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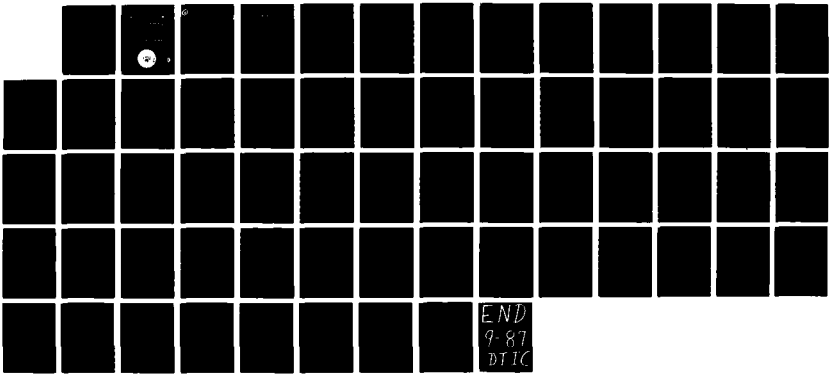

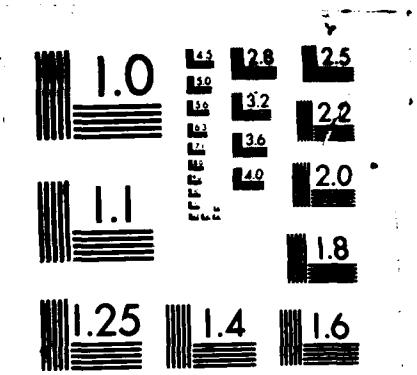

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NAVY EXPERIMENTAL DIVING UNIT
REPORT NO. 6-87

A REVIEW OF PHYSIOLOGICAL AND PERFORMANCE LIMITS IN SATURATION DIVING: 1968 - 1983

By

Wolfram H. Enseleit, ENS, MC, USNR

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DEPARTMENT OF THE NAVY
NAVY EXPERIMENTAL DIVING UNIT
PANAMA CITY, FLORIDA 32407-5001

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A review of published literature on the topic of physiological and performance limitations in saturation diving was conducted. Emphasis was directed to the deep diving experience of the United States, France, Great Britain and Norway between 1968 and 1983. Changes at depth in the following systems were reviewed: respiratory, cardiovascular, renal, neurological, neuropsychological, and musculoskeletal, among others. The findings of this review indicated that impairments in neurological (HPNS) and neuropsychological functioning are at present the most significant determinants in prescribing the limits to which man may descend in hyperbaric environments.

ABSTRACT

A review of published literature on the topic of physiological and performance limitations in saturation diving was conducted. Emphasis was directed to the deep diving experience of the United States, France, Great Britain and Norway between 1968 and 1983. Changes at depth in the following systems were reviewed: respiratory, cardiovascular, renal, neurological, neuropsychological, and musculoskeletal, among others. The findings of this review indicated that impairments in neurological (HPNS) and neuropsychological functioning are at present the most significant determinants in prescribing the limits to which man may descend in hyperbaric environments.

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PHYSIOLOGICAL AND PERFORMANCE LIMITS IN SATURATION DIVING

GLOSSARY

AMTE (PL)	Admiralty Marine Technology Establishment (Physiological Laboratory)
ATA	atmospheres absolute
B	bar
°C	degrees Centigrade
CO_2	carbon dioxide
d	day
EEG	electroencephalogram
EKG	electrocardiogram
FSW	feet of sea water
ft	foot
g	gram
gm/L	grams per liter; density calculated using body temperature of 37°C, pressure of 760 mmHg plus gauge pressure, and $pp0_2$ of .21 ATA.
h	hour
He	helium
HPNS	High Pressure Nervous Syndrome
Hz	Hertz
K	potassium
m	meter
MSW	meters of sea water
N_2	nitrogen
Ne	neon
NUTEC	Norwegian Underwater Technology Center
O_2	oxygen
REM	rapid eye movement
RNPL	Royal Navy Physiological Laboratory
T4	Thyroxine
UBA	underwater breathing apparatus
W	watt

A REVIEW OF PHYSIOLOGICAL AND PERFORMANCE LIMITS IN SATURATION DIVING: 1968 - 1983

I. PURPOSE.

Current needs and interests in extending military and civilian diving operations to greater depths require the identification of physiological and performance limits for saturation divers, if and where they exist. Concern for the effectiveness of the divers and their safety at or beyond such limits is paramount.

The U.S. Navy has identified the need to determine the physiological limits for saturation dives. Identification of limiting physiological factors may then be correlated with such variables as:

a. Maximum depth of the dive

b. Maximum duration of the dive (seal to seal)

c. Maximum duration of excursion(s)

d. Maximum time at depth

e. Maximum sustained number and frequency of excursions per diver per saturation

This information is required to modify or extend existing operational procedures and thereby optimize diver efficiency, effectiveness and safety.

The purpose of this paper is to review the literature of deep diving (>300 MSW, 1000 FSW or 31 ATA) to identify physiological limits of saturation divers--if they exist, how they affect the diver and how they can be modified. Recommendations are submitted in light of this information.

II. INTRODUCTION

A. Saturation Diving Requirements

Current U.S. Navy requirements for underwater activity require such missions as ship repair and maintenance, ship salvage, retrieval of equipment, searches and surveys, special warfare operations, explosive ordnance disposal and submarine rescue. Although 99% of Navy dives occur shallower than 200 FSW (Vorosmarti, 1980; Berghage, 1980), operational procedures exist for dives to 1,000 FSW, with greater depths attainable with one atmosphere (1 ATA) systems (Vorosmarti, 1980).

Commercial deep diving requirements reside predominantly in the off-shore oil and gas industry (Elliott, 1980). In 1980, the average depth of commercial saturation dives in the North Sea was 500-600 FSW with much deeper open sea dives possible (e.g. 1574 FSW by the French; Berghage, 1980). The impetus for extending existing diving limits was presented in earlier workshops (Halsey et al, 1975; Daniels et al, 1980).

B. Medical and Technical Problems with Deep Diving

In the context of attempts to extend saturation diving to deeper depths, numerous medical problems have become evident, including compression arthralgia, high pressure nervous syndrome (HPNS), fluid and electrolyte imbalance, psychiatric disorders, respiratory compromise, long term neurological effects and others (Halsey et al, 1975; Fructus, 1980). Technical problems which have occurred center on diver breathing and thermal protection, adequate underwater breathing apparatus (UBA), diver monitoring, and environmental control (Vorosmarti, 1975; Elliott, 1975).

Consideration of these problems has led Lambertsen (1980) to list the following limiting factors in deep saturation diving:

(1) Temperature (both hypo- and hyper-thermia)

(2) Compression/hydrostatic pressure (especially on neurologic functioning)

(3) Inert gas exchange (including O_2 tolerance, rate of elimination and isobaric counter diffusion)

(4) Breathing gas density

He also issued the following caution: "Diving is not simply passive exposure to gas pressure in a chamber or simply breathing underwater. Therefore, prediction of limitations must be concerned not only with the absence of convulsions, or unconsciousness but also with the quality of thought and the capacity for useful physical action".

C. Experience in Deep Diving (1968-1983)

Since 1968, experimental dry and wet chamber dives, as well as open sea dives have been undertaken to test the limits of deep diving. These experiments have provided a tremendous body of literature regarding the physiological, psychological and performance changes associated with diving at great depths. Man's horizon has been extended to 2250 FSW (686 MSW) in a dry chamber (Atlantis III; Bennett, 1981) and to 1650 FSW (501 MSW) in the open sea (JANUS IV; Fructus, 1980). The physiological and performance data generated from these and the many other deep dives have formed the basis for predictions of limitations to saturation diving.

1. French Diving Experience:

Hyperbaric studies in France have been undertaken using both military and commercial (COMEX) diving volunteers to investigate the effects of varying compression schedules and different inert gas mixtures on various physiological, neurological and performance parameters to optimize deep

saturation dive procedures. These experiments from 1968-1983 are listed in Table IA. These multiple diving series were developed to extend both bottom time and maximal depth in exploration dives (PHYSALIE I-VI, time 20-80 min; depth 335-610m), saturation dives (SAGITTAIRE I-IV, 2-10d; 300-610m) and working dives (JANUS IIIA, IIIB, IV, 6-16d; 390-501m) (Fructus & Rostain, 1978). In addition, these series were instrumental in the development of modifications to companion schedules for deep dives to include the intermediate stages (PHYSALIE IV, SAGITTAIRE and III), or exponential (i.e., decreasing rate of compression with increasing depth) compression profiles (JANUS IIIA and SAGITTAIRE II), or both (JANUS IIIB, PHYSALIE V and VI, SAGITTAIRE VI, DRET 79/131, ENTREX V AND VIII) (Fructus & Rostain, 1978; Rostain et al, 1984; Torok, 1984). They also furthered advances in the use of breathing mixtures containing nitrogen (Trimix) in deep dives (PHYSALIE I-IV, CORAZ I-III, JANUS IV, "Selection", DRET 79/131, ENTEX V and VIII). Both exponential compression with stages and use of Trimix were developed to reduce occurrence of HPNS at depth (Rostain et al, 1984; Rostain et al, 1982; Fructus & Rostain, 1978; Fructus et al, 1976; Rostain & Lemaire, 1984; Bennett et al, 1981b; Bennett et al, 1981a).

2. <u>British Hyperbaric Studies</u>:

In addition to a few earlier deep dives (in 1969 and 1970), the Deep Dive Series at AMTE(PL) was started in 1975 to improve techniques in

TABLE IA

French Hyperbaric Studies, 1968-1983

Rate of Compression

	DIVE NAME	DATE	NO. OF DIVERS	MAX DEPTH	BOTTOM TIME	COMPRESSION TIME	COMPRESSION STAGES	DECOMP. TIME (h/d)	BREATHING MIXTURE (B=Bar)	TOTAL TIME
1.	PLC-1	1968	2	335m	17 min	133 min	+	94 1/2h	He/N_2 3%/O_2 3.7%	97h
2.	PLC-3	1968	2	330m	20 min	85 min	-	91 h	He/N_2 4.5%/O_2 2.7%	92 1/2h
3.	PHYSALIE I	1968	2	335m	20 min	113 min	+	97 1/2h	He/N_2 4%/O_2 2.6%	100h
4.	PHYSALIE II	1968	2	360m	14 min	115 min	+	114 1/2h	He/N_2 4%/O_2 1.8%	117h
5.	PHYSALIE III	1968	2	365m	8 min	123 min	-	138 1/2h	He/N_2 5%/O_2 1.9%	141h
6.	PHYSALIE IV	1968	2	300m	10 min	180 min	*****	103 1/2h	He/N_2 5.7%/O_2 1.8%	106h
7.	PHYSALIE V	1970	2	520m	100 min	74 1/2h		8 1/3d	He/O_2 .42 B/O_2(N_2 0.15%)	12 1/2d
8.	SAGITTAIRE I	1971	4	300m	7 2/3 d	164 h		5 1/2d	He/O_2 .42 B/O_2(N_2 0.15%)	17d
9.	SAGITTAIRE II	1972	2	500m	100h	49 h	-	8d	He/O_2 .40 B/O_2(N_2 0.17%)	14d
10.	PHYSALIE VI	1972	2	610m	80 min	233 h	*	9 1/2d	He/O_2 .40 B/O_2(N_2 0.17%)	17.1d
11.	SAGITTAIRE III	1973	4	300m	15d	4 2/3d		7d	He/O_2 .40 B/O_2(N_2 0.17%)	27d
12.	JANUS IIIA	1974	3	460m	6d	50h	-	7d	He/O_2 .40 B/O_2(N_2 0.15%)	15d
13.	SAGITTAIRE IV	1974	2	610m	50h	10 3/4d	**	10d	He/O_2 .40 B/O_2(N_2 0.15%)	22.6d
14.	JANUS IIIB	1974	3	395m	6d	50h		10d	He/O_2 .42 B/O_2(N_2 0.19%)	48d
15.	CORAZ I	1975	3	300m	4d	4h		6d	He/N_2 9%/O_2 .426	15d
16.	CORAZ II	1975	2	300m	4d	4h		6d	He/N_2 4.5%/O_2 .426	15d
17.	CORAZ III	1975	2	300m	33h	4h		6d	He/N_2 4.5%/O_2 0.42	8d
18.	CORAZ IV	1975	2	300m	3 1/3d	4h		6d	He/O_2 .42 B/O_2(N_2 0.15%)	10d
19.	JANUS IV	1976	8	400/460m***	9d/7d	25h		8 3/4d	He/N_2 4.8%/O_2 .42	26d
20.	SELECTION	1979	8	450m	48h	38h		20 2/3d	He/N_2 4.8%/O_2 0.42	14d
21.	FRENCH NAVY SSS6	1979		300m	10d				He/O_2	
22.	DRET 79/131	1980	8	450m	50d	38h	****	12 1/2d	He/N_2 4.8%/O_2 0.48	
23.	ENTEX V	1981	4	450m	12d	38h	****	12 1/2d	He/N_2 4.8%/O_2 0.48	
24.	ENTEX VIII	1982	4	450m	12d	38h	****	12 1/2d	He/N_2 4.8%/O_2 0.48	
25.	ENTEX IX	1983	4	450m	12d	38h	****	12 1/2d	He/N_2 .7 ATA/O_2	

\+ Staged Compression.

* Stops: 45h at 340m, 14h at 535m and 14h at 565m (Rostain & Naquet, 1978)

** Stops: 17h at 200m, 45h at 400m, 23h at 550m and 23h at 580m (Rostain & Naquet, 1978)

*** Open Water Dive: 10 min excursions to 501m (Bennett, 1980)

**** Stops: 150 min at each 100m with N2 injection at beginning of stages (Rostain et al, 1984; Rostain et al, 1982; and Rostain & Lemaire, 1984).

***** Stops: 16h at 1150 FSW, 16h at 1510 FSW (Fructus et al, 1976)

saturation dives and perform physiological studies. Table IB lists these studies from 1969-1981. This series of dives has provided further information on techniques to prevent HPNS, as well as detailed insights into other physiologic parameters, including endocrine, thermoregulatory and metabolic derangements at depth (Garrard et al, 1981; Doran & Garrard, 1984; Torok, 1980; Morrison et al, 1976; Torok, 1984b).

3. USA Hyperbaric Studies (Duke University, University of Pennsylvania, U.S. Navy):

American experience in deep saturation diving has evolved from studies undertaken by commercial, university and military centers. Table IC lists these studies from 1968-1982. Many of these studies are noteworthy for their comprehensive approach (Predictive Studies III and IV; USN 1800 ft; Atlantis I-IV), for their extensive experience with Trimix (Duke dives; Atlantis I-IV), and for establishing the current depth record for experimental dives (to 686m; Atlantis III). Such studies have been instrumental in advancing current concepts in deep diving research (Torok, 1984a).

4. Norwegian and Other Hyperbaric Studies:

Hyperbaric studies in Norway (NUTEC) were instituted to solve problems in operational diving related to national and oil industry interests. Consequently, their experiments dealt more with technical aspects than physiological problems in deep saturation diving. Table ID lists these studies from 1980-1983. Although difficulties were encountered with HPNS and narcosis in these studies, important data were obtained for operational (wet) dives at depth, particularly regarding thermoregulation (Vaernes et al, 1984a; Vaernes et al, 1984c).

TABLE IB

British Hyperbaric Studies, 1969-1981

	DIVE NAME	DATE	NO. OF DIVERS	MAX DEPTH 300m/	BOTTOM TIME	COMPRESSION TIME	STAGES	DECOMP. TIME (h/d)	BREATHING MIXTURE	TOTAL TIME
1.	RNPL/Swiss	1969	3	340m	75h/5h	30 min - 5m/min		88h	He/O_2	7d
2.	RNPL/1500 Ft	1970	2	457m	10h	3.5d			He/O_2	17d
3.	AMTE/PL 5	1976	2	300m	7.6d	2.3d	-	10.3d	He/O_2	20.2d
4.	AMTE/PL 6	1977	2	300m	6.8d	1d	-	10.2d	He/O_2	18d
5.	AMTE/PL 7	1977	2	420m	2d	8.4d	*	15.6d	He/O_2	26d
6.	AMTE/PL 8	1978	2	420m	3.4d	6.5d	*	15.5d	He/O_2	25.4d
7.	AMTE/PL 96	1979	2	540m	2.7d	3.2d	+**	20.2d	He/O_2	26.1d
8.	AMTE/PL CDI	1980	2	300m	3d	3d		9d	He/O_2	15d
9.	AMTE/PL 126	1980	2	660m	36h	3.5d	+***	35d	He/O_2	40d
10.	AMTE/PL 13	1981	2	540m	6h	6d	+	23d	He/O_2	35d

Ref: Daniels et al, 1980; Buhlman et al, 1970; Morrison et al, 1976; Torok, 1980; Doran & Garrard, 1984; Garrard et al, 1981.

∞

\+ Staged compression.

* Compression at 60 MSW/d in 6 increments separated by 2h (Torok, 1984a).

** Compression to 180 MSW (day 1), 300 MSW (day 2), 420 MSW at 5m/min (day 3), 3m/min to 480 and 1 m/min to 540m (day 4), (Torok, 1984a).

*** Compression to 420m (day 1), hold at 420 (day 2), to 540m (day 3) and to 660m (day 4), (Torok, 1984b).

TABLE IC

American Hyperbaric Studies, 1968-1982

DIVE NAME	DATE	NO. OF DIVERS	MAX DEPTH	BOTTOM TIME	COMPRESSION TIME	STAGES	DECOMP. TIME (h/d)	BREATHING MIXTURE	TOTAL TIME
1. USN/Duke	1968	5	305m	77.5h	24.5h		12d	He/O_2	
2. AIRCO/IUC	1968	4	339m	5 min				He/O_2	
3. Predictive Studies III	1972	4	366m	6d	10d		9d	He/O_2*	
4. Duke 1000 Ft	1973	4	305m					He/O_2	
5. Duke Trimix	1973	4	305m	1h	27 min		97h	He/N_2 18%/O_2	
6. Access II	1973	5	305m	30 min	10 min			He/N_2 13%/O_2	
7. USN 1600 Ft	1974	6	488m	8d	6d		19d	He/O_2	32d
8. Duke 1000 Ft	1974	5	305m	2h	33 min			He/N_2 10%/O_2	
9. Predictive Studies IV	1976	4	488m	1.6h	4h	+ **	10d		
10. Duke/AMTE(PL)	1976	2	400m	3 min	4h			He/N2 6%/O_2	
11. USN 1400 Ft	1976	6	427m	11d++	4d		17d	HeO_2	32d
12. USN 1500 Ft	1977	6	457m	4d	16d		16d	HeO_2	36d
13. Atlantis I	1979	3	460m	4d	12 1/3h	+ ***		He/N_2 5%/O_2	
14. USN 1800 Ft	1979	6	549m	5d	3 3/4d	****	27d	He/O_2	40d
15. Atlantis II	1980	3	650m	24h	3d	+ ***	18d	He/N_2 9%-5%/O_2	
16. Atlantis III	1981	3	650m/686m	4d/24h	7d/9h	+ ***		He/N_2 10%/O_2	
17. Atlantis IV	1982	3	650m					He/N_2 5%/O_2	

Ref: Daniels et al, 1980; Lambertsen, 1976; Peterson & Wright, 1976; Lambertsen et al, 1978; Bennett, 1975; Bennett, 1981; Torok, 1984a; Dougherty & Schaefer, 1969).

\+ Staged Compression.

++ Includes upward excursions on 3 days to 1200 FSW.

* Included gas density experiments to He/O_2 depth equivalent 1520m (Lambertsen, 1976).

** After 3 1/2h compression to 1200 FSW, 22h hold at 1200 FSW before excursion(s) to 1600 FSW in 20-40 min (Bennett, 1980).

*** Compression at decreasing rates (18m/min-0.25 min) to 460m, with 1-2h stops at 305, 355, 400 and 430m (Atlantis I-IV), proceeding at decreasing rates (0.2m/min-0.05m/min) to 650m with 2-14h stops at 500, 560 and 611m (Atlantis II-IV) (Bennett et al, 1981).

**** Compression profile included varied rates: 0 to 198m at 6m/h, 198 to 305m at 12.3m/h, 305 to 488 at 8m/h, 488 to 549 at 4.5m/h, with an 8d stop at 198m and stages at 271m (overnight), 427m (1 day) and 463m (8h) (Bennett, et al, 1981).

Hyperbaric studies at other centers are also included on Table ID; CEMA-CERB dive (1972) provided further information regarding respiratory function at deep (51 ATA) $He/0_2$ exposures (Broussolle et al 1976). The Japanese 300m saturation dive (SEADRAGON IV) provided additional physiological data relevant to operational diving by including exercise studies and water immersion at depth (Ohta et al, 1981; Matsuda et al, 1981; Nakayama et al, 1981).

D. Cautions

These hyperbaric studies between 1968 and 1983 as listed on Tables IA-ID constitute the data base of physiological and technical factors modified by diving to depths greater than 1000 FSW. Medical and operational limitations and capabilities are derived from this data base. In turn, policies and procedures for deep saturation diving are formulated based on these assessments. For example, in French studies, the appearance of severe, limiting HPNS symptoms on rapid, linear compressions on $He/0_2$ to depth (PLC-1, PLC-3) led to the development of exponential compression profiles (SAGITTAIRE II) with intermediate stops (SAGITTAIRE IV) and use of Trimix (CORAZ I-III, "Selection"). HPNS symptoms were less severe and less limiting on these latter dives, thus extending the maximum depth and duration at depth attainable (Fructus, 1980).

TABLE ID

Norwegian Hyperbaric Studies; 1980-1983, Other Hyperbaric Studies

DIVE NAME	DATE	NO. OF DIVERS	MAX DEPTH	BOTTOM TIME	COMPRESSION TIME	COMPRESSION STAGES	DECOMP. TIME (h/d)	BREATHING MIXTURE	TOTAL TIME
1. Deep EX-80	1980	3	300m		4 3/4h	+*		He/02	
NUTEC		3	300m		4 3/4h	+**		He/N_2 10%/O_2	
2. Deep EX-81	1981	3	500m	9d	26 3/4h	+***		He/02	
NUTEC		3	500m	13d	41 1/3h	+****		He/N_2 10-2%/O_2	
3. Seaway on-Shore Trial	1983	6	350m	5d		*****	11d	He/02	
NUTEC									
4. CEMA-CERB III	1972	2	500m	1.5d	18h	******		He/02	
5. SEADRAGON IV	1979	4	300m	14d	30h	*******	12d	He/02	

REF:

1. NUTEC: Torok, 1984a; Varnes et al, 1984a; Vaernes et al, 1984b; Myrseth et al, 1984; Vaernes et al, 1984c; Vaernes et al, 1983; Vaernes et al, 1982.

2. OTHER: Broussolle et al, 1976; Ohta et al, 1981; Matsuda et al, 1981; Nakayama et al, 1981.

\+ Staged Compression.
* Compression at decreasing rates (6.0-1.5m/min), held for 8h 41min at 250m.
** Compression at decreasing rates (6.0-1.5m/min).
*** Compression at decreasing rates (6.0-2.0m/min), 3h hold at 216m.
**** Compression at decreasing rates (9.0-0.1m/min), 2 shallow 10 min stages, and five 2-9 deep stages (wet dives in He/O_2 at depth).
***** Linear compression with stages at 216m and 296m; (wet chamber dives at depth) (Myrseth, et al, 1984).
****** Compression to 500m after 27h hold at 400m.
******* Compression linear at 10m/h with 14h stops at 100m and 200m (Ohta, et al, 1981); head out immersion studies at depth.

However, a cautionary note must be made: Interpretation and comparison of different studies must take into account the unavoidable problems inherent in these studies. Foremost of these problems is the necessarily limited number of subjects per study (N=2 to 8; $\bar{X}$=3.3). In light of the marked variability of responses of individuals in the same study (Bachrach & Bradley, 1973; Bennett, 1973; Vorosmarti, 1985; Torok, 1980; Spaur, 1980; Vaernes et al, 1984a; Rostain et al, 1984; Vaernes et al, 1983; Vaernes et al, 1982; Rostain et al, 1981; Lambertsen et al, 1978), as well as the variability of response of the same individual in different dives (Rostain & Naquet, 1978; Naquet & Rostain, 1980), statistical treatment of the data becomes less useful and observations become more anecdotal in nature. Another inherent problem in these studies is their lack of comparability on technical grounds. Obvious and subtle differences exist among these studies with regard to diver selection and training, physical plant, compression profiles, breathing gas mixtures, bottom depth and duration, tasks, performance assessment techniques and tools, environmental monitoring and control, decompression profiles, etc. These myriad variables do not necessarily preclude cogent and reasonable comparisons from being made. But again, caution must be exercised.

III. Physiological Changes in Deep Diving

A. Respiratory Changes at Depths >1,000 FSW

Compromise of respiratory function was anticipated in deep saturation diving based on experience at lesser depths (Lambertsen, 1976). Increased gas

density, with its attendant flow restrictions, mechanical restriction imposed by increased hydrostatic pressure, and increased work of breathing at depth were expected to be limiting factors on ventilatory ability and especially work/exercise tolerance. Numerous studies were undertaken to investigate these limitations: USN 1400 ft (Dwyer et al, 1977; Spaur, 1980); USN 1600 ft (Spaur et al, 1977; Spaur, 1980); CEMA-CERB III (Brousselle et al, 1976); Predictive Studies III (Peterson & Wright, 1976; Lambertsen, 1976); RNPL 1500 ft (Morrison et al, 1976); Predictive Studies IV (Lambertsen et al, 1978); SEADRAGON IV (Ohta et al, 1981; Matsuda et al, 1981), Atlantis I and II (Salzano et al, 1981); USN 1800 ft (Bennett et al, 1981; Spaur, 1980), ENTEX series (Hyacinthe et al, 1984); NUTEC On Shore Trial (Vaernes et al, 1984a); AMTE/PL 7 and 8 (Torok, 1980).

1. Static Lung Volume Changes at Depth.

At 300m (SEADRAGON IV) slight increases in vital capacity (V_C), increased expiratory reserve volume (ERV), decreased inspiratory reserve volume (IRV) and unchanged tidal volume (VTS) have been observed in resting divers (Ohta et al, 1981). At 1600 FSW, functional residual capacity (FRC) was increased (Spaur et al, 1977). At depths of 460m and 650m (Atlantis I and II, respectively) increases in V_T and dead space (V_D) were observed (Salzano et al, 1981). These changes suggest that "ventilatory reserve", i.e. IRV, becomes progressively limited with increasing hydrostatic pressures. However, the prediction has been made that pulmonary mechanical function is sufficient to depths of 5000 FSW (Peterson & Wright, 1976).

In contrast, head out water immersion at 300m (SEADRAGON IV) produced decreases in V_C and ERV, but increased inspiratory capacity (I_C). Therefore, immersion appears to complicate lung mechanics at depth.

2. Changes in Pulmonary Ventilation at Depth.

Pulmonary air flow is decreased at depth: maximum voluntary ventilation (MVV), maximum expiratory flow rate (MEFR) and maximum inspiratory flow rate (MIFR) were decreased at 300m (Ohta et al, 1981; Dougherty & Schaefer, 1969), 400m (Peterson & Wright, 1976; Lambertsen, 1976) and 1600 FSW (MVV by 45%; Spaur et al, 1977). These changes appear to be related to depth and gas density (Peterson & Wright, 1976; Comparesi & Salzano, 1980; Fagraeus, 1981, Ohta et al, 1981). Moreover, an inverse exponential relationship between MVV and gas density has been proposed (Camporesi & Salzano, 1980). Decreased Functional Expiratory Volume (FEV) (1.0/FVC at depth; Ohta et al, 1981) suggests that decreased ventilatory flow rates are due to dynamic airway compression; flow becomes effort-independent (Camporesi & Salzano, 1980; Fagraeus, 1981). Maintenance of adequate flow, therefore, requires increased respiratory work to overcome airway and tissue resistance. The capability of meeting this excess work of breathing requirement ultimately determines the adequacy of respiratory function. The dependent factor in this process appears to be gas density. Predictive Studies III reported no limitation on pulmonary function when gas density was varied from 1 to 25 gm/L (Lambertsen, 1976). They concluded that respiratory function is not a limiting factor in dives at >25 gm/L (500 FSW equivalent; Peterson & Wright, 1976).

Notwithstanding, decrements in pulmonary ventilation do occur. However, adaptation after stabilization at depth, allowing for some recovery of ventilatory ability and MEFR, have been observed (Dougherty & Schaefer, 1969; Peterson & Wright, 1976; Lambertsen et al, 1978).

Lambertsen (1976) has suggested that immersion may offer additional compromise to pulmonary ventilation by increasing resistance and decreasing compliance. This is supported by SEADRAGON IV, in which head out immersion at 300m produced further ventilation decrements (Matsuda et al, 1981), in agreement with an earlier, shallower study (Thalmann et al, 1979).

3. Work Tolerance and Respiratory Function at Depth

Exercise tolerance of the already burdened respiratory system at depth has been studied to estabish maximum work performance limits in saturation diving. However, the results of these studies are widely varied. In SEADRAGON IV (300m) both maximum exercise ventilation (V_E) and maximal work capacity were decreased (Ohta et al, 1981). Similarly, in AMTE/PL 7 and 8 (420m) V_E was reduced (to 60% MVV) but work at 150-200 W was performed, limited only by dyspnea (Torok, 1980). In Atlantis III (686m) work at 200 W for 5 min was tolerated (Bennett et al, 1981). During the USN 1400 ft (open sea) dive, 75 W work was generated (representing a 30% decrease from surface control), limited by dyspnea (Dwyer et al, 1977). In contrast, in Atlantis I and II (460m, 650m) V_E was increased, while moderate exercise was limited by dyspnea (Solzano et al, 1981). In Predictive Studies III (366m) work capacity

was decreased, but no respiratory dysfunction was observed (Lambertsen, 1976). Despite these disparities, Fagraeus (1981) has proposed that limits for "useful work" lie at 1600 FSW in the open sea and 2132 FSW in chamber simulation. Additional considerations include the possibility that improved diver selection and training (specifically to increase V_T) may increase work capability at depth (Hyacinthe et al, 1984), and that inspiratory assistance may lead to improved work capabilty under pressure via decreased inspiratory work of breathing (Fagraeus, 1981).

4. Dyspnea

The subjective sensation of breathlessness has been encountered by divers in many of these studies: NUTEC On Shore Trial Dive (350m), AMTE/PL 7 and 8 (420m), USN 1400 and 1800 ft dives, Predictive Studies IV (488m), Atlantis I-IV (460m-686m). Dyspnea on exertion limits work performance; it becomes more severe and more debilitating as depth increases, progressing to dyspnea at rest at extreme depths. The origin of dyspnea at depth is not certain; Spaur et al (1977) proposed mechanical causes; Salzano et al (1981) suggested a relationship to respiratory heat loss; Camporesi & Salzano (1980) have observed that inspiratory dyspnea is improved with Trimix. Factors deemed conducive to dyspnea at depth include increased breathing awareness, increased respiratory work and abnormalities in the respiratory muscles (Fagraeus, 1981). Dyspnea is considered a limiting factor with moderate exertion at 1400-1600 FSW and at rest at 1800-2100 FSW (Vorosmarti, 1985; Spaur, 1980).

Additional subjective respiratory complaints of divers at depth included obligate mouth breathing, coughing and sneezing, difficulty coordinating swallowing and breathing, and sudden momentary airway obstruction during rapid inspiration in Atlantis II at 650m (Bennett et al, 1981; Salzano et al, 1981). These symptoms were attributed to the extreme gas density levels in these studies, though increased nasal congestion (Fructus & Rostain, 1978) may contribute to obligate mouth breathing.

B. Cardiovascular Changes at Depths >1000 FSW

Extreme hydrostatic pressure can produce cardiac arrest in animals (Lambertsen, 1980). A high pressure bradycardia, presumably via the direct action of pressure on the impulse generating mechanism of the heart, has been noted (Halsey et al, 1975). However, in man these dysfunctions have not been observed. At 300m (SEADRAGON IV) resting heart rate (HR), stroke index (SI) and cardiac index (CI) were unchanged from surface controls. Through exercise, HR at depth was less than surface HR control (Ohta et al, 1981). At 488m (Predictive Studies IV) no EKG or cardiac function abnormalities were observed; HR varied appropriately for activity (Lambertsen et al, 1975; Lambertsen, 1980). Interestingly, a minor bradycardia has been observed in man at 31 ATA with head-out immersion (Matsuda et al, 1981). This, however, would indicate intact reflex mechanisms rather than dysfunction; immersion increases venous return, which increases stroke volume and cardiac output, which produces a decrease in heart rate via increased vasovagal feedback.

C. Gastrointestinal Changes at Depths >1000 FSW

Divers at depths >1000 FSW have, to varying degrees, proportional to depth suffered myriad gastrointestinal (GI) symptoms including nausea, vomiting, increased salivation, "nonspecific epigastric sensation", stomach cramps, diarrhea, anorexia, aversion to food and severe gastrocolic reflex (Bachrach & Bradley, 1973; Bennett, 1980; Spaur, 1980). These symptoms are currently considered to be autonomic (parasympathetic) manifestations of the HPNS (Naquet & Rostain, 1980) to be discussed later (Section III, E.3 below). The observations that negative nitrogen balance can be reversed by dietary supplementation at 540m (Doran & Garrard, 1984) suggest that GI function is sufficient at high pressures. Depth limitation based on GI symptomotology has not been established. Certainly, central nervous manifestions of the HPNS (i.e. motor and higher cortical functions) supersede the autonomic (GI) manifestations in determining diving limits.

D. Renal Fluid and Electrolyte Changes at Depths >1000 FSW

In a 14d saturation dive to 300m (SEADRAGON IV) divers demonstrated a hyperbaric diuresis and a natriuresis, with resultant hemoconcentration and minor (1%) weight loss; nocturia was also noted (Nakayama et al, 1981). Immersion at this depth appeared to augment free water clearance via hemodynamically-mediated inhibition of ADH release; however, prostaglandin (PGE_2) mediated reduction of tubular absorption may also have contributed (Ohta et al, 1981). At 366m for 6d (Predictive Studies III) a similar

diuresis, with attendant increased serum osmolarity (but a kaliuresis rather than a natriuresis) was observed (Lambertsen, 1976; Lambertsen et al, 1978). In a 1.6h excursion to 488m from 366m (Predictive Studies IV) diuresis and kaliuresis were also observed. However, serum osmolority and electrolytes were decreased at depth and within normal limits post-excursion, corresponding to changes in ADH and aldosterone levels (Lambertsen et al, 1978). The discrepancy between these two studies may merely reflect differential time constraints on the regulatory hormones, ADH and aldosterone; aldosterone effects being prolonged and slower to change in response to hemodynamic stimuli than ADH. These studies indicate that hormonal regulation of renal function is operable at depth and that adaptation toward normalization of fluid and electrolyte balance occurs in the face of increased hydrostatic pressure. Water loss, reflected in up to a 10% weight loss in some studies (Halsey et al, 1975) is an easily remedied problem. As such, no depth limitation associated with renal functions/fluid and electrolyte balance has been established.

E. Neurological Changes at Depths >1000 FSW

1. High Pressure Nervous (Neurological) Syndrome (HPNS)

Diving to depths of 400-500m can produce incapacitating neurologic derangements in man including nausea, tremors, dysmetria, imbalance, motor incoordination, loss of manual dexterity and loss of alertness. Progressing to deeper depths can lead to vertigo, marked

indifference to the environment and marked confusion. Further experimental studies in man and animals have delimited the effects of high pressure on neural function, identified as the high pressure nervous syndrome (HPNS) (reviews: Hunter & Bennett, 1974; Bennett, 1982). HPNS produces characteristic alterations in motor, spinal, cerebellar, vestibular, sensory and autonomic functions, EEG changes and altered psychomotor, affective and cognitive functions in man (Naquet et al, 1975; Hempleman, 1975; Lambertsen, 1976, Fructus, 1980); in animals extreme HPNS leads to convulsions and death (Smith, 1975; Halsey, 1975).

Several problems are encountered in attempts to overcome this obstacle to extending the limits of diving: (1) The mechanism of HPNS is not known. The "critical volume hypothesis" is probably too simple (Miller, 1980). The mechanism appears much more complex, probably involving multiple stages in its development and multiple sites of action (Brauer, 1980); (2) Individual susceptibility to HPNS varies as to depth of onset, types of symptoms, and severity of symptoms; (3) HPNS develops as a function of both absolute pressure and compression rate, and is modified by temperature and adaptation; and (4) HPNS can have long term neurological effects, including post-dive tremor, depression, attention deficit and forgetfulness (Fructus, 1980).

However, significant progress has been made in reducing the breadth and severity of HPNS symptoms. Limitations imposed by HPNS have been markedly relaxed by (1) selection of least susceptible divers; (2) use of

suitable rates of compression; (3) use of excursions from saturation at shallower depths; (4) use of narcotics, e.g. N_2 (Trimix); and (5) allowing for adaptation at depth (Bennett, 1980). The advances have been remarkable such that man can now work below 500m in the sea and to 700m in a chamber (Fructus, 1980).

2. Motor Function Changes at Depths >1000 FSW

Tremors, fasciculations and myoclonus are early manifestions of HPNS. They have been observed in many of the earlier deep dives, particularly in those using rapid, non-stop compression and heliox ($He/0_2$) breathing mixtures: RNPL 1500 ft; PLC-3, PHYSALIE V, SAGITTAIRE II, JANUS IIIA, USN 1600 ft and 1800 ft, AMTE/PL8 and 9b, Predictive Studies IV, Deep EX 80 ($He/0_2$) and 81 ($He/0_2$), NUTEC On Shore Trial, and Atlantis I. These motor disturbances have become manifest with wide individual variability (Lambertsen et al, 1978), though they appeared consistent, even characteristic ("tremor signatures") within individuals (Bachrach & Bradley, 1973). They have not generally been considered incapacitating--"more irritating than disabling" (Bachrach & Bradley, 1973). However, they probably have contributed to fatigue, psychomotor performance decrement (Morrison et al, 1976), mood changes (Bennett et al, 1981) attention lapses and sleep disturbances (Spaur, 1980).

HPNS tremors include both intention tremors and postural tremors. These types are differentiated by their dissimilar responses to compression profile manipulation, narcotics (N_2) and adaptation. Intention tremors are not extinguished as successfully by exponential compression with stages, use of trimix, and with time at bottom as are postural tremors (Vaernes et al, 1983; Bennett et al, 1981). Intention tremors are characterized by 8-12 Hz oscillations occurring early (at 200-300 m), affecting distal extremities first but progressing up the limbs with increased depth, and persisting at depth; severity varies proportionally with compression rate (Fructus & Rostain, 1978). These tremors apparently are not due to impaired respiration, CO_2 retention or temperature; nor do they correlate with EEG changes (Bachrach & Bradley, 1973). Postural tremors observed at depth are characterized by 8-10 Hz oscillations and body sway similar to that seen in ethanol intoxication and thyrotoxicosis. Disturbances in proprioception, spinal reflexes, cerebellar, vestibular and/or extrapyramidal function may underlie their development (Harris & Bennett, 1980; Torok, 1980; Bachrach & Bradley, 1973).

Fasciculations and myoclonic jerking generally appear at 200-300m with rapid compression, or beyond 500m with slow compression. Fasciculations precede myoclonics, but both predominantly affect the upper limbs. At increased pressures they ascend the limbs, sometimes also affecting the neck and face (Fructus & Rostain, 1978; Fructus, 1980). Proprioceptive disturbances, and motor neuron hyperexcitability and/or spinal hyperreflexia may underlie their development (Harris & Bennett, 1981; Bachrach & Bradley, 1973).

Tremors, fasciculation and myoclonus have been completely avoided in some deep dives [Deep EX 80 (Trimix), Atlantis II, Coraz I] and significantly reduced in others (Atlantis I, III and IV, AMTE/PL 6 and 7, "Selection", Coraz II and III, Janus IIIB and IV, Entex V and VIII). These motor symptoms were intensified by exercise at depth (USN 1800 ft), by changing the breathing mixture from Trimix to HeO_2 at depth (Deep EX 80 and 81) and by rapid excursions from deep saturation levels (Predictive Studies IV, RNPL 1500 ft). However, adaptation to these symptoms was observed after 2-24h at depth (Predictive Studies IV, PHYSALIE III) or on subsequent excursions (Predictive Studies IV). However, post dive persistence of tremors has also been observed in some divers (Fructus, 1980).

Therefore, it appears that depth limitation due to motor dysfunction may be overcome by procedural modifications, i.e. the use of exponential compression profiles with stages, the use of trimix, and the use of lag periods to allow for adaptation at depth.

3. Coordination, Balance and Autonomic Changes at Depth

Dysmetria, incoordination and subsequent psychomotor decrement have been observed at depth in many dives at COMEX (Fructus, 1980), in Predictive Studies IV (Lambertsen et al, 1978), Predictive Studies III (Lambertsen, 1975, Hamilton, 1976) and Deep EX 81 He/O_2 group (Vaernes et al, 1983; Vaernes et al, 1984c). Dysmetria and incoordination have been observed at depths of 300-400m, subject to individual variation of susceptibility, and

reversibility with decompression (Fructus & Rostain, 1978). Underlying cerebellar lesions caused by HPNS have been suggested to provoke these changes (Torok, 1980). However, adaptation has been observed at depth (Bennett, 1980); trimix and staged compression also appeared to ameliorate the decrement (Fructus, 1980). Diver motivation, experience and pre-dive training also reduce these decrements. Incoordination at depth, therefore, is not considered a limitation to deep diving.

Vertigo and imbalance have commonly been observed at depths greater than 300m (Bachrach & Bradley, 1973; Spaur, 1980). Though individual susceptibility to these vestibular symptoms varies, they are generally not incapacitating by themselves, even to 1600 FSW (Lambertsen et al, 1978). However, nystagmus and autonomic symptoms, including nausea, vomiting, diaphoresis, salivation, headaches, colic pain and diarrhea (see Section IIIB), also become manifest in association with vestibular derangement, adding to psychomotor decrement, mood changes and altered cognitive abilities (Halsey et al, 1975). The mechanism of vestibular function disturbance under pressure is not certain; direct effects on the vestibular end organ have some experimental support (Torok, 1980); caloric, indirect and other effects have also been proposed (Bachrach & Bradley, 1973). Vestibular symptoms subside with time at depth; severity is reduced by slow, staged compression profiles, and by increased diver experience, training and motivation (Lambertsen et al, 1978). Therefore, with careful diver selection, appropriate compression and allowance for adaptation, the symptoms of vestibular dysfunction (e.g. vertigo, imblance) and autonomic symptoms are not limiting at depth.

4. Auditory and Visual Function at Depth

Concern has been expressed that Eighth Nerve Disturbance (END) in divers may result in auditory dysfunction, ranging from low frequency hearing loss to unilateral or bilateral deafness (Hempleman, 1975). In Predictive Studies III (Lambertsen, 1976) and IV (Lambertsen et al 1978) no auditory dysfunction was observed. The only other reported auditory disturbance was a single case of Meniere's disease during decompression in the RNPL 1500 ft dive (Morrison et al, 1976), unrelated to HPNS.

Further, at depth divers may experience visual disturbances including tunnel vision and a previously reported sudden-onset lack of eye focus (Naquet et al, 1975). In Predictive Studies IV (Lambertsen et al, 1978) small, transient and insignificant changes were noted in visual accommodation and acuity at depth; field of vision and extraocular eye movements were unchanged. In Predictive Studies III (Lambertsen, 1976) no visual changes were observed.

No auditory or visual disturbance is considered limiting to the depth of saturation diving.

5. EEG Changes at Depths >1000 Feet

Increasing hydrostatic pressure produces significant changes in brain wave activity in man consisting of increased presence of slow waves (Delta and Theta) and decreased presence of fast waves (Alpha and Beta) (Bachrach & Bradley, 1973). These EEG changes are considered characteristic of the HPNS (Naquet et al, 1975). Though the onset and degree of EEG changes are subject to individual variability, they tend to correlate predictably with susceptibility to other HPNS symptoms (Rostain et al, 1984). Test dive (to 180m) EEG changes have been used as a screening tool for diver selection in some deep dive series (Bennett, 1980). However, the significance of these EEG changes has been debated; Theta activity changes do not correlate well with tremors (Bachrach & Bradley, 1973), nor with performance (Lambertsen et al, 1969). Increased slow wave activity with decreased fast wave activity do, however, seem to correlate with "microsleep" or loss of vigilance. Increased Delta activity which occurs in Stage I sleep, appears to correlate with the observed tendency to fall asleep without external stimuli at depth; decreased Alpha activity appears to correspond to decreased alertness (Torok, 1980).

Experience in deep dives has been varied. Characteristic EEG changes were maximal with arrival at 460m in Atlantis I, but improved with time (Bennett et al, 1981). Adaptation was also observed in Deep EX 81 (500m) after 48h at depth (Vaernes et al, 1984c). Trimix appeared to prevent EEG changes in Deep EX 80 (300m) compared to heliox (Vaernes et al, 1982), but not in Deep EX 81 (Vaernes et al, 1983). Other trimix dives (Duke 1000 ft and

Atlantis II) reported little or no EEG changes (Bennett, 1980; Bennett et al, 1981), though EEG changes developed progressively after a delay in Janus IV (Naquet & Rostain, 1980; Gardetti & Rostain, 1981). Less EEG changes were noted with slower compression rates in AMTE/PL 6-8 (Bennett, 1980). However, EEG changes persisted post dive in Deep EX 80; this persistance was variable and ranged from hours to weeks post dive (Vaernes et al, 1982). Unusual paroxysmal EEG discharges have also been reported (Bennett, 1980; Torok, 1984).

Therefore, EEG changes and their associated somnolence appear to be useful diagnostic and predictive measures of susceptibility to other HPNS symptoms at greater depths. Adaptation, appropriate compression rates and trimix appear to modify their onset and severity. However, limitations to deep diving are not clearly established on this basis.

F. Neuropsychological Changes at Depths >1000 FSW

Mental status changes in divers at great depths are indicative of perturbation of higher cortical function in man. These mental correlates of underlying neurophysiological disturbances compromise human performance and therefore may constitute a significant limitation to saturation diving. Alteration of cognitive and perceptual abilities at depth has been studied intensively; the ultimate goal has been to maximize human performance capability, keeping in mind the complex interaction between environmental, equipment, human and procedural factors involved (Bennett, 1973; Reilly & Cameron, 1973). Objective assessment of diver mental status, including

perceptual, cognitive, memory and psychomotor abilities (Fletcher, 1977), however, has not been uniform. Various tools utilized include: SINDBAD (Reilly & Cameron, 1973), PMS (Vaernes et al, 1984a), Halstead-Reitan battery (Becker, 1984) and others (Carter, 1977). Therefore, caution must be exercised in making close comparisons, e.g. when verbal vs. manual responses have been used or when different skills/tasks have been used to describe the same mental function.

1. Psychomotor Changes at Depth

Disturbance of the diver's psychomotor function has been considered a major limitation to performance capability at depth. Psychomotor decrement has been observed to include mental slowness, prolonged reaction time, increased errors, depressed visualmotor speed and coordination, as well as decreased manual dexterity. These decrements, in sum or in part, were observed in most COMEX dives, Predictive Studies III and IV, USN 1500 and 1800 ft dives, Atlantis I and II, RNPL 1500 ft, AMTE/PL 126, Deep EX 80 and 81 and On Shore Trial Dive. These decrements appeared to vary as a function of depth; in the AMTE/PL 126 dive to 660m only small psychomotor decrement was observed at or below 300m (Logie & Baddeley, 1983); finger dexterity was decreased by 9% at 300m in the On Shore Trial Dive (Vaernes et al, 1984b) and decreased by 23% at 500m in Deep EX 81 (Vaernes et al, 1983). Individual variability, however, appears to be pronounced; e.g. in "Selection" dive at 450m a psychomotor decrement of 6-10% was determined, yet in Atlantis I at 460m, the decrement was 40-50%. Diver motivation appears to be an important

factor (Parker, 1969). Nausea, tremors and vertigo may also contribute to observed performance. Adaptation to psychomotor disturbance has been observed at depth (Rostain et al, 1982), though the recovery of function was slower than that for other HPNS symptoms [e.g. tremors (Lambertsen et al, 1978; Vaernes et al, 1984c)]. Trimix appeared to add to psychomotor decrement at depth when compared to He/O_2 (Vaernes et al, 1982). Immersion at depth also increased the impairment (Carter, 1977). These changes are reversed with decompression. Subjective reports of psychomotor decrement persisting days to months post dive eventually disappeared also (Becker, 1984).

Mental slowness, prolonged reaction time and decreased dexterity are variably manifested derangements of psychomotor function often associated with HPNS at depth. Individual training and motivation, as well as adaptation at depth, can alleviate but not eliminate these symptoms. Trimix appears to aggravate the symptoms via narcotic effects of N_2. Diver selection and training may offer the best means of reducing the limitations on performance these changes impose at depth.

2. Thought Changes at Depth

Abnormal thought content was observed in only one study. In Deep EX 81 (500m) visual and auditory hallucinations were reported 12 hr following change of breathing mixture from 10% trimix to heliox on day 4 at bottom (Vaernes et al, 1984c). Post dive complaints of difficulty thinking

and putting thoughts together (Spaur, 1980; Becker, 1984) were not substantiated by objective testing in a long term follow-up (Becker, 1984).

3. Cognitive Changes at Depth

Cognitive function disturbances have been noted in many but not all divers at depth, affecting their attention and concentration, arithmetic and grammar abilities, reasoning and memory. These disturbances have limited performance in deep dives.

Deficits in attentiveness and concentration ability have been observed in numerous COMEX dives (Fructus, 1980), Deep EX 80 and 81 (Vaernes et al, 1983; Vaernes et al, 1984c), USN 1800 ft (Bennett et al, 1981) and Predictive Studies IV (Lambertsen et al, 1978). These deficits were variable, they intensified with increasing depth (Vaernes et al, 1983), but regressed completely with time at depth (Rostain et al, 1984). Slower compressions resulted in less severe deficits (Fructus, 1980). Concentration difficulty was more common with trimix than with He/O_2 (Vaernes et al, 1984c). Inattentiveness, somnolence and/or microsleep appear to correlate with specific EEG changes, namely increased slow wave activity with increasing depth (see Section III.E.5). Post-dive difficulties with attention have been reported (Spaur, 1980), which may persist for long periods. One of six divers displayed measurable attention decrement three years after the USN 1800 ft dive (Becker, 1984).

Temporary impairment of intellectual function in saturation divers has been observed in decreased arithmetic, grammar and reasoning abilities. These decrements appeared to vary proportionately with increasing depth, e.g. no deficit was observed in the RNPL 1500 ft dive (Morrison et al, 1976); a 4-30% decrease in arithmetic was seen in the Deep EX 81 500m dive (Vaernes et al, 1984) and a 40% decrease was observed in Atlantis III at 686m (Bennett, 1981). In some cases, cognitive decrements were more pronounced during compression and stabilized at depth (Vaernes et al, 1984b). Adaptation at depth has also been observed, with progressive recovery from initial deficits (Bennett et al, 1981) even to surface control levels with sufficient time (Vaernes et al, 1984c). However, the presence of N_2 in the breathing mixture appears to have detrimental effects on intellectual function. In Predictive Studies III, intellectual cognitive decrements were observed with N_2, but not with Ne or He at depth (Lambertsen, 1974). In Deep EX 81 reasoning ability was more depressed in the trimix group than the heliox group (Vaernes et al, 1983). In addition, recovery of intellectual abilities by adaptation at depth was suppressed by N_2 in Deep EX 81; but with change of the breathing mixture from trimix to heliox, this suppression was lifted (Vaernes et al, 1984c). Intellectual disabilities are intensified by immersion at depth (Carter, 1977). Post-dive difficulties with intellectual cognitive abilities, including decreased arithmetic ability have been reported (Spaur, 1980). Long term follow-up of these sequelae, however, showed that arithmetic, grammar and reasoning decrements are recovered.

Memory losses are also considered manifestions of the HPNS in saturation divers (Bachrach & Bradley, 1973). Both short-term memory (Torok, 1980) and long term memory (Spaur, 1980) deficits have been reported. Short term memory decrement was more pronounced than long term memory decrement in the On Shore Trial dive at depth (Vaernes et al, 1984b). However, short term memory decrement improved with time on trimix in Deep EX 80, while long term memory decrement did not until the breathing mixture was changed to He/O_2 (Vaernes et al, 1982). Long term memory decrement was measurably greater in the trimix group compared to He/O_2 in Deep EX 81 (Vaernes et al, 1983). Therefore, N_2 narcotic effects appear to aggravate long term memory deficits associated with HPNS. Post-dive complaints of forgetfulness by saturation divers (Vorosmarti, 1985) may represent a persisting deficit. In one of six divers of the USN 1800 ft dive decreased incidental memory was observed (Becker, 1984).

Cognitive functions appear to be generally depressed at depth, though wide individual variability has been observed. Effects on concentration, intellectual function and long term memory may be accentuated by narcotics at depth. Immersion further impairs cognitive function at depth. Decrements on intellectual abilities appear to be reversible.

4. Mood Changes at Depth

Altered affect has been observed in divers at depths greater than 31 ATA. These mood changes included hostility, decreased sense of well-being, depression, lethargy, excitement and euphoria (Fructus, 1980; Bennett et al, 1981; Vaernes et al, 1983; Curley et al, 1979). Mood changes during Atlantis I improved with time at depth (Bennett et al, 1981). No mood changes were reported in Atlantis II (Bennett et al, 1981). Mood changes appear to be reversible with no late sequelae (Becker, 1984) and correlate with diver stress (Curley et al, 1979).

5. Sleep Changes at Depth

Sleep disturbances are considered manifestations of the HPNS in divers at depth. Disturbed sleep quality has been reported in several dives: AMTE/PL 126 (Logie & Baddely, 1983), Predictive Studies IV (Lambertsen et al, 1978), several COMEX dives (Rostain et al, 1981; Fructus et al, 1976) and Atlantis I and III (Bennett et al, 1981; Bennett, 1981). Subjectively, divers have reported nightmares and vivid dreams (e.g. of levitation). Objective evidence of altered sleep cycles has also been obtained (Rostain et al, 1981). These include increased number of sleep cycles, but a lightening of sleep, especially during compression, by increased number and duration of waking spells. Also, Stage I and Stage II sleep increased at the expense of Stage III and Stage IV; REM sleep also became unstable and decreased. In the

same study, increased waking and Stage I persisted during decompression, Stage III increased reflexly and Stage IV and REM decreases were less. These changes may reasonably lead to reduced diver efficiency for waking tasks due to sleep deprivation stresses.

G. Musculoskeletal Changes at Depths >1000 FSW

Fatigue and weakness are commonly encountered by divers at deep depths. The effects of hyperbaric pressure on muscles both directly and indirectly may contribute to these symptoms. Though maximum voluntary contraction is not altered, the tetanic force of muscle is increased at elevated pressure (Bolstad et al, 1984). The increased muscle force generated via neuronal hyperexcitability under pressure (Section III.E.2) or isochemic changes may therefore underly the accelerated muscle fatigue observed at depth. In Deep EX 80, measurably reduced hand grip strength at depth in He/O_2 trimix (Vaernes et al, 1982) suggests that suppressible neuronal excitation drives the accelerated muscle fatigue.

Knee pain occurred in one of three divers in Deep EX 81 at 300m on trimix but improved with further descent (Vaernes et al, 1983); severe and persistent joint pain following exercise has also been observed in some USN divers at depths beyond 300m (Spaur, 1980). These are manifestations of pressure arthralgia which can develop at shallower depth; the mechanism may involve a pressure-induced osmosis which causes dehydration of the articular

cartilage leading to lack of adequate joint lubrication and a predisposition to cavitation phenomena (Hempleman, 1975). The change may not be reversible, though symptoms may subside somewhat. Post-dive persistence of joint pain has been observed (Spaur, 1980).

Aseptic bone necrosis is a chronic hazard for divers, potentially leading to debilitation, osteoarthritis and neoplastic disease (Walder, 1980). The incidence of bone lesions increases directly with increasing depth, such that deep saturation divers are especially susceptible. The mechanisms of bone necrosis may involve impacted bubble emboli or fat cell changes due to pressure such that intramedullary bone circulation is compromised.

H. Other Changes at Depths >1000 FSW

Hematological abnormalities observed in deep saturation divers include a leukocytosis and thrombocytopenia at 1500 FSW (Morrison et al, 1976), increased hemolysis at 660m (Doran & Garrard, 1984) and red cell abnormalities including appearance of echinocytes and spiculed ovoid cells, as well as decreased erythrocyte carbonic anhydrase activity.

Hepatic "embarrassment" has been suggested in one study in which abnormalities in enzyme levels (elevated ALT and AST, intermediate 5' nucleotidese and normal alkaline phosphatse and RGT) were interpreted as indicating hepatocyte leakage (Doran & Garrard, 1984). They further proposed that some features of HPNS may be attributable to hepatic damage, via an hepatic encephalopathy.

Elevated blood T4 levels observed post-excursion (Lambertsen et al, 1978) and during decompression from deep (540-660m) saturation (Doran & Garrard, 1984) are believed to represent a mild thyrotoxic state which may contribute to hyperbaric fatigue phenomena. This thyrotoxicosis is probably due to reduced clearance rather than pituitary-axis stimulation (Garrard et al, 1981; Doran & Garrard, 1984). Blood levels of stress hormones (cortisol, norepinephine) are also elevated at depth (Torok, 1980). Insulin levels are variable (Torok, 1980; Lambertsen et al, 1978).

Negative nitrogen balance has been demonstrated in divers at depths greater than 300m (Torok, 1980; Garrard et al, 1981; Doran & Garrard, 1984). The deficit is estimated at 4-8g/day, but is reversible with decompression. Diet supplementation can reverse the negative nitrogen balance in some divers at depth (Doran & Garrard, 1984). Elevated thyroid and adrenocortical hormone stimulation may in part underly this imbalance (Garrard et al, 1981).

Thermoregulation is stressed at depth. Heat loss is accelerated at the body surface as a function of ambient temperature and activity level, and via the lungs as a function of breathing gas temperature and increased heat capacity of He under pressure (Lambertsen, 1980; Lambertsen, 1975; Vaernes et al, 1984c). Heat production is also increased at depth via endocrine, metabolic and work components (Garrard et al, 1981). This acceleration in heat exchange results in a significant narrowing of the

thermal comfort range for divers at depth (Lambertsen, 1980) and can significantly reduce diver performance. Significant decreases in psychomotor and cognitive function were observed with a decrease in core temperature of 0.3°C; shivering was observed at a core temperature decrease of 0.9°C with further decrement in performance (Vaernes et al, 1984c). Thermogenic regulation, therefore, is disturbed at depth but may not be limiting with effective control of the thermal environment (Garrard et al, 1981).

IV. Summary of Changes at Depths >1000 FSW and Their Limitations

A. Respiratory

Increasing hydrostatic pressure imposes progressive restrictions on pulmonary mechanics. Though tidal volume (V_T) may be unchanged or slightly increased, dead space (V_D) is increased and inspiratory reserve volume (IRV) is decreased significantly. Ventilatory reserve is compromised by these changes in static lung volumes. Immersion at depth imposes additional restrictions by further decreasing vital capacity (V_C) and expiratory reserve volume (ERV).

Pulmonary ventilation is progressively compromised at increasing depths secondary to increased gas density and airway resistance. Maximum voluntary ventilation (MVV) varies as an inverse exponential function of gas density. Maximum expiratory flow rate (MEFR) is further compromised by dynamic airway compression. These changes require an increased work of

breathing in order to maintain adequate tissue oxygenation. Immersion at depth further compromises pulmonary ventilation by increasing tissue resistance, augmenting airway compression and decreasing compliance; thus, adding to the work of breathing. However, adaptation, defined as recovery of function after stablilization at depth, allows for some improvement in ventilatory ability observed between 300 and 500m.

Current experimental evidence suggests that the limits of deep diving due to pulmonary mechanical and ventilatory restrictions lie in excess of 5000 feet heliox equivalent (1500m) in chamber (dry) environments and somewhat less with immersion. These limits lie well beyond the 1983 achieved levels of 700m (chamber) and 500m (open sea).

Increased work of breathing and dyspnea reduce the exercise tolerance and work capability of divers. Exercise tolerance varies with ventilatory capacity (V_E), which is limited by the MVV. Diver selection and training may improve V_E at depth, resulting in improved exercise tolerance. Inspiratory assistance, by reducing the work of breathing, may also improve exercise tolerance and work capacity at depth. Dyspnea is invariably encountered, limiting exertion as depth increases and progressing to dyspnea at rest at extreme pressures. Control of breathing gas temperature and the presence of N_2 in the breathing mixture (Trimix) improve dyspnea.

Limitation to deep diving due to exercise intolerance coincides with the 1983 achieved depths of 700m in a chamber environment and 500m in the open sea. Limitation due to dyspnea presently appears at 450-500m with moderate exertion, 600-700m at rest. These limits may be extended by diver selection and training, breathing mixture control and use of Trimix.

B. Cardiovascular

Cardiovascular function is not significantly altered with increasing hydrostatic pressure. Cardiovascular response to exercise and immersion is appropriate at depth. Limitations to saturation divers due to cardiovascular function lie beyond 5000 FSW (1500m).

C. Gastrointestinal (GI)

GI symptomotology develops in divers at depth secondary to neurological disturbances. Symptoms are reversible and improved by techniques which alleviate neurologic dysfunction. GI disturbances are not presently limiting factors in deep diving.

D. Renal/Fluid and Electrolyte

Increasing hydrostatic pressure produces a hyperbaric diuresis with attendant free water and K losses, secondary to renal perfusion and reflex endocrine (ADH and aldosterone) changes at depth. Adaptation at depth

alleviates these changes and restores fluid and electrolyte balance, though minor water loss continues. Immersion at depth augments free water clearance by similar mechanisms. Water losses at depth contribute to weight loss in saturation divers. These changes, however, are not limiting at present in deep diving.

E. Neurological

Hyperbaric pressure has direct disruptive effects on the nervous system producing a variety of neurologic derangements collectively defining the high pressure nervous syndrome (HPNS). HPNS constitutes the major physiological limitation for saturation divers, though advances have been made.

Motor manifestions of neurologic dysfunction include tremors, fasciculations and myoclonus. Tremors are further subdivided into intentional and postural types. Neuronal hyperexcitability via direct hyperbaric effects probably underly these motor disturbances, though more complex mechanisms are possible. Motor dysfunction occurs early in compression (200-300m), varies proportionately with depth and rate of compression, and is subject to wide individual variability. These disturbances contribute to fatigue, psychomotor and cognitive decrements, mood changes and sleep disturbance at depth. Tremors, fasciculations and myoclonus become less severe at depth via adaptation (improvement in postural more than intention tremors). They may be ameliorated by using slow, staged compression profiles and narcotics (N_2 in trimix). Tremors may persist post-dive in some individuals.

Cerebellar, vestibular and autonomic manifestations of neurological dysfunction in the HPNS include dysmetria, incoordination, vertigo, imbalance, vomiting, sweating, headaches, colic and diarrhea. Direct pressure effects on the cerebellum and vestibular end organ may underly these symptoms; negative symptoms arise secondary to vestibular dysfunction. Collectively, these symptoms occur at depths >300-400m, subject to wide individual variability. They contribute to psychomotor and cognitive decrements, as well as mood changes at depth. Adaptation occurs at depth, and slow, staged compression profiles reduce their severity. Diver motivation, training and experience also reduce their severity. Trimix reduces cerebellar symptoms (dysmetria and incoordination), but not vestibular and autonomic symptoms.

Significant alternations in auditory and visual function are not observed at the present limits of deep diving experience.

HPNS produces characteristic changes in the EEG, namely increased slow wave (Theta and Delta) activity and decreased fast wave (Alpha and Beta) activity. These changes correlate with inattentiveness or microsleep in divers at depth, but not tremors or performance decrements. EEG changes also correlate with susceptibility to other HPNS symptoms and can be used as a screening tool in diver selection. Onset and severity of EEG changes show significant individual variability. However, adaptation occurs at depth. EEG changes are reduced or avoided by slow compression profiles and use of trimix. EEG changes may persist post-dive in some divers (10-30%) but they eventually disappear. Unusual paroxysmal EEG changes are rare but have been observed at depth.

These neurologic disturbances have been observed to varying degrees to the limits of 1983 diving experience (700m chamber, 500m open sea). Current experimental evidence suggests that through the use of diver selection, exponential compression profiles with stages, lag time to allow for adaptation and the use of narcotics (N_2 in trimix) these disturbances may be ameliorated or avoided such that the present limits of deep diving can be extended. The presence of post-dive sequelae, however temporary, recommends caution.

F. Psychological

Psychological disturbances are also considered to be manifestations of the HPNS in divers at depth, including psychomotor and cognitive dysfunction, as well as thought, mood and sleep disturbances. Derangements in these areas constitute a major limitation to diver performance at depths >300m.

Psychomotor dysfunction includes mental slowness, prolonged reaction time, increased errors, decreased visual motor speed and coordination, and decreased manual dexterity. These disturbances become manifest as a function of depth, subject to wide individual variability. Diver motivation and training reduce the decrements. Adaptation at depth is slower than for neurological symptoms. Trimix adds to the psychomotor decrement at depth. Immersion at depth also aggravates psychomotor dysfunction. Recovery generally occurs with decompression, though short-lived persistence of these disturbances post-dive may occur in some individuals.

Cognitive function compromised at depth includes attention/ concentration ability, intellectual function and memory. Inattentiveness and concentration difficulties correlate with EEG Theta and Alpha wave changes. They are more severe with increasing depth, though individual variability is pronounced. Adaptation at depth is complete. Slow compression reduces this severity. Concentration difficulty, however, is more pronounced with trimix than heliox. Long-term attention difficulties may persist post-dive in some individuals. Intellectual decrements include decreased arithmetic, grammar and reasoning abilities. Intellectual decrements vary among individuals. Maximal decrement may occur at the bottom or at intermediate stages. At saturation depth, slow adaptation can lead to complete recovery of function. Trimix aggravates intellectual dysfunction, as does immersion at depth. Short-term persistence of intellectual decrement post-dive may occur in some individuals. Both short-and long-term memory deficits occur at depth. Short-term memory improves via adaptation at depth. Long-term memory deficit is aggravated by trimix. Both improve with decompression though long-term persistence of memory deficit post-dive may occur in some individuals.

Abnormal thought content is rare, though a single incident of visual and auditory hallucinations at depth has been reported. Post-dive difficulty in "putting thoughts together" may occur in some individuals, but is eventually extinguished.

Mood changes at depth include hostility, depression, decreased well-being, lethargy, excitement and euphoria generally related to stress. These changes improve via adaptation at depth and during decompression. Post-dive depression in some individuals is short-lasting.

Sleep disturbances, including vivid dreams and nightmares, occur in some divers. Sleep cycle disturbances include increased number and duration of waking cycles, increased Stage I and Stage II sleep and decreased Stage III and Stage IV. REM sleep becomes unstable and decreases also. During decompression Stage III increases reflexly. Stage IV and REM deficits may persist, post-dive.

These neuropsychiatric disturbances have been observed to varying degrees to the limits of present deep diving experience. Use of slow, staged compression profiles, longer lag times at depth to allow for the slower adaptation from psychomotor and intellectual decrements, and diver selection and training may improve performance at greater depths. But use of narcotics (N_2 in trimix) would seem to only aggravate these disturbances. Long and short-term sequelae of these dysfunctions suggests that the 1983 outer limits of 700m (chamber) and 500m (open sea) may not be extended easily.

G. Musculoskeletal

Fatigue and weakness at depth may be due to direct effects of pressure on muscle, accelerating the rate of muscle fatigueability. Narcotics (N_2 in trimix) may prevent this process by suppressing neuronal excitation of the muscle.

Compression arthralgia develops in some individuals at depth via pressure-induced fluid movements in joints rendering articulae cartillage more susceptible to damage. Though generally reversible, this damage may persist post-dive. Compression arthralgia may represent an unavoidable hazard of deep diving.

Aseptic bone necrosis develops in some divers after a long latency period. Incidence varies directly with depth. The mechanism of the lesion may involve compromised intramedullary bone circulation due to impacted bubble emboli and/or narrow fat cell changes. Bone lesions potentially lead to osteoarthritic or neoplastic disease. Aseptic bone necrosis may represent a chronic, unavoidable hazard of deep diving.

H. Other

Hematological changes include a reversible leukocytosis and thrombocytopenia, as well as a poikilocytosis which may persist post-dive. The significance of this change is not known.

Hepatic embarrassment occurs at depth and may contribute to some features of HPNS.

A mild thyrotoxic state occurs following excursions and during decompression from saturation dives. This may contribute to fatigue, increased metabolism and thermogenic disturbances.

Negative nitrogen balance occurs at depth, estimated at 4-8 g/d. The deficit is reversible with decompression. Dietary supplementation may reverse these losses.

Thermal exchange is accelerated at depth. Heat loss is increased at the body surface and via lung losses. Heat production is also increased.

The thermal comfort range of divers becomes narrow. Slight reductions in body temperature are associated with psychomotor and cognitive function decrements. Decreased thermoregulation may not be limiting with effective control of ambient and breathing gas temperature.

V. Recommendations

On the basis of these physiological and performance considerations in deep diving, the following general recommendations can be made in order to maximize diver safety and performance capability at depth:

(1) Compression rates should be exponential; i.e., slower rates with increasing depth.

(2) Compression rates should include intermediate stages.

(3) Time for physiological and psychological adaptation should be allowed after arrival at depth.

(4) Diver selection should consider motivation, and trainability.

(5) Diver training and conditioning should be maximized to improve performance at depth.

(6) Use of trimix must be controlled to balance its beneficial effects on neuromuscular function with its adverse effects on psychomotor and cognitive functions.

(7) Adequate hydration should be maintained to compensate for water losses.

(8) Dietary supplementation should be implemented to counteract negative nitrogen balance.

(9) Temperature control of the environment and breathing gas mixture must be maintained within strict limits.

(10) Inspiratory assistance should be used to reduce the work of breathing at depth.

(11) Post-dive follow-up should include studies relative to chronic changes, particularly bone necrosis, and neuropsychological status.

VI. Conclusions

A review of physiological derangements associated with deep saturation diving indicates that neuropsychological function and its impairment at depth is the most significant determinant in prescribing the limits to which man may descend in hyperbaric environments. The 1983 limits of 700m (chamber) and 500m (open sea) may be unsurpassible without the development of procedures and techniques to alleviate or avoid psychomotor and cognitive decrement at depth.

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