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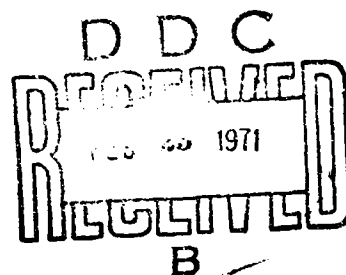
RECOVERY OF THE RESPIRATORY SYSTEM FOLLOWING BLAST INJURY

**Edward G. Damon, John T. Yelverton,
Ulrich C. Luft, and Robert K. Jones**

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**Technical Progress Report
on**

Contract No. DA-49-146-XZ-372



**THIS WORK, A PHASE OF INVESTIGATIONS DEALING WITH
THE BIOLOGICAL EFFECTS OF BLAST FROM BOMBS, WAS SUPPORTED
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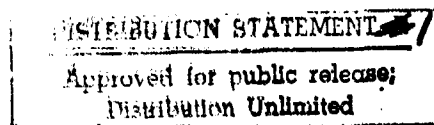
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October 1970



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FOREWORD

This report presents the results of studies of the tempolabile response of the pulmonary system of sheep exposed to blast pressures. The study is a sequel to an earlier work in which the acute effects of blast injury on pulmonary function were investigated.

The findings may be of interest to those involved in the treatment of chest injuries, the analysis of weapons effects, or in industrial or military medicine.

This study is a part of a broad program in the field of blast and shock biology designed to obtain data for use in prediction of hazards from explosions and in the development of a sound basis for the prognosis and treatment of blast injuries.

ABSTRACT

The pattern of recovery of the respiratory system from blast injury was investigated in sheep exposed to overpressures in a shock tube. Measurements of the pH and blood gas tensions, determinations of the venous-admixture (Q_s/Q) and the alveolar-arterial oxygen gradient (A-a) O_2 were conducted before and at intervals up to 132 days following injury. There was an immediate marked increase in Q_s/Q , reduction in PaO_2 , and a moderate increase in (A-a) O_2 , with very little change in the pH or PCO_2 of the arterial blood. The greatest recovery was evident within 24 hours with further gradual improvement seen 2, 7, 14, and 21 days after exposure. After the 21st day, most of the animals exhibited virtual complete recovery of the functional efficiency of the pulmonary system as tested at rest.

ACKNOWLEDGMENTS

The authors wish to acknowledge the able technical assistance of Mr. Charles S. Gaylord and Mr. Glen L. Paxton for conducting the shock-tube exposures and obtaining the pressure-time measurements, Dr. Ernest A. Henderson for veterinary surgical support, Mr. Takeshi Minagawa for the illustrations, and Mrs. Berlinda Martinez for typing the manuscript.

Finally, the senior author would like to express sincere appreciation to Dr. Donald R. Richmond whose consultations and encouragement greatly facilitated the conduct of this study.

The experimental work discussed in this manuscript was conducted according to the principles enunciated in the "Guide for Laboratory Animal Facilities and Care," prepared by the National Academy of Sciences-National Research Council.

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RECOVERY OF THE RESPIRATORY SYSTEM FOLLOWING BLAST INJURY

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INTRODUCTION

Most studies of the physiological effects of blast injury to the lungs which have been conducted to date were designed to assess the relation between the extent of lung injury and the immediate or early changes in pulmonary function.¹⁻⁸ We are not aware of any investigations in which pulmonary function test parameters have been followed for more than 24 hours following the detonation.

Previous reports have indicated that there is usually an increase in rate and reduction in the depth of respiration occurring soon after exposure.³ Furthermore, there is an increase in the venous-arterial shunt which generally correlates with the level of blast lung injury.⁶ The purpose of this study was to extend observations made in the previously cited reference including changes in the pulmonary function in sheep occurring with time after exposure to "sharp"-rising overpressures of "long" duration in the sublethal or near-lethal range.

METHODS

General

Ten young adult ewes having a mean body weight of 40 kg. were exposed singly with their left sides against the endplate of a 42-72-inch-diameter shock tube⁹ to "sharp"-rising reflected pressures ranging from 33 to 45 p. s. i. g. with positive-phase durations of 173 to 228 msec. The ambient pressure at exposure was 12 p. s. i. a.¹⁰

Pulmonary function tests, including determinations of the venous-arterial shunt ($\dot{Q}_s/\dot{Q}$), alveolar-arterial O₂ gradient (A-a)O₂, arterial-alveolar (end-tidal) CO₂ gradient (a-A)CO₂, oxygen tension (PaO₂), carbon dioxide tension (PaCO₂), and pH of the arterial blood, were conducted without anesthesia from 2 to 13 days prior to exposure, on the day of exposure (usually within 1 hour after the blast), at post-exposure intervals of 1 day, 2, 7, 14, and 21 days and, on some of the animals with patent catheters, at irregular intervals up to 132 days post-shot. All animals were sacrificed for studies of the gross pathology at the end of the study or when the arterial catheter became plugged. The pulmonary-function test procedures have been previously described.⁸ Blood samples were drawn from an indwelling arterial catheter inserted through the femoral artery into the aorta.¹¹ The end-tidal CO₂ was measured by a Beckman

Spinco Model LB-1 CO₂ Meter with Linearizer, and the O₂ and CO₂ concentrations of expired gases and calibration gases were determined by the micro Scholander technique.¹² The blood samples were analyzed at 37°C. on an Instrumentation Laboratories Model 113-SI Ultra-Micro pH, P_{O₂}, and P_{CO₂} Blood Analyzing System, and the values were corrected to the species' body temperature (39°C.).

RESULTS

The major results of the study are given in tables 1 through 5 of appendix A and illustrated in figure 1. The changes in venous-arterial shunt ($\dot{Q}_s/\dot{Q}$), expressed as a percent of the cardiac output, are recorded in table 1 of appendix A and illustrated in figure 1(a). The pre-shot mean value was 6.7 with a range of 4.6 to 8.8 percent. Post-shot tests were generally conducted within 1 hour of the exposure, and all animals, except Sheep No. 811, exhibited an increase in $\dot{Q}_s/\dot{Q}$ at this time. The greatest post-exposure recovery was evident on day 1 with further gradual improvement seen on days 2, 7, and 14. By day 14, four of the eight animals tested had $\dot{Q}_s/\dot{Q}$ values within the pre-shot range, whereas the other four still had a residual increase in the venous admixture.

A similar pattern of change was evident from the arterial oxygen tension (PaO₂) data listed in table 2 and illustrated in figure 1(b). There was moderate hypoxia immediately after exposure with progressive recovery during the subsequent 14 days.

The data in tables 3 and 4 indicate that there was little or no change in the mean value of the carbon dioxide tension and pH of the arterial blood in these animals. The data on the alveolar-arterial oxygen difference (A-a)O₂, given in table 5, show a mild increase in the mean value of this parameter which reached its peak on the first post-exposure day and returned to pre-exposure levels after the 21st day. There were no consistent changes in the other pulmonary function test parameters recorded during the study.

DISCUSSION

Lung injuries resulting from exposure to blast pressures would be expected to produce pathophysiological effects which, for some undetermined period of time, would become worse due to the progressive development of pulmonary hemorrhage and edema, then stabilize and begin to improve. The improvement in the functional efficiency of the pulmonary system with time may be a twofold process, including an initial physiological compensation involving a reduction in ventilation of non-perfused alveoli and reduction in blood flow through the vasculature of non-ventilated alveoli. The second, and somewhat slower process of recovery, includes the reversal of pulmonary edema, resolution and clearing of pulmonary hemorrhage, and the progressive repair of damaged tissues. The sharp reduction in venous admixture which occurred in these animals 24 hours after exposure may be largely due to compensatory vasoconstriction reducing

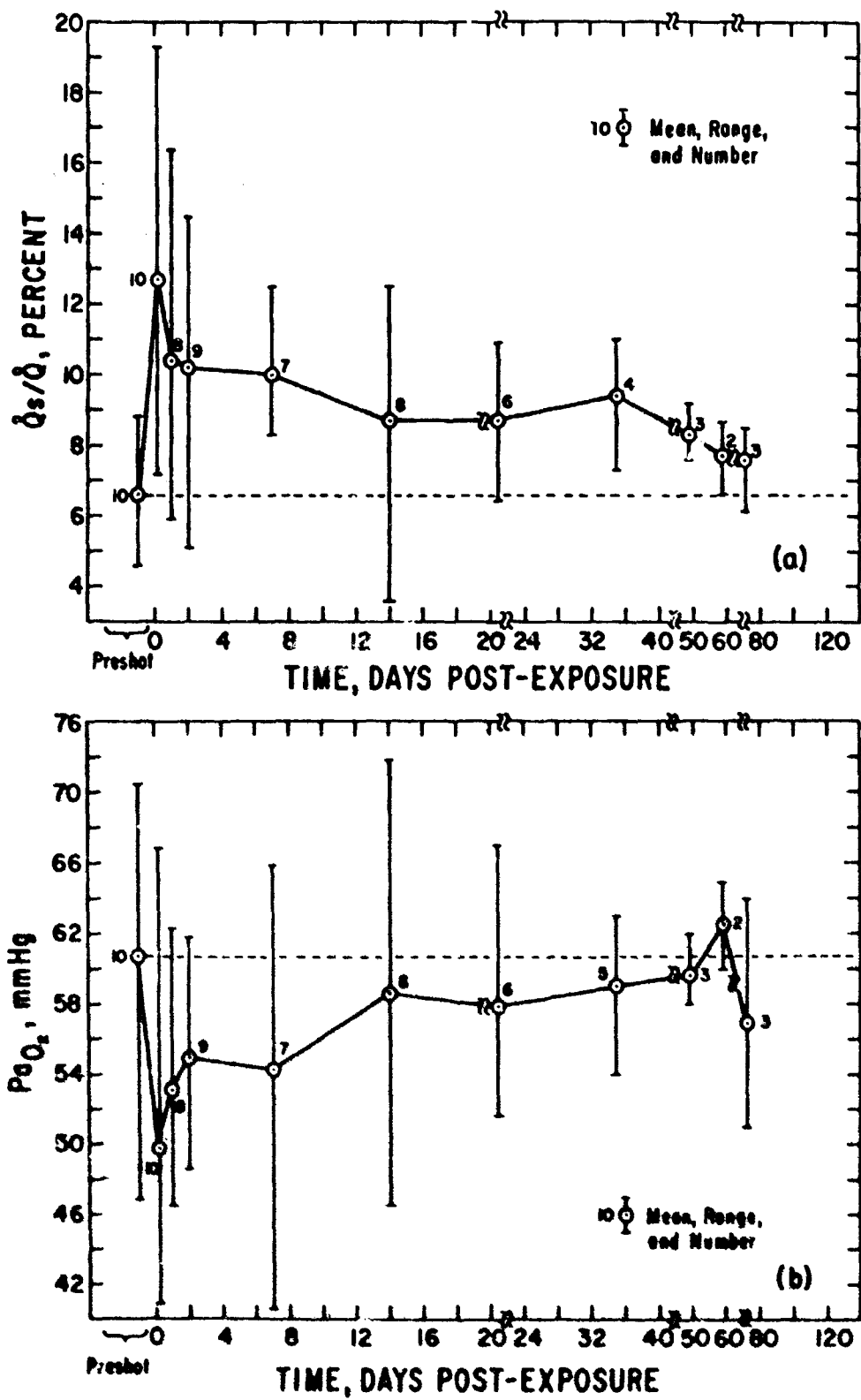


Figure 1. -- Effects of Blast Pressure on Venous-Arterial Shunt ($\dot{Q}_s/\dot{Q}$) and Arterial Oxygen Tension (P_{aO_2}) in the Sheep.

the blood flow through damaged segments of the lung in which the alveolar spaces and airways are filled with hemorrhage and are therefore not open to ventilation; whereas, the slower recovery seen on subsequent days is the result of progressive clearing of hemorrhage, edema, and the restoration of the pulmonary parenchyma through the healing processes. Earlier studies have shown that by 7 days' post-exposure there is marked clearing of the pulmonary hemorrhage, and by 21 days' post-exposure the lungs are usually entirely clear of hemorrhage.¹³ However, one of the sequela of blast injury to the lungs is the occurrence of multiple fibrotic foci, or fine scars, which apparently persist unchanged beyond a period of 2 months.¹³ The ultimate fate and functional significance of such scars is unknown. There was very little to indicate a chronic functional effect of the lung injuries sustained by these animals. Although two of the animals exhibited latent increases in the $\dot{Q}_s/\dot{Q}$ at 94 and 132 days (table 1, Animal Nos. 794 and 771, respectively), the PaO_2 , PaCO_2 , pH, and $(\text{A-a})\text{O}_2$ all were within the normal range (tables 2 through 5). It should be emphasized, however, that the animals in this series were exposed to blast pressures in the sublethal or near lethal range and the results might be far different for those surviving exposure to overpressure at or above the mid-lethal range. Also, the lack of residual effects on the pulmonary function test parameters in this study might be expected because of the tremendous compensatory capacity of the lung, and further resolution of this question must await the use of pulmonary function studies during exercise or other more sensitive tests in the future. Nevertheless, the recovery of the functional efficiency of the pulmonary system within the first 2 to 3 weeks following blast injury is quite remarkable.

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APPENDIX A
TABULATED DATA

TABLE 1
EFFECTS OF BLAST PRESSURES ON
VENOUS-ARTERIAL SHUNT IN THE SHEEP

Sheep No.	Body Wt., kg	Reflected Shock Pressure, psig	Venous Arterial Shunt, Percent																
			Pre-Shot	~1 hr Post-Shot	Days, Post-Exposure														
					1	2	7	14	21	28	35	49	59	66	72	80	86	94	132
825	41	45	5.4	11.1	9.3	9.2	8.3	7.7	7.0										
821	39	42	5.7	14.6	13.4	12.8	12.5	12.5	10.9	11.8	10.1	8.0	8.7	8.0	8.2	5.8	5.8		
771	50	40	5.4	9.9	7.7	7.8		6.5	6.5						8.5				9.3
750	43	40	7.3	14.9		14.5	10.1	7.5											
000	41	40	6.7	9.6															
794	36	38	6.6	12.1	10.7	10.4	9.4	10.0	7.8		11.0	9.2						11.3	
811	43	35	8.2	7.2	5.9	5.1													
715	38	39	8.8	15.8	13.4	13.0	11.4	11.4	10.4		9.0								
646	39	34	7.6	19.3	16.4	13.2	9.8	10.2	7.5		7.3	7.6	6.6		6.1				
570	30	33	5.1	13.8	6.8	5.8	8.5	3.6											
Mean	40	36	6.7	12.7	10.4	10.2	10.0	9.7	8.7		9.4	8.3	7.7		7.6				

TABLE 2
EFFECTS OF BLAST PRESSURES ON
ARTERIAL OXYGEN TENSION IN THE SHEEP

Sheep No.	Body Wt., kg	Reflected Shock Pressure, psig	Arterial Oxygen Tension (Atr), mmHg														
			Pre-Shot	~1 hr Post-Shot	Days, Post-Exposure											94	132
					1	2	7	14	21	28	35	49	59	66	72	80	86
825	41	45	55	48	52	54	56	50	54								
821	39	42	58	50	51	53	52	53	52		63	58	60	63	55	54	67
771	50	40	65	47	53	56		67	59	60					51		60
750	43	40	68	53		58	53	63									
000	41	40	70	55													61
794	36	38	60	49	50	51	51	53	55	54	59						
811	43	35	63	67	62	61											
715	38	34	47	46	48	53	41	47	61	59							
646	39	34	61	41	47	49	62	66	67	61	61	62	65		64		
570	30	33	59	43	61	62	66	72									
Mean	40	38	61	50	53	55	54	59	58	59	59	60	63		57		

TABLE 3
EFFECTS OF BLAST PRESSURES ON
ARTERIAL CARBON DIOXIDE TENSION IN THE SHEEP

Sheep No.	Body Wt., kg	Reflected Shock Pressure, psig	Carbon Dioxide Tension (Air), mmHg														
			Pre-Shot	~1 hr Post-Shot	Days, Post-Exposure											94	132
					1	2	7	14	21	28	35	49	59	66	72	80	86
825	41	45	36	31	32	33	37	32	32								
821	39	42	32	36	31	34	38	33	36		35	39	35	36	33	37	35
771	50	40	29	28	27	31		33	34		34				32		
750	43	40	24	33		30	33	40									
000	41	40	38	32													
794	36	38	36	34	37	35	35	33	32		32	33					31
811	43	35	35	38	27	30											
715	38	34	31	30	26	33	37	33	31		30						
646	39	34	36	37	29	32	33	32	31		34	32	31		36		
570	30	33	32	35	33	33	34										
Mean	40	38	33	33	30	32	35	34	33		33	35	33		34		

TABLE 4

EFFECTS OF BLAST PRESSURES ON
ARTERIAL BLOOD pH IN THE SHEEP

Sheep No.	Body Wt., kg	Reflected Shock Pressure, psig	pH of Arterial Blood (Air)																
			Pre-Shot	~1 hr Post-Shot	Days, Post-Exposure											94	132		
					1	2	7	14	21	28	35	49	59	66	72			80	86
825	41	45	7.47	7.46	7.48	7.42	7.47	7.48	7.39										
821	39	42	7.43	7.43	7.52	7.44	7.45	7.46	7.47		7.41	7.36	7.46	7.57	7.43	7.39	7.40		
771	50	40	7.45	7.41	7.45	7.43		7.51	7.45						7.53			7.41	
759	43	40	7.47	7.49		7.47	7.56	7.48											
000	41	40	7.48	7.46															
794	36	38	7.47	7.55	7.47	7.47	7.41	7.50	7.50		7.51	7.45					7.45		
811	43	35	7.47	7.45	7.44	7.42													
715	38	34	7.47	7.47	7.29		7.47	7.50	7.51		7.42								
646	39	34	7.48	7.47	7.58	7.52	7.46	7.47	7.42		7.42	7.45	7.40		7.44				
570	30	33	7.44	7.45	7.41	7.45	7.41	7.42											
Mean	40	38	7.46	7.46	7.46	7.45	7.46	7.48	7.46		7.44	7.42	7.43		7.47				

TABLE 5

**EFFECTS OF BLAST PRESSURES ON
ALVEOLAR-ARTERIAL OXYGEN DIFFERENCE
(A-a)O₂ IN SHEEP BREATHING AIR**

Sheep No.	Body Wt., kg	Reflected Shock Pressure, psig	Alveolar-Arterial Oxygen Difference (Air), mm Hg																
			Pre-Shot	~1 hr Post-Shot	Days, Post-Exposure														
					1	2	7	14	21	28	35	49	59	66	72	80	86	94	132
825	41	45	42	26	42	28	25	48	34										
821	39	42	33	40	37	32	42	28	40		14	12	19	4	23	29	18		
771	50	40	30	32	33	34		23	31		35				39			23	
750	43	40	35	37		32		40	23										
800	41	40	18	24															
795	36	38	18	44	41	32	42	42	39		39	12					20		
811	43	35	33	19	25	23													
715	38	34	48	42	44	41	37	57	23		33								
646	39	34	22	44	36	48	24	21	24		5	23	18		10				
970	50	33	8	26	22	14	9	15											
Mean	40	38	29	33	35	32	30	34	31		28	16	19		24				

APPENDIX B
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The pattern of recovery of the respiratory system from blast injury was investigated in sheep exposed to overpressures in a shock tube. Measurements of the pH and blood gas tensions, determinations of the venous admixture (Q_v/Q) and the alveolar-arterial oxygen gradient $(A-a)O_2$ were conducted before and at intervals up to 132 days following injury. There was an immediate marked increase in Q_v/Q, reduction in PaO_2, and a moderate increase in $(A-a)O_2$, with very little change in the pH or P_{CO_2} of the arterial blood. The greatest recovery was evident within 24 hours with further gradual improvement seen 2, 7, 14, and 21 days after exposure. After the 21st day, most of the animals exhibited virtual complete recovery of the functional efficiency of the pulmonary system as tested at rest. /		

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