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THE EFFECTS OF INTERMITTENT POSITIVE PRESSURE RESPIRATION ON OCCURRENCE OF AIR EMBOLISM AND MORTALITY FOLLOWING PRIMARY BLAST INJURY

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Prepared for:

Defense Nuclear Agency

January 1973

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HEADQUARTERS
Defense Nuclear Agency
Washington, D. C. 20305

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1. ORIGINATING ACTIVITY *(Corporate author)*

Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico

2a. REPORT SECURITY CLASSIFICATION

UNCLASSIFIED

2b. GROUP

3. REPORT TITLE

The Effects of Intermittent Positive Pressure Respiration on Occurrence of Air Embolism and Mortality Following Primary Blast Injury

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*

Final Report

5. AUTHOR(S) *(First name, middle initial, last name)*

Edward G. Damon, Ernest A. Henderson, and Robert K. Jones

6. REPORT DATE

January 1973

7a. TOTAL NO. OF PAGES

32

7b. NO. OF REFS

16

8a. CONTRACT OR GRANT NO.

DASA 01-70-C-0075

b. PROJECT NO. NWED Code: M

c. Task and Subtask Code: A012

d. Work Unit Code: 03

9a. ORIGINATOR'S REPORT NUMBER(S)

9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*

DNA 2989F

10. DISTRIBUTION STATEMENT

Approved for public release; distribution unlimited.

11. SUPPLEMENTARY NOTES

12. SPONSORING MILITARY ACTIVITY

Director
Defense Nuclear Agency
Washington, D.C. 20305

13. ABSTRACT

Twenty beagle dogs were exposed in pairs to airblast on the endplate of a 42-inch diameter shock tube. One dog of each pair then was given intermittent positive pressure respiration (IPPR) for 2 hours with 100 percent oxygen, and the other dog was maintained on 100 percent oxygen for 4 hours in a hyperbaric chamber at a chamber pressure of 14 p.s.i.a., after which she was given IPPR with 100 percent oxygen for 2 hours. The mortality, time of death, and incidence of arterial air embolism in these two groups then were compared with those of 10 untreated control animals that previously had been exposed to airblast in the same way as those in the treatment groups. The mortality was 60 percent in the untreated control group, 80 percent in the immediate IPPR group, and 50 percent in the delayed IPPR treatment group. There was one case of air embolism (14-minute fatality) in the untreated control group, three cases of air embolism (5-minute, 15-minute, and 22-minute fatalities) in the immediate IPPR group, and none in the delayed IPPR group. The mean survival time for the fatalities was 12.4 hours for the untreated control group, 2.3 hours for the immediate IPPR group, and 9.9 hours for the delayed IPPR group. Thus, the results indicate that the use of IPPR immediately following blast injury may result in an increase in the incidence of air embolism, increase in mortality, and a reduction in survival time; whereas, when used after a delay of 4 hours, IPPR resulted in neither an increase in incidence of air embolism nor in mortality but did result in a shortening of survival time.

DD FORM 1 NOV 65 1473 REPLACES DD FORM 1473, 1 JAN 64, WHICH IS OBSOLETE FOR ARMY USE.

UNCLASSIFIED

Security Classification

Ia

UNCLASSIFIED

Security Classification

14. KEY WORDS	LINK A ROLE	LINK A WT	LINK B ROLE	LINK B WT	LINK C ROLE	LINK C WT
Blast injury						
Blast biology						
Thoracic trauma						
Inhalation therapy						
Intermittent positive pressure respiration (IPPR)						
Pulmonary contusion						
Traumatic air embolism						
Ib						

UNCLASSIFIED

Security Classification

DNA 2989F
January 1973

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This work was supported by the Defense Nuclear Agency
under NWED subtask MA012-03

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Contract No. DASA 01-70-C-0075

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FOREWORD

This report presents the results of initial studies on the effects of inhalation therapy in the treatment of blast injury in the beagle dog. Specifically, the effects of administering intermittent positive pressure respiration (IPPR) either immediately following blast exposure or after a delay of 4 hours were explored. The effects of the two modes of treatment on mortality and the occurrence of arterial air embolism compared to that of untreated, blast-injured control animals were investigated.

The findings may be of interest to those involved in the treatment of thoracic trauma, 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 the prediction of hazards from explosions and in the development of a sound basis for the prognosis and treatment of blast injuries and related trauma.

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.

ACKNOWLEDGMENTS

The able technical assistance of Mr. Ben Jojola III and Mr. John Viney, the assistance of Mrs. Berlinda Martinez by typing and helping to edit the manuscript, and the assistance of Mr. Emerson Goff for the illustration are gratefully acknowledged.

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INTRODUCTION

Injuries produced as a direct result of exposure of the body to a pressure pulse emanating from a detonation or from some other blast source are referred to as primary blast injuries (reference 16). Such injuries are seen most often in organs where there are differences in density of adjacent tissues and are especially evident in gas-containing structures (references 1 and 16). Hence, the lungs are very susceptible to blast injury, and many of the pathophysiological effects of blast injury can be traced directly or indirectly to pulmonary injuries (references 1 through 5, 8, 9, and 16). There is usually extensive pulmonary hemorrhage, and those that die within a few minutes after exposure to a blast wave frequently exhibit arterial air emboli (references 1, 6, and 16). The air bubbles evidently gain access to the circulation through the disrupted tissues of the blast-injured lungs and may contribute to early lethality if they involve the coronary or cerebral circulation. Those with blast injuries frequentlv exhibit respiratory distress. In such cases, the use of some form of oxygen therapy is mandatory and the use of a respirator to assist the ventilation seems desirable. However, it has been pointed out previously that the use of positive pressure inhalation therapy may be contraindicated because of the risk of injecting additional quantities of air into the circulation, thus adding to the hazards of air embolism (reference 16). There is also the hazard that full expansion of the blast-injured lung may result in an increase in the extent of the pulmonary hemorrhage, and if alveoli have ruptured into the plural space this may allow formation of a tension pneumothorax. In spite of these theoretical objections to the use of positive pressure ventilation, this continues to be an important mode of therapy in the treatment of traumatic thoracic injuries (reference 13), and the extent of the risks involved previously has not been investigated experimentally.

The purpose of this study was to assess the effects of intermittent positive pressure respiration (IPPR) when administered to beagle dogs immediately after blast injury or after a delay of 4 hours following exposure to air blast, with special reference to the mortality and the incidence and extent of air embolism in these animals as compared with that of untreated blast-injured controls.

METHODS

A total of 20 female beagle dogs having a mean body weight of 9.8 kg with a range of 8.5 to 11.25 kg and an age of 1 to 3 years were utilized in this study. The animals were grouped in pairs on the basis of age and were exposed in pairs on shelves on the endplate of a 42-inch diameter shock tube to reflected shock waves. One dog of each pair then was given treatment for 2 hours with IPPR with 100 percent O_2, and the other dog was placed in a hyperbaric chamber and treated for 4 hours with 100 percent oxygen at a chamber pressure of 14 p.s.i.a. (note that this pressure was approximately equivalent to that of ambient pressure at sea level), after which she was placed on the respirator and IPPR was administered for 2 hours with 100 percent oxygen. To administer IPPR to the animals, a cuffed endotracheal tube was placed without anesthesia and the trachea then was suctioned to remove excess blood, after which the endotracheal tube was connected to a Bird Mark VII pressure-cycled respirator. The end-inspiratory pressure was set at 50 cm H_2O, and the machine was set to ventilate the animal in accordance with its own inspiratory demands. In addition to these animals, 10 similar female beagle dogs, which previously had been exposed to airblast in connection with a previous study in the same manner as those in the current study, were utilized as untreated controls (reference 10). The animals that died were examined grossly soon after death, and the survivors were sacrificed 7 days after injury and similarly examined.

Air Blast Exposures

The 42-inch diameter air-driven shock tube and pressure-time instrumentation utilized in these studies previously have been described (reference 14). The pressure-time waveform was recorded from two piezoelectric pressure transducers mounted side-on in the walls of the shock tube, 3 inches and 6 inches upstream from the endplate directly above the backs of the animals. The same compression chamber pressure (85 p.s.i.g.) was utilized in this study as in a previous study from which the untreated control data were taken. The ambient pressure at exposure was 12 p.s.i.a. (reference 11), and the duration of the positive phase of the reflected shock wave was ~140 msec (reference 15).

The animals were placed on shelves on the endplate and secured by means of harnesses constructed of cotton webbing, with their left sides pressed tightly against the endplate of the shock tube. Thus, the overpressures to which the animals were exposed were "sharp-rising" overpressures of "long" duration (reference 15).

RESULTS

The mean reflected shock pressure to which the animals were exposed was 49.9 p.s.i.g., with a range of 49.2 to 51.3 p.s.i.g. The mean reflected shock pressure to which the untreated controls were exposed was 50.8 p.s.i.g., with a range of 50.1 to 51.8 p.s.i.g. Thus, there was a small difference (0.9 p.s.i.g.) between the mean pressure to which the controls and the treated animals were exposed ($.01<p<.025$), even though the compression chamber pressure of the shock tube was the same (85 p.s.i.g.) in each group.

Table 1 lists the reflected shock pressure to which the animals were exposed on each shot, gives the time of death of each fatality, and also indicates the presence or absence of arterial air embolism at time of death. The mortality was 6/10 (60 percent) in the untreated control group, 8/10 (80 percent) in the immediate IPPR group, and 5/10 (50 percent) in the delayed IPPR treatment group. In the untreated control group, one animal exhibited arterial air embolism (14-minute fatality). In the immediate IPPR group, three animals with times of death of 5 minutes, 15 minutes, and 22 minutes had arterial air embolism. Two of these animals (5-minute and 22-minute fatalities) had greater quantities of intravascular air than that seen in the untreated control. These animals had air bubbles scattered throughout the arteries of the mesenteries, in the walls of the diaphragm, in the coronary and cerebral arteries, and in the pulmonary veins. None of the animals given IPPR treatment after a delay of 4 hours exhibited air embolism. The mean survival time for the fatalities was 12.4 hours in the untreated control group, 2.3 hours in the immediate IPPR group, and 9.9 hours in the delayed IPPR group.

Figure 1 further illustrates that in the two treatment groups most of the deaths occurred soon after termination of the IPPR treatment. In the immediate IPPR group, four animals died while still on the respirator. This includes the three animals that died with air embolism, as indicated above, plus one animal that died 2 hours and 5 minutes following blast exposure near the end of the treatment period. No intravascular air could be detected in this animal, and the extent of the pulmonary hemorrhage and edema (as indicated by the lung weight

TABLE 1.--The effects of immediate IPPR treatment, delayed IPPR treatment, or of no treatment on occurrence of air embolism and survival times of beagles exposed to airblast

Test No.	Reflected Shock Pressure, p.s.i.g.	Immediate IPPR Treatment			Delayed IPPR Treatment		
		Time of Death	Air Embolism	Lung Wt., % of Body Wt.	Time of Death	Air Embolism	Lung Wt., % of Body Wt.
1	49.4	3 hr	-	3.54	20 hr	-	NR**
2	49.1	5-1/2 hr	-	3.99	10 hr	-	NR**
3	49.4	4 hr 50 min	-	3.62	6 hr 15 min	-	4.18
4	49.3	22 min	Massive	2.65	7 hr 6 min	-	2.97
5	51.2	15 min	Moderate	3.08	S*	-	1.63
6	51.3	5 min	Massive	2.32	S*	-	1.51
7	49.3	S*	-	1.52	S*	-	1.29
8	49.6	2 hr 5 min	-	3.24	S*	-	1.73
9	50.7	2-1/2 hr	-	3.73	S*	-	1.80
10	49.3	S*	-	1.78	6 hr 10 min	-	3.40
Mean	49.9	2.3 hours			9.9 hours		
Range (49.2 to 51.3)							

*S Denotes survived until sacrificed 7 days postshot.
**NR Denotes not recorded because of postmortem decay.

TABLE 1. --(Continued)

Test No.	Reflected Shock Pressure, p.s.i.g.	Immediate IPPR Treatment		
		Time of Death	Air Embolism	Lung Wt., % of Body Wt.
Untreated Controls:				
1	51.8	S*	-	1.32
2	51.8	14 min	Extensive	3.47
3	51.0	24 hr 25 min	-	4.52
4	50.2	1-1/2 hr	-	2.56
5	51.4	26-1/2 hr	-	3.57
6	50.8	20 hr	-	NR**
7	50.4	S*	-	1.66
8	50.3	S*	-	1.44
9	50.1	S*	-	1.67
10	50.2	1 hr 47 min	-	3.89
Mean	50.8	12.4 hours		
Range (50.1 to 51.8)				

*S Denotes survived until sacrificed 7 days postshot.
**NR Denotes not recorded because of postmortem decay.

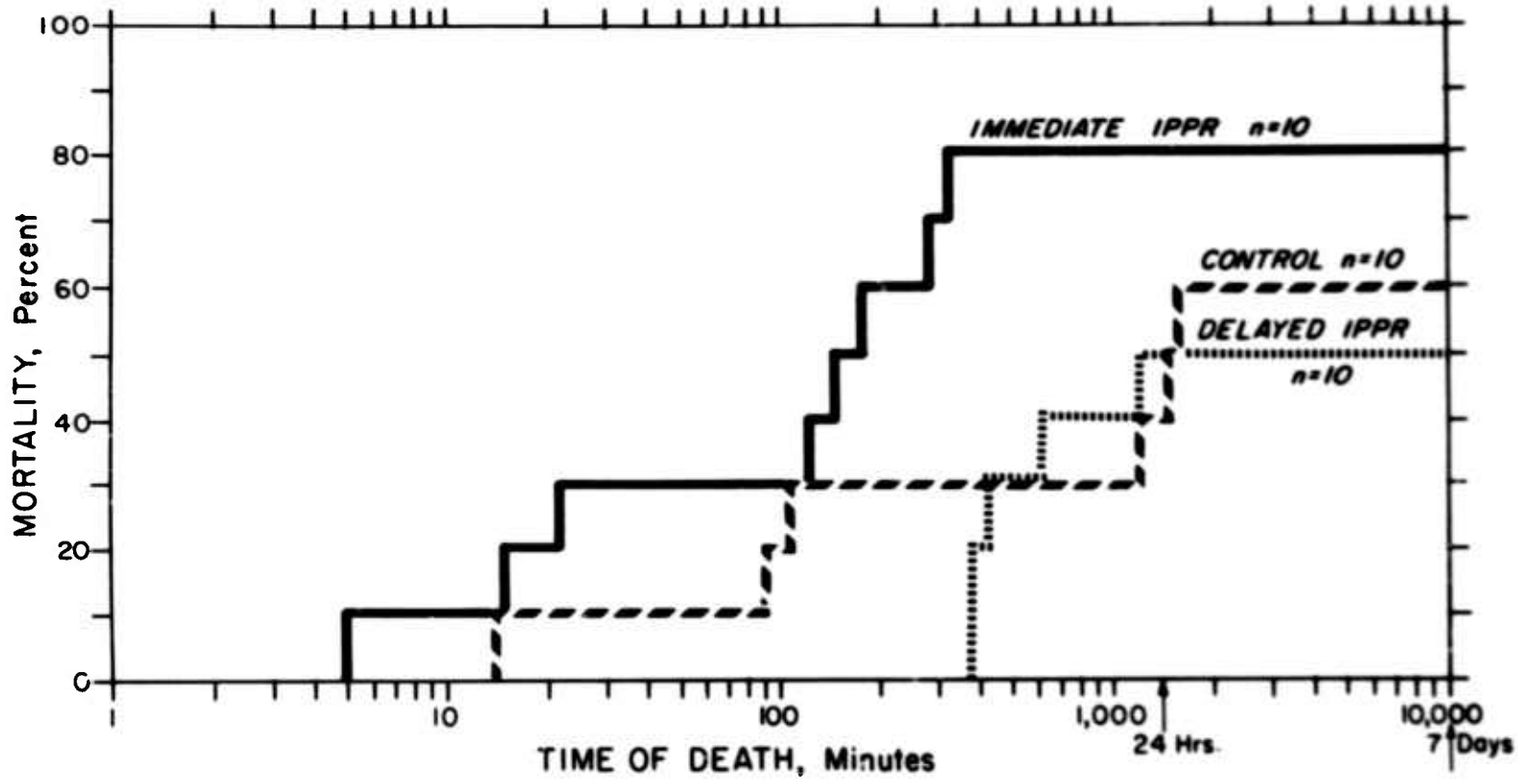

Figure 1. --Death distribution of beagle dogs receiving IPPR immediately following exposure to air blast, or after a delay of 4 hours as compared to that of untreated, blast-injured controls.

data presented in table 1) was somewhat less than that seen in the animals that died at later periods of time. The remaining four fatalities in this group died at times ranging from a few minutes to a few hours after removal from the respirator. In the delayed IPPR treatment group, none of the animals died during the 4-hour delay period during which time they were breathing 100 percent oxygen in a chamber at a pressure of 14 p.s.i. absolute, and none died while on the respirator. The fatalities in this group died within a few minutes to a few hours after removal from the respirator.

The lung weight data presented in table 1 reflect the extent of the pulmonary hemorrhage and edema in each animal. Animals in all groups that died a few hours after injury generally exhibited confluent hemorrhage of more than 90 percent of the entire lung, whereas those that died within a few minutes exhibited a less extensive development of pulmonary hemorrhage and edema and, thus, generally lighter lung weights than in those that died at later time intervals. The survivors exhibited a remarkable clearing of the pulmonary hemorrhage when sacrificed 7 days after injury. The mean lung weight, expressed as a percentage of the body weight, for the survivors in all three groups was 1.58 percent as compared with a mean value of 3.42 percent for the fatalities from all three groups. These data may be compared to a mean value of 0.77 percent for 116 beagle controls which were sacrificed without exposure to trauma (reference 7).

DISCUSSION

In order to assess the possible hazards of IPPR treatment following blast injury, the end-inspiratory pressure purposely was set at a somewhat higher level (50 cm H_2O) than that usually employed in inhalation therapy. Although the differences in mortality and incidence of air embolism between the groups were not statistically significant, the results indicate that IPPR when administered soon after blast injury may promote both the development of arterial air embolism and an increase in intrapulmonary hemorrhage as a result of distention of the lung, thus leading to early lethality or death soon after termination of the treatment. It may be that the animals in the immediate IPPR group that exhibited air embolism had some air emboli before being placed on the respirator and that the effect of IPPR was to increase the quantity of air passing through pre-existing alveolovenous or bronchovenous fistulae. The fact that none of the animals in the delayed IPPR treatment group had air emboli would seem to indicate that positive pressure respiration at the pressure utilized in this study (50 cm H_2O) neither promotes the formation of new fistulae through which air can pass into the circulation nor reopens pre-existing fistulae providing it is administered after a delay of 4 hours. However, the fact that the fatalities in the delayed IPPR group died with times of death shorter than that of the untreated controls may indicate that IPPR when administered 4 hours after injury still may promote the development of pulmonary hemorrhage and edema as a result of distention of the blast-injured lung and result in death soon after removal from the respirator. Furthermore, the use of IPPR at the pressure utilized here may have resulted in an increase in the pulmonary arterial pressure, an effect that scarcely could be tolerated by an animal whose pulmonary vascular resistance was already elevated as a result of blast injury to the lungs.

Previous studies have indicated that the use of hyperbaric oxygen therapy results in an increase in the survival and recovery of guinea pigs, rabbits, and beagles following blast injury (references 10 and 12). Thus, in areas where hyperbaric facilities are available, the treatment of choice for blast injury may be the use of hyperbaric oxygen therapy with spontaneous respiration rather than

the use of a respirator. The results of the current study suggest that, where hyperbaric facilities are not available, 100 percent oxygen with spontaneous respiration should be used initially to alleviate the hypoxia of the blast-injured patient. However, if this approach fails to maintain a P_{O_2} near the normal range, positive pressure ventilation may be employed later. Regardless of the type of respirator utilized (volume-cycled or pressure-cycled), the machine should be set to deliver oxygen at the lowest possible positive pressure that can be used to maintain the P_{O_2} and P_{CO_2} of the arterial blood within the normal range in order to avoid over-distention of the injured lung. It may be that the duration of IPPR treatment (2 hours) employed in the current study was too short and that the positive pressure (50 cm water) was too high to result in therapeutic benefits to the animals.

In view of the wide-scale use of respirators in the treatment of thoracic trauma and in view of the results of the current study, future investigations should be conducted to determine the optimum delay before use of positive pressure respiration, the optimum duration of treatment, and the optimum pressure level to be employed. Also, the effects of continuous positive pressure respiration in the treatment of thoracic trauma should be explored.

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