LABYRINTHINE DYSFUNCTION DURING DIVING

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Undersea Medical Society

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**Abstract:**
This report presents the proceedings of the First Undersea Medical Society Workshop on "Labyrinthine Dysfunction During Diving," held at Duke University Medical Center on 1 and 2 of February 1973. There were presentations and discussions dealing with all aspects of the subject problem.
The following areas were stressed:
- Anatomy and function of the Vestibular end organ.
- Clinical evaluations of the vestibular dysfunction.
- Symptoms, signs and pathology of vestibular endorgan disease.
- General history in vestibular disorders in diving.
- Vestibular and auditory problems during - compression and decompression.
- Vestibular disorders during stable deep depths.
- Labyrinthine disorders in divers.
- Prevention and management of vestibular orders in diving.
LABYRINTHINE DYSFUNCTION

Joseph C. Farmer, Jr., M.D.

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Program

Labyrinthine Dysfunction During Diving

Workshop sponsored by the Undersea Medical Society, Inc.

February 1-2, 1973 - Duke University Medical Center, Durham, North Carolina

Chairman: Joseph C. Farmer, Jr., M.D., Assistant Professor, Division of Otolaryngology, Department of Surgery and F. G. Hall Laboratory for Environmental Research, Duke University Medical Center

February 1st

8:15 a.m. Opening Remarks - Dr. David H. Elliott, President, Undersea Medical Society

8:20 a.m. Welcome - Dr. William R. Hudson, Professor and Chief, Division of Otolaryngology, Duke University Medical Center

8:25 a.m. Introduction - Dr. Farmer

8:30 a.m. Functional Anatomy of the Vestibular Endorgan - Dr. H.O. Barber, Professor of Otolaryngology, University of Toronto

8:50 a.m. Functional Anatomy of the Central Vestibular System - Dr. Brian McCabe, Professor and Chairman, Department of Otolaryngology, University of Iowa

9:10 a.m. Discussion and Questions

9:20 a.m. Break

9:30 a.m. Clinical Evaluation of Vestibular Dysfunction - Dr. McCabe

9:50 a.m. Electronystagmography - Principles and Clinical Uses - Dr. Barber

10:20 a.m. Discussion and Questions

10:30 a.m. Break

10:40 a.m. Symptoms, Signs and Pathology of Vestibular Endorgan Diseases - Dr. Barber
February 1st

11: a.m.  Symptoms, Signs and Pathology of Central Vestibular Diseases - Dr. McCabe

11:20 a.m.  Discussion and Questions

11:30 a.m.  Break

11:40 a.m.  Current Concepts of CNS Dysfunction in Diving -
Dr. Peter Bennett, Professor of Anesthesiology and
F. G. Hall Environmental Laboratory, Duke University
Medical Center

12:00 noon  Discussion and Questions

12:20 p.m.  Lunch

1:40 p.m.  General History of Vestibular Disorders in Diving -
Dr. Robert S. Kennedy, LCDR, MSC, USN - Head, Human
Factors Engineering Branch, Naval Missile Center,
Point Mugu, California

2:00 p.m.  Vestibular and Auditory Problems During Compression -
Dr. Carl Edmonds, MRCP, DPW, School of Underwater
Medicine, Royal Australian Navy

2:30 p.m.  Discussion and Questions

2:50 p.m.  Vestibular Disorders During Stable Deep Depths -
Dr. W. K. H. Sundmaker, University of Pennsylvania, and
Dr. C. J. Lambertsen, Director, Institute of Environmental
Medicine, University of Pennsylvania

3:20 p.m.  Discussion and Questions

3:40 p.m.  Investigations of Vestibular Interactions in the High Pressure
Neurological Syndrome - Dr. James G. McCormick, Research
Assistant Professor of Otolaryngology and Director of
Otolaryngology Laboratories, Department of Surgery, Bowman Gray
School of Medicine, Winston-Salem, N.C., and Dr. Ralph W.
Brauer, Director, Wrightsville Marine Bio-Medical Laboratory,
Wilmington, N.C.

4:10 p.m.  Discussion and Questions

4:30 p.m.  End of 1st day. Visitors can tour the F. G. Hall Laboratory
for Environmental Research
February 2nd

8:30 a.m. Vestibular and Auditory Problems During Decompression - Dr. Farmer
8:50 a.m. Discussion and Questions
9:00 a.m. Vestibular Symptoms Associated with Deep DIVING - Dr. Barber
9:20 a.m. Discussion and Questions
9:30 a.m. Vestibular Dysfunction in Commercial DIVING - Dr. Theodore D. Langley, Ocean Systems, Inc., Union Carbide Technical Center, Tarrytown, New York
9:50 a.m. Discussion and Questions
10:00 a.m. Break
10:10 a.m. Labyrinthine Disorders in Divers: Some Experiences and Activities with the Royal Navy - Dr. P. R. A. Coles, Institute of Sound and Vibration Research, University of Southampton and Consultant to the Institute of Naval Medicine, Alverstoke, United Kingdom
10:50 a.m. Discussion and Questions
11:00 a.m. Panel: Prevention and Management of Vestibular Disorders in DIVING - Drs. Coles, Barber, Edmonds, and McCabe, plus Dr. William R. Braithwaite, Assistant Medical Research Officer, U. S. Navy Experimental DIVING Unit, and Dr. William H. Spaur, Commander, MC, USN, Senior Medical Officer, Experimental DIVING Unit. Chaired by Dr. Farmer.
12:00 noon Discussion and Questions by Attendees
12:20 p.m. Lunch
1:30 p.m. Panel: Needed Research Concerning Vestibular Problems in DIVING - Drs. Barber, McCabe, McCormick, Coles, and Edmonds. Chaired by Dr. Farmer.
2:30 p.m. Questions and Discussion by Attendees
2:50 p.m. Final Recommendations. Panel: Drs. Barber, Bennett, McCormick, McCabe, Coles, Edmonds, Spaur, Braithwaite, and Farmer
3:50 p.m. Questions and Discussion by Attendees
4:00 p.m. Adjourn
REPORT TO THE UNDERSEA MEDICAL SOCIETY ON WORKSHOP HELD AT DUKE UNIVERSITY MEDICAL CENTER FEBRUARY 1 & 2, 1973, ENTITLED: LABYRINTHINE DYSCONJUNCTION DURING DIVING

Date of Report: April 10, 1974

First Day - Morning: (*)

The first morning discussion was concerned with the functional anatomy and physiology of the vestibular system led by two recognized experts in these fields, Dr. H. O. Barber, Professor of Otolaryngology, University of Toronto, and Dr. Brian McCabe, Professor and Chairman, Department of Otolaryngology, University of Iowa. The morning session was ended with the presentation by Dr. Peter Bennett on the Current Concepts of CNS Dysfunction in Diving.


Dr. Bennett's presentation concerned the general concepts of central nervous system physiology in diving. He covered the previous developments in this field and discussed in detail the current concepts of physiology of the high pressure nervous syndrome. This is summarized in a later publication entitled "The High Pressure Nervous Syndrome During Human Deep Saturation and Excursion Diving," by A. Bachrach and P. B. Bennett; Forsvarsmedicin: Volume IX, No. 3, July, 1973, pages 490-495.

(*) Follow program outline as presented earlier.
First Day - Afternoon:

Presentation by Dr. Robert S. Kennedy concerning the General History of Vestibular Disorders in Diving. Dr. Kennedy's comments are covered completely in his N.M.R. I. Research Report number N-406.03.5000-BAK-9, August 10, 1972, report number 1, entitled "A Bibliography of the Role of Vestibular Apparatus Underwater and Pressure: Content Oriented and Annotated." A later N.M.R.I. research report of this same number, report number 3, dated March, 1973, specifically covers the material which Dr. Kennedy covered in his presentation at the workshop. This presentation briefly reviewed the literature relating to vestibular function in diving and introduced the above bibliography. It was the author's opinion that the role of the vestibular system in compressed air work was currently underestimated. This presentation, plus the broader bibliography, called attention to the incidence of vestibular involvement in diving and provided a reference to the background material felt essential for understanding future studies of vestibular problems in diving.

The next afternoon presentation was by Dr. Carl Edmonds concerning Vestibular and Auditory Problems during Compression. This is summarized in the book: Otological Aspects of Diving by Edmonds, Freeman, Thomas, Tonkin, and Blackwood. Australian Medical Publishing Co., Lt., New South Wales, 1973. Dr. Edmonds discussed the possible mechanisms of vestibular injury during the compression phase of shallow, no-decompression, dives and discussed means of prevention. His presentation is also summarized in the article "Vestibular Injury During Diving," by J. C. Farmer, Jr., and W. D. Thomas, Forsvarsmedicin, Volume IX, number 3, July, 1973, pages 396-403.

The next presentation, Vestibular Disorders during Stable, Deep Depths, was given by Drs. W. K. H. Sundmaker and C. J. Lambertsen. This work has not
yet been published. It concerned instances of unilateral losses of vestibular function while at stable deep depths, but shortly after the onset of breathing a mixture which contained a second inert gas. Dr. Joseph Idicula was present during the discussion and postulated the etiology of these injuries as the production of gas bubbles at membrane interfaces resulting from the counter-diffusion of two inert gases between adjacent fluid filled spaces. A reference to this topic is: Graves, D. J., Idicula, J., Lambertsen, C.J., and Quinn, J. A.: "Bubble Formation Resulting from Counter-Diffusion Super Saturation: A Possible Explanation for Isobaric Inert Gas "Urticaria and Vertigo": Phys. Med. Biol., 18, No. 2, 256-264, 1973.

The next presentation was by Dr. James C. McCormick and Dr. Ralph W. Brauer concerning the Production of Cochlear and Vestibular Injuries in Laboratory Animals with Deep Helium-Oxygen Exposures. Noteworthy is that Dr. McCormick's research suggested the possible protective effect of heparin administration. This work is summarized in: McCormick, J. G., Philbrick, T., Holland, W. and Harrill, J. A.: "Diving Induced Sensori-Neural Deafness: Prophylactic Use of Heparin and Preliminary Histopathology Results." Laryngoscope, 83, 1483-1501, 1973.

Second Day - Morning:

The program was opened by a presentation entitled Vestibular and Auditory Problems During Decompression by Dr. Joseph C. Farmer. This presentation is summarized in the enclosed copy of the above referred to article by Farmer and Thomas. After questions and discussions, Dr. Barber gave a brief review of his experience with patients suffering vestibular symptoms after decompression from deep helium-oxygen dives. These cases were very similar to the cases discussed by Dr. Farmer. Dr. Ted Langley then discussed the experience of Ocean Systems, Inc.
with vestibular dysfunction in diving. His cases were also very similar to the cases presented by Dr. Farmer. Dr. P. R. A. Coles then discussed the British Royal Naval Experience with Labyrinthine Disorders in Diving. His discussion is summarized by his later publication entitled "Labyrinthine Disorders in British Navy Diving: Forsvarmedicin, Vol. IX, No. 3, July 1973, pages 428-433.

The next item on the program consisted of a panel discussion of The Prevention and Management of Vestibular Disorders in Diving by Drs. Coles, Barber, Edmonds, McCabe plus Dr. William R. Braithwaite and Dr. William H. Spaur from the U. S. Navy Experimental Diving Unit. It was Dr. Spaur’s opinion that the selection of appropriate diving tables was the most crucial item needed to prevent vestibular disorders in diving. Dr. Spaur then discussed the Experimental Diving Unit’s efforts to develop an appropriate table to prevent this problem.

As a result of these efforts, he indicated that the U. S. Navy was trying to avoid very rapid ascents for the first 150 to 100 feet during decompressions from exposures to depths deeper than 300 to 400 feet. For deeper dives, classical saturation decompression schedules and diving systems were being used. He felt that if operational needs called for decompressions with shorter times than the classical saturation decompression schedules, means should be available to promptly recompress a diver who might develop vestibular or auditory problems during decompression. Recompression capabilities should include the means to recompress back to the depth of the dive and to provide appropriate life support systems for a subsequent saturation decompression after the initial recompression treatment.

Dr. Braithwaite felt that the biggest part of prevention was detection. The major effort needed in detection was the education of physicians and divers
in the diving community of the problem of vestibular and auditory injury during decompression and the proper utilization of electronystagmography and audiometry. Dr. Coles felt that ENG monitoring was definitely needed. In addition to earlier detection of vestibular injury during decompression, it would provide possibly earlier detection of the disoriented diver in underwater conditions.

A major recommendation of all the panel members was that adequate baseline vestibular and auditory pre-dive testing should be done on all divers. Basic vestibular tests should consist of testing for spontaneous nystagmus in two or three positions and bilateral warm caloric testing. It was recommended that the Undersea Medical Society might consult the Subcommittee on Equilibrium of the American Academy of Ophthalmology and Otalaryngology as regards the development of pre-dive vestibular evaluations.

Next the panel was asked to discuss the management and treatment of vestibular disorders during diving. It was generally agreed that no drugs were known to be useful in reversing endorgan or central vestibular injuries occurring during decompression. The only useful pharmacological agents which were known to suppress the observed nystagmus of vestibular dysfunction in non-diving situations were Valium, given as a stat dose of 15 mgs., or Innovar made up of Droperidol and Fentanyl. These agents give only symptomatic relief and should be administered only with careful monitoring of vital signs because of CNS depressive effects. It was felt that the more commonly used antimotion sickness drugs, frequently antihistamine derivatives, were not of value in treating significant vestibular dysfunction. The possible use of heparin, as the experiments of Dr. McCormick suggested, was discussed. It was agreed that use of this drug for apparent vestibular decompression sickness should not be advocated at this time until
further investigations had more specifically elucidated the mechanisms of decompression sickness in the labyrinth.

As to the needed depth of treatment for recompression of vestibular decompression sickness it was agreed, after much discussion, that diving facilities should be able to recompress a diver injured in this manner back to the deepest depth of the dive. The need to raise the partial pressure of oxygen in the treatment breathing mixture was suggested. The question was raised as to whether oxygen might be involved in injury from counter-current diffusion. Dr. Idicula indicated that his experiments had discounted the role of oxygen in this mechanism of injury. It was advised that the recompression treatment depth should be the depth of relief by ENG monitoring even if one had to return to the bottom depth. All panelists agreed that medical therapy such as heparin, dextran, steroids, or "vasodilators" were contraindicated and that our current state of knowledge did not justify the use of these agents for vestibular decompression sickness.

Dr. Barber led a discussion of the needs for proper electronystagmographic evaluation. This involved complete understanding of the characteristics of amplifying equipment plus the use of a darkened room with a standard mental alerting technique. He suggested several mathematical tests. He also advised examinations for spontaneous nystagmus with eyes closed in the dark plus examinations in the erect, supine, right lateral, and left lateral head positions with the avoidance of twisting of the neck. He also suggested the use of a rotating drum for investigating of optokinetic nystagmus. For caloric testing, he advised the use of bilateral 44° irrigations if time did not permit the classical bithermal irrigations.
Second Day - Afternoon:

The panel discussed the question of Needed Research in the Field of Vestibular Problems in Diving. It was felt that in addition to the above screening studies of the diving population, studies of blood coagulation and lipid chemistry should be done. Also, there should be a greater dissemination of knowledge in this field among the diving community.

The final session consisted of a panel who made final recommendations concerning the problem of vestibular injury during diving. These are summarized as follows:

I. Incidence:

The panel in general felt that the incidence of labyrinthine dysfunction during decompression is higher than previously indicated. The reasons for this are felt to be related to an increased awareness of this problem with deeper dives over recent years and a possible increase in susceptibility of the inner ear to injury during decompression from deep helium-oxygen dives. The summary of the literature by Dr. Kennedy suggested that decompression accidents occur in about 10% of all dives and vestibular accidents comprise about 5% of these.

It was felt by Dr. Carl Edmonds and others that the frequency of difficulty with middle ear pressure equilibration during compression was high; however, the incidence of injury to the inner ear during compression and shallow dives requiring no decompression was probably low.

II. Prevention:

Several steps were recommended for prevention of injury to the vestibular organs during decompression. All members of the panel agreed that careful monitoring and screening of the diving population with particular attention to standardized vestibular tests done by qualified investigators was essential, not only to future research, but to possible prevention. All panel members felt that improved
decompression profiles were very important. Dr. Spaur particularly noted that in the experience of the Experimental Diving Unit, inner ear injuries did not occur during the U. S. Navy saturation decompression profiles. It was agreed that more careful instructions should be given to all divers so that they might be more aware of vestibular and/or cochlear symptoms at any time during diving and more prompt reporting of these difficulties would occur.

It was agreed that forceful Valsalva maneuvers in attempts to equilibrate middle ear pressure during compression were potentially harmful. Divers should be trained to use other maneuvers to clear the ears such as the Toynbee or the Frenzel maneuvers. Dr. McCormick, with the concurrence of Drs. Barber and McCabe, felt that in those situations where there was an urgent need for compression of an individual who was having difficulty with middle ear pressure equilibration on the surface, 8 mgs. of Medrol, administered orally would be helpful. The use of topical vasoconstrictor agents in the nose in these situations was also suggested. It was Dr. Edmonds' opinion, with the concurrence of Dr. Farmer and others, that dives should be aborted at the first signs of difficulty with middle ear pressure equilibration during descent. It was also suggested by Dr. Peter Bennett that improved compression rate standards should be developed. Until further information is obtained regarding the biological effects of inert gas switching at stable deep depths, Dr. Bennett advised that future changes in inert gases during decompression should be characterized by smaller increases in the introduced inert gas partial pressure over a longer period of time. Dr. Braithwaite also advised the slowing of decompression rates during such switches.

III. Needed Investigations:

The first recommendation for needed investigations was more careful monitor-
ing and screening of the diving population with the performance of standardized pre-dive otological testing by qualified investigators. Also, greater dissemination of knowledge of otologic injury, both vestibular and cochlear, in diving was needed. The use of the planned journal sponsored by the Undersea Medical Society for this purpose was recommended. On future experimental dives for investigating various dive profiles, monitoring of cochlear and vestibular functions should be done and any dysfunction noted well documented.

A major effort in investigating vestibular function in animals during various and multiple diving conditions was recommended. In addition to function monitoring during various dive profiles and gas mixtures, the investigation of blood coagulation and lipid chemistries during these conditions should be undertaken. Also, more extensive histological studies of the inner ears of animals during various diving conditions should be done.

IV. Treatment:

The panel agreed that treatment would depend upon the stage of diving in which injury apparently occurred since the mechanism of injury was probably different depending upon whether the injury occurred during compression at stable deep depths, or decompression. For injuries during compression and shallow, no decompression dives, Drs. Edmonds, Barber and McCabe felt that a 12 to 24 hour period of bed rest followed by exploratory tympanotomy for cases which were not significantly improving or becoming worse. For cases which occurred during decompression, it was generally agreed that immediate recompression should be performed with the first signs of vestibular and/or cochlear dysfunction. The recompression should be followed with electronystagmography and should proceed up to the depth of the dive. Dr. Spaur felt that there was no precise rules known regarding the
optimum recompression profile. He advised using a standard treatment Table V. Dr. Bennett felt that the recompression should proceed at least to the depth of relief. It was felt that there was better criteria for recompression of apparent vestibular decompression sickness was needed.

For treatment of those cases occurring during deep, stable depths, shortly after inert gas switching, it was felt that not enough evidence was at hand as to the mechanism of these injuries in order to precisely define treatment. Until newer knowledge was gained, it was agreed that the best treatment was avoiding such inert gas switching at deep depths and particularly during inert gas switching at decompression, the switch should be made with smaller delta P of inert gases over a longer period of time. If vestibular symptoms occurred during these instances, immediate recompression should be undertaken. If vestibular symptoms occur shortly after inert gas switching at stable deep depths, the switch to the new inert gas should be immediately discontinued and the divers placed on a breathing mixture containing high partial pressure of oxygen with the same inert gas which was present prior to the switch.

As for other type of therapy such as drugs, it was agreed that no specific drugs were known which might specifically treat cases of vestibular decompression sickness. Drs. Barber and McCabe advised the use of valium for symptomatic treatment. For cases of vestibular dysfunction within the compression phase of shallow, no decompression dives, it was advised that the best treatment was prevention of significant pressure differentials across the tympanic membrane and round and oval windows and the use of Medrol and nasal vasoconstrictors in those cases which there was an operational need for the dive to proceed in individuals who could not clear their ears before the dive started.
It was also felt that in view of the subsequent physiology and course of symptoms after a permanent vestibular injury as discussed by Dr. McCabe, adequate evaluation after such an injury for future diving suitability required evaluation by a specialist in the vestibular disorders, including the use of appropriate electronystagmography. It was agreed that individuals who were demonstrated to have suffered permanent injury to one of the vestibular end organs should not be exposed to future underwater or hyperbaric conditions.