, was very rare. Nor was there any obvious relationship between adherence to safety regulations and such training. On the contrary, it was a Royal Australian Navy trained diver who introduced the deep underwater oxygen regime (60 foot, 18metre) for decompression, in the belief that it was a conventional and established technique.

Of some interest was the divers' own assessments regarding their decompression profiles and requirements.* The following assessments were made by the divers:-

24.5% stated they never did dives that required decompression.
41.1% stated they rarely did dives requiring decompression.
34.4% stated they commonly did dives requiring recompression.

The above information must be appreciated as being very subjective. When the divers were asked to describe what type of formal decompression regime they followed, the following results were obtained:-

13.8% claimed they followed the U.S. Navy Diving Tables.
8.6% claimed they followed a modified U.S. Navy Diving Table.
0.7% claimed they followed the Royal Navy Diving Tables.
17.8% followed a decompression meter.
59.2% admitted they followed no formal decompression tables.

There were 69 instances of decompression sickness that were treated by recompression therapy in a recompression chamber. Of these, 39 appeared to be neurological in nature.

Other diseases which could well have influenced some of the interpretations of the survey, included both near drowning and carbon monoxide poisoning. In 23% of divers, there was an episode which could have led to drowning, and which resulted in the diver aspirating a large quantity of sea water, but in only one case was there actually a loss of consciousness following this. In 19% of the divers in the survey, there was a history of carbon monoxide poisoning, but in only one case did this lead to unconsciousness.

As there was only one case each of carbon monoxide poisoning and near drowning resulting in unconsciousness, it was presumed that these illnesses would not have contributed significantly to the overall result of the survey.

The greatest depth to which the divers had dived for abalone, which could well be a measure of the diver's personality or knowledge as well as his diving ability, shows a normal distribution. The depths, which have almost certainly been "rounded off" by the divers, are as follows:-

40 ft.	2.7%	50 ft.	4.1%
60 ft.	7.5%	70 ft.	9.5%
80 ft.	12.9%	90 ft.	12.9%
100 ft.	19.7%	110 ft.	6.1%
120 ft.	15.6%	130 ft.	3.4%
140 ft.	4.1%	150 ft.	1.4%

Over the last few years, there has been an increasing use of oxygen, with far more oxygen being used in some areas, e.g. South Australia, than others, e.g. the eastern coast of Australia. The overall incidence of the availability and use of oxygen on the boat is as follows:-

Never	78.1%
Surface	8.6%
Underwater (less than 30 ft/9m.)	7.9%
Underwater (greater than 30 ft/9m.)	5.3%

It can be seen that 13.2% of the divers used oxygen underwater, either for treatment or prevention of decompression sickness. The 5.3% who used it in excess of 30ft., restricted the depth to a maximum of 60 ft., even though this has obviously been exceeded in some specific case reports.**

If one compares the diver's claim that he did not do dives which required recompression, together with the assessment of the dive profile as estimated in the decompression stress, one finds the following information:-

Of the 37 divers who stated that they never did dives that required decompression, 5 of them routinely required decompression staging of less than 60 minutes, and 4 of required decompression staging of 60 minutes or more, based on their own stated dive profile.

Of the 62 divers who claimed they rarely dived in such a manner as to require decompression staging, 23 routinely required decompression of less than 60 minutes, whereas 6 required decompression of 60 minutes or more.

The corollary of this would seem to be that, of the 99 divers who believed they never or rarely dived in such a manner as to require decompression staging, according to the U.S. Navy Tables, 28 would have routinely required decompression of less than 60 minutes, whereas 10 would have required decompression of 60 minutes or more. Thus, over one third of this group of divers were unaware of their decompression requirements.

__Summary__ This survey encompased 152 professional abalone divers, with the average diver being aged 38, having a body mass index of 25, having dived for 12 years as an abalone diver, usually to a depth of up to 50 ft. on an average day, with over 5 hours underwater, for over 105 days per year.

More than half the divers omitted required decompression on their average dive. 38 of the 99 divers who believed that they did not require decompression, in fact, did have such a requirement, but they were unaware of it. Over half of the divers had gone to 100 ft. or more to catch abalone.

They averaged over 4 cases of decompression sickness per diver, and there were 39 instances of obvious neurological decompression sickness in the group. Recompression therapy was administered in a pressure chamber in 69 instances, but many more were treated underwater.

They averaged 2 cans of beer per day, and 7 cigarettes per day. Over 13% used oxygen underwater, for either decompression or therapy or both. 5.3% used it in excess of 30 ft.

REFERENCES

NASIMOTO I, KOBAYASHI K, GOTOH Y. 1986. An appraisal of dive profiles in shellfish divers with reference to the risk of decompression sickness. Proceedings of the European Undersea Biomedical Symposium 1985, Sweden. In Press

*DECOMPRESSION TECHNIQUES

By far the most common method of determining what diving profiles were possible, was to try it and see. A fairly common variation is to gradually increase the diving duration or depth until minor symptoms develop (often not considered by the diver to be decompression sickness as they were not severe enough!) and then withdraw to the non eventful profiles.

Other claims were more picturesque e.g.
Regarding diving durations at any specific depth - "I double them".
"I dive until my arms and shoulders get stiff"
"If I can't see the bottom from the top, I don't go".
A clicking in the right shoulder indicated to one diver that he reached his maximum permissible depth.

There is a widespread belief amongst abalone divers that they acquire a partial immunity against decompression sickness because of their excessive diving. They presume that decompression sickness is reduced by 50% after a weeks work at any specific depth, and by 60% after 2 weeks (Reference: Knight M. Abalone Diving in Tasmanian Waters, 1985)

Case Report. Dived all day at 60 ft on his birthday, and then noted on returning to the boat, that he had a few aches in his joints. He could not steer the boat, became unconscious, then woke up with a great deal of pain. He could not see very well but "battled" the boat back to shore. He was given oxygen which improved his state very rapidly, and after he took the oxygen off he became very bad again. He used it on and off for the rest of the night.

Case Report. Every night for the first three or four days of diving after a spell the diver noticed parasthesia, numbness, vague aches and pains. Decompression approach was to extend diving until he got obviously bent, then to back track a little.

Case Report. Never had bends, but often gets severe headaches, especially after diving too deep, like 55 feet for 5-6 hours. Also gets deep aches in the bones of the right hip, left shoulder and left knee, for a few days.

**OXYGEN BREATHING

Case Reports.

While breathing oxygen at about 40 ft. his lips go "funny" and he notes a tingling or numbness over his body.

Uses oxygen underwater, because of his navy training. The maximum depth and duration would be one hour at 50 ft. During this time he continues his collecting of abalone, and sometimes notices his right arm twitching and jerking, loss of sight, the appearance of starlight objects, twitching in the mouth and body. He claimed never to have lost consciousness underwater, however, other observers state this is not correct.

Lost consciousness after a few minutes (probably many minutes as he half filled his ab bag) at about 60 ft.

Never takes oxygen in excess of 60 ft, and tries to reduce the oxygen duration even at shallower depths, to less than three hours.

After breathing oxygen for more than ten minutes at 60 ft., his eyes went swimmy and fuzzy and tended to twitch. Knows that it is then time to quit.

Chapter 4

General Recommendations from the Survey

Perhaps the most common criticism voiced by the divers, was an absence of technical and diving information. The second was a distrust of officialdom. The third involved conflict amongst local divers, representatives and commercial distributors of the product.

Uniform legislation and control of the industry is impossible because of the contentious commonwealth/state rights and responsibilities.

Because of the interaction of these concerns, and because they aggrivate each other, remedies are not simple. In my opinion the divers, the representatives and the fisheries organisations are all easilly misunderstood by each other. Most had a genuine interest in promoting both the divers welfare and the industry promotion. Isolation and a lack of understanding of each others problems resulted in much suspicion. This would not usually concern an occupational health and medical survey, except that it may interfere with implementation of recomendations, and has inevitably influenced these recommendations.

Recommendations

1. Periodic seminars between the divers and practically orientated experts be made available (not obligatory). This is already undertaken in some areas, and they have been well attended. Health and diving safety subjects could be integrated with equipment and fisheries subjects, so that the overall attractiveness would be heightened.

Non medical subjects could include: Compressor technology, maintainance and testing; diving equipment; abalone recruitment; marketing; etc. The lecturers in some of these subjects would be funded by their commercial firms or government departments.

The small number of lecturers could be organised to visit 6-7 areas consecutively, in a two week period and cover all abalone diving groups. With adequate warning, diving could be suspended for 2 days to allow for these seminars and other local meetings. Such an arrangement would be efficient as regards lecturer participation, as preparation of one lecture would allow 6-7 presentations. Questions from one area could then contribute to presentations in subsequent areas.

2. Periodic literature in the form of an annual or semi-annual newsletter covering practical subjects in non-technical terminology, and with contributions by experts in a variety of subjects related to abalone harvesting, should be available to all licenced divers.

The subjects covered should include the same as those referred to in the above seminars, and could even be transcripts of them. It could also be used to inform divers of such meetings. The literature should be sent direct to the diver, to ensure that local administrative difficulties or political animosities do not intrude.

3. Specific practices which are inherently dangerous, such as the inappropriate use of oxygen underwater, could be discouraged by implementation of the above recommendations.

4. A Central Registry which is able to compile and analyse medical records of divers, would allow a longitudinal study of health problems with this form of professional diving. This would also be of great value to the individual diver as it would allow for early baseline medical data to be compared with subsequent

assessments. It would also ensure a standard of investigation and reporting that is quality controlled.

Individual medical information would only be available to the diver or a representative designated by him (e.g. local doctor).

This Central Registry would be comparable to U.K. Decompression Sickness Panel and the Japanese group from the Saitam Medical School. In Australia, it would have to be an independent organisation, unassociated with state or commonwealth government departments. Otherwise it will be interpreted with suspicion by the very divers it is intended to help.

CHAPTER 5

DYSBARIC OSTEONECROSIS IN DIVERS - A REVIEW

C. J. Lowry

INTRODUCTION

Infarction of areas of bone associated with exposure to pressure, be it in air or water, has been recognised since the turn of the century. A causal relationship was first suggested by Twynam in 1888 in a case report of a caisson worker constructing the Iron Cove bridge in Sydney although in retrospect the man appeared to be suffering from "septic" necrosis.

Although there were many reports of bone necrosis occurring in caisson workers, the first report on a diver appears to be by Grutzmacher in 1941 (quoted in Elliott and Harrison 1971). Detailed studies of the prevalence in divers were not undertaken until the 1960's and 1970's and a wide range of incidence has been reported.

Although rare, several cases have been reported in aviators not exposed to hyperbaric conditions.

Various names (see table 1) have been given to this disease but recently the term dysbaric osteonecrosis has gained precedence as it clearly distinguishes the causal relationship with pressure from the other causes of bone necrosis.

TABLE 1

SOME SYNONYMS FOR DYSBARIC OSTEONECROSIS
Caisson arthrosis
Caisson disease of bone
Hyperbaric osteonecrosis
Barotraumatic osteoarthroplathy
Avascular necrosis of bone) *
Ischaemic necrosis of bone) *
Aseptic necrosis of bone) *
Arthritis deformans
Diver's bone rot
Diver's crumbling bone disease

* To be distinguished from other causes of bone necrosis (see Table 2)

TABLE 2

SOME DISORDERS ASSOCIATED WITH ASEPTIC NECROSIS OF BONE

1. Decompression sickness
2. Trauma (eg fractured neck of femur, dislocated hip)
3. Corticosteroid effects
4. Arteriosclerosis, Raynaud's phenomenon (occlusive vascular disease)
5. Alcoholism
6. Gout
7. Hyperlipidaemas and obesity
8. Platelet disturbances (eg idiopathic thrombocytopaenia)
9. Gaucher's disease
10. Pancreatitis
11. Polycythemia/marrow hyperplasia
12. Sickle cell disease and other haemoglobinopathies
13. Syphilis
14. Sarcoidosis
15. Rheumatoid arthritis and osteoarthritis
16. Paget's disease
17. Idiopathic (unknown cause)

AETIOLOGY

The exact mechanism for the production of bone necrosis due to hyperbaric exposure has not been elucidated. Whether the incidence of bone lesions is more related to the cumulative effects of hyperbaric exposures or the statistical chance of a single event, is unknown.

The most widely held belief is that dysbaric osteonecrosis is due to the decompression phase and that it is a long term manifestation of decompression sickness. There are, however, numerous variations on this theme. It is sometimes suggested that the infarction of bone is caused by arterial gas emboli produced during decompression. Certainly "silent" bubbles can be detected by Doppler techniques during clinically safe decompression schedules. Several series show a relationship between clinical decompression sickness and the subsequent development of bone necrosis. However it is usually a relationship to either type I Decompression Sickness (DCS) or total DCS rather than serious CNS (type II) DCS which are more likely to be associated with intra arterial bubbles.

Others propose that large amounts of nitrogen are taken up by the fat in bone marrow during longer pressure exposures. During or after decompression, gas is liberated from this fatty tissue and its expansion interferes with blood supply within the non compliant bony tissue. Bubbles have been found in the large venous sinusoids in animal experiments and at post mortem, and may well have obstructed venous outflow from marrow, leading to areas of infarction (Kawashima & Tamura 1983).

Changes secondary to intravascular bubbles, be they arterial or venous, may then take place - such as platelet clumping and intravascular coagulation causing further vascular obstruction. This is supported by the post dive observation of increased platelet adhesiveness and decreased platelet count after certain dive profiles (Davidson 1975, Walder 1980).

There may be a critical period of bone ischaemia after which pathological changes become irreversible. This may help to explain the somewhat "hit or miss" nature of this disease.

Another somewhat more controversial theory attributes the damage to the osmotic changes leading to fluid shifts during pressure changes, especially rapid compression.

Fat embolism from disturbance of cells by disruption or regional hypoxia has also been implicated in the development of infarction.

It is possible that a number of factors may combine to produce necrosis in a given situation and that the aetiology is complex or multifactorial.

Any theory must account for the following observations:-

1) DO may follow a single exposure to pressure

2) Although there appears to be a relationship between DCS and DO not all divers with DO have a history of DCS and not all divers who suffer DCS develop DO

3) Not all divers at high risk develop DO

Various animal models have been developed to study the aetiology of DO because of the obvious difficulties in early detection and monitoring of such a capricious and chronic disease.

One must have reservations in extrapolating the results of these studies to humans.

The importance of establishing the mechanisms involved lies in the development of effective methods of both prevention and treatment.

PREVALENCE:

Prevalence figures should be taken as a rough guide, since different radiological techniques and diagnostic criteria could have been used by the radiologists employed in each survey. There have been numerous reports of the prevalence of DO in compressed air and workers. These appear to show a reducing incidence with the development of more conservative decompression schedules. The difficulty in obtaining adequate follow up of workers at risk is often reported. For example at the Clyde tunnel only 241 men were surveyed a total of 1362 workers (55% left after 4 months). Of those surveyed 19% had lesions and more than half were juxta-articular. In 1972 the Medical Research Council Decompression Sickness (MRC DCS) Panel had X-rays of 1674 workers of which 19.7% had positive lesions.

In 1972 a study by Jones & Behnke on the B.A.R.T. tunnelling project in San Francisco showed no reported clinical or x-ray evidence of necrosis. All workers had pre-employment X-rays and those with lesions were excluded. The pressure exposure ranged from 9-36 psi with only one decompression per day. However the follow up period was relatively short.

In divers there is a reported prevalence of 5 to 65%, with different types of diving. (See Table 3)
The lower incidences are reported in military series

where strict decompression schedules are adhered to, as with many commercial diving operations. In the self employed fisherman-divers of Japan and Hawaii, the divers undertake relatively deep dives, with long bottom times and little or no decompression.In these series prevalences up to 65% have been reported (Wade et al 1978).

In a mixed group of 110 sport and professional divers in Australia 28 had definite or suspected lesions of DO although only a small number were juxta-articular and none were severe (Williams & Unsworth 1976).

TABLE 3 REPORTED PREVALENCE OF DYSBARIC OSTEONECROSIS IN DIVERS

Report	Type of Diver	Total No	% Positive
* Ohta & Matsunaga 1974	Japanese shellfish	301	50.5%
Fagan & Beckman 1976	Gulf coast commercial	330	27%
Elliott & Harrison 1976	Royal Navy	350	4%
Harvey & Sphar 1976	U.S. Navy	611	2.5%
Wade et al 1978	Hawaiian diving fishermen	20	65%
Davidson 1981	North Sea commercial	4422	4.4%
Kawashima & Tamura 1983 *	Japanese shellfish	747	56.4%

Footnote: Kawashima & Tamura survey is an extension of the Ohta & Matsunaga survey and they have divers in common.

CLINICAL FEATURES

The earlier stages of DO are usually asymptomatic and are detected by bone scan or x-ray. There may be a history of decompression sickness and/or repeated inadequate decompression.

Symptoms develop when the articular surface is damaged due to underlying osteonecrosis and the development of osteoarthritis in the joint. However, some workers have reported symptoms prior to the development of x-ray changes. The lesions are almost always in the shoulder or hip and may occasionally be bilateral. It has rarely been reported in other joints, eg ankle (Kuipers 1985).

The progressive joint destruction leads to steadily increasing pain and limitation of movement, the features of degenerative osteoarthritis. Whether divers with head, neck and shaft lesions are more prone to develop the joint-endangering juxta-articular lesions is not yet established.

Lesions away from the articular surface of hip or shoulder generally remain asymptomatic although several cases of sarcomatous change have been reported in compressed air workers.

RADIOLOGY

Radiological examination remains the basis of detection and assessment of progress of lesions.

The detection of early lesions demands high quality x-rays of long bones, demonstrating the fine trabecular detail. Early lesions may be difficult to differentiate from bone islands, bone cysts and other variations from normal.

Some studies have shown a higher incidence of bone islands amongst divers whilst others dispute this. (Ohta and Matsunaga 1974, Kawashima et al 1983, Davidson et al 1977). When these lesions have been studied histologically they have not shown necrosis (Davidson 1977). Bone islands are said to be present in up to 40% of the normal population (Davidson 1981).

Radiological surveys should include views of shoulder, shaft of humerus, hips, shaft of femora and knees. The projections recommended by the U.K. Medical Research Council Decompresson Sickness (MRC DCS) Registry are described briefly as follows:

(a) An antero-posterior projection of each shoulder joint. A 30 x 25 cm film is recommended. The patient is placed in a supine position with the trunk rotated in an angle of approximately 45 degrees to bring the shoulder to be radiographed in contact with the table. This arm is partially abducted and the elbow is flexed. Centre 2.5 cm below the coracoid process of the scapula, and cone to show as much humerus as possible, bringing in the lateral diaphragms to show only the head and shaft of the humerus. This view should show a clear joint space, and the acromion should not overlap the head of the humerus;

(b) An antero-posterior projection of each hip joint. A 30x 25 cm film is recommended. The patient is placed in a supine position with the feet at 90 degrees to the table top. The edge of the gonad protector should be as near the femoral head as possible, but not (in any way) obscuring it. Centre the cone over the head of the femur, that is, 2.5 cm below the midpoint of a line joining the anterior, superior iliac spine and the upper border of the pubic symphysis;

(c) An antero-posterior and lateral projection of each knee. An 18 x 43 cm film is recommended. Centre at the level of the upper border of the patella. The field should include the lower third of the femur and the upper third of the tibia and fibula. The gonads should be protected. As fine trabecular detail is required underpenetration must be avoided.

The MRC DCS Register system of classification as described by Davidson (1976) is widely recognised. Lesions are divided into juxta-articular (A) and head, neck and shaft (B). These are further subdivided as shown in Table 4. Symptoms are usually only reported corresponding to A4 and A5 lesions.

TABLE 4

U.K. MRC DCS PANEL

CLASSIFICATION OF RADIOLOGICAL LESIONS

JUXTA-ARTICULAR A

A1 Dense area with intact articular cortex

A2 Spherical segmental opacity

A3 Linear opacity

A4 Structural failure

- (a) translucent subcortical band
- (b) collapse of articular cortex
- (c) sequestration of cortex

A5 Osteoarthritis

HEAD, NECK AND SHAFT B

B1 Dense areas (not bone islands)

B2 Irregular calcified area

B3 Translucencies and cysts

B4 Cortical thickening

PATHOLOGY

When examined histologically the area of necrosis is much more widespread than evident radiologicaly. The marrow is acellular and osteocytes are absent from the lacunae. There is evidence of healing and revascularisation with unabsorbed dead bone in the centre of some trabeculae. The revascularisation stops short of completion and a band of dense fibrous tissue is found at the border with necrotic tissue (Davidson 1976). Adjacent trabeculae on the healing side are thickened due to the formation of new bone on the surface of dead trabeculae (Calder 1982).

The articular cartilage usually appears normal in he early stages of the disease but damage occurs when the underlying bone trabeculae collapse. Evidence of osteoarthritis then appears in long standing cases.

MANAGEMENT

The appropriate course of action for divers with type B lesions is not yet established. There is no clear evidence that such divers are more prone to develop type A lesions but they may indicate that their diving pattern is somewhat hazardous. At present those divers should be advised of the x-ray findings and of the need for regular follow up. Some modification of their diving profile may be indicated.

Divers with type A lesions should be encouraged to stop provocative diving but advised that this will not prevent the progression of the disease process. Avoidance of heavy loads and manual work, for up to 12 months,has been suggested to allow reparative processes to take place in order to avoid collapse of the articular surface. Some workers suggest a more active management of the early stages especially of the hip. The emphasis is on early diagnosis by Technetium bone scan (^{99m} Tc), intra-osseous marrow pressure measurements and phlebography,and cone biopsy of the femoral head. It has been suggested that the latter procedure may be therapeutic in relieving intramedullary pressure and arresting progress of the disease (Kawashima and Tamura 1983, Kuipers 1985). With A4 type lesions, correctional osteotomy has been employed. For A5 lesions the only appropriate therapy to alleviate symptoms and improve range of movement is by total joint replacement with a prosthesis.

PREVENTION

Short of abstinence there is probably no guaranteed method of avoiding dysbaric osteonecrosis, although it is most unlikely in diving shallower than 10 metres.

Reduction of the incidence of DO may be achieved by following recognised decompression schedules, oxygen decompression, avoidance of long dives or diving deeper than 30 metres. Education in diving physiology and medicine amongst the commercial diving population would therefore be valuable.

Early diagnosis by routine radiological surveys should allow greater knowledge of the disease process and help identify the divers at risk. Detailed information about all divers should be compiled in a central registry in each country and should include pre-exposure and regular x-rays as well as a personal, medical, occupational and diving history. Risk groups or predisposing factors could thus be identified.

Monitoring of serum ferritin or urinary hydroxyproline excretion has been suggested but these methods lack sensitivity and do not locate the lesion. They may be of more value in testing new decompression protocols.

The use of bone-seeking radio-isotopes such as Technetium methylene diphosphate (^{99m}C MDP) Tc) pyrophosphate is of value in identifying lesions prior to x-ray changes and may be positive a few weeks after the causal incident Beckman et al 1984). Such investigation, while not feasible on a widespread scale, may be of considerable value in selected situations such as after clinical decompression sickness or omitted decompression.

CONCLUSION

Dysbaric osteonecrosis is a serious problem amongst commercial divers affecting a relatively young fit population and endangering their livelihood. Much more information is required in order to be able to advise and manage divers who may develop this disorder.

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Chapter 6

DYSBARIC OSTEONECROSIS - A SURVEY OF ABALONE DIVERS

C.J.Lowry, F.M. Traugott and M.W. Jones

Dysbaric osteonecrosis (DO) is an occupational disease of caisson workers and divers. Although the incidence varies widely in various reports, surveys from other parts of the world reveal a much higher incidence of DO in fishermen-divers than in their military or commercial counterparts (Ohta & Matsunaga, 1974; Wade et al, 1978; Harvey & Sphar, 1976; Davidson, 1981).

Most of the theories of aetiology of DO (see Chapter 5) relate to the consequences of inadequate decompression. Studies have indicated possible relationships between the incidence of DO and age (Kawashima, 1976), inadequate decompression (Ohta & Matsunaga, 1974; Wade et al, 1978) and deep or experimental diving (Harvey & Sphar, 1976).

The diving pattern of the abalone fisherman described in this study appears in Chapter 3. Briefly, compared to other forms of commercial diving the fisherman-diver spends longer periods of time underwater per day and dives more days per year. Notwithstanding, the Australian abalone diver would not appear to dive as deep as his counterpart in Japan (Ohta & Matsunaga, 1974) or the diving fishermen of Hawaii (Wade et al, 1978). The prevalence of DO in those series was 50% and 65% respectively. The larger Japanese series showed an increasing incidence of DO with age and number of cases of decompression sickness.

The purpose of the present study was to determine the incidence of DO in an Australian population of abalone divers and describe any dependence DO may have with such factors as age, tobacco and alcohol consumption, and diving exposure.

METHOD

The long bone x-rays of 108 Australian abalone divers were collected and assessed according to the British Medical Research Council Decompression Sickness (MRC DCS) panel guidelines (Davidson, 1976). In most cases, x-rays from divers had been previously undertaken at local medical centres and later forwarded on for central reporting such that independent diagnoses were received from two radiologists. Those divers who had not had any or adequate previous x-rays were encouraged to attend one of several large hospital radiology departments in Sydney, Melbourne, Adelaide, Hobart and Launceston. X-rays so obtained were also then sent to Sydney for further reporting. For the majority of x-rays the interpretation of the central radiologist (M.W.J.) formed the basis of the present study. Diving and medical data were not available to the reporting radiologists.

Radiological Technique and Interpretation

The radiographs obtained were antero-posterior shoulder views with internal and external rotation of the humeri, antero-posterior views of both proximal femora and antero-posterior and lateral views of both knees including adjacent long bone shafts.

Abnormalities were classified according to the MRC DCS panel classification of Dysbaric Osteonecrosis. Small single, well defined bone densities, usually slightly away from the adjacent joint surface without any other abnormal findings, were regarded as incidental bone islands and not reported at all by some radiologists. The variable trabecular thickening often seen in the femoral neck superiorly at the insertion of the joint capsule or in the medial base of the femoral neck adjacent to the lesser trochanter was regarded as normal and not reported.

In film interpretation there was concern not to over diagnose dysbaric osteonecrosis. Abnormalities were regarded as suspect or definite. Suspect lesions were alterations in bone appearance consistent either with normal variation or early bone lesions, and also clear abnormalities consistent with osteonecrosis but for which trauma would also need to be considered as the cause. Definite lesions were those abnormalities typical of dysbaric osteonecrosis previously described in the literature (Davidson, 1976; Elliott & Harrison, 1971).

Personal and diving information from 85 of 108 abalone divers studied were obtained by interview and questionnaire. (Refer to chapter 3 for a more detailed description of the method.) This data included age, body mass index, total years of diving, maximum depth dived per day, average daily maximum depth, amount of time spent diving per day and per year, number of incidents of decompression sickness, tobacco and alcohol consumption.

Statistical Methods

The data was statistically analysed using Minitab (Ryan et al., 1985) and performed on a PDP Vax 11/750 computer. Standard chi-square tests and one way analysis of variance (Snedecor & Cochran, 1980, Sokal & Rohlf, 1969) provided the statistical tools in which to assess the likelihood that diving or social factors were in some way responsible for the occurrence of dysbaric osteonecrosis.

RESULTS

Of the 108 divers x-rayed, 35 were found to have or be suspected of having bone necrosis in one or more sites resulting in an overall prevalence of 32% (Table 1). Both juxta-articular (A) and head, neck and shaft (B) lesions were included. Bone islands, if reported, were considered innocent and thus counts thereof were appended to form the no lesion group.

Closer scrutiny of these lesions by location, type and number (Table 2) reveals that the overall occurrence of definite lesions are similar in the leg and in the arm. Despite this the number of B lesions found in the leg was 2.3 times greater than that in the arm. By contrast, definite A lesions were 3.7 times more prevalent in the arm than in the leg. No difference could be shown for suspect B lesions between locations. Similarly, little difference by location could be ascribed for the prevalence of either bone islands or no lesions. Where lesions of either type A or B were observed there was a 5 to 12 times greater likelihood that multiple lesions of similar type would exist. There were no suspect A lesions observed in the arm of any diver.

Table 3 describes the prevalence of any type of lesion occurring simultaneously in either the arm or the leg of these divers. In the majority of cases, B lesions observed in the arm were associated with B lesions also in the leg. By contrast, where A lesions occurred in the arm they did so in isolation, there being essentially no similar lesions found in the leg.

Of the 108 abalone divers studied in this trial diving and selected social histories (smoking and alcohol consumption) were completed on 85 of these. This data is presented in Table 4.

This group of mean age 38 ± 7.3 (± standard deviation) years had dived on average for a total of 16 ± 6.9 years, the latter 12 ± 6.7 years actively involved in abalone diving. They would dive on average to a maximum depth of 54 feet and remain underwater for 5.2 ± 1.00 hours per day. This diving profile would be repeated for 100 ± 32.3 days per year contributing to a total abalone diving experience of 7266 hours. Figures relating to alcohol and cigarette consumption indicate that 12 ± 15.6 cans/week (375 mls of approximately 5% alcohol) and 7 ± 11.1 cigarettes per day were consumed by these divers. The proportion of divers who drank alcohol and/or smoked was 90% and 39% respectively.

One way analysis of variance of the possible factors responsible for the occurrence of any lesion (irrespective of type or location) was performed and is presented in Table 5. Factors which may affect dysbaric osteonecrosis include age ($P<0.05$), total years diving ($P<0.05$) and decompression stress ($P<0.05$). All other parameters examined showed no significant relationship to the prevalence of lesions.

A similar one way analysis of variance tests for all the above factors was performed focussing on the arm and leg independently. A significant relationship existed between alcohol consumption and the event of a femur lesion ($P<0.05$). No such relationship occurred in the arm.

DISCUSSION

The long bone x-rays of 108 abalone divers were reviewed. 35 divers were found to have definite or suspected lesions of dysbaric osteonecrosis, giving a prevalence of 32%. This figure is much lower than those previously reported for other comparable diving groups. For instance, in Japanese shellfish divers (Kawashima and Tamura, 1983) 56% of 747 divers had at least one lesion whilst in Hawaiian fishermen divers (Wade et al, 1978) in 20 selected divers of a population of 450 divers, 13 divers (65%) had lesions. These differences may be explained by differences in the diving profile. The latter two groups dive considerably deeper than the Australian abalone diver. However, the prevalence in the present study is comparable to the 27% reported in Gulf coast commercial divers (Fagan and Beckman, 1976) and to the 25% in a mixed group of sport and professional divers in Australia (Williams and Unsworth, 1976). In much more controlled exposures of military divers, lower figures have been reported. For example, in the Royal (Elliott and Harrison, 1976 and the United States Navies (Harvey and Sphar, 1976) 4 and 2.5% prevalences have been reported respectively. A large series on 4422 North Sea commercial divers also revealed a relatively low incidence of 4.4% (Harrison, 1981).

In our series, juxta-articular lesions were much more common in the shoulder than the hip which is in accord with previous diver surveys (Ohta and Matsunaga, 1974; Wade et al, 1978). Also we found a greater number of B lesions were found in the femur. Davidson (1981) reported 66% of all lesions occurred in the femoral shaft.

A relationship between "decompression stress" and the occurrence of lesions is suggested by our data. If "inadequate" decompression is a causal factor in the development of DO, then one would also expect a relationship between prior decompression sickness and DO. It must be admitted that both these factors are somewhat subjective relying on the diver's and observer's interpretation. It is our impression that many divers probably do not recognise minor episodes of decompression sickness.

The correlation between femoral lesions and alcohol consumption may also be significant in view of incidence of aseptic necrosis of the femoral head in (non-diving) alcoholics. It is possible that alcohol and decompression may be cofactors in the development of lesions in certain individuals. However, the results should be interpreted with caution since any observed relationship may not necessarily imply cause and effect.

As in other surveys of prevalence the figures are dependent on radiological technique and interpretation especially in the very early stages of the disease. A few of the older x-rays were insufficiently penetrated to allow assessment of fine trabecular structure so that some early lesions may have been missed.

In some instances, an inadequate length of the femur was incorporated in the radiological projection. A high proportion of B lesions have been reported in other surveys in the lower femur (Davidson, 1981). Thus we may have missed some of these B lesions.

The differential diagnosis between bone islands and early lesions is notoriously difficult. Therefore, a sample consisting of 4 sets of x-rays were forwarded for critical review to Dr J.K. Davidson, Radiological Member, MRC Decompression Sickness Panel. These series showed divers with multiple bone islands. The diagnosis of bone islands were confirmed by Dr Davidson. As bone islands were regarded as normal they were not reported by some radiologists and the reported prevalence is thus an underestimate.

A major concern of the investigators was that potentially important data was unavailable due entirely to inadequate provision of facilities for retention of x-rays by local authorities. Where radiologists reports only were still available, these were not included in the series.

Some of the divers who were said to have symptoms which would point to arthritis of the hip or shoulder were reluctant to participate in this survey, for reasons discussed elsewhere in this book (Chapter 7). For all the above reasons, it is felt that our reported prevalence is probably an underestimate of the true incidence of this disorder in Australian abalone divers. Notwithstanding, this series represents approximately one third of active abalone divers in Australia.

In summary, the survey highlights the need for some central data collection area for long bone x-rays so that valuable information is not lost and a clearer picture of this disorder amongst divers may be established. The divers themselves need to be made more aware of this disorder and encouraged to present at least every two years for a formal skeletal radiological survey. In many instances the x-rays ~~would need to~~ should be performed more often and perhaps other investigations such as bone-seeking radioisotope scans would be indicated. Where lesions are found their significance should be discussed with the diver. An appropriate course of action may then be recommended. This may range from a modification of his diving profile or shallower diving to complete cessation of diving. The decision about his diving future should then be made by the diver himself and with as much information as we can currently supply him.

Table 1

Prevalence of dysbaric osteonecrosis in Australian abalone divers.

	n	%
No lesions or bone islands	73	67
Suspect or definite lesions	35	32
Total	108	100

n = number of observations; lesions were taken to include Suspect or definite A and B lesions occurring in either the arm or the leg.

Table 2

Frequency of arm and leg lesions (described by type and number) exhibited on x-rays taken from each of 108 abalone divers shown in Table 1.

		Number of lesions			
Location	Type of lesion	Nil	1	>1	All
Arm					
	No lesions	72			72
	Bone Islands		5	7	12
	Suspect B lesions		1	6	7
	B lesions		0	6	6
	A lesions		3	8	11
	All	72	9	27	108
Leg					
	No lesions	64			64
	Bone islands		11	7	18
	Suspect B lesions		1	7	8
	B lesions		1	13	14
	Suspect A lesions		0	1	1
	A lesions		0	3	3
	All	64	13	31	108

Table 3

Counts of individual divers having both arm and leg lesions (described by type only) exhibited on x-rays taken from the same 108 Australian abalone divers as in Tables 1 and 2.

	Type of leg lesion						
Type of arm lesion	No lesion	Bone Islands	Suspect B lesions	B lesions	Suspect A lesions	A lesions	All
No lesion	50	11	4	6	0	1	72
Bone islands	6	6	0	0	0	0	12
Suspect B lesions	2	0	4	0	1	0	7
B lesions	1	0	0	4	0	1	6
A lesions	5	1	0	4	0	1	11
All	64	18	8	14	1	3	108

Table 4

Mean (± SD), minimum and maximum values of diving profile, alcohol and smoking habits of 85 Australian abalone divers.

PARAMETERS	MEAN	SD	MIN	MAX
Age(years)	38	7.3	22	55
Body mass index	25.0	2.80	21	33
Total years diving (years)	16	6.9	0	35
Total years abalone diving(years)	12	6.7	0	24
Maximum depth dived per day(feet)	54	17.8	20	100
Duration of diving per day(hours)	5.2	1.00	1.50	8.00
Days dived per year (days)	100	32.3	40	200
Total abalone diving experience(hours)	7266	4212	775	24000
Decompression sickness (number)	6	14.7	0	99
Alcohol consumption (cans per week)	12	15.6	0	70
Smoking habits (cigarettes per day)	7	11.1	0	40

SD = Standard deviation of the mean, MIN = minimum value, MAX = maximum value. For the purposes of this analysis time dependent diving parameters have been adjusted to the date when each individual diver's x-rays were taken. Factors corrected include age, total years diving, total years abalone diving and total abalone diving experience.

Table 5

Relationship between the likelihood of developing a lesion (irrespective of type or location) and diving and social history, in the diving population studied.

Parameter	F	P
Age	6.84	<0.05
Body mass index	1.67	NS
Years diving	6.77	<0.05
Years abalone diving	2.05	NS
Maximum depth dived per day	0.10	NS
Dive hours per day	3.08	NS
Days dived per year	3.64	NS
Decompression stress	5.49	<0.05
Decompression number	0.01	NS
Alcohol	3.88	NS
Smoking	0.04	NS

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Chapter 7

A5 - DYSBARIC OSTEONECROSIS REQUIRING JOINT REPLACEMENT

by C.LOWRY and C.EDMONDS

Introduction: The early diagnosis of dysbaric osteonecrosis is made more difficult by the fact that there is not always consistency in the radiological technique - despite the excellent recommendations laid down by the Medical Research Council Decompression Sickness panel of the United Kingdom.

Also, even when the correct radiological procedure has been performed, there are sometimes inconsistencies in the radiological interpretation of early or doubtful lesions.

Perusal of the statistics from the M.R.C. survey on 4,030 professional divers who had long bone x-rays, reveals that the more difficult diagnostic diagnoses are the very ones which are more frequently encountered. The same data shows the rarity of juxta-articular lesions(Calder)

Total number of divers screened	4030	100%
Normal results or non diving abnormalities	3973	94.1
Suspected shaft lesions (possible early sign)	61	1.5
Definite shaft lesion	95	2.4
Suspected joint lesion	42	1.0
Definite joint lesion, with joint surface intact	32	0.9
Definite joint lesion with damaged joint surface	4	0.1
Joint damage with disability	2	0.05
Joint damage leading to surgery	1	0.025

Of note, is that despite the 36 definite joint lesions and the 95 suspected joint lesions, making a total of 131 possible joint lesions, there were only 2 cases of disability and one case leading to surgery.

Ohta and Matsunaga in 1974, and Amako et al in 1974, described a survey on the shellfish divers of the Ariake sea, in Japan. Because the divers were exposed to compressed air diving twice a day, for 4 hours each and with a one hour surface interval, and because they had no concept of decompression staging, decompression problems were expected - and found. They averaged 4 deaths per year with diving accidents. There was a prevalence of disabling bone necrosis with structural failure (grades 4b and 5 of the M.R.C. classification) in 11 cases - all unilateral; 7 in the humeral head and 4 in the femoral head. Osteoarthritis was more often in the hips (14 joints) than the shoulder (1 joint).

From the Japanese survey, it appeared that the incidence of dysbaric osteonecrosis increased in proportion to the duration of diving experience and the working depth. Juxta-articular lesions were seen only in men who had dived over 5 years and deeper than 20 metres (67 feet).

There was a statistically significant relationship between the incidence of dysbaric osteonecrosis and decompression sickness, but no relationship between the sites. No difference was found between the incidence of dysbaric osteonecrosis in divers who had spinal decompression sickness, nor in those who consumed alcohol. There was no significant difference between the incidence on the right and left sides.

AUSTRALIAN ABALONE DIVER SURVEY

The methodology and analysis of this survey has been described previously.

The problem of diagnosis of indefinite lesions was in evidence in the abalone diver survey. Perusal revealed both cases of false negative and false positive results, as well as some indefinite and unclassifiable lesions. Review of some of the more doubtful films by Dr J.K. Davidson, Radiological member of the U.K. MRC Decompression Sickness Panel tended to confirm the diagnosis of our consultant radiologists.

During the survey it became apparent that there was a significant incidence of severe joint disease amongst the divers and ex-divers.

The non standardisation of the radiological procedures, irregular (and sometimes absent) radiological examinations and difficulties in storage and reporting of the X-rays, contributed to the difficulty in assessing the full clinical importance of dysbaric osteonecrosis in this occupational group. It was aggravated by the problematical effects of selection.

It was well known, and reported frequently to us during this survey, that the most serious cases of dysbaric osteonecrosis with severe joint disability, had moved away from the diving industry, and were no longer available for examination. Also, with even greater frequency, it was reported that the divers who were still active in this occupation, but who had very significant joint disability, decided not to have X-rays or to present themselves during the survey, for fear of medical confrontation and the possibility that action may be taken to prohibit them from diving on medical grounds.

In fact, the latter was neither contemplated or possible, but the fear was widespread amongst the divers, and it was impossible to influence this belief in order to get access to the divers concerned.

Another factor tending to reduce our reported incidence of A5 lesions requiring surgery, amongst this population, was the extreme reluctance of the divers to undergo any form of medical assessment or treatment. Also, although the greatest number of operations were performed on the hip (8 out of 8), it was very evident from observation of the divers' actions (e.g. trying to lift up a glass of beer in the pub), that in many cases the shoulders were grossly affected - but presumably the divers were able to tolerate this more than the incapacity of hip disease and the associated immobilisation.

Thus, the group of divers whom we saw were very selected, biased to demonstrate a lesser significance of this disease.

One of the common statements of those who are affected by joint damage of dysbaric osteonecrosis, is that they feel so much more relieved when working underwater, with neutral buoyancy, than trying to engage in terrestial physical activity where the effects of gravity make the joint symptoms worse.

A diver scheduled for surgery, but taken by a shark pre-operatively, was to have had a shoulder replacement.

Despite the above factors weighing heavily against eliciting serious cases of dysbaric osteonecrosis, there were a suprising number of cases in which surgery was indicated or was being arranged, or who had surgery because of this disability within the previous five years.

In this population there were five divers who had hips replaced because of gross damage from dysbaric osteonecrosis. In three the operation was bilateral and in two it was unilateral.

Because of the diverse nature of the diving, and the isolationist tendencies of the divers involved, it is very likely that other cases of bone necrosis have required surgery and have had this performed. There is unfortunately no central registry which would allow this information to be collated.

In all the surgical cases, hip replacement was performed because the articular cartilege had long since been destroyed i.e. there were no cases of "early" surgery with an intact articular surface.

Because there have been at least 5 divers previously operated on for A5 dysbaric osteonecrosis, and one scheduled for this treatment, there is a minimum prevalence of 2.0%. With nine joints requiring a joint replacement, the prevalence was 3.0%.

Review of the comparative M.R.C. figures revealed an prevalence of 0.025% for both, amongst the 4030 divers.

None of these divers had been exposed to helium or saturation diving (McCallum and Harrison,1982), and none have been treated with steroids, during recompression therapy (Black et al,1981).

Conclusion: The number of cases of severe disability suffered by this occupational group of divers, appears to be in excess of that reported in most other series.

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Chapter 8

PERMANENT HEARING LOSS DUE TO DIVING

A REVIEW

Carl Edmonds

LITERATURE

Excellent reviews on the development of knowledge of this subject have been made by Farmer [1983] and Molvaer [1980].

The presence of a permanent hearing loss in caisson workers has long been established, and was described by Bert [1878] and Snell [1896] last century. Boot [1913] coined the phrase "caisson worker's deafness" to describe a hearing loss which was either acute or chronic, temporary or permanent. Lang et al more recently [1971] investigated caisson workers under 40 years of age, and found that 60% had hearing defects. There was a variety of reasons why caisson workers should be affected with such a high incidence of hearing abnormalities, and these included barotrauma, decompression sickness, infections and noise damage.

Rozsahegyi [1975] demonstrated a loss of hearing in caisson workers, especially at the 4K Hz. He also found hearing impairment in 11 of his cases of decompression sickness, with tinnitus in 8 [1959].

Before 300 years B.C., Aristotle described tympanic membrane rupture in divers. Although there were many anecdotal case reports of permanent hearing loss with diving, the earlier surveys on divers and submariners revealed little or no significant permanent hearing loss.

Shilling and Everley, in 1942, performed a comprehensive survey on auditory acuity following exposure to pressure in U.S. navy submariners. They concluded that an acute and temporary loss of auditory acuity was associated with aero-otitis media (ear squeeze, middle ear barotrauma), whereas there was little or no effect due to exposure to air pressure, per se. Permanent damage to hearing was said to have been particularly in the higher frequencies, and in cases where damage to the middle ear had been extensive. They also demonstrated that there were rare cases of loss of auditory acuity associated with compressed air illness (decompression sickness).

Haines & Harris [1946] highlighted the seemingly contradictory statements regarding the effect that diving had on auditory acuity. They pointed out that some authors claimed a high tone loss, others a low tone loss; some stated that deafness may be severe and permanent, others that the auditory acuity was regained in a matter of hours. They also stressed that an audiogram demonstrating impaired acuity did not necessarily reflect a cause or relationship with diving activity, unless a previous base line audiogram had been performed. They stressed the common effects of noise, gunfire, blast, disease, etc. They discredited previous work on these grounds and performed their own prospective study on hyperbaric exposure in submarine escape training personnel.

Haines and Harris found no serious or significant effect of middle ear barotrauma on auditory acuity unless the middle ear was filled with blood unmixed with air, and that deafness was then more a result of dampening of the ossicles, than of pathological changes. Minimal hearing loss was noted both in asymptomatic subjects exposed to hyperbaria and in those who suffered lesser grades of middle ear barotrauma. The duration of these changes was not noted, but the inference was that they were temporary. In those subjects with symptoms of ear pain associated with compression, low tones may or may not have been affected, but for frequencies above 2000 Hz, some loss of acuity was usual. It was also noted that improvement of acuity was possible in some, but subjective sensation or loss of hearing was often an artifact, and that rupture of the tympanic membrane resulted in an acuity loss of only 5-10 db.

Extrapolation from the absence of permanent effects with a single exposure, to infer safety with multiple exposures, was unwarranted. But that is what happened. It was analagous to exposing a large number of patients to a small dose of X, finding no serious effects and therefore concluding that a large dose of X was safe.

Coles and Knight [1960] performed a survey on 54 Royal Navy divers and although there was a demonstrable hearing loss, they attributed this to gunfire and noise damage, as the 11 divers who were not subjected to these had normal hearing. Although one could question both the methodology and conclusions of this survey, it had the advantage of assessing experienced divers, and not "one time exposures".

The hearing losses which were transitory and related to middle ear barotrauma of descent, were verified by later workers, (Zannini et al,1972; Edmonds et al,1973;). The transitory conductive hearing loss of the lower frequencies, and the temporary threshhold shifts of the higher frequencies, are not subjects covered by this review.

Nevertheless, disorders associated with diving were increasingly being shown to produce permanent hearing loss. These included noise, by Coles and Knight [1961] and Molvaer et al, who reported on the inner ear damage from diving helmets [1982], pressure chambers [1981] and diving tools [1981]. Decompression sickness was incriminated by Buhlmann and Waldvogel [1967], Harris [1971] and Farmer et al [1976]. Inner ear barotrauma was described by Freeman and Edmonds in 1972, with the pathological concommittants of labyrinthine window fistula verified by Edmonds et al [1973]. Inner ear haemorrhage followed by the development of maladaptive new bone formation was shown to underlie the permanent damage, by Money et al [1985].

Despite the current acceptance of the disorder of permanent hearing loss as an occupational complication of diving, there was very little evidence of the incidence of this disorder amongst divers. The inference from the surveys referred to above was that it is low. Brady et al [1976] comparing 97 U.S. Navy divers with comparable age groups of civilians, showed virtually no differences due to the military diving environment, the occurrence of barotrauma, the type of diving equipment used, or the duration of diving.

One of the problems with assessing the hearing loss that may be attributable to diving, rests with the difficulty in obtaining a population which is adequately exposed to the diving. Brady et al used U.S. Navy divers who had as little experience as two months diving. Also, in the same series, there was no indication of the amount of diving, or the type of diving performed by this population, even though they would have had accurate log books available. It is well known that many U.S. Navy divers may spend relatively little time under water, and a great deal of time in their non diving naval pursuits. This is certainly so as the diver becomes more senior in rank.

That particular survey did demonstrate the hearing assessments on U.S. Navy divers but was, correctly, not used to extrapolate the effects of extensive diving. The U.S. Navy divers also tended to descend to a certain depth, at which they they performed a single duty or task, and then ascended. Such a diving profile would have been subjected to very little in the way of barotrauma stress.

However, Zannini et al [1972] showed that there was a significant permanent hearing loss especially in the 4000-8000 Hz range compared to non-divers. Just as Coles and Knight attributed the permanent high frequency deafness of many of their divers to coincidental noise and gunshot trauma in their earlier years, so has Farmer attempted to explain Zannini's findings.

Cross & Mayo [1979] found high frequency hearing impairment, most pronounced at 6000 Hz, much more frequently in North Sea divers than in non divers, regardless of the divers' experience.

Molvaer & Lehmann [1985] carried out a cross sectional study on 164 professional divers. A close correlation was shown to exist between hearing impairment and increasing age, increasing diving experience and acoustic trauma. The hearing impairment in divers was more often in the left than the right, and was predominantly in the left when the impairment was bilateral, consistent with the observations of Rozsahegyi and Lang. The harmful effects of cigarette smoking on hearing loss was demonstrated, and many references were cited verifying this association in the otological literature. They concluded that the hearing of divers at a young age wass better than that in a standard population, probably due to selection procedures. In the fourth decade of life, however, the high frequency in divers was at the same level as the standard population, suggestive of a faster deterioration in the divers. They also concluded that smoking contributed to the observed hearing impairment.

The excellent cross-sectional survey of professional divers performed by Molvaer and Lehmann, even though it did show some probable effects of diving, suffered from having an extremely heterogenous group of subjects, as admitted by the authors. Some were navy hard hat divers, some were involved in underwater work procedures, some were deep divers working offshore in the saturation profiles, whereas others were inspection and shallow water construction divers. It was apparent in that survey that the diving range was from 1 to 43 years, the most common being 5 years (mean=10.2, S.D.=7.8). There was no indication as to the frequency of the diving performed in this survey.

To ascertain whether there was an appreciably high incidence of hearing loss in professional divers, a survey was performed by Edmonds and Freeman[1985] on a very special group of 28 professional abalone divers from a rural area in Australia. The subjects had performed extensive diving (averaging more than 13000 hours underwater) - comparable to the exposures of experienced caisson workers, and in conditions in which noise damage was unlikely. Making allowance for age, over 60% had serious sensorineural high frequency hearing loss. In half the cases it was unilateral and half bilateral.

These abalone divers of Australia were mainly ex-fishermen who took up diving when their traditional fishing became unprofitable. The average age was 37.5 [SD=7.7]. All spent in excess of 6 years full time abalone diving [average=13.0,SD=3.8].The diving was mainly 15-20 metres depth, 4 hours per day, 100 days per year. Hookah gear was used, with the divers performing strenuous tasks - dragging the long compressed air hoses and transferring the full bags of abalone to the boat.

An extension of the Edmonds and Freeman survey is reported by Edmonds and Traugott[1986] in an Australian Abalone Diver survey. Pure tone audiometry was performed after an otological history and examination on 152 professional abalone divers. The occupational history and otological assessments were subjected to a factorial analysis and showed minor, but interesting, associations that have previosly been demonstrated by others. The effects of age were as expected.

The slight predominance of hearing loss in the left ear, compared to the right at 4000 Hz., was statistically but was not clinically significant and was not observed on the "damaged" ears. An explanation of this variation between sides involved a variation in size of the cochlear aquedut leading to greater likelihood of inner ear barotrauma on the left. No support fot this hypothesis was obtained. It could well have been due to the right handedness of the general population and the hearing loss following rifle shooting (left ear being affected predominantly).

The major influence of moderate alcohol intake was not great and was in the low frequencies 500-2000 Hz., perhaps due to its effect on the upper respiratory tract. The effect of smoking was even less, reaching significance only in the 1000 Hz. frequency.

The hearing loss was severe in 42% of divers, slightly more bilateral than unilateral.

A comparison was made of the hearing loss of Australian abalone divers who had extensive diving experience, with those who were of a more heterogenous group described by Molvaer and Lehmann. The abalone divers had a much more extensive hearing loss than the Norwegian professional group, in the 4000, 6000 and 8000 Hz, but without any of the obvious explanations that could be used to describe the damage in the Norwegian group, e.g.helium and deep diving, noise exposure, gunfire, etc.

Abalone divers were more comparable to the caisson workers, in their degree of exposure to hyperbaric conditions, and showed a similar intensity of hearing impairment.

DISCUSSION

The one real argument against most of the surveys which have looked at hearing loss due to diving, was that there was little attempt to estimate the actual time spent underwater.

A comparison of the hearing standards performed during the different surveys on divers is very difficult to interpret. This is especially because there is such a great variability in the type and amount of diving performed, by the different samples being chosen. Of the more recent surveys, comparison between Molvaer and Lehmann's divers, who were professional but had a variable exposure to diving, showed the hearing loss to be much less significant and serious than that demonstrated by Edmonds' Australian abalone divers who had been exposed to an extensive diving experience. It would therefore seem that, on the basis of the amount of diving exposure, Molvaer and Lehmann's group would be similar to Zannini's and intermediate in exposure between those described by Brady et al (less exposure) and Edmonds (more exposure).

The Australian group of abalone divers were analogous in many respects to caisson workers described by previous clinicians. They had undertaken a great deal of exposure to dysbaric conditions, and therefore were more likely to suffer the consequences of decompression sickness and barotrauma, than their more conservative counterparts in other professional diving activities - such as navy divers, oil rig divers, etc. Because of their age and their relative freedom of exposure to noise environments and explosives (this group were basically fishermen) and because they did not wear helmets and rarely were exposed to recompression chambers, it is more difficult to dismiss the auditory deficit, as has been done with other diving groups. There was no recent diving exposure to explain the deficits on the basis of a temporary threshhold shift, and no evidence of middle ear barotrauma on otoscopy. They did not use helium, and rarely employed any underwater tool louder than an abalone iron.

With the above results, Edmonds agreed with the Italian workers, Zannini et al, in claiming that high frequency sensorineural hearing loss is an occupational disease of compressed air divers, and that its incidence is statistically significant and clinically serious.

CONCLUSION

Permanent hearing loss may result from compressed air diving, especially affecting the high frequencies. It may result from the dysbaric disorders of barotrauma and decompression sickness. Contributary factors include infections, noise exposure (compression chambers, helmuts and underwater tools), and explosions.

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Chapter 9

CHRONIC ENT DISORDERS, SURVEY RESULTS

Carl EDMONDS

Results

The range of normal hearing for an Australian population, measured by pure tone audiometry in good listening conditions has been presented in tables, corrected for age, by the National Acoustics Laboratory (1974). Any sensorineural hearing loss greater than the acceptable or normal hearing loss for each age group, in excess of these figures is considered compensatable.

In comparison to the National Acoustic Laboratory scales of normal hearing, the 152 abalone divers had the following results:-

Normal hearing	33 divers	22%
Hearing loss	119 divers	78%

Of the divers who do have hearing loss according to the N.A.L. (1974) scale, 64% have bilateral hearing loss, 18% have right sided hearing loss, and 18% have left sided hearing loss.

If one considers the Australian Standards Association recommendation for the minimum I.S.O. standard of hearing for divers and compressed air workers, then 64 (42%) of the 152 professional abalone divers surveyed had severe hearing loss, and should not have continued diving. Of these, 58 had severe sensorineural hearing loss and were able to be assessed clinically; 32 (55%) had bilateral sensorineural hearing deafness and 26 (45%) had unilateral sensorineural deafness. There was one of these with associated severe conductive deafness, due to otosclerosis, and a few had a relatively mild conductive loss

In an attempt to assess the aetiology of the sensorineural hearing loss, recourse was made to the clinical history and examination of the subjects, and to the diagnosis as made by consultant otologists to whom the diver had been referred either prior to the survey, or during it.

CAUSES
Otitic barotrauma - 14
Decompression sickness - 4
Noise-explosion or industrial deafness - 11
Multiple causes - multiple aetiologies - 15
Miscellaneous trauma (mastoid operation,1; head injury,2; otosclorosis,1) - 4
No aetiology discernible - 10
Infections - 2
Total - 58

Tinnitus was noted in a large number of the cases, and was often concurrently linked with the aetiology of the deafness, e.g. with gunfire effects, barotrauma, etc. There were another 6 cases of complaints of tinnitus, which were unassociated with hearing loss. It was associated with barotrauma effects, often recurrent. In two others, it was associated with other trauma, specifically gunfire and "pop music" concerts. There were 2 cases of recurrent perforated tympanic membranes, not included in the above group, because there ws not, by definition, a hearing loss in excess of the Australian Standards Association criterion. There were also 5 cases of recurrent performated tympanic membrane ruptures, that were included in the above 58 divers.

Apart from the 58 audiological impaired divers, there were other chronic ear problems, including 5 cases of recurrent vertigo associated with ascent, one with descent, and 3 following decompression sickness - none of which showed pure tone audiometry impairment.

Many of the above cases of pure tone audiometry impairment also complained of tinnitus and occasional or frequent episodes of vertigo associated with dysbaric changes.

One case of vertigo was associated with carbon monoxide toxicity, without any hearing loss.

11 cases suffered symptoms consistent with temporo-mandibular joint dysfunction, i.e. pain or restriction of movement of the jaw following repeated or extended diving operations.

In 2 of the divers, the development of vertigo at depths was associated with the sensation of impending loss of consciousness.

Another 2 cases of tinnitus were observed to follow noise exposure or gunfire.

There were 28 cases of hay fever, of which 10 were associated with pure tone hearing loss. Decongestants were employed regularly by 21 of the 152 divers.

In 6 of the 14 cases of pure tone hearing loss associated with barotrauma, it was related to "reversed ear", i.e. barotrauma during ascent. In one case, it was related to both barotrauma during both ascent and descent, whereas in 7 of the cases it was related to barotrauma of descent "ear squeeze". This was despite the much more common occurrence of middle ear barotrauma of descent, than middle ear barotrauma of ascent.

There were 4 cases requiring surgical intervention for chronic sinus pathology, one of a large mucocoele that was initially mistaken and investigated as a cranial tumour, and 2 cases of removal of nasal polyps.

Conclusion:

There is a great deal of chronic E.N.T. pathology associated with recurrent diving, varying from the very high incidence of sensorineural deafness, especially associated with middle ear barotrauma and its inner ear sequelae, but also including occasional cases of decompression sickness. The other pathology includes physiological problems such as vertigo and tinnitus, together with occasional cases of nausea and vomiting associated with vestibular dysfunction.

Other chronic pathology includes temporo-mandibular joint dysfunction, chronic sinus disease and otological infections.

CHAPTER 10

FACTORIAL ANALYSIS OF HEARING LOSS IN PROFESSIONAL ABALONE DIVERS

Carl Edmonds and Frans M. Traugott

Despite the current acceptance of the disorder of permanent hearing loss as an occasional occupational complication of diving (Edmonds et al, 1973; Farmer, 1983), there is very little evidence of the relative incidence of this disorder amongst divers.

Shilling and Everley (1942) performed a comprehensive survey on auditory acuity following exposure to pressure in U.S. navy submariners. They investigated 2751 men taking part in a 50 p.s.i. pressure test and evaluated the effects of those who developed aero-otitis media (middle ear barotrauma) with those who did not. Of these, 1866 were taking it for the first time. They concluded that an acute loss of auditory acuity was associated with aero-otitis media, whilst little or no effect resulted due to exposure to air pressure, per se. Permanent damage to hearing was thought to occur particularly in the higher frequencies, and also in cases where damage to the middle ear had been extensive. Further, they demonstrated rare cases of loss of auditory acuity associated with compressed air illness (decompression sickness).

Haines and Harris (1946) carried out audiometry pre and post pressurisation on 6149 U.S. navy personnel. They found no serious or significant effect of middle ear barotrauma on auditory acuity unless the middle ear was filled with blood unmixed with air, and then the deafness was more a result of dampening of the ossicles, than of other pathological changes. Minimal hearing loss was noted in some asymptomatic subjects exposed to hyperbaria, and sometimes in those who suffered lesser grades of middle ear barotrauma. The duration of these changes was noted, but the inference was that they were temporary. It was also observed that: improvement of acuity was possible in some, subjective sensation or loss of hearing was often an artifact, and rupture of the tympanic membrane resulted in an acuity loss of only 5-10 Db.

By contrast, Coles and Knight (1960) described a survey on 54 Royal navy divers and although there was apparent hearing loss, they attributed this to gunfire and noise damage, as 11 of the divers who were not subjected to these stresses had normal hearing. Although one could question both the methodology and conclusions of this survey, it had the advantage of assessing experienced divers, and not "one time exposures". The inference from the study above is that there is little or no permanent hearing loss due to diving.

However, Zannini et al (1972) studied hearing loss in 160 professional divers, mainly from the Italian military forces. They described permanent hearing loss especially in the 4000-8000 Hz range in divers compared to non-divers. They concluded that the perceptive (sensorineural) hearing loss was significantly related to the years of professional diving and to the difficulty in equalising pressure in the middle ear space.

Just as Coles and Knight (1960) attributed the permanent high frequency deafness of their divers to coincidental noise and gunshot trauma in their earlier years, so has Farmer (1983) attempted to explain Zannini and colleagues' findings.

Further studies by Brady et al (1976) compared 97 U.S. navy divers with civilian audiometry standards for comparable ages. The study examined the relationship between audiometric characteristics and a number of variables including the years of navy diving experience, previous noise exposure, previous barotrauma and types of equipment used, e.g. SCUBA or helmet. They showed virtually no differences in hearing due to the military diving environment, the occurrence of barotrauma, the type of diving equipment used, or the duration of diving. The navy diving experience ranged from two months to more than ten years. They concluded that diving performed by the United States navy, in general, did not have a detrimental effect upon hearing.

Cross and Mayo (1979) found high frequency hearing impairment, most pronounced at 6000 Hz, much more frequently in North Sea divers than in non divers, regardless of the divers' experience.

A more recent study performed by Edmonds and Freeman (1985) reported an audiometric survey on 28 very experienced Australian abalone divers, averaging 5200 hours underwater. Over 60% had severe sensorineural hearing loss, half unilateral and half bilateral. That preliminary study was then repeated and expanded into the current text.

At about the same time, Molvaer and Lehmann (1985) described data collected from Norwegian male professional divers with a heterogenous age, diving experience and diving practice. This was the most extensive and carefully analysed survey, to date in the literature. It comprised 164 divers, with an average age of 30.9 ±8.4 years, with a mean total number of diving years of 10.2 ± 7.8 years. The co-variance of age and diving experience was so closely associated that either could be used alone without changing any of the results. The conclusions drawn by Molvaer and Lehmann was that hearing at a young age is better in divers than in the standard population, probably due to selection procedures for the diving profession. By the fourth decade of life, however, the high frequency hearing in these divers is at the same level as the standard population, suggesting a faster deterioration of hearing in divers. Also it was thought both smoking and noise exposure were factors contributing to the observed hearing impairment.

Cigarette smokers comprised 47% in this group, with a mean loss of 5.5 decibels. When adjustments were made for the effects of age, diving experience and history assessment of noise damage then effect due to smoking revealed significant differences.

No other possible factors contributing to hearing loss from diving were analysed in their report. These apparent omissions included the incidence of decompression sickness, or other factors that could have aggravated decompression sickness such as alcohol intake, the body mass index and the decompression profiles being used. Also the actual amount of diving being performed (as opposed to the number of years in which the subject has been classified as a diver), i.e. number of diving days per year and the hours per day was not evaluated.

Methods

Professional abalone divers numbering 152 of mixed sex comprised this study. (For a detailed description of the methods applied to this study, refer to chapter 3.) They were all examined by pure tone audiometry using a recently calibrated Angus & Coote S.A.3 pure tone audiometer in a sound protected area, measuring hearing loss from 500 Hz to 8000 Hz I.S.O. in accordance with the Australian Standards Association. There was no recent diving exposure to explain the deficits on the basis of a temporary threshhold shift, and no evidence of middle ear barotrauma on otoscopy.

Statistical methods

Statistical analysis was carried out on a PDP VAX 11/750 computer using a suite of analytical programs including BMDP, Spida, Glim and Minitab. For inferential comparisons of diving related parameters and audiometric data standard Student's t-test, Chi-square, Analysis of Variance with repeated measures and stepwise regression procedures were employed(Snedecor & Cochran,1980;Sokal & Rohlf,1969).

Results

Diving and personal profile

Diving data and cigarette and alcohol consumption figures obtained from 152 professional abalone divers are shown in Table 1. All parameters described with the exception of decompression sickness, alcohol and cigarette consumption are normally distributed, the latter being positively skewed. This group, averaging 38±7.8 (± standard deviation) years of age, had been diving with compressed air for approximately 16.1±7.75 years of which the last 12.1±7.12 years of diving experience was largely undertaken by diving for abalone. On a typical diving day, this group would dive on average to a maximum depth of 50 feet for 5.2±1.05 hours per day. This routine would be repeated for 106±30.3 days per year thereby resulting in a total abalone diving experience for this group of 6669 hours. Recorded social habits indicate that 12.6±7.0 cans (375mls of about 5% alcohol) were imbibed per week, whilst 7.1±13.3 cigarettes were smoked per day. The number of smokers in this group totalled 50 (or 33%).

The corresponding diving and social data for ages 20 to 29, 30 to 39, 40 to 49, 50 to 59 and 60 to 69 years are shown in Table 2. Not surprisingly, abalone and total diving exposure differed significantly between age groups($P<0.05$); the more senior age groups displaying a greater diving experience. Although not significantly different between groups, the number of episodes of decompression sickness was less in both the 20 to 29 and 50 to 59 year age groups. Alcohol intake in the 50 to 59 year age group also appeared less than that of the others ; this group did not smoke. There were no other age related differences revealed in the diving history.

Effect on hearing performance

The mean hearing levels(decibells) according to ear, age and audiometric frequencies measured are shown in Table 3 for the same divers reported in Tables 1 and 2. There are no statistically significant age related differences in hearing acuity between the left and right ear at any single frequency measured in this study. Nevertheless, hearing acuity showed a marked detioriation in both ears with increasing age($P<0.001$). Further hearing loss was greatest in the 4000 to 8000 Hertz frequency range.

The overall effect of these hearing levels when compared with the minimum acceptable standard level of hearing in divers is shown in Table 4. Significant hearing losses occurred in 15 to 26% of the divers at high frequencies, whilst 1 to 6% of divers had hearing losses beyond acceptable levels at low frequencies. Chi-square analysis revealed no preferential loss in hearing performance between either the right or left ears at each frequency, despite, at high frequencies a larger number of divers displaying less than the admissable level of hearing in the left ear, than in the right. At low frequencies the converse was true.

In order to determine the extent of unilateral or bilateral hearing impairment in any one diver, a count of the number of divers having demonstrable hearing impediment (as defined by the Australian Standard) at one or more frequencies in either the left, the right or both ears was recorded (see Table 5.). Of the total group studied, 88(or 58%) were free from any hearing loss. Of the remainder, the majority (33 or 22%) were more affected in the left ear, whilst 9(or 6%) showed equal loss between ears.

A further analysis of potential disparity in hearing between ears was performed using a paired t-test on the raw hearing scores of either ear, not corrected for the Australian Standard (Table 6.). The only frequency in which a difference between ears occurred was at 4000Hz; in this case revealing a greater hearing loss in the left than in the right ear.

Relating the diving and social parameters to possible causes of hearing loss in these divers a stepwise regression analysis was performed for each ear and each frequency independently. The results are shown in Table 7. An overriding feature of these results is the strong effect of age on hearing loss at most frequencies in both ears. When age is accounted for in the model, alcohol and to a lesser extent abalone diving seem to affect hearing in both ears at low frequencies.Notwithstanding, there is a suggestion that body mass index may in some way be related to hearing loss at high frequencies.

Discussion

A comparison of the hearing standards performed during the different surveys on divers is very difficult to interpret. This is expecially because there is such a great variability in the amount of diving performed, in the different populations being assessed. In the earlier work, one exposure was enough to warrant inclusion in the survey. Even in the later series there was often no estimate of the amount of diving.

In fact, the one real argument against most of the previous surveys which have investigated hearing loss due to diving, has been the absence of an estimate of the degree of diving component.

For example, Brady et al (1976) used U.S. navy divers, some of whom had had as little as two months diving experience. It is well known that many U.S. navy divers may spend relatively little time underwater, and a great deal of time in their non diving naval pursuits. This is certainly so as the diver becomes more senior in rank. Nevertheless this study did illustrate the auditory acuity of U.S. navy divers, but could hardly be used to extrapolate the effects of extensive diving on this acuity. Further, the U.S. navy divers tend to descend to a certain depth, at which they perform a single duty or task, and then ascend. Such a diving profile would be subject to relatively little barotrauma stress.

Another major problem in assessing the value of the surveys available, is the diversity of diving experiences cited. Helmet and chamber diving would presumably predispose to noise damage, deep and helium diving to decompression sickness, shallow air diving to middle ear barotrauma. Attempts to analyse these data independently, result in a great reduction in the numbers available to reach significance.

The excellent cross-sectional survey of professional divers performed by Molvaer and Lehmann(1985), even though it did show some probable effects of diving, suffered by sampling from an extremely heterogenous group of subjects, as admitted by the authors. Some were navy hard hat divers, whilst others were involved in underwater work procedures, some were deep helium divers working offshore with saturation profiles, whereas others were inspection and shallow water construction divers. It was apparent in their survey that the diving range was from 1 to 43 years with a mean duration of 10.2 years and a standard deviation of 7.8. There was no indication as to the frequency of the diving performed in this series, but presumably some were very experienced.

As most of the surveys did not estimate how much actual diving was performed, it is hard to draw conclusions regarding the effects of extended diving in auditory acuity.

Comparison of the hearing loss of abalone divers who had extensive diving experience, as described by Edmonds and Freeman(1985), with those who were of a more heterogenous group described by Molvaer and Lehmann(1985), highlighted the much more serious hearing loss in the Australian abalone divers than the Norwegian professionals in the 4000, 6000 and 8000 Hz, but without the variety of explanations that could be used to explain the damage in the Norwegian group, e.g., noise exposure, gunfire, etc. The same could be said for the survey of Zannini et al (1972).

The current survey was designed not only to ascertain the degree of auditory acuity, but also to correlate this with a variety of otological, social and diving parameters. In this survey, the professional abalone divers of Australia were mainly ex-fishermen who took up diving when their traditional fishing became unprofitable. There was no element of litigation or compensation as the divers were all self-employed.

This group of divers were analogous in many respects to caisson workers described by previous clinicians. They had undertaken a great deal of exposure to dysbaric conditions, and therefore were more likely to suffer the consequences of decompression sickness and barotrauma, than their more conservative counterparts in other professional diving activities - such as navy divers, oil rig divers, etc. Because of their age and their relative freedom of exposure to noise environments and explosives [this group wer basically fishermen] and because they did not wear helmets and rarely wer exposed to recompression chambers, it is more difficult to dismiss th auditory deficit,as has been done with other diving groups There was n recent diving exposure to explain the deficits on the basis of a temporar threshhold shift, and no evidence of middle ear barotrauma on otoscopy They did not use helium, and rarely exmployed any underwater tool loude than an abalone iron.

**The authors gratefully acknowledge the statistical advice given by Michael Jones, Clinical Computing Division, and the secretarial assistanc of Ms Sarah Burns, Department of Anaesthetics, Royal North Shore Hospital St Leonards, Australia.

Table 1. Mean (±SD), minimum and maximum values of diving profile, alcohol and smoking habits of 152 professional abalone divers.

PARAMETERS	MEAN	SD	MIN	MAX
Age(years)	38	7.8	23	63
Body mass index	25.0	3.01	18.9	33.4
Total years diving (years)	16.1	7.75	0.4	35.0
Total years abalone diving (years)	12.1	7.12	0.1	31.0
Maximum depth dived per day (feet)	50	17.9	15	100
Duration of diving per day (hours)	5.2	1.05	1.5	9.0
Days dived per year (days)	106	30.3	28	200
Decompression sickness (number)	4.1	12.16	0	99
Alcohol consumption (cans per week)	12.6	7.0	0	70
Smoking (cigarettes per day)	7.1	13.3	0	99

SD = Standard deviation of the mean, MIN = mimimum value, MAX = maximum value.

Table 2. Mean (±SD) values of diving profile according to age in the same 152 abalone divers as in Table 1.

PARAMETERS	AGE (years) 20-29	30-39	40-49	50-59	60-69
n	24	61	57	9	1
Age (years)	27 (1.8)	35 (2.9)	43 (2.7)	54 (2.6)	63
Body mass index	23.5 (2.86)	25.1 (2.92)	25.2 (2.97)	26.9 (3.50)	24.4
Total years diving (years)	8.4 (5.56)	13.2 (6.21)	21.8 (4.4)	21.1 (10.7)	15
Total years abalone diving (years)	4.6 (4.05)	9.9 (6.39)	16.9 (4.36)	16.7 (8.56)	15
Maximum depth dived per day (feet)	56 (20.5)	46 (18.7)	53 (15.1)	52 (17.0)	30
Duration of diving per day (hours)	5.2 (1.26)	5.2 (1.08)	5.3 (0.95)	5.5 (1.02)	4.5
Days dived per year (days)	117 (33.2)	105 (30.1)	102 (30.3)	105 (18.7)	140
Total abalone diving experience (hours)	2675 (2503)	5001 (3517)	8885 (3755)	10632 (7830)	9450
Decompression sickness (number)	0.9 (2.43)	4.7 (15.9)	5.4 (10.7)	1.3 (2.35)	0
Alcohol consumption (cans per week)	10.0 (9.69)	13.8 (16.2)	13.9 (16.3)	4.3 (11.6)	0
Smoking (cigarettes per day)	6.1 (8.94)	5.7 (10.6)	10.0 (17.3)	0	20

n = number of observations, figures in brackets are one standard deviation above and below the mean.

Table 3. Audiometric hearing levels(decibels) at several frequencies in both the left and right ear described by age group in 152 abalonedivers.

	AGE (years)				
FREQUENCY(Hz)	20-29	30-39	40-49	50-59	60-69
n	24	61	57	9	1
Left ear					
500	16 (12.1)	15 (11.3)	15 (8.0)	14 (6.8)	30
1000	11 (4.6)	13 (11.2)	16 (9.4)	16 (6.0)	35
2000	10 (7.8)	15 (12.9)	16 (11.3)	22 (11.7)	50
4000	15 (8.5)	26 (21.9)	40 (24.1)	57 (22.8)	60
6000	32 (21.1)	34 (19.2)	49 (24.4)	67 (27.1)	65
8000	28 (21.0)	30 (21.2)	44 (27.6)	71 (28.7)	100
Right ear					
500	14 (7.2)	14 (7.8)	19 (15.8)	22 (11.2)	60
1000	10 (7.6)	12 (6.5)	18 (15.8)	27 (16.4)	55
2000	11 (6.5)	13 (11.0)	19 (17.2)	24 (17.6)	75
4000	16 (14.0)	20 (17.1)	34 (25.0)	56 (27.0)	100
6000	27 (15.5)	31 (18.1)	45 (25.6)	76 (25.2)	100
8000	23 (16.5)	30 (20.1)	42 (27.4)	62 (32.9)	100

n = number of observations, figures in brackets are one standard deviation above and below the mean.

Table 4. Number of professional abalone divers in this study displaying less than the minimum acceptable standard level of hearing in either the left or right ear as measured by audiometry at different frequencies.

AUDIOMETRIC FREQUENCY (Hertz)	STD (dB)	HEARING LEFT (dB)	RIGHT (dB)	χ^2	P
500	40	3	6	0.1	NS
1000	35	2	7	0.1	NS
2000	35	9	6	1.3	NS
4000	45	37	23	42.4	NS
6000	50	40	35	41.4	NS
8000	50	40	32	49.1	NS

dB = hearing level measured in decibels, STD = minimum standard level of hearing (Australian standard AS2299), χ^2 = comparison of Chi-square values for the difference between acceptable and unacceptable hearing levels in the left and right ears at each frequency, P = probability level, NS = not significant.

Table 5. Counts of the number of abalone divers having appreciable hearing loss (determined by Australian Standard AS2299) at multiple frequencies in the right, the left or both ears.

	Right ear							
	Number of frequencies							
Left ear No. of frequencies	0	1	2	3	4	5	6	Total
0	88	3	4	0	1	0	1	97
1	10	2	1	1	0	0	1	15
2	5	1	1	3	1	0	0	11
3	4	4	4	6	2	0	1	21
4	1	0	0	3	0	0	1	5
6	1	0	0	0	0	0	0	1
Total	110	10	10	13	4	0	4	151

Table 6. Statistical comparison for hearing acuity between left and right ears at different frequencies in the same total group as in Table

FREQUENCY (Hertz)	P
500	NS
1000	NS
2000	NS
4000	<0.05
6000	NS
8000	NS

Statistical comparison between right and left ear performed using paired t-test. P = probability level, NS = not significant. Values for hearing are raw scores uncorrected for the Australian standard.

Table 7. Dependence of diving and social parameters and hearing acuity in either the left or right ear measured at different frequencie[s]

FREQUENCY (Hertz)	LEFT EAR	F	RIGHT EAR	F
500	Alcohol	7.23	Age	12.78
1000	Abalone diving	11.74	Age	29.25
	Alcohol	8.30	Alcohol	3.89
	Smoking	4.98	Abalone diving	3.95
2000	Age	14.16	Age	23.23
	Alcohol	5.71	Alcohol	5.50
			TADH	4.62
4000			Age	50.79
			MDD	4.07
6000	Age	32.37	Age	52.4
	BMI	7.72		
8000	Age	32.04	Age	40.23
	BMI	7.32	BMI	5.89

Statistical analysis carried out using stepwise regression. F = F test valu[e] MDD = maximum depth dived per day, BMI = body mass index, TADH = total hou[r] spent abalone diving.

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Chapter 11

RECOMMENDATIONS REGARDING OTORHYNOLOGICAL PROBLEMS.

When an otological consultant was available during the survey, it was found that about one third of the divers either required or requested a consultation because of current otological or paranasal problems.

An appreciable number of the divers suffered from difficulties with routine middle ear equalisation during descent. This was surprising as one would have thought that natural selection would have removed such subjects much earlier in their diving career. Problems such as hay fever, perennial vaso-motor rhinitis, post-nasal drip, chronic nasal obstruction, sinus abnormalities,etc., were widespread.

The sequelae of these naturally occurring disorders included the following:-

The use of decongestants, both in spray and oral forms.
Middle ear barotrauma of descent (ear squeeze).
Middle ear barotrauma of ascent (reversed ear).
Vertigo, especially during ascent (alternobaric vertigo).
Permanent inner ear damage.
Nasal and paranasal pathology including nasal polyps, sinus polyps and mucocoeles.

The most serious problem was that of sensorineural hearing loss, and its associated tinnitus.

The major recommendation is that the professional diver should be examined annually by a clinician knowledgeable in diving medicine. The reason for this recommendation is that many of the divers are not aware of the safe and more efficient ways of equalising their middle ear cavities during descent, and are sometimes relying on decongestants which may then produce other pathology, including the predilection to middle ear barotrauma of ascent.

Particular attention should be paid to the otological problems during the annual diving medical examination, and this should be supplemented by a naso-otological assessment, by an otological specialist when indicated.

It is recommended that professional divers undertake a pure tone audiometry, encompassing at least the range 500 to 8000 Hz. In many cases, the only damage is in one or more of the 4000, 6000 or 8000 Hz. range, with the greatest problem at 6000 Hz.

With the preventative measures of adequate instruction in middle ear autoinflation, the initial base line pure tone audiogram and the annual audiometric assessment to ensure no progression of hearing loss, it should be possible to reduce or even completely prevent a great deal of the otological and paranasal pathology experienced by this occupational group.

Chapter 12

NEUROPSYCHOLOGICAL DAMAGE WITH DIVING.

CAUSES AND RECOMMENDATIONS

Carl EDMONDS

This survey applied itself specifically to investigating the chronic or long term neurological sequelae of compressed air diving. There were, however, many complaints made regarding the short term effects, lasting over some hours and following the diving operation. It may well be that the short term effects have a long term component to them, however is not known.

It is also possible that the divers will not show a great deal of change at this stage, but will show an earlier development of the usual signs of senility and dementia. This could only be demonstrated in a prospective survey.

It would be of interest to repeat many of the investigations that have been carried out in this survey, and on the same divers, during a later stage in their occupational exposure. Either the same or parallel forms of the same tests, could be employed. Also the use of more sophisticated investigations such as Positron Emission Tomography or Nuclear Magnetic Resonance may be of value in elucidating brain damage in the future.

The possible causes for both short term and long term psychological effects are as follows:-

Carbon monoxide and other gas contaminants.

Carbon monoxide is a toxic gas because it combines with the haemoglobin in the red cells, together with other enzymes in the body. This results in an interference with the ability to transfer oxygen and to use it within the body tissues.

The effects from carbon monoxide can be very varied, including such symptoms as mild or moderate confusion, dizziness, exhaustion and breathlessness, headaches, etc. Only in the severe cases, will unconsciousness develop. The symptoms will tend to disappear over a matter of hours after the contamination.

Breathing gas contaminants, can be produced by inefficient air compressors, and are not able to be adequately corrected by any known filtering system. A contaminant which is in the air prior to compression, or added during compression because of a fault in the system, or contributed by the storage system, is likely to soon overload any conventional filtering mechanism.

Carbon monoxide can, together with various nitrogen oxides, be produced by the following methods:-

1. Carbon monoxide contaminating the air being compressed, i.e. an outside source such as a compressor or a nearby motor. The commonest cause of carbon monoxide contamination is the incorrect positioning of the air intake, so that air is collected together with the exhaust of the compressor.

2. When oil in the compression cylinder is combined with high temperatures, the oil will burn (flashing) or break down (cracking), into carbon monoxide with or without hydrocarbons. The excessive heat may be due to irregularities in the cylinder, rings or piston, causing increased friction (hot spots). Heat may also be produced by the following

(a) restriction of the compressor intake, e.g. dirty filter, excessive length or inadequate width of the intake pipe, kinked intake hose, etc. Under these conditions the final compression stage must compensate for the lessened intake, with a high compression ratio.

(b) leaks between the compressor stages, via piping, loose fittings or head gaskets, or around the piston, will also require higher final stage compression ratios.

(c) compressor valve leakage between the stages.

(d) general or local overheating beyond the compressor's design limits. Any inequality or impairment of the cooling system can cause this.

Air contamination from residues within the storage vessel or bottle is not common. Residues of cleaning and scouring materials and scale formed by rusting can contribute vapour or dust if the cleaning operation is not conducted properly, or if the cylinder or storage vessel is allowed to deteriorate. Water may be introduced into bottles if they are left open after use.

Hypoxia from salt water aspiration and near-drowning.

This is particularly likely to happen with the Hookah equipment which is used by abalone divers, because of the relatively low pressure in the air line (this has some beneficial effects in reducing the wear on the compressor) and the use of regulators which are suited to this equipment. The SeaBee regulators are also very likely to produce salt water aspiration, because of the position of the demand valve and the exhaust valves. Salt water leaks into the exhaust valve when it does not function adequately, and is then likely to be inhaled as a fine aerosol spray.

Attention to the regulator is required, and when the diver does notice the symptoms of salt water aspiration, then either the regulator should undergo a full maintenance correction, or be changed to more satisfactory equipment. It is possible that divers with a constitutional predisposition to bronchiolar reactivity may react more to the sea water spray, than others.

Most divers are now aware of the symptoms of salt water aspiration, including the symptoms of shivering or "fever", anorexia, nausea or even vomiting, mild dyspnoea, cough, sputum, headaches, malaise and generalised aches and weakness. They are also aware that these symptoms are normally absent by the next day.

Hypothermia.

Because these divers have to be exposed to such cold water, often below 5 degrees Centrigrade, and because of the extended time under the water, hypothermia is not uncommon. Dry suits are expensive and not suitable for the rough work of abalone diving. Some of the divers have shown innovative techniques by utilising hot water flow beneath the wet suits, supplied from the diver's compressor. Unfortunately, this is not practical in other areas, and therefore hypothermia is probably a significant hazard with great deal of abalone diving.

The effects of hypothermia producing toxiconfusional states, disorientation and amnesia, is well known. If the deep body temperature is less than 35 degrees Centrigrade, there is an impairment of speech, fixation of ideas, sluggish reaction time and mental impairment. Depersonalisation, amnesia, confusion and delirium are possible. Unconsciousness is not likely until the deep body temperature drops much lower than this, e.g. to 27 degrees Centrigrade or less.

Decompression sickness.

The incidence of decompression sickness, and its related dysbaric osteonecrosis, demonstrates this group of divers is particularly prone to this disorder. It is certainly consistent with their diving profiles, and it is rather surprising that there is not a greater incidence of decompression sickness. Nevertheless, sometimes neurological manifestations are evident when the symptoms suggest only minor joint "bends". There have even been cases of undoubted and clinically significant neurological damage from depths as shallow as 10 metres. The brain damage that may be possible from this disorder has been demonstrated, although there is inadequate evidence yet that this damage is cumulative.

Dehydration.

In many cases the divers appear to have a very inadequate fluid intake, sometimes being restricted to a cup of coffee in the early morning before boarding their craft, and having little or no fluid between then and returning to the dock, some 4 to 8 hours later. This in itself, is likely to result in a very significant dehydration, but when one adds some of the other factors that they are exposed to, this problem is accentuated.

Immersion creates a condition which resembles the gravity free state experienced by astronauts. Changes occur in the circulatory system, resulting in weakness and orthostatic intolerance. Immersion reduces the volume of pooled blood in the leg veins. Also peripheral vasoconstriction will occur in response to any cold stress. These cause a central blood volume increase, leading to a water diuresis (increased urine output) and haemoconcentration due to a reduction in plasma volume. Also, the breathing of compressed gas is likely to aggravate the fluid loss from respiration.

Another factor with abalone diving, is that often there is an increased exercise demand, with increased sweating and increased respiration. Again, both result in a fluid loss with dehydration.

RECOMMENDATIONS

Short term neuropsychological effects

An investigation may be indicated into the short term effects of extensive diving, as performed by abalone divers. This could be achieved by a small group of investigators travelling with the divers during their occupational activity, or alternatively meeting them on the wharf immediately following their return, and before they have regained their normal functioning.

The investigations would have to include some form of psychometric testing (including short tests such as the Wechsler memory Scale) and other self-assessment questionnaires, together with any electrophysiological data that may be available (e.g. electroencephalography), deep body temperature and full biochemical and haematological screening.

Decompression Sickness and Oxygen Use

It would seem prudent to avoid dangerous decompression profiles as much as possible, making allowance for the occupational exigencies. It is of note that a great number of the abalone divers were not aware of their failure to follow established principles of decompression. With this in mind, regular seminars held for the divers, which they could attend on a voluntary basis, could only be of value.

The attempt by the divers to reduce decompression sickness by the use of oxygen may have conflicting effects. Although the oxygen certainly reduces the incidence of intravascular bubbles, and possibly also bubbles very close to the vascular system (fast tissues), its use under hyperbaric conditions i.e. underwater, can produce a whole series of other neuropathological damage, e.g. to the special senses and the higher functions of the brain. The danger of oxygen as a neurotoxic substance, if used under pressure, has not been fully appreciated by many of the divers. This would also be the subject of any educational campaign which organised seminars for the divers, by experts in diving medicine.

As a general rule, even if there are no symptoms of decompression sickness present, it may be prudent for the diver to use oxygen on the surface to reduce decompression problems - assuming that the normal precautions against fire have been taken. It is not proposed as a method of extending underwater endurance and it is not recommended for routine use underwater.

The obvious way of diminishing the influence of decompression sickness in producing neuropsychological sequelae is by prompt and effective treatment of the disease.

Gas Contaminants

It is recommended that educational lectures be given to the divers, by experts in compressor technology. Numerous questions asked of the medical experts revealed the need for this information.

In each diving area, there should be available to the divers, a method of measuring the presence of carbon monoxide and other contaminants (hydrocarbons), that may be present in the gas supplied by the compressor. The investigation only takes a few minutes and should be performed at least every month, and whenever there is any reason to suspect compressor abnormality.

Such detection kits are readilly available in Australia and include the Drager Multi Gas Detector unit, with carbon monoxide and hydrocarbon tubes, the AUER gas analysers available from M.S.A., and the Bendix Gastic Gas Detection Systems in many other countries. They have similar capabilities. These simple analysis systems, which can be utilised by any diver or boatman, can inform the diver that his compressor is not function adequately.

Dehydration

It would seem reasonable to advise, even without waiting for further investigations, that adequate fluids be taken both before the dive and during the diving day. Fluids such as coffee, which produce a diuresis, would not be as adequate as fruit juices.

Chapter 13 Chapter not available till after Sept 86

PSYCHOMETRIC SEQUELAE OF DECOMPRESSION SICKNESS

Carl Edmonds

Cases of acute decompression sickness described in this series were subjected to psychometric screening tests

A screening programme was designed which required that the divers complete two separate psychometric assessment techniques. The first was the ACER Higher Test WL, which is standardized on Australian populations, and which is designed to measure general ability as revealed by performance on material of a verbal nature. As the abilities tapped by the test are among those which are hypothesized to decline less significantly with age and to be least affected by the deteriorative process, the final result provided an indication of premorbid intellectual endowment.

The second test was the Revised Visual Retention Test [Benton] which is a clinical and research instrument designed to assess visual perception, visual memory and visuoconstructive abilities. Research findings have demonstrated significant relationship between Revised Visual Retention Test performance and general intelligence level, and between test performance and chronological age. The tests' normative data provide the basis for clinical interpretation within the framework of knowledge of age and estimate of premorbid intellectual endowment of the subject.

Given the subject's age and the estimate of premorbid intellectual functioning from the ACER Higher Test WL, an "expected score" appropriate to these factors was deduced for the Benton. Interpretation of any subject's performance was then made by comparing this "expected score" with the obtained score of the test itself. The degree of discrepancy between the scores then allowed an assessment of the performance as a possible indication of acquired impairment of cognitive function. If there was no intellectual impairment and no confusional state, there should be no wide discrepancy between the scores.

The psychometric testing performed was of a screening variety. It would not have been adequate to have delineated the nature of the neuropsychological pathology in any individual case. This would require far more extensive testing on each individual, and this was performed on a small number.

A significant decrement of performance in the Benton test, in comparison to the ACER Higher Test WL, would not have been expected in a normal group of subjects. It was however observed in 12 out of 32 such divers tested.

There was no suggestion from the above investigation that the abnormality was more than a temporary abberration.

The explanations for this observed abnormality included:

Neurological damage from decompression sickness,

Neurological damage from pre-existing disease or treatment modalities (hyperbaric oxygen, medication, etc)

Symptomatic depression

Psychological stress from the decompression illness or the therapeutic procedure (sleep deprivation or interference, secondary gain from illness, loss of self esteem etc)

Summary

This group of divers was selected for investigation because of their known exposure to decompression sickness within the preceding week. The results were, however, indicative of the interference with intellectual functioning soon after an episode of decompression sickness requiring and recieving treatment.

Chapter 1?

ELECTROENCEPHALOGRAPHIC and EVOKED CORTICAL POTENTIALS

Robert HJORTH , Carl EDMONDS and V.VIGN

INTRODUCTION

The problem of how to assess brain damage in divers, has nc clarified.

The importance of such an assessment is unquestioned, as th avoidance of the cause at an early stage. It would not ne deterioration of intelligence, but at least it would avoi existing situation.

It is likely that progressive intellectual deterioration dc certain amount of brain tissue has been destroyed. This i "threshhold effect"(Roth,1972). Only after this threshhol become possible to demonstrate the quantitative relations psychometric assessment and the cerebral damage.

It is for this reason that earlier and more objective measu are so important.

Useful investigations to pursue would be the electroencepha evoked cortical potential (E.C.P.) responses - visual and decided to investigate a small series of very experienced whether this type of investigation would detect the effec diving, before there was gross neuropsychological damage

It was also decided that, if the E.E.G. findings did show a of that in the general population, even though they could neuropsychological deterioration, then these cases would r prospectively and the survey extended to encompass more s

If abnormalities were not found in excess of the normal popu investigations would need to be considered, including comp and positron emission tomography, if and when these were fr presumed that the computerised tomography could demonstrat and the positron emission tomography could demonstrate imp function.

The other reason for initially looking at the E.E.G. is that sensitive to a variety of neurological insults to which di gas bubble formation within nerve tissue (decompression s embolic phenomenon (air embolism), hypoxia and carbon monc

LITERATURE

Because the popular description of the possible "divers' dem drunk" syndrome, and because the multiple cerebral vessel exist in the occupational diseases of both boxers and dive the E.E.G. investigations in both groups is relevant.

A number of studies described immediate or short term effect boxers after their contests (Courjon,1972). Most studies r abnormal records attributable to the fight, especially wit Similar disturbances of normal background rhythyms with th either generalised or focal slow wave abnormalities, may c injuries, even mild ones, outside the boxing ring.

It is not known whether these changes indicate permanent neurological damage, although tissue destruction may be reflected in persistent abnormalities in the E.E.G. It is well recognised, however, that extensive loss of cerebral tissue, following head injury, is entirely compatible with a normal recording.

Regarding permanent, cumulative brain damage, only a small number of E.E.G. reports on brain damaged boxers are available (Roberts,1969). In earlier studies, the most frequently recorded abnormality was an excess of theta activity, often low voltage and generalised, but occasionally focal and then most commonly in the temporal areas. There was otherwise no characteristic abnormality noted. Nor were the most severe abnormalities constantly related to the cases most severely disabled clinically. In fact, non-specific abnormalities of the kind described in most of these cases, are found in the E.E.G.'s of 10-15% of the population. In one study of brain damage in boxers, 168 boxers were compared with 150 controls of a similar age bracket and the proportion of abnormal recordings in both boxers and controls was no greater than was to be expected in any cross section of the population.

The progression of intellectual incapacity and disturbance in motor functions in cases of traumatic encephalopathy in boxers, some years after their retirement, probably reflects the changes associated with ageing in brains in which there has already been some depletion of neural functioning reserves (Roberts 1969).

Traumatic encephalopathy of boxers, the punch drunk syndrome, may be due to either single or repeated neurological insults. Although it undoubtedly needs to be diagnosed as early as possible, whether it can then be halted once the progression has commenced, is uncertain. It is presumed that the disease could be aggravated by continuing exposure. It is thought that the brain sustains either a contrecoup injury or a rotational strain, or both. This leads to haemorrhagic episodes upon which the clinical picture depends. The pathological changes affect the cerebral hemispheres, cerebellum and midbrain region, and have been well described.

A series of 40 boxers were exposed to computerised tomography (C.T.), and a positive correlation was found between the number of bouts, cerebral atrophy and ventricular enlargement. (Casson,1982). It was also found that 13 out of 15 professional boxers tested had C.T. or E.E.G. evidence of organic cerebral dysfunction (Casson 1984). In fact the C.T. scan may prove to be the most helpful in detection of early cases of brain damage (Miller,1984).

One survey of 150 professional divers in Poland (Kowski,1979), with an average age of 36 years and a 14 year exposure to diving, demonstrated E.E.G. abnormalities with a slight to medium abnormal change observed in 43%, and which were greater with the more years of diving experience. It was stated that 10% of the normal population would be expected to have such changes. It is not clear whether the increase in abnormalities with the increased diving experience was found independent of the effects of age.

Another study (Vaernes and Eidsvik,1982), which compared two groups of divers, 9 with a history of diving accidents and 15 accident free divers, showed that 8 of the 9 accident divers had abnormalities on neuropsychological tests implicating lesions of the higher levels of the central nervous system. It was stated that E.E.G. and brain stem auditory evoked potentials were taken for later studies, but no information regarding these were available and they have not been reported in subsequent literature.

The overall conclusion from reading the E.E.G. literature is that, although E.E.G.s may be sensitive to recent damage, they quickly become normal, so that E.E.G.s are of little value is assessing the result of chronic or past damage. Although the literature on evoked potentials is much smaller, it suggests that a different pattern occurs. The major use of evoked potentials has been in the study of multiple sclerosis and here we find that the evoked potentials are sensitive both to acute insult and also to chronic change. Thus, in visual evoked responses, optic neuritis causes a rapid slowing or disappearance of ev oked potentials and, although there may be some clinical improvement subsequently, evoked potentials remain permanently abnormal.

A striking finding is that visual evoked potentials are abnormal in a significant percentage of patients with multiple sclerosis who have no clinical evidence of optic nerve involvement and give no past history of optic neuritis or visual disturbance. (Halliday, 1982). If the effects of compression and decompression in divers were similar to those of multiple sclerosis, then one might expect that evoked potential studies would pick up a number of cases of sub-clinical damage in a situation analogous to that of multiple sclerosis.

METHODOLOGY

Subjects.

During an extensive investigation on professional Australian abalone divers, a group of 20, 10 from Victoria and 10 from N.S.W., were investigated by E.E.G., and half this number, those who were thought to be subjected to the most significant diving exposures, were investigated by visual and auditory evoked cortical potentials and somatosensory evoked responses from both upper and lower limbs.

Subjects who had a history of head injury or other cause of unconsciousness, such as near-drowning, hypoxia or carbon monoxide poisoning, were excluded from the survey.

Personal and diving information was elicited from both a standardised questionnaire and a personal interview to clarify the dive profile. Each diver was graded according to his customary diving pattern, obtained from the interview. The grading was as follows:

1= Dives less than 30 ft. and within conventional tables.
2= Dives in excess of 30 ft. but within conventional tables.
3= Dives in excess of the conventional tables, but nevertheless appearing as a reasonable and sensible dive pattern, e.g. if a diver were to spend two hours at 60 ft, followed by one hour at 40 ft. and one hour at 20 ft, without any significant surface interval, then one could argue that the tables have been contravened; however, one could equally argue that the 40 ft. stop would assist in the decompression to some degree, and that the 20 ft. stop would be probably adequate for subsequent decompression requirements.
4= Dives which required a decompression commitment of less than one hour, but which was routinely ommitted.
5= Dives which omit an hour or more of decompression staging.

E.E.G.

Electroencephalograms were performed using the Beckman Accutrace (either 8 or 16 channel machines). Electrodes (silver/silver chloride padded) were placed according to the 10-20 system. E.E.G.s were recorded with bipolar and referential montages. Paper speed of 30 mm/sec, time constant of 0.3 seconds and eye frequency filter of 70Hz were used. All subjects were made to hyperventilate for 2 minutes and were also given photic stimulation using a Grass model photic stimulation.

These were recorded in two separate neurological institutions, and read independently. Eight channel E.E.G.recordings were done with a number of montages, photic stimulation and hyperventilation were used.

Visual evoked responses:

The stimulus was a video monitor screen with checkerboard pattern reversal. The screen was 70 cm. from the subject's eyes and it measured 38 cm. square so that the angle subtended was approximately 30 degrees. The check size was 2 degrees per check. The reversal rate was 2 per second and 128 sweeps were recorded per study with an analysis time of 200 msecs., a low frequency filter of 10 Hz and a high frequency filter of 125 Hz. The recording electrodes were small subcutaneous needle electrodes and there were four channels averaged:-

Oz to Fz,
Cz to Fz,
5 cm. to the left of Oz to Fz,
5 cm. to the right of Oz to Fz.

Each eye was studied separately and all averages were duplicated. Measurements were made by the use of a cursor which the observer placed on the wave identified as P.100 being the dominant positive potential in the appropriate area of the graph. With this technique a W shaped potential may require half field stimulation in order to establish which of the two positive potentials is the true P.100 but in this series there was never any ambiguity about the identify of P.100. Laboratory normals have been determined on a group of controls between the age of 20 and 50 and the normal results are recorded on the table.

Somatosensory evoked responses from the upper limbs:

In the upper limbs each median nerve was stimulated using percutaneous stimulation at the wrist and with a strength just sufficient to cause a motor response in the thenar eminence. The stimuli were at 2 per second and usually 512 sweeps were averaged. Recording involved the averaging of 3 channels which were:

Erb's Point to Fz,
Posterior process of the second cervical vertebra to Fz,
Cc to Fz (where Cc is the contralateral somatosensory cortex which is considered to be 2 cm. behind C.3 or C.4 in the 10 20 E.E.G. system).

The analysis time was 50 msec, the low frequency filter was 10 Hz, and the high frequency filter 1.5 KHz. All averages were duplicated. The E.P. potential was identified from the recording at Erb's Point, the N.13 from the recording at the cervical spine and the N.20 from the contralateral cortex. For each potential latency to peak and amplitude (from base line) were measured.

Somatosensory evoked responses in the lower limbs:

The tibial nerve was stimulated behind the medial malleolus and the stimulus strength was just sufficient to cause a contraction in flexor hallucis brevis. Recording was by small needle electrodes placed over the appropriate site and the three channels averaged were:

First lumbar process to iliac crest,
First lumbar process to L.4,
Fz to Cz,
Cc to Ci.

Because of an administrative error, although the 20 E.E.G.'s were obtained, only 9 of the subjects were submitted to the E.C.P. responses.

RESULTS

Diving Data

The following information categorises the divers studied, and their diving experience.

E.E.G. E.C.P.

	N= 20	N= 9
Age (Av. & Standard Deviation)	40(SD=5.6)	42.7(SD=2.9)
Body Mass Index	26.0(SD=2.8)	26.3(SD=2.6)
Years diving	18.4(SD=5.4)	20.7(SD=4.1)
Years abalone diving	15.5(SD=5.7)	16.9(SD=6.4)
Usual (maximum) depth, in feet	49.1(SD=14.2)	52.8(SD=11.8)
Average diving duration in hours	5.2(SD=1.0)	5.0(SD=1.4)
Days diving per year	110.8(SD=32.7)	105.6(SD=31.7)
Decompression stress (graded 1-5)	4.0(SD=0.9)	4.4(SD=0.5)
Decompression sickness incidents	2.0(SD=1.7)	2.6(SD=1.42)

Of interest in assessing the results is that none of the divers had ever suffered from hypoxia or carbon monoxide toxicity to the extent of impairing consciousness. Of the 39 episodes of decompression sickness, only 14 were given recompression therapy in a chamber. There were 7 instances of decompression sickness affecting the central nervous system, in the subjects who were sent for evoked potentials, and one in a subject who was not. The alcohol consumption, estimated by questionnaire, revealed an average of 15.5 "cans" of alcohol (375ml with approximately 5% alcohol) per week.

E.E.G. results

The following abnormalities were noted.

Diver A, aged 51; 4909 abalone diving hours; decompression stress of 3; no cases of decompression sickness. An intermittent 8-9 Hz alpha rhythym was symmetrical. Generalised arrhythmic theta waves were seen. Hyperventilation and photic stimulation did not evoke abnormalities. Comment: This EEG showed minor generalised abnormalities.

Diver B, aged 39; 5500 abalone diving hours; decompression stress of 4; 3 cases of decompression sickness but not apparently affecting the neurological system. An intermittent 8 Hz. alpha rhythym was symmetrical. Generalised low voltage theta waves were seen intermittently. Arrhythmic 3.5-5 Hz waves were intermittently seen over either temporal areas. Hyperventilation and photic stimulation did not evoke abnormalities. Comment: This EEG shows mild to moderate bilateral abnormalities more over the temporal regions.

Diver C, aged 39;810 abalone diving hours; decompression stress of 5; 2 cases of decompression sickness, one of which included neurological symptoms. This diver had suffered his neurological decompression sickness, 12 days previously. He required 3 recompression therapies, but was said to be symptom free after that. An 8 - 9 hz alpha rhythym was present bilaterally. Small quantities of theta were seen and these were sufficient to make the record borderline abnormal. The theta fluctuated and was worse with drowsiness. Hyperventilation and photic stimulation did not add any information. Conclusion: a borderline record with only non-specific features.

A comparison of the "normal" divers with the "abnormal" divers, based on E.E.G., shows that there is no greater diving exposure as judged by; the hours spent abalone diving, decompression stress, or decompression sickness incidence, in the "abnormal" cases.

visual evoked responses:

The results of the visual evoked responses are shown in Table 1 with a typical tracing shown in Fig. 1. From this table it can be seen that the visual evoked responses are completely normal both individually and on average.

Somatosensory responses from the arms

The results of the somatosensory evoked responses from the upper limbs are shown in Table 2 and a typical tracing is shown in Fig. 2. Comparison with the normal values of Chiappa show that as a group the divers had slightly longer latencies in all three of the potentials. The most likely explanation for this is that divers as a group were taller and had longer arms than Chiappa's normal control group. There is also a suggestion that people who do a lot of heavy work with their arms may have slight slowing of conduction pepripherally. Calculation of the central conduction times, although not incorporated in the table, shows no slowing.

Somatosensory responses from the legs

The somatosensory evoked responses from the lower limbs are shown in Table 3 wih a typical recording in Fig. 3. There are two subjects where the recording has only been from one leg. In one of the subjects the reason was that one leg had suffered a compound fracture with damage to the nerves in the past. The other subject had one leg missed due to oversight. Chiappa's normal values are included at the bottom of the table and from this it can be seen that there again the distal conduction (from the ankle to the lumbar spine) is slow, and is slower in the divers than in the control group. This again would be best explained as due to difference in height and the table incorporates the average height for the subjects (174.8 cm) and for Chiappa's normal controls (166 cm.) The transit time which partly corrects for the height difference is almost identical on average to that of the normal controls.

DISCUSSION

This group of 20 divers had no reason for brain damage other than their diving exposure. None had suffered neurological damage from motor vehicle accidents, hypoxia, carbon monoxide poisoning, or other causes.

There were no known features which would have diminished this group's likelihood to decompression sickness or air embolism, the average age being 40 years, and a body mass index of 26. They had suffered 39 recognised instances of decompression sickness, and this is in a group which has a very low threshhold for recognising and reporting such an illness. There were 14 recompression therapies administered and 7 obvious instances of neurological decompression sickness.

The exceptional factor in this group was the exposure to excessive compressed air diving. On a typical diving day they would expose themselves to a decompression stress that would conventionally require decompression staging, but which was omitted. In 13 subjects this would be less than one hour omitted decompression,and in 4 subjects it would be in excess of this.

It is possible that if one used evoked potentials to study a larger number of divers and used controls that were very closely matched for age, sex, height and limb length, that subtle group differences would be discernible. However the present study indicates that there were no individuals outside the generally accepted normal range and certainly nothing like the striking slowing of conduction that has characterized multiple sclerosis.

This study suggests that cumulative neurological damage does not occur from excessive diving, or the neurophysiological tests selected were not sensitive enough to detect this damage.

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Chapter 15

INTELLECTUAL DETERIORATION WITH EXCESSIVE DIVING.

['PUNCH DRUNK DIVERS']

Carl Edmonds * and Jon Boughton **

ABSTRACT.

A survey was performed on a specific occupational group of compressed air divers - the professional abalone divers of New South Wales. One aspect of this survey included the use of psychometric screening tests to elicit evidence of impaired intellectual capacity, which may be related to the compressed air diving. Results of the survey indicate that there is evidence of intellectual impairment in almost half of this diving population. The fact the this diving group exposed themselves to much greater decompression stress than the more conventional professional diving groups, would suggest that these results should not be extrapolated to other diving populations. The results are supportive of the anecdotal beliefs that exist regarding this highly selective diving group, i.e. that a syndrome of reduced intellectual capacity [dementia or "punch drunkenness"] is present.

There is an anecdotal or folklore belief amongst many occupational diving groups, that a dementia syndrome, or "punch-drunk" effect, is produced by prolonged compressed air diving. The Diving Medical Centre in Sydney, Australia, was requested to advise on this subject, and to make recommendations which could allow preventative action to be taken by newer divers. Unfortunately, there was inadequate data for such a hypothesis, and therefore more information was sought regarding this problem.

During recent years the interest has mainly been on the effects of deep diving and its neuropsychological sequelae(1). Workshops and symposium in Luxembourg (1978), Bethesda (1981) and Stavanger (1983) highlighted this problem, without producing a consensus. Nevertheless, during the investigations of deep diving effects, there has been an apparent acceptance of neuropsychological complications of diving with compressd air at shallower depths. This disorder seemed to be presumed, without a great deal of objective or clinical evidence.

The belief that prolonged compressed air exposure resulted in intellectual impairment, with a variety of neurological and psychological disorders, was promoted by Rozsahegyi (2) in Hungary, with clinical observations on caisson workers who had neurological decompression sickness. Peters et al (3) in the U.S.A.,with Vaeres and Eidsvik (4) from Norway, supported this association in compressed air divers who had decompression sickness. Autopsies on divers, by Calder (1) in the U.K., suggested a diffuse cerebral pathology to explain these observations. In each of the above reports the brain damage, with the neuropsychological clinical sequelae, was specifically correlated to the existence of decompression sickness. The incidence, or even the presence, of the "punch drunk" syndrome amongst compressed air divers who had not been subject to gross neurological decommpression sickness, was not elucidated.

In a report on abalone diving in Australia (5), it was stated that 30% of the divers suffered chronic ear damage, 20% had dysbaric osteonecrosis and 10% had brain damage - but detailed analysis and assessments were not presented and so the observation, whether accurate or not, must be classified as conjecture.

An opportunity became available to carry out a basic clinical screening examination on a very special group of divers who could clarify the association of extreme diving exposure and the development of dementia. It was conjectured that such a group would be likely to show a much greater degree of morbidity than their more regimented colleagues. The hypothesis was that if this group of divers showed no degree of dementia, then it would be an unlikely or uncommon complication of compressed air diving, per se.

POPULATION STUDIED.

The professional abalone divers of New South Wales, Australia, had performed a great deal of compressed air diving and competed for a very limited catch. In this state of Australia, they formed a closed community. They were obliged to register for licences in 1980, and a prerequisite was to have had at least three years full time professional abalone diving, prior to the application. Thus all the divers had more than six years full time experience at the time the study was performed. No formal diving training was required. They tended to be ex-fishermen, from fishing families who had moved from their previously profitable and traditional occupation to the more lucrative abalone industry - taking with them their maritime skills, but little else.

The diving was strenuous, with the divers carrying bags of abalone and enduring the tidal drag on their long compressed air supply hoses, collecting bags of abalone that were then transferred to the boat when filled. Each diver would average approximately 100 days diving per year and on each diving day he would be under water for a total of about four hours, and this was unrelated in any way to his depth. There would usually be three or four brief surface intervals, usually only of ten minutes or so, but could be up to an hour - the time necessary to move his boat to an adjacent non-fished area. Most of the dives were between 15 and 20 metres, shallow depths only being possible in very good weather and with low seas [uncommon in that area]. No decompression staging was performed.

Despite the hazardous nature of the diving, the financial rewards were not great, and there was a considerable depletion of the accessible abalone beds. Because of this and other reasons, the licences were not able to be transferred or sold, but were waived when the diver decided not to continue his occupation. Thus the divers had to persevere with their diving to finance their heavy capital expenditures on boats and equipment, without opportunity to "sell out" [despite considerations of age or health], and with little opportunity to be redeployed to other occupations. Detailed records were not kept of the daily diving profile, and often neither watch nor depth gauge was worn. Most of the depths were known from the accurate maritime charts and the reported depth of the anchorages. Rarely was it possible to dive according to any plan or principle. Diving first in deeper water and then coming shallow would only be possible if the wind and sea abated during the day - the opposite to its customary activity.

METHOD.

The object was to employ psychometric screening tests, standardised and extensively used on the Australian population(6), to indicate the existance or otherwise of brain damage in divers. Tests in common clinical practice were selected, and it was not intended to apply sophisticated test batteries to demonstrate highly specific cognitive dysfunctions or conceptual abnormalities, at this stage.

The screening programme required the divers to complete two separate psychometric assessments. The first was the ACER Higher Test WL, standardized on Australian populations, and designed to measure general ability as revealed by performance on material of a verbal nature. As the abilities tapped by the test are among those which are hypothesized to not decline significantly with age and to be least affected by the deteriorative process, the final result provided an indication of premorbid intellectual endowment.

The second test was the Revised Visual Retention Test [Benton] which is a clinical and research instrument designed to assess visual perception, visual memory and visuoconstructive abilities(7). Research findings have demonstrated significant relationship between Revised Visual Retention Test performance and general intelligence level, and between test performance and chronological age. The tests' normative data provide the basis for clinical interpretation within the framework of knowledge of age and estimate of premorbid intellectual endowment of the subject.

Given the subject's age and the estimate of premorbid intellectual functioning from the ACER Higher Test WL, an "expected score" appropriate to these factors can be deduced for the Benton. Interpretation of any subject's performance is then made by comparing this "expected score" with the obtained score of the test itself. The degree of discrepancy between the scores allowed the results to be classified as either "no indication" of acquired impairment of cognitive function, "raise the question" of such impairment, "suggest" such impairment or show "strong indication" of such impairment.This is analagous to the approach in assessment of dementia in the clinical setting.

As well as the psychometric assessments, a record was made of the diver's age, number of years diving, number of days involved in professional abalone diving, number of times decompression sickness had been diagnosed, and his alcohol intake.

RESULTS.

The population studied was as follows:

There were 30 professional abalone divers with an mean age of 37.5 years [SD = 7.70]. They averaged 13.0 years diving [SD = 3.80], and the number of days diving per diver was 1523.5 [SD = 1270.50]. Two thirds of the divers admitted to alcohol ingestion, but only two of the 30 claimed to drink more than two bottles of beer per day, i.e. 1.5 litres. No divers admitted to the ingestion of spirits, and the observations of the investigators, who were staying with the divers in the same isolated residential hotel, were consistent with their claims.

In assessing the results of the psychometric screening tests, there were six exclusions necessary for technical reasons. One refused to do any psychological testing; one did not have his reading glasses and was therefore unable to perform the tests; one did not give his age; one missed out a whole page of one test, but was otherwise assessed to have "strongly suggestive" indications of impairment; one did not attempt the ACER Higher Test WL, but the Benton was poorly completed and indicative of impairment; one performed below the norms on one test and with questionable performance on the other, generating hypotheses of reading ability, visual difficulty, problems in attention span and comprehension instructions. Thus of the subjects excluded from formal analysis, approximately half demonstrated results or behaviour which was suggestive of deteriorated performance.

Of the remaining 24 divers there was no evidence of intellectual impairment in 12, one had results which required allocation to the "questionable" category. The results indicated impairment of acquired intellectual capacity in the remaining 11, four suggestive and seven strongly indicative.

As a quick screening device it appears, at least with this population, that definitive allocation to categories can be made with few equivocal cases.

Fifteen of the 28 divers reported decompression sickness, but this was not significantly correlated with the impairment of intellectual capacity. The total number of episodes of decompression sickness was 41.

DISCUSSION.

This group of divers was selected for investigation because of their known extreme exposure to dysbaric conditions, and their very provocative diving procedures. It was presumed that had this group not shown any evidence of intellectual impairment, then it would be unlikely to be of a significant degree in the more conventional air diving groups.

The range of environmental hazards which have been conjectured as possible aetiological agents producing dementia in divers include such factors as: decompression sickness, air emboli, carbon monoxide toxicity, hypoxia, high oxygen or nitrogen pressures, etc. No attempt was made to differentiate these factors during this survey as we intended only to ascertain whether there was reason to proceed further. Had results not shown any indication of dementia, further investigation would not have been warranted.

The psychometric tests performed in this survey, were of a screening type, able to detect only the more gross psychopathology, indicative of intellectual deterioration of a clinically relevant level(7). They would not have been adequate to have delineated the nature of the specific neuropsychological pathology in any individual case. This would require far more extensive test batteries on each individual. The results were, however, strongly indicative of a general impairment of intellectual capacity [dementia] in at least seven of the subjects tested, out of a total of 24, with four showing some evidence of this disorder. Demonstration of such impairment would not be expected in normal subjects and thus this finding in 11 out of 24 subjects is a cause of concern.

This group of divers have no gross or obvious neurological manifestations [they are all required to pass medical examinations each year]. There is no obvious non-diving environmental factor to explain the results.

Extrapolation from the psychometric testing on other groups of more conventional divers, would not have led us to suspect the results obtained during this survey. Previous work(8) suggested that intelligence is positively correlated with diving success, at least in the navy diver training courses, and the psychometric investigations performed on professional divers in the Australian navy did not show any evidence of intellectual impairment.

Because the results achieved indicate that there could be valid support for the hypothesis that compressed air diving can lead to impaired intellectual capacity [dementia], further studies are being performed on this and analogous groups throughout Australia. This particular group of divers comply less with formal decompression procedures than most other groups, although only about half the divers did report this disorder.

It is known that this incidence of decompression sickness is grossly under reported, as there is very little that can be offered to the divers in the way of recompression facilities, in that area. Also the training and awareness of these divers in identifying decompression sickness, is much below any acceptable standard. The only time they would report the disorder would be when it was very obvious.

It is appreciated the much of the information obtained did not have the accuracy that one would wish. Specific details regarding the extent of the diving exposure, either for individual divers or in total, must be considered mere approximations. So also would be the degree of alcohol consumption and the incidence of decompression sickness. Nevertheless, whether these figures are underestimates or overestimates, the implications of the psychometric testing still require explanation.

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Chapter 16

DOES NON CLINICAL DECOMPRESSION STRESS LEAD TO BRAIN DAMAGE IN ABALONE DIVERS

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ABSTRACT

Abalone divers are subject to considerable decompression stresses and could be at risk of progressive cognitive impairment. A sample of abalone divers were compared with a matched sample of fishermen on a battery of neuropsychological tests. No evidence of cognitive impairment was found in the divers despite evidence of their exposure to decompression stress. The implications for other professional divers and for recreational underwater divers who follow standard decompression protocols are reassuring.

Abalone are found on reefs and rocks off the southern coasts of Australia. The shellfish are well camouflaged and cling tightly to the rock if disturbed. While once available in the shallow waters off the coast, they have now receded to depths of 6 - 30 metres. Divers collecting these shellfish on a commercial basis must spend several hours at these depths breathing from compressed airlines. They experience repeated episodes of compression and decompression. The abalone divers and their families have noted that after a difficult day divers are often irritable, slow moving and apparently, confused as though they were punch drunk.

The "punch drunk" syndrome has also been reported in a number of occupational diving groups. (e.g. Symth,(1)). The syndrome is believed to be a consequence of rapid decompression. Decompression, when carried out too quickly, results in nitrogen bubbles coming out of solution in the blood, and these bubbles can interfer with cerebral circulation. Other mechanisms that could compound this syndrome in the abalone diver are carbon monoxide poisoning from the air compressor, and hypoxia due to salt water aspiration as the low pressure compressors and the regulators used produce a salt water mist in the inspired air.

The literature on the relationship between prolonged compressed air diving and permanent intellectual impairment is limited. Rozsahegyi(2), who first identified this association, reported clinical observations on caisson workers who had neurological decompression sickness. Peters, Levin and Kelly(3) and Vaernes & Eidsvik(4) further supported this association in compressed air divers with decompression sickness. These studies are restricted to divers who showed gross neurological decompression sickness. Only two studies to date have addressed the more general issue of the relationship between compressed air diving and neuropsychological impairment. A brief report(1) claimed that deep sea divers of the British Isles showed significant differences on memory and reasoning skills tests according to diving experience ; the less experienced the diver the better the performance on the tests. The report was brief, no data were presented, a comparison group was not included and it is unclear whether the effects of age were controlled for in the analysis. Secondly, in a report on abalone divers in Australia (5), it was stated that 30% of the divers suffered chronic ear damage, 20% had dysbaric osteonecrosis and 10% had brain damage. Again, data presentation and procedural information was brief.

The abalone divers of Australia are a particularly interesting group for the study of the relationship between compressed air diving and brain damage. These divers form a closed community and have performed a great deal of compressed air diving. They were obliged to register for licences in 1980, and a prerequisite for this licence was at least three years full time professional abalone diving. No formal diving training was required. Their diving is strenuous, with divers carrying heavy bags of abalone, enduring the tidal drag on their long compressed air supply hoses, and returning to the surface for three or four brief intervals to unload their bags or move their boats. Each diver would average approximately 100 days diving per year and on each diving day he would be under water for a total of about 4 - 6 hours, this duration being unrelated to the depth of the dive. Further, in the areas around the N.S.W./ Victoria border where the divers work the sea conditions can be dangerous. Seas often become rough in the afternoon which prevents the abalone diver from observing the safer diving protocol of deep diving in the morning and shallow diving in the afternoon to allow for more gradual decompression. Whatever the conditions, the abalone diver is is not renowned for strict adherence to decompression staging.

The present study was designed to test the cognitive neurological functioning of abalone divers on the south coast of N.S.W and the north coast of Victoria. They were compared with a group of fishermen matched for age, education, history of brain damage and reported alcohol consumption. Fishermen were chosen as the comparison group because they worked in the same industry, experienced similar lifestyles and working conditions, but did not dive.

Method

Subjects

We were able to test 32 of the 54 registered abalone divers in N.S.W and 16 of the 22 registered divers in Eastern Victoria, giving an overall response rate of 63%. All divers were tested at least 72 hours after their last dive, which avoided contamination from the more acute post dive effects of lethargy, confusion and memory loss. The 48 abalone divers were compared with a control group of 47 commercial fishermen. The fishermen's participtation was obtained by soliciting the cooperation of the crews of boats as they returned to berth in the ports of the south coast of N.S.W. While we have no way of knowing the response rate of the fishermen, it did appear to be similar, in that rather more than half the fishermen approached agreed to participate.

Materials

The battery of tests used to ascertain evidence of neurological impairment were as follows:

(1) The Symbol Digit Modalities Test (SDMT). This test, which is commonly used to screen for cerebral dysfunction, involves the

conversion of meaningless geometric designs into written and oral number responses(6).

(2) The Single and Double Simultaneous (Face-Hand) Stimulation Test(SDSS). This is a test of specific somatosensory function which determines the accuracy with which subjects can identify single and double simultaneous tactile stimulation applied to the cheek and/or hand. The SDSS is known be be most reliable when used in association with the SMDT(7).

(3) The Wechsler Memory Scale(8). This test is designed to detect memory impairment. We used 3 of the 7 subtests; namely logical memory, visual reproduction and paired associate learning. The logical memory test involves the recall of two passages and is believed to assess left temporal lobe function. The visual reproduction task involves drawing from memory three separate diagrams and is believed to assess right temporal lobe function. The paired associate learning task involves recall of 10 paired associates. This test assesses both memory of past associations and the acquisition of new associations. These three tests were chosen as they were the most sensitive and relevant to the needs of the present study.

(4) The Controlled Word Association test(9). This test measures verbal fluency and is believed to be a measure of frontal lobe function. The patients are asked to say as many words as they can think of which begin with F, then A, and finally S; omitting proper nouns, numbers and the same word with a different suffix. As a control procedure, the subjects are also asked to list as many animal names as they can think of in the required time.

Procedure:

On arrival at the testing unit, the abalone divers were required to fill out questionnaires on diving history, diving procedure, diving related illnesses, history of concussion or other 'brain damage' incidents, substance abuse, and education. Following questionnaire completion the neuropsychological testing was commenced. The fishermen were usually tested in a wharfside office and completed the same tests with the exception of the detailed diving questionnaire. If an office was not available testing was conducted on the fishing boats. The testing was conducted by a psychologist, psychiatrist or senior medical student; all of whom were trained to criterion on the tests. Testing took approximately 30 minutes. Results were scored, checked and verified without knowledge of group membership.

Results

The 48 divers had experienced 235 episodes of decompression sickness, and received recompression therapy for 36 of them. There were 8 cases of decompression sickness with neurological sequelae but none were proven permanent. There were 16 episodes of carbon monoxide toxicity and 10 episodes of salt water aspiration but none of the cases had resulted in unconsciousness. The interveiwers felt that these rates were underestimated. The data collected from the 48 abalone divers and the 47 fisherman on age, education, history of brain damage, and current level of alcohol intake is presented in Table 1. The only significant difference between the abalone divers and the fisherman was in age. This difference was an unfortunate artifact of skippers prefering to volunteer their younger deckhands as subjects.

Insert Table 1 & 2 about here

In Table 2 the performances of both groups are displayed for each test in terms of means and standard deviations, significance of difference between groups, and significance of difference after an analysis of covariance allowing for the effects of age. A total score was derived by summing the standard scores(z scores) for each test and the differences tested similarly.

The divers did less well than the fishermen on 5 of the 8 tests, significantly so on two tests, the logical memory and the paired associate learning sub-tests from the Wechsler Memory Scale. These differences remained significant when the effects of age were taken into account. The differences were small and could have been an artifact of the multiple significance testing. The scores which the abalone divers obtained were not poor enough to suggest neurological impairment. That is, their scores fell in the normal range for the two Wechsler subtests. As it was possible that these differences were due to the abnormally low scores of only some of the divers we looked at the number of abalone divers and fishermen whose scores fell two standard deviations outside the mean. There was no evidence that a sub-set of divers were neurologically abnormal.

The next possibility to be explored was whether low scores on the tests could be related to diving history or diving behaviour believed to be harmful. We looked at correlations between test scores and years of diving, depth of diving, hours of diving, reports of near drowning or carbon monoxide poisoning. There were no significant correlations between any of these variables and the scores on the two Wechsler tests on which divers scored significantly worse than fishermen. There were significant correlations between divestress and SDMTW (p = 0.046), divestress and SDMTO (p = 0.046), hours of diving and visual reproduction (p = 0.027), and near drowning/carbon monoxide poisoning and SDMTO (p = 0.03) and animal (p = 0.003). On all of these tests the divers had performed marginally, but not significantly, less well than the fishermen. Finally, each result was converted to a standardized score (Z score) and summed to allow a total score on the cognitive test battery to be calculated. After adjustment for the age discrepancy, no significant difference between groups was evident.

Discussion

Only 9 of the 48 (19%) abalone divers said they carried out conventionally acceptable dive protocols, and 31 of the 48 (65%) routinely failed to complete the required decompression procedures. As a result of these practices and the extraordinary amount of diving performed (average of 5.2 hours/day, 100.7 days/year and 15.3 years of abalone diving) neuropsychological complications of decompression should be overrepresented in this group. If this group was found to be unaffected then it would be unlikely that other, less exposed, groups of divers who complied with established safety dive protocols, would be significantly affected.

We were unable to demonstrate that abalone divers performed significantly less well on a neuropsychological test battery than did fishermen matched for education, alcohol intake, history of other head injury, and *post hoc*, for age. We conclude that despite accounts of brain damage following compressed air diving in other diving groups(1) and anecdotal accounts of the prevalence of irritability and confusion in abalone divers directly after diving, there is no evidence of any permanent intellectual decrement in this group.

Our null conclusion, however, leaves two observations unexplained; the divers' reports of irritability, slowness and confusion after a hard day of diving, and the association between dive stress and some aspects of neuropsychological test performance. First, although irritability, slowness and confusion may be evidence for transient neurological dysfunction, it could also be due to general exhaustion after a hard and difficult day of diving. Second, the correlation between performance on some tests and high dive stress might well indicate that the less cognitively able report more dangerous dives. This explanation is not incompatible with our initial hypothesis that dive stress may also result in neurological impairment. The divers' scores on the neuropsychological tests were within a normal range and did not differ from those of the fishermen.

The high incidence of dysbaric osteonecrosis and otological disorders found in this group of divers gives added evidence of the hazards of excessive diving. These disorders are attributable in whole or in part to inadequate decompression procedures(10). Decompression sickness which results in severe neurological damage does have neuropsychological sequelae(2,3,4). Nevertheless in this study, in which there is ample evidence of decompression inadequacy without gross neurological disease, support for accumulation of subclinical neuropsychological insults leading to a dementing process is not available. Thus other professional divers and recreational divers who follow the protocols should be safe from progressive brain damage as a consequence of their diving.

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TABLE 1: Means and standard deviations of features likely to affect test performance

	Divers		Fishermen	
Age in years	40	(7.6)	32	(10.8)*
Episodes of concussion etc.	0.4	(0.8)	0.4	(0.7)
Alcoholic drinks per day	2.8	(1.2)	3.6	(2.9)
Education level+	3.7	(1.2)	3.3	(1.3)

* $p < .05$

+ 3 = school certificate
  4 = school certificate plus trade training

TABLE 2: Means and standard deviations of the test scores for the abalone divers (N = 48) and the fishermen (N = 47) with significance of the difference (two-tailed test) with and without controlling for the age difference

	Divers		Fishermen		Significance uncontrolled	Significance controlled
Symbol Digit Modalities Test (written)	40	(10)	44	(9)	0.06	0.17
Symbol Digit Modalities Test (oral)	47	(9)	51	(10)	0.07	0.24
Single & Double Simultaneous Stimulation Test	0.06	(0.3)	0.06	(0.3)	0.90	-
Wechsler Subtest; Logical Memory	10	(3)	12	(3)	0.005	0.02
Wechsler Subtest; Visual Reproduction	10	(2)	11	(3)	0.26	0.87
Wechsler Subtest; Associate Learning	15	(3)	17	(3)	0.01	0.03
Controlled Word Association Test; F, A, S	34	(10)	33	(9)	0.78	0.92
Controlled Word Association Test; Animals	18	(3)	17	(3)	0.52	0.94
TOTAL (sum of Z scores)	-0.92	4.13	1.18	4.77	0.23	0.91

Critical significance level after correction for experiment-wise error rate p = .006

Chapter 17

MULTIPLE APTITUDE ASSESSMENTS ON ABALONE DIVERS

TERRY COULTON* AND CARL EDMONDS

INTRODUCTION

A prerequisite to demonstrating a deterioration in intellectual capacity, is an ability to measure this quality. Wechsler designed an intelligence test comprising two broad sections, verbal and performance, which in turn were combined to yield a full scale I.Q., and this is one of the most standardised of psychometric tests.

One of the major effects of factor analysis techniques has been the development of multiple aptitude batteries. These yield a profile of test scores for relatively independent abilities.

It is considered likely that any pathological damage to the brain would not affect all intellectual functions uniformly[1]. Some functions are believed to remain unaffected, whereas others are more sensitive to brain damage. The Wechsler scales (specifically the WAIS-R) are very applicable to a profile analysis, since all the subtest scores are expressed in directly comparable standard scores.

Three different types of approach are used for these tests. Firstly, there is the amount of scatter, or the extent of variation amongst individual scores on all the subtests. The rationale for this is that the inter-test variation should be larger in pathological than in normal cases. The second procedure involves the use of a "deterioration index" based on the difference between tests which are resistant to deterioration from brain damage, and those which are considered susceptible. The third approach is that of "score patterns" which may be associated with specific clinical syndromes.

One serious drawback to the WAIS-R, is that it requires individual administration, and therefore a group administered multiple aptitude battery test was selected, which had a high co-efficient of correlation with the WAIS-R. The multidimensional aptitude battery (MAB) incorporates some of the widely acknowledged positive features of the WAIS-R, into a structural format permitting group administration[2].

The MAB, like its counterpart in the Wechsler scales, has a profile of 10 subtests. It employs 5-choice multiple-choice items on all 10 subtests. Each requires 7 minutes of testing, and yields raw scores which are converted to scaled scores, corrected for age, with a mean of 50 and a standard deviation of 10.

To facilitate comparison with the WAIS-R, similar functions are assessed on the MAB and WAIS-R scales, even though in some cases the actual tasks might differ. For example, block design in the WAIS-R and the spatial subtest of the MAB, both measure spatial ability.

The MAB permits the identification of verbal, performance and full scale I.Q. equivalents. These represent, in combination with the subscale scores, a convenient, reliable summary of an individual's intellectual funtion.

Under the verbal I.Q, the tests include:-

Information
Comprehension
Arithmetic
Similarities
Vocabulary

Under the performance I.Q. the tests include:-

Digit Symbol
Picture Completion
Spatial
Picture Arrangement
Object Assembly

Methodology

67 professional abalone divers were examined in groups of 10 to 12, using the multidimensional aptitude battery (MAB) in accordance with the manual. All the divers were English speaking, and only one spoke with a trace of a foreign accent. A clinical history was taken to exclude brain damage or diseases likely to induce dementia. A full diving history was obtained and scored, as described elsewhere.

Results

Population

The population on which the MAB investigations were performed, had the following characteristics:-

N=67	Mean	Standard Deviation
Age in Years	36.8	8.1
Body Mass Index	24.7	2.8
Years Abalone Diving	10.6	8.2
Diving Days/Year	114.2	29.5
Diving Hours/Day	5.2	1.1
Usual Maximum Depth(feet)	43.8	16.1

These subjects reported only 22 diagnosed cases of decompression sickness, of which 10 were neurological. The recognition of more minor cases was grossly inadequate (judging by the interviews) and recompression facilities were not easily available.

Decompression profiles on their typical dives were considered inadequate in 40%, by U.S. navy tables. Decompression of less than one hour was routinely omitted by 22 divers, and in 5 divers it was more than this, on their typical dives.

They averaged 11.6 cans of beer (375ml of approximately 5% alcohol) per week, and only 28% consumed cigarettes.

Statistical analysis

The results of the MAB were :

VERBAL	Mean	S.D.	PERFORMANCE	Mean	S.D.
Information	44.5	7.7	Digit symbol	47.7	9.7
Comprehension	48.8	8.3	Picture completion	55.2	8.0
Arithmetic	50.3	9.2	Spacial	53.4	8.8
Similarities	48.7	9.2	Picture arrangement	50.2	10.2
Vocabulary	49.7	11.4	Object assembly	50.4	8.3
Verbal	97.7	12.7	Performance	101.7	13.2

TOTAL I.Q.= 98.7 (S.D.=12.3)

A multiple regression analysis was made against all diving co-variants and MAB subtest scores, verbal IQ, performance IQ, total IQ, and deterioration index ("dementia score"), corrected for age.

Information subtest - no statistical significance.
Comprehension subtest - no statistical significance.
Arithmetic subtest - no statistical significance.
Similarities subtest - the only significant correlations were with numbers of decompression sickness incidents (- 2.27) and with smoking (2.06).
Vocabulary subtest - no statistical significance.

Digit Symbol - no statistical significance.
Picture Completion - no statistical significance.
Spatial - no statistical significance.
Picture Arrangement - no statistical significance.
Object Assembly - no statistical significance.

A multiple regression analysis of the verbal IQ, the performance IQ and the total IQ all showed no relationship with any of the measurements performed. The deterioration index (dementia score) also showed no association with any of the measurements performed on the divers.

DISCUSSION

One of the outcomes from the use of factor analysis in the traditional intelligence tests, has been the development of multiple aptitude batteries. These are designed to provide a measure of the individual's ability in each of a number of different traits.

A separate score is obtained for each trait, and these scores may be used to compare with others, or included in an overall assessment. These batteries also incorporate into a comprehensive and systematic testing programme, much of the information formally obtained from the special aptitude tests [1]. The term "aptitude test" has been traditionally employed to refer to tests measuring a clearly defined segment of ability. The term "intelligence test" customarily refers to a single global score such as an IQ. Special aptitude tests measure a single aptitude, whereas multiple aptitude batteries measure a number of aptitudes, but provide a profile of scores, one for each aptitude.

The use of an aptitude profile permits research regarding patterns of abilities associated with different neuropsychological conditions. It also permits a basis for evaluating changes in intellectual ability that might occur in the period after brain trauma or psychopathology.

Such a broad clinical entity as "brain damage" probably has a varied and contradictory pattern available on these investigations and in different sub-groups, so as to make it very difficult to interpret. Nevertheless, such IQ scales tend to have a high reliability and a fair degree of validity for many purposes. They may be used to ascertain what type of aptitudes are required to be successful in any specific occupational field (e.g. professional abalone diving), as well as indicating, in a coarse manner, the possibility of brain damage.

The use of the Wechsler scales have been extraordinarily successful [2] for a number of reasons. They incorporate a diversity of tasks, not only verbal and school learning content, but performance and practical skills as well. They reflect conceptions of the nature of intelligence, which might be affected by a variety of disorders including neurosis, psychosis and brain damage. They have a high level of internal consistency, reliability, and in fact, these Wechsler scales have become the standard against which other test of intelligence have been appraised.

One important source of unreliability and bias in the measurement of aptitudes, is the subjectivity accompanying judgementally scored tests. The examiner can thus bias the outcome of those tests. The structured approach to scoring the MAB is designed to minimise this influence. It has been shown (Ryan, Profiteria and Powers 1983) that, regardless of one's experience level in psychological testing, scoring errors occur frequently, and detract from the accuracy of WAIS-R I.Q.s. Golden (1979) noted that WAIS verbal scales such as comprehension, similiarities and vocabulary are particularly difficult to score, with experienced test users disagreeing over borderline responses. It has also been shown (Donaghue and Sattler, 1971) that the examiners liking for the respondent affected the scoring. Thus there was an advantage in using a test in which the scoring was objective, as in the case of the MAB.

An examination of the MAB/WAIS-R in comparison with the WAIS/WAIS-R correlations indicates that the two sets correlate to about the same degree even though the MAB departs appreciably from the individually administered format of the Wechsler tests.

Comparison of Correlation Coefficients between the MAB and WAIS-R and the WAIS and WAIS-R

Subtest	MAB/WAIS-R (N=145)	WAIS/WAIS-R (N=72/70)
Information	.82	.84
Comprehension	.73	.62
Arithmetic	.89	.56
Similarities	.66	.52
Vocabulary	.89	.59
Digit Symbol	.45	.54
Picture Completion	.87	.47
Spatial/Block Design	.44	.80
Picture Arrangement	.86	.33
Object Assembly	.65	.40
Verbal Scale	.94	.82
Performance Scale	.79	.82
Full Scale	.91	.87

The inadequacy of using aptitude profiles as an estimate of intellectual deterioration is well appreciated. Usually the variation between different parts of the test has to be moderately large to reach statistical significance, e.g. comparison of the WAIS-R verbal and performance IQ's is approximately 10 points at the 0.05 level, and 13 points to reach the 0.01 level of statistical significance. This is also applicable in the comparison among the individual subtests.

Also, the non-specificity of the aptitude batteries to detect intellectual deterioration, has resulted in the development of specialised neuropsychological tests. These include the Benton Visual Retention Test and the Bender-Gestalt Test, involving perception of spacial relations and memory for newly learned material. Neuropsychological batteries such as the Halstead-Reitan and Luria-Nebraska are thought to demonstrate more specific deficits and indicate anatomical localisation of lesions. As yet, these do not have the wide usage of the aptitude batteries and are too time consuming to use as a widespread screening assessment for dementia.

CONCLUSION

The population surveyed in this investigation showed none of the features hypothesised to be present with serious intellectual impairment, as assessed by the Multidimensional Aptitude Battery.

* Gratefull acknowledgement for the acquisition, administration and scoring of this test battery is given to Mr Terry Coulton. Unfortunatelt Mr Coulton was not available during the presentation of this paper, and therefore any errors of commission or ommision are mine alone.

Acknowledgement must also be paid to Mr Frank Traugott who carried out the statistics and computer analysis. Carl Edmonds

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Chapter 18

PART 1. THE NEUROBEHAVIOURAL EFFECTS OF PROFESSIONAL ABALONE DIVERS

A.M.WILLIAMSON, B.CLARKE AND C.EDMONDS

PART 2. RELATIONSHIP BETWEEN NEUROBEHAVIOURAL FACTORS AND DIVING EXPOSURE

A.M.WILLIAMSON AND B.CLARKE

Professional diving is usually regarded to be a hazardous occupation due to health risks such as decompression sickness, air embolism, and carbon monoxide poisoning to name a few. Surprisingly, little is known, however, about the effects of working underwater on the nervous system and particularly central nervous system function. In a study of 33 commercial abalone divers from the Port Lincoln area of South Australia, nervous system function was examined using a battery of neurobehavioural tests. Their performance was compared to that of nondiver controls matched for age, sex, education level, job type, language abilities and cigarette and alcohol consumption. Abalone divers showed significantly poorer vision, learning and short term memory performance and increased tremor relative to their controls. The reaction time of abalone divers however, was as fast as or significantly faster than that of controls but their error rates were much higher indicating that abalone divers were sacrificing speed for accuracy. Despite their apparent risk-taking approach to these tests, the performance of abalone divers suggests impairment of nervous system function.

The effects of long term exposure to the underwater environment such as is encountered by professional divers are not well understood.

While the effects of deep diving has attracted a considerable amount of research attention[1] very few studies have focussed on the long term effects of shallow water exposure.

One area in particular, the effect of long term effects of repeated shallow water exposure on the nervous system is open to question. Even though there is a significant amount of clinical and anecdotal evidence that professional divers suffer damage to the nervous system, neurological and behavioural function has not been examined systematically.

A few scattered reports have detailed neurological effects of decompression sickness. One of the earliest described diffuse damage to a range of areas in the nervous system as well as neurotic and psychosomatic symptoms in caisson workers[2]. Subsequent work has shown that divers suffer similar nervous system[3,4] effects of decompression sickness. In the latter study neuropsychological testing revealed that 90 percent of commercial divers who reported at least one episode of decompression illness involving the central nervous system had abnormalities compared to commercial divers with no reported history of decompression sickness, and concluded that central nervous system disturbance following decompression sickness in divers is more common than had been previously appreciated.

There are a number of other potential causes of nervous system damage in professional divers however, the most notable being air embolism and carbon monoxide poisoning due to contaminated air. A study of the effects of near-miss diving accidents mostly due to air embolism[5] showed that these divers had poorer spatial memory and complex sensory-motor functions compared to both divers who had no reported accidents, and non-divers. The effects of carbon monoxide poisoning have not been studied in divers, however there is considerable evidence from other occupational groups that even low-level exposure to carbon monoxide produces significant reductions in vigilance, some visual functions and time estimation abilities[6].

It is likely that the nervous system is at risk in divers who are exposed to these hazards. None of the studies conducted to date however have attempted to ascertain the extent of exposure or the circumstances which led up to the damage nor to determine systematically whether particular neurobehavioural functions are being damaged.

This study was an attempt to investigate neurobehavioural function in a working group of professional abalone divers. The tests used were from a battery designed to investigate the effects of various occupational hazards and has been successful in detecting the neurobehavioural effects of occupational exposure to inorganic mercury[7] and inorganic lead[8].

Method:

1. Description of participants:

Thirty-three professional abalone divers were tested. All were males from the Port Lincoln area of South Australia. Most, (22), were currently working but one-third were ex-divers. Of the ex-divers most had only recently given up diving and many still did relief diving. Most of the group did casual work in conjunction with their diving such as fishing and farming since they dived an average of only 91.8 days (SD=35.6) per year.

A group of 33 male non-divers were also tested for comparison purposes. This control group was matched with the diver group in terms of age, education level and their consumption of cigarettes and alcohol. Virtually, all members of both groups (100% of divers, 91% controls) were either born in Australia or had been residents for more than 15 years. Only one participant, originally from Britain, had been a resident for less than 10 years. As far as possible, the control group was selected to match the jobs, both diving and casual, of the diver group. For example, if the diver also did a skilled job eg, Electrician, a skilled worker was chosen as his control.

2. Neurobehavioural tests

The tests were chosen on the basis of information processing theory[9] and consisted of the following:

1. Critical Flicker Fusion: This is a perceptual test in which a critical flicker fusion (CFF) threshold is obtained for each eye. An electronic device was used similar to that described elsewhere[10]. Presentation of the light source was by a descending method of limits[11].

   Seven trials were presented to each eye. Each trial began with the presentation of a steady light, the switching frequency of which decreased at a constant rate of 2Hz/s until terminated by the subject pressing a button, indicating first awareness of flicker. The mean critical flicker fusion threshold value was calculated using the last five trials for each eye.

2. Hand Steadiness: This is a psychomotor coordination test. This apparatus consisted of two 17.5 cm x 5.75 cm plates bolted onto a single perspex back and held vertical at an adjustable height by a retort stand. Each plate independently formed an electric circuit when hit or touched by a stylus. The subject was required to hold the stylus in a 5 mm diameter hole between the two plates with his arm outstretched keeping it as steady as possible for 1 min. The number and duration of touches on upper and lower plates were recorded independently for the three consecutive 20 second periods.

3. Bourdon-Wiersma test: This is a test of sustained attention and psychomotor speed[12]. The subject was required to mark every group of four dots on a page of 50 rows of 25 groups of three, four or five dot groups as quickly and as accurately as possible in a 5 minute time period. The total number of groups examined (targets tracked) and the number of missed 4 dot groups (errors) were noted and expressed as a percentage of the total number of groups on the page.

4. Digit Symbol test: This is a measure of psychomotor performance using paper and pencil. It is a subtest of the Wechsler Adult Intelligence (WAIS) test battery[13]. It consists of a sheet containing 100 small blank squares arranged in four rows. Each square is paired with a randomly assigned number from one to nine. Above these rows is a printed key that pairs each number with a different nonsense symbol (eg. 7=^). Following a practice trial on the first seven squares, the subject was required to fill in as many of the blank squares with the symbol that is paired to the number above the space in 90 seconds. The number of correct responses was calculated and converted to an age-related standard score obtainable from the WAIS-R manual.

5&6 Reaction Time and Sternberg Tasks: The apparatus has been described elsewhere[14]. It was used for both reaction time and Sternberg tasks.

a) Reaction time. Subjects were tested in a simple reaction time paradigm. Beginning with the index finger of the preferred hand on the starting point, subjects were required to press the button closest to the preferred hand as quickly as possible when a O appeared on the display (the stimulus was always a O). Twenty trials were given. Mean reaction time was calculated from the last ten. After completion of the Sternberg task, a further ten trials were given and the mean calculated.

b) Sternberg Task. A set of two, three, four or five randomly selected digits from 0-9 was presented orally to the subject and he was asked to remember it. A digit was then displayed on the response box to which the subject was required to respond with the YES button if the digit was included in the memory or positive set or the NO button if the digit was not (negative set). A total of 48 presentations were made in which each type of digit set (ie: two, three, four or five digits) was represented twice and each individual set was tested six times, three each for positive and negative responses. Mean reaction time was calculated for each type of digit set.

7. Short Term Memory (STM) (Paired-associates). The test items consisted of five pairs of high (real) association (greater than 90% associative value) alphabetic trigrams selected from the list compiled by Archer[15]. The trigram pairs were presented individually using a slide projector at a rate of 1 per 5 s. Once all five pairs had been shown, the first member of each pair was shown in a random

order and the subject was required to write down the matching member. This sequence was repeated until all five trigrams were recalled correctly or following 6 trials. The number of trigrams correctly recalled on the first trial and the number of trials to criterion or the failure to reach criterion were recorded.

8. Long Term Memory (LTM): This test was a continuation of the paired-associate test described above. Without prior warning, the subjects were asked to recall the real and nonsense trigrams they had learned earlier (approximately 1.5h before). The first member of each trigram pair were presented. Number of trigrams correctly recalled was recorded.

3. Procedure

Diving histories were obtained by interviewing each diver and a questionnaire was completed by each participant to cover general biographical information such as age, education level, ethnic background, waking and sleeping habits, cigarettes and alcohol consumption and medication. Three tests, the Paired associates STM test, the Bourdon-Wiersma test and the Digit-symbol test, were carried out in groups of 5 or 6. The remainder were performed individually. In all cases the group tests were carried out first to allow sufficient time between learning and the LTM test for the Paired Associates. All other tests were carried out in random order.

4. Statistical Analysis

There were two basic comparisons of interest; the difference between the divers (including current and ex-divers) and their controls on each test and the difference between performances of current divers and ex-divers on each test. In virtually all cases the analysis was performed by t-tests or analysis of variance. For the Paired associates STM and LTM tests, however, the Wilcoxon rank sum test[16] was used because the distribution of scores clearly departed from normal and the Trials to Criterion measure was ordinal.

For two tests, the Bourdon-Wiersma and the Digit-Symbol, matched controls were not available at the time of writing. Consequently, for the Digit-Symbol test each individuals score was converted to the age-corrected standard score available for the test from large population assessments and then the diver group was compared to the population norm. Unfortunately, there are no population standards from the Bourdon-Wiersma test so the only comparison reported for this test was between current and ex-divers.

Results

Analysis of the effectiveness of the matching process (see Table 1) showed that divers and controls were not statistically significantly different in terms of age ($X^2_{(3)}=0.64$,N.S.) or education level ($X^2_{(3)}=0.41$,N.S.). Similar percentages of the two groups smoked, and the amount smoked per week was not statistically significantly different ($t_{(32)}=0.82$,N.S.). More divers admitted to drinking alcohol than non-divers but the amount consumed was not significantly different ($t_{(32)}=0.62$,N.S.).

Similar analysis between current and ex-divers showed the ex-divers were older ($X^2_{(3)}=14.98$ $p<0.01$), but their educational backgrounds were comparable ($X^2_{(3)}=3.39$,N.S.). Only one ex-diver smoked and similar numbers in the two groups admitted to consuming alcohol. Ex-divers, however, consumed significantly greater amounts of alcohol ($t_{(10)}=5.7$, $p<0.01$).

Comparison of the diving characteristics of current and ex-divers (see Table 2) showed that they were not significantly different in terms of length of diving exposure ($t_{(32)}=1.44$, N.S.) the number of instances of decompression sickness ($X^2_{(4)}=2.42$, N.S.) nor the incidence of neurological symptoms with decompression sickness ($X^2_{(1)}=0.02$, N.S.). They were different, however in the maximum depth reached on an average dive ($t_{(32)}=2.06$, $p<0.05$). Current divers typically dived much deeper than ex-divers.

The diver group had significantly lower CFF thresholds than controls as shown by analysis of variance (2 factor, repeated measures on one factor, group main effect ($F_{(1,64)}=10.99$, $p<0.01$). There was no difference in the threshold of each eye for either group (eye main effect ($F_{(1,64)}=1.1$, N.S.) and the deficiency in the diver group was the same for each eye (interaction effect $F_{(1,64)}=0.18$, N.S.).

A similar comparison between current and ex-divers (2 factor analysis of variance, repeated measures on one factor) showed no significant differences between them (group main effect $F_{(1,31)}=2.85$, N.S.) for either eye (interaction $F_{(1,31)}=0.48$, N.S.) nor between the thresholds for either eye (eye main effect $F_{(1,31)}=0.77$, N.S.). (See Table 3).

Analysis of reaction time by t tests showed no significant differences between divers and controls ($t_{(32)}=1.71$, N.S.) nor between current and ex-divers

($t_{(31)}=0.46$,N.S.). (See Figures 1 and 2).

The results of the Hand Steadiness test were analysed by two 3 factor analyses of variance (repeated measures on two factors, See Table 4). Significantly more off-target touches were made by divers than controls (group main effect $F_{(1,64)}=10.61$, $p<0.01$), particularly to the lower plate (interaction $F_{(1,64)}=7.99$, $p<0.01$). Off target touches were significantly more frequent to the lower plate compared to the upper plate in both groups (plate main effect $F_{(1,64)}=103.01$, $p<0.01$) but there was no change in off-target touches over the period of the test for either group (time main effect $F_{(2,128)}=1.79$,N.S.).

The groups were not significantly different overall in terms of the time they spent off-target in this test (group main effect $F_{(1,64)}=2.83$,N.S.). Again, the lower plate was the site of most time off-target for both groups (plate main effect, $F_{(1,64)}=82.89$, $p<0.01$) but by the diver group in particular (interaction $F_{(1,64)}=4.15$, $p<0.05$). There was no change in time off-target over the period of the test for either group as before ($F_{(2,128)}=1.15$,N.S.).

Comparison of current and ex-divers on this test (see Table 5) revealed no significant differences in their hand steadiness for either off-target touches

($F_{(1,31)}$=0.01,N.S.) or time off target ($F_{(1,31)}$=1.57, N.S.). The lower plate was the site of most off-target movement in both groups ($F_{(1,31)}$=50.3,$p<0.01$) for time; ($F_{(1,31)}$=75.8, $p<0.01$) and for touches but to the same extent in both groups ($F_{(1,31)}$=0.98, N.S.; $F_{(1,31)}$=3.6, N.S. for time and touches respectively).

For the Bourdon-Wiersma test as mentioned earlier, the only comparison that could be made was between current and ex-divers. Analysis by t tests showed that there were no significant differences between these groups either in terms of the number of targets searched over the time of the test ($t_{(31)}$=0.14,N.S.) or the number of targets missed (errors, $t_{(31)}$=-0.11,N.S.) (see Table 6).

Compared to the population's standard score, divers showed significantly poorer performance on the Digit Symbol test ($t_{(31)}$=2.74, $p<0.01$), however, again, within the Diver group, there was no significant difference between the performance of current and ex-divers ($t_{(31)}$=0.65, N.S.). (See Table 7).

The results of the Sternberg test are shown in Figures 1 & 2. Lines of best fit were calculated for speed of response against the size of the memory set for both positive (actively remembered or "Yes" items) and negative (items not in memory set or "No" items) memory sets for each group. Comparison of the slopes of the lines for Divers and Non-divers showed that for the positive set only, the cognitive component of the test took significantly longer for the diver group (positive set, $t_{(64)}=4.83$, $p<0.01$; negative set, $t_{(64)}=-1.94$ N.S.). Analysis of the intercepts also showed significant differences between divers and controls for both memory sets (positive set, $t_{(64)}=-11.07$, $p<0.01$; negative set, $t_{(64)}=-2.86$, $p<0.01$) but in both cases divers were faster than non-divers. Divers made significantly more errors on this test than controls (1.58 ± 1.12 for divers and 0.62 ± 0.95 for controls, $t_{(64)}=-3.28$, $p<0.01$). (See Table 8).

Comparison of current and ex-divers showed significant differences for cognitive and movement (intercept) components for the positive memory set (slope, $t_{(31)}=3.66$, $p<0.01$; intercept, $t_{(31)}=3.81$, $p<0.01$). There were no significant differences within the divers for the negative set (slope, $t_{(31)}=0.31$, N.S.; intercept, $t_{(31)}=1.00$,N.S.). Similarly, the number of errors was no different between the two diver groups (1.41 and 1.91 for current and ex-divers respectively, $t_{(31)}=1.22$,N.S.).

There were no differences between the performances of divers and non-divers on the short term memory measure of the Paired Associates test, the number of trigrams correct on the first round (Wilcoxon rank sum test z=-0.58,N.S.). Divers, took significantly more trials to learn the set of trigrams than controls (z=2.42, p<0.01). While more divers failed to reach criterion of all correct (39.3% compared to 27.3% for Non-divers), there was no significant difference in long term memory test performance between divers and controls (z=-1.2, N.S.).

The same comparisons for the Paired Associates group of tests showed no significant differences between current and ex-divers for the STM test (number correct, z=0.76,N.S.) trials to criterion (z=0.19,N.S.) nor the LTM test (z=0.48,N.S.). (See Table 9)

DISCUSSION

Abalone divers clearly showed poorer performance on a number of neurobehavioural tests than would be expected based on that of carefully matched non-divers. This was true for both currently working and retired divers. In particular, abalone divers showed poorer CFF thresholds, poorer hand steadiness, lower digit symbol scores, were less able to learn new material, and in the Sternberg memory test made more errors and showed a lesser ability to cope with increasing memory loads compared to controls. Nevertheless, abalone divers performed as well or better than non-divers in a few tests, namely the Paired Associates STM and LTM tests, the negative memory set in the Sternberg test (ie: items not actively being remembered) as well as the reaction time test and the motor component of the Sternberg test (intercept).

Interpreting this pattern of results from an information processing viewpoint, the most clearcut finding is of depressed CFF thresholds. Reduced CFF thresholds may indicate lowered cortical arousal[17] and/or depressed visual function either at the retina or in the intermediate visual pathway[18].

This suggestion of visual disturbance in professional divers is in keeping with reported visual affects of decompression sickness in caisson workers[2] which were attributed to "retinal aeropathy". Visual impairment may also be due to inappropriate use of oxygen for treatment of decompression sickness or for carbon monoxide toxicity. It is known that oxygen under pressure can be neurotoxic and, in particular, affect the retina in humans[19].

The finding of poorer hand steadiness is consistent with the report of intentional tremor in divers with known histories of decompression sickness[4] and of psychomotor impairment in divers who had air embolism and/or hypoxia[5]. In this study however the only psychomotor effects were increased tremor. Reaction time abilities were at least as good as those of non-divers in the Reaction time test and even better than non-divers as shown by the motor components of the Sternberg test.

It is possible that differences in motivation may be responsible for the superior response times of the diver group. The reaction time test is a simple test of motor speed whereas the Sternberg test is a test requiring both speed and accuracy. Divers may be able to react faster than non-divers on this test because they were sacrificing speed for accuracy. Similarly, divers made significantly more errors than non-divers indicating either that they often moved before they had made an appropriate decision, or that they were not remembering the information correctly in the first place, or both.

The results of the Paired Associates STM test did not indicate any deficiencies in the Short term memory of divers compared to controls however the results of the Sternberg memory test show that divers did not deal with increasing memory loads as well as controls. The Sternberg test makes it possible to distinguish the cognitive or thinking components of a memory test from problems of responding[14]. Sternberg's theory predicts that the slope of the line which describes the relationship between memory set size and reaction time is an indicator of the relative increase in reaction time with each additional memory item and therefore reflects cognitive elements in the test such as encoding of the information and item matching. Thus the steeper the slope, the more time the individual takes to cognitively process the information and the slower the memory process. Since divers had significantly steeper slopes for the actively remembered items (positive set or Yes items) than controls it must be concluded that they needed more time to process this information before making a response. Both previous studies of divers[4,5] found that the affected divers were characterised by memory problems.

On the other hand, in this study, the cognitive component of the information not actively remembered (negative set or No items) was no different from that of controls. Sternberg's theory maintains that when presented with an item the individual searches the actively remembered set (Yes set) of items first before deciding that his/her response should be Yes or No. Consequently the slope for the No responses is usually about the same or steeper than that of the Yes responses. For the control group in this study this was true, but the divers were considerably faster in their cognitive responses to the No items than to the Yes items. This suggests again that the divers were trying for speed rather than accuracy. It is likely that they were only partially completing the search through the remembered set before making the decision in the negative. This would explain too, why such a high percentage (74.1%) of errors were of the No-should-have-been-Yes type.

It is likely that professional abalone divers are a unique group quite apart from any of the neurobehavioural health effects of their chosen occupation. Abalone diving, like other potentially dangerous occupations eg firefighters[20] probably attracts a high proportion of risk-takers. The results on the tests in this study, and possibly from other studies as well, may reflect this performance bias. Therefore the performance of professional divers on tests that can be influenced by incentive or motivational differences, eg tests requiring speed like reaction time, maybe confounded by their use of different strategies such that deterioration in neurobehavioural functions may be difficult to detect.

Nevertheless, there were detectable differences between divers and their matched controls in visual, short term memory and some psychomotor abilities which are consistent with findings from previous studies and are suggestive of impairment of nervous system function.

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TABLE 1: Comparison of characteristics of current and exdivers and nondiver. Controls showing percentages means and standard deviations or medians and ranges where appropriate.

	Current Divers	Exdivers	Total Divers	Non-divers
Age (median years)	35.0 (21-48)	44.5 (40-53)	40.0 (21-53)	40.0 (20-59)
Education (median years)	11.8 (6-16)	11.2 (6-17)	11.5 (6-17)	12.1 (6-17)
% Smoking	31.8	10.0	24.2	27.3
No.cigarettes smoked per week (mean)	20.7 (6.1)	25.0 (n.a)	21.3 (5.8)	20.1 (4.6)
% Using alcohol	72.7	80.0	80.0	54.5
Alcohol/wk (mean,gms)	84.8 (93.5)	157.8 (116.9)	115.0 (103.8)	107.0 (82.94)

TABLE 2: Comparison of the diving experience of current and exdivers showing extent of exposure, the maximum depth reached on a typical dive and the incidence of decompression sickness per individual with and without neurological symptoms.

	Current Divers	Exdivers	Total Divers
Exposure (mean, hrs)	4685 (3603)	6350 (2877)	5205 (3436)
Maximum depth (mean, ft)	68.6 (16.1)	56.5 (15.8)	64.8 (16.8)
Incidence of decompression sickness per individual (median)	3.5 (0-100)	4 (0-15)	3.75 (0-100)
% reporting neurological symptoms with decompression sickness	50	40	45

TABLE 3: Results of the Critical Flicker Fusion test showing means and standard deviations for divers and controls and current divers and exdivers.

	Divers n=33	Nondivers n=33	Current divers n=22	Exdivers n=11
CFF Left eye	34.1 (4.7)	37.9 (4.5)	34.9 (4.7)	32.6 (4.6)
CFF Right eye	33.7 (4.89)	37.4 (5.00)	34.7 (4.3)	31.6 (5.6)

TABLE 4: Hand steadiness test results for divers and controls showing means and standard deviations for time off target and the number of off-target touches to the upper and lower plate in each consecutive 20 second time period.

		Divers Time Period			Controls Time Period		
		1	2	3	1	2	3
Off-target touches	Upper	23.9 (9.3)	21.8 (9.8)	21.2 (13.7)	18.9 (8.8)	18.3 (8.7)	16.6 (9.3)
	Lower	33.4 (9.9)	34.8 (11.6)	33.9 (14.6)	24.8 (11.6)	25.9 (9.4)	23.1 (10.5)
Time off target	Upper	19.8 (10.5)	18.8 (10.4)	17.7 (12.9)	16.8 (13.1)	16.1 (9.9)	15.7 (13.7)
	Lower	32.5 (17.7)	31.6 (13.5)	31.6 (21.7)	26.0 (17.3)	25.2 (15.7)	22.3 (16.8)

TABLE 5: Results of the hand steadiness test showing means and standard deviations for off-target touches and time off target of upper and lower plate for each of three consecutive 20 second time period comparing current and exdivers.

		Current divers Time period			Exdivers Time period		
		1st	2nd	3rd	1st	2nd	3rd
Off-target touches	Upper	22.8 (9.5)	21.5 (9.8)	19.8 (11.2)	26.1 (7.9)	22.4 (9.3)	23.9 (16.8)
	Lower	34.1 (9.1)	36.4 (12.1)	33.6 (12.6)	32.0 (10.8)	31.7 (8.9)	34.4 (17.4)
Time off target	Upper	18.5 (8.2)	18.0 (8.1)	16.0 (8.6)	22.1 (13.4)	20.5 (13.3)	21.1 (17.9)
	Lower	30.9 (12.6)	30.7 (11.3)	27.3 (11.1)	35.7 (24.1)	33.4 (16.4)	40.0 (31.8)

TABLE 6: Results of the Bourdon-Wiersma test showing means and standard deviations for number of targets tracked and percentage errors for current divers and exdivers.

	Current Divers	Exdivers
Targets tracked	147.64 (27.5)	146.18 (27.6)
% errors	10.6 (6.73)	10.1 (6.74)

TABLE 7: Digit symbol test results for Total diver group and for current and exdivers showing means and standard deviations.

	Total diver group	Current	Exdivers
Scaled score	8.9 (2.09)	8.8 (2.17)	9.2 (1.91)

TABLE 8: Errors on the Sternberg memory test for comparisons between divers and nondivers and current and exdivers. Means and standard deviations are shown.

	Divers	Nondivers	Current divers	Exdivers
Sternberg test errors	1.58 (1.12)	0.62 (0.95)	1.4 (1.01)	1.9 (1.1)

TABLE 9: Results of the Paired Associates test showing medians and ranges for the comparisons; Divers with nondivers and current divers with exdivers. The STM measure, number correct, Trials to criterion and percentage of each group reaching criterion are shown as well as the LTM number correct measure.

	Divers	Nondivers	Current Divers	Exdivers
STM, no.correct	2.0 (0-4)	2.0 (0-5)	2.0 (0-4)	2.0 (0-3)
Trials to criterion	4.5 (2->6)	3.0 (1->6)	3.5 (2->6)	3.0 (2->6)
% reaching criterion	60.6	75.6	63.6	50.0
LTM, no.correct	2.5 (0-5)	3.0 (1-5)	3.0 (0-5)	2.0 (1-5)

Figure 1: Results of Reaction Time and Sternberg memory tests, showing mean reaction time for Divers (D) and Non-Divers (C), and lines of best fit for Sternberg memory tests showing Yes responses (Y) for Divers and Non-Divers, and No responses (N) for Divers and Non-Divers.

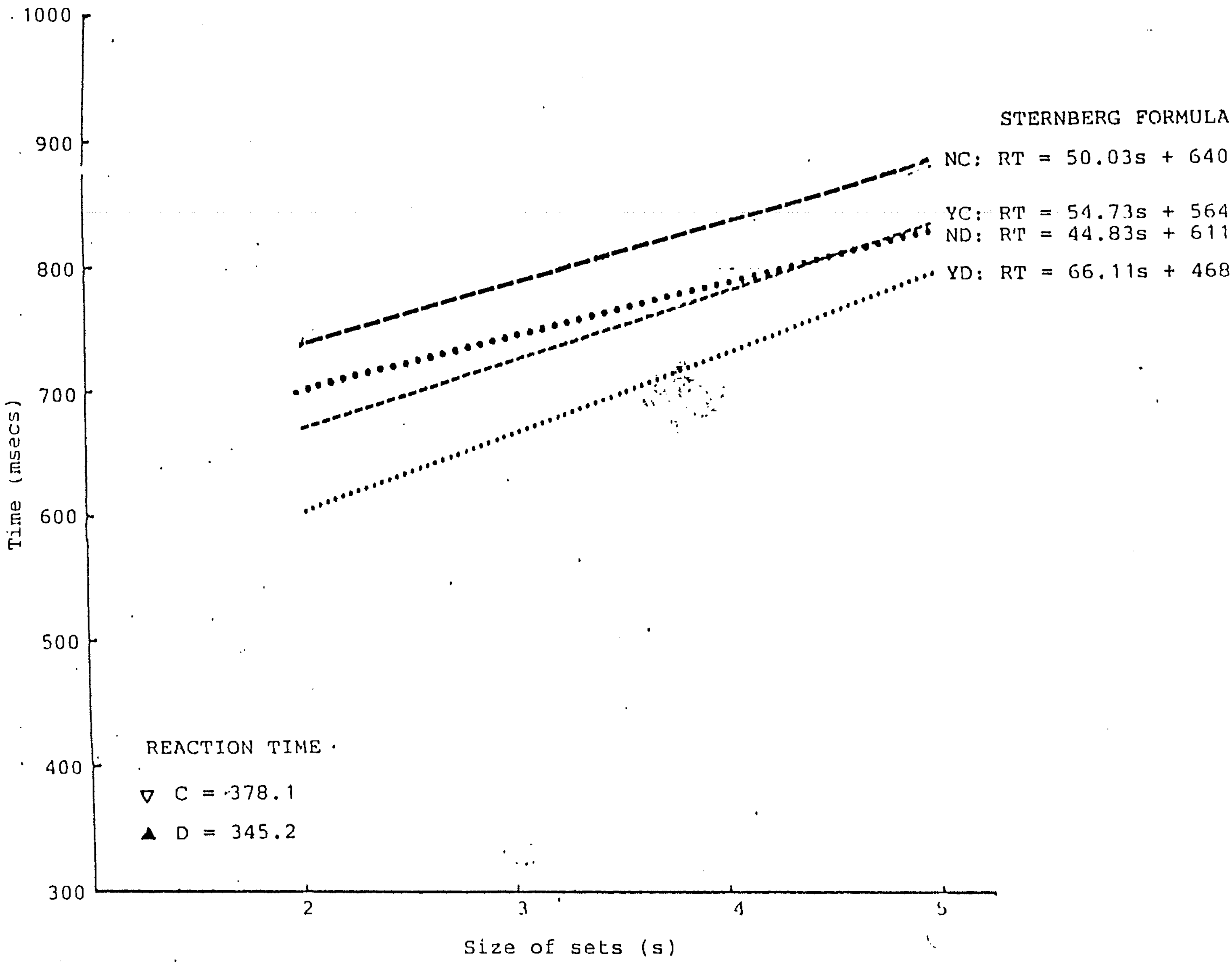
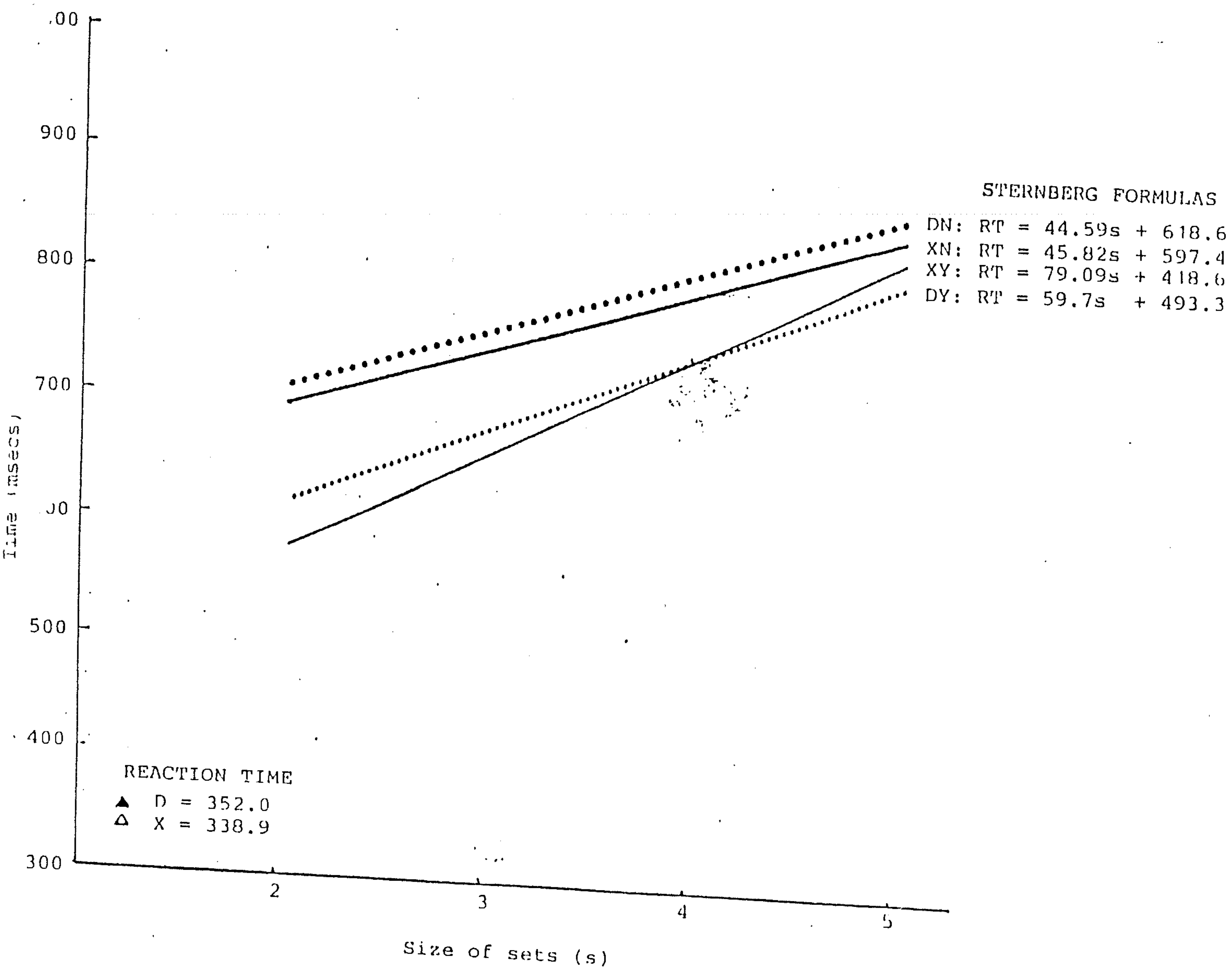


Figure 2: Results of Reaction Time and Sternberg memory tests, showing mean reaction time for Divers (D) and Ex-Divers (X), and lines of best fit for Sternberg memory tests showing Yes responses (Y) for Divers and Ex-Divers, and No responses (N) for divers and Ex-Divers.



The findings of Part I suggest that there is a relationship between various aspects of the Diver's exposure to the underwater environment and such neurobehavioural functions as vision, memory and hand-eye coordination. In this part of the study, the aim was to investigate the nature of this relationship in order to attempt to establish whether the observed deficiencies in the above neurobehavioural functions can be related to the Diver's occupation and to establish their causes if possible. By taking divers from different geographical areas a broad range of diving experiences were obtained due to differences in the diving terrain and local habits. This provided a wide variation in many of the diving variables of interest such as the maximum depth of the dive and whether or not oxygen is commonly used to aid decompression.

1. Description of participants:

Forty-six divers from Tasmania, 12 from southern New South Wales, and 22 from the Port Lincoln area of South Australia were tested. All were male with ages ranging from 23 years to 51 years (mean=34-5 years). All were active Divers at the time of testing.

2. Procedure:

As in Part I of the study, data was collected on the diving history of each participant by interview and general biographical information and details of daily routine by questionnaire.

The tests were the same as in Part I and were carried out in the same way. Additional diving information was gathered for this section. As well as the amount of diving per year, information was collected on the number of years they had dived, and the typical dive including its length, depths reached, whether decompression stops were required and how this problem was overcome and whether oxygen was used to aid decompression. Information was also gathered on the number of incidences of decompression sickness with and without neurological symptoms. Thus 6 diving variables were used. These were exposure or the number of hours dived in the individual's working life (calculated by hours per day x days per year x total years dived) the maximum depth that was achieved on an average, the number of incidences of general decompression sickness and the number of incidences of decompression sickness with neurological symptoms and their degree of decompression stress or the amount of risk each diver subjected himself to on a typical dive. For this measure a five-point scale was used in which, in order of increasing risk, 1= no decompression diving eg diving to less than 10 metres, 2= diving within the US navy decompression tables to depths greater than 10 metres, 3= diving in which recompression was "logical" eg diving in deeper water for first part of the working day then moving to shallower water but no meter etc used, 4= diving which could require decompression stops of up to 60 minutes on average but not done, 5= diving requiring decompression stops of more than 60 minutes on average but not done. The final diving variable was named the Oxygen Factor and was measured on a four point scale where 1= never used it, 2= oxygen used only on surface, 3=oxygen used underwater to 10 meters, 4= oxygen used underwater to depths in excess of 10 meters.

3. Statistical Analysis:

In this part of the study the question of interest was the relationship between the amount of exposure to the underwater enviroment and the neurobehavioural deficits observed in the earlier part of the study. Multiple linear regression analysis (Lewis-Beck, 1980) was used to determine which diving variables and/or what combinations of variables best "explained" the neurobehavioural results.

RESULTS.

The divers from each location were very similar on a number of demograhpic variables. There were no differences in age ($F_{(2,74)}$=2.85, N.S), education level ($X^2_{(6)}$=7.63, N.S), the percentage who smoke ($X^2_{(2)}$=0.29, N.S) and the amount smoked ($F^{(2,21)}$=0.16, N.S.), the amount who take medication ($X_2^{(2)}$=0.32, N.S) and the number who cansume alcohol ($X_2^{(2)}$=1.99, N.S). Tasmania divers reported consuming significantly more alcohol than the other groups ($F_{(2,63)}$=3.15, p<0.05).

There are however large differences between locations on the diving variables. The NSW Group had dived for significantly more hours than the other two groups ($^{(2,73)}$=11.19, p<0.01). The Tasmanian divers dived much shallower on average than either South Australian or NSW divers ($F_{(2,73)}=11.03$, $p<0.01$). The incidence of Decompression sickness was very different between the locations ($F_{(2,73)}=7.14$, $p<0.01$). The South Australian Divers had a much higher incidence than divers at other locations with the Tasmanian divers having very little decompression sickness. The results for neurological decompression sickness were similar. With half of the divers from South Australia reporting neurological symptoms, but none or virtually none in the other two groups ($X^2_{(6)}=27.6$, $p<0.01$). Not surprisingly in terms of diving styles, the risk of Decompression sickness was similar to its incidence. South Australian divers were more at risk than either of the other groups with Tasmanian divers significantly lowest in risk ($X^2_{(8)}=34.0$, $p<0.01$). Oxygen use too differed significantly between the groups ($X^2_{(10)}=72.34$, $p<0.01$). South Australian divers were the only group to use oxygen to any appreciable extent.

Many of the diving variables were interrelated (see Table 7). The three decompression sickness variables, general incidence and incidence with neurological symptoms and decompression stress were all significantly and positively related indicating that as would be expected decompression sickness increases when risky diving is undertaken and that neurological symptoms are more likely to be included when decompression sickness is frequent. These variables were also all significantly related to the maximum depth reached and the use of oxygen. Again, as expected, decompression sickness risk increases with increasing depth, as does oxygen use.

The only independent diving variable was length of exposure which was not related to any other. Length of exposure was however related, not surprisingly, to age and also to the number of cigarettes smoked indicating that divers who had dived longest smoked the most. Higher levels of smoking was also associated with high decompression stress. The only other significant relationship between diving and demographic variables was between the incidence of decompression sickness and medication. Divers with higher incidences of decompression sickness were less likely to take medication.

Eight variables were selected as predictors in a multiple linear regression analysis. These included the six diving variables as well as age and education level since these last two are well-established as potential confounders in measures of neurobehavioural function (Lezak, 1983). The results of this analysis are shown in Table 8 . Twelve measures were explained by at least one of the predictor variables. With the exception of Sternberg errors all measures were explained by the quality of diving variables rather than the quantity of diving measure, exposure.

Reaction time and Bourdon-Wiersma targets tracked were explained by maximum depth. CFF-both eyes, Paired Associates-STM, Sternberg intercepts of the No response, and Bourdon -Wiersma-targets tracked were predicted by one of the decompression sickness variables. Oxygen use predicted the measure slopes of the Yes responses of the Sternberg test.

Five measures, CFF left eye, Bourdon-Wiersma-targets tracked, Sternberg slopes for the yes responses, Sternberg errors and Trials to criterion on Paired associates test were also predicted by either of the demographic variables used. For two measures, lower time off-target in the Hand Steadiness test and the Digit Symbol test, demographic variables were the only significant predictors. The remaining measures were not significantly explained by any of the predictor variables.

As there were significant differences between Tasmanian divers and divers from the other two locations in alcohol consumption, stepwise linear regressions were attempted with this additional variable as a predictor. The only measure to be changed was the STM measure of the Paired Associates test. The equation then became $Y=-0.005$ X $ALC/WK+1.86$ ($r=-0.37$, $F_{(1, 38)}=5.87$, $p<0.02$). This variable was not included in the original predictor set as it resulted in a significant reduction in sample size since a relatively large number of divers did not drink and because it made little difference to the final outcome for the they dived since they encountered the greatest depths and were experiencing the greatest decompression sickness risk. This was manifest in their high decompression sickness rates. The NSW divers on the other hand were experiencing greatest risk from the amount of diving they had done. On average, they had dived three times more than either of the other two groups. The Tasmanian divers however typically dived in relatively shallow water and their length of exposure was relatively short. The inclusion of divers from these three locations therefore provided the necessary wide range of both lengths of exposure and diving styles.

Diving style was undoubtedly the major influence on neurobehavioural function. Most tests were predicted by variables relating to diving technique. Performances on the CFF test were related to the incidence of neurological symptoms in decompression sickness, with poorer CFF test performance by divers with high frequencies of symptoms. This reinforces the finding in part 1 of lower CFF thresholds in Divers compared to nondivers and again those of a previous study of decompression sickness (Rosahegyi, 1959). The fact that age was also a predictor variable for CFF thresholds with the left eye can be attributed to the acknowledged decline in CFF thresholds with age which becomes pronounced after about 55 years of age (Misiak, 1967). As all divers in this study were well below this age it is likely that this result reflects only a slight deterioration due to age which is only observable in one eye. Even the older exdivers in part 1 showed no difference in CFF thresholds compared to the younger current divers.

The reaction time and Bourdon -Wiersma tests, both perceptual-motor tasks, were predicted by the variable, maximum depth. Divers who reached greater depths tended to have faster reaction times and track more targets in the Bourdon -Wiersma, test perhaps reflecting the risk-taking characteristics of divers as discussed in part 1. For the Bourdon -Wiersma test, decompression stress was also a significant predictor but in the opposite direction, divers who subjected themselves to greater risk of decompression sickness tracked fewer targets. This finding would appear to negate the risk-taking hypothesis mentioned above. However, decompression stress is a much better predictor of the risk of decompression sickness than is the maximum depth variable ($r=0.41$ for decompression stress and $r = 0.3$ for maximum depth). Therefore, the relationship between targets tracked in the Bourdon -Wiersma test and the decompression stress variable reflects the disabling effects on perceptual-motor performance in divers subjecting themselves to conditions which produce decompression sickness.

The results for the Sternberg test show that decompression stress is again a predictor and that the greater the risk for decompression illness, the poorer the performance on both positive and negative set items. Exposure also emerged as a predictor of performance for the negative memory set, such that divers with longer exposure showed poorer performance on this measure. For Sternberg errors, too, exposure was an influencial variable but for this measure more errors were made by the divers with the shortest exposure. This result suggests, as argued in Part 1, that the greater speed achieved by Divers in this study reflects a speed-for-accuracy tradeoff in which Divers risk correct responses in favour of increasing speed.

For the group of memory measures in the Paired Associates test only the STM measure showed any relationship with diving variables (ie: maximum depth) however this variable was removed when alcohol consumption was added in to the equation. The more alcohol the diver consumed the fewer paired associates he remembered. This is not surprising as there is abundant evidence that short term memory abilities can be disrupted by chronic and acute alcohol consumption (Birnbaum and Parker, 1977).

The other two potential confounding variables of interest, age and education level were influencial on a number of measures both alone and in concert with the diving variables. Education level made the most impact, being a predictor for the Bourdon-Wiersma targets tracked measure and for both Sternberg memory test measures in conjunction with diving variables. For the *Digit symbol test, the Trials to criterion measure of the Paired Associates test and one measure of the Hand Steadiness test, time off-target to the lower plate, education level was the only significant predictor. For all measures, increasing education level was associated with better performance. It is known that people unused to handling pencils and/or with few skills do poorly in the Digit Symbol test (Lezak, 1983) so this finding is not surprising. Similar factors are likely to be responsible for the relationship between education and the other tests.

The relationship between age and CFF thresholds has already been discussed, however the error measure of the Sternberg test was also related to age, such that more errors were made by older divers.

GENERAL DISCUSSION

Abalone Divers showed deficits in neurobehavioural function compared to Nondivers some of which seem to be related to their occupation. Specifically, Divers showed poorer visual function, poorer psychomotor performance, lower ability to learn new information and reduced abilities to cope with memory loads compared to Nondivers. Of these functions, visual function (CFF thresholds), some of the psychomotor abilities, namely reaction time and the Bourdon-

Wiersma test, and memory as shown by the Sternberg test results were all affected by aspects of their occupational exposure. Differences between Divers and Nondivers in the Digit Symbol test, the Paired Associates learning measure, Trials to Criterion and the Hand Steadiness appear to be due to aspects of their education rather than their occupation.

Abalone diving appeared to affect neurobehavioural function mostly through diving styles rather than length of exposure to the underwater environment. Divers whose techniques increase their risk of decompression sickness were most affected. So it seems that the neurobehavioural affects in Abalone Divers are due to the effects of decompression sickness and not to other potential hazards in their underwater exposure.

It could be argued that the differences between Abalone Divers and controls arise not due only to their occupational exposure and that, in fact the contribution of the Divers occupation to their lowered performance is very small. While this must certainly be the case, it is extremely likely that the Diving variables are underestimates of the true situation. Divers could only estimate their dive pattern, the incidence of neurological symptoms of decompression sickness and of decompression sickness itself for example. It is likely that many episodes were not recalled and, if the symptoms were slight, were not even acknowledged to themselves. In addition, many divers may have been reluctant to reveal incidents nor their true dive profiles in case of repercussions which interferred with their livelihood.

The overall effect of these factors would be to depress the strength of the relationship between diving style and performance.

It should be noted that Abalone Divers showed a tendency to risk-taking which is perhaps not surprising given the inherent dangers in their work. Nevertheless, this characteristic clearly affected their performance on a number of the neurobehavioural tests used in this study and presumably in other studies as well. Not only should this be taken into account when interpreting results from other Diver groups, but it should also be recognised when determining safety limits for Abalone Divers as an occupational group. In knowing that the Divers may be high risk-takers, greater emphasis should be placed on educating them about proper safety precautions, ensuring regular health checks particularly preemployment and adequate medical first aid should be available in the vicinity of diving locations.

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TABLE 1 : Comparison of characteristics of the Port Lincoln, South Australia Diver group and Non-Diver controls.

	Divers	Non-divers	
Age (median years)	40.0 (range 21-53)	40.0 (range 20-59)	$X^2_{(3)}=0.64$ N.S.
Education (median years)	11.47	12.09	$X^2_{(3)}=0.41$ N.S.
% Smoking	24.2	27.3	
No. cigarettes smoked per week (mean)	21.3	19.0	$t_{(15)}=0.82$, N.S.
% using alcohol	72.7	54.5	
Alcohol/wk (mean gms)	118.6	99.91	$t_{(15)}=0.62$, N.S.

TABLE 2 : Results of the Critical Flicker Fusion test showing means and standard deviations for Divers and Controls and Current Divers and Exdivers.

	Divers n=33	Nondivers n=33	Current divers n=22	Exdivers n=11
CFF Left eye	34.1 (4.7)	37.9 (4.5)	34.9 (4.7)	32.6 (4.6)
CFF Right eye	33.7 (4.89)	37.4 (5.00)	34.7 (4.3)	31.6 (5.6)

TABLE 3 : Results of the Bardon-Wiersma test showing means and standard deviations for number of targets tracked and percentage errors for Current Divers and Exdivers.

	Current Divers	Exdivers
Targets tracked	147.64 (27.5)	146.18 (27.6)
% errors	10.6 (6.73)	10.1 (6.74)

TABLE 4 : Digit symbol test results for Total diver group and for Current and Exdivers showing means and standard deviations.

	Total diver group	Current divers	Exdivers
Scaled score	8.9 (2.09)	8.8 (2.17)	9.2 (1.91)

TABLE 5 : Results of the Paired Associates test showing medians and ranges for the comparisons; Divers with Nondivers and Current divers with Exdivers. The STM measure, number correct, Trials to criterion and percentage of each group reaching criterion are shown as well as the LTM number correct measure.

	Divers	Nondivers	Current Divers	Exdivers
STM, no.correct	2.0 (0-4)	2.0 (0-5)	2.0 (0-4)	2.0 (0-3)
Trials to criterion	4.5 (2->6)	3.0 (1->6)	3.5 (2->6)	3.0 (2->6)
% reachin criterion	60.6	75.6	63.6	50.0
LTM, no.correct	2.5 (0-5)	3.0 (1-5)	3.0 (0-5)	2.0 (1-5)

TABLE 6 : Errors on the Sternberg memory test for comparisons between Divers and Nondivers and Current and Exdivers. Means and standard deviations are shown.

	Divers	Nondivers	Current divers	Exdivers
Sternberg test errors	1.58 (1.12)	0.62 (0.95)	1.4 (1.01)	1.9 (1.1)

TABLE 9 : Demographic characteristics of Abalone Divers from Port Lincoln, South Australia, South coast of NSW and Tasmania

	Diving Locations		
	South Australia	N.S.W.	Tasmania
Age(yrs)	34.9	38.0	33.2
Education(Md yrs)	11.1	10.38	10.9
% Smoking	31.8	25.0	30.4
No. smoked per day	20.7	17.0	19.6
% taking medication	15.0	8.3	14.0
% consuming alcohol	85.0	100.0	90.7
Alcohol(gms/wk)	84.8	92.2	178.7

TABLE 7 : Comparison of diving-related variables for divers at each geographical location.

	Location		
	South Australia	N.S.W.	Tasmania
Exposure(hrs)	4685	12,130	4631
Maximum depth reached (ft)	68.6	61.2	45.1
Incidence of Decompression sickness (no.times per individual)	13.5	2.0	0.4
% experiencing neurological decompression sickness symptoms	50.0	9.0	0
% Decompression stress risk			
Low(1-2)	13.6	18.2	51.2
Intermediate(3)	0	18.2	20.9
High(4-5)	86.4	63.6	27.9
% Oxygen use			
Never Used	36.4	81.8	92.9
Surface only	9.1	18.2	4.8
Surface and to 30 ft	18.2	0	2.4
Used below 30ft	36.4	0	0

TABLE 8 : Correlations between exposure variables and between exposure variables and demographic variables for all Abalone Divers

	Exposure	Maxdepth	Decompression Incidence	Neurological	Stress	Oxygen Use
Exposure	-	-	-	-	-	-
Maxdepth	0.01	-	-	-	-	-
Deco. Incidence	0.01	0.30+	-	-	-	-
Neurological	0.17	0.29	0.73	-	-	-
Stress	0.01	0.59	0.41	0.41	-	-
Oxygen use	-0.05	0.49	0.52	0.61	0.53	-
Age	0.53	-0.12	0.04	0.21	0.002	0.09
Education	-0.17	0.001	0.04	0.003	0.05	-0.09
Smoking* consumption	0.46	0.12	0.14	0.10	0.60	0.15
Alcohol* consumption	-0.05	-0.13	-0.12	-0.09	-0.12	-0.05
Medication	-0.1	0.14	0.40	0.16	0.19	0.27

* Based on n=20 and n=61 for smoking and alcohol respectively. Remaining correlations based on n=70.

Correlations underlined are statistically significant $p<0.05$.

TABLE: REGRESSION COEFFIENTS AND CONSTANTS FOR EQUATIONS RELATING PREDICTOR VARIABLES AND NEUROBEHAVIOURAL MEASURES.

TEST OR MEASURE.	EXPOSURE.	MAXIMUM DEPTH	INCIDENCE	NEUROLOGICAL	DECOMPRESSION STRESS	OXYGEN USE	AGE	EDUCATION LEVEL	CONSTANT	MULTIPLIER
CRITICAL FLICKER FUSION:							-0.29			
LEFT EYE.				-2.5					47.8	0.62
RIGHT EYE.				-2.8					37.5	0.49
REACTION TIME:		-0.09							41.59	0.29
HAND STEADINESS :										
OFF TARGET TOUCHES.										
-UPPER PLATE										
-LOWER PLATE										
TIME OFF TARGET										
-UPPER PLATE										
- LOWER PLATE.								-1490	1353	0.32
BOURDON-WIERSMA:										
TARGETS TRACKED		0.09			-2.4			1.98	31.88	0.38
ERRORS										
DIGIT SYMBOL								0.98	4.86	0.36
STERNBERG MEMORY					0.09			-0.11	2.85	0.42
POSITIVE SET					0.09			-0.15	3.16	0.51
NEGATIVE SET							0.09		-1.23	0.34
ERRORS	$-8x10^{.5}$									
PRED ASSOCIATES									1.07	0.36
STM NO CORRECT			0.02					-1.26	10.94	0.28
TRIALSTO CRITERION										
LTM NO CORRECT										

Chapter 19

LONG TERM NEUROPSYCHOLOGICAL EFFECTS OF COMPRESSED AIR DIVING

A REVIEW

LINDA HAYWARD

Neuropsychological assessment procedes with gathering the above information and testing across a variety of specific behaviours. Many neuropsychological tests have been devised to specifically assess brain behaviour relationships. Often a test is developed using patients with known cortical lesions and then the test performance of groups of people with different types of brain damage is compared. These tests are soundly constructed to test specific abilities and are not the same as asking a patient if they have memory problems, concentration problems etc.

Some of the literature on neuropsychological effects of decompression sickness do not actually use true neuropsychological tests. Rozsahegyi(1959) for instance presents only patient reports of symptoms like forgetfulness. These subjective accounts do not provide the basis for asserting the presence of neuropsychological damage.

Tests borrowed them from other areas of psychological interest (e.g. educational and aptitude testing) are sometimes used for assessing brain-damage. These tests have been used largely because of the appeal of using standardised, reliable tests with good normative data. Although these tests can be useful as part of a test battery, they were not designed for neuropsychological purposes and their use as a sole diagnostic tool is limited, largely because performance on a test or subtest is dependent on a number of skills, a decrement in any one of which can produce poor performance.

The Wechsler Adult Intelligence Scale (WAIS) is one test that has been widely used. It is a test of general intellectual ability that provides a total IQ score that is a composite based on subtest performance. The IQ score is derived from 11 subtests, six of which are associated more with verbal abilities and the other five with nonverbal ones. However the subtests themselves are relatively complex in that peformance is dependent on a number of skills. For most normal people, performance on each of the subtests is fairly consistent, but with brain damaged people performance on the subtests can show marked variation. It turns out that different profiles or patterns of performance on the WAIS subtests are consistent with lesions in different parts of the brain and would prompt the neuropsychologist to do further assessment. The WAIS total IQ scores, however, are not especially useful in neuropsychological diagnosis. Further, there are some types of brain damage, for example mild right hemisphere and frontal lobe damage, for which a patient may produce a normal subtest profile on the WAIS (Lezak, 1983).

The Multidimensional Apptitudes Battery or MAB (a test similar to the WAIS but administered to groups not individuals) was used for the assessment of divers abilities by Edmonds and Coulton (1986). These authors found no real relationship between test performance and various diving indices. Unfortunately, as already explained, these tests were not designed for fine grained neuropsychological diagnosis, especially if the aim of a study is to pick up the early (and perhaps subtle) effects of damage due to cumulative dive stress rather than the acute effects of damage due to neurological decompression sickness.

In clinical practice, neuropsychological assessment usually is done by giving the client a battery of tests. Test batteries commonly include some measure of IQ like the WAIS, but also include a variety of tests each sampling different aspects of behaviour. Tests of memory, expressive and receptive languge, motor skills, sensation and perception, visuospatial skills, and executive functions would form the basis for any neuropsychological test battery. Inconsistencies in performance across tests, and the patterns of performance provide the basis for diagnosis.

Diagnosis of brain damage must however be made in the context of accounting for the effects of a number of variables. First, there is great variation between individuals in performance on many of these tests. People who have a better than average IQ score, for instance, are also likely to perform better than average on tests of memory and executive functions, conversely those with below average IQ scores will also tend to perform below average on some other tests. To complicate matters further, variables such as age and education are also highly correlated with test performance. These issues present difficulties when comparisons between groups of people, or of a person against normative data are made. Some method of taking into account age, education and ability level must be employed, for instance, by using controls matched to the diving group on these varariables, or by using a statistical procedure (e.g. multiple regression) to control for the effects of confounding variables. As will be discussed later, this is an issue that has not always been adequately dealt with in the studies under review.

A final point about performance on neuropsychological tests is that the chronicity of the brain insult is an important variable in determining test performance. Generally speaking, acuteness of injury is associated with much more marked decrements in performance than would be seen at som later time. For instance, a person who has had a stroke will perform more poorly soon after the event than they will two years later. During those two years, the greatest cognitive gains will occur during the first six months (Bond, 1979), with performance levelling off after that. As a consequence, chronic damage (the residual effects of brain insult) can be more difficult to detect than acute damage. Furthermore, tests performed shortly after an incident will be an overestimate in many cases of the amount of damage that will remain in the future, the exception being degenerative neurological conditions. Again, some of the studies under review do not report the time since last decompression sickness (Vaernes and Eidsvik,1982; Boughton and Edmonds, 1985; Andrews et al, 1986; Edmon and Coulton, 1986; and Williamson and Clark, 1986) and if they do report it (Levin, 1975; and Peters, Levin and Kelly, 1977) they make no attempt to control for the time since injury. The only article which clearly restricts itself to the chronic effects of decompression illness is that by Rozsahegyi (1959). Unfortunately, this the article provides the weake evidence, since it has no comparison group and does not use proper neuropsychological tests.

Studies where a major concern has been the cumulative effects of exposure to compressed air diving face a slightly different problem. The effects they are seeking to detect are probably quite subtle and therefore require fine grained neuropsychological assessment. As mentioned earlier, the use of the WAIS or similar tests, or single tests of brain damage, are not appropriate choices for tests to detect subtle effects.

Methodological Issues

In order to demonstrate a relationship between a set of environmental conditions like dive stress or decompression sickness and some behavioural outcome, a number of conditions must be met. It is probably easiest to provide an explanation of these requirements in the context of pointing out what is wrong with the typical methodologies that have been used.

One approach used by some earlier researchers in this area was to simply document cases of sufferers who were divers and put their psychological and health problems down to some aspect of diving (e.g. Rozsahegyi,1959). Such an approach always leaves open the opportunity for alternative explanations of the observations. For instance, it could be argued that that a particular patient or set of patients were not representative of the diving population. An example of this would be where people who are seeking compensation for injuries compose the cases studied, as was the case for divers in Levin (1975). Such people, it could be argued, have potential to gain from being found to be abnormal and might not produce a performance on tests consistent with their ability. The other problem with simply reporting observations is that the potential for the observer to unwittingly bias his observations, perhaps by selective reporting of some symptoms and not others, is not controlled.

A more sophisticated approach to the question of whether diving accidents or exposure to compressed air diving causes brain damage is to compare divers to some other group (a control or comprison group). This strategy certainly has more merit than the preceding one, but, the way in which both the divers and the other group are selected is critical. It is necessary that the two groups differ from each other only in the presence or absence of the variable of interest, e.g. decompression sickness. If this is not the case, observed differences in performance between groups could arise from any number of the ways in which they differed. Further, it is necessary that subjects are selected in such a way as to reduce the type of bias arising from them not being representative of the population to which generalisations are to be made. Examples of inappropriate selection of groups are provided by Levin (1975) and Peters, Levin and Kelly (1977). Levin (1975) presents the neuropsychological test results o 15 divers who had complained of symptoms following a diving accident and for most of whom litigation was pending. Judgements were made such that 5 divers who were considered not to have CNS involvement (the criterion for this judgement was not clearly specified) were compared to 6 divers who were, because their "complaints of CNS symptoms were substantiated by the test battery" (p.VI-234). A further 4 divers were excluded from comparison "with equivocal neuropsychological findings"(P. VI-243). Although it is not quite clear in the paper, it appears that the groups were determined by performance on the very tests they were later compared on. Under such circumstances it is not at all surprising that Levin reports differences in performance between the two groups, since they were selected in such a way as to maximise any difference. The discarding of 4 of the divers' results, for whatever reason, only served to maximise the difference between groups.

Peters, Levin and Kelly (1977) divided a sample of 19 divers who underwent neuropsychological testing into two groups based on their performance on the neuropsychological tests. Seven were deemed to be "impaired". Eight who were considered not to be impaired were used as the control group and the responses of 4 patients were not included because of response bias. The impaired and "control" groups were compared on the neuropsychological tests, and not surprisingly the two groups differed. Because of the way subjects were allocated to groups in these studies, the findings do not enable a conclusion to be drawn about the effect of decompression sickness on brain-behaviour relationships. The difficulty of interpretation is further increased by the fact that people seeking compensation for their diving accident were used as subjects, and are arguably different in terms of motivation, at least, from the population of divers who have had diving accidents.

Even where researchers go to some lengths to get a control group from a non-diving population, care still has to be taken in selection of the controls. One of the main reasons for this is that age, education and ability are highly correlated with test performance. Thus, it is important that the comparison group does not differ from divers on these variables, and if they do it is important to statistically correct for the influence of such variables. Some studies in the literature report control groups which differ from divers in some critical way, but either make no attempt, or use an inappropriate strategy, to address the problem. Vaernes and Eidsvik (1982), for instance, used three groups, a diving accident group, a diving non-accident group, and a "reference" or control group. Unfortunately, the control group consisted of data collected in the USA on a large number of subjects, from a different culture and language group. The authors argued that since their accident group was the same age (36 yrs) as the reference group" the observed difference between the accident and non-accident group is unrelated to age"(p. 803). Further, when looking at IQ scores, they comment that both groups are within the "average" range. However the non-accident group had a higher mean IQ (111) than the accident group (106), as well as being significantly younger (26 yrs) than the accident group (36 yrs). This line of reasoning is quite inadequate. Usually norms for neuropsychological tests are given for different age groups, especially when samples are large, as they were for the reference group, and it is not correct to cite average age for the reference group as if further comparisons were reasonable. This is especially so since other published normative data for the tests that Vaernes and Eidsvik (1982) used show that some of the tests are clearly affected by age and I (e.g. Fromm-Auch and Yeudall,1983; Klove, 1974 and Cauthen, 1978). These sorts of differences between groups should have been adjusted for by statistical procedures (e.g. analysis of covariance) where the effects fo age and IQ are partialled out of the results, and any remaining variation in test performance is investigated for the effects of diving accidents. Because of the existence of differences between groups, other than the occurrence or not of diving accidents, it is always possible that the observed differences were due to differences in age and/or IQ and not the variable of interest, diving accident.

Second, because a good neuropsychologial assessment incorporates a numbe of tests, the size of the groups needs to be quite large in order for rea differences to be picked up. This is so because each statistical analys carried out on the data takes into account the possibility of chance differences occurring for that test. The more statistical tests that are done, the more the researchers increase their chance of picking up differences between groups due to chance. If the sample size is reasonab large, this problem can be accounted for by tightening up the criteria f accepting that a difference is real (e.g. Bonferroni Adjstment, Hall and Bird, 1985). This strategy has only been used in part in one of the published studies (Andrews et al, 1986), even though all the studies the have control groups have carried out a large number of comparisons on the same data set.

The problems outlined above have bedevilled to a greater or lesser extent all the published work so far. These problems make it difficult to reach any conclusion about the effects of either diving accidents or dive stres on neuropsychological functioning in divers. Rather than review in detail all the findings of studies to date, I will briefly outline the findings of two recent studies, which although problematic do at least fulfil two important criteria. First, they both contain comparison groups of non-divers and second, they both employ neuropsychological tests which are sensitive to subtle effects of brain damage. These studies are also interesting in that their subjects were abalone divers, a group who do a large amount of diving and who often do not comply with safety dive protocols and adequate decompression procedures.

Divers in Andrews et al (1986) did significantly less well (after age corrections) than controls on only two of eight measures of various aspects of neuropsychological functioning. These were two tests from the Wechsler Memory Scale, logical memory and paired associate learning. The differences were small, however, and the divers scores were within norma limits. Correlations between tests and variables such as years of diving depth of diving, hours of diving, reports of near drowning or carbon monoxide poisoning and dive stress, revealed no relationship between diving variables and performance on the logical memory scale and paired associate learning. There were significant correlations between dive stress and the Symbol Digit Modalities Test (SDMT-written and oral), hours of diving and the visual reproduction test from the Wechsler Memory Scale, and between near drowning or carbonmonoxide poisoning on the oral part of the SDMT and the controlled word association test for animal words. The size of the correlations were not reported so it is not possible to comment on how large these associations were. Further, since approximately 40 correlations were calculated without appropriate statistical adjustment, it is doubtful whether any of these correlations would have achieved significance. They also investigated whether the two groups differed in terms of having members whose performance was abnormally low, and found no evidence for a subset of divers with abnormal scores. The authors quite rightly concluded that in their study there was no evidence for "the accumulation of subclinical insults leading to a dementing process".

Williamson and Clark (1986) report two studies, one looking at whether divers differ from controls in the performance on a variety of tests, and a second looking at the relationship between test performance and various diving indices. The latter study is particularly interesting in that it makes an attempt to separate out the effects of simple exposure to the underwater environment from risks and dangers associated with particular styles of diving. In the first study they found that the divers did as well or better than controls on some tests (reaction time, some memory and motor tasks) and worse than controls on visual, short term memory and some psychomotor skills. As in other studies, no attempt was made to control for the number of statistical tests carried out, so some of the reported significant findings may be spurious. They do, however, point out that the way in which divers choose to complete the tests differs from controls, in that they are more likely to take risks, substituting speed for accuracy, for instance. This sort of difference in motivation, needs to be taken into account when interpreting test results, and may lead to difficulty in detecting deterioration in neuropsychological functioning.

The second study by Williamson and Clark focussed on whether decrements in neuropsychological functioning are associated with any of a number of diving related variables. They looked at six diving variables: exposure (the number of hours dived in their working life); the maximum average depth; the incidence of general decompression sickness; the incidence of decompresson sickness with neurological symptoms; the degree of decompression stress (the risk associated with a typical dive); and an oxygen factor (related to whether or not oxygen is used to assist decompression). They found that the major influence on test performance was from variables related to diving technique or the quality of diving, rather than by the simple quantity of diving. However, only a small amount of variability in test performance is accounted for by the diving variables, so that the observed associations are quite weak. Apparently there are other factors that contribute to decrements in neuropsychological performance, that have not yet been elucidated.

Conclusion

The issue of whether decompression sickness, or various indices of dive stress are associated with decrements in neuropsychological functioning still remains unresolved. Given the many problems with the studies to date it is not possible to conclude one way or the other, whether diving accidents and/or chronic exposure to compressed air diving are related to decrements in neuropsychological performance. It seems likely that if there are effects on neuropsychological functioning due to exposure over time to risks taken when diving, they are probably not large (at least in the short term) and they probably only affect a subset of individuals. If this is the case, then the design of future research needs to accommodate the possibility that effect sizes and base rates will be small. The possibility of subtle effects could be clarified by undertaking a longitudinal study, in which divers and a matched group of controls are repeatedly tested over a number of years, perhaps at the same time as annual medical checks. Over this time information should be gathered about diving history, as well as other relevant information (e.g. alcohol consumption). If the question of interest is to detect early and subtle changes in functioning, then appropriately sensitive tests sampling different aspects of behaviour should be used. Since the amount of time available to carry out such an assessment will be limited for practical reasons, careful choice of tests is required. It would be wise to obtain a measure of general intellectual functioning, however, using an instrument like the WAIS is too time consuming. There are a number of short tests available which are correlated with WAIS performance and which could be used to estimate IQ (e.g. Mill Hill Vocabulary Scale).The Symbol Digit Modalities Test, a test of general cerebral efficiency, only takes 90 seconds to administer and is sensitive to brain dysfunction would also be worth including. Tests of verbal and non-verbal memory, perceptual functions, sensation, motor skills and executive functions should also be administered. Lezak (1983) provides a compendium of neuropsychological tests, from which the interested researcher can select tests that assess those areas of functioning of interest and which are most economical in use of time.

Large samples would need to be obtained since some individuals may be mor sensitive than others to the effects of decompression sickness and thus base rates of people with neuropsychological impairment may be low. Large samples will be needed in order to provide enough statistical power to:

a) detect low base rates of neuropsychological dysfunction and

b) statistically adjust for the number of comparisons that have to be mac between groups on different tests and over time.

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Chapter 20

MISCELLANEOUS MEDICAL DISORDERS

CARL EDMONDS

Abalone divers are susceptible to the same disorders as other divers, but because of the excessive time underwater they are likely to have a higher incidence of these. Because of the exceptional exposures, some diseases may only be seen in this type of diver.

The problems of otological disease, dysbaric osteonecrosis and possible long-term neuropsychological sequelae of compressed air diving, have been discussed earlier. In this chapter a pot pourri of other diseases are mentioned.

In doing the survey a number of divers made specific complaints, which were recorded. Although they did not fit into the standardised format, and although no assessment could be made as regards the incidence of these symptoms (because the other divers were not asked about them), it is considered that some of them were frequent enough to make them noteworthy, whereas others were of general interest.

Stories of abalone holding divers underwater until they drown, are thought to be fictitious.

MARINE ANIMAL INJURY

The cold water suitable for abalone growth is also the same as that which is infamous for the presence of the feared white pointer (Isuridae) shark.

One abalone diver was bitten in half during his diving near Bruny Island, Tasmania, by a white pointer. Another recieved fractured ribs when he was rammed by a shark. Many divers were said to have been bailed up by sharks, and sometimes by dolphins and seals.

In January,1974 an abalone diver was attacked and mauled off Streaky Bay, South Australia, by a shark that was presumed to be a white pointer.

While swimming near Neptune Island in South Australia, at Christmas 1983, a white pointer shark swam straight up to one diver, with its mouth wide open. He pushed his abalone bag into its mouth, lacerating his right hand in the process. The shark accepted the offer, and swam away with it. There was minor permanent scarring of the diver's hand.

Octopuses sometimes competed for the abalone catch, and many of the divers had coincidental encounters with sea urchins, eels, stingrays, scorpion fish and jellyfish.

ABALONE ALLERGIES

Abalone poisoning is classically due to the ingestion of the liver and perhaps other viscera - a rare activity in countries other than Japan. The photodynamic toxin seems to develop especially during the spring months. It causes a rapid onset of a histamine like skin disease, with burning, itching, redness, blister and wheal formation (urticaria) and swelling of the tissues, especially in the areas exposed to sunlight. Photosensitisation is otherwise rare as a result of foods of animal origin, although not uncommon with drug reactions. Ulceration can develop (Halstead, 1965).

The development of a skin allergy to the abalone can be disabling to both diver and sheller. It has resulted in a number of men leaving the industry, and the victim can be so sensitised that merely touching any part of his skin with the abalone meat, shell or blood would produce blisters within minutes. Some divers avoid touching the abalone with ungloved hands, cannot assist in the shelling or other preparation, will not remove their diving clothes until all abalone are packed and cannot touch any part of the animal.

Allergic reactions can become more serious with increased contact, and generalised or systemic symptoms are possible. Asthmatic reactions are sometimes severe and have required hospitalisation in abalone divers, especially if induced by the inhalation of the dust of abalone shells.

Unfortunately, consumption of the abalone steak can produce allergy reactions with a diffuse itchy red rash spreading over the body and developing blisters, wheals (urticaria) and generalised swelling. Two of the divers ironically were prevented from ingesting their catch because of this disease. This reaction is not restricted to the photosensitised areas as described above. Even the exposure to abalone cooking can precipitate the attack in sensitised divers. Review of the literature revealed similar observations by others (Horesh, 1943).

Other dermatological disorders are common, including minor abrasions and cold exposure in the marine environment, causing "supersensitive hands". Abrasion from the wet suit with excessive swimming (often on the ankles and backs of the knees) can be prevented with the use of pantihose and leotards. Urine rash over the lower body and fungal infections due to the water logged skin of the feet were noted.

Following a number of consecutive days abalone diving, the hands may show considerable desquamation ("flaking fingers"). This is reduced by the use of hand creams morning and evening during the diving activity.

MEDICAL DISEASE

Some divers would be considered by most medical authorities to be medically unfit for professional diving. For example;

CARDIOVASCULAR ABNORMALITIES

Case Report. This 48 year old diver had his last major myocardial infarction 18 months previously. Since then he has reduced his alcohol consumption and ceased cigarette smoking.

Case Report. Three years ago one diver developed angina pectoris, precipitated by exercise and eating a heavy meal, and sometimes occurring spontaneously. He was treated for his associated hypertension with hypotensive drugs, failed medically for his pilot's licence, and was using a large amount of Anginine.

The angina was more often precipitated by the struggle of getting into his wet suit than diving, which he considered effortless! However, he often did take a tablet just prior to "suiting up", and noted that the angina was more likely to develop if he swam hard at the start of the dive. If he gradually worked up to the maximum effort, it was not as bad. He had open heart surgery with a triple by-pass. He continued diving, although "taking it easy".

His current diving is up to 60 ft. for one to one and a half hours, and then less than 30 ft. for a variable time. He also continued to take Anginine, before diving. This was combined with Neosynephrine nasal spray, Sinutabs, etc., as he considered his major diving problem was difficulty in equalising his middle ear spaces and sinuses. In approximately half his dives he would develop epistaxis. Advice was given regarding the cessation of smoking, techniques for equalising his middle ears, and the use of a non-sympathomimetic nasal spray e.g. a topical corticosteroid , for his allergic nasal problems - and disposal of his abalone licence as soon as possible. There is evidence that he took action on all recommendations except the last.

RESPIRATORY DISEASES

Chronic respiratory disease was present in a suprising number of divers. Seven had asthma, with four regularly taking Ventolin and one Intal. Another had a past history of asthma. Bronchospasm was present on ascultation in two divers without a clear asthmatic history. Two had chronic bronchitis, one with cough and expectoration each morning and one with low pitched rhonchi and crepitations probably related to heavy marihuana use.

DRUGS

Probably about 20% use marihuana, half on a frequent basis.

Regular use of decongestants (mainly Sudafed and sympathomimetic nasal sprays was used by 8 divers; 1 had trouble with middle ear barotrauma of ascent (reversed ear) and 1 with tinnitus post dives.

Case Report. This diver often uses marihuana and observed that, for the first 10-20 feet, he felt an increase in "expansive" sensations, with perhaps decreased orientation. By 40 ft any beneficial effect was all gone. He therefore does not dive while " stoned", because it is a waste of money and gives no real pleasure.

Case Report. Diver used numerous non-prescription drugs including marihuana, cocaine and narcotics. His belief is that marihuana taken after a dive helps decrease the decompression sickness - "it slows the metabolism down".

Case Report. Worried whether his lower limbs, which are extremely cold for twelve hours or more after diving, could be due to the diving or to the Indian hemp that he smokes daily.

PSYCHIATRIC DISORDERS

There are possibly two cases of suicide known amongst the abalone divers, but specific details were not available and nor were they sought. Psychiatric disease is not well accepted by this group.

One episode of attempted suicide was ascertained, with the diver purposefully exceeding the tables. He blamed diving for his broken marriage.

One man suffered a very severe manic phase of a manic depressive psychosis, requiring both lithium and other drugs, but continuing his diving throughout. Another claimed to be chronically unhappy and depressed. One had a "nervous breakdown" lasting 12 months, especially associated with aggression.

ORTHOPAEDIC PROBLEMS

Recurrent low back pain ("Banana back") intermittently incapacitating the diver and related to diving activities was volunteered by four divers. Some claimed the carrying of a weight belt (needed to counteract the buoyancy effects of the wet suit) put a strain on the lumbar spine, and substituted a corset like weight belt or a weight vest, which distributed the weight over a greater area, supported the spine and kept it less mobile.

Symptoms of osteoarthritis unassociated with dysbaric osteonecrosis: 5.

PHYSIOLOGICAL OBSERVATIONS

Report. "The deeper you dive and the longer you dive, the more your vision changes after you get back to the surface. You notice that the reds and oranges will stand out and glare far more."

Episodes of the carotid sinus syndrome, with cervical discomfort, dizziness and fainting were noted by a few divers, but were remedied by cutting or modifying the tightness around the neck of the wet suit.

DISEASES DUE TO DIVING

FATALITIES

There were probably 10 deaths from abalone diving
Shark attack 2
Carbon monoxide toxicity 1 (possibly with other drugs contributing)
Drowning and entrapment 1
Pulmonary barotrauma 2
Pulmonary barotrauma and decompression sickness 1
Decompression sickness: 3

CARBON MONOXIDE TOXICITY

Carbon monoxide poisoning leading to unconsciousness in one diver was possibly the cause of 50% mistakes in graphaestheia testing (the only one to show such abnormality). In another it had repeatedly precipitated a migraine type headache.

Four other divers claimed to have had conventional symptoms of carbon monoxide toxicity, but not sufficient to cause loss of consciousness. One diver lost his brother who was also an abalone diver, in a diving fatality due to carbon monoxide poisoning.

BAROTRAUMA

Ear barotrauma. See chapter 10.

Gastrointestinal barotrauma

Four divers complained of repeated eructation of gas and vomiting during or after ascent (one diver vomited after diving every day!). Air swallowing was a possible cause of the collection of air into the stomach, but a more likely cause was the ear equalisation by swallowing when the diver was in the inverted position.

Pulmonary barotrauma

There was a past history of survival after pulmorary barotrauma in 4 of the divers in the survey, with pneumothorax in one, air embolus in one, mediastinal emphysema in one, and a combined pneumothorax and air embolism in one.

DECOMPRESSION SICKNESS SEQUELAE

Case Reports.

1.Very severe vertigo, headaches and balance problems for six months after decompression sickness.

2.Developed a severe bend in his right hip, five months previously and the pain remained severe ever since the bend. No investigation performed.

3.Recurrent pains in the left shoulder following decompression sickness. Bone scan positive.

4.Diver restricts diving to 30-35 ft, because deeper than this causes his "left side to go numb with pins and needles", whilst under the water. He thought it followed a spinal decompression sickness lesion.

5.Claimed he dived to 140 ft. for abalone, on a garden hose attached to the compressor! One post dive syndrome was diagnosed as the Guillian-Barre syndrome, and treated as such for many weeks before he gradually improved.

6.Spinal decompression sickness during abalone diving, superimposed on poliomyelitis as a child, resulted in extensive neurological disease with almost complete paraplegia, and severely painful muscular spasms for the subsequent eight years.

7.Lost vision post dive, with 2 distinct and almost identical episodes 2 and 6 years previously.

8.Grand mal epilepsy followed decompression sickness.

9.Severe delirium with decompression sickness resulted in a diver being refused treatment because of his "difficult personality".

10.Left leg paresis and impaired balance persisting after an episode of decompression sickness three years ago, but not interfering with diving.

POST DIVING SYNDROMES

Possible neuropsychological symptoms, of a temporary nature, were volunteered as common after diving. The importance of these were not appreciated during the design of the survey, and therefore the information collected was not in a specified format and there were no designated questions asked regarding this. Nevertheless many spontaneous complaints were offered by the divers and their wives e.g.

I have a high feeling and somewhat disorientated.
Conversation is lacking. He drives in a daze.
Appears to have a loss of memory also. I must have solitude.
Loses strength after diving. Sleeps like the dead.
He will get insomnia unless he forces himself to stay up and be active after the dive.
Sits and stares, does nothing (many times).
Looks at, but does not take in, the television.
We keep an old car that he can bash into with an axe and vent his anger .
After diving I suffer a very bad temper. I am highly irritable and fly off the handle almost irrationally over the slightest incident.

Both the wives and the fishing authorities agree that the diver should not be involved in any decisions or discussions on the afternoon of the dive. Even when the topic is agreeable, it is likely that the diver will be either unhelpful or disinterested.

Salt water aspiration syndrome, also known amongst the divers as "night fever", was very common, with symptoms related to the breathing a fine spray of sea water during the dive. This was attributed to the low line pressure used with Hookah compressors and the unusual configuration of the exhaust value in the popular See-Bee regulator.

The main complaints were sweating profusely on the night of the dive, the "body like a radiator", etc.

Case Report. Diver uses a See-Bee regulator and even at 10 ft. if he dives for a few hours, then half to one hour after the dive he develops an aching "in every bone and joint in my body", together with anorexia and temperature up to 104 degrees Fahrenheit. It lasts about four hours.

The salt water aspiration syndrome was often remedied by repairing the regulator or the substitution of a new regulator.

Shoulder crunching.

There were four divers who complained of noisy shoulder movement after long durations at shallow depths. It was conjectured that this symptom, which was confirmed and complained about by one spouse), could have been due to release of gas into the slow tissues around joint spaces and be analagous to aviators who ascend after being saturated at ground level, and develop gas in the articular joint fluid.

Case Report. After extended dives, i.e. 4 - 6 hours or more at all depths (his maximum is 45 ft), the diver notices "a squelchy, squelchy shoulder" usually for up to 2 hours and with the right being greater than the left. It is produced when the arms are being lifted up, e.g. while taking off his jumper. during the dive, he certainly uses his right arm more, prising off the abalone. He has never noted this symptom with any other type of manual work, and it has never been painfull.

Temporo-mandibular Joint Dysfunction.(Edmonds et al,1982;Knight 1985).11 cases Known as "jaw seizure" by the divers, it is well described by the following:

Case Report. The jaw seizes and the mouth wont open, you can not get the regulator out or in. If the regulator is removed to fill the parachute, you cannot open the mouth to replace it. This is disconcerting.

Case Report. After using borrowed equipment, I have to force my mouth open, lever an apple in and make myself chew it until function is restored. I consume a lot of apples.

Case Report. After two or three days work, I can't chew for some hours after the dive. I smoke dope after the dive, and this seems to help

UNCONSCIOUSNESS WHILE DIVING

Two episodes of unconsciousness with free diving, during spear fishing championships. One episode resulted in cardiac arrest for a period of about three minutes.

UNUSUAL MEDICAL PROBLEMS

Dives with a prosthetic (blown glass) eye. No problems were noted.

Notices a hoarse voice for 2-3 days after each dive.

Has a club foot; tends to get bends involving the left shoulder and his club foot. Uses a small flipper on his small foot.

Large hiatus hernia, extending into the thoracic cavity was detected 12 months previously and caused no trouble with his diving and was not associated with gastrointestinal barotrauma.

Swallows a lot of sea water associated with abdominal discomfort and vomiting post dive.

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Chapter 21

RECOMMENDATIONS - Summary

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GENERAL RECOMMENDATIONS

1. Periodic seminars between the divers and practically orientated experts be made available (not obligatory). This is already undertaken in some areas, and they have been well attended. Health and diving safety subjects could be integrated with equipment and fisheries subjects, so that the overall attractiveness would be heightened.

Non medical subjects could include: Compressor technology, maintainance and testing; diving equipment; abalone recruitment; marketing; etc. The lecturers in some of these subjects would be funded by their commercial firms or government departments.

The small number of lecturers could be organised to visit 6-7 areas consecutively, in a two week period and cover all abalone diving groups. With adequate warning, diving could be suspended for 2 days to allow for these seminars and other local meetings. Such an arrangement would be efficient as regards lecturer participation, as preparation of one lecture would allow 6-7 presentations. Questions from one area could then contribute to presentations in subsequent areas.

2. Periodic literature in the form of an annual or semi-annual newsletter covering practical subjects in non-technical terminology, and with contributions by experts in a variety of subjects related to abalone harvesting, should be available to all licenced divers.

The subjects covered should include the same as those referred to in the above seminars, and could even be transcripts of them. It could also be used to inform divers of such meetings. The literature should be sent direct to the diver, to ensure that local administrative difficulties or political animosities do not intrude.

3. Specific practices which are inherently dangerous, such as the inappropriate use of oxygen underwater, could be discouraged by implementation of the above recommendations.

4. A Central Registry which is able to compile and analyse medical records of divers, would allow a longitudinal study of health problems with this form of professional diving. This would also be of great value to the individual diver as it would allow for early baseline medical data to be compared with subsequent assessments. It would also ensure a standard of investigation and reporting that is quality controlled.

Individual medical information would only be available to the diver or a representative designated by him (e.g. local doctor).

This Central Registry would be comparable to U.K. Decompression Sickness Panel and the Japanese group from the Saitam Medical School. In Australia, it would have to be an independent organisation, unassociated with state or commonwealth government departments. Otherwise it will be interpreted with suspicion by the very divers it is intended to help.

DYSBARIC OSTEONECROSIS.

Annual or bi-annual long bone X-rays should be encouraged because of the alarming incidence of severe and disabling lesions in this group.

The X-rays should be arranged only at selected hospitals, where strict adherence to the MRC Decompression Sickness Panel guidelines is maintained and where technical skill and experience is available. Specifically attention must be paid to correct shielding of the gonads, and viewing an adequate length of femur.

The films should be interpreted by selected radiologists who adhere to the MRC reporting system and who have expertise in this disorder. The films must then be held by an independent body which acts as a registry where films are interpreted a second time, dispatched if required for further assessment, and then retained until the diver leaves the industry, when they should be returned to him.

RECOMMENDATIONS REGARDING OTORHINOLOGICAL PROBLEMS.

The major recommendation is that the professional diver should be examined annually by a clinician knowledgeable in diving medicine. The reason for this recommendation is that many of the divers are not aware of the safe and more efficient ways of equalising their middle ear cavities during descent, and are sometimes relying on decongestants which may then produce other pathology, including the predilection to middle ear barotrauma of ascent.

Particular attention should be paid to the otological problems during the annual diving medical examination, and this should be supplemented by a naso-otological assessment, by an otological specialist when indicated.

It is recommended that professional divers undertake a pure tone audiometry, encompassing at least the range 500 to 8000 Hz. In many cases, the only damage is in one or more of the 4000, 6000 or 8000 Hz. range, with the greatest problem at 6000 Hz.

With the preventative measures of adequate instruction in middle ear autoinflation, the initial base line pure tone audiogram and the annual audiometric assessment to ensure no progression of hearing loss, it should be possible to reduce or even completely prevent a great deal of the otological and paranasal pathology experienced by this occupational group.

RECOMMENDATIONS ON NEUROPSYCHOLOGICAL FUNCTIONING

Short term neuropsychological effects

An investigation may be indicated into the short term effects of extensive diving, as performed by abalone divers. This could be achieved by a small group of investigators travelling with the divers during their occupational activity, or alternatively meeting them on the wharf immediately following their return, and before they have regained their normal functioning.

The investigations would have to include some form of psychometric testing (including short tests such as the Wechsler Memory Scale) and other self-assessment questionnaires, together with any electrophysiological data that may be available (e.g. electroencephalography), deep body temperature and full biochemical and haematological screening.

Decompression Sickness and Oxygen Use

It would seem prudent to avoid dangerous decompression profiles as much as possible, making allowance for the occupational exigencies. It is of note that a great number of the abalone divers were not aware of their failure to follow established principles of decompression. With this in mind, regular seminars held for the divers, which they could attend on a voluntary basis, could only be of value.

The attempt by the divers to reduce decompression sickness by the use of oxygen may have conflicting effects. Although the oxygen certainly reduces the incidence of intravascular bubbles, and possibly also bubbles very close to the vascular system (fast tissues), its use under hyperbaric conditions i.e. underwater, can produce a whole series of other neuropathological damage, e.g. to the special senses and the higher functions of the brain. The danger of oxygen as a neurotoxic substance, if used under pressure, has not been fully appreciated by many of the divers. This would also be the subject of any educational campaign which organised seminars for the divers, by experts in diving medicine.

As a general rule, even if there are no symptoms of decompression sickness present, it may be prudent for the diver to use oxygen on the surface to reduce decompression problems - assuming that the normal precautions against fire have been taken. It is not proposed as a method of extending underwater endurance and it is not recommended for routine use underwater.

The obvious way of diminishing the influence of decompression sickness in producing neuropsychological sequelae is by prompt and effective treatment of the disease.

Gas Contaminants

It is recommended that educational lectures be given to the divers, by experts in compressor technology. Numerous questions asked of the medical experts revealed the need for this information.

In each diving area, there should be available to the divers, a method of measuring the presence of carbon monoxide and other contaminants (hydrocarbons), that may be present in the gas supplied by the compressor. The investigation only takes a few minutes and should be performed at least every month, and whenever there is any reason to suspect compressor abnormality.

Such detection kits are readilly available in Australia and include the Drager Multi Gas Detector unit, with carbon monoxide and hydrocarbon tubes, the AUER gas analysers available from M.S.A., and the Bendix Gastic Gas Detection Systems in many other countries. They have similar capabilities. These simple analysis systems, which can be utilised by any diver or boatman, can inform the diver that his compressor is not function adequately.

Dehydration

It would seem reasonable to advise, even without waiting for further investigations, that adequate fluids be taken both before the dive and during the diving day. Fluids such as coffee, which produce a diuresis, would not be as adequate as fruit juices.

Research

A longitudinal survey to determine possible neuropsychological damage could be combined with the above medical and seminar program to further determine the degree and type of damage possible from excessive diving. A combined neuropsychological and electrophysiological study with the inclusion of the more recent neurological investigations such as CT scans, NMI and PET may ultimately be required.