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AD NUMBER

ADA800326

CLASSIFICATION CHANGES

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FROM:

AUTHORITY

Chemical Corps Medical Labs, Army Chemical
Ctr ltr, 29 Jan 1954; US Army Edgewood
Arsenal ltr, 1 Feb 1973

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CHEMICAL CORPS
MEDICAL DIVISION
ARMY CHEMICAL CENTER
MARYLAND

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

by

John A. Zapp, Jr.

APR 12 1951

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FOREWORD

This series of reports summarizes war investigations carried out by Medical Division in collaboration with Division 9 of the National Defense Research Committee. While primarily related to flame warfare the lessons learned should prevent or reduce the injuries incident to many types of major fires. In order to make the results of these investigations more readily available, Dr. John E. Zapp, Jr. was engaged to put the reports in more popular form and to eliminate all material having a classification higher than Restricted. He is eminently qualified for this task having been stationed as a U.S.P.H.S. officer at Medical Division during the war with responsibilities for these studies. He is now Assistant Director, Haskell Laboratory of Industrial Toxicology, E.I. du Pont de Nemours & Company, Wilmington, Delaware.

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THE TOXICOLOGY OF FIRE

PART I. Assessment of Toxic Factors Created by Fire.

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ARMY CHEMICAL CENTER, CHEMICAL CORPS, MEDICAL DIV., WD.
(MEDICAL DIVISION SPECIAL REPORT NO. 4)

THE TOXICOLOGY OF FIRE

JOHN A. ZAPP, JR. APRIL '51 109PP. TABLES, DIAGRS, GRAPHS, DRUGS

MILITARY OPERATIONS (24) WARFARE, FLAME
STRATEGY AND TACTICS (6) FLAME THROWER ATTACKS - CASUALTIES

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Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART I. Assessment of Toxic Factors Created by Fire.

SUMMARY

The military importance of flame attack on fortified Japanese positions in the Pacific theater led the Medical Division, Army Chemical Corps, in cooperation with the National Defense Research Committee, to undertake an investigation into the casualty producing factors in flame attack.

Early pilot experiments left some doubt as to whether heat or a toxic atmosphere was the primary lethal factor in killing men subjected to flame attack in enclosed fortifications when the victims showed no visible signs of surface burning. The experiments first carried out by the Medical Division were designed to measure quantitatively and continuously before, during and after flame attack the air temperature and the concentration of various gases in the atmosphere (O_2 , CO, CO_2 , and hydrocarbons) which might contribute to the production of casualties. The methods finally adopted are described.

Following the initial experiments on a poorly ventilated Japanese type bunker, experiments were conducted on other structures of different ventilation characteristics, and on targets in the open.

In order to correlate the environmental changes recorded by the measuring instruments with their physiological effects a certain number of goats were exposed under rigid medical supervision and pertinent physiological observations were made. In a further effort to elucidate the relative importance of the various factors, animals were exposed to the effect of the separate factors, singly and in combinations of two or more. Problems of protection against flame attack were also studied.

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART I. Assessment of Toxic Factors Created by Fire.

I. HISTORICAL

While flame warfare played but a small part in operations carried out by United States forces in the European theater of World War II, its role in the Pacific theater assumed increasing importance to United States forces as they proceeded with the attack and reduction of strongly fortified Japanese island positions. Japanese fortifications were often so constructed as to be almost impregnable against attack with artillery or mortar shell, aerial bombs, grenades, or small arms. It was discovered empirically that flame throwers were often effective in killing the occupants of such fortified positions. In the meanwhile, flame warfare was being used extensively by Canadian forces in the Northwest European theater, and somewhat less extensively by British forces. It was noticed in both the European and Pacific theaters that the occupants of fortifications subjected to flame attack were sometimes killed without visible evidence of being burned.

It was important from the military point of view to discover what factors other than the direct effect of contact with burning fuel were responsible for killing the occupants of fortifications attacked with flame. Hence the primary problem was to determine what lethal factors other than its incinerating effect might be present in petroleum fuel burning inside a closed or partially closed structure.

An intensive effort was made in the field to determine the cause of death in the unburned casualties from flame attack. A pathology team from No. 1 Canadian Mobile C. W. laboratories accompanied several infantry operations in which flame throwers were used. Canadian medical services were alerted to the problem as well. The Medical Division, Army Chemical Corps, sent Captain George C. Ham, M.C., to the Pacific theater for the purpose of gathering similar data. It was found impossible, however, in either the European or Pacific theater to establish conclusive evidence as to cause of death because the chaotic conditions of the battlefield necessitated too long a delay before the bodies could be carefully examined, or because incidental damage to the bodies from other weapons after the flame attack rendered the cause of death doubtful. After two Canadian officers were wounded while studying the effect of flame attack under fire, it was decided that the problem could best be studied in experimental establishments at home.

Pilot experiments revealed that burning fuel discharged from a flame thrower into a poorly ventilated fortification was not always completely combusted within the fortification. It was therefore easy to postulate the presence of carbon monoxide as a possible lethal factor. Along with

carbon monoxide one would expect to find carbon dioxide, a deficiency of atmospheric oxygen, hot air, particulate matter (oily smoke), and possibly steam. In addition to these there might be present irritant gases resulting from the partial decomposition of petroleum fuel.

The relative importance of these factors was unknown. It was, therefore, decided to attempt to discover the nature of the lethal factors actually present in flame attack, and to evaluate their relative importance. Since poorly ventilated Japanese fortifications were at the moment of primary military interest, initial interest centered on study of the effects of petroleum fires in poorly ventilated structures. Subsequently the scope of the investigations was enlarged to cover the lethal factors involved in flame attack of moderately and well ventilated positions.

The study of the lethal factors involved in flame attack was initiated in this country in the spring of 1944 when Dr. Alan R. Moritz and co-workers of Harvard University, working under contract with Division 9, National Defense Research Committee, agreed to carry out a series of pilot experiments. At about the same time, the Canadian government also undertook a series of experiments involving the flame attack of a model Japanese bunker.

There was immediately available for Dr. Moritz's work certain facilities of the Factory Mutual Experiment Station at Norwood, Mass. A concrete fireproof room of 600 cu.ft. capacity, which could be made nearly airtight, was chosen for the first experiments. Gasoline was placed in shallow pans, which completely covered the floor of the room, and was ignited by an electric spark. Samples of the atmosphere were collected in evacuated flasks through long tubes leading through the wall of the conflagration chamber. Air temperatures were taken in several locations in the chamber by means of continuously recording sensitive thermocouples.

The pilot experiments indicated that when the conflagration chamber was poorly ventilated, the gasoline fire was quickly extinguished by lack of oxygen, but that in no case did the oxygen concentration in the atmosphere drop below 14%, the carbon monoxide concentration exceed 1% or the carbon dioxide concentration exceed 4%. Air temperatures as high as 900°C. were recorded but were not maintained above 400°C. for more than 20 seconds. In conflagrations where the chamber was ventilated, the gasoline burned completely and there was no evidence of significant oxygen depletion or development of significant amounts of carbon monoxide or carbon dioxide in the atmosphere.

On the basis of these pilot experiments, Moritz and his co-workers concluded that heat was probably the most important casualty producing factor in gasoline conflagrations. They accordingly directed their efforts to an investigation of the time-energy relationships in the production of casualties by exposure to heat. Much of this work has now been published (1,2,3,4,5,6,7,8,9) and will not be described in detail in this report.

At approximately the same time as the Moritz experiments, the Canadian government carried out a series of experiments involving flame thrower attack of a model Japanese bunker at the Suffield (Alberta) Experiment Station. In the course of these studies goats were placed in the bunker in such a position that they were shielded from the flame itself. Deaths occurred in some of these goats, and in the majority of those succumbing a lethal concentration of carbon monoxide was found in the blood (10,11).

The Canadian data indicated that the products of combustion might play a more important role as a lethal factor in flame attack than had been suspected on the basis of Moritz's pilot experiments.* Because of unfavorable meteorological conditions at the Suffield Experiment Station, it was agreed that the Medical Division, Army Chemical Corps, with the support of Division 9, National Defense Research Committee, should undertake an intensive investigation into the lethal factors involved in flame attack of enclosed fortifications. A Flame Attack Section, of the Toxicological Research Laboratory, Medical Division, Army Chemical Corps, was accordingly set up in the summer of 1944 under the command of Captain Boris B. Rubenstein, M.C., and subsequently of Captain George C. Han, M.C., and of Captain L. B. Hobson, M.C. As there was immediately available at Edgewood Arsenal, Maryland, a replica of the Japanese bunker which had been used at Suffield (Fig. 1) it was decided to utilize it for the first experiments.

At the same time, a study of the construction details of captured Japanese and German fortifications was carried out by the author, and as a result, four different types were reproduced at Edgewood Arsenal (Figs. 2,3,4, and 5). The diversity of the types permitted considerable variation in the conditions attendant to the flame attack.

It was decided that the first objective of the Edgewood group should be to obtain quantitative information concerning the physical and chemical changes occurring inside a bunker subjected to flame attack. Interest was initially centered in the changes taking place in locations sheltered from the effects of direct flame. The measurements which were considered most important were as follows:

1. Air temperature and calorific bombardment at various locations in the bunker.
2. Oxygen, carbon monoxide, and carbon dioxide content of air within the bunker.
3. The concentration of unburned hydrocarbons and other gases developed within the bunker.

*The discrepancy in the results of Moritz and of the Canadians turned out to be due to the difference in combustion characteristics of petroleum fuel statically ignited in an enclosed building and burning petroleum fuel injected into such a building.

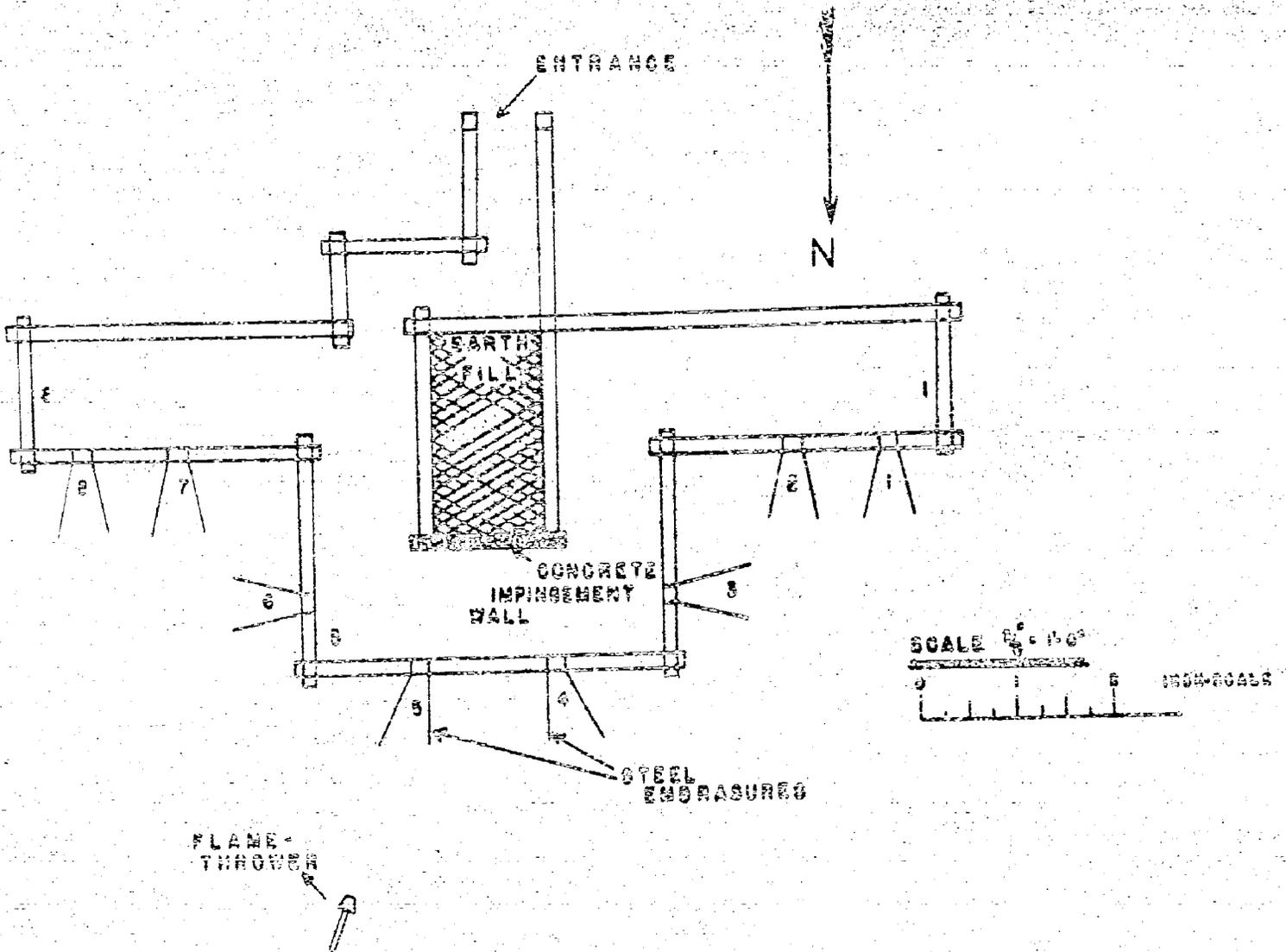
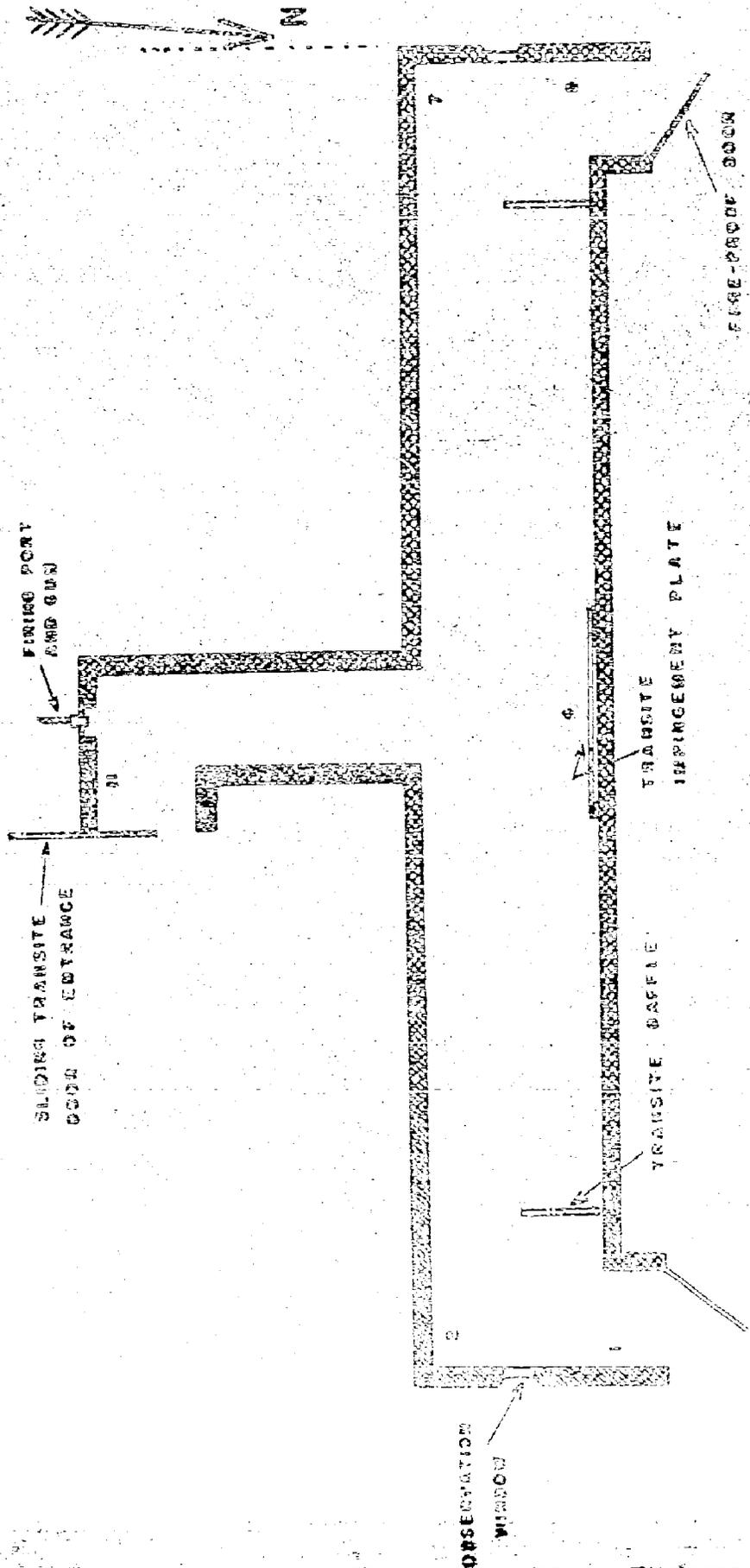


FIGURE 1.- JAPANESE-TYPE "B" BUNKER
 "B" FIELD
 EDGEWOOD ARSENAL, MD.

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SCALE 1/4" = 1'

FIG. 2 - JAPANESE-TYPE AIR RAID SHELTER
KING'S CREEK AREA
EDGEWOOD ARSENAL, MD.

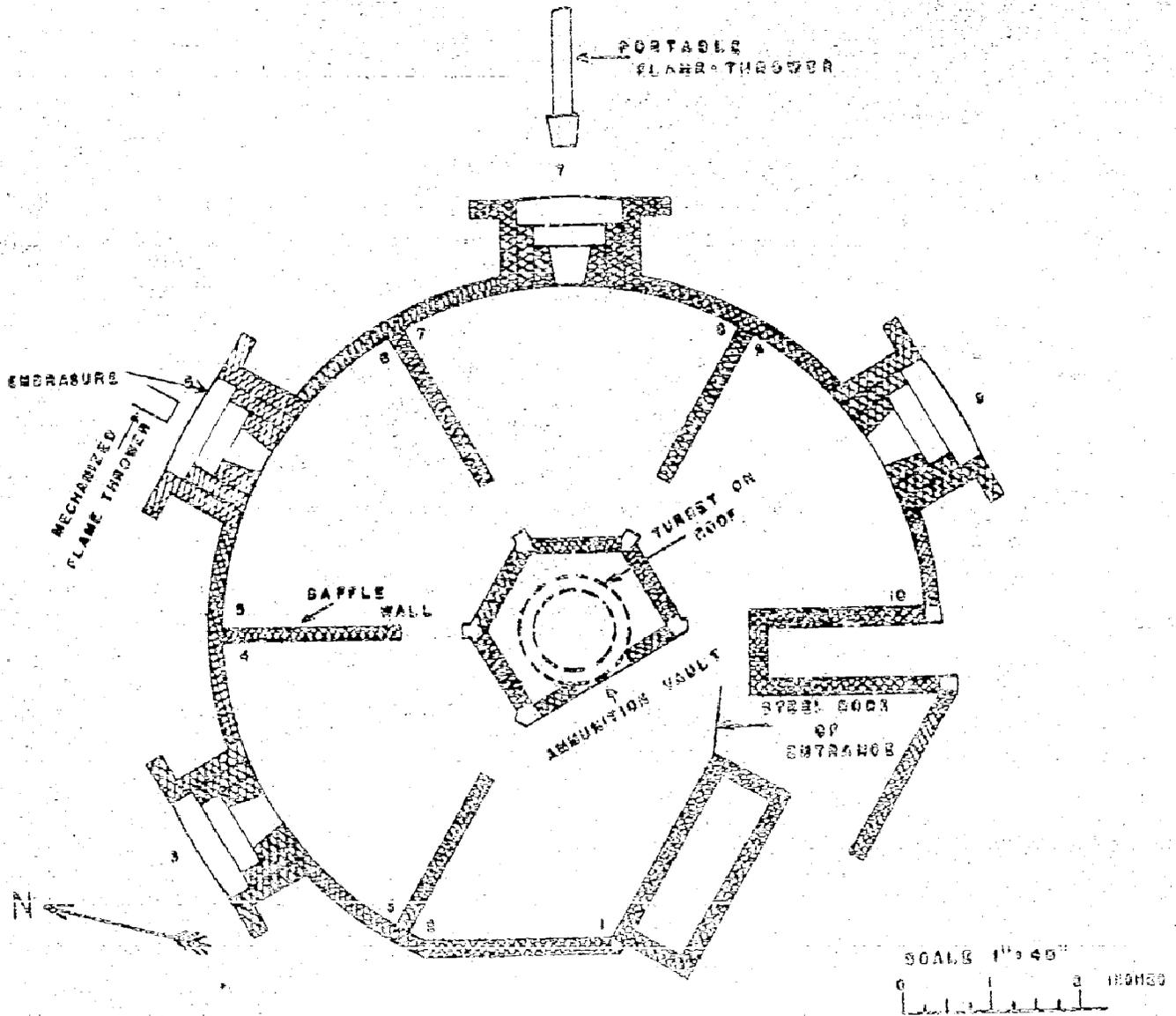
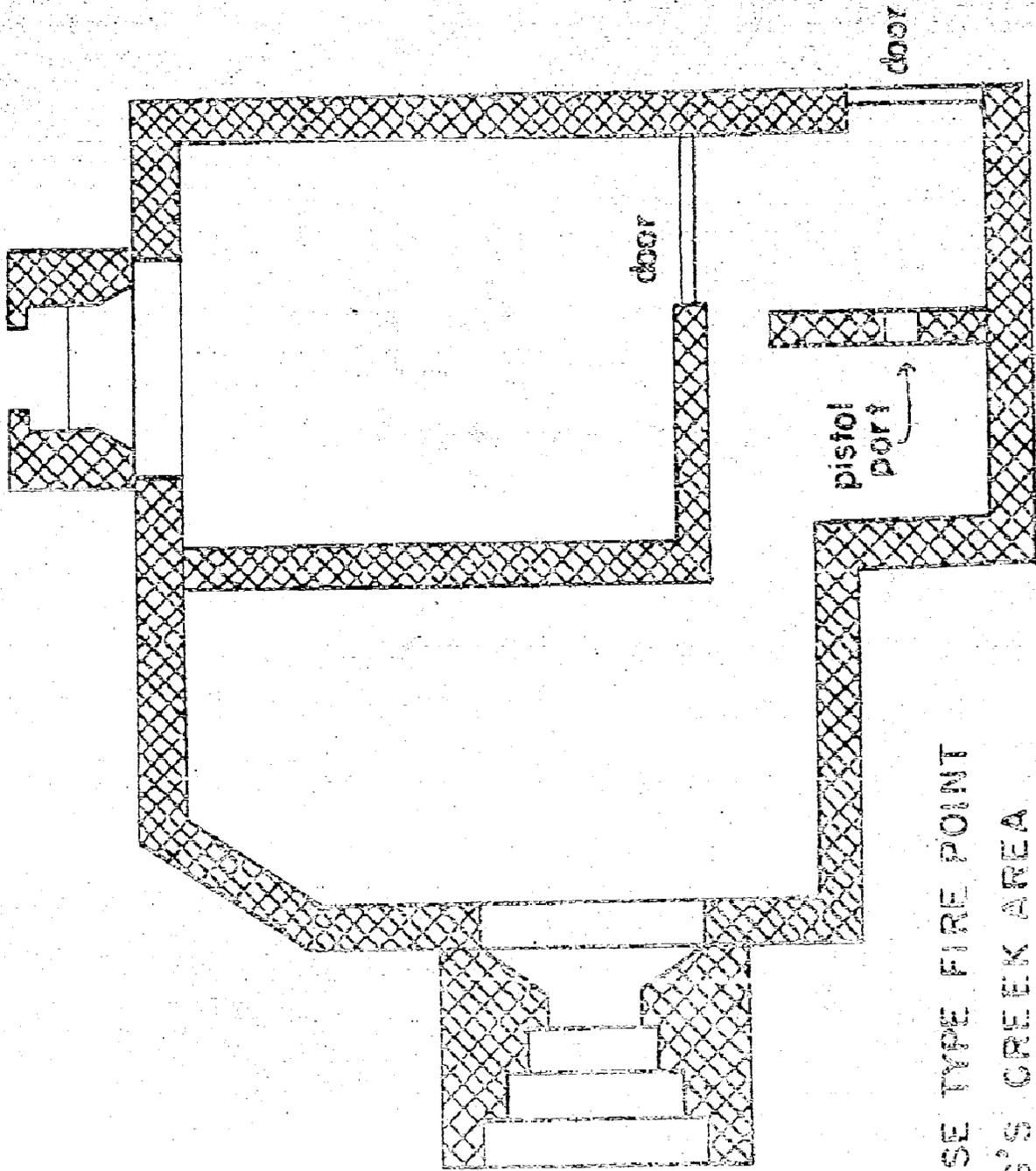


FIGURE 3.- GERMAN TYPE BLOCKHOUSE
KING'S CREEK AREA
EDGEWOOD ARSENAL, MD.



JAPANESE TYPE FIRE POINT
KING'S CREEK AREA
EDGEWOOD ARSENAL, MD.

FIGURE 4

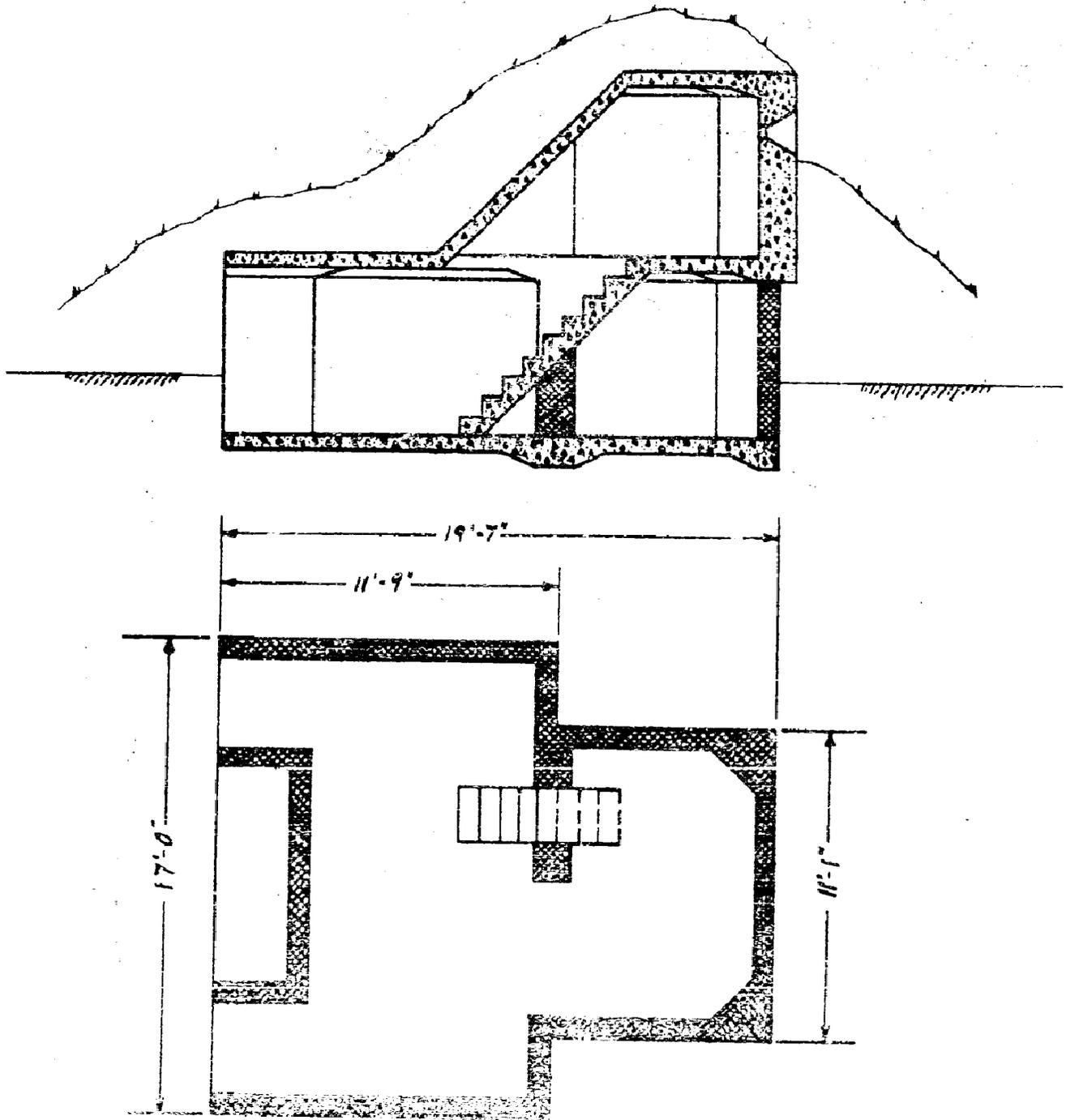


FIGURE 5

TWO LEVEL JAPANESE FORTIFICATION

Ideally, all of the above variables were to be measured continuously, before, during, and after flame attack. With the exception of caloric bombardment, this proved to be practicable. Subsequently, the Flame Attack Section developed methods for making physiological studies of animals exposed to flame attack and extended its investigations to cover the flame attack of moderately and well ventilated positions.

Having measured both the environmental changes produced by flame attack and having studied the net effect of these factors on animals, it was desirable to attempt to evaluate the relative importance of the lethal factors singly and in combinations of two or more. These investigations were undertaken by the Clinical Research Section, Medical Division, Army Chemical Corps; under Captain P. R. Dumke, M.C.

II. EXPERIMENTAL

The methods used for measuring the environmental and physiological changes produced in enclosed structures by flame attack are described below.

A. Environmental Changes

1. Heat

(a) Ambient air temperature

Changes in air temperature were measured by means of a chromel-alumel welded thermocouple whose output was amplified and recorded continuously. In order to insure sensitivity to rapid changes in temperature, a fine thermocouple was required. No. 28 (12.6 mil) chromel and alumel wires were threaded through two channels of a porcelain tube and allowed to extend about one quarter of an inch from each end. To these 12.6 mil wires, No. 36 (5 mil) wires were welded, chromel to chromel and alumel to alumel. The 5 mil wires were then welded together to give a 5 mil thermocouple with 12.6 mil leads. From the other end of the porcelain tube the 12.6 mil wires, insulated by asbestos fiber tubing, went to the cold junction where they were connected to copper leads and immersed in an ice water bath. The copper leads from the cold junction were placed in series with a 1000 ohm potentiometer to permit compensation for line and thermocouple resistances. The output of the thermocouple was then amplified through a General Motors, breaker type, D.C. amplifier whose output was recorded on an Esterline Angus Recorder. Thermocouples of this construction gave a linear output up to 900°C. with a response time of about 0.5 seconds. The error of the air temperature measurement with this equipment was about 1%.

(b) Caloric bombardment

Copper calorimeters were used in an attempt to measure the amount of heat bombarding the occupants of a fortification. These consisted of copper cylinders of known mass and surface area which hung in the fortification. The ambient and radiant heat striking these cylinders dur-

electrically on a thermocouple brazed to the interior of the cylinder. While these calorimeters proved to be satisfactory under conditions involving relatively slow changes in temperature, the time lag in response was too great for them to be of much value during the usual brief flame attack under simulated battle conditions. They were accordingly not used for the bulk of the experiments.

2. Oxygen concentration in the atmosphere

The per cent oxygen in the atmosphere was measured by means of the Cities Service M. H. Heat Prover, which incidentally is also equipped to measure per cent combustible gases (mainly hydrocarbons and carbon monoxide). Gas Samples were drawn from the bunker through a one-eighth inch pipe by a one-quarter h.p. pump sampling at a flow rate of 10-12 liters/min. with a negative line pressure of two inches of mercury. A second pump which is an integral part of the Heat Prover, drew a measured sample from this main sample line and delivered it to the oxygen meter.

The oxygen meter of the Heat Prover consists essentially of a Wheatstone bridge circuit, two arms of which are made up of heated, catalytic, platinum wire, and two arms of variable resistances and hot wires. The gas sample drawn from the bunker is mixed with excess hydrogen, generated in the meter by a sodium hydroxide cell operating at constant voltage. On coming in contact with the heated catalytic platinum wire, all of the oxygen in the sample combines with hydrogen, and the heat evolved by the reaction increases the temperature of the wire and unbalances the bridge. The degree of unbalance is measured on a milli-voltmeter graduated to read in per cent oxygen.

To make a continuous record of the per cent oxygen, the milli-voltmeter was disconnected and the degree of unbalance of the Wheatstone bridge was measured on a General Electric photoelectric recording galvanometer having a full scale deflection for 210 microamperes. The accuracy of the oxygen determination is $\pm 0.5\%$, i.e., if $X =$ per cent oxygen, the instrument measures it to within $X \pm 0.5\%$.

3. Carbon monoxide and carbon dioxide concentrations

The carbon monoxide and carbon dioxide concentrations in the atmosphere were measured by means of Leeds and Northrup Selective Gas Analyzers, which are designed to measure the infrared absorption of dipolar gases. The instrument used was essentially that described by W.G. Fastie and A.H. Pfund (12) as their final instrument.

a. Determination of carbon monoxide

In the determination of carbon monoxide, a beam of infrared radiation passes through the gas sample, after which it is split in two and passes through two conical gas filters, one of which contains pure oxygen and the other pure carbon monoxide. After passing through the gas filters, both beams pass through an additional compensating filter containing carbon dioxide

to eliminate the effect of the presence of that gas on the carbon monoxide analyses. The divided beam then falls upon a differential thermopile which measures the difference in energy of the two beams.

The infrared absorption is maximal in the beam passing through the gas filter containing pure carbon monoxide, while the amount of absorption in the beam passing through the oxygen filter depends on the concentration of carbon monoxide in the gas sample. In practice, the output of the differential thermopile is made zero initially (in the absence of carbon monoxide in the air sample) by the application of a bucking potential. Subsequent appearance of carbon monoxide in the air sample unbalances the thermopile by reducing the amount of radiation passing through the oxygen filter.

The output of the analyzers was amplified and recorded continuously on Esterline Angus recorders. The relation of output to carbon monoxide concentration was determined by analyzing gas mixtures containing known percentages of carbon monoxide. The air sample was pulled through the analyzer at a flow rate of 10 liters/min. The accuracy of the determination is proportional to the percentage of carbon monoxide in the sample, so that if X = per cent carbon monoxide, the analytical error is approximately $\pm 0.05X$ %.

b. Determination of carbon dioxide

The method for the determination of carbon dioxide is exactly analogous to that for the determination of carbon monoxide, except that the two gas filters contain oxygen and pure carbon dioxide, respectively, and the additional carbon dioxide filter is, of course, eliminated. Since the concentration of carbon dioxide in our experience was always sufficiently greater than that of carbon monoxide to eliminate serious interference from the presence of the latter, an additional compensating filter containing carbon monoxide was unnecessary.

The sampling rate, method of calibration, and accuracy of the determination were the same as with carbon monoxide.

Since neither carbon monoxide or carbon dioxide are consumed in the process of analysis, the same air sample was pulled through carbon monoxide analyzers and carbon dioxide analyzers in series.

4. Concentration of unburned hydrocarbons

The Cities Service M. H. Heat Prover, mentioned above, consists of two parts, one for the determination of oxygen as already described, and another part for the determination of unburned combustible gases. The latter would be mainly unburned hydrocarbons and carbon monoxide in gasoline conflagrations. The construction and operation of the combustible gases meter is similar to that of the oxygen meter except that the gas sample is mixed with

excess air permitting complete oxidation of combustible gases over the heated catalytic platinum wire. Since carbon monoxide was being determined simultaneously by means of the infrared analyzer, the concentration of combustibles other than carbon monoxide could be estimated.

5. Other possible toxic gasses

In order to determine whether any possible toxic constituents of the atmosphere were being overlooked, gas samples taken from a bunker subjected to flame attack were analyzed by the National Bureau of Standards, by means of the mass spectrometer. The results of two analyses are shown in Table I.

TABLE I

Analysis of Gas Samples From Edgewood Bunker

<u>Component</u>	<u>Sample #1</u> <u>Mole Per cent</u>	<u>Sample #2</u> <u>Mole Per cent</u>
Methane	0.47 ± 0.05	0.48 ± 0.05
Acetylene	0.03 ± 0.01	0.05 ± 0.01
Ethylene	0.38 ± 0.03	0.45 ± 0.04
Propylene	0.04 ± 0.01	0.06 ± 0.01
Acetaldehyde	0.14 ± 0.01	0.18 ± 0.02
Butenes	0.04 ± 0.01	0.02 ± 0.01
Benzene	0.05 ± 0.01	0.07 ± 0.01
Toluene	0.01 ± 0.005	0.01 ± 0.005
Neon	0.11 ± 0.01	0.06 ± 0.01
Nitrogen	77.6 ± 0.1	77.7 ± 0.1
Carbon Monoxide	2.38 ± 0.03	2.91 ± 0.03
Carbon Dioxide	8.03 ± 0.02	10.38 ± 0.02
Oxygen	9.59 ± 0.02	6.53 ± 0.06
Argon	1.10 ± 0.01	1.10 ± 0.01

It can be seen from Table I that aside from carbon monoxide, carbon dioxide, low oxygen, and some hydrocarbons, only acetaldehyde appeared to be worthy of consideration as a possible toxic constituent.

B. Experimental Animals

The instrumental data obtained by the methods described above pointed to the important lethal factors associated with flame attack, but the quantitative data could only be evaluated by correlation with physiological and pathological changes associated with a given set of environmental changes. For this purpose experimental animals had to be used.

Of the available experimental animals, the goat was chosen because it was felt that the response of the goat would closely approximate the response of man under the conditions of the experiments. The goat has proved to be a satisfactory animal for experiments with toxic gases. The effects of heat on the goat had not, to our knowledge, been investigated prior to World War II, but in this respect also, the goat possessed favorable qualifications.

Heat can produce damage in two ways: (a) by local injury or destruction of the skin (burns), and (b) by elevating the body temperature without necessarily attendant skin damage (hyperpyrexia). In the latter case the rise in body temperature should be related to the amount of heat absorbed per unit of body weight (cal./kg.). On the other hand ambient and radiant heat reaches the body from the surrounding atmosphere by virtue of the caloric bombardment of the body surface. It is thus apparent that a small animal with a relatively large surface (target) area per unit of body weight would be more vulnerable to the effects of heat than would a larger animal with its smaller ratio of body surface to body weight.

A comparison of the ratio of surface area to body weight for various experimental animals calculated by using Meeh's formula and Benedict's constants is given in Figure 6, and it can be seen that this ratio for a goat weighing about 75 lbs. is not significantly different from that of man. The relationship is only approximate since the applicability of the formula to the goat has not been verified experimentally.

The animals used for the experiments on the toxicology of fire were adult goats weighing from 25 to 138 lbs., and averaging 64 lbs. The goats were examined by a veterinarian before use and were rejected if there was any evidence of disease or abnormality.

Since the long hair of the goats afforded significant though variable protection from ambient heat, all animals were clipped from knees and hock joints to the head before exposure to flame attack. Clipping was done with an electric clipper which left about one-eighth inch of hair on the animals.

The goats were fastened in the structures subjected to flame attack by a chain attached to a leather collar. The chain was long enough to allow them to fall without choking if they became prostrate before removal. Following flame attack, the animals were not removed from the structure under attack, until the concentrations of carbon monoxide and oxygen and the air temperature had returned to harmless levels.

COMPARISON OF BODY WEIGHT & SURFACE AREA

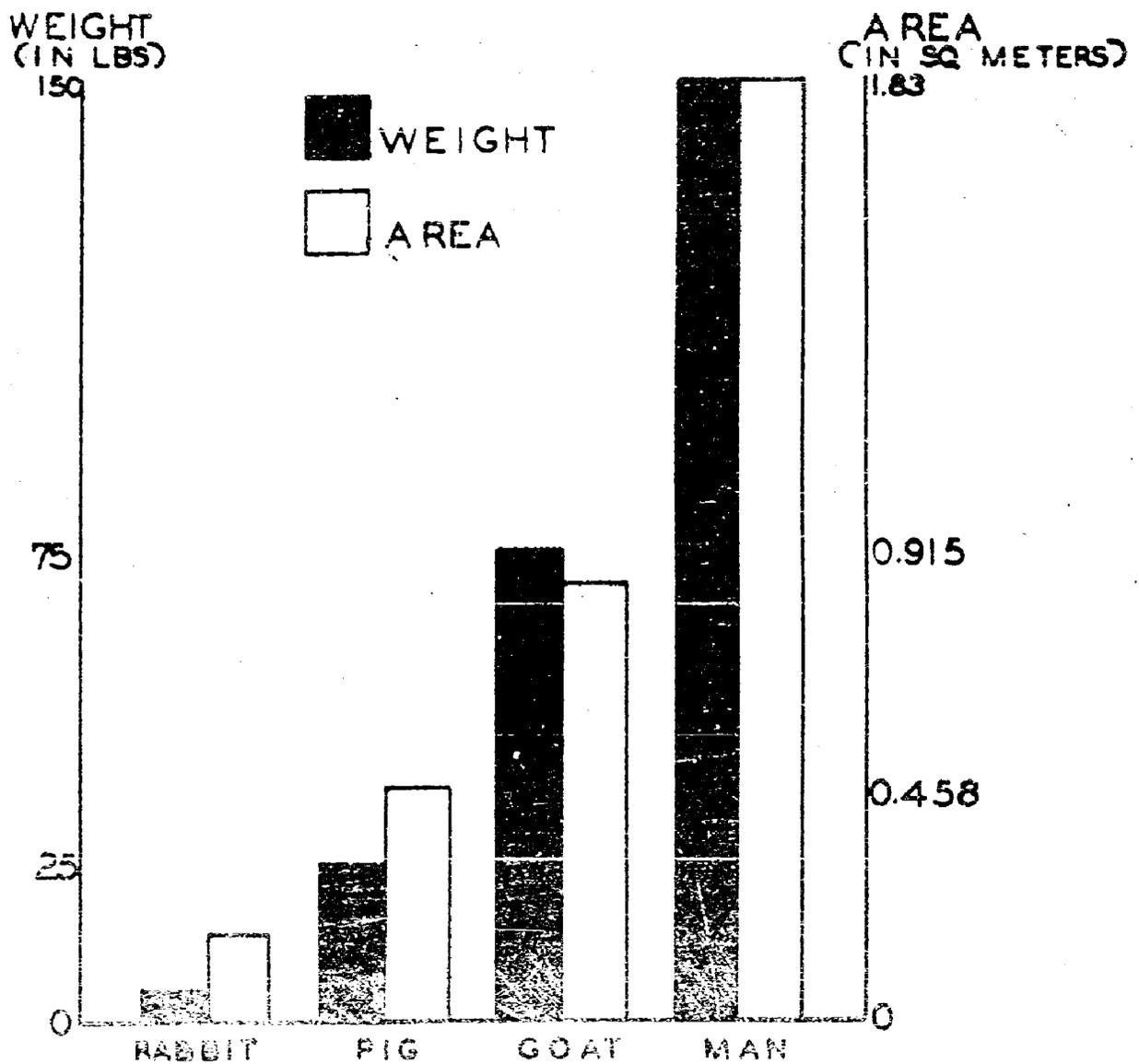


FIGURE 6

At the time of removal, the animals were immediately examined by a veterinarian. Any animal which was dead on removal or died within ten minutes after removal was classified as a fatality. All animals which were alive ten minutes after removal were classified as survivors regardless of the severity of their injuries. All severely injured survivors were destroyed by electrocution immediately after the ten minute observation period.

C. Physiological Observations

Physiological observations on goats exposed to flame attack were a necessary adjunct to the instrumental measurements of the environmental changes. Due to the number of experiments which had to be carried out, it was not possible to make detailed observations on all animals. The end results of an experiment were the number of animals killed (as previously defined) or surviving. However, in a large number of animals the body temperature, blood oxygen concentration, and blood carbon monoxide concentration following exposure to flame attack were measured.

In most cases, rectal temperature was measured immediately after removal of the animals from the structure attacked by flame, the instrument used being an ordinary glass laboratory thermometer. In a few experiments, rectal temperature was recorded continuously by means of an iron-constantan thermocouple coupled to a recording galvanometer. Blood oxygen and blood carbon monoxide were measured by the method of Scholander and Roughton (13). Blood hemoglobin was measured photometrically by means of a Klett-Summerson Photoelectric colorimeter, and blood methemoglobin was measured spectrophotometrically by means of a Colson Spectrophotometer.

Additional measurements were made in special cases, which will be described as occasion arises in subsequent papers.

D. Pathology

A representative number of animals were examined post mortem for gross and microscopic pathology. The micropathology was interpreted by Major A. M. Ginzler, M.C., Chief, Pathology Section, Medical Division, Army Chemical Corps.

E. Synthetic Experiments

By means of the techniques described above, a considerable amount of descriptive information relative to the environmental changes produced by flame attack was obtained, and this information was correlated with physiological changes produced in experimental animals. The information did not provide a complete explanation of the relative importance of several lethal factors which occurred simultaneously. It was, therefore, decided to approach the problem by exposing goats to each of the known lethal factors at first and then to expose them to combinations of two or more. This work was carried out by the Clinical Research Section, Medical Division, under the direction of Captain Paul R. Durko, M.C.

F. Protection Against Flame Attack

Methods of protecting personnel against the effects of flame attack were given serious consideration, not only by the Army and Division 9, NDRC, but also by Division 11, NDRC. Experiments conducted at the Army Chemical Center, Maryland, involved studies of the effectiveness of protective clothing and of heat protective ointments. These experiments will be described in a subsequent paper.

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THE TOXICOLOGY OF FIRE

PART II. Factors Involved in Injury or Death
From Exposure to Direct Flame.

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Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART II. Factors Involved in Injury or Death From
Exposure to Direct Flame.

SUMMARY

Goats exposed in the open to the effects of aerial bombs charged with thickened gasoline frequently died within an hour or two. While they usually sustained severe cutaneous burns, these burns were not of a type which is known to produce such rapid death. Examination of the casualties revealed that death was not due to inhalation of toxic gases or a deficiency of oxygen in the atmosphere. Pathological examination revealed, in addition to the skin burns, damage to the respiratory tract and pulmonary congestion and edema, which presumably resulted from the inhalation of heated air and smoke.

Goats exposed in the open to burning fuel discharged from a flame thrower also frequently died within an hour or two. They also sustained severe skin burns, but did not show damage to the respiratory tract. There was, however, evidence of central respiratory failure in some goats and in others there was an increase in serum potassium concentration to levels ordinarily considered lethal.

It is believed that in both types of exposure to direct flame in the open, death was caused by heat, a factor which Moritz and co-workers had shown to be a sufficient cause of rapid death when animals were exposed for longer times to temperatures lower than those which existed for a few seconds in these experiments.

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART II. Factors Involved in Injury or Death From
Exposure to Direct Flame.

I. HISTORICAL

It was indicated in the first part of this paper that several factors may be involved in causing injury or death to the occupants of enclosed fortifications subjected to attack by flame throwers. These are chiefly heat, carbon monoxide, lack of oxygen, carbon dioxide, hydrocarbon vapors and possibly other toxic gases. On a priori grounds it would be expected that the toxic gases would be most effective in poorly ventilated fortifications and less effective as the degree of ventilation of the fortification was increased. The extreme case of good ventilation would be flame attack of personnel in the open. Similarly, one might expect heat to be most important as a casualty factor under cases of maximum ventilation.

As a part of investigations sponsored by Division 9, National Defense Research Committee, Moritz, Henriques, and co-workers undertook an experimental investigation of the effects of heat, uncomplicated by toxic gases in the production of casualties and death (1,2,3,4,5,6,7,8). These studies have done much to increase our knowledge of the genesis and mechanism of heat injury. That part of the Moritz and Henriques experiments which bears most closely on injury by fire involved the cutaneous exposure of pigs to circumambient (air) and circumradiant heat in an oven (4) and the respiratory exposure of dogs to hot air and steam (8).

Moritz and Henriques studied the effect on pigs of exposure for varying lengths of time to air temperatures ranging from 70°C. to 550°C. In the extremes, exposures of the order of 15 minutes to an air temperature of 80°C. or of the order of 30 seconds to air temperatures greater than 500°C. were capable of causing acute hyperthermic death. During exposure the animals breathed air at room temperature. The mechanisms involved in the production of death at the lower and higher temperature extremes, however, were somewhat different. In the long exposures at low temperatures there was little or no cutaneous burning and death appeared to result from peripheral vascular collapse with attendant progressive hypotension. In the case of brief exposure to high air temperatures, there was always severe general cutaneous burning with circulatory failure of central rather than peripheral origin. The cause of the central circulatory failure was traced to the rapid liberation of potassium from erythrocytes in the heated cutaneous and subcutaneous tissues, with consequent damaging effect of the liberated potassium on the heart (4, 6,7).

In the above experiments, the heat was applied to the skin and not to the respiratory tract. In another series of experiments, the effect of heat on the respiratory tract, uncomplicated by cutaneous exposure to heat, was investigated (8). Anesthetized dogs were made to breathe either oven-

heated air, or flame or steam. If breathed in the normal fashion, the result was a severe pharyngeal edema which resulted in obstruction of the respiratory passages (and hence asphyxia) within an hour. In these cases, however, heat damage to the lungs was not evident. When the heated atmosphere was delivered by means of an insulated trans-oral cannula into the trachea below the larynx, the following effects were noted: (a) Moderate to severe tracheitis without deep pulmonary injury when hot dry air or flame was breathed; and (b) tracheitis and mild to severe thermal injury of the lungs when steam was breathed. The difference in the effect of hot air and steam was attributed to the fact that dry air has little heat capacity, and gave up its heat before reaching the lungs, whereas steam has more heat to give up and lost heat more slowly, so that enough heat could be delivered to the lung to cause thermal injury.

These experiments showed definitely how heat, uncomplicated by the effect of inhalation of a toxic atmosphere, could be a cause of death or severe injury in the circumstances attending flame attack.

On the battlefield, men were being subjected to flame attack in the open and in many cases were being severely injured (or killed) as a result. Two very practical problems arose thereby. How could the destructive power of flame attack be minimized by the adoption or utilization of protective devices? How should injuries sustained as a result of flame attack be treated, i.e., as heat injury, injury from toxic gases, or both?

The experiments of Moritz, Henriques, et al., while very important, did not completely answer these questions because: (a) Moritz's extreme air temperature was about one-half that of flame temperature; and (b) the possibility of injury from inhalation of toxic products of combustion could not be arbitrarily excluded, even in the case of personnel attacked by flame in the open. It could not be concluded with certainty, therefore, that the mechanisms of heat death revealed by Moritz's experiments would be those responsible for killing or injuring personnel attacked by flame - even in the open.

The following experiments were carried out therefore to study the effect of flame attack in the open on goats, and to evaluate the toxic factors thereof. The justification for these rather drastic experiments was simply that the information gained might save the lives of Allied soldiers, and that such information could be gained in no other feasible way.

II. EXPERIMENTAL

A. Materials and Methods

The experiments dealing with the effects of flame in the open fell into two broad categories: (a) Those in which the flame was delivered onto the target by means of aerial fire bombs charged with thickened gasoline; and (b) those in which the flame was delivered onto the target by means of a flame thrower. Both of these weapons were being used on the battlefield.

The fire bombs were dropped onto a target area from aircraft, and ~~some were tethered within the area, both on the surface of the ground and in~~

explained in Part I of this paper the goats were clipped so as to leave a uniform amount of hair, corresponding roughly to the amount of protection afforded a man by a suit of cotton coveralls.

The target area was entered within 30 to 90 seconds after the bomb detonated, and blood samples were taken on selected dead or prostrate animals for determination of oxygen and carbon monoxide content and oxygen capacity and occasionally for other analyses. All survivors were examined immediately by a veterinarian, and if injured, were painlessly destroyed on the spot. Selected goats were examined for gross and micropathology.

In the second group of experiments, a single goat was tethered in a location adjacent to a 1/8 inch iron pipe, placed upright and with the open end 3 feet 8 inches from the ground. This pipe was supported by a larger one to which a chromel-alumel thermocouple was attached. The thermocouple was shielded from possible direct impingement of burning fuel by a small piece of transite. Gas sampling and analysis, and recording of air temperature were done by the methods described previously (Part I of this paper).

The goats used were healthy adult clipped animals as described elsewhere.

An initial blood sample of 10 ml. was drawn from the jugular vein, and the rectal temperature was taken by means of an ordinary laboratory thermometer inserted about two inches. In most experiments, electrocardiographic leads of the non-polarizable, plate type supplied with the Sanborn Cardiette were taped to each foreleg and the left hindleg about two-thirds of the distance from the hoof to the axilla or groin. Ordinary electrode paste was used and the skin was not shaved. Initial electrocardiograms were taken prior to the flame attack.

In some experiments a cardiac thermistor, mounted at the end of a piece of rubber tubing 18 inches long and the size of a No. 5 French catheter was passed through a 12 gauge needle inserted into the right jugular vein until the thermistor was at the junction of the venae cavae. Previous trials under fluoroscopic observation guided the placement of the thermistor. On occasion, the respiration rate was measured by means of a carbon pile pneumograph, which was placed about one-third the distance from the sternum to the vertebra at the level of the xiphisternum. The carbon pile unit was held firmly against the ribs by means of a strap passing around the thorax.

The electrodes, thermistor leads and pneumograph were insulated from heat by heavy paddings of fiberglass wool and cloth held close to the body surface.

The duration of exposure to flame was of the order of five seconds, after which time the goats were dead or unconscious. Survivors were under the constant observation of a veterinarian and a physician and were painlessly destroyed if they showed any signs of discomfort or return to consciousness.

Blood samples taken after exposure were used for the determination of serum potassium and sodium by means of the Flame Photometer, plasma proteins by the falling drop technique, and blood pigments and gases by standard analytical methods. The hematocrit was estimated roughly by using a centrifuge to pack the cells of the heparinized blood.

Immediately after the death of each subject, the chest was opened, and blood was withdrawn from each auricle. The lungs were removed, drained carefully of blood, and were weighed. The blood, water, and ash contents of the lungs were measured subsequently in each animal. A gross autopsy was performed, and tissue specimens were taken for micropathological examination.

B. Results

1. Fire Bomb Experiments

Fire bombs of the type used in these experiments scatter chunks of burning thickened gasoline over a roughly elliptical area of ground. For a few seconds following detonation, there exists in the vicinity of the bomb, a large cloud of burning vapor which has been aptly described as a fireball. After the fireball has dissipated the chunks of fuel burn on the ground for several minutes. Goats outside the area of burning fuel were not injured. Animals inside the area of burning fuel were liable to two sorts of injury: (a) Radiation burns from the fireball; and (b) actual physical contact with pieces of burning thickened gasoline.

Following the detonation of a fire bomb, the goats were reached within 30-90 seconds by observers who extinguished any fuel in contact with the animals. While skin contact with burning fuel produced severe local burns as might be expected, these were of the type which usually does not result in immediate or even rapid death. Yet 34% of the goats on the surface of the ground and 2% of the goats in slit trenches were dead within 10 minutes after detonation of the bomb, and 46% of those on the surface and 33% of those in trenches were prostrate and unconscious. It appears likely, therefore, that death or severe injury was associated primarily with the cloud of burning vapor or fireball.

The oxygen and carbon monoxide content of the blood of goats who were alive but unconscious following the bomb burst are given in Table I. It is evident from the data of Table I that carbon monoxide was not a sufficient cause of death since the arterial blood levels are well below those ordinarily considered lethal. The low oxygen saturation may be associated with the slow and gasping type of respiration of the animals and does not necessarily indicate that they breathed an atmosphere strikingly deficient in oxygen.

A crude measure of air temperatures attained was made by scattering temperature indicating pellets* over the target area. These pellets never indicated temperatures above 600°F., but much higher air temperatures undoubtedly existed for two or three seconds, and the time lag for melting of the pellets must be taken into account.

*Tempil-Pellets - The Tempil Co., New York, N.Y.

Oxygen and Carbon Monoxide Contents of Goats Exposed to Effects of
the Fire Bomb

Trial No.	O ₂ Saturation (%)	CO Saturation (%)	O ₂ Capacity vols. (%)
7	77.2	8.14	11.9
10	83.2	10.4	17.0
11	42.7	7.0	14.0
11	44.8	12.1	14.9
23	44.8	12.1	14.9

Gross and microscopic pathological studies were made of 14 goats. Findings in the individual animals are listed in Table II.

The significant findings were mainly confined to the respiratory system, and formed a characteristic pattern although there was considerable variation from one animal to another. Laryngeal edema was present to some extent in all but six of the fourteen goats. In six animals the edema of the laryngeal mucosa produced complete or almost complete occlusion of the larynx, and in four more there was partial occlusion. All of the goats showed slight to considerable reddening of the mucous membrane of the trachea and main bronchi, but in only one instance did an exudative pseudomembrane develop. Several presented a deposit of carbon that lined the trachea and extended through the bronchial tree, but this was not a consistent finding. In almost all cases there was at least a slight amount of frothy edema fluid in the bronchi and lower trachea; in some instances, considerable edema fluid poured from the bronchi and filled the trachea with foamy fluid.

The lungs showed a general increase in size and weight, half of the group showing a decided increase in the lungs. Body weight ratio beyond the normal range of 0.8 to 1.2%. This was associated with congestion of the lungs varying from patchy involvement, chiefly of the lower lobes, to almost diffuse involvement of all lobes; and a similarly variable degree of pulmonary edema. In some animals the cut surfaces of the lungs were considerably congested but presented only a slight or moderate amount of edema fluid on pressure, while in others a considerable amount of edema fluid exuded freely from cut sections and filled the bronchi and trachea. Microscopically, the lungs showed prominent hyperemia of the interalveolar septal capillaries, and congestion of the small blood vessels. Edema of the lungs varied from the occurrence of an occasional patch in sections of some animals to diffuse involvement of almost all alveoli in sections of others. Where more advanced, it was usually accompanied by some degree of perivascular connective tissue edema and lymphatic dilatation. Atelectasis was not a significant gross feature of the

TABLE VI
Pathologic Findings in Brains Exposed in the Open to Plague

Animal No.	Site Burned	Sex or Age	General Condition	Larynx	Trachea and Bronchi	Lungs	Weight of Body (gms.)	Color
K 10 C	5th degree burn of entire exposed side.	10 mts.	Feet, not protrude	Normal	Normal	Moderate hyperemia. No significant edema. Several foci hemorrhages.	-	Blood bright red.
K 20 C	Normal; 1st degree burn of exposed side and head.	1 1/2 hr.	Normally (slurred) protrude	Marked edema and congestion almost completely occluded.	Congested. Marked amount of frothy fluid and cartilage in trachea and bronchi.	Moderate congestion. Moderate congestion, especially of lower lobes. Lungs fairly crepitant; edema - 1 plus.	1.80	Ed and thick and edema slightly redder than normal.
K 21 C	4th degree burn of entire exposed side.	15 mts.	Protrude immediately dead at 15 mts.	Marked edema and congestion almost complete occlusion.	Congested. Moderate amount of frothy fluid and cartilage in trachea and bronchi.	Moderate to heavy congestion, most marked in lower lobes. Microscopically, patchy hyperemia and partial atelectasis. Edema - 1 plus.	0.85	Ed and O ₂ - 14.7% Sat. CO - 20.7% Sat. (0.75 vol. %)
K 22 C	2nd and 4th degree of entire exposed side and head.	1 1/2 hr.	Normally protrude	Considerable edema with partial occlusion.	Carbon in trachea and main bronchi. Moderate amount of frothy mucus and carbon particles.	Moderate congestion, most marked in lower lobes. Edema slight, patchy - 1 to 2 plus.	1.25	Ed and O ₂ - 17.1% Sat. CO - 20.6% Sat. (0.75 vol. %)
K 23 C	Heavy severe 4th degree burn of face, head, neck and legs.	6 mts.	-	Moderate edema; not occlusive.	Normal; contains frothy mucus and carbon particles.	Heavy red, mottled and with patchy congestion areas, more marked in lower lobes. Moderate patchy edema - 1 plus.	1.46	Edema interstitial edema of lungs.
K 24 C	2nd and 4th degree burn of head and body.	6 mts.	-	Marked edema.	Moderate redness. Moderate amount of frothy fluid exuding from bronchi. Carbon in trachea and bronchi.	Considerable, patchy congestion. Edema 2.4% locally lower lobes, on slight pressure 3 to 4 plus.	2.4%	Interstitial edema of lungs, especially lower lobes.
K 25 C	Generalized 3rd and 4th degree burn.	4 mts.	-	Moderate laryngeal edema with partial occlusion.	Large amount of frothy fluid pouring from bronchi.	Marked patchy congestion. All lobes show marked edema - 4 plus.	2.25	Interstitial edema of lungs.
K 26 C	Generalized 3rd and 4th degree burn of head, neck and legs.	1 hr.	-	Essentially negative.	Small amount of frothy edema fluid in bronchi.	Moderate congestion and edema of lungs, patchy chiefly in lower lobes. Edema - 2 plus.	2.35	-
K 27 C	Generalized 4th degree burn of head, neck and legs.	11 mts.	Protrude immediately after attack	Marked edema moderate congestion.	Slight redness in edema fluid in bronchi.	Marked, patchy degeneration of all lobes. Locally, scattered patches hemorrhagic with areas of partial atelectasis. Other areas of hyperemia and partial atelectasis in alveolar sections.	1.32	-
K 28 C	Generalized 3rd and 4th degree burn.	60 mts.	Protrude	Marked edema.	Congested filled with frothy edema fluid.	Marked congestion of all lobes. Marked edema, with occasional foci of emphysema from out surface - 3 to 4 plus.	2.69	Interstitial edema of lungs toward hilum.
K 29 C	Ditto	60 mts.	Ditto	SLIGHT edema.	Congested. Considerable froth in trachea and bronchi.	Marked congestion and edema of all lobes - 6 plus.	2.11	Ditto
K 30 C	Ditto	5 hr.	-	Moderately edematous	Froth present.	Considerable patchy congestion but relatively little edema in lungs. Patchy areas of partial atelectasis, microscopically. Edema - 1 to 2 plus.	1.17	-
K 31 C	Ditto	2 hr.	Rebound when also inflated	Moderately edematous	Froth present; carbon in fur as air source.	Considerable patchy congestion of all lobes; moderate edema - 1 plus. Slight patchy areas of atelectasis microscopically.	1.27	-
K 32 C	Ditto	20 mts.	-	Severe edema, with almost complete occlusion.	Little congestion. Slight amount of mucus and froth.	Moderate congestion; slight edema - 2 plus. Patchy hyperemia and atelectasis in apex of upper lobes.	1.00	-

pulmonary pathology, but areas of partial atelectasis were not an uncommon finding microscopically. Their occurrence appeared to be related to a stage of distinct capillary hyperemia and early transudation of fluid into the alveoli, and they were generally absent in sections of areas of more advanced edema. The appearance of the bronchi was not suggestive of bronchoconstriction, although the bronchi sometimes contained collections of desquamated epithelial cells which, possibly together with some inflammatory exudate, may have contributed to partial obstruction of such bronchi. Epithelial desquamation occurred chiefly in the trachea and main bronchi, and little or not at all in the smaller bronchi. Actual epithelial necrosis was not observed. Occasionally, granules of carbon pigment were observed as far distally as the respiratory bronchioles and atria. No systematic histologic study was made of the other organs since there was no gross evidence of pathology.

2. Flame Thrower Experiments

a. Changes in the atmosphere

The results of fifteen trials in which air temperature, oxygen, carbon monoxide, carbon dioxide, and hydrocarbon concentrations were measured continuously are shown in Table III. Two values are recorded for each variable: The peak or extreme change; and the Ct or Tt product, which is the integral of the curve of gas concentration, C, (or air temperature, T) versus time, t. The integration of the curves was done mechanically by means of a planimeter, and was carried out from t=0 at the time of firing to the time when the atmospheric condition had returned to essentially normal limits (usually five minutes or less). In the case of oxygen, which decreased in concentration as a result of the conflagration, the results are expressed in terms of ΔCt where ΔC is the change in oxygen concentration expressed as a positive number. Thus, the greater ΔCt , the greater the oxygen deficit over the given period of time.

The Ct values were calculated to give some index of the dosage of a given inhaled gas, for C (vols./100 volumes) times t (minutes) times respiration rate, R (volumes for minute), has the dimensions of quantity of gas. If respiration rate is assumed to be constant from one animal and experiment to another over the period in which the gas is respired, Ct should be proportional to the dose of the inhaled gas. While the assumptions may not be and probably are not strictly justified, Ct was as close an approach to a dosage quantity as was practical. The quantity ΔCt for oxygen representing as it does a deficiency of an essential atmospheric constituent rather than the presence of a noxious constituent, may still be considered in a sense as representing a dosage of an injurious quantity. Tt by the same reasoning may be imagined as a dosage of heat, although the heat probably acts mainly on the surface of the body and relatively little by virtue of its inhalation.

In some trials the flame appeared to impinge directly on the sampling point, and in these (Trials DF 4, DF 5, DF 6, DF 9, DF 10, DF 11, DF 13, DF 14, and DF 16) peak air temperatures above 800°C. were recorded. Considering that the thermocouples used do not reliably indicate the true temperature in the vicinity of 1000°C., the temperatures recorded

TABLE III

Changes in Atmosphere Produced by Flame of Hydrocarbon Fuels Fired From Flame Thrower in Open

Fl	Peak Temp. °C.	Temperature Rise °C. x min.	O ₂ Depletion		CO Production		CO ₂ Production		CH ₄ Production	
			Peak Vol. %	Δ % x min.	Peak Vol. %	% x min.	Peak Vol. %	% x min.	Peak Vol. %	% x min.
1	165	37.5	0	0	0	0	0	0	0	0
2	175	22.9	20.1	0.1	0.1	Trace	0.7	0.2	0	0
3	565	37.9	28.9	1.1	1.0	Trace	2.2	0.4	0.3	Trace
	815		35.3		1.6		3.4	0.4	0.8	
4	1015	262.0	10.1	1.5	7.4	1.7	17.0	5.4	7.8	1.4
5	1030	Thermocouple broke	6.2	4.1	Instrument failed				6.3	2.6
6	1075	136.0	15.3	0.6	2.5	0.1	0	0	1.3	0.2
7	520	45.6	19.1	0.3	0.4	0.1	0.7	0.3	1.2	0.2
8	145	15.9	0	0	0	0	0	0	0	0
9	965	254.0	1.3	3.6	9.3	1.6	17.0	4.2	15.7	2.4
10	680	117.1	12.6	1.1	2.7	0.4	6.3	2.0	3.2	0.5
11	1000	142.9	5	2.2	4.2	0.7	8.9	2.0	9.8	1.4
12	80	19.6	20.4	0.1	0	0	0.4	0.2	0	0
13	675	157.7	13.3	1.6	0.8	0.1	2.5	0.9	1.0	0.1
14	925	515.4	12.3	1.1	1.5	0.2	4.9	0.9	2.5	0.3
15	1000	1150.0	0	14.0	6.3	1.8	10.8	9.7	19.4	6.7

RESTRICTED

In these trials, in which flame apparently struck the sampling point, there was also a marked depletion of oxygen, and carbon monoxide was present in concentrations which reached as high as 9.3% (DF 9). Unburned hydrocarbons were also present even though combustion was taking place in the open air. The concentration of the various gases returned to normal levels, however, almost immediately after the flame passed, so that the Ct values were low.

b. Changes in animals

All goats exposed to flame showed a diminished response to pain and appeared to be unaware of what was happening around them. None was convulsant, but all but one were prostrate. Reflexes were diminished terminally, but were normal otherwise.

Respiration varied from animal to animal and from time to time with each goat (Table IV). The most common type of breathing was labored, slow, and noisy, of variable depth, and subsided gradually into a series of slow gasps. Every goat had a thick tenuous nasal discharge, which did not seem to interfere greatly with respiration. Most animals also salivated excessively.

The estimated extent of the body surface which was severely burned varied from 35 to 100%. This amount of burning generally is fatal in human beings.

The rectal temperature of the goats often did not rise at all and never exceeded 42°C. (Table IV). The cardiac temperatures were higher than the rectal by 2.1 to 4.1°C., and in some were above the level of hyperthermia found to be fatal in pigs by Moritz and his group (7).

The heart rate always rose during exposure to flame, and remained high for a variable length of time. Most of the animals which died soon after exposure showed electrocardiographic changes usually associated with cardiac anoxia, i.e., terminal bradycardia, conduction defects of various types, ectopic foci, and variable arrhythmias (Figs. 1a and 1b). One goat (Fig. 2) had a slow ventricular fibrillation described as occurring in potassium intoxication (7). This animal also had high levels of blood potassium (Table VI).

The blood carbon monoxide did not rise to dangerous levels in any of 5 goats exposed to direct flame, on whom analyses were made (Table V). Three of the five showed zero per cent saturation, and the remaining two 4.5 and 6.5%, respectively. The final oxygen saturation in these same goats ranged from 4.0 to 77.9% but the low oxygen saturations cannot be attributed to oxygen deficiency in the atmosphere because all of the goats breathed atmospheric air for at least several minutes before death. Since respirations ceased in these animals before effective heart action stopped, it is probable that the anoxemia was of the stagnant type.

TABLE IV

Changes in Physiological Measurements During Exposure to Flame

Trial or Goat No.	Body Temperature (°C.)		Heart Rate (Change in beat per min.)	Respiration (Change in rate per min.)
	Rectal	Cardiac		
DF 16	+0.1	-	-	-
DF 17	-	-	-	-
DF 18	+0.8	-	-	-
DF 19	-0.1	-	+65	-
DF 21	0	-	+30	-90
DF 22	0	-	+90	-57
DF 23	-0.1	-	+25	+7
DF 24	+1.2	-	-	-
DF 25	+1.1	-	-	-
DF 26	+1.5	-	+100	+20, -49
DF 27	-0.3	+4.0	+15	-
DF 28	-0.1	+3.9	+92	-27
DF 29	-1.0	-	+60	+178, -58
DF 30	+0.9	-	+53	+52, +164
DF 31	+1.1	-	-	-28
DF 32	+2.3	-	-	+228, -216
DF 33	+1.1	+4.1	-	+12, -3
DF 34	-0.1	+3.4	+70	-10
DF 35	+0.5	+2.1	+48	-48

TABLE V

Carboxyhemoglobin, Oxyhemoglobin, pH, Carbon Dioxide Capacity, and Methemoglobin in Goats Exposed to Flame

Trial or Goat No.	Arterial Blood Gases		CO ₂ Capacity (Vol. %)			Methemoglobin (gm./100 cc.)		
	(% Saturation)		pH		Initial		Final	
	H ₂ O	H ₂ O ₂	L.	Rt.	L.	Rt.	L.	Rt.
DF 16	4.5	77.9						
DF 17	0*	67.0**						
DF 19			7.26	7.20				
DF 21	6.5	16.1						
DF 23					53.0	44.3	43.4	
DF 25	0	4.0					1.82	4.88
DF 26					29.3	44.3	55.6	
DF 28	0	9.5						2.26
DF 32			7.36	7.32			7.12	

* Femoral Artery
** Carotid Artery

TABLE VI

Plasma Proteins, Serum Potassium and Sodium, and Serum Hemoglobin (Hemolyzed) in Goats Exposed to Flame

Trial or Goat No.	Plasma Proteins (gm./100 ml.)			Serum Potassium (mg./100 ml.)			Serum Sodium (mg./100 ml.)			Serum Hemoglobin (mg./100 ml.)		
	Initial	Final	Rt.	Initial	Final	Rt.	Initial	Final	Rt.	Initial	Final	Rt.
DF 16	-	-	-	-	-	-	-	-	-	-	-	-
DF 17	-	-	-	-	-	-	-	-	-	-	-	-
DF 19	9	9.5	-	20.4	31.9	-	373	306	-	-	-	-
DF 21	7.2	7.4	-	20.4	41.2	-	355	340	-	430	1300	-
DF 22	7.6	6.4	6.6	17.0	25.1	-	357	357	-	-	-	-
DF 23	7.4	7.9	7.8	15.9	31.9	29.7	371	350	286	130	960	490
DF 24	6.8	7.9	8.2	13.7	33.9	29.7	350	350	360	50	180	180
DF 25	7.9	9.3	9.7	16.0	35.3	54.0	337	342	322	0	270	130
DF 26	-	-	-	13.7	27.4	47.9	320	300	310	0	320	180
DF 27	8.1	7.3	6.7	13.1	35.9	39.8	315	380	335	500	790	430
DF 28	-	-	-	33.7	68.7	72.5	375	330	330	310	1350	1760
DF 29	-	-	-	12.6	17.2	24.0	295	322	295	500	1120	1350
DF 30	-	-	-	17.9	58.5	56.5	366	305	366	400	1080	950
DF 31	-	-	-	19.1	58.7	83.7	368	330	342	0	130	220
DF 32	-	-	-	15.8	16.9	25.0	353	344	356	50	9000	180
DF 33	-	-	-	17.9	43.3	-	311	311	353	0	360	220
DF 34	-	-	-	14.4	27.5	47.5	300	389	324	220	310	220
DF 35	-	-	-	12.6	35.4	23.7	350	356	-	-	-	-

Figure 1a

ELECTROCARDIOGRAM OF COAT DF 21

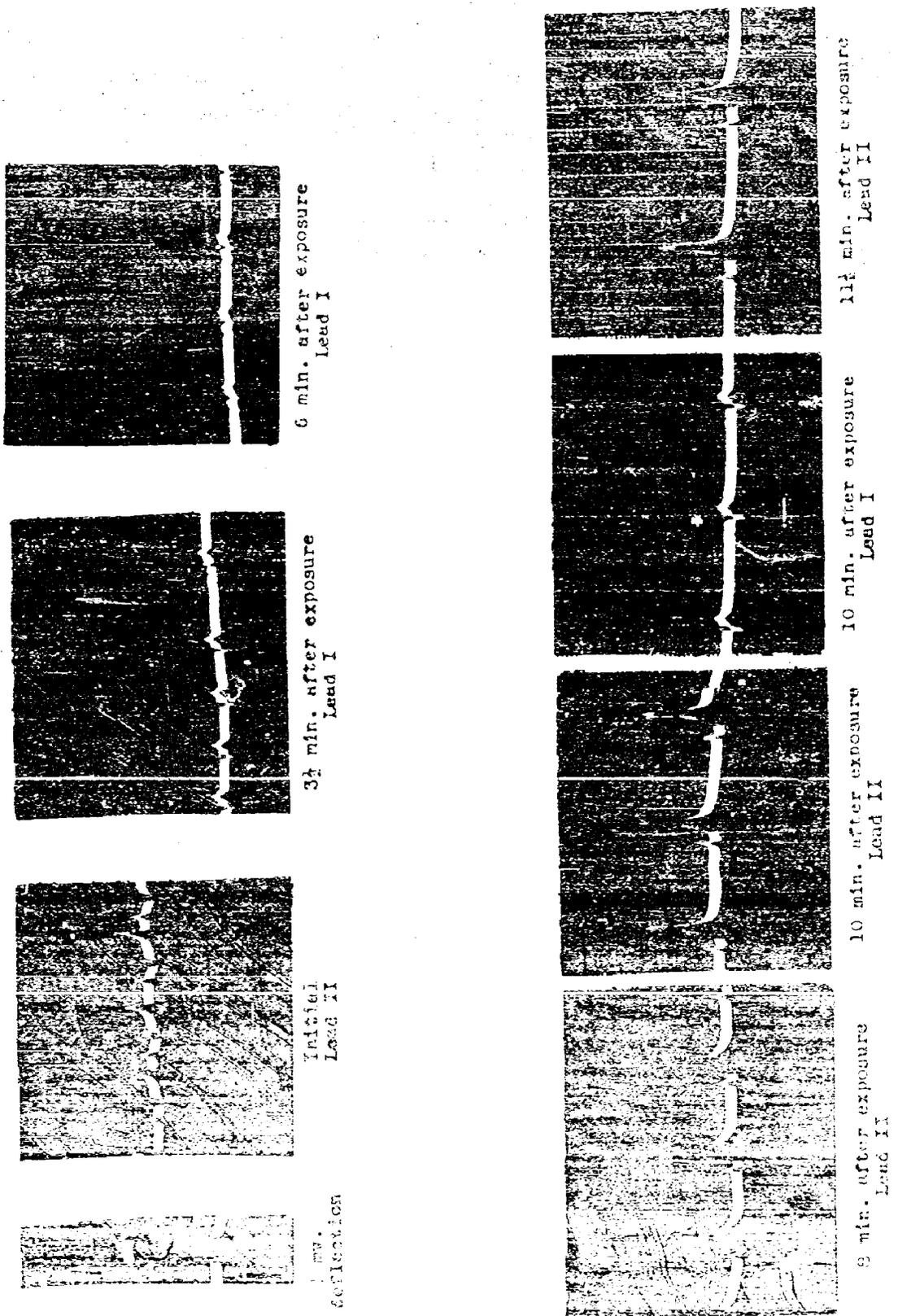
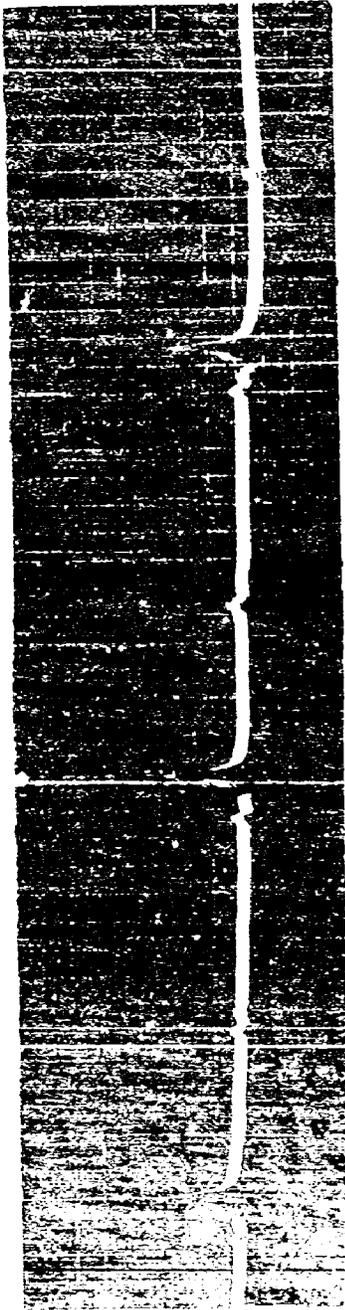


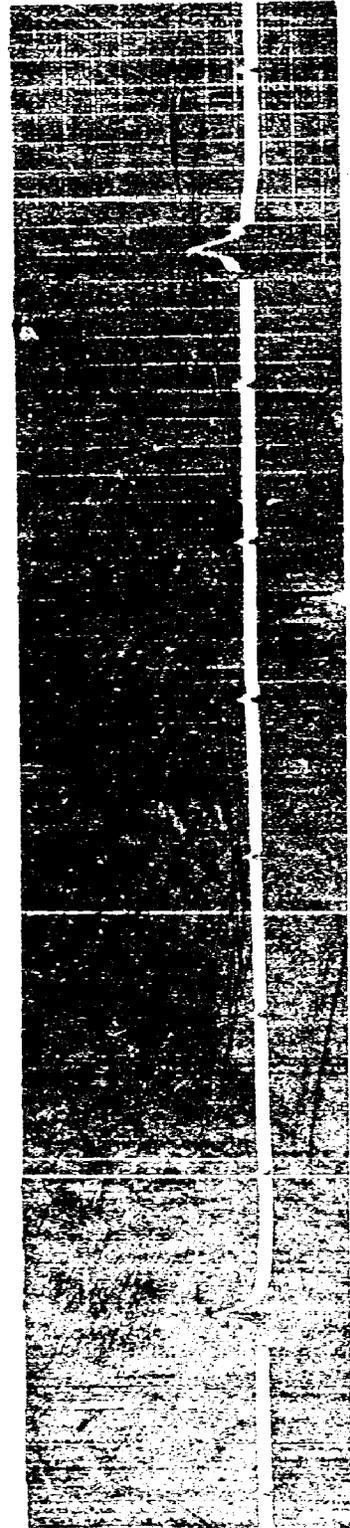
Figure 1b
ELECTROCARDIOGRAM OF GOAT DF 21



12 1/2 min. after exposure
Lead II

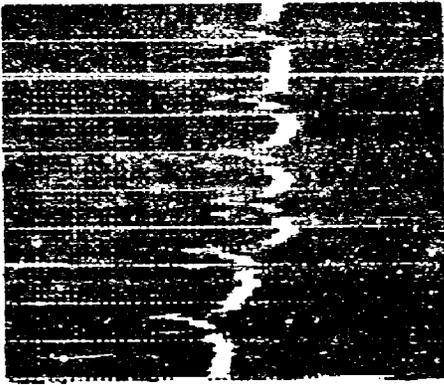


12 1/2 min. after exposure
Lead II

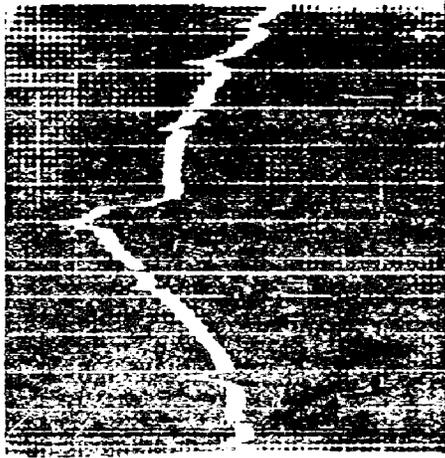


16 1/2 min. after exposure
Lead II

Figure 2
ELECTROCARDIOGRAM OF GOAT DF 28



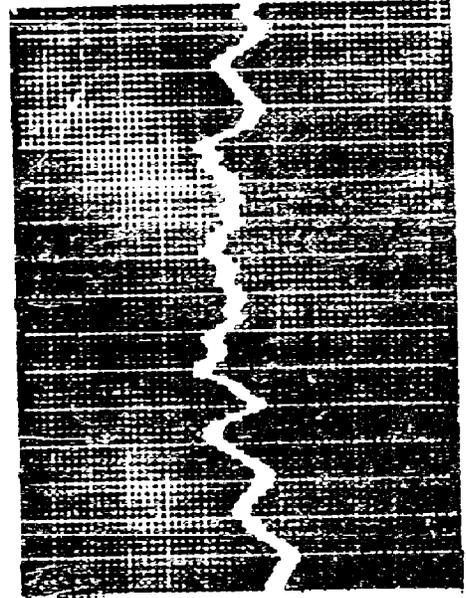
3 1/2 min. after exposure
Lead II



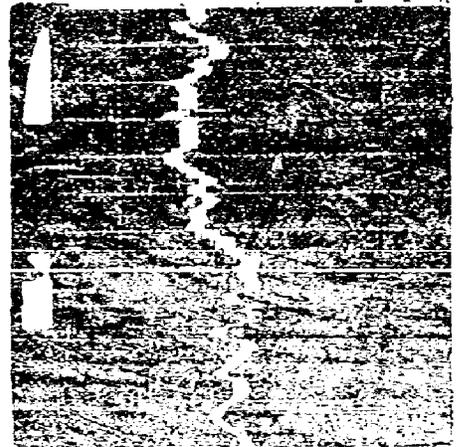
Initial
Lead II



1 mv.
deflection



12 1/2 min. after exposure
Lead II



11 min. after exposure
Lead II

The serum potassium concentration was always elevated after exposure to flame (Table VI). The average initial value for serum potassium was 17.13 mg./100 ml. and the range 12.5 to 33.7 mg./100 ml. (blood drawn from the jugular vein). Blood samples after death were taken as quickly as possible by opening the chest wall and withdrawing blood from each auricle. While sometimes in good agreement, the samples from the two auricles showed at other times marked differences in the concentration of serum potassium as well as other blood constituents. No explanation for this difference was apparent.

Only four goats (DF 25, 28, 30, and 31) had final serum potassium levels above 50 mg./100 ml., the level usually considered to be lethal.

The serum sodium concentration averaged 343 mg./100 ml. before exposure. The final samples (Table VI) varied from 389 to 286 mg./100 ml. and in many instances the terminal sodium concentration was lower than the initial.

The amount of hemoglobin in the serum (i.e., from hemolysis) did not parallel the potassium level although it varied widely, and was generally higher in the left auricle than in the right. The amount of hemoglobin in whole blood before and after exposure to flame is shown in Table VII. The concentration of hemoglobin increased after exposure, the increase varying from about one to fifty per cent.

A rough hematocrit determination was made in some experiments. The results (Table VII) showed wide variations after exposure to flame, and poor correlation with changes in the concentration of hemoglobin. The variation in plasma protein concentration before and after exposure can be seen from the data of Table VI. The changes were not marked, and there was no correlation between the concentration of plasma proteins and hemoglobin or the hematocrit after exposure to flame.

Gross pathological examination of 15 goats dying from exposure to direct flame showed obvious burning to the superficial layers of the skin and mucosa. The larynx was slightly edematous in a few goats and the trachea was occasionally hyperemic. The lungs of a number of the animals appeared to be hyperemic to a variable degree, and generally were cyanotic. There was no gross edema or hemorrhage.

Micropathology was confined almost entirely to the skin, which showed necrosis of the outer layers. The dermis was usually coagulated through at least the outer half, but only one section (from goat DF 28) showed necrosis of the entire thickness of the dermis. Examination of the heart, respiratory tract, spleen, liver, small intestines, adrenal, kidney and pituitary, showed very little beyond the minor pathology seen in the organs of a group of control goats considered to be in good health.

TABLE VII

Hemoglobin and Hematocrit in Goats Exposed to Flame

Trial or Goat No.	Hemoglobin (gm./100 ml.)			Hematocrit (rough - %)		
	Initial	Final		Initial	Final	
		L. Aur.	Rt. Aur.		L. Aur.	Rt. Aur.
DF 16	7.74	10.88	11.10			
DF 17	9.44*	12.31**				
DF 19	9.75	13.20	12.35			
DF 21	11.27	12.65	13.16			
DF 22	10.43	13.31	12.60			
DF 23	10.35	11.7	12.35	45	50	-
DF 24	10.2	12.4	13.0			
DF 25	7.20	7.86	7.42	30	30	40
DF 26	9.59	14.80	13.96	40	60	60
DF 27	8.14	8.84	9.72	45	40	45
DF 28	7.45	7.92	10.59	25	30	35
DF 29	9.27	8.20	9.40			
DF 30	9.28	8.79	11.80	40	40	50
DF 31	8.78	9.00	9.90	35	40	45
DF 32	9.90	10.40	10.12			
DF 33	10.3	12.6	-			
DF 34	9.68	12.0	12.0	40	55	50
DF 35	9.84	11.04	10.70			

*Femoral artery

**Carotid artery

III. DISCUSSION

A review of the results of the experiments described above, both with the aerial bomb charged with thickened gasoline and the flame thrower, reveals both similarities and differences. There was not, in either set of experiments, evidence that toxic gases or atmospheric anoxia played any significant part in producing casualties or death. In both sets, there was evidence that intense heat was the predominant lethal factor present.

The effects of exposure to flame from the aerial bomb were somewhat different, however, from the effects of exposure to flame from the flame thrower. The only explanation for these differences would appear to be in the different physical characteristics of the respective fuels or in their state of dispersion during combustion, since their chemical composition did not differ in any material degree.

Goats dying from exposure to the effects of the fire bomb, showed consistent pathology of the respiratory tract, such as laryngeal edema, tracheitis, and pulmonary congestion and edema. Goats killed by exposure to flame from the flame thrower, on the other hand, showed no respiratory tract pathology in 14 of 15 goats examined. In both sets of experiments the pathologists expressed the opinion that the immediate cause of death was not readily apparent from the anatomical lesions found on gross and micro pathological examination.

The lesions of the respiratory tract found in the first set of experiments could have been caused only by the inhalation of a heated atmosphere. It will be recalled that there existed for a few seconds after the burst of the fire bomb a "fireball" which was apparently composed of burning hydrocarbon vapor. In some animals there was a deposit of carbon which lined the trachea and extended through the bronchial tree. This would indicate merely the inhalation of smoke, but it is not unreasonable to assume that the smoke was very hot.

In the flame thrower experiments, there was no significant involvement of the respiratory tract. It should not be inferred, however, that this difference would always exist between the effects of fire bombs and flame throwers, since it appears to depend on physical phenomena which might well vary under slightly different conditions of exposure. In fact, Canadian investigators have reported finding pulmonary edema in goats subjected to flame attack in the open by flame throwers (9,10).

Physiological studies were not practicable in the fire bomb experiments, but observations of various sorts were made on goats exposed to the flame thrower. These observations were consistent with the hypothesis that heat was the sole important lethal factor in deaths resulting from direct exposure for a few seconds to flame. The two principal effects of heat which must be considered are: (a) local cutaneous burns; and (b) general increase in body temperature.

Moritz and his group described two mechanisms of death in pigs exposed to heat (7). When the animals were heated slowly, death occurred as a result of peripheral vascular collapse with attendant progressive hypotension. In brief exposures to high air temperatures, there was circulatory failure of central rather than peripheral origin. The cause of the central circulatory failure was attributed to the rapid release of potassium from erythrocytes in heated cutaneous and the subcutaneous tissues and the consequent damaging effect of the potassium on the heart.

The goats exposed to the flame thrower did not fall clearly into either class. The heart action in some animals stopped before or at about the same time as the respirations. Two of the goats in this group had serum potassium values above the presumed lethal level and one (DF 28) an electrocardiogram (Fig. 2) which showed ventricular fibrillation that might have been caused by the hyperpotassemia. In other goats, the respirations ceased well in advance of the last heart beat, and all of the electrocardiograms were, in the terminal phase, of the anoxic type. Only one goat of this group had a high serum potassium value.

It would appear from these data that in some cases at least, the liberation of potassium into the blood stream is an important mechanism in death from exposure to flame. It is not, however, the only mechanism since most of the goats which were killed by flame did not show a final serum potassium concentration at or above the lethal level, and since respiratory arrest preceded cardiac arrest.

The data on the goats do not allow an accurate evaluation of the role of peripheral vascular collapse. The presence of shock, however, was indicated by the hemoconcentration (Table VII) which was observed with fair regularity.

Thus, while no definite statement can be made as to the exact cause of death in animals exposed to the effects of flame in the open, it appears from the data on hand that heat per se is the primary lethal factor, that respiratory involvement may or may not be present depending on the particular conditions of the conflagration, but that respiratory involvement is not a necessary accompaniment of fatal exposure.

One can, therefore, generalize that long (15 minute) exposure to an air temperature of 80°C. can kill by causing acute peripheral vascular collapse (Moritz et al.), short exposures (30 seconds) to temperatures of 500°C. can kill by causing a rapid release of potassium into the blood stream (Moritz et al.), and that exposures on the order of 5 seconds to temperatures approximating that of flame (these experiments) can kill by several mechanisms which include hyperpotassemia, hyperthermia, and shock.

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RESTRICTED

THE TOXICOLOGY OF FIRE

PART III. The Creation of Toxic Atmospheres by Fire

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Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART III. The Creation of Toxic Atmospheres by Fire

SUMMARY

Experiments in which flame throwers charged with petroleum fuel were fired into a poorly ventilated structure (a Japanese-type bunker) are described. In portions of the structure where burning fuel did not penetrate, the oxygen concentration of the atmosphere was temporarily reduced to levels as low as zero, carbon monoxide reached as high as 3.5%, carbon dioxide was high as 10%, combustible hydrocarbons as high as 10%, and air temperature as high as 375°C.

Goats exposed to these alterations in the atmosphere frequently died, and there was a fair correlation between the animal mortality and the severity of the changes in the atmosphere as measured by the integrals of the gas concentration:time curves and air temperature:time curves.

Physiological examination of selected goats dying from exposure to flame attack in the bunker revealed in the majority of cases the presence of enough carbon monoxide in the blood to be considered lethal. There was also a deficiency of blood oxygen, a rise in rectal temperature, and hemoconcentration. Pathological examination revealed pulmonary edema in many of the goats. The significance of these changes is discussed.

It is pointed out that in conflagrations encountered in civilian life similar atmospheric conditions may prevail.

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART III. The Creation of Toxic Atmospheres by Fire

I. HISTORICAL

Part I of this paper pointed out the practical importance of flame warfare in World War II, and the desirability, from the military point of view, of gaining more information as to the mechanisms, other than contact with burning fuel, by which flame produced casualties or killed. Flame warfare was especially effective in the attack of enclosed fortifications such as dugouts and bunkers, and it was noted in both the European and Pacific Theaters that the occupants of fortifications subjected to flame attack were sometimes killed without visible evidence of being burned.

These observations led to the hypothesis that flame might kill or produce casualties by virtue of heat (radiant and circumambient) or by the creation of a toxic atmosphere, i.e., one deficient in oxygen and containing toxic constituents such as carbon monoxide, carbon dioxide, hot particulate matter, steam, and possibly irritant gases resulting from the partial decomposition of petroleum fuel.

The relative importance of these lethal or casualty factors could not be assessed on a priori grounds or investigated on the field of battle, so a program of experimental investigation was set up as described in Part I of this paper. It was felt that heat should be the decisive casualty-producing factor under conditions of good ventilation, such as flame attack in the open, where there would be little opportunity for the accumulation of toxic gases or a deficiency of oxygen. Part II of this paper describes experiments which established the correctness of this assumption. The present paper describes experiments which show that a toxic atmosphere can be created by fire burning under conditions of poor ventilation and that injury or death may result from exposure to such atmospheres.

II. EXPERIMENTAL

A. Materials and Methods

The experiments to be described were carried out by firing a flame thrower at a Japanese-type log bunker so that burning fuel entered the bunker through one of its embrasures. The bunker was built mostly below ground, and had 8 embrasures opening approximately one foot above ground level. Each embrasure had an area of 1 sq.ft. The only other opening was a rear door approximately 3.5 by 5 feet, which led into the bunker through a baffled passage. The volume of the bunker was 3400 cu.ft., and it can be characterized as a poorly-ventilated structure.

The interior of the bunker was divided into three compartments by means of baffles but the compartments were not closed off from one another by doors. Burning fuel fired into one compartment was retained in that compartment by the baffles, but flame and smoke penetrated to the other compartments. Reasons of military security have prohibited the reproduction of a drawing of this bunker, and it is, therefore, impossible to visualize the experimental setup exactly. However, the flame thrower was fired into the central compartment of the bunker. Goats and sampling equipment were placed in each of the adjacent compartments at positions which may be labelled Position 1 and Position 8. Goats in these positions usually did not sustain severe skin burns when the flame thrower was fired into the central compartment of the bunker.

The goats at Positions 1 and 8 were tethered adjacent to sampling points which consisted of a thermocouple for measuring air temperature, and a pipeline for drawing off samples of air for analysis. As described in Part I of this paper, continuous records could be obtained of the air temperature, and of the concentration of oxygen, carbon monoxide, carbon dioxide, and combustible hydrocarbons, before, during, and after a shoot. In a few experiments, where small quantities of fuel were used, goats were exposed at Position 5, a location closer to the embrasure at which the flame thrower was aimed than were Positions 1 and 8. No measurements were made of air temperature or gas concentrations at Position 5.

Two fuels were used in these studies. Both were composed of petroleum hydrocarbons, but differed in viscosity. For purposes of description, the one fuel will be characterized as "thick" and the other as "thin" fuel. Varying degrees of change in the atmosphere of the bunker were produced by varying the amounts of each of these fuels fired into the bunker.

The procedure followed in each trial was to place the goats in the desired positions, start the sampling apparatus, and fire the flame thrower.

As soon as the analytical instruments indicated that the atmosphere was again essentially normal, usually about five minutes after firing, an exhaust fan was moved into the rear doorway and the residual smoke was quickly exhausted. The bunker was then entered and the animals removed. Those dead on removal or dying within ten minutes after removal were arbitrarily classified as having been killed by the exposure. Those surviving ten minutes after removal were arbitrarily classified as surviving the exposure. Any survivors which suffered burns or other injury were painlessly destroyed on the spot.

Thirty-one goats which died as a result of exposure to flame attack were selected for physiological study in order to correlate the instrumental measurements on the atmosphere with the effect of the atmosphere on the animals. Rectal temperatures were taken and blood was drawn from the jugular vein just before the animals were placed in the bunker prior to firing. Immediately after removal from the bunker, the rectal temper-

ature and another blood sample were taken. This final blood sample was taken from a carotid artery exposed by rapid dissection. Each 10 ml. blood sample was placed in a tube containing 200 mg. of potassium oxalate, stirred well, and placed on ice in a dark box. The arterial samples were collected under mineral oil and determinations were always completed on the day of the trial. The analytical methods are described in Part I of this paper. Selected animals were also examined for gross and micro-pathology.

B. Results

The firing of the flame thrower into the central compartment of the bunker resulted under the conditions of these experiments in essentially complete combustion of the fuel, and produced at Positions 1 and 8 a rise in air temperature which reached a maximum of 400°C., a fall in oxygen concentration to a minimum of zero; a rise in carbon monoxide to a maximum of 3.5%, a rise in carbon dioxide to a maximum of 10%, and a rise in the concentration of combustible hydrocarbons to a maximum of 8.5%. The time trend of changes in air temperature, oxygen concentration, carbon monoxide concentration, and combustible hydrocarbon concentration are shown in Figs. 1 to 4, which show the extremes and average change in forty trials in which the same quantity of fuel, thick or thin, was fired into the bunker.

It was recognized that two factors were probably involved in the effect of the alterations in the atmosphere on the experimental animals, namely, the intensity and duration of the abnormality.

Attempts were made to correlate animal mortality with the peaks of the temperature, time and gas concentration-time curves, but the correlations were not impressive. It was then decided, following the reasoning presented in Part II of this paper, to integrate the curves mechanically by means of planimeters and thus determine Tt and Ct products which might prove to be more significant with respect to animal mortality.

The curves of the individual trials which made up the composite curves of Figs. 1 to 4 were integrated from $t=0$ (time of initial deflection) to $t=4$ (4 minutes after initial deflection). Four minutes was chosen because it was felt that the gas and temperature values were so close to normal after 4 minutes in these particular trials that integration over longer time intervals would not be significant.

There was of course a time lag in pulling the gas samples through the sampling line but this was corrected for by taking $t=0$ at the time of the instrumental indication of the first change in composition of the atmosphere after firing.

The Tt and Ct values obtained are represented in the histograms of Figs. 5 to 8. In these, the concentration of carbon monoxide and of combustible hydrocarbons is measured as volumes per cent above zero, the concentration of oxygen is measured as change in volumes per cent below 20,

OXYGEN CONCENTRATION IN BUNKER Z TO Z+4 MINUTES FIELD TRIALS 30-70

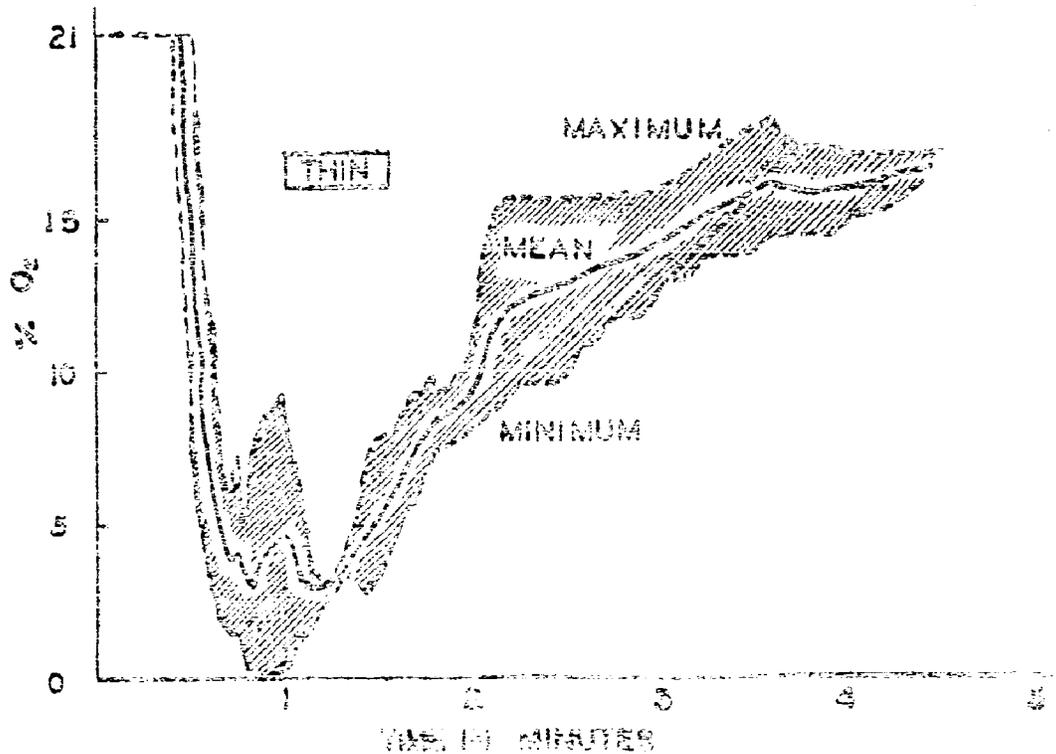
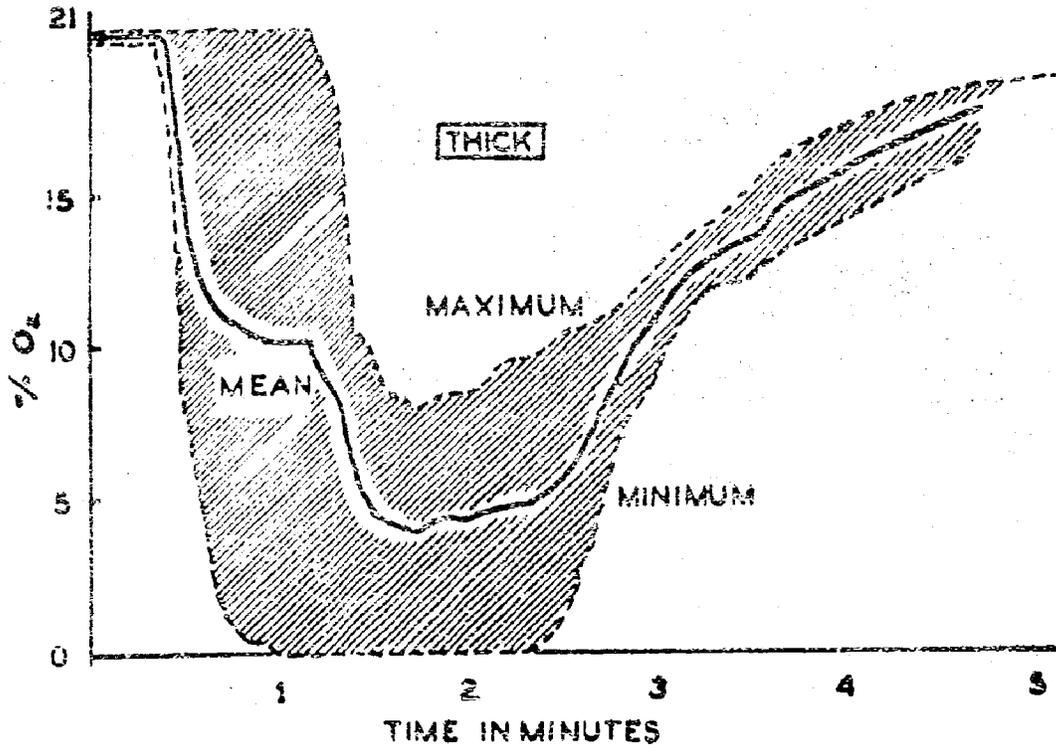


FIGURE 1

COMBUSTIBLES CONCENTRATION IN BUNKER Z TO Z + 4 MINUTES FIELD TRIALS 30-70

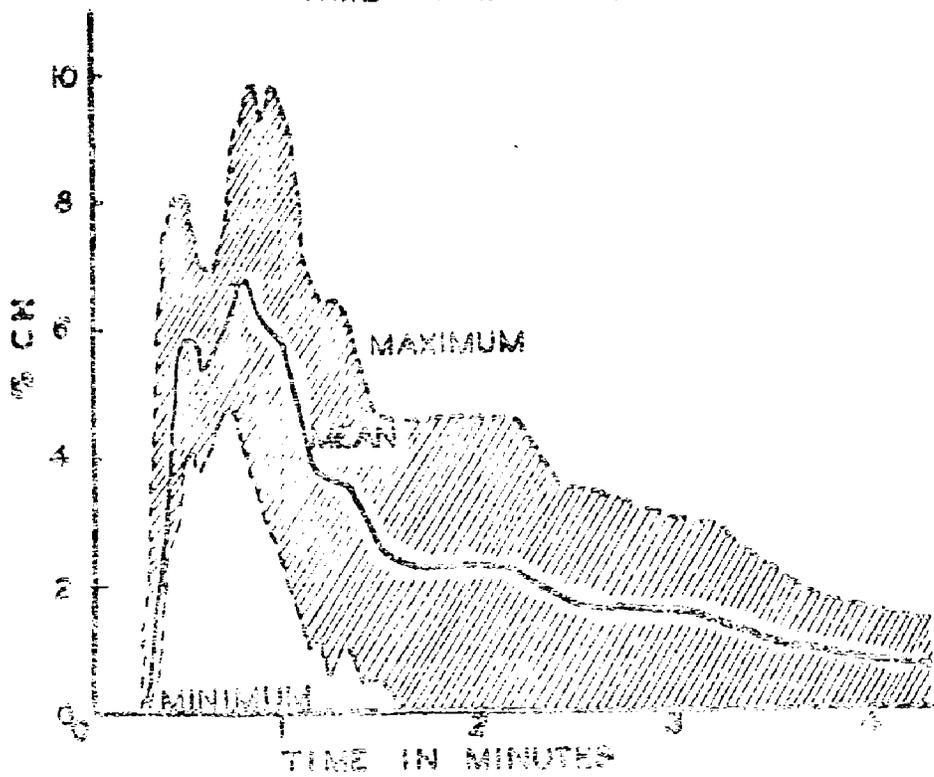
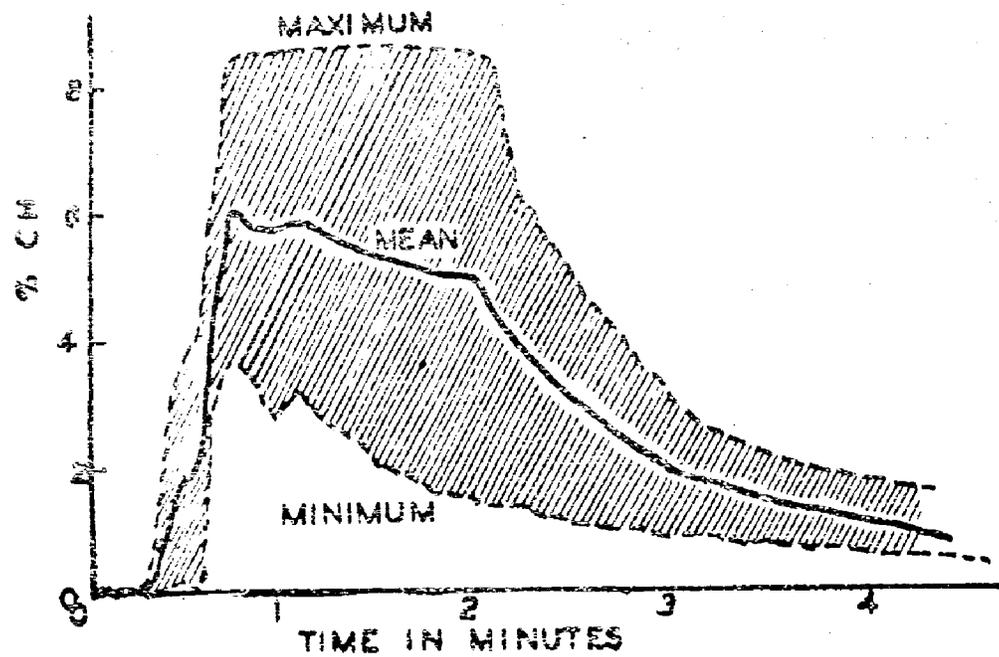


FIGURE 2

CARBON MONOXIDE CONCENTRATION IN BUNKER Z TO Z + 4 MINUTES

FIELD TRIALS 30 - 70

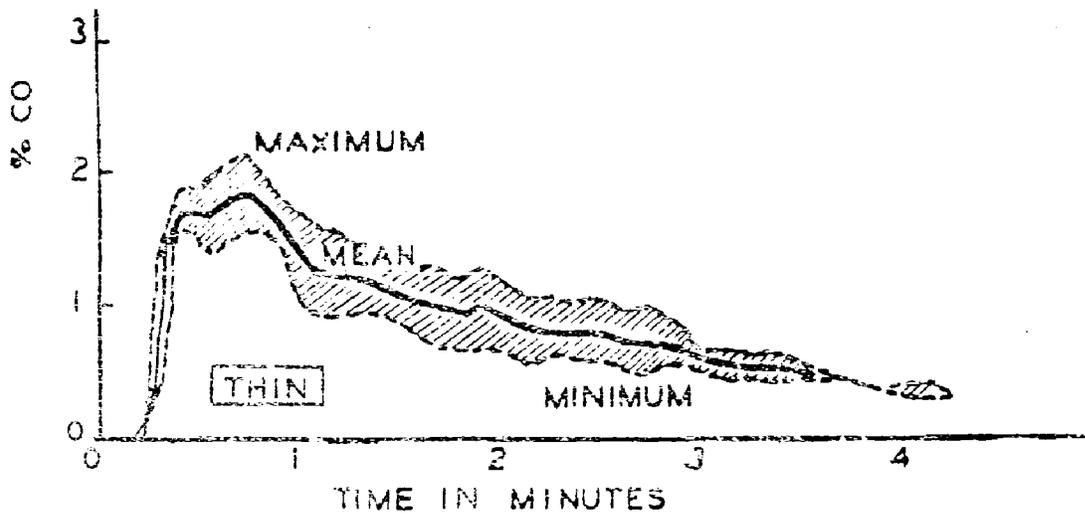
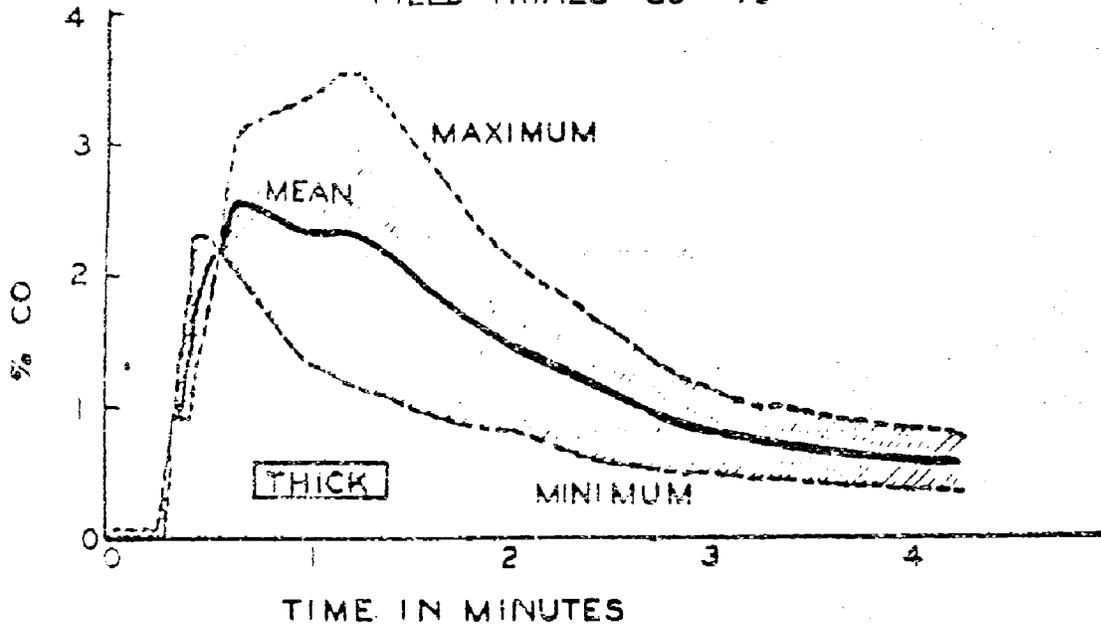


FIGURE 3

AIR TEMPERATURE IN BUNKER Z TO Z+4 MINUTES FIELD TRIALS 30 - 70

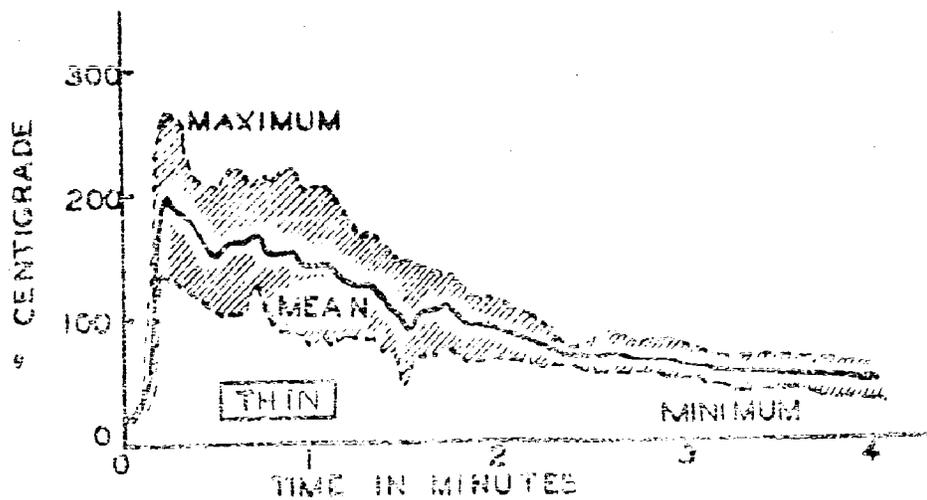
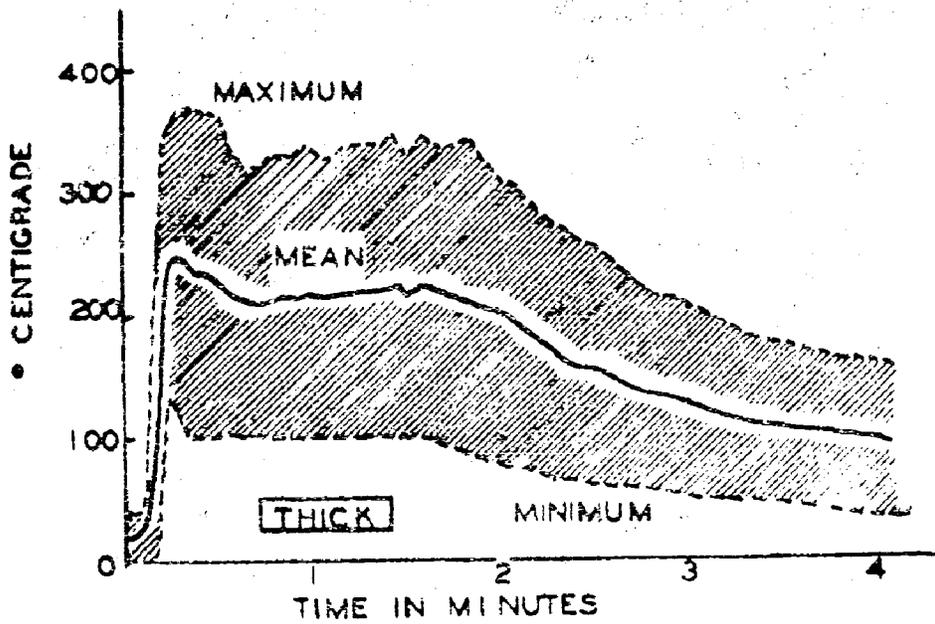


FIGURE 4

COMPARISON OF MEAN OXYGEN $\Delta C \times T$ IN BUNKER

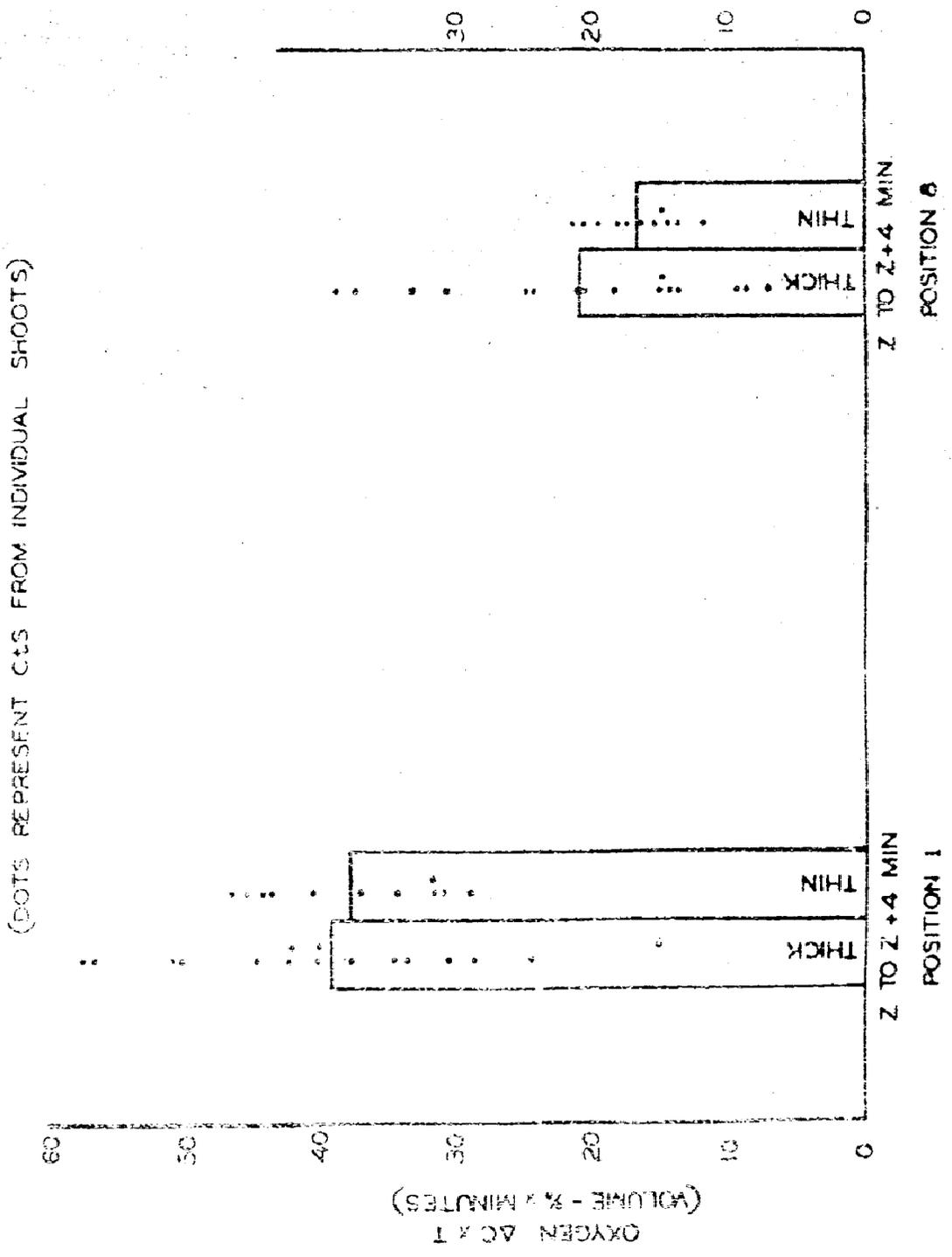


FIGURE 5

COMPARISON OF MEAN CARBON MONOXIDE CT IN BUNKER

(DOTS REPRESENT CTS FROM INDIVIDUAL SHOOTERS)

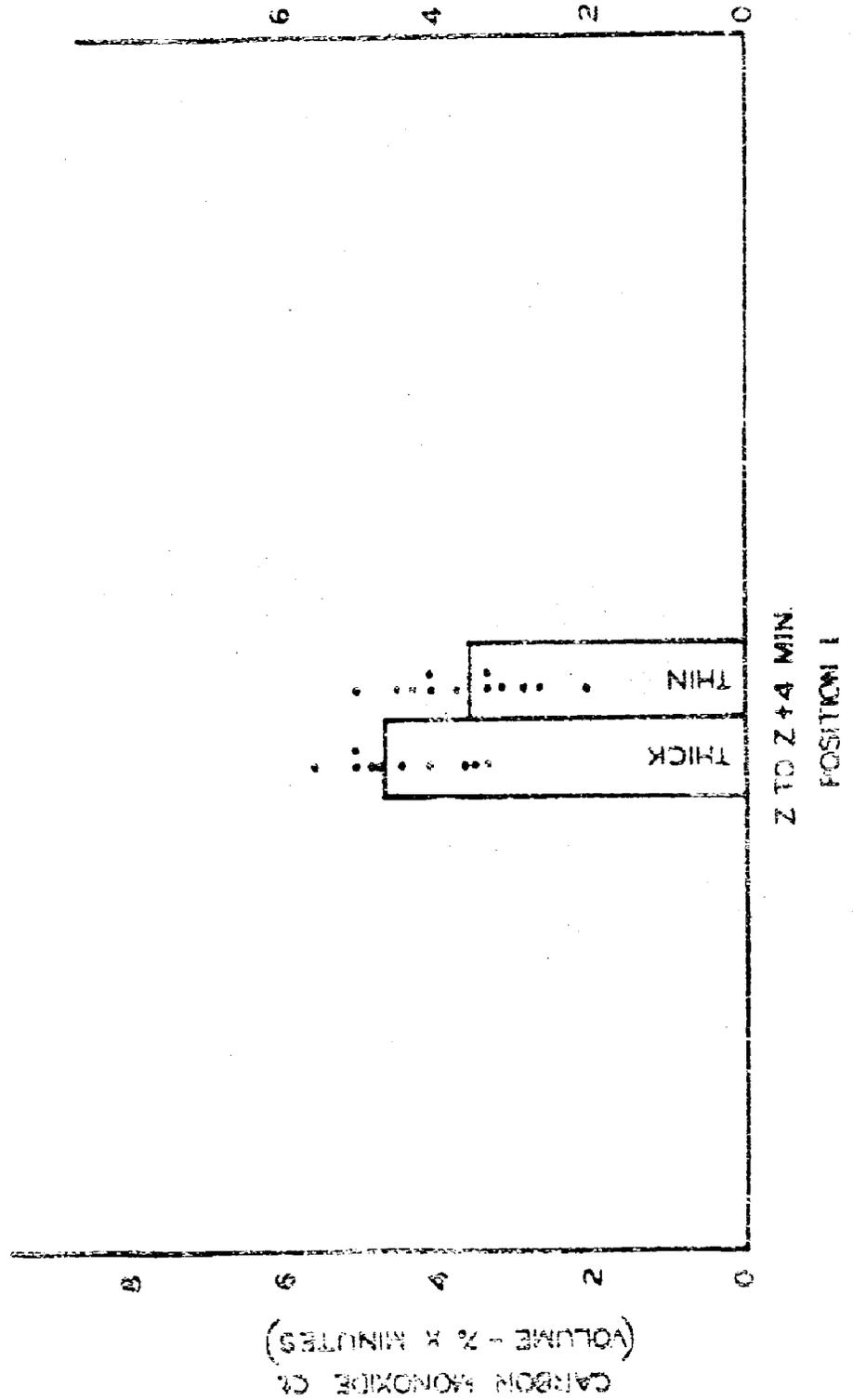


FIGURE 6

COMPARISON OF MEAN COMBUSTIBLES IN BUNKER

(DOTS REPRESENT CUS FROM INDIVIDUAL SHOOT)

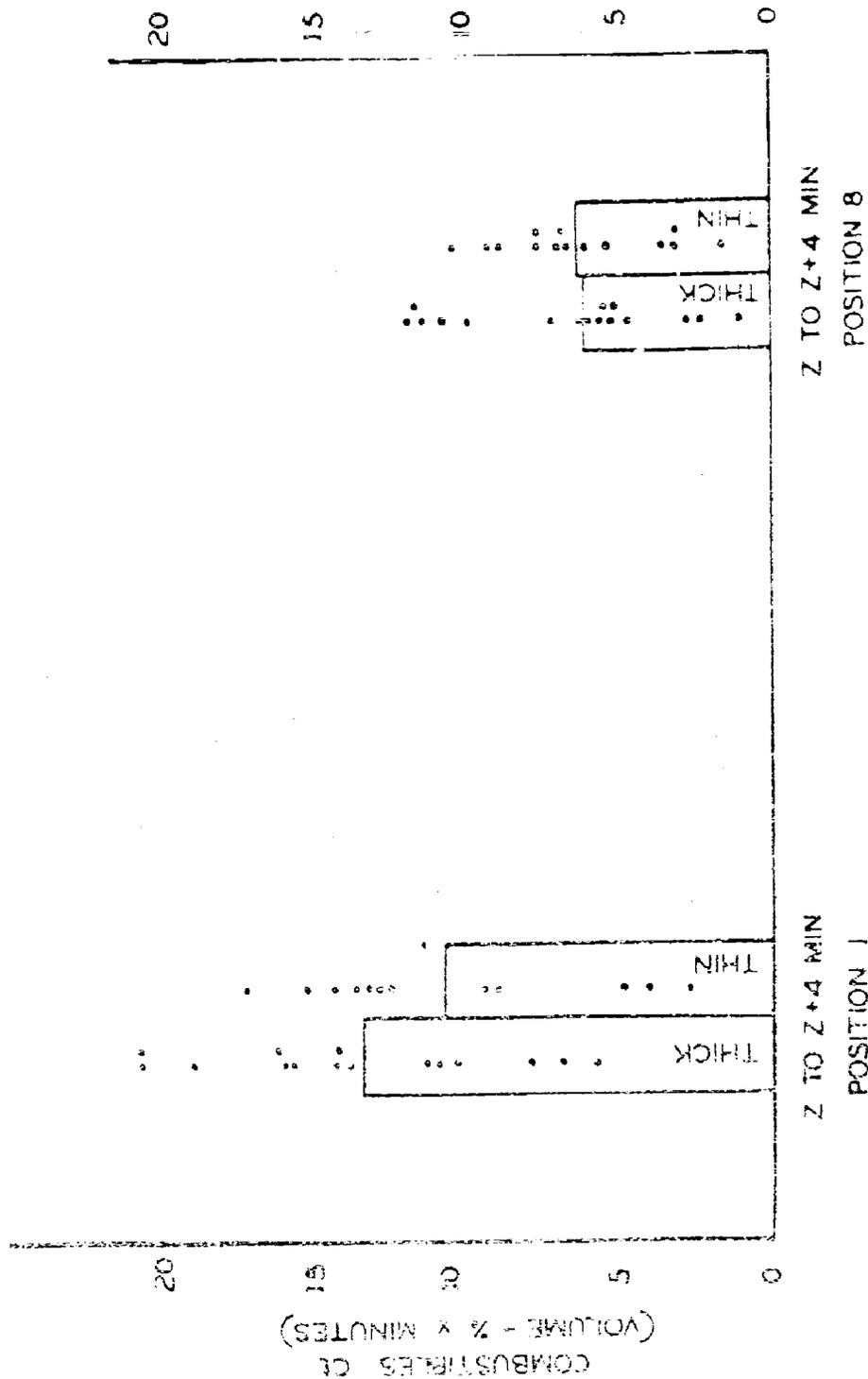


FIGURE 7

COMPARISON OF MEAN TEMPERATURE X TIME PRODUCT IN BUNKER

(DOTS REPRESENT T1'S FROM INDIVIDUAL SHOOTERS)

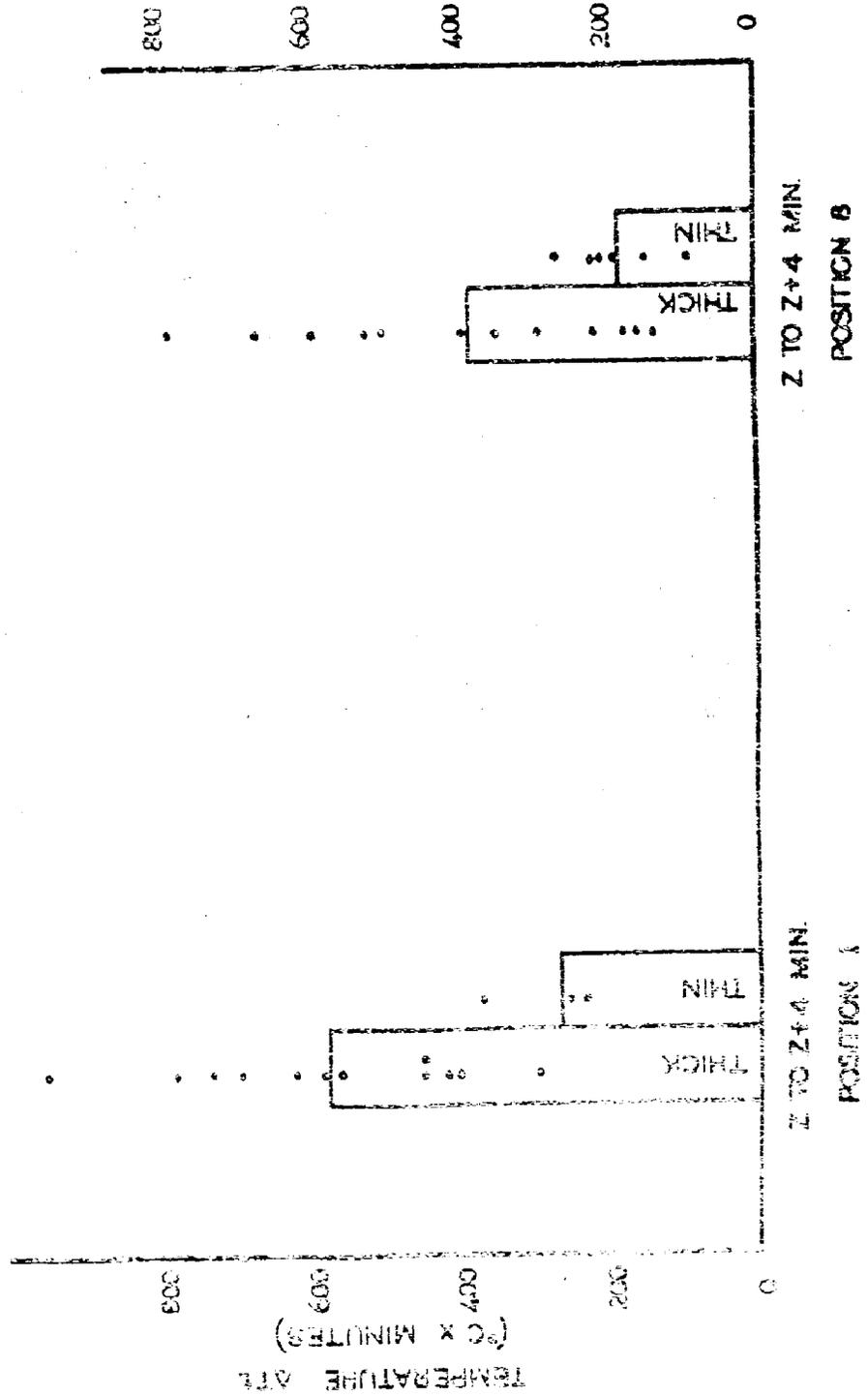


FIGURE 6

and temperature as °C. above zero, since the initial environmental temperature on the instrument scale could not be distinguished from zero °C. The height of each histogram represents the mean value for thick and thin fuels, respectively, and the superimposed dots show the scatter of the individual values. It can be seen from Figs. 5 to 8 that the integrals of the temperature:time and concentration:time curves were fairly consistent, and that there was no essential difference between the two fuels used so far as alterations in the atmosphere of the bunker were concerned.

No curves or histograms are shown for carbon dioxide. This gas was not measured in many of the trials because apparatus for its continuous analysis was not available. The maximum peak CO₂ concentration, when measured was approximately 10% and the average peak approximately 7%. As with the other gases, the concentration of carbon dioxide fell to essentially normal levels within 4 minutes.

The effect of the changes in the atmosphere on animal mortality can be seen more clearly in another group of experiments in which the amount of fuel fired into the bunker was varied so that a wide range in the severity of conditions was produced. The data are shown in Tables I and II. It had been decided by this time that the continued measurement of combustible hydrocarbons could not provide any further valuable information so data are not given for this variable. On the other hand, the integrals of the carbon dioxide concentration:time curves (CO₂ Ct's) are given. In each field trial, there resulted a set of gas Ct's and a Tt in two positions, Position 1 and Position 8. Two goats were exposed in each position in every trial so that there occurred a goat mortality of 2/2, 1/2, or 0/2 corresponding to each set of Ct's and Tt.

The correlation between the gas-temperature values and goat mortality is shown in Figs. 9 to 12. It can be seen that there was a fair correlation between each of the variables measured and animal mortality. However, the variables heat, oxygen, carbon monoxide, and combustible hydrocarbons are highly correlated with one another, so that the correlation between any one of the variables and goat mortality cannot indicate a necessary cause and effect relationship between that particular variable and death. These factors will be discussed in more detail in a subsequent paper.

The effect of fatal exposure on the goats, as revealed by changes in the blood and organ pathology, gives some information about the causes of death. Thirty-one goats which were dead on removal from the bunker were studied for changes in carboxyhemoglobin, oxyhemoglobin, total hemoglobin concentration, and rectal temperature. Twenty-one of these were examined for gross and micropathology, as were also two additional goats which were alive but moribund when removed from the bunker, and which were sacrificed.

TABLE I
Thick Fuel

Field Trial No.	Goats Killed Pos.1 Pos.8	CO		ΔO_2		CO ₂		T (°C.)	
		Vol-% x min. Pos.1 Pos.8	T x min. Pos.1 Pos.8	T x min. Pos.1 Pos.8					
130	2/2	13.60	3.19	117.5	63.8	65.5	45.3	1343	1164
144	2/2	15.20	12.94	174.7	88.6	91.1	58.7	1884	1913
145	2/2	7.80	4.44	111.7	58.6	50.6	32.9	1305	953
136	2/2	9.16	4.6	112.8	71.1	59.4	32.8	1375	392
135	2/2	14.82	4.9	154.0	69.0	74.1	32.5	1624	927
146	2/2	11.2	3.2	130.3	62.4	65.4	33.0	1648	---
147	2/2	12.5	11.0	148.3	113.2	89.4	67.1	1739	2223
158	2/2	7.88	2.84	93.2	50.2	43.0	28.9	1465	1155
172	2/2	13.01	3.85	142.6	86.1	50.4	34.6	1655	1016
175	2/2	14.56	12.58	112.5	115.5	45.3	57.1	402	1264
176	2/2	9.62	2.27	122.2	52.2	50.5	21.0	873	464
155	0/2	0	0	0	0	0	0	0	0
157	2/2	4.7	1.6	69.7	34.8	36.3	16.6	937	397
140	1/2	5.4	3.13	73.3	45.8	40.8	26.7	844	520
141	2/2	4.1	1.75	64.0	24.6	30.1	14.0	660	439
142	1/2	3.24	1.84	55.8	29.2	38.6	15.2	747	440
143	1/2	2.64	1.21	47.3	26.3	27.8	18.7	542	348
157	1/2	6.19	2.77	76.6	41.9	40.8	25.2	1025	1072
148	2/2	3.5	2.0	35.8	33.0	35.4	17.6	739	521
149	2/2	7.3	2.3	67.0	48.1	43.0	20.3	---	514
156	2/2	4.93	2.74	67.1	41.1	35.7	22.5	959	835
160	2/2	5.57	2.13	81.4	50.4	49.0	32.6	1254	1161
162	2/2	4.21	2.40	70.0	33.4	42.3	21.7	969	1107
159	2/2	4.93	2.79	87.8	45.9	37.2	22.5	1146	1121
173	2/2	8.25	1.53	100.6	31.6	43.6	13.4	1346	523
174	2/2	6.73	2.68	109.8	63.8	45.8	27.5	533	691
150	0/2	2.7	1.8	37.1	36.4	25.6	16.0	588	357
151	0/2	1.6	.9	28.5	18.9	16.8	10.1	407	265
169	1/2	3.27	.84	46.7	12.2	18.8	5.7	551	243
170	2/2	4.68	1.49	59.2	33.7	26.0	13.9	473	253
153	0/2	3.1	2.1	54.0	42.3	26.3	18.0	763	514

TABLE II

Thin Fuel

Field Trial No.	Gents killed		CO		Δ O ₂		CO ₂		T (°C.)	
	Pos.1	Pos.8	Vol-% x min. Pos.1	Vol-% x min. Pos.8	Vol-% x min. Pos.1	Vol-% x min. Pos.8	Vol-% x min. Pos.1	Vol-% x min. Pos.8	T x min. Pos.1	T x min. Pos.8
112	2/2	1/2	4.71	4.56	89.4	64.3	-	-	867	852
119	2/2	1/2	14.5	4.1	129.1	71.9	85.2	29.5	1636	996
120	2/2	2/2	15.6	9.2	141.8	81.2	99.1	45.2	1870	1167
127	2/2	1/2	11.5	5.3	100.5	56.0	98.7	31.8	1561	620
128	2/2	2/2	12.9	5.6	126.7	69.4	103.9	32.7	1651	1034
109	0/2	0/2	0	0	-	-	-	-	0	0
115	0/2	0/2	-	-	-	-	-	-	0	16
110	0/2	0/2	.85	0	8.3	-	-	-	83	32
114	0/2	0/2	2.67	.88	37.9	15.8	-	-	369	269
116	0/2	0/2	1.82	.63	27.7	12.3	-	-	300	236
111	2/2	0/2	3.26	1.89	50.6	-	-	-	-	661
113	2/2	0/2	3.97	2.37	74.7	38.6	-	-	773	676
117	2/2	0/2	3.02	4.39	109.3	65.1	-	-	1095	937
123	2/2	0/2	7.9	3.4	99.4	57.4	74.3	32.0	1054	810
125	2/2	0/2	8.3	3.4	89.6	45.3	59.3	18.6	1153	765
126	2/2	0/2	6.8	2.7	67.6	29.8	42.2	13.7	859	561
121	1/2	1/2	6.2	3.3	67.9	39.5	39.7	20.0	771	548
122	1/2	1/2	5.1	1.7	61.8	29.4	36.6	5.4	736	375
132	2/2	1/2	16.5	2.3	123.6	28.6	-	11.2	1299	394
134	2/2	0/2	17.3	2.6	139.9	28.3	-	12.3	1777	414
161	2/2	0/2	9.80	1.67	84.1	24.8	29.6	11.6	1590	743
130	2/2	0/2	5.2	1.7	49.8	20.8	25.2	12.5	414	245
133	1/2	0/2	4.4	1.7	54.6	19.6	-	8.8	585	242
164	2/2	0/2	8.49	1.90	88.7	34.3	22.4	15.3	1517	560
166	2/2	0/2	9.08	1.76	94.1	28.8	38.9	15.1	1890	598
163	2/2	0/2	3.59	1.39	44.2	17.8	22.0	7.7	511	259
165	2/2	0/2	5.23	1.00	53.7	17.8	22.4	8.6	489	275
167	2/2	0/2	19.52	2.49	159.2	35.7	71.4	17.4	1145	593
166	2/2	2/2	21.21	3.12	184.2	44.7	82.6	20.1	978	657
171	2/2	2/2	14.99	3.54	160.7	58.6	54.1	31.6	1955	765

FIGURE 10

RELATION BETWEEN OXYGEN
Ct AND GOAT MORBIDITY

O = Thin Fuel
X = Thick Fuel

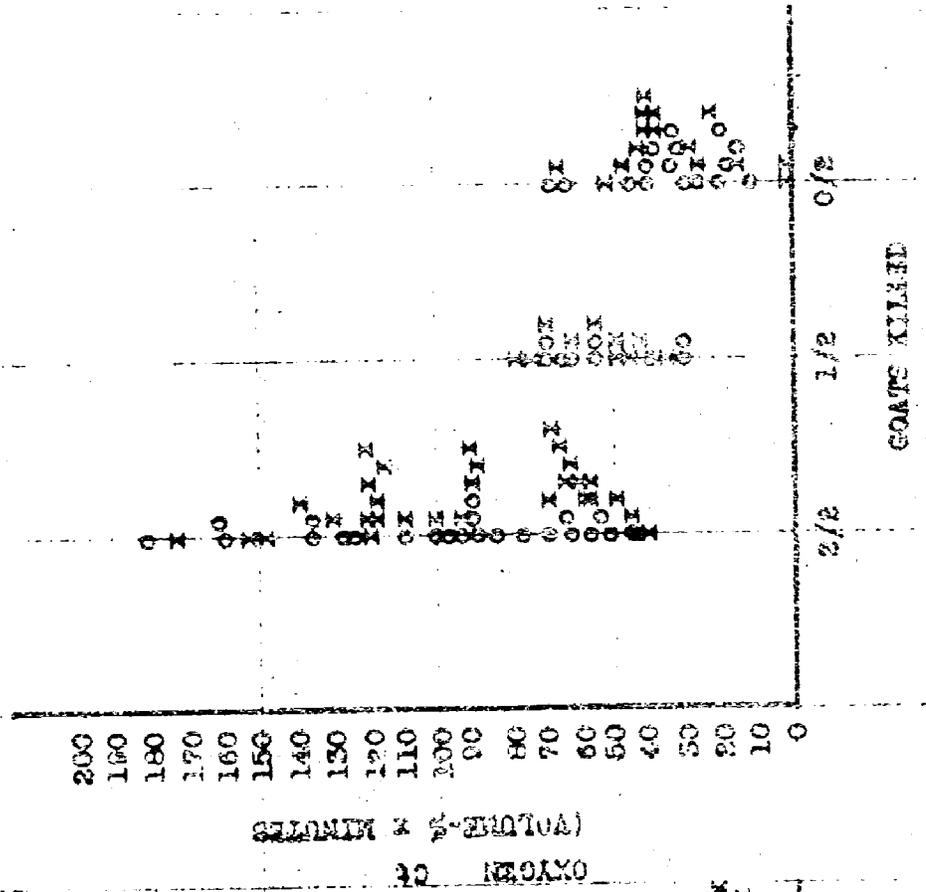
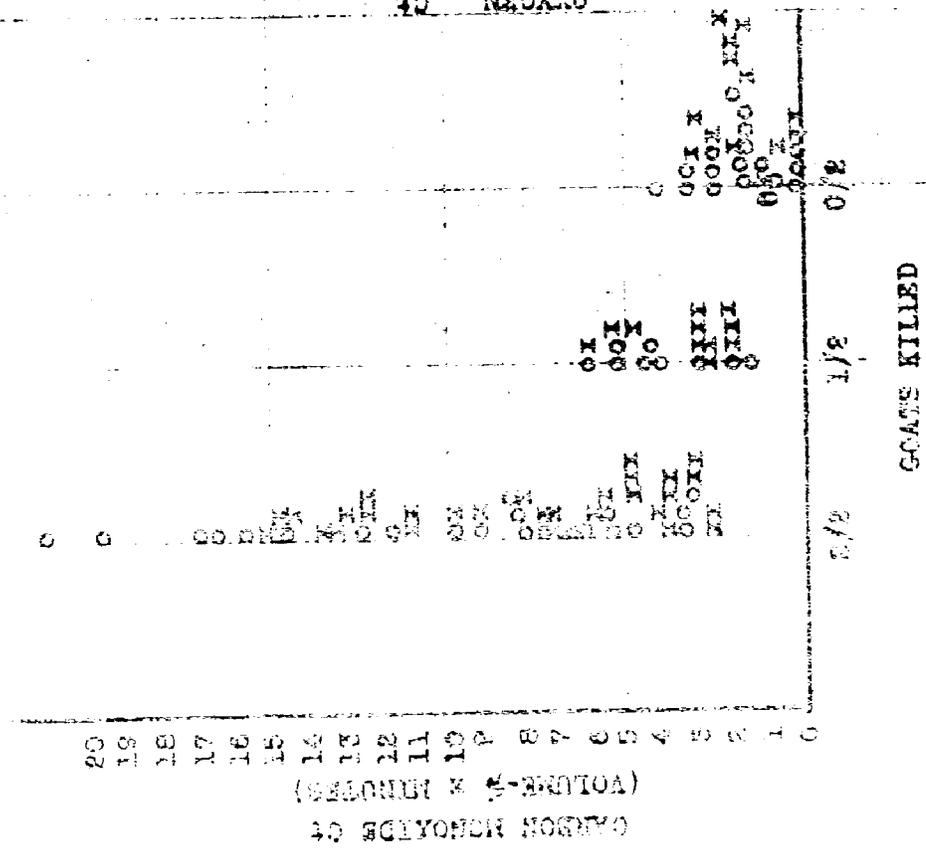


FIGURE 9

RELATION BETWEEN CARBON
MONOXIDE Ct AND GOAT MORBIDITY

O = Thin Fuel
X = Thick Fuel



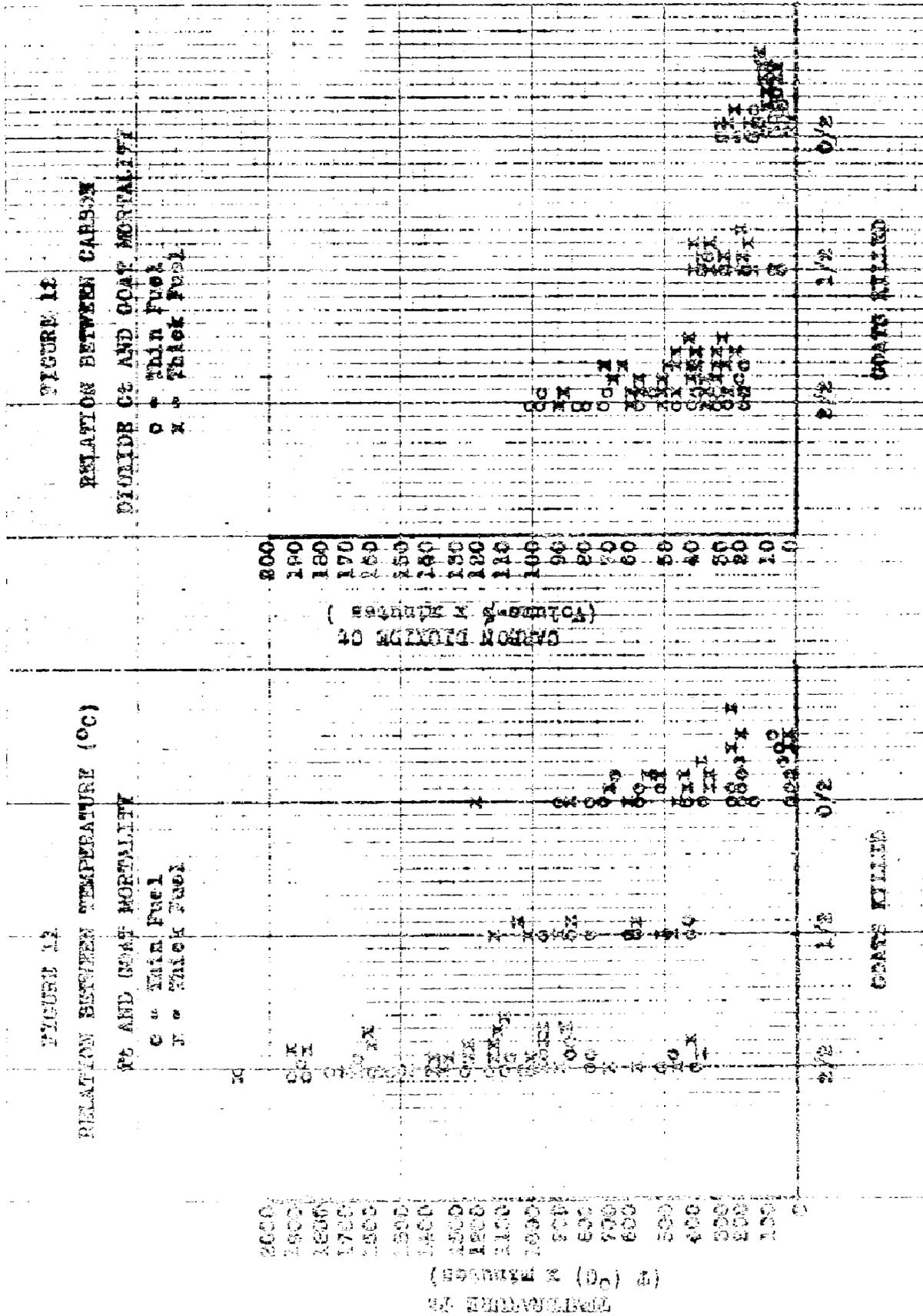


Fig. 13 shows the amount of carbon monoxide in arterial blood (% HbCO). The height of the histogram indicates the average % HbCO, and the individual observations which make up the average are shown as superimposed points. The per cent carboxyhemoglobin was calculated from the equation:

$$\% \text{ HbCO} = \frac{C_{\text{CO}}}{C_{\text{p}}} \times 100$$

where C_{CO} was the carbon monoxide content of the blood (in volumes per cent) and C_{p} the oxygen capacity of the blood (in volumes per cent).

It can be seen from Fig. 13 that there was no substantial difference in the % HbCO found in goats dying at Position 1 and Position 8, but those dying at Position 5 had considerably less HbCO. It will be recalled that Position 5 was closer to the flame than the others and that, therefore, heat was probably the dominant lethal factor in that position.

The amount of oxygen in the arterial blood (% HbO₂) is shown in Fig. 14. There is good agreement in the values obtained at Positions 1, 8, and 5, and all values are of doubtful significance since it is possible that there was utilization by the tissues of oxygen in the blood if respirations ceased before circulatory arrest or of uptake of oxygen by the blood if the animals breathed pure air before dying. Fig. 15 shows the per cent change in the hemoglobin content of the blood. There was usually some hemoconcentration although a few goats showed a decrease in hemoglobin content. The histogram shown by solid lines for Position 5 shows the average per cent hemoconcentration if the extreme value of 62.1% is excluded. The dotted extension of the histogram shows the effect on the mean of including this extreme value. The per cent hemoconcentration was calculated as:

$$C = \frac{\text{Hb}_f - \text{Hb}_i}{\text{Hb}_i} \times 100$$

Where Hb_i is the initial total hemoglobin content (in gm./100 ml.) and Hb_f the final total hemoglobin content.

The increase in rectal temperature of the goats is shown in Fig. 16, from which it can be seen that there was considerable variation from goat to goat at Position 1, and a possible tendency for greater elevations at Position 5.

The methemoglobin content of four goats before and after flame attack is shown in Table III. The amount was small and insignificant initially and was not increased after exposure.

Gross and microscopic pathological examinations were made on twenty-one goats which died immediately as the result of exposure and on two which were moribund when removed from the bunker and which were sacrificed. Of the twenty-three animals, only three showed third degree skin burns, ten showed second degree burns, eight showed first degree burns.

Figure 13. Amount of Carbon Monoxide in Arterial Blood (% HbCO) of Goats Killed by Flame Attack

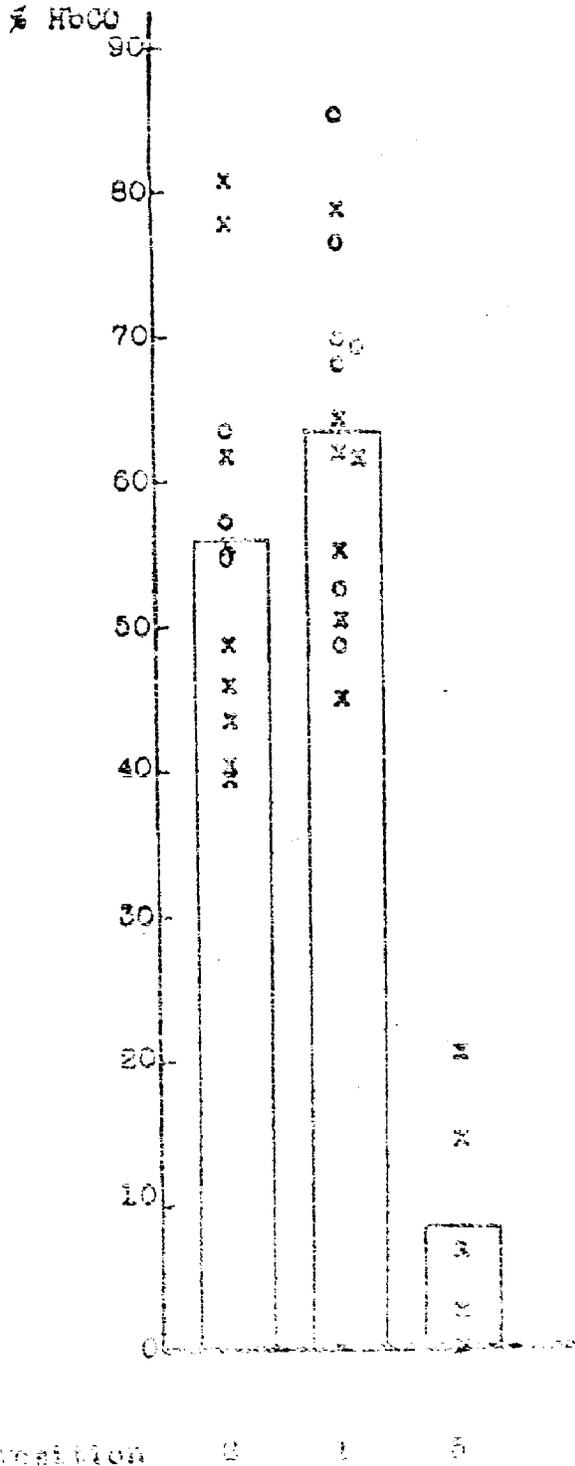
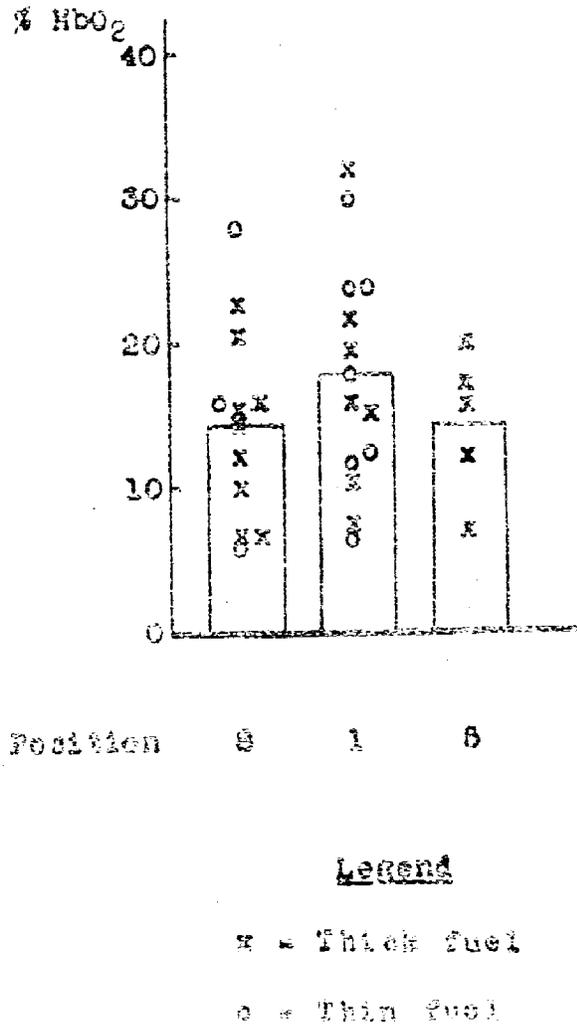


Figure 14. Amount of Oxygen in Arterial Blood (% HbO₂) of Goats Killed by Flame Attack



Legend

x = Thick fuel

o = Thin fuel

Figure 15. Percent Change in Hemoglobin Content of Blood of Goats Killed by Flame Attack

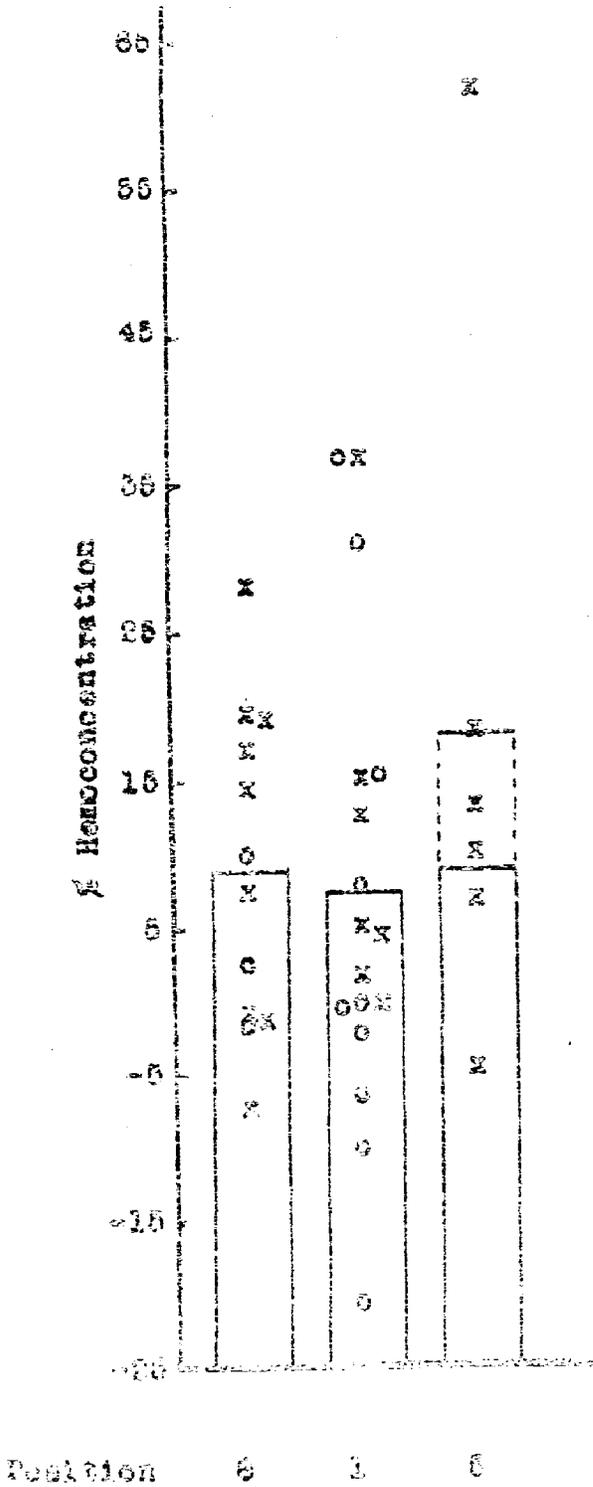


Figure 16. Increase in Rectal Temperature ($^{\circ}$ C) in Goats Killed by Flame Attack

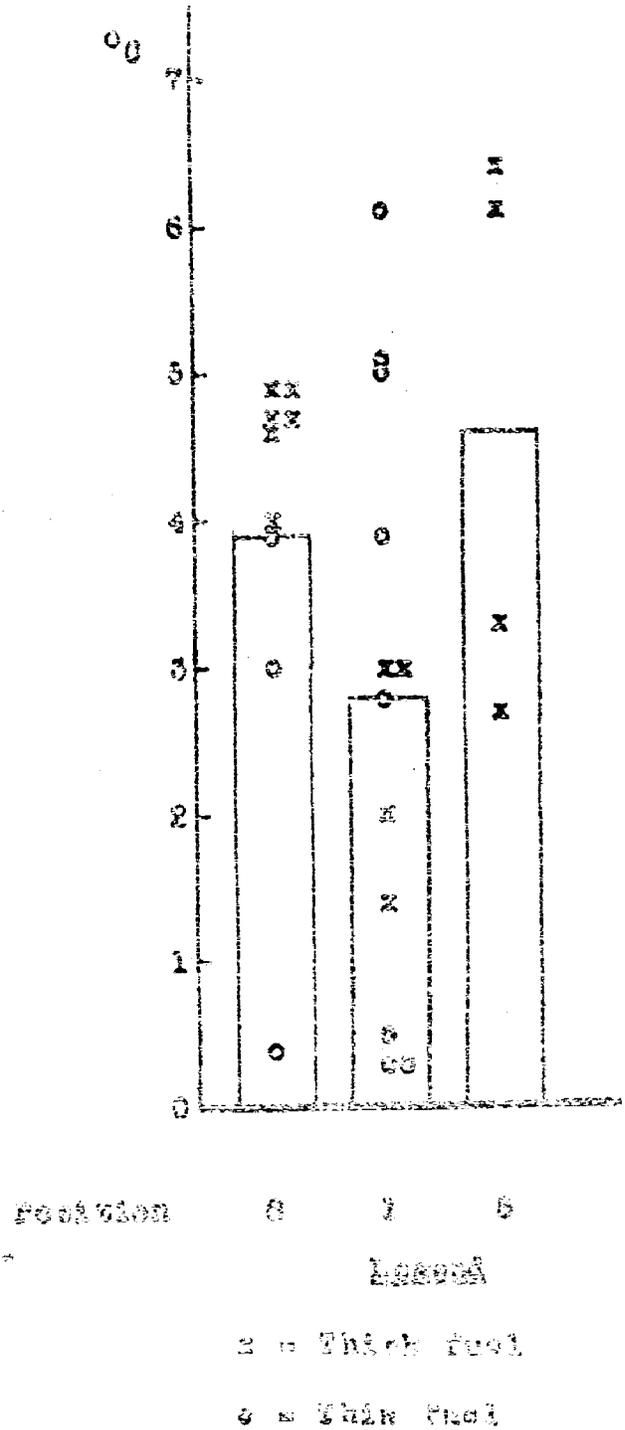


TABLE III

Methemoglobin Content of Goats Before and After
Exposure To Flame Attack

Animal No.	% Methemoglobin	
	Initial	Final
L4	1.82	0
L8	1.47	0.88
N3	1.34	1.57
N4	1.14	0.73

and two showed no evidence of skin burns. Aside from the skin burns, the remaining pathology was essentially confined to the respiratory tract. The lungs, grossly, showed edema and congestion varying from mild focal hyperemia and patchy edema of the lower lobes to severe, almost diffuse involvement of most of the parenchyma; in the more severe instances, the lungs were large, heavy, boggy, or almost "meaty" in consistence and reddish-purple in color, with considerable edema fluid exuding from cut surfaces, and frothy edema fluid in the bronchi and trachea. From a combination of gross and microscopic examinations, pulmonary edema was estimated as considerable or severe in eight of the twenty-three deaths, moderate in nine, and absent in six.

In about half the deaths in this group, there was slight to pronounced edema of the laryngeal mucous membrane resulting in partial, or, in the more severe instances, apparently complete laryngeal obstruction. The impression was that the instances of laryngeal edema were most likely to occur when considerable moisture was present in the bunker, which may have resulted in the generation of steam when the flame entered. The larynx and trachea contained deposits of carbon which often extended for a considerable distance distally.

Significant gross changes were absent in other organs of the animals in the group examined, except for the frequent occurrence of a cherry red color of the tissues. The brains were not examined.

III. DISCUSSION

It is a matter of general knowledge that a quietly burning flame, such as a lamp or candle, will be extinguished if the oxygen concentration of the atmosphere falls below 15%. This fact has been utilized in mine rescue operations as a test for the presence of an atmosphere containing sufficient oxygen to support life. Moritz et al.(1) likewise demonstrated that when gasoline in large open trays was statically ignited in a poorly ventilated space, combustion ceased when the oxygen concentration fell to a minimum of 14.6%.

When burning petroleum fuel is fired under pressure into a poorly ventilated space, such as a bunker or similar fortification, the conditions of combustion permit of a much more effective use of the atmospheric oxygen. It can be seen from Fig. 1 that the average minimum oxygen concentration in a series of forty trials was around 5%, and that it fell in some trials to a level not significantly different from zero.

The more efficient utilization of oxygen in the flame thrower experiments is also reflected in the concentration of the products of combustion. Where Moritz et al. (i) for example, found a peak carbon monoxide concentration of 1.4% in their experiment with statically ignited gasoline, the average peak in the forty trials with the flame thrower was around 2% and the maximum peak was 3.6%.

Complete combustion of petroleum fuel should result in the end products carbon dioxide, water, and heat. In this sense, combustion was not complete in the flame thrower experiments just described, since carbon monoxide and unburned hydrocarbon vapor were both present in the atmosphere of the bunker, and both are products of the partial combustion of petroleum fuels. A detailed analysis of gas samples taken from the bunker was shown in Table I of Part I of this report.

Neither the peak carbon monoxide concentrations nor the minimum oxygen concentrations encountered in the bunker would be compatible with life if they persisted, but the duration of the most severe conditions was very short and atmospheric conditions usually returned to normal within four to five minutes. There was, moreover, no striking correlation between the peaks of the gas - temperature curves and animal mortality. It was, therefore, decided that a measure of dosage which took account of both the intensity and duration of the abnormality in the atmosphere should be calculated. Accordingly, following the reasoning presented in Part II of this paper, the gas-concentration-time curves were integrated and the areas expressed as Ct's (or Δ Ct in the case of oxygen). The air temperature-time curves were likewise integrated and the results expressed as Tt's (Figs. 5 to 8).

The data of Tables I and II, when plotted in Figs. 9 to 12, show a reasonably good correlation between the magnitude of the Ct's and Tt and goat mortality ($2/2$, $1/2$, or $0/2$). It can be seen that for each variable (oxygen, carbon monoxide, carbon dioxide, and air temperature) there is an upper limit above which both goats were always killed, and a lower limit below which no goat was ever killed. There was also an intermediate range in which the mortality was quite variable, as might be expected where only two animals were exposed at a time.

The data of Figs. 9 to 12 bring out a danger which is inherent in experiments of this type. The correlations between the respective Ct's or Tt and mortality do not permit the inference that any one of these was an important cause of death. This stems from the fact that all of the variables were highly correlated with one another, and hence

if any one causal factor showed a good correlation with mortality, all of the other factors would likewise show a good correlation by virtue of their dependent variation with the causal factor. The possibility of synergistic action must also be considered. For example, Henderson and Haggard (2) have listed the physiological effects of CO Ct's (recalculated in terms of volumes per cent x minutes) as follows:

<u>CO Ct</u>	<u>Effect</u>
1.5	No perceptible effect
3.6	A just perceptible effect
5.4	Headache and nausea
9.0	Dangerous

It will be noted from Fig. 9 that goats died at CO Ct's above 1.5 and that no goat survived a CO Ct above 5.5. The reason for the discrepancy between these results and those of Henderson and Haggard might possibly be in differences of concentration and duration of exposure since the rule $Ct=k$ does not usually hold over an extended time and concentration range, but the main reason is more likely to be the contributing effect of heat, anoxia, etc., to the mortality in the present experiments.

The physiological observations on 31 goats killed by exposure at Positions 1, 8, and 5 to the results of flame attack, provide some basis for speculation as to the toxic factors involved in the death of the animals.

Fig. 13 shows that the carboxyhemoglobin saturation averaged around 60% and ranged, in goats exposed at Positions 1 and 8, from 40 to 87%. Henderson and Haggard (2) also list the physiological effects associated with various amounts of carboxyhemoglobin in the blood, as follows:

<u>% CO Hemoglobin</u>	<u>Physiological Effects</u>
10	No appreciable effect, except shortness of breath on vigorous muscular exertion.
20	No appreciable effect in most instances, except shortness of breath, even on moderate exertion; occasionally slight headache.
30	Decided headache; irritable; easily fatigued; judgment disturbed.
40 to 50	Headache; slight confusion; collapse and fainting on exertion.
60 to 70	Unconsciousness; respiratory failure and death if exposure is long continued.
80	Rapidly fatal.
Over 80	Immediately fatal.

A comparison of this tabulation and the data of Fig. 13 shows that lethal levels of carboxyhemoglobin probably existed in goats exposed at Positions 1 and 8 in the average experiment and almost certainly existed in the extreme experiments.

If an atmosphere containing carbon monoxide was simultaneously deficient in oxygen, the lethal blood level of carbon monoxide would presumably be lowered. Fig. 14 shows that the amount of oxygen in the arterial blood was indeed low, a finding which is compatible with the oxygen deficiency which undoubtedly existed in the atmosphere. The figures for per cent oxyhemoglobin must be taken with reservations, however, since it is possible that they are too high (if oxygen was added to the blood by the breathing of relatively pure air before death) or too low (if oxygen was lost from the blood to the tissues by virtue of the occurrence of respiratory arrest prior to circulatory arrest).

In any event, it seems probable that the amount of carbon monoxide in the blood coupled with the deficiency in oxygen would be sufficient in the majority of cases to cause death without any reference to the effects of heat. The five goats dying at Position 5 (Fig. 13), however, had very little carboxyhemoglobin, and it appears likely that these were killed so quickly by exposure to heat that there was no opportunity for carbon monoxide asphyxia.

The heat exposure factor cannot be neglected for any of the exposures at Positions 1, and 8, however, since the majority of goats showed an increase in rectal temperature which averaged 3 to 4°C., and was only slightly less than the average increase in rectal temperature of the goats exposed at Position 5. These data, while establishing the fact that there was exposure to heat, do not allow too close interpretation since it was established in trials where goats were subjected to flame attack in the open that rectal temperatures do not always parallel the temperature of blood in the deep arteries or the heart.

Both carbon dioxide and combustible hydrocarbons were present in the atmosphere during the exposures, and these may have had some effect on the goat mortality. Evidence that will be discussed in more detail in a subsequent paper, however, lead us to believe that these gases were of secondary rather than primary importance.

In general, the goats which were dead on removal from the bunker showed some hemoconcentration (Fig. 15). Pathological examination of 23 selected goats generally revealed some pulmonary edema. This has also been observed in Canadian experiments with goats exposed to attack by flame throwers (3); in previously reported experiments in which goats were exposed to the effects of fire bombs (2); in some of the human victims of the Coconut Grove disaster (4,5); and in dogs exposed to the inhalation of steam (6).

The causes of pulmonary edema are many, but in the present experiments it was the impression that pulmonary edema was most severe in those experiments in which there was considerable moisture in the bunker, which may have been converted into steam.

These experiments establish the fact that dangerous or lethal atmospheres may be produced by the combustion of petroleum under conditions of imperfect ventilation, and it is believed that similar conditions of ventilation may be encountered in civilian disasters. Since the products of the incomplete combustion of petroleum (7), are qualitatively similar to those of other combustible materials listed by Easton (8), it would appear that the present observations may have general applicability to conflagrations involving limited ventilation and do much to explain the "extreme toxicity that appears to be displayed by fire gases under certain circumstances" (8).

On the basis of the experiments reported, heat, carbon monoxide, and a deficiency of oxygen appear to be the chief lethal factors. It does not seem to be necessary, therefore, to postulate the presence of other toxic gases such as nitrous fumes, phosgene, hydrogen cyanide, or hydrogen sulfide (although these may be present and significant in special circumstances) in order to account for the death of casualties in conflagrations where skin burns are absent, or appear to be of minor importance.

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RESTRICTED

THE TOXICOLOGY OF FIRE

PART IV. The Effect of Ventilation

RESTRICTED

RESTRICTED

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART IV. The Effect of Ventilation

SUMMARY

Experiments are described in which a flame thrower was discharged into an experimental building designed to be either poorly ventilated or well ventilated. With poor ventilation, 5 gallons of fuel killed 26 of 96 goats exposed and the evidence indicated that carbon monoxide and a deficiency of oxygen were the predominant lethal factors. Heat was probably also important, although the same amount of heat, uncomplicated by carbon monoxide or anoxia, did not, in another experiment, kill any of four goats.

When the same building was well ventilated, no goats were killed when quantities of fuel less than 16 gallons were fired. Heat was apparently the chief lethal factor under conditions of good ventilation.

Trials were also carried out in which a flame thrower was discharged into three abandoned mine tunnels. These experiments showed that the toxic gases of combustion can travel by convection a considerable distance from their source under favorable conditions. The travel of these gases, however, is interrupted or diverted by sources of vertical ventilation or by the interposition of relatively simple mechanical barriers.

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART IV. The Effect of Ventilation

I. HISTORICAL

It has been shown in Parts I, II, and III of this paper that the factors responsible for rapid death as a result of exposure to petroleum conflagrations are different depending on whether the conflagration takes place in the open or in an enclosed poorly-ventilated structure. Under conditions of poor ventilation, heat, carbon monoxide, and oxygen deficiency are the chief lethal factors, while heat alone is the dominant lethal factor in conflagrations in the open air. Death in these experiments, was rapid, frequently occurring within 10 minutes after exposure to the conflagration. Thus, the delayed death which often occurs after severe burns did not enter into the picture.

This paper will discuss some of the qualitative and quantitative effects of ventilation on the lethality of such petroleum conflagrations as produce rapid death.

II. EXPERIMENTAL

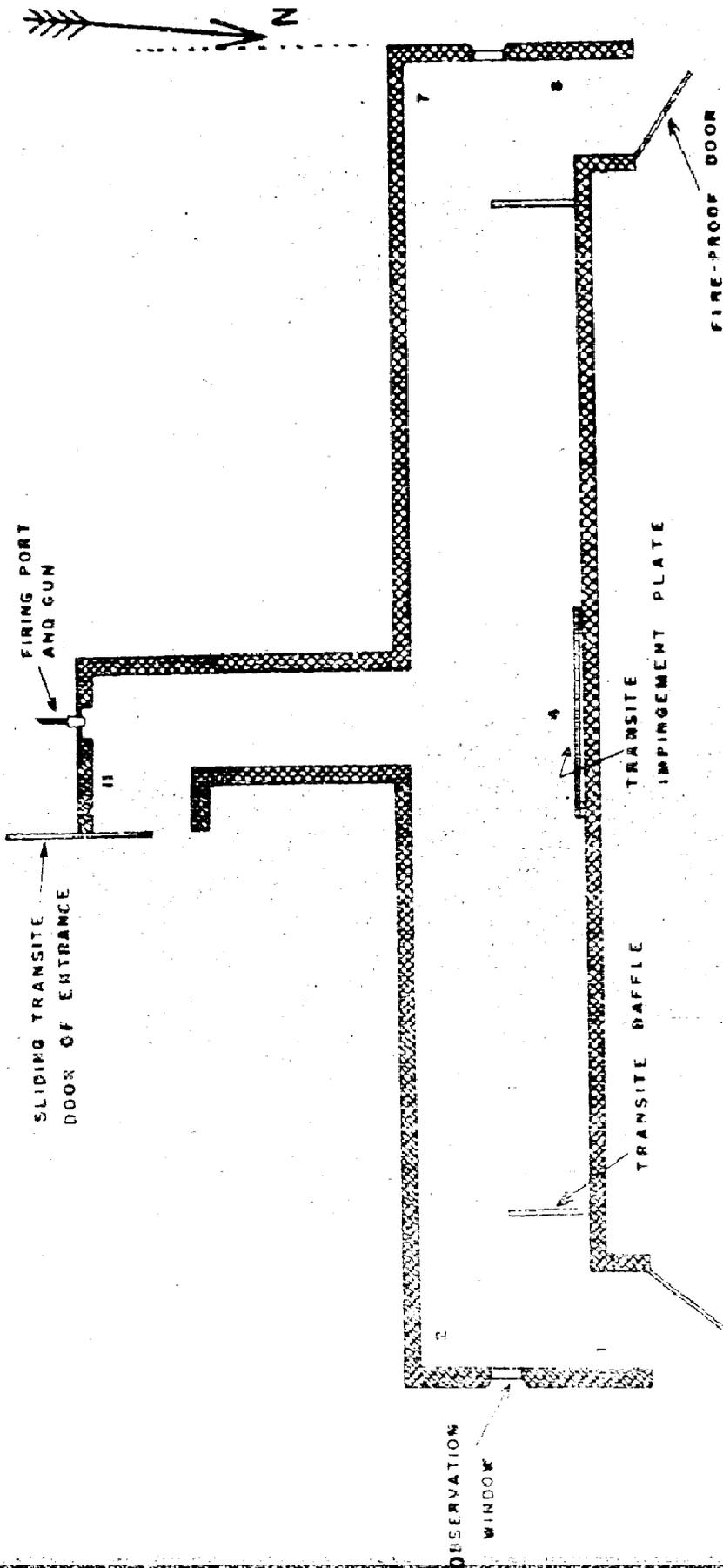
A. Materials and Methods

The experiments carried out were of two types: (a) Those in which burning fuel was discharged from a flame thrower into an experimental building whose ventilation could be varied; and (b) those in which burning fuel was discharged into mine tunnels having different ventilation characteristics.

The experimental building was erected in the shape of a T as shown in Fig. 1. It was constructed of concrete block, and provision was made for discharging the fuel into the base of the T as indicated in Fig. 1. Doors at Positions 1, 8 and 11, extending from floor to ceiling, could be opened to any desired degree in order to change the ventilation characteristics of the structure.

Actually, results with only two degrees of ventilation will be described. In the first case, which can be characterized as "poorly ventilated", the doors at Positions 1 and 8 were completely closed while the door at Position 11 was left partially open leaving a slit 20 inches wide extending from floor to ceiling. In the second case, characterized as "well ventilated", all three doors were fully opened.

Provisions were made for continuously recording air temperature, oxygen concentration, and carbon monoxide concentration at Positions 1, 2, 7 and 8, but it developed that no consistent differences in



EXPERIMENTAL BUILDING

FIGURE 1

atmospheric conditions were produced among these four positions, so that in most experiments samples were taken either from Positions 2 and 7 or Positions 7 and 8. The methods of continuously analyzing for oxygen, carbon monoxide and air temperature are described in Part I of this report.

Healthy adult goats, prepared as previously described (Part I of this paper) were tethered adjacent to sampling points at Positions 1, 2, 7 and 8 in some of the trials so that the atmospheric conditions could be correlated with animal mortality.

Preliminary experiments with the building "poorly ventilated" were carried out to determine the approximate quantity of petroleum fuel that had to be discharged from a flame thrower in order to produce barely lethal conditions at Positions 1, 2, 7 and 8. A number of trials were then conducted with this selected quantity of fuel. The doors of the building were then opened, and a further series of trials were conducted, in which the amount of fuel fired into the building was varied.

The second type of experiment involved firing fuel from a flame thrower into three mine tunnels of different natural ventilation characteristics. These tunnels had been bored into the sides of hills in the vicinity of Dugway Proving Ground, Utah, for the purpose of mining metals and were subsequently abandoned. They were predominantly horizontal, although in some cases as will be described, the tunnels communicated with vertical shafts. In structure, these tunnels were not unlike some of the caves which were defended by Japanese troops in the Pacific.

It was not practical to make continuous analyses of atmospheric gases or air temperature in the mine tunnels because of their inaccessibility, so that spot samples were usually taken with Bureau of Mines vacuum bottles, or by pulling air from the sampling point into collecting bottles by means of a 1/8 inch. i.d. pipe and a vacuum pump. The samples were subsequently analyzed either by the Bureau of Mines, or the Army Chemical Corps.

Since mine safety lamps and carbide lamps will not burn in atmospheres containing less than 16% and 12% oxygen, respectively (1), these lamps were used in all trials to determine whether the oxygen concentration had fallen below the respective critical percentages during a trial, as well as to determine the time after firing when sufficient oxygen was present to allow safe employment of the carbon monoxide absorbing canister of the MSA type N mask. The carbon monoxide concentration was roughly determined as desired with the Hoolamite apparatus(1).

Goats weighing 60 to 100 lbs. and prepared in the usual manner were employed throughout. Blood samples were drawn from the external jugular vein before the trials on some of the animals, and within five minutes after removal from the tunnel on most of the animals surviving the flame attack. Samples of blood were withdrawn from the left ventricle of all goats which were removed dead from the tunnels. All blood samples were immediately expelled from the syringes, under oil, into test tubes containing oxalate, and were stored in a cool thermos jug during transportation to the laboratory. All analyses were completed within 18 hours.

As in trials previously described in Parts II and III of this paper, the petroleum fuels used in the present trials varied little in chemical composition but rather markedly in viscosity, so that some could be characterized as "thick" and others as "thin". Since the data of previous trials failed to reveal any significant difference in the physiological effects of the "thick" and "thin" fuels, no distinction will be made among them in describing the present trials, either in the experimental building or in the mine tunnels.

B. Results

1. Trials in the experimental building (Fig. 1)

a. Trials with the building poorly ventilated

Twenty-four trials were carried out in which 5 gallons of fuel were fired directly into the building through an opening adjacent to Position 11 (Fig. 1). Oxygen concentration, carbon monoxide concentration and air temperature were recorded continuously at Positions 7 and 8 during and after the firing of the flame thrower until atmospheric conditions had returned to approximately their pre-trial status. Goats were exposed during the first 12 trials, 2 at each of Positions 1, 2, 7 and 8, or 8 goats per trial.

Following the reasoning presented in Parts I, II and III of this paper, the curves relating gas concentration to time, and air temperature to time following a trial were integrated mechanically by means of planimeters in order to obtain Ct and Tt products which experience had shown to be significantly related to animal mortality. The curves were integrated from $t=0$, time of initial instrumental deflection, to $t=x$, the time when the instruments showed an essentially normal atmosphere.

The results of the 24 trials are shown in Table I. The goat mortality in the first 12 trials was 26 of 96 goats exposed. As stated previously, only those goats were counted as dead which were dead on removal or died within 10 minutes after removal from the building. The CO Ct's, the ΔO_2 Ct's and Tt's associated with the 27% animal mortality are shown in Table I. The average CO Ct at Positions 7 and 8, averaged over all 12 trials during which goats were exposed, was 2.89, the ΔO_2 Ct 66.65, and the Tt 647.79. When the individual Ct's and Tt's were averaged over both Positions 7 and 8 and all 24 trials, the results were: CO Ct, 3.53; ΔO_2 Ct, 65.41; and Tt, 666.71.

b. Trials with the building well ventilated

Fourteen trials were carried out in which all 3 doors to the building were fully open. The flame thrower was fired from the same position as in the trials with poor ventilation, but the quantity of fuel fired was varied from 4 gallons to 24 gallons. In these trials, samples were taken at Positions 2 and 7, and one goat was exposed at each of Positions 1, 2, 7 and 8 in nine of the trials while one goat was exposed at each of Positions 2 and 7 or 1 and 8 in the remaining 5 trials. The results of the experiments in terms of animal mortality, CO Ct, ΔO_2 Ct and Tt, are shown in Table II.

TABLE I
Field Trials in Experimental Building (Poorly Ventilated)

Id	Goats Killed				CO Ct		ΔO ₂ Ct		T (°C.) x min.		
	P1	P2	P7	P8	Total	Vols.% x min. Pos.7	Pos.8	ΔVols.% x min. Pos.7	Pos.8	Pos.7	Pos.8
2	0/2	0/2	2/2	0/2	2/8	1.96	2.71	66.20	58.10	686.6	672.0
3	1/2	2/2	2/2	0/2	5/8	3.39	3.78	82.64	78.74	894.3	956.8
4	0/2	0/2	2/2	2/2	4/8	2.55	3.42	87.44	76.22	803.2	736.6
5	0/2	0/2	2/2	1/2	3/8	1.96	2.99	73.29	70.86	881.3	855.0
6	0/2	0/2	0/2	1/2	1/8	2.49	3.28	67.02	56.29	583.7	600.0
7	1/2	0/2	0/2	0/2	1/8	2.57	3.65	68.11	54.24	----*	591.7
8	0/2	0/2	0/2	2/2	2/8	3.00	3.65	73.95	64.97	505.7	636.5
9	1/2	1/2	1/2	1/2	4/8	2.40	3.30	67.24	63.64	560.3	611.6
10	0/2	0/2	0/2	0/2	0/8	1.76	2.53	59.07	58.54	543.86	619.51
11	0/2	0/2	1/2	1/2	2/8	2.93	3.62	73.64	64.12	518.57	580.42
12	0/2	0/2	1/2	1/2	2/8	2.77	2.94	58.34	55.48	463.23	517.07
13	0/2	0/2	0/2	0/2	0/8	2.57	3.19	57.75	62.59	566.0	578.86
14	-	-	-	-	-	3.32	5.68	66.78	68.14	713.6	702.4
15	-	-	-	-	-	3.55	4.50	65.27	69.87	713.6	691.2
16	-	-	-	-	-	3.99	4.26	70.80	63.44	798.4	702.4
17	-	-	-	-	-	3.41	4.45	65.67	64.06	821.1	680.0
18	-	-	-	-	-	3.45	3.15	56.67	43.76	----*	647.3
19	-	-	-	-	-	4.14	2.93	64.44	61.95	713.6	641.6
20	-	-	-	-	-	3.41	3.25	56.42	61.54	755.3	614.4
21	-	-	-	-	-	4.19	4.19	71.59	64.75	833.3	683.2
22	-	-	-	-	-	5.06	3.98	78.17	71.65	899.4	720.0
23	-	-	-	-	-	4.72	3.76	76.52	64.02	853.7	692.8
24	-	-	-	-	-	4.22	4.53	70.86	61.95	819.5	766.4
25	-	-	-	-	-	3.48	3.00	54.44	55.63	702.3	649.6

Instrument Failure

TABLE II
Field Trials in Experimental Building (Well Ventilated)

Field Trial	Gal. Fired	Goats Killed								CO Ct		Δ O ₂ Ct		T (°C.) x min.		Tt
		P1	P2	P7	P8	Total	Pos.2	Pos.7	Pos.2	Pos.7	Pos.2	Pos.7	Pos.2	Pos.7		
223	4	7	0/1	0/1	-	0/2	0.04	0.14	0.62	2.28	-*	141.0				
224	8	0/1	0/1	0/1	0/1	0/4	0.09	0.20	1.24	4.45	-*	254.4				
225	12	0/1	0/1	0/1	0/1	0/4	0.48	-	3.52	24.51	441.4	820.7				
226	16	0/1	1/1	1/1	1/1	3/4	0.96	-	-*	-*	781.7	894.0				
226	16	0/1	-	-	1/1	1/2	-	1.25	9.69	-	1045.0	1167.1				
227	20	1/1	1/1	1/1	1/1	4/4	0.34	3.42	4.34	56.56	859.8	1156.2				
227	20	1/1	1/1	1/1	0/1	3/4	1.57	1.55	8.27	22.33	1607.6	-				
228	20	0/1	1/1	0/1	0/1	1/4	0.28	0.14	4.96	1.55	1224.7	584.6				
229	20	0/1	1/1	1/1	0/1	2/4	0.11	0.14	2.79	1.96	1272.9	431.0				
230	24	0/1	1/1	1/1	1/1	3/4	0.58	1.55	8.89	-*	1697.2	652.9				
232	24	0/1	1/1	1/1	1/1	3/4	-*	-*	-*	-*	1112.8	1083.9				
234	16	0/1	0/1	0/1	0/1	0/2	1.75	0.80	16.47	18.56	570.5	542.9				
233	20	1/1	1/1	1/1	1/1	2/2	0.70	1.54	9.69	26.19	846.5	670.2				
235	20	1/1	1/1	1/1	1/1	2/2	1.66	1.21	15.67	23.51	662.0	710.1				

*Instrument Failure

These data are further summarized in Table III, in which goat mortality, average CO Ct, average ΔO_2 Ct, and average Tt are related to the quantity of fuel fired. It can be seen that the CO Ct and ΔO_2 Ct with any quantity of fuel up to 24 gallons did not approach the values obtained with 5 gallons of fuel fired into the same structure when poorly ventilated. CO Ct appeared to reach a limiting value when 16 or more gallons of fuel were fired, while ΔO_2 Ct appeared to reach a limiting value when 12 or more gallons of fuel were fired. Animal mortality increased with the amount of fuel fired, although no animals died when the quantity fired was less than 16 gallons. The Tt product increased directly with the quantity of fuel fired, from 4 to 24 gallons. When 12 gallons of fuel were fired into the well ventilated building, the Tt product was approximately the same as that obtained when 5 gallons of fuel were fired into the same building with poor ventilation.

TABLE III

Relation of Quantity of Fuel Fired to Effects Produced in Well Ventilated Building

Gals. Fuel	Goats Killed	Average CO Ct	Average ΔO_2 Ct	Average Tt
4	0/2	0.09	1.45	141.0
8	0/4	0.15	1.88	264.4
12	0/4	0.48	14.02	631.1
16	4/8	1.16	13.36	833.5
20	14/20	1.05	14.82	902.3
24	6/8	1.07	(8.89)*	1136.7
(5)**	(26/96)**	(2.89)**	(66.65)**	(647.8)**

*Single reading - not an average. Other readings lost due to instrumental failures.

**Comparative data with building poorly ventilated - Cf Table I

No physiological observations other than classification as dead or alive were made on the goats in these trials.

2. Trials in mine tunnels

a. Tunnel No. 1

The plan of Tunnel No. 1 is shown in Fig. 2. This tunnel was 173 feet in length, and ended in a pit or winze whose floor was approximately 18 feet below the level of the main tunnel. A side tunnel branched off the main tunnel at a distance of 128 feet from the main floor and sloped upward to a point approximately 12 feet above the floor of the main tunnel. The single main portal was the only source of ventilation of this tunnel. Pipe lines of 1/8" i.d. were run to points A, B, and C

TUNNEL NO. 1



Top View



Side View

SCALE IN FEET
0 10 20 30 40

FIGURE 2

in the tunnel (Fig. 2). A sheared goat was tethered at each of Positions A, B, C, and D. The flame thrower was fired into the mouth of the tunnel from a range of ten yards.

Flame was observed in the tunnel for approximately ten minutes, but little flame came out of the entrance. After ten minutes, however, unignited vapor was seen to come out along the bottom of the tunnel to the portal, where it ignited just outside the cave.

The tunnel was entered by observers wearing oxygen breathing apparatus sixteen minutes after firing. At this time, visibility was only 2 to 3 feet throughout the mine. Heat was noticeable only in the region where the actual combustion took place. Beyond the first turn of the tunnel the air was cool.

Table IV shows in relation to the number of minutes after firing the gas concentrations which were obtained through the pipe line system as well as by means of Bureau of Mines vacuum bottles, and also shows the condition of the goats. As indicated, observations were made to determine whether the flame safety lamp or carbide lamp placed at various positions within the tunnel before the fuel was fired were or were not still burning, and the density of the smoke was estimated.

Goat A was reached by the observers 19 minutes after the end of firing and was found dead but unburned. The carbide lamp was not burning, and a vacuum bottle sample was taken at a level of $2\frac{1}{2}$ feet above the floor. At 22 minutes, Goat B was found alive and unburned. The carbide lamp at Position B was not burning. At 23 minutes, Goat D was found dead, but, like the others, unburned. The carbide lamp was not burning. The goat at Position C in the bottom of the pit was reached 24 minutes after the end of firing and was dead. The flame safety lamp at Position C was not burning, and a vacuum bottle sample was taken at a level of $1\frac{1}{2}$ feet above the floor of the pit.

One hour after firing, the air of the cave within 10 feet from the portal was irritating to the eyes although visibility was markedly improved, and the flame safety lamp burned normally. A Hoolamite determination at this point indicated approximately 0.4% carbon monoxide.

Seventy-five minutes after the end of firing, the flame safety lamp would burn anywhere in the tunnel except when lowered into the pit at Position C. The animals were then removed from the tunnel by men wearing appropriate respiratory protection. The goat at Position B was dead at this time.

Blood was drawn from the left ventricle of each of the dead animals within 5 minutes after removal from the tunnel, for hemoglobin, hematocrit, oxyhemoglobin and carboxyhemoglobin determinations. The results of the analyses are shown in Table V.

TABLE IV

Tunnel No. 1

Time of day	Sample	Position A			Position B			Position C			Position D		
		%CO	%CO ₂	%HC	%CO	%CO ₂	%HC	%CO	%CO ₂	%HC	%CO	%CO ₂	%HC
1 st	end of car-	0.2	15.6	4.6	1.8	8.1	7.9	-	0.0	20.4	0.1	-	-
2 nd	125 - 142	0.6	4.2	11.2	0.2	13.4	4.8	-	0.0	18.8	1.4	-	-
5 th		0.8	7.2	9.0	0.6	10.0	7.2	-	0.2	16.3	3.0	-	-
10 th		0.4	12.8	4.6	0.6	12.4	5.4	-	0.6	13.0	5.2	-	-
15*		0.4	16.0	3.5	0.2	14.4	4.4	-	0.6	12.4	5.2	-	-
19**	-22 ft. ##	0.7	14.4	4.3	0.7								
20*	above floor of tunnel	Goat A - Dead, Car-											
22**	-5 ft. ##	0.0	16.9	2.7	0.2	16.1	3.4	-	0.4	8.9	4.9	-	-
23 - 13 ft.	above floor of tunnel	Goat B*** alive, uncon-			0.6	16.0	3.4	0.6					
24**	-22 ft. ##	conscious. Carbide lamp											
25	above floor of pit	not burning.											
74**		0.04	20.4	0.4	0.1								
76**		0.03	20.6	0.3	0.1								
79**	-42 ft. ##	Goat B - Dead											
below floor of tunnel													
132*		0.0	21.0	0.0	0.0	20.6	0.2	-	0.6	14.5	3.8	-	-

Goat D-Dead. Carbide lamp not burning.

Goat C-Dead. Flame safety lamp not burning.

0.7 12.8 5.2 0.7

0.6 14.3 4.1 0.6

0.6 14.5 3.8 -

Samples taken through pipe lines.

Samples taken with Bureau of Mines Vacuum Bottles. (Samples taken at level of tunnel end of sampling pipe unless otherwise noted.)

Goat placed on raise 13 ft. above floor of main tunnel - when observed after firing goat had struggled to a level 3 ft. above floor of main tunnel.

Visibility unlimited, smoke detectable.

Visibility less than 50 ft. with electric miners' lamp.

Visibility less than 3 ft. with electric miners' lamp.

TABLE V
Blood Studies on Goats in Tunnel No. 1

Position	Observation Time	Condition	Hemoglobin gm./100 ml.		Hematocrit		%Hb O ₂ Final	%Hb CO Final
			Init.	Final	Init.%	Final%		
A	19 min.	Dead	10.7	11.7	42	39	0.3	74
B	22)	Unconscious						
	76)	Dead	10.0	8.3	31	27	9.7	64
C	24	Dead	---	8.3	--	26	1.2	71
D	23	Dead	9.0	11.4	31	36	0.3	79

The ventilation in Tunnel No. 1 was entirely by convection, i. e., air entering the tunnel from the outside being warmer than the air in the tunnel, rose to the ceiling and traveled along the ceiling until it cooled. Air left the tunnel from the bottom. It is interesting that the gases of partial combustion were carried back throughout the 173 feet of the tunnel by this simple mechanism. It is obvious that the amount of carbon monoxide in the blood coupled with the existence of a deficiency in atmospheric oxygen would be sufficient to cause death. Heat was apparently not a significant lethal factor at the points where the goats were exposed.

b. Tunnel No. 2

Fig. 3 shows that this tunnel had a single portal leading to a main tunnel 548 feet in length. In addition, there was a curving side tunnel 217 feet in length, which branched off from the main tunnel 34 feet from the entrance. The long tunnel had several fissures of various sizes at different points throughout its length. These are indicated in Fig. 3 by small cross marks, either singly or connected with a solid line to indicate, in the latter instance, a fissure which transected the entire tunnel. The shorter tunnel was similar to Tunnel No. 1 in that there were no fissures or other openings except the portal where it joined the main tunnel.

Trial No. 1. As indicated in Table VI, goats were placed at Positions A, B, C, and D. Carbide lamps were placed at Positions C and D and a flame safety lamp at Position A. The flame thrower was fired into the main portal from a distance of 17 feet, using the same quantity of fuel as in Tunnel No. 1. During a 10 minute period after the end of firing, flame alternately flashed out of the opening of the mine, and then into the tunnel so deeply that no fire appeared at the entrance. Eleven minutes after firing, three men with oxygen masks and goggles entered the mine.

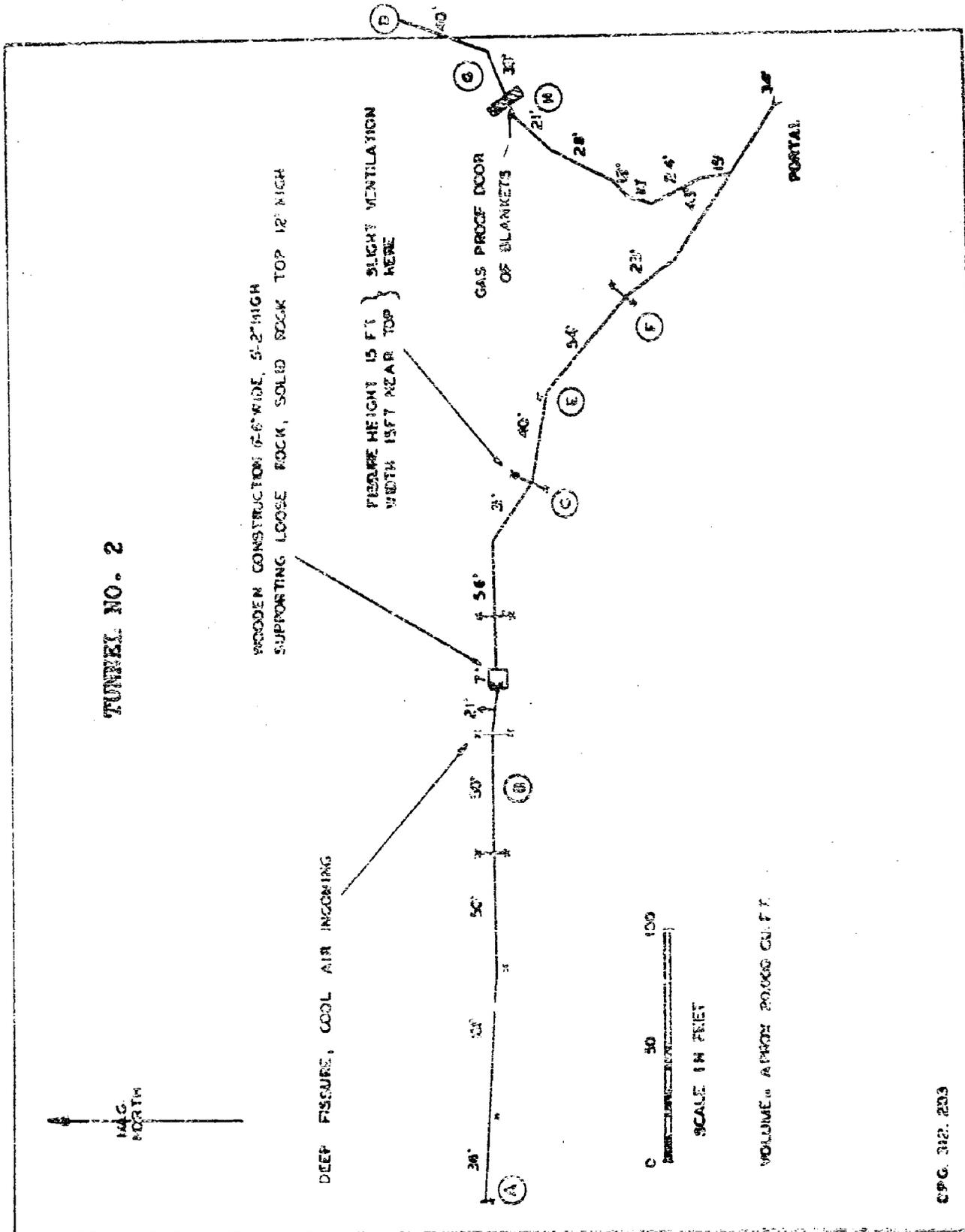


FIGURE 3

TABLE VI
Tunnel No. 2

Position	Time after end of firing - min.	Smoke	%CO	%O ₂	%CO ₂	%CH	Goat	Lamp
C	14	*	0.0	20.9	0.1	0.0	Alive	Carbide lamp burning
B	16	None	0.0	20.8	0.1	0.0	Alive	-
A	19	None	0.0	20.9	0.1	0.0	Alive	Flame safety lamp burning
D	20	**	1.3	14.9	3.3	0.4	Dead	Carbide lamp burning
D	64	*	0.1	19.6	0.9	0.1		
C	70	None	0.0	20.8	0.1	0.0	-	

* Visibility less than 50 ft. with electric miners' lamp.

** Visibility less than 3 ft. with electric miners' lamp.

At junction of tunnels 33 minutes after end of firing the Flame Safety Lamp would not ignite and carbon monoxide concentration was 0.1 by Hoolamite.

NOTE: All samples taken at level of the head of standing goat (approximately 2 ft. above floor level) by means of Bureau of Mines Vacuum Bottles.

Table VI shows the time after firing at which various positions were reached, the condition of the lamps, and the location and sampling time of the gas samples. As noted in Table III, the smoke was extremely dense throughout the length of the 217 foot side tunnel leading to Position D, whereas, in the main tunnel, the density of the smoke diminished after passing the first fissure, was only moderate at Position C and was undetectable at Positions A and B. The entire tunnel system was cool except for a section extending along the main tunnel for a distance of 50 feet from the portal.

Only the goat at Position D was found dead. This animal was found to have 0.8% oxyhemoglobin, and 69% carboxyhemoglobin in a post mortem blood sample. As in the trial in Tunnel No. 1, heat was apparently not a factor in the death of this goat. The goats at positions A, B, and C, were apparently unaffected in any way.

Trial No. 2. The results of the first trial suggested that the ventilation supplied by the various fissures in the main tunnel might be reducing the effectiveness of the flame attack. There was no trace of smoke on the walls or ceiling of the main tunnel by the time one reached the wooden construction half way in the 548 foot main tunnel, whereas, in the 200 foot side tunnel which had no fissures, smoke could be detected on the ceiling all the way to Position D.

It was decided, therefore, to pack all detectable fissures with wet straw and burlap. In addition, a standard army "gas-proof" door was constructed of scrap lumber and 2 G.I. blankets in the side tunnel at a point 148 feet from the main portal (Cf. Fig. 3). Goats were placed at Positions A, B, C, E, F, G, and H as indicated in Fig. 3. Carbide lamps were placed at 5 positions as indicated in Table VII, and a flame safety lamp was placed at Position G. The same quantity of fuel was fired as in Trial No. 1. Thirteen minutes after firing observers entered the tunnel wearing protective equipment. The condition of the goats, lamps, and smoke are indicated at each position in Table VII.

The gas analyses for samples taken at Positions B and C in Trial No. 2 indicate the greater effectiveness of this trial over Trial No. 1. (Compare Table VI and VII) so far as the main tunnel was concerned. The goat behind the "gas-proof" door at Position G was unharmed, the flame safety lamp was burning and there was no detectable smoke in the tunnel beyond the "gas-proof" door when this portion of the tunnel was first entered. Analysis of the blood of the goat at Position G showed 3.7% carboxyhemoglobin, but this was after the goat traversed the section of the tunnel between the "gas-proof" door and the main portal, so that much or all of this carbon monoxide could have been breathed during removal. The blood carboxyhemoglobin concentrations of the goats at Positions C, E, F, G, and H, are shown in Table VIII.

TABLE VIII

Blood Carbon Monoxide Concentration
In Goats - Trial No. 2

Goat	Condition	%Hb CO
C	Alive	5.7
E	Collapsed	27.2
F	Unconscious	25.4
G	Alive	3.7
H	Dead	86

The contrast between the condition of goats at G and H is striking testimony to the effectiveness of simple protective measures.

TABLE VII
Tunnel No. 2

Position	Time after end of firing - min.	Smoke	%CO	%O ₂	%CO ₂	%CH	Goat	Lamp
A	19	None	0.0	20.9	0.1	0.0	Alive	Burning
B	22	None	0.1	18.8	1.6	0.0	Alive	-
C	24	*	0.1	18.5	1.7	0.1	Alive	Not burning, reignited
E	26	*	0.3	16.4	3.0	0.2	Collapsed(2)	Not burning, reignited
F	27	*	0.4	15.5	3.8	0.3	Unconscious (2)	Not burning, reignited
H	31	**	0.5	15.4	3.6	0.4	Dead	Not burning, reignited
G(1)	32	None	0.1	20.2	0.5	0.0	Alive	Flame safety lamp burning

- (1) Behind "gas-proof" door.
(2) Recovered completely.

* Visibility less than 10 ft. with electric miners' lamp.
** Visibility less than 3 ft. with electric miners' lamp.

NOTE: All samples taken at level of the head of a standing goat (approximately 2 ft. above floor level) by means of Bureau of Mines Vacuum Bottles.

c. Tunnel No. 3

Tunnel No. 3 is shown in Fig. 4. It consisted of a main tunnel and several branching tunnels. Approximately 200 feet from the portal, a 20-foot tunnel branched off, terminating at Position C, at which point a vertical shaft rose 30 feet and connected with a horizontal tunnel 20 feet long, which terminated with a second vertical shaft extending 20 feet upward to the surface of the ground. Goats and lamps were placed at Positions A, B, C, D, and F as indicated in Table IX. The flame thrower was fired into the main portal from a distance of 15 feet, using the same quantity of fuel as in the other tunnels. Seventeen minutes after the end of firing, observers entered the tunnel wearing oxygen protective equipment. At this time, all goats were alive and all lamps were burning. There was a moderate amount of smoke from the portal to Position C, but none distal to the point where the side tunnel leading to Position C branched off.

TABLE IX

Tunnel No. 3

Position	Time after end of firing - min.	Smoke	%CO	%O ₂	%CO ₂	%CH	Goat	Lamp
F	26	*	0.4	17.8	1.9	0.4	Dead	Carbide lamp not burning
C	27	**	0.1	19.6	0.8	0.1	Alive	Carbide lamp not burning
A	28	None	0.0	19.9	0.3	0.0	Alive	Carbide lamp not burning
B	30	None	0.1	19.3	0.5	0.1	Alive	Carbide lamp burning
D	32	None	0.1	20.1	0.5	0.1	Alive	Carbide lamp not burning

* Visibility less than 10 ft. with electric miners' lamp.

** Visibility unlimited, smoke detectable.

NOTE: All samples taken at the level of the head of a standing goat (approximately 2 ft. above floor level) by means of Bureau of Mines Vacuum Bottles.

Thirty minutes after the end of the firing an equal quantity of fuel was again fired into the tunnel. Twenty-one minutes after the end of the second firing, observers again entered the tunnel wearing protective equipment. All goats were alive except the one at Position F, but all carbide lamps were extinguished except for the one at Position B. Table X shows the blood carboxyhemoglobin concentrations of the goats.

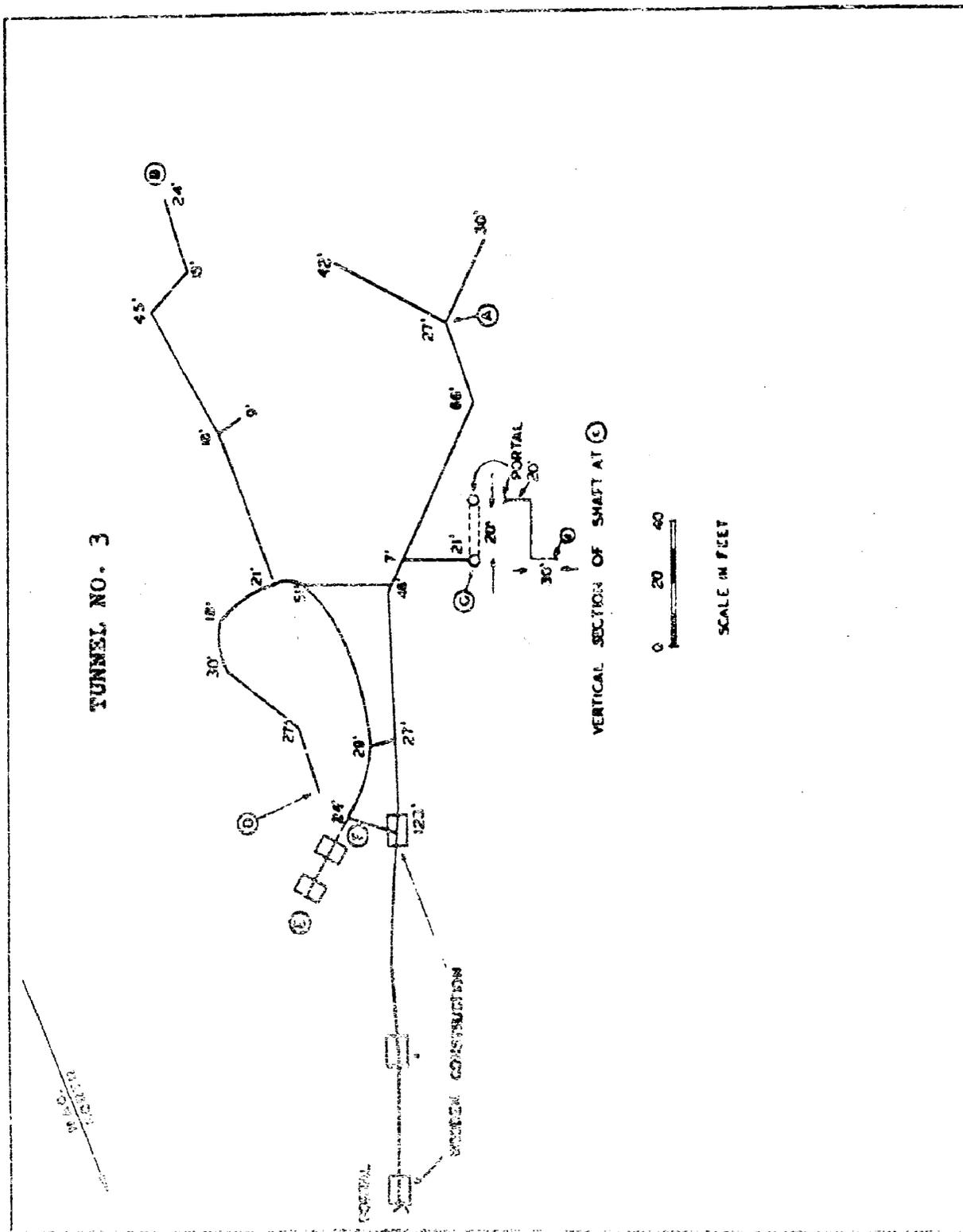


FIGURE 4

TABLE X
Blood Carbon Monoxide Concentration of
Goats - Tunnel No. 3

Goat	Condition	%Hb CO
A	Alive	0
B	Alive	5.7
C	Alive	16.9
D	Alive	-*
F	Dead	63.7

*Sample lost

III. DISCUSSION

The experiments described in this paper serve to show two important facts: First, that the gases of partial combustion can be very effective in producing a lethal atmosphere in poorly ventilated structures, even at a considerable distance from the actual conflagration; and, secondly, that a given quantity of burning fuel is more dangerous to life in a poorly ventilated structure than in a well ventilated one.

The trials conducted in the experimental building are of interest because heat was an important lethal factor both when the building was poorly ventilated, and when it was well ventilated. Nevertheless, the quantity of fuel required to produce a time x temperature product of approximately 600 degree minutes was 5 gallons under conditions of poor ventilation and 12 gallons under conditions of good ventilation. A dangerous depletion of atmospheric oxygen or dangerous build-up of carbon monoxide comparable to that produced by 5 gallons of fuel under conditions of poor ventilation could not be achieved under conditions of good ventilation, even with quantities of fuel up to 24 gallons.

If, therefore, one was exposed to a conflagration at relatively close quarters, as was the case with the goats in the experimental building, one would have to worry most about toxic gases under conditions of poor ventilation and about heat under conditions of good ventilation. For a given quantity of burning fuel, the chances of survival would be better under conditions of good ventilation.

The experiments carried out in the mine tunnels serve to illustrate how far the toxic gases of partial combustion can be carried by currents. In Tunnel No. 1, lethal conditions were produced at the extreme end of the tunnel, 173 feet from the portal and heat was obviously not a factor in any

The experiments in Tunnel No. 2 illustrate the effectiveness of vertical ventilation in acting as a barrier to the horizontal movement of the gases of combustion, and they also illustrate the relative ease of stopping the travel of these gases by providing a simple mechanical barrier such as the "gas-proof" door constructed of two G.I. blankets.

The third tunnel differed from the first two in having a vertical shaft to the outside at the end of a branch from the main tunnel. The experiments carried out in Tunnel No.3 show the effect of this vertical shaft in directing the path taken by the smoke (and toxic gases).

It is obvious from the experiments in these tunnels, that a relative remoteness from the point of an actual conflagration within an enclosed structure, and where no heat is experienced, cannot guarantee safety unless one is separated from the conflagration by sources of ventilation which serve to draw off the toxic gases, or by a mechanical barrier to the travel of these gases.

IV. BIBLIOGRAPHY

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RESTRICTED

THE TOXICOLOGY OF FIRE

PART V. An Evaluation of the Toxic
Factors in Conflagrations

RESTRICTED

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART V. An Evaluation of the Toxic Factors in Conflagrations

I. HISTORICAL AND THEORETICAL

It has been shown in previous parts of this paper that the mechanisms by which petroleum conflagrations produce casualties which terminate in rapid death are different, depending on the conditions of ventilation which attend the conflagration. Where ventilation is very good, as in conflagrations in the open, heat is the dominant lethal factor, and death is the result of hyperthermia. Where ventilation is poor, however, death is apparently the result of anoxia, attributable to respiration of an atmosphere containing carbon monoxide, carbon dioxide, smoke, unburned hydrocarbons, and deficient in oxygen. Heat may play some part in affecting the toxicity of the atmosphere, but under other conditions may contribute nothing to the toxicity of the gas mixture making up the ambient atmosphere.

A very important objective of these experiments was to provide information on the relative effectiveness of the different lethal factors concerned, in order to guide research on protective and therapeutic measures. It was relatively easy to say when heat alone was the dominant lethal factor, but when a toxic atmosphere was involved, it was not easy to say which constituent of the atmosphere was the dominant toxic factor. Some of the difficulties in evaluating the toxic factors have already been discussed.

It was decided, therefore, to investigate the problem by synthesis. The Clinical Research Section, Medical Division, Army Chemical Corps, was presented with the results of the experiments previously described in which death of goats was apparently due to carbon monoxide and anoxia, and was asked to expose goats to the known lethal factors singly, and then in various combinations, using intensities and duration of exposures comparable to those actually observed.

Since conflagrations of petroleum fuels under conditions of poor ventilation do not result in complete combustion of the fuels, unburned hydrocarbon vapors had to be considered as possible lethal agents. It had been noted by several investigators that serious cardiac arrhythmia and even death following ventricular fibrillation occurred sometimes during chloroform and cyclopropane anesthesia, particularly when surgery was begun during the earlier stages of anesthesia (4-8). Several investigators noted that the intravenous injection of epinephrine, as a substitute for the sympathetic stimulation of premature surgery, could produce similar disturbances in conjunction with the inhalation of certain volatile

hydrocarbons and halogenated hydrocarbons (9-11). It was thought, therefore, that the inhalation of hydrocarbon vapors during a petroleum conflagration, coupled with the intense emotional stimulation likely to occur during exposure to conflagrations, might well result in a situation analogous to premature surgery or epinephrine injection during inhalation of hydrocarbon anesthetic.

The Pharmacology Section, Medical Division, Army Chemical Corps, headed by Major A. Z. Gilman, was asked to investigate this point (12). Dogs were, therefore, exposed to the inhalation of hydrocarbon vapor in concentrations similar to those encountered in poorly ventilated conflagrations, and were injected with epinephrine in order to stimulate the sympathetic nervous system.

The experiments involved were as follows.

II. EXPERIMENTAL

A. "Synthetic" Experiments

Goats (or in a few experiments, pigs) were usually restrained in the supine position on an animal board. An inflatable cuff was placed around the chest or upper abdomen and connected to a tambour which recorded on a kymograph. One of the femoral arteries was cannulated under local anesthesia for the purpose of taking blood samples and recording blood pressure. Blood samples were collected in a heparinized syringe, sealed with mercury, and kept on ice until analyzed.

Oxygen, carbon monoxide, and carbon dioxide content, and oxygen capacity of the blood were measured on the Van Slyke-Neill apparatus. Hemoglobin was determined on each sample by a modification of the Evelyn-Malloy (1) cyanmethemoglobin method.

The gas mixtures were made up in two 100 liter gasometers, and the composition of the gas was checked with the Orsat apparatus. The gasometers were connected by 1 inch bore tubing to a snugly fitting head mask, and the animal inspired directly from the gasometers. Continuous readings were made of minute volume of respiration and respiratory rate.

No practical method was found for heating animals as rapidly as they were heated in experiments with the flame thrower. As a compromise the trunk of the animal was wrapped in two layers of heavy felt and turkish toweling, and was encircled with three coils of inductotherm cable. Heating was continued for approximately 20-40 minutes, until the rectal temperature measured 42.5°C. or higher, at which time gassing was started. Internal temperatures immediately after death, measured above the liver, were 1° - 2°C. higher than the rectal temperatures.

Most of the blood gas data were obtained on animals gassed until all respiratory or cardiac activity had ceased. Some animals were gassed for three minutes only on the assumption that this might represent a reasonable time period for exposure to the effects of a conflagration. Such

animals were allowed to breathe room air, without artificial respiration, following exposure.

The pig was used in a few experiments because of its previous use in the extensive heat studies of Moritz and co-workers.

1. Results - "Synthetic" experiments

a. Toxicity of low oxygen mixtures

Goats were subjected to an atmosphere of pure nitrogen or nitrogen containing up to 4.4% oxygen, since oxygen concentration found in previous experiments ranged from a minimum of zero to an "average" (Ct/t) of 4.4% over the exposure period. The results are summarized in Table I. The four goats which breathed 4.0 - 4.4% O₂ were still alive at the end of 10-15 minutes when the experiment was terminated for technical reasons, but the others breathed the respective gas mixtures until all respiratory and cardiac activity ceased.

During the first 20-30 seconds of gassing, the goats were usually quiet; violent convulsive movements then followed, and were accompanied by hyperpnea and apparent loss of consciousness. After 1-1½ minutes, respiratory volume diminished and convulsive movements decreased in frequency and magnitude. The depression of respiration was coincident with a cessation of the regular type of breathing followed by a period of apnea lasting several seconds, which in turn was followed by a series of gasps at increasing intervals until death occurred.

Changes in cardiac action paralleled closely the respiratory changes. After pronounced tachycardia in the second half minute, there was frequently a brief cardiac arrest at the time of apnea. Subsequently cardiac action was characterized by marked arrhythmia. The heart beat was generally audible at the chest wall for 6-8 minutes, and electrocardiographic evidence of activity was present for as long as 18-20 mins. In one instance, ventricular fibrillation resulting in sudden death occurred before respiration had ceased (onset at 2¼ minutes).

Arterial oxygen saturation fell precipitously to a level of approximately 13% at the end of 1 minute when the animal breathed 99.5 + % nitrogen, the level then decreased further to less than 10% saturation at time of death. With less oxygen deficient atmospheres the arterial saturation was maintained between 10% and 25% until respiration ceased when the saturation fell sharply below 10%.

b. The toxicity of carbon monoxide - low oxygen mixtures

It was considered that 2.7% carbon monoxide might represent a reasonable "average" concentration to which victims of a conflagration might be exposed. Five goats were, therefore, exposed to a mixture

TABLE I
The Effects of Low Oxygen Mixtures on Respiration and Arterial
Oxygen Saturation in Unanesthetized Goats

No. of Goats	% O ₂ in Mixture	Cessation Regular Respiration (Min.)		Last Gasp		Minute Volume (liters/min.)		Arterial Oxygen Saturation (%)				
		Min.	Max. Avor.	Min.	Max.	1 Min.	2 Min.	1 Min.	2 Min.			
3	0-0.4	0.83	2.0	1.42	2.67	5.66	3.62	8.2-31.0	1.0-17.3	9.1-21.6 Av. 16.3	7.2-16 Av. 10.4	-----
5	0.8-1.8	1.0	2.92	1.76	2.75	5.75	4.87	9.3-23.7	2.8-11	12-32 Av. 20.1	9-29.7 Av. 17.0	6.5-23.2 Av. 11.8
3	3	3.09	6.42	4.56	5.33	9.50	6.91	10.6-15.3 18.6	14.1- 18.6	24-26 Av. 25.2	16-23.8 Av. 19.4	10.9-23.5 Av. 15.3
9	4-4.4	2.0	13.6	-----	4.25	15.2	-----	3.7-14.1	2.5-19.9	-----	-----	-----

of 2.7% carbon monoxide in air. The results, expressed as averages for each series, are shown in Table II.

TABLE II
The Effect of Carbon Monoxide - Low Oxygen Mixtures

Gas Mixture	2.7% CO	2.7% CO	3% O ₂ *
	in air	in N ₂	in N ₂
Number of Animals	5	9	3
Respiration Ceased (min.)	3.03	1.9	4.56
Last Gasp (min.)	7.24	4.8	6.91
Arterial Oxygen Saturation (%)			
During Gassing			
0.5 min.	--	54	
1.0 "	48.5	30	25.2
3.0 "	13.8	10	--
5.0 "	7.6	6	15.3
Arterial CO Saturation (%)			
During Gassing			
1.0 min.	44.2	48	--
3.0 "	74.2	82	--
5.0 "	88.2	90	--

*Data of Table I.

The combined effects of carbon monoxide and low oxygen were studied by exposing 9 goats to 2.7% CO and 3.0% O₂ in nitrogen. The results, expressed as averages, are also shown in Table II. The effects of exposing 3 goats to 3.0% O₂ in nitrogen are shown in the last column of Table II for the purposes of comparison.

It can be seen that 2.7% CO in air is slightly more lethal than 3.0% O₂ in nitrogen in that respiration ceased and the last gasp occurred slightly earlier with exposure to the carbon monoxide. Arterial oxygen saturation was lower after 5 minutes gassing with 2.7% CO (7.6%) than after 5 minutes gassing with 3.0% O₂ (15.3%).

The combination of 2.7% carbon monoxide with 3.0% oxygen in nitrogen proved to be more lethal than either carbon monoxide alone or low oxygen alone, as might be predicted from theoretical considerations. It can be seen from Table II that respiration ceased at an average time of 1.9 mins. and the last gasp occurred at an average of 4.8 mins. when the 2.7% carbon monoxide with 3% oxygen in nitrogen was used. Similarly, arterial oxygen

saturation reached a lower level (6%) and arterial carbon monoxide saturation reached a higher level (90%) after 5 minutes gassing with the mixture, than with either the low oxygen or the carbon monoxide component.

c. The effect of carbon dioxide

Carbon dioxide levels did not exceed 10% in the conflagrations studied, even under conditions of relatively poor ventilation, and since there is ample evidence that this level is not lethal in itself no exposures were made to carbon dioxide in air alone. It was recognized, however, that the stimulating effect of 5 - 10% carbon dioxide on the respiration and the rate of gaseous exchange in the lung, and the effect of carbon dioxide on the dissociation of hemoglobin from oxygen and carbon monoxide, might have some effect on the toxicity of gas mixtures containing carbon monoxide and deficient in oxygen.

Five goats were exposed to an atmosphere containing 2.7% carbon monoxide, 3.0% oxygen, and 7.0% carbon dioxide. The results, expressed as averages, are shown in Table III.

TABLE III

The Effect of Carbon Dioxide on the Toxicity of High Carbon Monoxide - Low Oxygen Mixtures

Gas Mixture	2.7% CO 3.0% O ₂ 7.0% CO ₂ in N ₂
Number of Animals	5
Respiration Ceased (min.)	1.25
Last Gasp (min.)	4.1
Arterial Oxygen Saturation	
During Gassing 1.0 min.	20
3.0 "	7
5.0 "	5
Arterial CO Saturation	
During Gassing 1.0 min.	72
3.0 "	89
5.0 "	91

It can be seen from the data of Table II and Table III, that 7.0% carbon dioxide had a slight and probably insignificant effect on the time of death, final arterial oxygen saturation, or final arterial carbon monoxide saturation, of goats gassed with 2.7% CO and 3.0% O₂ in N₂. Loss of oxygen from the blood and increase in carboxyhemoglobin were accelerated, but at the end of 5 mins. the concentrations reached were essentially the same as in the absence of carbon dioxide.

d. The effect of artificial fever on the toxicity

Artificial fever was induced in 21 goats, 4 of which died within 10-20 mins. from fever alone, and apparently because of respiratory failure with precipitous fall in blood oxygenation before circulatory failure occurred.

The goats which survived induction of artificial fever had rectal temperatures at the end of heating which ranged between 42.6 and 43.6°C. It was noted that there was some decrease in arterial oxygen saturation after induction of fever, the initial average O₂ saturation being 91.4% and the average after induction of fever 86.2%. While the reasons for the decrease in oxygenation during induction of artificial fever were not investigated the decrease in affinity between hemoglobin and oxygen at elevated temperatures may be a factor (2).

e. The effect of artificial fever on the toxicity of lethal gas mixtures

Heated goats were gassed with the mixtures previously described, and the results expressed as averages for each series are shown in Table IV.

TABLE IV

Effect of Heating on Goats Exposed to Lethal Gas Mixtures

Gas Mixture	4.2% O ₂ in N ₂	2.7% CO in air	2.7% CO 3.3% O ₂ in N ₂	2.7% CO 3.0% O ₂ 7.0% CO ₂ in N ₂
Number of Animals	4	3	8	2
Respiration Ceased (min.)	2.1	2.5	1.25	1.08
Last Gasp (min.)	3.7	4.4	2.85	2.78
Arterial O ₂ Saturation				
Minutes of Gassing				
0.5	29.5	67.1	24	24
1.0	14.4	41.2	12.2	15
3.0	5.4	14.6	2.9	2.2
5.0		6.6		
Arterial CO Saturation				
Minutes of Gassing				
0.5		21.4	13.7	27
1.0		44.6	29.3	48
3.0		71.8	37.8	49
5.0		79.2		

A comparison of these data with those of Tables I, II, and III shows that heat had a pronounced effect in increasing the susceptibility of goats to the lethal gas mixtures used. Time of death was reduced to less than 3

minutes in animals breathing 2.7% CO and 3% O₂ in N₂, with or without 7.0% CO₂; 4.2% O₂ killed in 3.7 minutes, compared with an average survival time exceeding 10 minutes in unheated animals; and 2.7% CO in air killed in 4.4 minutes as compared with 7.24 minutes in goats not subjected to heat.

Arterial oxygen saturation fell much more rapidly in the heated animals with all gas mixtures. Carboxyhemoglobin, on the other hand, increased more slowly than in unheated animals, except for the group getting 2.7% CO in air. At death, the blood of heated animals exposed to 2.7% CO and 3.0% O₂ contained only 37.8% carboxyhemoglobin and that of animals exposed to 2.7% CO, 3.0% O₂, and 7.0% CO₂ contained 49% carboxyhemoglobin as compared with saturations of about 90% in unheated animals (Tables II and III).

f. The comparative effects of low oxygen and high carbon monoxide on goats and pigs

In order to get data on another species than the goat, and because of the extensive heat studies of Moritz and co-workers on pigs, it was decided to gas pigs with 2.7% CO and 3.0% O₂ in N₂ and with 2.7% CO, 3.0% O₂, and 7.0% CO₂ in N₂. The results, expressed as averages, are shown in Table V.

TABLE V

Effects of Low Oxygen and High Carbon Monoxide on Pigs

Gas Mixture	2.7% CO 3.0% O ₂ 7.0% CO ₂ in N ₂		2.7% CO 3.0% O ₂ in N ₂	
	Number of Animals	5		3
Respiration Ceased (min.)	1.17		2.0	
Last Gasp (min.)	4.3		4.2	
Arterial O ₂ Saturation				
Minutes of Gassing	1.0	15	36	
	2.0	8	9	
	5.0	6	4	
Arterial CO Saturation				
Minutes of Gassing	1.0	76	49	
	2.0	84	78	
	5.0	88	92	

Comparison of the data of Table V with those of Tables II and III shows that the response of pigs to these gas mixtures was not appreciably different from that of goats.

g. Survival rates after 3 minutes exposure to gas mixtures

A series of animals were gassed for 3 minutes, and then were allowed to breathe room air, since this gave, in most cases, Ct products as great as or greater than those observed in field experiments (Part III of this paper). A large majority revived spontaneously as shown in Table VI. Most survivors were on their feet and appeared normal at the end of 15 minutes after gassing. Three of the 5 deaths occurred during gassing.

B. Experiments with gasoline vapor and epinephrine

The experiments with gasoline vapor and epinephrine have been described by Chenoweth (15) elsewhere, and will merely be reviewed briefly here.

Healthy male dogs weighing 5.6 to 20.4 kg. were anesthetized with 35 mg./kg. of sodium pentobarbital given by intraperitoneal injection. Gasoline vapor in air was delivered to the dogs through a tracheal cannula, and the concentration of combustible gases in the mixture was recorded continuously by drawing a sample of the inspired air through a Cities Service M. H. Heat Prover. Blood pressure changes were followed by means of a cannula inserted in the femoral artery and attached to a Huertle manometer, and electrocardiograms were taken on a Sanborn "Cardiette".

The procedure used was to take tracings of the electrocardiograms and blood pressure before administration of the gasoline vapor was begun. Gasoline vapor was then administered in such manner as to duplicate as closely as possible the concentration-time relationships found in experiments with flame thrower attack of a poorly-ventilated bunker. A second electrocardiogram was taken during administration of the gasoline vapor, and then, after an interval of 60-120 seconds, 0.01 mg. epinephrine was injected intravenously.

Ventricular fibrillation occurred in 12 of 36 trials, involving 21 dogs, in which the gasoline vapor concentration reached peaks between 4 and 8 volumes per cent. Ventricular fibrillation could be induced in the remaining dogs either by increasing the challenging dose of epinephrine, or by increasing the concentration of gasoline vapor, or both.

The mechanism of production of ventricular fibrillation under these circumstances has been discussed elsewhere (15,16). While no firm conclusions were reached, it appeared that ventricular fibrillation usually results when an ectopic complex occurs in the period of late systole or early diastole.

TABLE VI
Survival Rates of Animals Exposed for 5 Minutes to Lethal
Mixtures of Carbon Monoxide and Low Oxygen

No. Animals	Gas Mixture	Regular Respiration Ceased (over)	Collapse	Arterial Oxygen Saturation at 5 min.	Carbon Monoxide Saturation at 5 min.	Animals No. Recovered	% Recovered
5	Goat 100% N ₂	1 min. 19 sec.	-----	10.0	-----	5	33
5	Goat 2.7% CO 3.0% O ₂	1 min. 54 sec.	58 sec.	10.3	81.6	7	77
9	Goat 2.7% CO 3.0% O ₂ 7 % CO ₂	1 min. 25 sec.	49 sec.	10.2	89.7	7	77
4	Pig 2.7% CO 3.0% O ₂ 7 % CO ₂	1 min. 27 sec.	-----	7.0	86.0	4	100

III. DISCUSSION

The ultimate aim of research on the causes of rapid death in petroleum conflagrations resulting from flame thrower attack was to provide information on the relative effectiveness of the different lethal factors concerned. Except for flame attack in the open or under conditions of abundant ventilation, where heat proved to be the determining factor, it was not possible to decide from the data of field trials the relative importance of the different lethal factors. The reasons for the difficulty will be described in more detail below, but the "synthetic" approach of exposing animals to the lethal factors singly and in various combinations as described in this paper was adopted in order to clarify the situation.

A. "Synthetic" experiments

1. Low oxygen mixtures

Although, as shown in Table I, breathing low oxygen mixtures caused rapid and profound changes in respiration and blood oxygen, even total oxygen lack was generally insufficient to kill in a period of 3 mins. ($\Delta O_2 Ct = 60$). This is shown by the observation that the last gasp occurred, on the average, 217 seconds after the onset of gassing with cylinder nitrogen, and that 5 of 6 goats exposed to cylinder nitrogen for 3 mins. recovered. Animals breathing less anoxic mixtures might die fortuitously as by ventricular fibrillation.

The effects of oxygen - deficient mixtures on minute volume of respiration were extremely variable. Many factors undoubtedly played a part in respiratory changes and differences in minute volume appeared to have no significant effect on survival or blood carbon monoxide or oxygen content at the oxygen and carbon monoxide concentrations employed. The effect of anoxia on the goat should parallel that of anoxia on man, since Barcroft and associates have reported that the oxygen dissociation curves of goat and human hemoglobin are very similar(14).

2. Carbon monoxide - low oxygen mixtures

Since the concentrations of carboxyhemoglobin and oxyhemoglobin, when hemoglobin is exposed to atmospheres containing carbon monoxide and oxygen are proportional to the relative partial pressures of the gases, it can be predicted that low oxygen in the inhaled mixture would favor the formation of carboxyhemoglobin. The additive effect of low oxygen and carbon monoxide would be more apparent, however, if the concentrations used were less drastic in effect than those reported here. In other words, lethal concentrations of reduced hemoglobin and carboxyhemoglobin were reached within very few minutes when low oxygen mixtures or 2.7% carbon monoxide in air were employed separately. However, blood gas concentration changes were slightly more rapid when the combination was used.

3. Effect of carbon dioxide

The action of carbon dioxide in stimulating respiratory volume and circulatory rate is likewise overshadowed by the high concentration of carbon monoxide and low concentration of oxygen used; in addition, there was insufficient time in most experiments for carbon dioxide to exert its stimulating action.

4. Effect of artificial fever

Numerous factors undoubtedly contribute to the increased susceptibility of heated animals to toxic gas mixtures. The fact that carboxyhemoglobin concentrations at time of death in heated animals was much lower than in unheated animals probably indicates that the animals died of the combination of other factors before carboxyhemoglobin concentration had time to reach maximal levels.

B. Effect of gasoline vapors and epinephrine

The inhalation of 4-8 volumes per cent of gasoline vapors followed by the intravenous injection of 0.01 mg. epinephrine in dogs anesthetized with sodium pentobarbital resulted in fatal ventricular fibrillation in a certain proportion of the dogs. This concentration of gasoline vapors could well be encountered during petroleum conflagrations, and the emotional disturbance attending exposure to such conflagrations might well result in the liberation of sufficient epinephrine to sensitize the myocardium to ventricular fibrillation. While this mechanism of death must be considered as possible in petroleum conflagrations, the evidence of the field trials previously reported, and the synthetic experiments reported here would suggest death usually occurs as the result of anoxia due to oxygen deficiency, and the presence of carbon monoxide in the atmosphere, and heat. The hydrocarbon-epinephrine mechanism, however, is of considerable importance in other applications and has been discussed in detail by Chenoweth (15) and by Garb and Chenoweth (16).

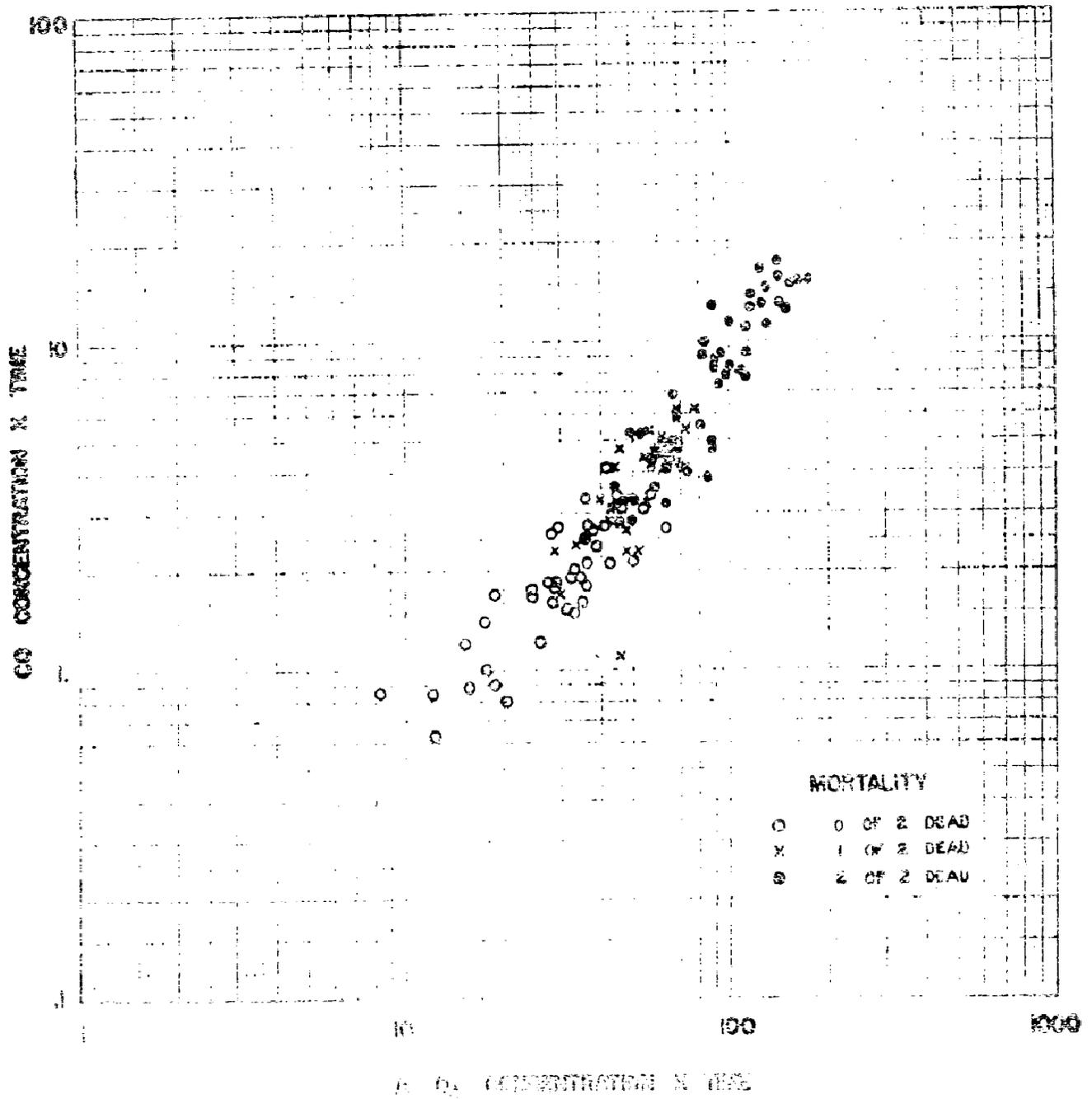
C. General

It appears from the experiments here reported, that low oxygen, high carbon monoxide and heat are the chief lethal factors responsible for death in exposure to conflagrations under conditions of poor ventilation. Carbon dioxide, while potentially capable of increasing the toxicity of these lethal factors, apparently contributed little to their toxicity under the conditions of these experiments. Another possible lethal mechanism, that of hydrocarbon vapors plus the release of epinephrine, leading to ventricular fibrillation, was apparently overshadowed by anoxia in actual conflagrations.

In this connection, it is interesting to consider certain difficulties inherent in the interpretation of the experimental data derived from field trials in a poorly-ventilated fortification. Figs. 1 to 6 show the various correlations between $\log \Delta O_2 Ct$, $\log CO Ct$, $\log CO_2 Ct$, and $\log Ft$, taken two at a time. The numerical correlations are shown in Table VII.

GOAT MORTALITY

LOG CO C1 VS LOG Δ O₂ C1 IN BUNKER



REQUIRE 1

GOAT MORTALITY

LOG Δ O₂ GA VS LOG TEMPERATURE TIME PRODUCT IN BUNKER

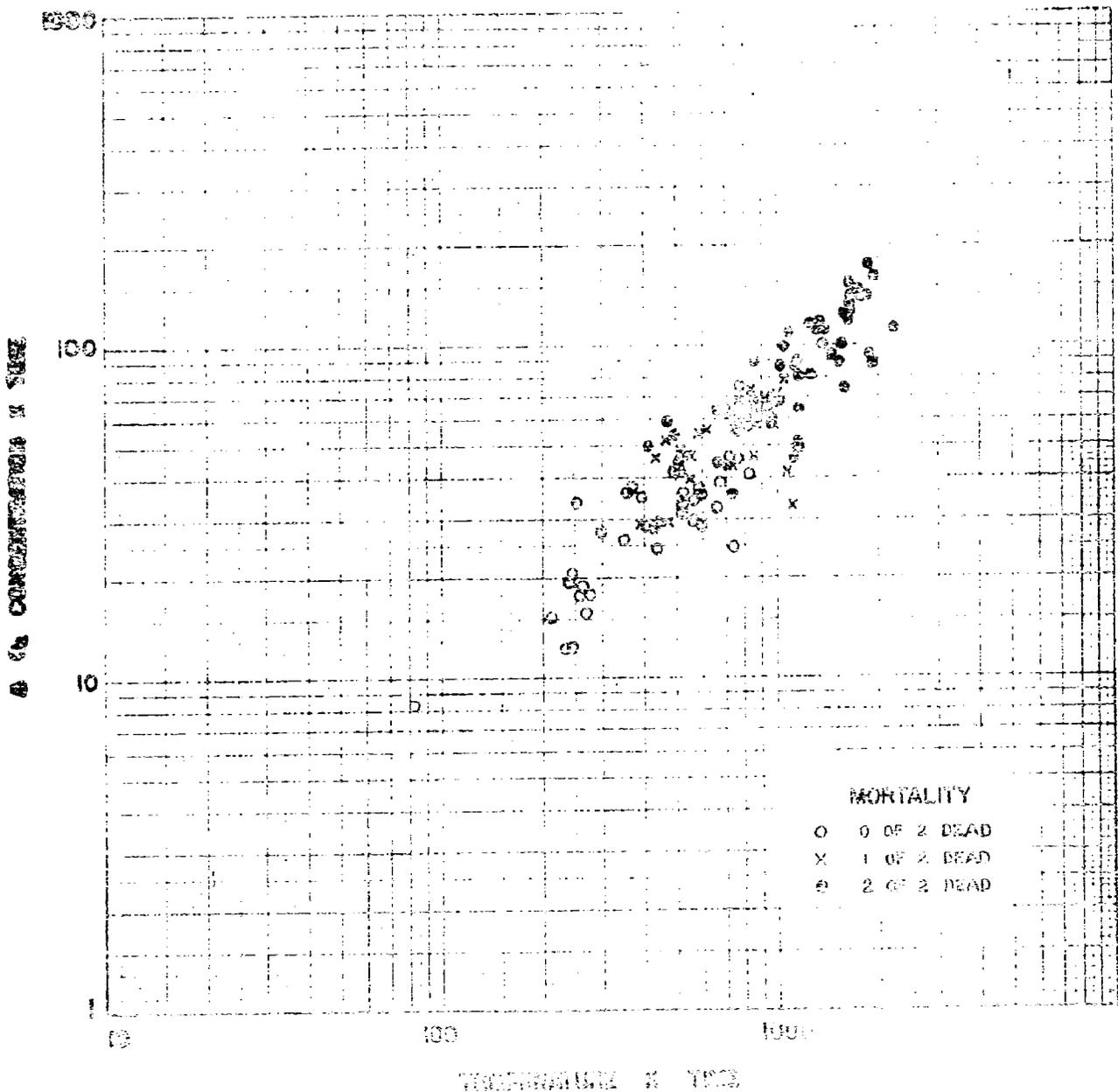


EXHIBIT B

GOAT MORTALITY

LOG ΔO_2 Ct VS LOG CO_2 Ct IN BUNKER

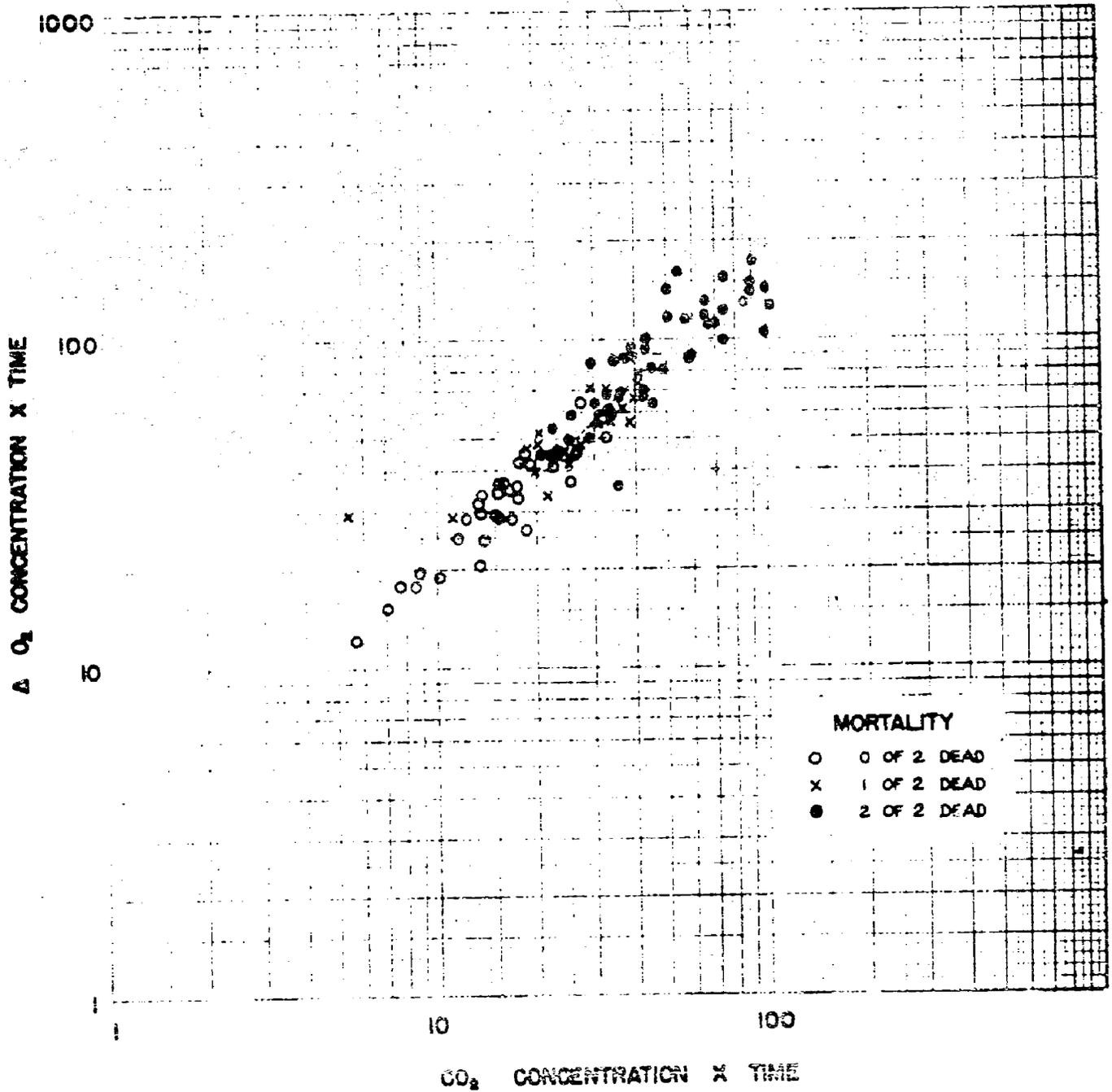


FIGURE 3

GOAT MORTALITY

LOG CO CL VS LOG TEMPERATURE TIME PRODUCT IN BUNKER

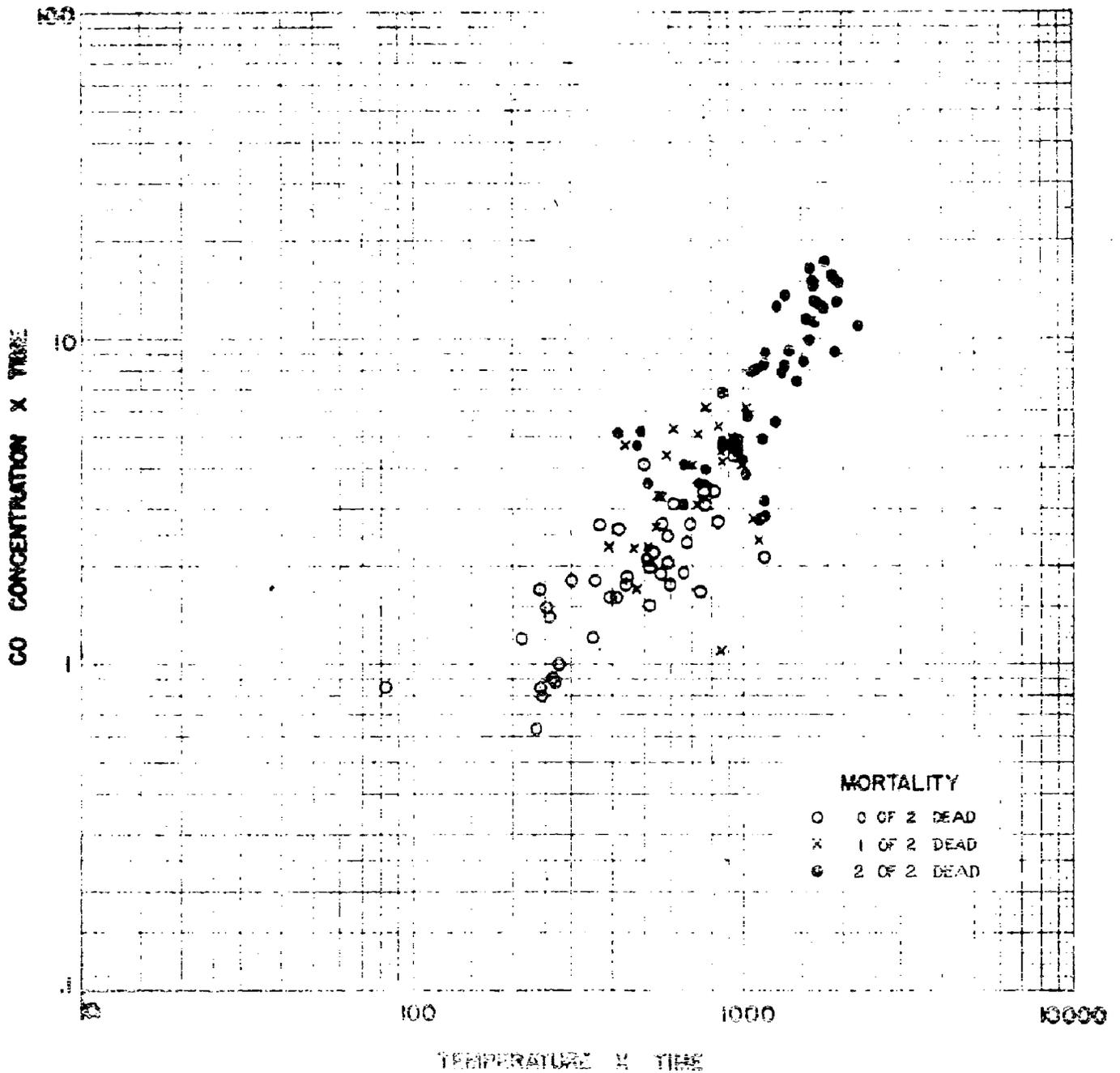


FIGURE 4

GOAT MORTALITY

LOG CO CT VS LOG CO₂ CT IN SLURRY

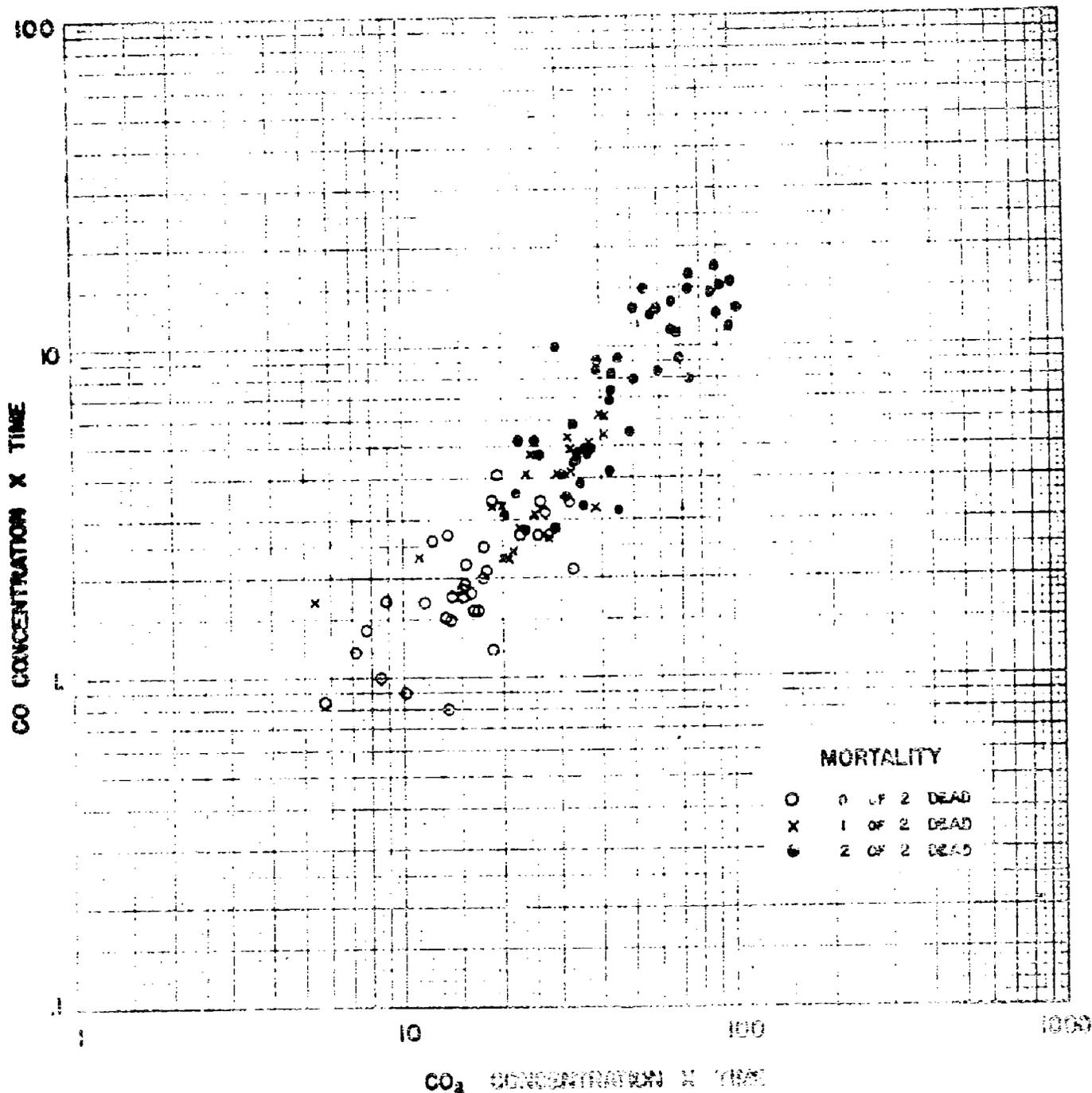


FIGURE 5.

GOAT MORTALITY

LOG CO₂ Ct VS LOG TEMPERATURE TIME PRODUCT IN BUNKER

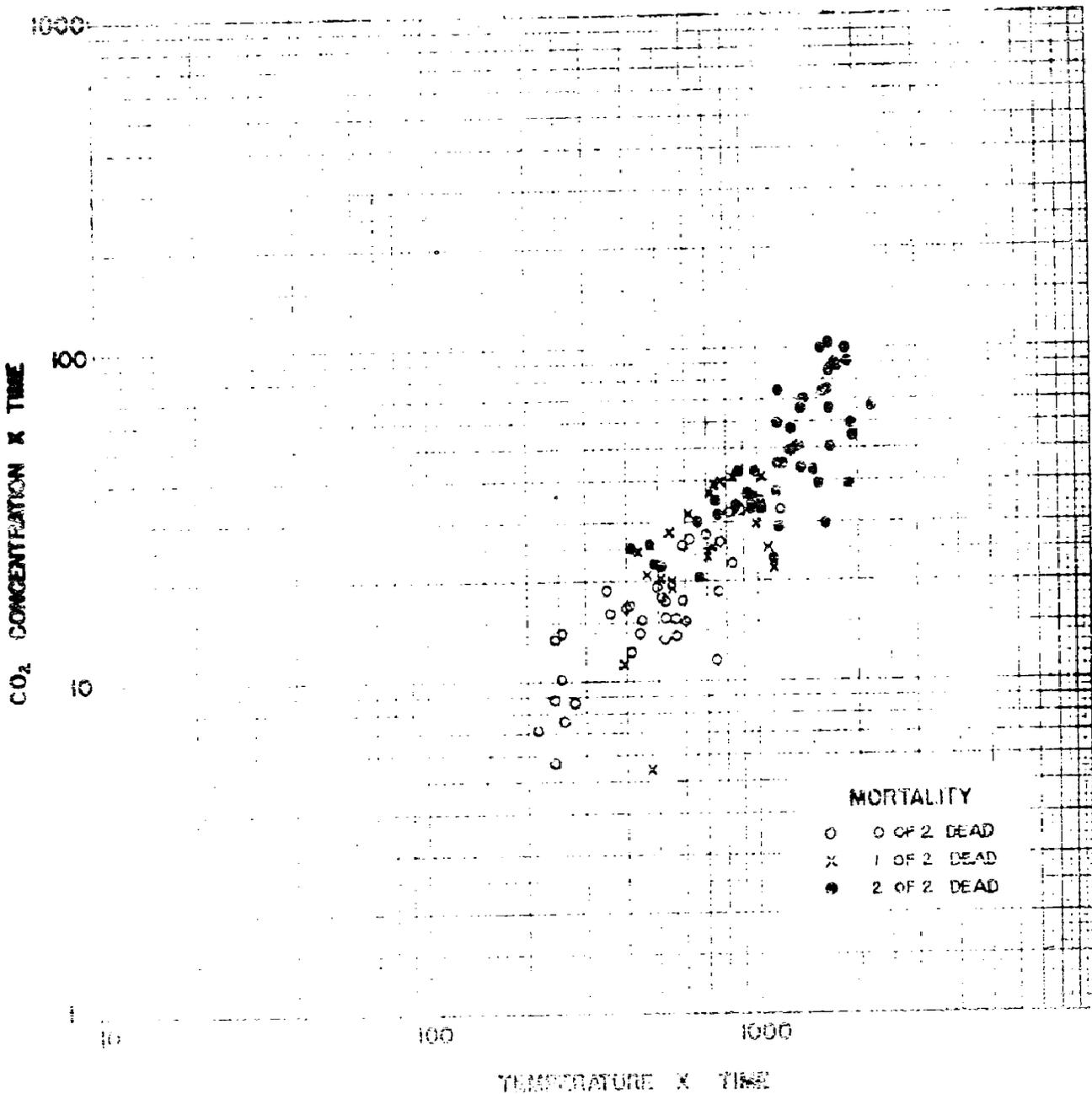


FIGURE 6

TABLE VII

Correlations Between Different Lethal Factors
Produced by Flame Attack in a Bunker

Factors	Correlation Coefficient (r)
Log Δ O ₂ Ct:log CO Ct	0.94
Log Δ O ₂ Ct:log Tt	0.91
Log Δ O ₂ Ct:log CO ₂ Ct	0.91
Log CO Ct:log Tt	0.86
Log CO Ct:log CO ₂ Ct	0.88
Log Tt :log CO ₂ Ct	0.69

It can be seen that strong positive correlations exist between each pair of factors, and by reference to Figs. 1-6, that each factor singly could be positively correlated with animal mortality. If only one of these lethal factors, e.g., Tt, had been measured in these experiments and a correlation determined between log Tt and goat mortality ($r=0.86$), it might have been tempting to speculate that heat was the obvious lethal factor. Similarly, if only CO₂ had been measured a correlation would have shown equally good agreement between log CO₂ Ct and goat mortality. Thus, these experiments illustrate the danger of drawing inferences as to causation from correlations where the variables correlated are not independent of other variables which may be concerned with causation. It would be legitimate, of course, to say that log CO₂ Ct might be as useful for predicting goat mortality in these experiments as log Tt, or log Δ O₂ Ct, or log CO Ct, but that is as far as one could go.

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RESTRICTED

THE TOXICOLOGY OF FIRE

PART VI. Protective Measures Against Injury by Fire

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Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART VI. Protective Measures Against Injury by Fire

SUMMARY

Protection against fire involves bodily protection against heat and respiratory protection against oxygen deficiency and the inhalation of toxic gases.

Protection against very high temperatures for very short periods (less than 1 second) can be achieved by very simple means such as a layer of heat protective ointment or thin cloth. As the duration of exposure increases, the requirements for a protective body covering become more rigid, the best devised during the war protecting against flame temperatures for more than one minute. Moist coverings become very dangerous if they are heated sufficiently to generate steam and if the steam can reach the skin.

Respiratory protection against oxygen deficiency and the inhalation of carbon monoxide appears to be of paramount importance in any poorly ventilated conflagration. The protective measures involved should also be effective for protection against any incidental toxic gases that might occur (such as nitrous oxides, phosgene or sulfur dioxide) from the thermal decomposition of certain chemicals.

Ventilation is a good protective measure for personnel in the immediate vicinity of a conflagration in that a plentiful supply of fresh air prevents the development of an oxygen deficiency and the formation of carbon monoxide as well as dilutes any incidental toxic gases that might be present.

A second measure of protection consists in setting up a barrier between exposed personnel and the conflagration. Any barrier that excludes smoke is likely to also exclude other toxic gases.

The amount of protection afforded by various types of respirators or gas masks is also discussed. Canister masks or any other devices which depend on an adsorbent for the removal of toxic gases from the atmosphere cannot be used safely in an atmosphere deficient in oxygen. The air-line or self-contained oxygen masks, on the other hand, when functioning properly are safe even in oxygen-deficient atmospheres or atmospheres containing dangerously toxic gases.

Medical Division Special Report No. 4

THE TOXICOLOGY OF FIRE

PART VI. Protective Measures Against Injury by Fire.

I. HISTORICAL AND THEORETICAL

Previous Parts of this paper have shown that the rapid death which can occur as a result of exposure to petroleum conflagrations may be due to heat, or to the inhalation of a toxic atmosphere, or to a combination of these. Ventilation determines which of these factors predominates. If ventilation is good, heat is the dominant lethal factor and rapid death results from a general or local elevation of the body temperature. The delayed death which usually occurs as the aftermath of extensive surface burns of the body was not a subject of study in these investigations. If ventilation is poor, inhalation of a toxic atmosphere is the dominant lethal factor, and death is the result of anoxia which stems from a primary oxygen deficiency aggravated by the presence of carbon monoxide and to a certain extent, carbon dioxide. Heat may be present as well as a toxic atmosphere and contributes to the anoxia.

Protection against injury as a result of exposure to conflagrations involves, therefore, protection of the body against heat, or protection of the respiratory tract against anoxia and carbon monoxide or both.

II. PROTECTION AGAINST HEAT

Heat reaches the body primarily by convection (ambient heat) and by radiation (radiant heat). Both types of heat are present during conflagrations, radiation occurring either from the flame itself or from objects heated by the flame, while ambient heat comes from physical contact with the heated atmosphere. Protection of the body against total heat, therefore, can be achieved by insulation with a protective layer which does not readily conduct ambient heat to the skin, and which reflects radiant heat or absorbs it in its external layers.

The duration of exposure to heat as well as the intensity of exposure determines, to a great extent, the nature of the protection required. Very brief exposures (less than 1 second) to very high temperatures such as occur in explosions result in flash burns. The temperatures in the exploding gases of ordinary munitions may reach 3000°C. and most of the heat energy is in the form of radiation. Flash burns may be extensive and fatal depending upon how much of the body is exposed, but death is seldom or ever immediate. Longer, though brief, exposures to high temperatures may result in rapid hyperthermic death. Moritz and co-workers (1) showed that 30-second exposures to air temperatures of the order of 500°C. produced a somewhat different type of fatal damage to pigs than longer (10 minute) and equally fatal exposures to temperatures of the order of 200°C. In some experiments with petroleum conflagrations,

5 second exposures to temperatures of the order of 1000°C. were fatal to goats, but the mechanism of death differed slightly from that described by Moritz for pigs. In these instances the cause of death was systemic or local hyperthermia although the exact mechanism by which death was accomplished differed from one type of exposure to another.

It is probably a fair generalization to say that the shorter the duration of exposure to heat, the simpler the protection required. Experience at Pearl Harbor showed that flash burns were prevented by ordinary cotton T shirts. The bulk of the heat produced in a flash combustion is radiant (infrared) and the thin T shirts were able to provide enough reflection and absorption of heat to prevent burns to the underlying skin.

Protective creams and ointments also have a limited value in protecting against heat - particularly radiant heat. Ointments which contain metallic flakes, for example, reflect both infrared and visible radiation well if the wave length of the radiation is small compared to the diameter of the metallic flakes. Metal bearing ointments were not suitable for military use because of their high reflection of visible light, which was incompatible with camouflage requirements. Nevertheless, both the Army and Navy produced anti-flash ointments which were capable of reflecting or absorbing a high proportion of incident radiation, both infrared and ultraviolet.

Radiant heat reflected away from the body has no physiological effects, but absorbed heat raises the temperature of the ointment, and since this is in intimate contact with the skin, the heat absorbed in the ointment can be readily transferred to the skin unless: (a) It is dissipated in an endothermic reaction, or in vaporizing an ointment constituent; (b) or the ointment is a poor heat conductor; or (c) the ointment has a high specific heat so that a relatively large quantity of heat is required to raise the temperature.

It appeared for a time that substances which would effervesce by volatilizing water of crystallization on heating, such as borax, would be advantageous ingredients of heat protective ointments, since the effervescent action is both endothermic and generates a layer of gas which is a poor conductor of heat. It was found, however, that such ointments tended to produce steam burns of the underlying skin which were more severe than would have been the case if the skin were dry but otherwise unprotected. While ointments and thin cloth are able to protect against very high temperatures of very short duration, they cannot afford much protection against longer exposures, even at lower temperatures. For this type of exposure, a thicker layer of insulation is required.

So far as protective clothing alone is concerned, it is possible to devise types which can withstand flame temperatures for short periods of time. The familiar asbestos suits of oil well fire fighters are a typical example. These are not combustible and conduct heat poorly, but in as short a time as 10 seconds of exposure to flame they may become thoroughly heated throughout their thickness, and, of course, burn the underlying skin. Flame protective suits, like firebrick, do not burn but can become very hot. It was not uncommon during the war to find that cotton clothing worn under the fire fighters' suits was charred even though the outer protective suit showed no signs of heat damage. In one extreme experiment conducted by the Medical Division, a department store mannikin was dressed in a protective suit of the best obtain-

able design and was exposed to the direct blast of a flame thrower. The suit was unharmed but the dummy itself was badly charred. This is not intended to minimize the value of protective clothing. With standard available clothing it is possible to sustain exposure to flame for a matter of seconds without injury. With the best protective clothing developed during the war, the period of exposure could be extended to more than a minute. This time, while short, may be all that is needed to escape from danger or perform some important act of defense. Since special fire protective clothing is not readily available, however, it was of interest to study the degree of protection provided by ordinary woollen or heavy cotton cloth.

During the experiments on the effect of exposure of goats to fire bombs dropped from an airplane, the amount of protection afforded by an ordinary woollen G.I. blanket or a shelter half was studied. Goats were prepared and exposed as in the experiments previously described, but were covered with a blanket or shelter half. It was found that dry covers tended to ignite and, if they did, offered less than no protection since severe burns resulted from contact with the burning cover. Wet covers did not ignite and gave considerably more protection than no cover at all. The best protection, however, was given when the protective covers, wet or dry, were removed from the animals by remote control immediately after the fireball resulting from detonation of the bomb had passed. The results of this protection can be seen in the summary of Table I, in which goats were classified in one category as either dead or prostrate or in another category as injured or not injured. Thus, while the protection afforded by a woollen blanket or a shelter half during an exposure of several seconds to flame itself was not absolute, it was significant and life-saving in many cases.

TABLE I

Summary of Effects of Fire Bombs on Protected and Unprotected Goats on the Surface of the Ground and in Slit Trenches

Animals	Dead or Prostrate	Injured or Uninjured
Unprotected on Surface	80%	20%
Protected on Surface	31%	69%
Unprotected in Trench	35%	65%
Protected in Trench	9%	91%

It must be realized that protective coverings applied to the body are valuable for only a limited period of time, the limitation being the time necessary for thermal equilibrium to occur between the outer to the inner surface of the covering. Wet coverings offer better protection against heat than dry coverings, because they will not ignite so long as they are wet and because some of the absorbed heat is used up in the endothermic process of vaporizing the water, but if heated to the point where steam is

produced, they may cause very severe burns to the underlying skin. Dry woolen or cotton covers ignite fairly readily, and if ignited can cause very severe burns. The best practical use of protective coverings involves removal as soon as possible after exposure to severe heat is over.

The importance of protection against heat varies with the degree of ventilation attending the conflagration. Where the ventilation is good, as in conflagrations in the open or in well ventilated buildings, protection against heat alone is required since toxic gases are either not produced or do not accumulate. Under conditions of poorer ventilation protection against heat as well as against a toxic atmosphere may be necessary.

III. RESPIRATORY PROTECTION

In discussing respiratory protection in conflagrations, a distinction must be made between the toxic atmosphere produced by the ordinary products of partial combustion - i.e., carbon monoxide, carbon dioxide and a deficiency of oxygen - and those produced by the thermal decomposition of certain organic chemicals. Examples of the latter would be nitrous oxides from the decomposition of nitrocellulose, or phosgene from the decomposition of carbon tetrachloride, or sulfur dioxide from the combustion of sulfur compounds. Gases of this type may or may not be present in any given conflagration depending on what is burning or is subjected to high temperatures. They were not present in the petroleum conflagrations described in the present series of papers. Nevertheless, the inhalation of a toxic atmosphere was a very important lethal factor for goats exposed to conflagrations under conditions of poor ventilation. Death, under these circumstances, was usually the result of anoxia, arising from primary oxygen deficiency and from the inhalation of carbon monoxide, and aggravated by heat. And regardless of whether special toxic gases like nitrous oxides or phosgene might be present in a given conflagration, these ordinary lethal factors leading to anoxia are still present and must be reckoned with. Fortunately, protection against special toxic gases as well as the ordinary toxic atmospheres of conflagrations requires essentially the same measures.

The first measure of respiratory protection to be considered is ventilation. If air can be admitted freely to a fire, partial combustion leading to oxygen deficiency and the production of carbon monoxide cannot occur, and any special toxic gases that might be produced can be held to low concentrations by the process of dilution. The free flow of fresh air into a building incidentally reduces the time \times temperature product (Tt) produced by the combustion of a unit amount of fuel within the building, thus minimizing another lethal factor of exposure to conflagrations. Ventilation is a good protective device so far as personnel in the immediate vicinity of the fire are concerned, but ventilation in another sense of the word may be a source of danger.

Gases produced in the vicinity of a fire may be conducted away from the fire by ventilation processes, either natural or forced, and there is danger, therefore, that they may be conducted to places that might be relatively remote from the fire itself and there produce casualties among exposed persons.

Even in a mine tunnel, gases travel by convection currents far from their original source. Air conditioning or forced ventilation systems could spread fire gases for a considerable distance. Incidents have been known to occur in fires in mines or on ships in which fire gases have been carried through forced ventilation systems to remote locations and have resulted in fatalities. Similar instances are a relatively common occurrence in homes, where gases from a defective coal furnace may permeate the house, injuring or killing some of the occupants. In summary, good ventilation in the vicinity of a fire prevents the occurrence of toxic atmospheres, but in the presence of an insufficient supply of air at the site of combustion, toxic gases may be transferred by whatever ventilation processes exist to places distant from the fire itself.

A second method of protection against toxic fire gases consists of setting up a barrier between the fire and the persons exposed. Experiments have been described in which the familiar "gas-proof door" of civilian defense days, consisting of a pair of blankets hung across a mine tunnel completely protected a goat on the far side of the barrier while another goat on the near side died. Wooden or metal doors would make an even better barrier than blankets.

Fortunately, smoke, which is relatively harmless in itself, accompanies the other fire gases. Smoke is visible and has a characteristic odor whereas carbon monoxide and lack of oxygen give no early sensory warning of their presence. It would appear sound to say that if one can keep out of smoke or can exclude smoke from one's vicinity by any barrier of any material, any toxic fire gases that might be present would also be excluded. Too often too little attention is paid to exposure to smoke, forgetting that dangerous atmospheres may accompany the smoke.

Sometimes a barrier to the passage of fire gases and smoke may be intangible. It will be recalled that in a previous paper it was shown (Part IV) that crevices extending upward in a mine tunnel to the outside air caused the smoke and fire gases to sweep upward and prevented their further travel into the mine tunnel.

General respiratory protection by ventilation or barriers is not always possible, so that situations may arise where personnel must enter an atmosphere known to be at least potentially dangerous. Such personnel must rely on gas masks or respirators.

There are two basic types of respirators. These are: (a) Respirators which depend on some adsorbent such as active carbon in a canister to remove toxic gases from the atmosphere; examples are the military gas mask or the MSA "All-Service" mask. (b) respirators which supply an atmosphere independent of the immediate environment of the person wearing the mask; examples are the "air-line" mask in which fresh air is pumped through a hose into the facepiece of the mask, or the self-contained oxygen mask which pure oxygen is delivered into the facepiece from a small cylinder of oxygen or from a chemical oxygen generator. The first type of mask is obviously of little use where there is a deficiency of oxygen in the

atmosphere. While wearing it may delay death by removing carbon monoxide or other toxic gases, it cannot supply oxygen which is necessary for life. An even more severe limitation exists on the use of a wet towel or handkerchief. It cannot supply oxygen or remove carbon monoxide. At best it might filter the inspired air and remove some smoke. Canister masks should only be worn where the presence of sufficient oxygen can be demonstrated. Specifically they should not be worn in any atmosphere which will not support the flame of a candle or mine safety lamp, or carbide lamp. If such flames burn steadily it may be presumed that at least 12% oxygen is present and this is sufficient to at least support life. It should be pointed out that in certain locations where there is little natural ventilation such as mine tunnels, local pockets of air may exist which are toxic and deficient in oxygen. Persons dependent on canister masks must, therefore, proceed under such circumstances with the greatest of caution and only when accompanied by a mine safety lamp or other reliable indicating device which will indicate a dangerous oxygen deficiency.

The air-line or self-contained oxygen masks, on the other hand, supply their own atmosphere to the lungs of the wearer, and thus are safe even in oxygen deficient atmospheres or atmospheres containing dangerously toxic gases.

For the sake of completeness, a few words should be said about the inhalation of heated air. Moritz, Henriques, and McLean (2) have shown that the inhalation of very hot dry air produces severe burns of the upper respiratory tract and laryngeal edema because all of the available heat is lost in the upper part of the respiratory tract. The inhalation of steam, on the other hand, produces thermal injury to the entire respiratory tract including the lungs, because steam has more heat to give up to its surroundings than does dry air. Hence some heat still remains to be delivered when the inspired air reaches the lungs.

It was our impression, however, from the present series of experiments on petroleum conflagrations that heat damage to the respiratory tract was of minor importance except in animals in the immediate vicinity of the flame, and even these apparently died of a systemic hyperthermia rather than from laryngeal edema or pulmonary injury.

IV. BIBLIOGRAPHY

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ARMY CHEMICAL CENTER, CHEMICAL CORPS., MEDICAL DIV., MD.
(MEDICAL DIVISION SPECIAL REPORT NO. 4)

THE TOXICOLOGY OF FIRE

JOHN A. ZAPP, JR. APRIL '51 109PP. TABLES, DIAGRS, GRAPHS, DRWGS

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