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# RESEARCH MEMORANDUM

REVIEW OF THE TOXICOLOGICAL PROPERTIES OF PENTABORANE,  
DIBORANE, DECABORANE, AND BORIC ACID

By Joseph M. Lamberti ✓

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Cleveland, Ohio

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## NATIONAL ADVISORY COMMITTEE FOR AERONAUTICS

WASHINGTON

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RESEARCH MEMORANDUMREVIEW OF THE TOXICOLOGICAL PROPERTIES OF PENTABORANE,  
DIBORANE, DECABORANE, AND BORIC ACID

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## SUMMARY

A review is presented on the toxicity of several boron hydrides and boric acid from available data in chemical and medical literature as well as from classified information of various military agencies.

The boron hydrides are extremely toxic as compared with other chemicals, and their modes of action within the human body are unknown. Primary effects of exposure include disturbance of the central nervous system for pentaborane and decaborane and pulmonary edema and hemorrhage for diborane. Prolonged exposures to these three compounds may cause damage to the kidneys and liver.

Tentative maximum allowable concentration for continuous exposure is 0.5 and 1.0 parts per million for pentaborane and diborane, respectively. The exposure concentration of decaborane lies midway between that of pentaborane and diborane. In a comparison, pentaborane is considered more toxic than hydrogen cyanide, while the toxicity of diborane may be compared with that of phosgene.

Although boric acid has long been used as a mild antiseptic, recent literature indicates poisoning of the human body by boric acid and boric oxide. The reported lethal dose (ingestion) for adults is 15 to 20 grams and for children, 5 to 6 grams. Many of the fatal cases reported in the literature involve application of boric acid over large areas of the denuded skin. Indications are that the boric acid is probably not absorbed through the intact skin but rapidly through the abraded skin.

Data are also presented comparing the toxicity of these compounds with other common laboratory chemicals. Information concerning protection for personnel engaged in working directly with these compounds is briefly discussed.

  
INTRODUCTION

A review of the available literature on the toxicity of the boron hydrides as well as boric oxide was necessary for research programs involving high-energy fuels at the NACA Lewis laboratory. A brief examination of the literature (ref. 1) showed that the boron hydrides were toxic. There was a need, therefore, to compile a survey in terms understandable both to the chemist and the engineer working with these hazardous materials.

This report discusses the relative toxicities of the boron hydrides and the specific toxicological effects of pentaborane, diborane, and decaborane, and reviews various phases of protection for personnel working directly with these compounds. Data are also presented on the toxicity of boric acid and boric oxide.

When using the information described in this report, the following points should be observed:

- (1) The report is intended primarily for the chemist and the engineer working directly with the boron hydrides. Consequently, detailed medical data, description, and terminology are avoided. A limited glossary of general medical terms has been included in appendix A for convenience.
- (2) The present report is not intended to represent a complete, comprehensive treatise on the toxicity of the boron hydrides. The data were collected from pertinent reports in available chemical and medical literature. The original reference should always be consulted for any detailed, specific information concerning any item discussed herein.
- (3) Isolated cases of boron hydride exposures are not reported as such, except in one or two cases. The generalizations made whenever possible may not be strictly accurate since some are based on relatively few cases.
- (4) Discussion of first-aid procedures or personnel protection is limited to those procedures followed at the Lewis laboratory. They are not to be construed as "official", but rather as "common sense" procedures which have been found satisfactory at this laboratory.

## FACTORS INFLUENCING INTERPRETATION OF TOXICITY DATA

There are many factors influencing toxicity that must be taken into consideration and some of the more important factors are discussed in the following paragraphs.



### Mode of Action and Effect of Exposure

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It is necessary to distinguish between mode of action and effect of exposure before a proper evaluation of the toxicity of a chemical is made. Mode of action is the mechanism by which a normal physiological function is disturbed by the toxic agent. Effect of exposure is the pathology which may result from this disturbance. For example, these two factors may be illustrated with a case of carbon monoxide poisoning (ref. 2). In normal respiration, oxygen combines with the hemoglobin of the blood to form oxyhemoglobin. This compound dissociates in the body into reduced hemoglobin and oxygen which is available for the tissues. In the mode of action of carbon monoxide poisoning, carbon monoxide combines with the hemoglobin at a rate 210 times faster than that of oxygen to form carboxyhemoglobin (the combination of carbon monoxide and hemoglobin), a more stable compound than oxyhemoglobin. Consequently, the more carboxyhemoglobin that is present in the blood stream, the less oxyhemoglobin would be formed for respiratory purposes.

The effect of exposure to carbon monoxide poisoning is asphyxiation. The length and severity of exposure will determine the degree of damage to the nervous system and circulation.

### Method of Exposure

A poisonous material may enter a human body intravenously, subcutaneously, by ingestion, inhalation, or skin absorption. When a comparison between two chemicals is made, the same method of exposure should be used in both cases. Also, the method of exposure will, in most cases, determine whether the action of the toxic agent will be of a local or systemic nature.

### Acute and Chronic Effects

An important question that cannot be easily answered is whether an exposure to a toxic agent in relatively large concentrations for short periods of time will produce the same physiological and pathological effect as exposures of smaller concentrations for longer periods of time. There are many factors which influence the final result, such as, concentration of the toxic agent, method of exposure, rate of distribution of the toxic agent within the body, chemical reactivity of the toxic agent within the blood stream, and rate of elimination of the toxic agent (ref. 2). The following examples will illustrate these points.

The halogenated hydrocarbons are extensively used as industrial solvents and refrigerants. Acute effects of exposure generally involve

irritating action on the respiratory tract and, if severe, on the central nervous system (ref. 2). However, the chronic results of exposure may be liver and kidney damage (ref. 2).

Carbon tetrachloride has many uses as a solvent and cleaner. Poisoning by this chemical may not only cause pulmonary and cardiac disturbance but also injury to the kidney and liver (refs. 2 and 3). The toxicological effects of this compound are probably not only due to the carbon tetrachloride molecule itself but also to its degradation products which form within the body, namely, hydrochloric acid and phosgene (ref. 3).

Benzene is an important compound used throughout the chemical industry. Its toxicity is dependent on two important facts, its insolubility in body fluids, and its excellent fat solvent properties. Since it is insoluble in the body fluids, equilibrium between the amount in the blood stream and the vapors in the air is rapidly attained; therefore, elimination is fairly rapid following a brief exposure. However, elimination is much slower after a long exposure because benzene is entrapped in the fatty tissues of the body (ref. 3).

#### Personal Factors

Age. - As a general rule, infants and children are more susceptible to the effects of chemical poisoning than adults. For example, younger people are more apt to get boric acid poisoning than older people (ref. 4).

Immunity. - A person may build up an immunity to certain toxic materials with repeated exposure to them. This tolerance depends on the chemical and particular physiological make-up of the individual. For example, it is believed that an immunity to chlorine can form by repeated exposures to low concentrations of it (ref.-2).

Health. - Persons who are ill or have recuperated from a recent illness are more susceptible to chemical poisoning than persons enjoying good health. Carbon tetrachloride poisoning, for example, is more severe in people with a history of kidney or heart disease (ref. 3).

#### Animal Experiments

Certain phases of the current medical research programs involve laboratory animals. Care must be taken in the extrapolation of the results of these experiments to include humans. In addition to the preceding factors which influence the toxicity of any given compound, the species of animals used and sometimes the different strains of the same species affect the results of a toxicity study (ref. 5).

Detailed principles of general industrial hygiene and toxicology as well as specific toxicities for individual chemicals are adequately described in the excellent texts and handbooks listed in references 2, 3, and 5 to 8.

#### TOXICOLOGY OF BORON HYDRIDES AND BORIC ACID

Important physical properties of pentaborane, diborane, and decaborane are listed in table I; a review of their toxicological effects is as follows:

##### Pentaborane

Toxicity. - The mode of action of pentaborane within the human body is unknown (refs. 9 and 10). However, primary effects of exposure involve the central nervous system (refs. 9 to 11); prolonged exposure may also cause damage to both the liver and the kidneys (ref. 10). Skin contact with this compound causes a severe chemical burn; the effects of ingestion are unknown. The most frequent cause of dangerous exposure to this material is inhalation (ref. 9).

The results of animal experimentation (ref. 9), obtained from inhalation tests, show that pentaborane is more toxic than diborane and hydrogen cyanide. Ingestion tests (refs. 9, 10, 12, and 13) with laboratory animals show convulsions, coma, and finally death in a relatively short time. Further animal tests (ref. 13) show that the lethal dose (LD<sub>50</sub>) for a 2-hour exposure with pentaborane was 14.1 parts per million for male mice and 19.5 parts per million for male rats. (The LD<sub>50</sub> concentration is that concentration in which 50 percent of the test animals died.) These same (ref. 13) studies indicate that there may be an accumulative toxic effect build up in the rats with repeated subacute exposures to this compound. Also, detoxification did not seem to occur within the animal body when exposed to low concentrations. There is no information available regarding the accumulative toxic or detoxification effects of pentaborane on humans.

Symptomatology. - The first general symptoms shown are typical in many chemical exposure poisonings. These may include dizziness, blurred vision, nausea, fatigue, and nervousness (refs. 9 to 11). However, the more characteristic symptoms of a severe pentaborane exposure are the abnormal muscular contractions or twitching of any part of the body followed by convulsion and then coma (refs. 9 to 11). After the convulsion-coma state, difficult breathing, blue coloration of the skin, poor muscular coordination, and imperfect articulation of speech may be observed (ref. 11).

Blood chemistry. - In a study (unpublished data compiled by Callery Chemical Company) based on 14 cases of pentaborane exposure, the following facts about blood chemistry were generally observed:

- (1) An elevation of nonprotein nitrogen and blood urea nitrogen (indication of kidney disorder)
- (2) A positive cephalin-cholesterol test (indication of liver dysfunction)
- (3) A positive thymol turbidity test (indication of liver dysfunction also)
- (4) A rise of creatinine level (indication of kidney disorder)
- (5) A high leucocyte count (white blood cells)

Urinalysis. - Based on the same 14 cases mentioned previously, the following urinalysis observations were noted:

- (1) Albumin strongly positive (indication of kidney disorder)
- (2) Low percentage of excretion of phenolsulfonphthalein dye (another indication of poor kidney function)
- (3) Sugar in relatively large amounts

The significance of these medical terms and tests are explained in appendix A of this report which is followed by a discussion (appendix B) of a severe case of pentaborane poisoning.

#### Diborane

Toxicity. - Within the human body the mode of action of diborane is unknown (refs. 9 and 10), but the primary effect of exposure is pulmonary irritation (ref. 9). Prolonged exposure to the material may also cause damage both to the kidney and liver (refs. 10 and 14). Ingestion and inhalation effects on humans are not definitely known; however, animal experimentation reveals that diborane compared with phosgene ranks below pentaborane and decaborane in its toxicological effects (refs. 9 and 10).

Additional animal studies revealed pulmonary edema (presence of serous fluid in lung tissues) and hemorrhage, tracheal and lung congestion, and liver and kidney damage (refs. 9, 10, 14, and 15). The LD<sub>50</sub> value for the rat is 50 parts per million for an exposure time of 4 hours (ref. 14).

Symptomatology. - The symptoms of a diborane poisoning generally include respiratory disturbances and those of the familiar "metal fume fever" syndrome, which consists of shivering or shaking of the voluntary muscles followed by sweat and pallor of the skin (refs. 9, 10, and 11).

#### Decaborane

Available data show no cases of human exposure to decaborane alone (ref. 10). Most of the information that is available and discussed in the following paragraphs is based largely on animal experimentation (refs. 9, 10, and 12 to 17).

Toxicity. - In humans the mode of action of decaborane within the body is not understood (refs. 9 and 10). Available data (ref. 15) indicate that it behaves somewhat like pentaborane, but to a lesser degree, in its toxicological effects. In general, toxicity studies show that recovery from pentaborane exposure is more rapid than from decaborane (ref. 12). Primary effects of exposure are disturbances of the central nervous system; prolonged exposure may cause liver and kidney damage (refs. 16 and 17). These same tests revealed corneal damage, hemorrhagic adrenal glands, and cardiovascular disturbances. A cumulative toxic effect may be exhibited by animals with repeated small exposures to decaborane. From the results of a study (ref. 13) laboratory tests generally indicated a cumulative toxic effect of decaborane. Also, detoxification did not occur within the animal body. There is no quantitative information on the cumulative or detoxifying action of decaborane in humans.

The LD<sub>50</sub> concentration for mice was 36.5 parts per million for an exposure time of 4 hours. Death of the animals occurred within 24 hours after the exposure (ref. 12).

Symptomatology. - Symptoms exhibited by animals exposed to decaborane may be compared with those exhibited by animals exposed to pentaborane (muscular incoordination, convulsions, and coma) (refs. 12, 13, 16, and 17). Additional clinical observations (ref. 17) include increased red-blood-cell count and hematocrit (measure of the relative volume of the plasma to the volume of the red blood cells), and increased number and percentage of the polymorphonuclear white blood cells.

#### Boric Acid

Toxic effect on humans. - Boric acid has been used extensively in the past as a mild antiseptic; it has never been considered as an industrial poison. However, the literature within recent years has reported several deaths from this compound not only by ingestion but also by application over large areas of the denuded skin (refs. 4 and 18 to 24).

In 86 recognized clinical cases of boron poisoning (including several methods of exposure and various age levels of patients) reported by reference 4, the mortality rate was about 50 percent. Infants seemed to be more susceptible to boron poisoning than adults.

The most characteristic symptoms of boric acid poisoning are gastrointestinal disorders and the erythematous (redness of skin) rash. In severe cases disorders of the central nervous system are noticed also (ref. 9). Symptoms of poisoning may not occur for several days or weeks after intoxication (refs. 4 and 19). Also, these same investigators report that boron may accumulate in the kidneys, liver, and brain. The reported lethal dose (ingestion) for adults is 15 to 20 grams and 5 to 6 grams for children (ref. 8).

Toxic effect on plants. - Boron is necessary for the normal growth of many of the common plants (refs. 25 to 28). However, little is known about its role in plant metabolism. Experiments show that amounts of boron beyond the actual need of the specific plant are definitely toxic. The toxicity varies with each individual plant. For instance, corn is injured by 0.1 parts per million, while cotton can tolerate up to 10 parts per million of boron (refs. 4 and 27). Irrigation waters which contain 0.5 to 2.5 parts per million are toxic to citrus trees (ref. 27).

Toxic effect on animals. - In the animals studied (ref. 4), boron was not essential to any function of mammalian tissues. The acute toxicity varied from 0.8 to 2.0 grams per kilogram of animal body weight. Boric acid was probably not absorbed through the intact skin, but rapidly through the abraded skin. These same tests also showed that boron accumulated in the brain, liver, and fatty tissues of the animal body after prolonged small doses or acute overdoses of boric acid. Repeated injections of boric acid did not affect the blood-forming system of these animals.

Toxic effect on fish. - The literature is rather scarce concerning the toxicity of boron in fish. The results of one study (ref. 28) showed that the rainbow trout is definitely harmed by concentrations of 80,000 parts per million of boric acid; whereas, concentrations of 6250 parts per million were fatal for the rudd within 18 hours.

Safe concentrations in water supplies. - For domestic water supplies, boron limits of 20 to 30 parts per million (ref. 28) have been recommended. For irrigation waters, a boron limit of 0.4 to 0.5 parts per million (ref. 28) was reported, since above this concentration some plants may be injured.

## COMPARISON OF TOXICITY OF BORON HYDRIDES WITH OTHER CHEMICALS

In order to show some relation between the toxicity of the boron hydrides and other chemicals, figure 1 presents these chemicals in their order of toxicity. For convenience, a logarithmic scale is used. This comparison is based on the allowable working concentration in parts per million. The calculation of parts per million is explained in appendix A. It is assumed that these values are based on an 8-hour working day. The values shown in figure 1 were taken from volume II of reference 2. It should be stressed that these numerical values are not absolute. They are average values for the "average" person. Figure 1 shows how extremely toxic pentaborane, diborane, and decaborane are when compared with other chemicals.

## PERSONNEL PROTECTION

## Allowable Concentrations

The median-detectable concentration (ref. 29) and the maximum-allowable concentration (ref. 30) for pentaborane, diborane, and decaborane are listed as follows:

Compound	Median-detectable concentration		Maximum-allowable concentration, ppm
	mg/cu m	ppm	
Pentaborane	2.5	0.97	0.5
Diborane	3.7	3.27	1.0
Decaborane	.35	.07	---

## Monitoring Devices

Since maximum-allowable concentrations of pentaborane, diborane, and decaborane are extremely low, it is essential to have some sort of monitoring device for personnel protection. Monitoring devices and their feasibility are discussed in references 30 to 36.

At the Lewis laboratory, the amperometric borane detector (ref. 31) and the hand-operated pentaborane detector (ref. 32) are used. The amperometric borane detector built at Lewis according to the specifications in reference 31 can detect 1.5 to 2.0 parts per million of diborane. The hand-operated pentaborane detector can detect 1 to 2 parts per million.

Since these monitoring devices detect concentrations above the permissible working levels, Lewis personnel, even if they are wearing gas masks, are instructed to leave work areas as soon as these detectors give an indication of vapors in the atmosphere.

#### Gas Masks

Evaluation of gas-mask absorbents for pentaborane and diborane is given in reference 37. In removing a 0.5-percent pentaborane concentration from the air, Whetlerite and activated carbon proved satisfactory; activated alumina and zinc-chloride-impregnated alumina were poor. For diborane, the Mine Safety Appliance "All Service" or Model S canister was satisfactory in removing concentrations of diborane ranging from 0.5 to 3 percent. However, no canister was satisfactory for diborane concentrations above the flammable limits.

Gas-mask protection against decaborane is discussed in reference 17. In removing less than 0.01-percent decaborane concentration from the air, silica gel was the best absorbent; activated carbon was also a good absorbent.

Air-supplied masks with full-face shields are always used at the Lewis laboratory by personnel entering work areas that may have high concentrations of these compounds.

#### Wearing Apparel

At the Lewis laboratory, personnel working directly with the boron hydrides wear the following apparel:

- (1) Chemical safety goggles and full face shields
- (2) Fireproof or leather jackets and trousers
- (3) Asbestos and/or rubber gloves
- (4) Masks as required

#### TREATMENT FOR EXPOSURE TO BORON HYDRIDES

##### Minor Exposure

Workers who accidentally spill small amounts of the boron hydrides on their skin or into their eyes should thoroughly flush the affected parts of the body with water and promptly leave the contaminated area. The contaminated clothing should be removed and destroyed.

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### Major Exposure

Unfortunately, for major exposure no specific therapy is known for poisoning by any of the boron hydrides discussed in this report. At present, therapy can only be symptomatic.

In cases of pentaborane and decaborane exposure where muscular twitching is noted, a short-term barbiturate can be used to control any impending convulsions. Barbiturates should be used with caution because of "synergic respiratory depression" (refs. 9 and 10). A late complication may be pulmonary edema, in which case, oxygen therapy should be used (refs. 9 and 10).

In cases of diborane poisoning, oxygen therapy should be used to help combat the pulmonary edema that may develop (refs. 9 and 10).

In the case of the boron hydrides, there is no specific therapy known to combat boric acid poisoning. Symptomatic therapy (ref. 21) can include intravenous administration of fluids and plasma or blood, antibiotics, and possibly adrenal cortical extracts.

### SUMMARY

The toxic properties of pentaborane, diborane, decaborane, and boric acid are summarized as follows:

1. Effects of pentaborane exposure involve the central nervous system. Prolonged exposure may cause damage both to the kidneys and the liver. It is considered more toxic than diborane and hydrogen cyanide.
2. Diborane poisoning causes pulmonary edema and hemorrhage. It can be compared with phosgene (a war-time poison gas), and is less toxic than pentaborane and decaborane in its effects.
3. The accurate toxicological effects of decaborane are unknown but may be compared with those of pentaborane.
4. Boron intoxication may occur from the absorption of boric oxide or boric acid by the broken skin of the body. Continued exposure may cause boron accumulation in the kidneys, liver, and brain.

Lewis Flight Propulsion Laboratory  
National Advisory Committee for Aeronautics  
Cleveland, Ohio, August 21, 1956

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## APPENDIX A

## MEDICAL TERMS

The medical terms used in this report are listed alphabetically and explained as follows (refs. 38 through 41):

Albumin. - Albumin is a protein which, if detected in the urine, generally indicates a kidney dysfunction.

Blood count. - The red blood cells (erythrocytes) are the oxygen carriers of the blood. The normal count varies from  $4\frac{1}{2}$  to  $5\frac{1}{2}$  million and 6 million per cubic millimeter for women and men, respectively.

The white blood cells (leucocytes) have several functions, one of the most important being phagocytosis (ingestion and destruction of bacteria). The average count is 10,000 white cells per cubic millimeter. These cells may be subdivided as shown in the following table (ref. 40):

Type of cell	Percent of cells	Total number/ cu mm
Neutrophils	60-70	4000-6000
Basophils	0.5-1	50-100
Eosinophils	1-3	100-300
Lymphocytes	20-40	2000-4000
Monocytes	2-6	200-600

Blood urea nitrogen (B.U.N.). - Urea is the end product of protein (nitrogen) metabolism. The normal range in the blood is 10 to 15 milligrams per 100 milliliters. High blood urea levels are present in increased protein metabolism, toxic and febrile conditions, dehydration of the blood, and various kidney disorders (ref. 41). In extensive liver damage, the blood urea may fall below normal (ref. 41).

Cephalin - cholesterol flocculation test. - The cephalin - cholesterol flocculation test is a test of liver function. The principle of the test consists in adding a mixture of cephalin cholesterol to a patient's serum. Flocculation (positive reaction) occurs in cases of liver dysfunction. Positive reactions may also be seen in nephritis associated with abnormal protein concentrations (ref. 40).

Central nervous system. - The central nervous system is made up of the brain and spinal cord.

Creatinine. - Creatinine is the end product of creatine (amino acid) metabolism and is a metabolite associated with energy metabolism in muscle physiology. Blood creatinine levels may be used as an index to the filtration power of the kidney. Elevated blood levels are found in severe kidney disorders. The normal blood creatinine range is 1 to 2 milligrams per 100 milliliters of blood; the normal urinary level is 1 to 1.2 grams per 24 hours (ref. 41).

Nonprotein nitrogen (N.P.N.). - The nitrogenous components and their normal levels (mg/100 ml of blood) that make up the nonprotein-nitrogen fraction of the blood are as follows (ref. 41):

Blood components	Mg/100 ml of blood
Urea nitrogen	10-15
Creatinine	1-2
Uric acid	2-3.5
Creatine	3-7
Amino acid nitrogen	5-8
Ammonia nitrogen	0.1-0.2
Undetermined nitrogen	4-10
Total nonprotein nitrogen	25-35

The nonprotein nitrogen of the blood generally determines the filtration power of the kidney; however, a small deviation from the normal level in a determination is not as significant as a deviation in the levels of such individual components as urea or creatinine. High nonprotein-nitrogen levels (nitrogenous retention) indicate functional disorders of the kidney.

Parts per million. - The mathematical expression used to determine the concentration of a toxic agent in air is shown as (ref. 2, vol. I)

$$\text{Parts per million} = \frac{24,450 \times (\text{mg/liter of toxic agent})}{(\text{Molecular weight of toxic agent})}$$

Phenolsulfonphthalein test. - The phenolsulfonphthalein test determines the excretory ability of the kidneys (ref. 42). A certain quantity of dye is injected intravenously into the patient, and the appearance time as well as the amount excreted are noted at various intervals. An increase in appearance time and a decrease in excretory rate indicate kidney dysfunction (ref. 42).

Polymorphonuclear white blood cells. - The polymorphonuclear white blood cells are blood cells which have nuclei of many forms.

Semioliguria. - Semioliguria is the reduced daily output of urine.

Thymol-turbidity test. - The thymol-turbidity test is a check of liver function. In the cases of liver dysfunction, turbidity (positive reaction) results when thymol reagent is added to the blood serum.

## APPENDIX B

## PENTABORANE POISONING

A severe pentaborane exposure as reported by the Callery Chemical Company (unpublished data) is summarized in part as follows:

## Symptomatology

- (1) Convulsions (severe after 2 hr)
- (2) Hiccoughs (began 12 hr after poisoning, lasted 1 week)
- (3) Memory disturbance (1 week from initial attack)
- (4) Fatigue (2 days, then gradual diminution)
- (5) Headache (lasting 2 weeks, old history also)
- (6) Nerve pain (right-ear area, peripheral neuritis)
- (7) Nausea (from exposure time to 5 days)
- (8) Semioliguria (3 to 6 days)
- (9) Temperature (99° to 100° F)

## Physical Findings

The physical findings after pentaborane exposure are normal except as follows:

- (1) Tachycardia on admission, lasting 24 hours; normal electrocardiogram (Tachycardia is the excessive rapidity in the action of the heart, and electrocardiogram is a graphic tracing of the contraction of the heart muscle.)
- (2) Occipital neuritis posterior to the right ear (onset in 7 days; lasted 1 week)

- (3) Rise in the blood pressure, which would read on admission, 100/46; on the 4<sup>th</sup> to 18<sup>th</sup> day, 168/110 (observed when the nonprotein nitrogen and blood urea nitrogen were at their highest); on the 20<sup>th</sup> day, 138/100; and on the 34<sup>th</sup> day, 124/90.

#### Blood Chemistry and Urinalysis

Variations from the normal levels of several components of the blood as a function of time are shown in figure 2. It should be noted that there was a sharp increase in these blood components within 1 week after the exposure. After this rise they began to subside to normal levels. However, in the case of the cephalin-cholesterol flocculation test normal levels were not achieved for the time shown (fig. 2).

Figure 2(b) shows the various tests performed on the urine of the same individual. Variations from the normal levels of these tests are plotted as a function of time. The greatest changes were noticed within 1 week.

#### Hematology

Pentaborane poisoning affects the blood analysis as shown by the following:

(1) Hemoglobin (the normal value obtained depends on the method of determination; by the Sahli method, 13.8 g per 100 cc of blood) was present at its highest value as 13 grams and its lowest as 11.5 grams.

(2) White blood count (normal value, 7000 to 10,000 cells per cc) was high with a reading of 32,000 and low with 7150.

(3) Lymphocytes (a variety of the white blood corpuscles which make up 20 to 40 percent of the total white blood cell count) ranged from a high of 30 percent to a low of 21 percent.

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TABLE I<sup>a</sup>

Name	Formula	Physical state	M.P., °C	B.P., °C	Stability	Hydrolysis
Diborane	B <sub>2</sub> H <sub>6</sub>	Gas	-165.6	-92.5	Fair	Very fast
Pentaborane	B <sub>5</sub> H <sub>9</sub>	Liquid	-46.8	58	Good	Slow
Decaborane	B <sub>10</sub> H <sub>14</sub>	Solid	99.5	213	Very good	Very slow

<sup>a</sup>Taken from ref. 34.

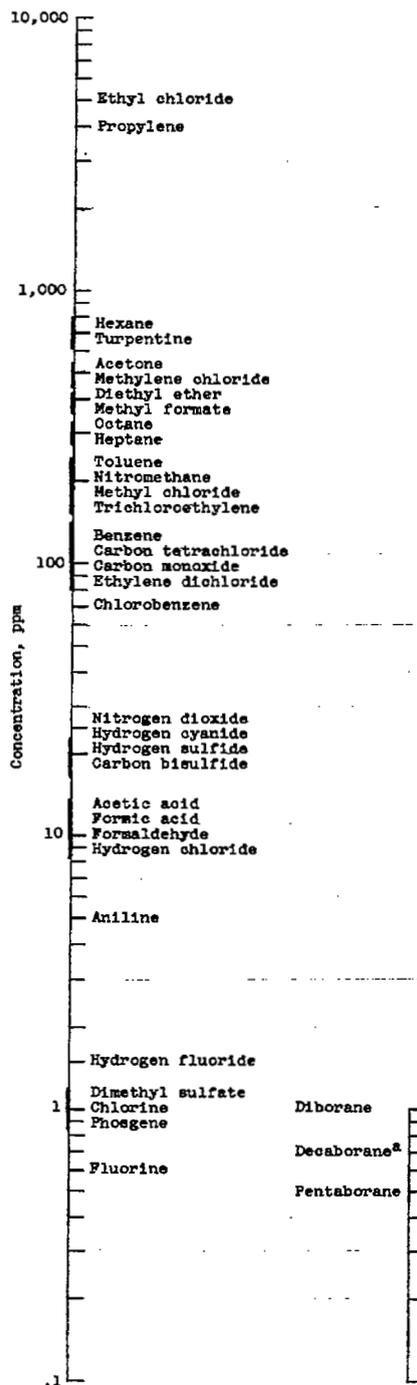
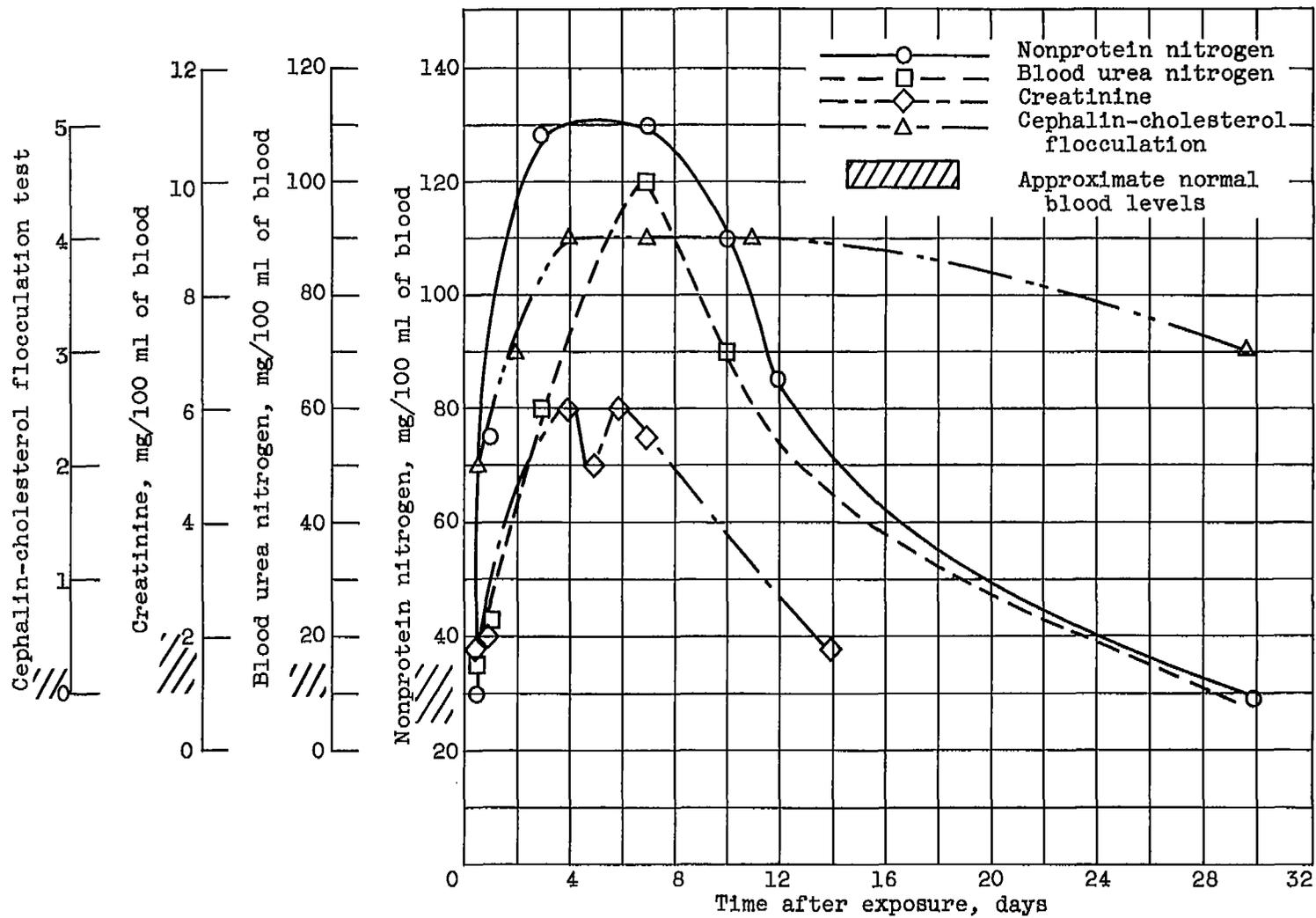


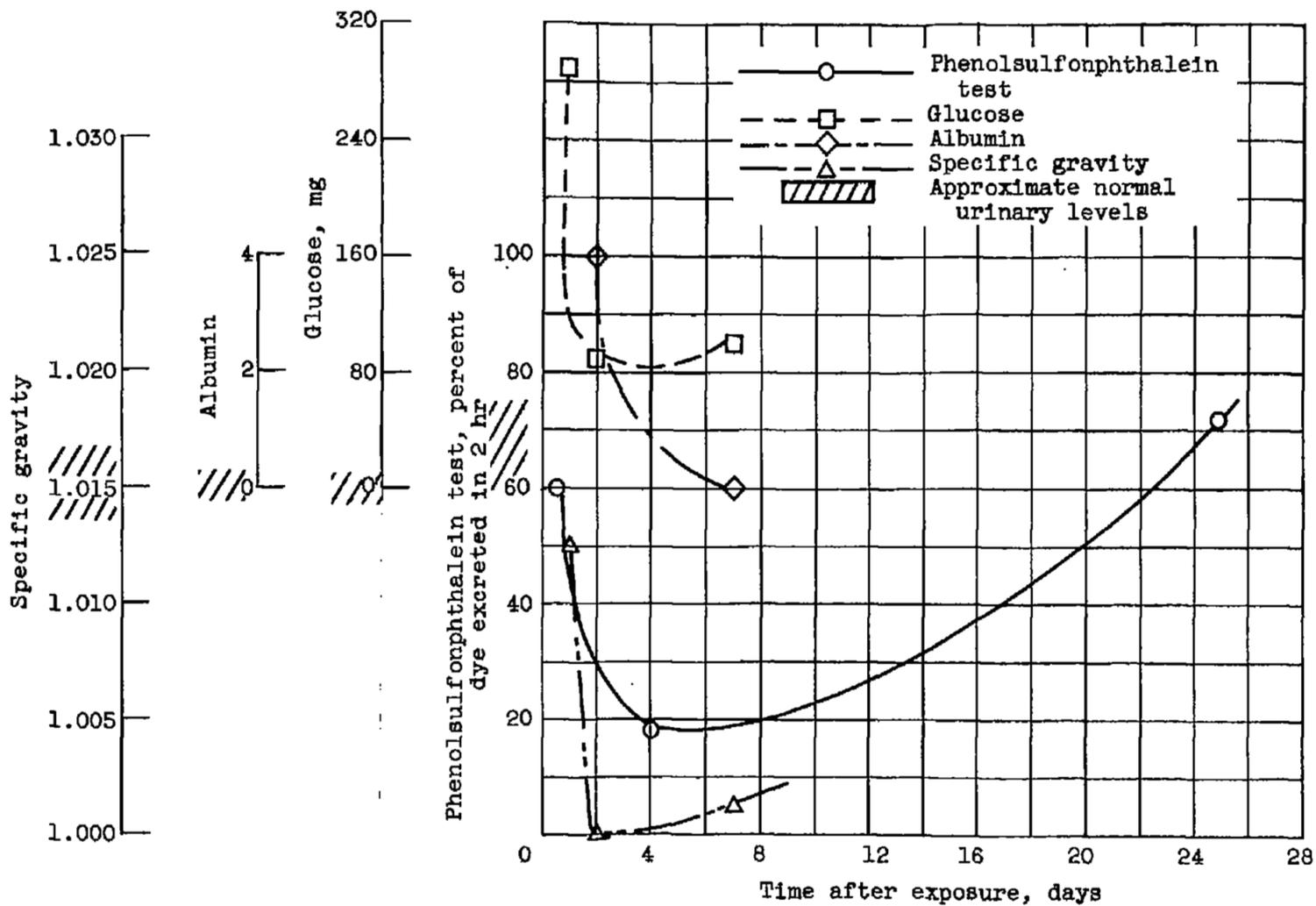
Figure 1. - Comparison of allowable working concentrations, for an assumed 8-hour working period, of common laboratory chemicals with diborane, decaborane, and pentaborane.

<sup>a</sup>Approximate. The toxicity of decaborane is intermediate between diborane and pentaborane.



(a) Blood analysis.

Figure 2. - Analyses following a severe case of pentaborane poisoning.



(b) Urinalysis.

Figure 2. - Concluded. Analyses following a severe case of pentaborane poisoning.

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