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13. ABSTRACT (Maximum 200 words) Because of their high compliance, the lungs are probably more vulnerable to damage by high intensity, low frequency underwater sound than any other part of the human anatomy. The objective of this research was to study the effects of low frequency (50-1200 Hz) underwater sound on the lungs to assist in estimating safe exposure conditions for swimmers and divers. To accurately assess the risk, it is necessary to have a complete understanding of the vibrational response of the lungs as well as knowledge of the potential damage mechanism. Lung resonances in an animal model (pigs) and humans were measured at low intensities using NIVAMS (Non-Invasive Vibration Amplitude Measurement System). The results from the human subjects indicated that the fundamental lung resonance was below the lowestfrequency used (50 Hz) and that a secondary resonance appeared in the 100 to 200 Hz range for subjects with their head above the surface. Also an attempt was made to determine damage mechanisms by exposing animals to high intensity sound while submerged. There was no damage detected for the three pigs exposed to sound pressure levels up to 177 dB.

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Final Report for

Navy Contract N00014-93-1-1263

Response of the Lungs to Low Frequency Underwater Sound

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Introduction

Because of their high compliance, the lungs are probably more vulnerable to damage by high intensity, low frequency underwater sound than any other part of the human anatomy. The objective of this research was to study the effects of low frequency (50 - 1200 Hz) underwater sound on the lungs and to estimate safe exposure conditions for swimmer and divers. In order to accurately assess the risk, it is necessary to have a complete understanding of the vibrational response of the lungs as well as knowledge of the potential damage mechanism. It is of principle importance to determine the resonance frequencies of the lungs, because for a given low frequency excitation, the vibration of the lungs (and hence the risk of damage) will be highest at resonance. These resonances must be measured underwater, not in air, since the resonance frequencies will be affected by the additional fluid loading underwater. The lung resonance frequencies may also be a function of depth and the air volume in the lungs.

The original research plan consisted of four tasks involving developmental and research effort:

1. Lung resonance measurements on an animal model,
2. Lung resonance measurements on humans,
3. Lung damage threshold study on an animal model, and
4. Analytical modeling to correlate the lung resonance measurements in humans to the animal model so safe exposure criteria can be based on the damage thresholds for the animal model.

This plan involved measuring the lung resonance of an animal model as well as in humans. Only the animal model would later be used to attempt to establish damage

thresholds. A human size animal model was needed since the scaling of the mechanics and physiology of the lung is not well understood. The domestic pig was chosen as it reasonable represents human physiology and is commonly used.

The plan was to develop the lung resonance measurement technique on the animal model in a small tank (500 gal), both with the head of the animal above the water surface and with the head underwater. Quantitatively, the data collected during the small tank developmental phase were compromised due to the large relative size of the lung volume to the tank volume. Therefore, measurements were also to be made in a large acoustic tank (32,000 gal), both head out and immersed to mid depth (~7 ft). Unfortunately, the lung resonance measurements on the animal model in the large tank were never made.

Next, the plan was to make lung resonance measurements on humans, head out and immersed, in the same large acoustic tank under the same conditions for direct comparison. Then measurements were to be made in the Ocean Simulation Facility (wet pot) at the Navy Experimental Diving Unit in Panama City, FL to examine the effects of depth on lung response. Unfortunately, again, this plan was not followed. Measurements were made on three subjects with their head out in the large tank at Georgia Tech, and measurements were made on six subjects, head out and submerged to 10 ft, in the test pool (30 ft x 15 ft x 15 ft) at the OSF.

The lung damage study involved exposing animal subjects to high intensity, low frequency underwater sound in the acoustic tank at Georgia Tech. During exposure, the animals were clinically monitored for physiological changes. Following exposure, the animals were sacrificed and necropsied. The lungs were examined both macroscopically and microscopically for damage. The goal was to ascertain the damage mechanism to the lungs from underwater sound as well as the sound level at threshold necessary to cause the damage.

Lung Resonance Measurements - Animals

Lung resonances in animals and humans were measured using the NIVAMS (Non-Invasive Vibration Amplitude Measurement System) [Fig. 1]. The NIVAMS uses ultrasound to measure, *in vivo*, the vibration of tissue and organs induced by low frequency underwater sound (Cox and Rogers, 1987). The advantages of the NIVAMS are that it is non invasive in that no surgery is required to make the measurement and it is non intrusive in that the vibrational response is unaltered by the measurement process.

The NIVAMS consisted of three subsystems - the underwater sound generator, the ultrasonic transmitter and receiver, and the transducer positioner. The low frequency underwater sound was created by a wave generator connected to an underwater transducer (J-9). The transmitted 1 MHz ultrasound was created by a high frequency wave generator connected to an ultrasonic transducer. A second identical transducer was used as a receiver, whose signal is viewed on a spectrum analyzer (continuous wave, frequency domain) or an oscilloscope (pulse-echo, time domain). The two transducers were mounted in a fixture that was positioned with an XYZ translator and rotator for the animal study. For the human study, the transducers were positioned by the subject with manual translators. The transducers were positioned to view the surface of the lung through the intercostal space.

The pulse-echo NIVAMS mode was used to locate and identify organs in the body of the subject. The transmitted signal consisted of single 1 MHz sine wave pulses at a 1 KHz repetition rate. This ultrasound traveling through the body is reflected at tissue interfaces. The relative difference in characteristic impedance on the two sides of the interface determines the amplitude of the reflection. Since the characteristic impedance of the air filled lung is much different than that of the tissue in the intercostal space, the lung surface returns a large echo [Fig. 2]. The transducers were positioned to

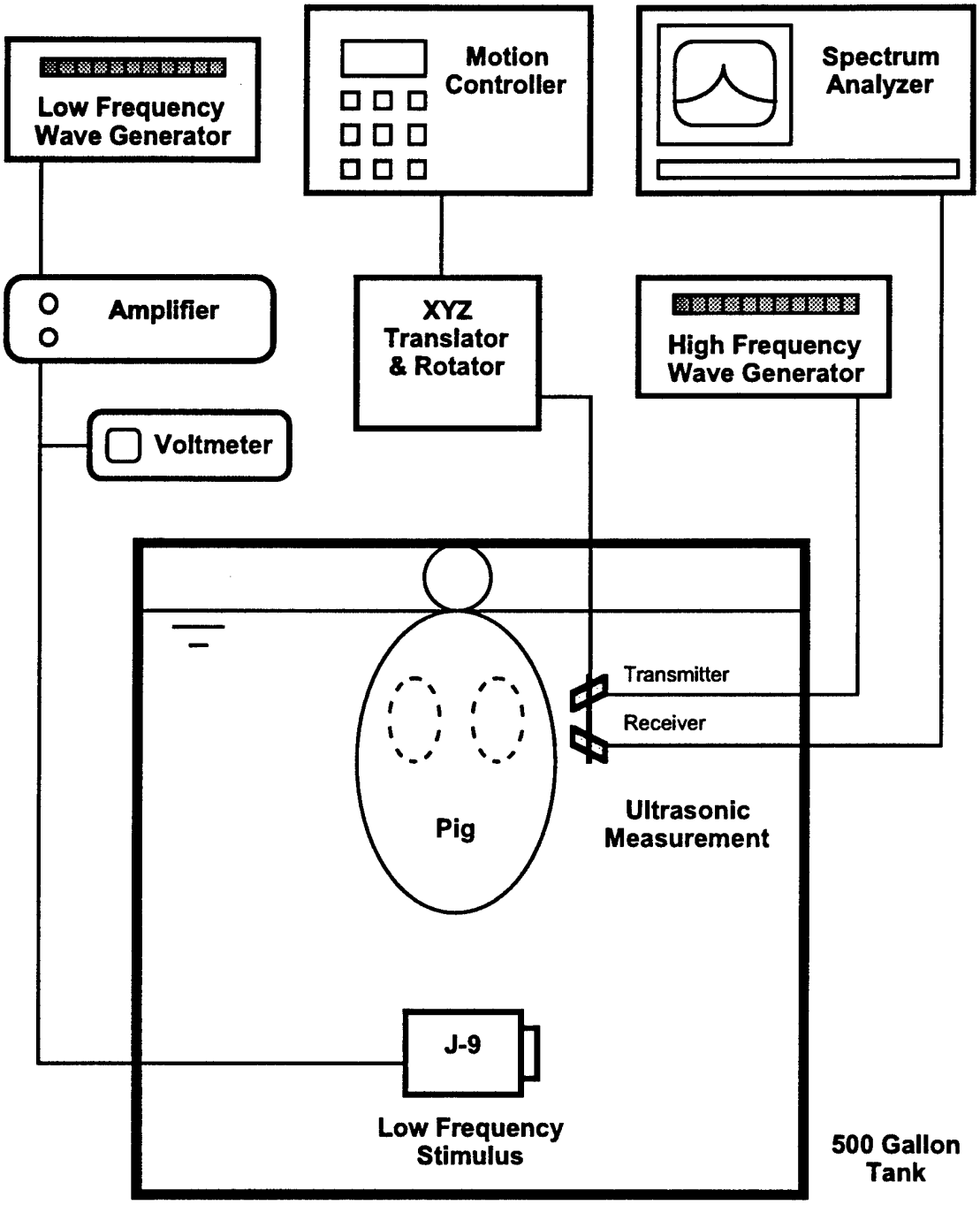

Figure 1. The NIVAMS as used to measure lung resonance in pigs.

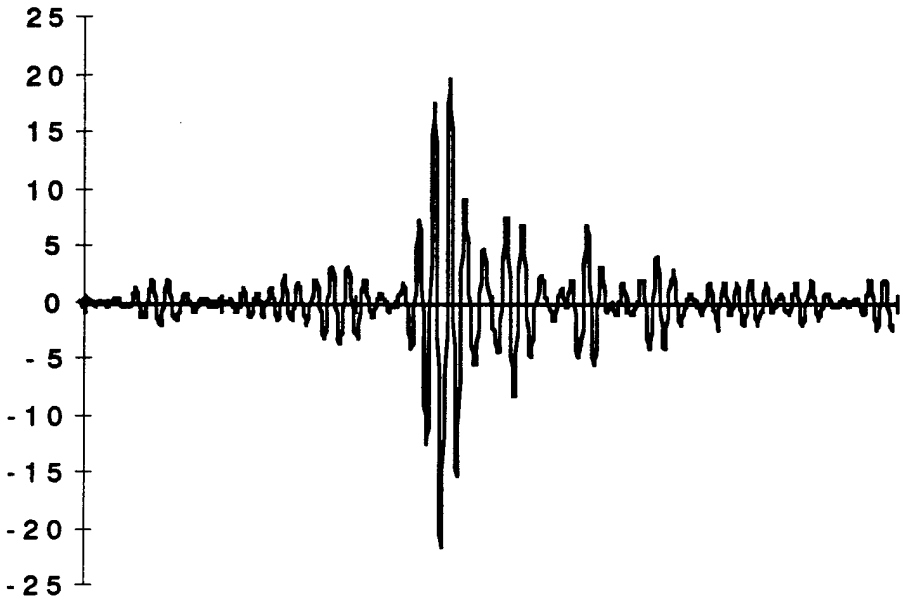

Figure 2. The echo from a single sine wave transmission using NIVAMS in the pulse-echo mode. The largest component is the reflection from the lung. Other echo components are due to various tissue interfaces in the ultrasound propagation path. The ordinate units are arbitrary.

maximize the reflected signal from the lung surface and minimize all other signals. The rigid rib also returns a substantial echo, but can be discriminated from the compliant lung, as the one is 180 degrees out of phase from the other.

The continuous wave NIVAMS mode was used to measure the amplitude of vibration of the reflecting surface. The reflected ultrasound is phase modulated by the moving interface. In the frequency domain, some of the energy from the 1 MHz transmitted signal is shifted to side bands at the ultrasound frequency (1 MHz) plus and minus the underwater sound frequency (shown at 50 Hz) [Fig. 3]. The amplitude of vibration can be calculated from the relative amplitude of the side bands to the center

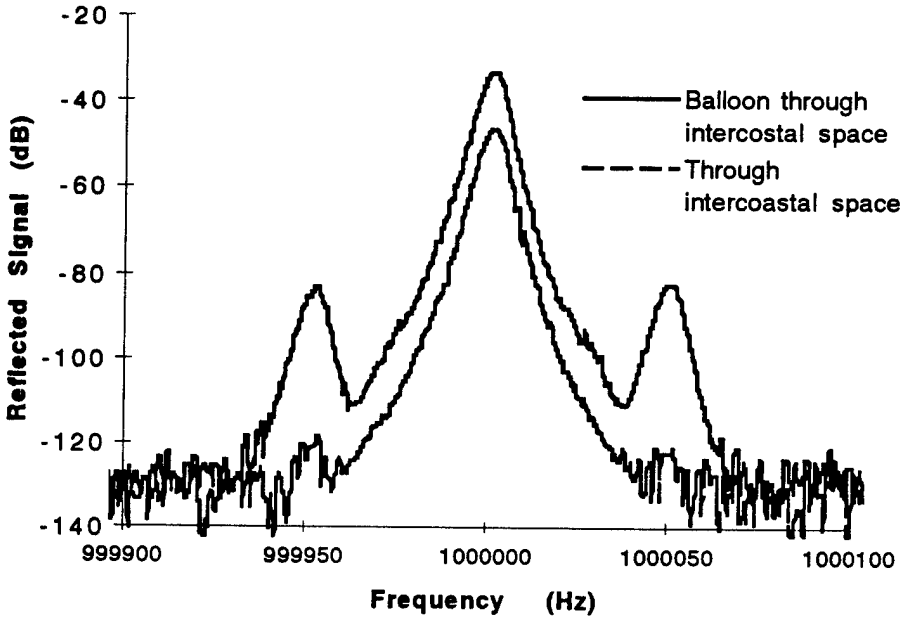

Figure 3. The spectrum of a continuous wave NIVAMS signal. The top curve represents the reflection off the surface of a balloon oscillating at 50 Hz while looking through the tissue of the intercostal space. The displacement amplitude of the balloon's oscillation is proportional to the relative amplitude of the side lobes (at 1 MHz + or - 50 Hz) to the center frequency (at 1 MHz). For the bottom curve, the balloon was removed.

frequency. By varying the underwater sound stimulus frequency in discrete steps, measurements were made to generate a frequency response curve for the lungs.

The initial research effort was directed toward developing the technique to measure lung resonance on the animal model in the small tank (500 gal), both with the head of the animal above the water surface and with the head underwater. An underwater breathing apparatus was also developed at this time to maintain the animal while submerged.

The Porcine Underwater Breathing Apparatus (PUBA) concept is fairly simple. The normal animal ventilator was sealed in a pressure chamber. The pressure level inside the chamber was regulated by an exhaust tube open at the depth of the animal's chest. Therefore, the pressure surrounding the ventilator is at the same ambient pressure as the lungs of the animal, and the airway pressure is kept relatively normal.

Table 1 lists the progression of the program which involved using 16 pigs on 25 days in the laboratory. The use of a mechanical ventilator to maintain the animal while anesthetized allowed measurements to be taken after inspiration, when the lung volume would be the largest, and after expiration, when lung volume would be the smallest. Although the small size of the tank limits the validity of the data quantitatively, the measurements yield useful qualitative information.

Figure 4 shows two frequency response curves for a single animal measured on the same day. The close correlation between the two frequency sweeps illustrates the repeatability of the measurements.

The acoustic particle motion in the small tank caused by the low frequency sound stimulus was calculated from measured pressure data. The amplitude of this particle motion was found to be much less than the amplitude of the vibrational motion measured with the NIVAMS [Fig. 5].

An attempt was made to measure the surface of the lung at different locations to determine if the response to low frequency acoustic pressure is uniform. The top curve of Figure 6 was measured with a lateral orientation between the ribs. The bottom curve was measured with a ventral orientation beneath the sternum. The measured frequency response was similar for the two surfaces of the lung.

Table 1. Progression of the Development of NIVAMS and PUBA in the Small Tank

Exp. #	Date	Pig #	Data	Landmarks:
1	Oct 28	960	-	First pig
2	Nov 23	960	-	Pig bar holds animal vertically.
3	Nov 29	978	-	2.25 MHz transducers.
4	Dec 7	978	-	1 MHz transducers.
5	Dec 9	1011	-	Pig died after removed from bucket.
6	Dec 13	1012	-	Pig overheated in water above 100° F
7	Dec 14	978	R: E, I	Ultrasound imaging located ribs and lungs.
8	Dec 20	1021	R: E, I	-
9	Dec 21	1022	R: I(2)	-
10	Dec 28	1023	R: I; S	Measured skin motion.
11	Jan 3	1021	-	First look toward diaphragm
12	Jan 6	1022	-	-
13	Jan 10	1023	D: E(2), I(2)	Data from diaphragm direction.
14	Jan 13	1057	D: E	Pig died during tests.
15	Jan 18	1058	D: E, I; R: E	Data from both orientations.
16	Jan 20	1059	-	PUBA mask #1; animal pulled due to apoxia.
17	Jan 24	1058	-	PUBA mask #2; animal pulled due to leaking trachea tube cuff.
18	Jan 27	1078	-	PUBA mask #2; Pig would not stop twitching. Used long tubes.
19	Jan 31	1079	R: E	No mask; long tubes.
20	Feb 4	1080	R: E, I(2)	Lid on PUBA; Data taken with pig fully submerged; pig submerged to 11" for 20 min.
21	Feb 7	1079	R: E	Pig was submerged and then died.
22	Feb 11	1080	R: E(2)	Two sweeps at the same location.
23	Feb 14	1111	R:	
24	Feb 17	1112	R: E, I	Huge echoes, Used new ultrasound
25	Feb 21	1113	R: E, I(3)	

R - lung between ribs
D - lung through diaphragm
S - skin
E - lung after expiration
I - lung after inspiration

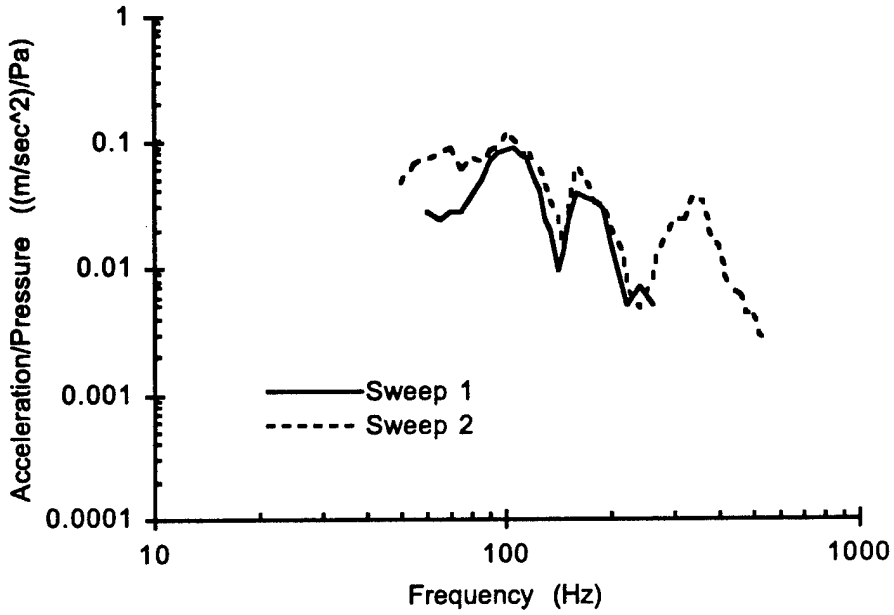

Figure 4. Two frequency response curves for the response of the pig's lungs to low frequency underwater in the 500 gallon tank.

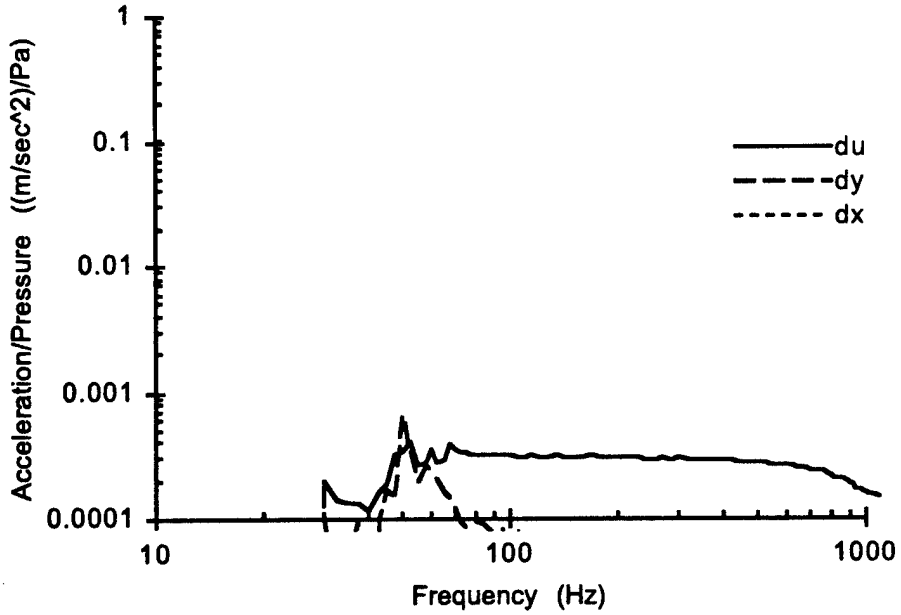

Figure 5. Acoustic particle motion in the 500 gallon tank calculated from measured pressure data. The du direction is vertical and dy and dx are normal in the horizontal plane.

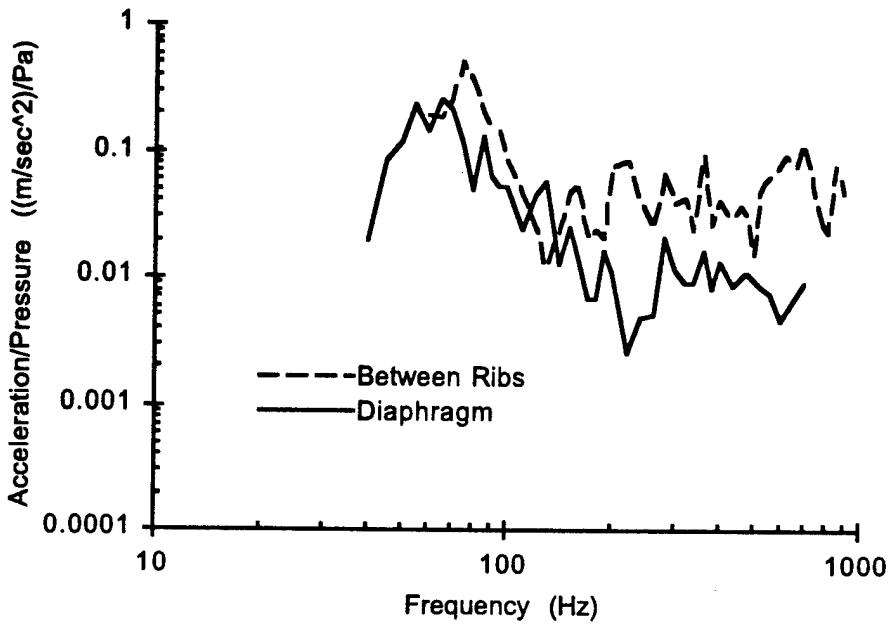

Figure 6. Frequency response of the lungs of a pig measured at two locations. The top curve was measured from a lateral orientation between the ribs. The bottom curve was measured from a ventral orientation beneath the sternum.

The frequency response was measured for an animal subject after having died during preparation [Fig. 7]. This response was significantly reduced at the lower frequencies. This reaffirms the need to use live, intact subjects to obtain relevant data.

The results from the developmental part of this task demonstrated that using NIVAMS to measure lung vibrations would yield reasonable and reliable data. The next logical step would have been to make measurements with the animal at the surface and submerged in a large volume of water. Unfortunately, at the sponsor's request, finishing this task was postponed indefinitely.

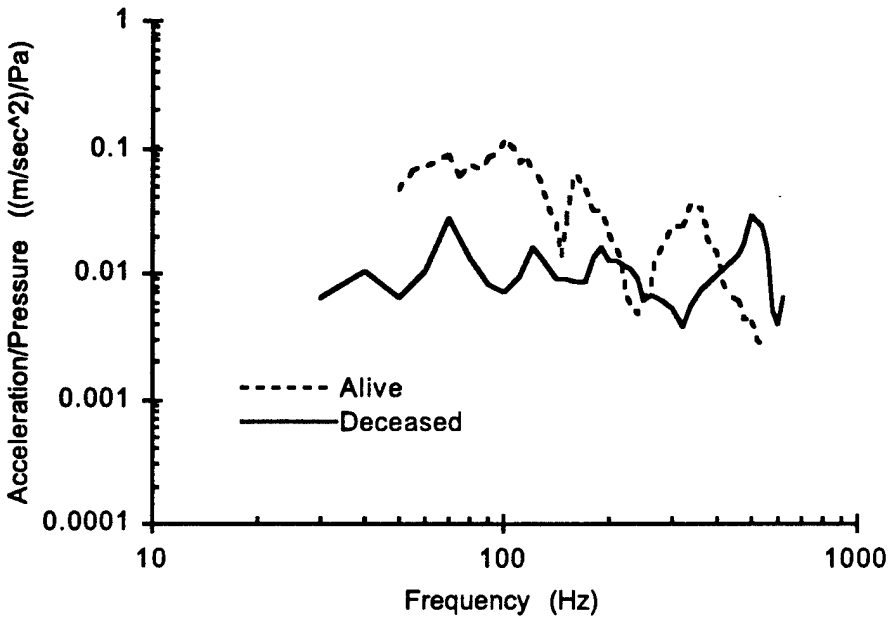

Figure 7. The measured frequency response of a recently (~1 hr) deceased pig as compared to the data from Figure 4. The curves are from different animals on different days.

Lung Resonance Measurements - Humans (Georgia Tech)

The initial developmental work on the use NIVAMS to measure lung vibrations in humans was carried out in the large acoustic tank at Georgia Tech. Although measurements were made on three subjects, only one data set is of interest. For that subject, the vibrational response of a rib overlying the lung was measured in addition to the motion the adjacent lung surface. Figure 8 shows that the responses are distinctly different.

Lung Resonance Measurements - Humans (NEDU)

Lung resonance measurements using NIVAMS were made on a team of six divers in the test pool at NEDU during the week of May 23-27, 1994. Each diver was tested on

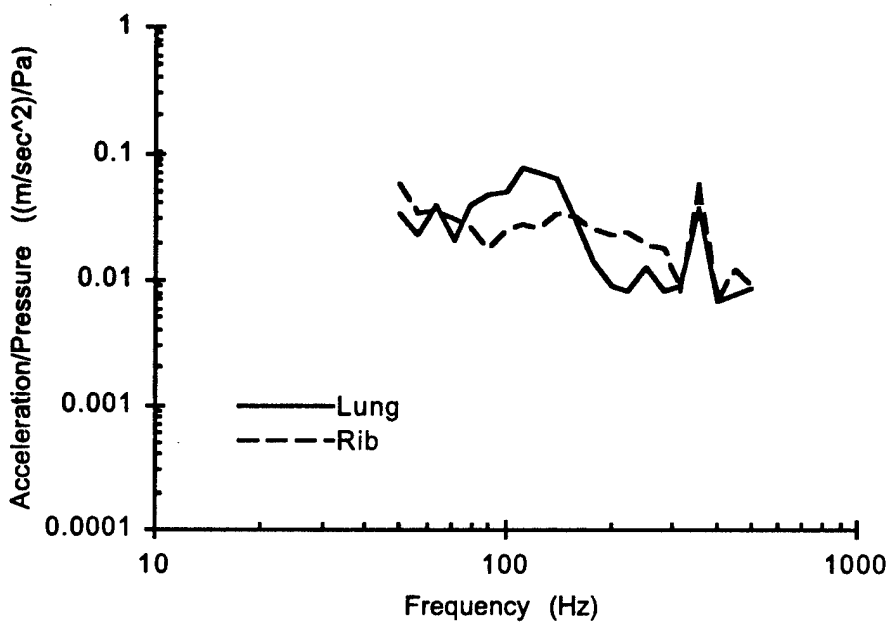

Figure 8. The response of a rib versus the underlying lung in a human subject.

two consecutive days under each of the following conditions: 1) Head-out, with a substernal depth of approximately 8.5 inches, and 2) Submerged to a substernal depth of approximately 10 feet. Vibration amplitudes were measured at discrete frequencies from 50 to 500 Hz in 1/6 octave steps, using a sound pressure level of not more than 130 dB (re: 1 μPa). While underwater, the subjects used a MK-20 diving rig. The diving rig was not used for the head-out study.

In this application a chair and translator system was constructed to position the ultrasonic transducers relative to the subject. To locate the lung, the NIVAMS was used in a pulse-echo mode. The diver sat in the chair and grossly oriented the transducers to point between the ribs under his left arm. Then fine adjustments were made using the translator system. The diver viewed an oscilloscope trace of the echo signal on a TV monitor mounted in a waterproof enclosure attached to the chair. Reflections off the lung

were differentiated from reflections off the rib by the phase of the signal (the rib signal was inverted compared to the lung signal) and the location in space (the ribs are closer to the surface of the skin than the lung). Vibration amplitude measurements were made using the NIVAMS in a continuous wave mode. Individual frequency measurements required a ten-second trial during which the diver held his breath. On the diver's ready signal, the low frequency sound was turned on, the spectral analysis of the reflected signal was completed, then the low frequency sound was turned off. The subject breathed normally between measurements. During this time the data was recorded and the low frequency changed. One to two sweeps in frequency were made per subject per day. This data is shown in Figures 9-11.

The filled symbol data were for the subjects with their head-out. The general shapes of the curves were consistent, indicating a resonance in the 100-200 Hz range. This is probably not the fundamental lung resonance, however. The fundamental resonance was probably below the frequency range of this study. This secondary resonance between 100 and 200 Hz is consistent with the animal data. With the diver immersed in 10 feet of water, this resonance is reduced in amplitude, as shown by the open symbol data. This is a common feature for the data from all subjects: a resonance in the 100 to 200 Hz range head-out whose magnitude is reduced when the subject is underwater. Especially in subjects A and D, it also appears that the frequency of this secondary resonance is raised as the subject is submerged. Data is shown for five of the six subjects. For both the head-out and immersed tests, the subjects were instructed to hold their breath during the individual measurements to obtain constant lung volume. The sixth subject did not do this (bubbles were noted during the underwater tests), so the data on subject F were rejected.

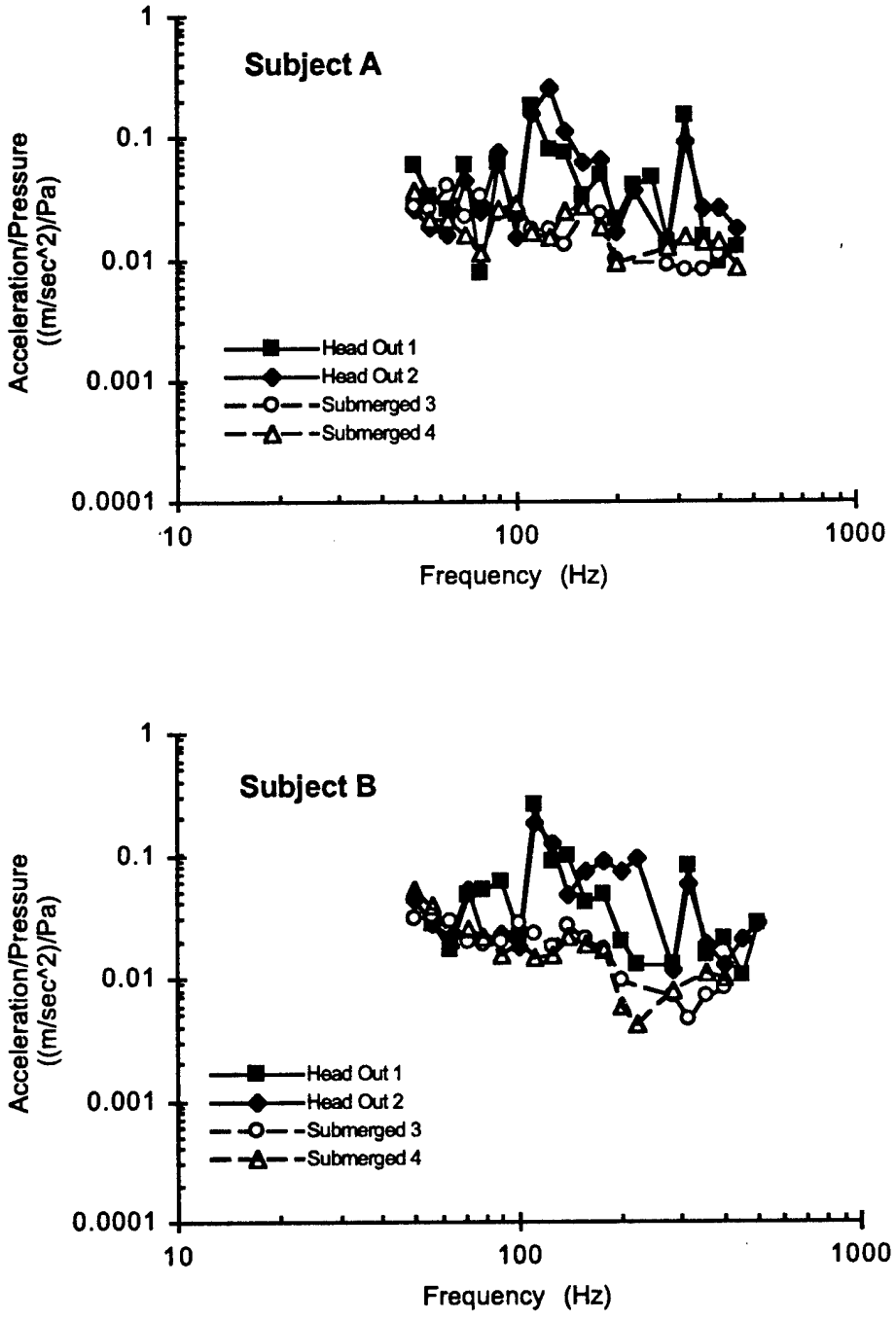

Figure 9. The measured frequency response of the lungs of divers A and B to underwater sound.

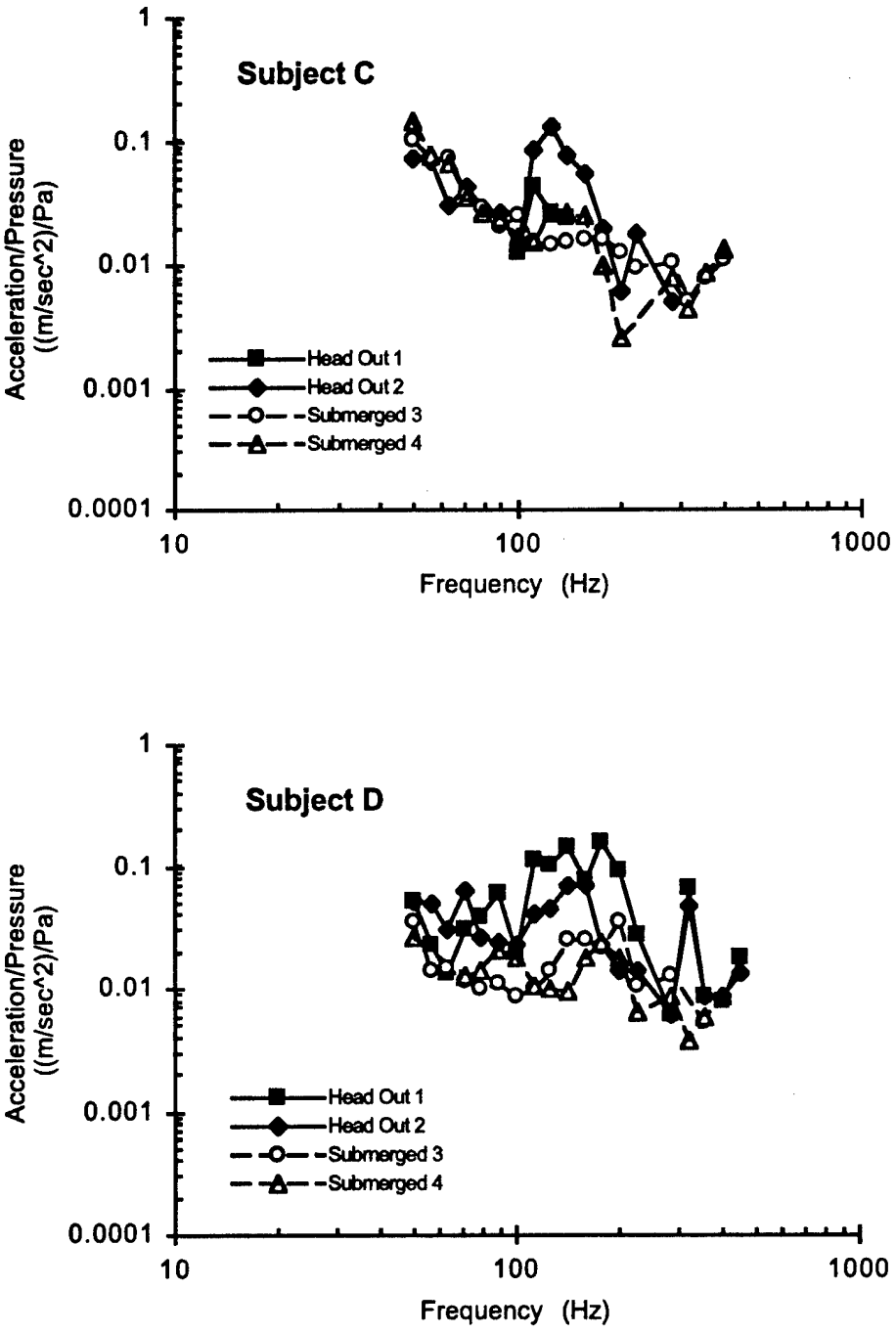

Figure 10. The measured frequency response of the lungs of divers C and D to underwater sound.

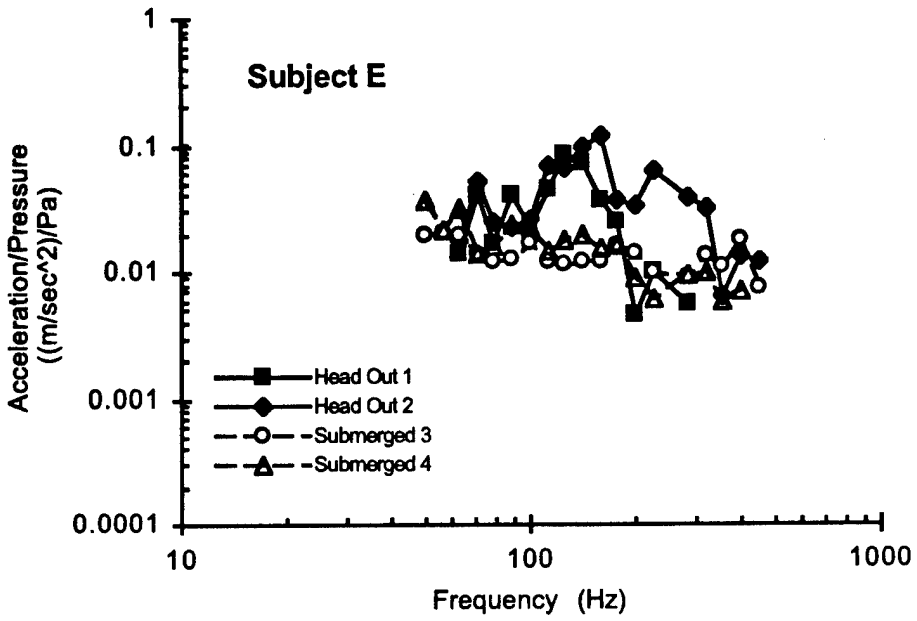

Figure 11. The measured frequency response of the lungs of diver E to underwater sound.

Results from NIVAMS Study

The results from the NIVAMS study were:

1. The NIVAMS was able to measure the motion of the lungs in response to low frequency underwater sound.

2. The measured lung motion differed significantly from the acoustic particle motion.

3. The measured lung motion differed significantly from the motion of the overlying ribs.

4. The response from a recently deceased animal differs significantly from a live animal.

5. For humans at the surface, there is a resonance in the 100 to 200 Hz range.

6. This secondary resonance is of lower amplitude and possible higher frequency when the subject is submerged to a 10 foot depth.

Exposure Study

At the sponsors request, we attempted to determine if 170 dB underwater sound damaged the lungs of pigs. The test involved anesthetizing the animal and submerged it in a vertical orientation to mid-depth (6' 8") in the 32,000 gallon acoustic tank. The test subject was then exposed to the high intensity, low frequency sound for 5 minute periods at discrete frequencies between 100 and 400 Hz. The subject was then removed from the water, sacrificed, and necropsied to determine the effects of the exposure. Table 2 summarizes the progression of this task involving the use of 18 animals.

The anesthetic regimen chosen and the ventilation management of the animal produced unexpected complications and had a strong effect on the outcome of some of the tests [Table 3]. Several animals suffered respiratory alkalosis due to improper ventilation management. Other animals have suffered hypotension, exacerbated by immersion in the water. Blood pressure management by eliminating the vasodilators given to the animals along with the initial sedatives and other measures helped minimize the hypotension. Both respiratory alkalosis and hypotension could have been responsible for the appearance of edema in the lungs of the animals. Unfortunately, initial damage to lung tissue from sound exposure may also be identified by the appearance of interstitial or alveolar edema.

Three animals were exposed to underwater sound while under anesthesia using pentobarbital. During the exposure, the animals had normal cardiovascular and acid-base status. The lung tissue appeared normal after exposure.

Table 2. Progression of Exposure Study

Exp #	Date	Pig #	Test	Survival	Anesthesia Record	Pathology Report	Blood Gas	Blood Tests	EKG Traces	Comments
1	Feb 28	1114	control	yes	X	-	X	-	-	1st PUBA diving pig (7 ft deep); no necropsy.
2	Mar 3	1115	sound	no	X	X	X	X	X	Died on extraction from tank after exposure. Pathology indicated respiratory alkalosis (high blood pH) and hypotension during experiment. Sections from lung were normal.
3	"	1116	control	yes	X	X	X	X	X	Pathology indicated respiratory alkalosis and hypotension during experiment. Sections from lung were normal.
4	Mar 8	1119	-	-	-	-	-	-	-	Terminated before immersion - gastric distress.
5	"	1120	-	-	-	-	-	-	-	Terminated before immersion - high blood pH.
6	Mar 9	1117	-	no	X	X	X	X	-	Died during descent to depth. Pathology indicated severe hypotension. Portions of the lung were atelectatic. No significant gross or histological lesions were observed.
7	Mar 10	1148	-	-	X	X	X	-	-	Terminated before immersion - high blood pH.
8	Mar 14	1150	-	no	X	X	X	X	-	Died during head out immersion. Pathology indicated cardiovascular failure from bradycardia and hypotension.
9	Mar 16	1149	control	yes	X	X	X	X	-	Pathology showed the right lung lobes contained approximately 20 petechia scattered over the pleural surface with a majority of the hemorrhages occurring in the middle lobe. The cause of the pulmonary hemorrhage was unknown.
10	Mar 17	1147	-	no	-	-	-	-	-	Died during intubation.

Exp #	Date	Pig #	Test	Survival	Anesthesia Record	Pathology Report	Blood Gas	Blood Tests	EKG Traces	Comments
11	Mar 18	1154	sound	no	X	X	X	X	X	Died on extraction from tank after exposure. Extraction from tank was performed incorrectly. The animal was exposed to an excessive respiratory pressure (40 cm water) for several minutes during and after extraction. Pathology indicated congestion and edema in the lung lobes.
12	Mar 21	1155	sound	yes	X	X	X	X	X	Pathology indicated severe pulmonary edema involving all the lung lobes. The edema may have been due to early decompensation of cardiac output with decreased stroke volume causing increased pulmonary capillary hydrostatic pressure and fluid leakage.
13	Mar 22	1156	-	-	-	-	-	-	-	Terminated before immersion - poor experimental subject.
14	Mar 28	1167	-	-	-	-	-	-	-	Terminated at surface - arterial line not patent.
15	Mar 31	1211	sound	no	X	X	X	X	X	Died after extraction from tank after exposure, probably due to early extubation. Lung lobes were normal.
16	Apr 4	1213	sound	yes	X	X	X	X	-	Pathology found a small area of acute bronchopneumonia that was present prior to the test. No other abnormalities were observed.
17	Apr 11	1210	-	-						Terminated before immersion - no arterial line.
18	Apr 18	1212	sound	yes	X	X	X	X	-	Pathology found multifocal red foci (3 to 7 mm in diameter on the left lobes. The left and right dorso-caudal lung lobes were dark red to purple. Histopathology was normal. Lab results indicated muscle trauma and stress. The clinicopathological correlation was the sound exposure did not cause significant abnormalities.

Table 3. Effects of Anesthetic Regimen and Underwater Sound on Cardiovascular Status, Acid-Base Balance and Pathologic Changes in the Lungs of Pigs

Anesthetic Regime	N	Experimental Conditions [1]	Cardiovascular Status Normal/Total	Acid-Base Status Normal/Total	Lung Status Normal/Total
Isoflurane	5	A	0 / 3 [2]	1 / 2 [2]	2 / 5 [3]
Isoflurane	2	B	1 / 2	1 / 2	1 / 2 [3]
Isoflurane	3	C	1 / 3	2 / 3	1 / 3 [3]
Pentobarbital	3	C	3 / 3	3 / 3	3 / 3

Notes:

[1] Experimental Condition A - Animals died while submerged or shortly after being prematurely removed from the tank. Not exposed to sound.

Experimental Condition B - Controls. Animals survived sham sound procedures at proper water depth in tank.

Experimental Condition C - Sound-exposed animals survived whole body sound exposure at proper water depth in tank.

[2] Cardiovascular and acid base data not available for 2 animals.

[3] Abnormal condition of the lung consisted of edema in all such cases.

The animals were exposed to sound in a cylindrical water tank, 13 ft 4 in deep by 20 ft diameter. The animals were suspended in the center, above a USRD J-15-3 sound projector. The exposure sound pressure levels were estimated to be from 161 to 177 dB for the frequencies listed. The pressure levels were limited by the source strength and the configuration of the test tank.

Table 4. Sound Exposure Levels and Results for Animals under Pentobarbital Anesthesia

Pig #	Weight (lbs)	Frequency (Hz)	Pressure Level (dB re: 1 µPa)	Exposure Time (min)	Gross Pathology	Histologic Pathology
1211	107	100	161	5	Normal	Normal
		150	166	5		
		200	167	5		
		250	164	5		
		300	175	5		
		400	162	5		
1212	99	100	161	5	Normal	Normal
		150	166	5		
		200	167	5		
		250	164	5		
		270	168	5		
		300	175	5		
		400	162	5		
1213	97	270	177	5	Focal areas mottled red to purple.	Acute broncho-pneumonia present prior to experiment.
		270	177	5		
		270	177	5		
		270	177	5		
		270	177	5		
		270	177	5		

Results from Exposure Study

There was no detectable damage to the lungs from the sound at the frequencies and levels used on the 3 animals exposure while under anesthesia using pentobarbital. This is consistent with a previous study by Sweeney, Duykers, and Percy (1975) [Table 5] at lower frequencies (40 to 80 Hz) and similar sound pressure levels.

Table 5. Sound Exposure Levels and Results from Sweeney, Duykers, and Percy (1975)

Pig #	Weight (lbs)	Frequency (Hz)	Level (dB re: 1 μPa)	Exposure (min)	Side R	Side L	Gross Pathology	Histologic Pathology
1	184	42	144	1.5	-	X	None	-
		70	171	1.5	X	-		
2	163	40	158	6.0	-	X	Blood in	-
		40	158	5.0	X	-	Thorax	
3	160	43	160	12.0	X	-	None	-
		43	160	18.0	-	X		
4	163	48	167.5	12.0	X	-	None	-
		48	167.5	18.0	-	X		

Unresolved Issues

1. Depth dependence of the fundamental resonance. The head out to 10 ft depth change for the study on lung resonance in humans was not adequate to characterize the effects of depth on resonance frequency. Although 10 ft of water represents a 33 % change in ambient pressure, the simplest gas resonator model predicts a change of only 15 % in the resonance frequency. It would be difficult to detect a change of this magnitude from the existing data.

2. Damage threshold as a function of frequency. There was no detectable damage from the exposure conditions used with the small number of test subjects. Therefore, it is impossible to determine damage thresholds.

3. Damage mechanisms. The assumption was made that by the appearance of interstitial or alveolar edema or hemorrhage after exposure would be the most sensitive indicator

of damage (Fung *et al.*, 1988). This assumption was never tested nor was it understood how sound could cause edema.

Direction of Future Research

Although we were able to obtain some useful information from the large animal study, we believe the research should be continued with a small animal model for the following reasons. The relative size of the lung volume to the tank volume compromised the data for the frequency response measurements made on the animals in the small tank (500 gal). The modal response of the small tank changed significantly when the animal was introduced. We were unable to accurately determine the magnitude of the pressure stimulus applied to the lung. Initially we had planned to make the NIVAMS measurements on the animal model in our large acoustic tank (32,000 gal) but were unable due to time constraints. Now we suggest measuring the lung response of a small animal model in the small tank. The use of a small animal should allow characterization lung motion at multiple locations on the same subject to determine if the motion is uniform over the lung surface.

We were unable to generate a high intensity sound stimulus over the frequency range of interest in our large acoustic tank using available acoustic sources. The animals were exposed at maximum levels and showed no signs of damage. Since acquiring a larger source to generate higher sound pressure levels would be expensive, other methods which cause lung motion should be explored. The air filled lungs are thought to respond to low frequency sound pressure by uniform volume expansion and contraction. This volume expansion results in strain of the lung tissue. Instead of generating high levels of sound pressure, volume expansion and compression can be applied directly to the animal. The animal can be placed in a fluid filled, rigid walled chamber with an electrodynamic underwater sound projector flanged to one wall. The volume displacement generated by

the motion of the transducer's piston will be coupled directly to the animal's lungs through the fluid, because the lungs are the most compliant component of the system. Previous NIVAMS measurements provide the transfer function between free field acoustic pressure and volume displacement for the lungs. A volume displacement coupler could allow simulation of high levels of acoustic pressure without the need for a large tank or a high intensity source. The smaller the animal, the higher the level of acoustic pressure.

Small animals are more cost effective for establishing damage thresholds over a wider range of exposure conditions. A large number of animals will be required to reasonably establish damage thresholds. Also a large number of animals are required in the development of the test apparatus and animal management procedures. Keeping an animal alive, healthy, and anesthetized underwater has proven to be a nontrivial task. Not only does the underwater breathing apparatus have to be adapted to a smaller animal, adequate ventilation parameters have to be established for the new animal using the proper monitoring equipment (i.e. blood gas analyzer). In the previous high intensity exposure study, 18 animals were used, but only three valid data points were collected.

Unfortunately, there is a disadvantage in using a small animal model. Since the damage mechanisms are not known a priori, the correct scaling for the mechanics and physiology of the different lung sizes must be determined.

The lung resonance measurements on humans also needs to be extended by testing subjects at higher simulated depths in the OSF. Modelers have predicted that the resonance frequency of the lungs will change with ambient pressure (some say up, others say down). If the resonance frequency of the lungs increases with depth, then the fundamental resonance, which is now out of the band of interest in this study, may move

in band. The sound pressure threshold for lung damage is probably much lower when the lungs are driven at their resonance frequency.

Underwater Sound and the Vestibular System

Although this research program has concentrated on the effects of acoustic pressure, a recent study on the effects of sound exposure on divers indicated that the vestibular system may be stimulated by low frequency underwater sound with potentially adverse results. Vestibular responses to air-borne acoustic stimuli are known as the Tullio phenomenon. Vogel *et al.*, (1986) suggested that the vestibular responses (vertigo, nystagmus-like eye movements) may be due to abnormal excitation of the otolith organs. In a study by Erlich and Lawson (1980), ten otologically normal subjects demonstrated the presence of the Tullio phenomenon to air-borne sound as measured by electronystagmographic testing. Vestibular responses can also be elicited using a vibrator applied to the head (Lackner and Graybiel, 1974).

Acoustic particle motion, rather than acoustic pressure, may be the relevant component of the sound stimulus to the vestibular system. Underwater, the skull is well coupled to the medium in which the acoustic waves propagate, so it moves like the acoustic particle motion. The motion of the otoliths of the vestibular system, acting as inertial masses, will lag behind the motion of their macula (which moves with the skull). This relative motion between the otoliths and their macula can cause temporary or permanent damage to the sensory system. In previous experiments on high intensity sound exposure of fish, the cilia of the hair cells of the macula were sheared off (Enger, 1981; Cox, 1987).

The effect of high intensity acoustic particle motion on the vestibular system can be studied in the following manner. The stimulus can be administered by placing the

animal in a small fluid filled vessel. The entire vessel is then be driven with a shaker to simulate the acoustic particle motion of high intensity free field acoustic pressure. Vestibular function testing, before and after exposure, evaluates the effects of the sound stimulus.

Bibliography

Cox, M. (**1987**). "An experimental investigation of the mechanics of the peripheral auditory system in goldfish," Ph.D. thesis, George W. Woodruff School of Mechanical Engineering, Georgia Institute of Technology, Atlanta, GA.

Cox, M. and P.H. Rogers (**1987**). "Automated noninvasive motion measurement of auditory organs in fish using ultrasound," J. Vib. Acoust. Str. Reliab. in Dsgn. **109**, 55-59.

Enger, P.S. (**1981**). "Frequency discrimination in teleost - central or peripheral?" in *Hearing and Sound Communication in Fishes*, edited by W.N. Tavloga, A.N. Popper, and R.R. Fay (Springer-Verlag, New York), pp. 243-255.

Erlich, M.A. and W. Lawson (**1980**). "The incidence and significance of the Tullio phenomenon in man," Otolaryngol. Head Neck Surg. **88**, 630-635.

Fung, Y.C., R.T. Yen, Z.L. Tao, and S.Q. Liu (**1988**). "A hypothesis on the mechanism of trauma of lung tissue subjected to impact load," J. Biomech. Engr. **110**, 50-56.

Lackner, J.R. and A. Graybiel (**1974**), "Elicitation of vestibular side effects by regional vibration of the head," Aerospace Med. **45**, 1267-1272.

Vogel, P., W. Tackmann, and F.-J. Schmidt (**1986**). "Observations on the Tullio phenomenon," J. Neurol. **233**, 136-139.