The oral temperature of the patients at the time of the onset of symptoms was looked at, but no pattern was identified (Figure 8).

TABLE 3
ONSET OF SYMPTOMS vs NUMBER OF DIVES

Number of dives	Number of incidents	Percentage
1 - 10	100	73%
11 - 20	17	12%
21 - 30	2	1.5%
31 - 40	7	5%
41 - 50	7	5%
51 & above	1	1%
No data	3	2%

DISCUSSION

Few authors have reported on the incidence of oxygen toxicity symptoms other than seizures. Ellis and Mandal (1983) in their review of 87 clinical patients, reported side effects in 18 patients: anxiety (43%), nausea/vomiting (13%), dyspnoea (12%), convulsions (5%), paraesthesiae (5%), and perspirations(30%). Donald (1947) found, in experiments with divers exposed to pressures of 3 ATA or greater, the following incidence of symptoms: convulsions (9.2%), twitching of lips (60.6%), vertigo (8.8%), nausea (8.3%), respiratory disturbances (3.8%), twitching of other parts other than lips (3.2%), sensations of abnormality (3.2%), visual disturbances (1%), acoustic hallucinations (0.6%) and paraesthesiae (0.4%). Both of these studies show far greater occurrence rates than here at MIEMSS. In the case of Ellis and Mandal, patients were treated in a monoplace chamber at 2 ATA and in Donald's study the subjects were divers breathing oxygen at depths 3 ATA or greater.

In our study all patients were treated in a multiplace chamber at depths of 6 ATA for decompression sickness (DCS) and Air Embolism, 2.8 - 3.0 ATA for Gas gangrene/Aerobic and Anaerobic infections and Carbon monoxide poisoning/ Smoke inhalation and 2.0 - 2.45 ATA for all other conditions. Thus comparisons of these studies are difficult due to the types of subjects and the different treatment protocols.

At MIEMSS the overall incidences of oxygen toxicity symptoms is much lower than in either of these studies. The highest incidence was that of seizures in patients with air embolisms (4.35%), a group that is prone to seizures just by the nature of their injury, followed by patients treated for gas gangrene or anaerobic and aerobic infections, our most septic patients, with an incidence of seizures being (1.6%) and an incidence of seizures in carbon monoxide poisoning/ smoke inhalation patients of (1.36%). In these 3 categories, patients were treated at depths ranging from 2.8 - 6.0 ATA, a range in which oxygen toxicity is a much greater problem. All other conditions were treated at lower pressures and as would be expected there was a much lower incidence of oxygen toxicity related problems (Figures 1-7 show the full details).

CONCLUSIONS

We have found that the incidence of oxygen toxicity related symptoms at MIEMSS is low, and is easily managed by removal from oxygen when symptoms occur, adjusting air break schedules and in some cases by pre-medication with Diazepam for subsequent dives.

REFERENCES

Donald KW. Oxygen Poisoning in Men. B*ritish Medical Journal*. 24 May 1947: 712-717.

Ellis ME & Mandal BK. Hyperbaric Oxygen Treatment: 10 years experience of a Regional Infectious Diseases Unit. Journa*l of Infection*. 1983; 6: 17-28.

MAMMARY IMPLANTS AND DIVING

RD Vann, GS Georgiade and RE Riefkohl

FG Hall Laboratory,
Department of Anesthesiology, and Department of Surgery
Box 3823
Duke University Medical Center
Durham, North Carolina 27710 USA.

This paper was presented at the 1985 Joint Conference of the Undersea Medical Society Annual Scientific Meeting and the Tenth Annual Conference on Clinical Application of Hyperbaric Oxygen, held 11- 1 ? June 1985 at Long Beach, California, USA.

The Divers' Alert Network has had several enquiries from sport divers concerning mammary implants and decompression safety. As little pertinent information could be found in the literature, an experimental study was conducted to determine if in vitro decompression of mammary implants would cause bubble formation and, if so, how extensive this bubble formation would be.

METHODS

Six mammary implants were tested in four simulated dives. Experiments were conducted with air at a temperature of 20° ± 2°C in a pressure chamber having an internal volume of about 10 cu ft. The implants were exposed to the desired pressure-time profile and then removed from the chamber for observation. The number of bubbles present and their sizes were estimated about once per hour for 5 to 8 hours and at 20 hours post decompression. The volume change of the implant was determined by submerging it in a water-filled container of known volume and measuring the change in container weight since the end of decompression. This method was accurate to a volume change of 1-2%. The maximum volume change occurred 4 t 5 hours after decompression.

RESULTS

Table 1 gives a summary of the results.

The first simulated dive was conducted at a depth of 120 feet of seawater (FSW) with a bottom time of 67 hours followed by immediate decompression to the surface. While this is an unrealistic exposure for divers, it was used because the extent to which bubbles would form was unknown.

TABLE 1
SUMMARY OF EXPERIMENTAL RESULTS

Dive Number	Dive Profile	Implant Number	Experiment Number	% Volume Change	Number of Bubbles	Bubble Sizes(cm)
1	120'/67hr	1	1	47	-	-
2	25'/65hr	2	1	7	-	-
			2	8	-	-
			3	7	-	-
		6	1	3	14	0.02-0.3
			2	1	48	0.02-1.0
			3	1	65	0.005-1.1
3	60'/60min	5	1	0	20	0.04-1.0
			2	3	60	0.02-0.8
			3	1	50	0.02-1.3
4	120'/15min	3	1	1	-	-
			2	3	17	0.2-0.5
	3 hr Surface Interval		3	2	15	0.02-0.6
		4	1	1	-	-
			2	2	170	0.02-2.5
	120'/6 min		3	4	-	-

With a severe exposure, even minimal bubble formation would be difficult to overlook. Many large bubbles developed resulting in a volume increase of 47%. No further tests of this dive were conducted.

The second simulated dive was to a depth of 25 FSW for at least 65 hours followed by immediate decompression to sea level. A marine scientist might experience such an exposure during a saturation dive in an underwater habitat. Two implants were exposed 3 times each. Upon decompression, the first implant had volume changes of 7, 8 and 7%. Fewer bubbles formed in the second implant which had volume increases of 3, 1, and 1%. As many as 65 bubbles developed in these experiments ranging from 0.005 cm to over 1 cm in diameter. The smaller bubbles were spherical while the larger ones tended to be oval or disk-like sheets of gas.

The third series of experiments tested a dive to 60 FSW for 60 minutes. This is a US Navy no-stop diving exposure limit which is routinely used by sports divers. In three exposures-of a single implant, volume increases of 0, 3, and 1% were noted. Bubbles were more numerous and larger in the second and third experiments.

The last series of experiments exposed two implants to a repetitive no-stop dive profile specified by the US Navy Repetitive Dive Tables. The first dive was to 120 FSW for 15 minutes followed by a surface interval of 3 hours. The second dive was to 120 FSW for 6 minutes. In three experiments with the first implant, volume changes of 1, 3, and 2% were observed. The second implant had volume changes of 1, 2, and 4%. After the third experiment with this repetitive dive profile sheets of gas as large as 2.5 cm were seen.

DISCUSSION

The bubble formation observed in these experiments was greater than would occur in vivo for several reasons. In the simulated dives, the implants were instantaneously exposed to the full oxygen and nitrogen partial pressures present in a diver's breathing gas. This does not occur in vivo because there is a circulatory delay in the transport of nitrogen between lungs and tissue and because metabolism reduces the oxygen tension in tissue to below the partial pressure in the lungs.

It has been demonstrated that extensive bubble formation can occur in mammary implants after severe compressed air exposure. Bubble formation leading to a volume increase of several percent might occur after shallow saturation diving. Single and repetitive no-stop dives might lead to the formation of a few bubbles but probably not to an appreciable change in implant volume.

Another significant difference between the in vitro and in vivo environments is the rate at which bubbles are absorbed. A bubble in tissue or in an implant surrounded by tissue is