THE EFFECT OF METHYL ALCOHOL FUMES ON BRIGHTNESS DISCRIMINATION IN THE WHITE RAT

by

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CONTENTS

I. INTRODUCTION

This investigation was conducted at the psychological laboratory of the University of Kansas under the supervision of Dr. W. S. Hunter to whom I am very grateful for guidance and criticism during the experimental work and for suggestions concerning the preparation of this thesis. Experimentation was begun in March, 1924, and discontinued in May, 1925. The experiment was begun as a repetition and extension of a short study made in this laboratory in 1919 by Marion R. Bills and Oakland Maupin in which their purpose was to determine by an objective method the effect of methyl alcohol fumes on brightness discrimination in the white rat. In my own study, after a short period of preliminary experimentation, it was found advisable to make a change in the method of procedure used by Bills and Maupin. This change will be described later in this paper. However, the problem as I studied it is essentially the same as that of Bills and Maupin.

This thesis represents but one phase of the problem of the toxicity of methyl alcohol, that is, visual defect as a result of the inhalation of the fumes of pure methyl alcohol. The influence of such factors as age, length of the period of exposure, method of administering the alcohol, whether by absorption through the skin, by injection or by ingestion, the species of animal, as well as histological examinations remain for future investigation. The present

work does not pretend to complete the investigation is so large a field, but merely serves to advance the information concerning the problem, and at the same time it may be indicative of research possibilities for the future.

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II. HISTORICAL SUMMARY

The present work is concerned with two main problems, brightness discrimination and methyl alcohol poisoning. Accordingly, the literature bearing upon each topic is treated separately.

A. Brightness discrimination:

But very little work has been done on the problem of brightness discrimination in animals, and a very small percentage of what has been done has been upon brightness discrimination in rats. In most cases birds have been used.

Lashley (3) using rats, and Bingham (1) using chicks studied visual discrimination of size and form, but did not touch upon the problem of brightness.

Lashley (4) in some control tests in his study of the color vision of birds used white light, and found the birds to be markedly photopositive. When two white lights were used, the chicks showed a tendency to go to the brighter of the lights, but their discrimination was very inaccurate. They showed a slight preference for the brighter of two lights of intensities three to one, but they never made ten successive reactions without error.

Coburn (2) found the crow able to distinguish between differences of brightness, size and form. He made no attempt to determine the differential thresholds.

Tugman (7) in her study upon light discrimination in the

sparrow found that when the intensity of one stimulus plate was .098 Hefner unit, and the other one less, the least discriminable difference was .015, .020 and 1035 H. U. Under the same conditions human thresholds varied from .009 to .013 H. U.

Yerkes and Eisenberg (9) trained ring-doves to choose the brighter of two lights before different colored lights of the same intensity were offered.

Reeves (6) shows that rats distinguish between stationary and moving lights.

Pearce (5) trained rats to react to light against darkness using methods similar to those used by Hunter in a study of sound. She found that a visual response is learned more rapidly than an auditory one. Similar results have been obtained in studies of the conditioned reflex.

Yerkes (8) studied brightness discrimination in the dancing mouse. In preliminary experiments using black and white cardboards as stimuli, he conducted a series of tests to determine which stimulus the mouse chose prior to training and without punishment. The results indicate a slight preference for black. Using punishment and reward, a discrimination between black and white was then set up through training. Yerkes (p. 110) says, "The habit of turning in a certain direction or of choosing by position can be formed more readily than a habit which depends upon visual discrimination."

The differential limen was found with gray papers. The

mice could be trained to discriminate between Nendel gray papers No. 10 and No. 20, but not between No. 10 and No. 15. Again using transmitted light, Yerkes found Weber's law to hold for brightness vision of the dancer.

In pointing out the increase in efficiency of brightness discrimination with training, Yerkes says (p. 128), "At the beginning of the experiments, a difference of one-half did not enable the mouse to discriminate as certainly as did a difference of one tenth after she had chosen several hundred times." The limit of discrimination was between one tenth and one fifteenth for the one mouse that was used in these tests.

Bills and Maupin (21), using the Yerkes discrimination box and brightness apparatus, trained their rats to discriminate light from darkness. They then gradually decreased the intensity of the light until the threshold for brightness was found. The limen measured in terms of amperes of the current to the 25-watt mazda lamp was approximately .13.

In general, the results of all investigations of brightness discrimination in rats indicate that rats are able to distinguish light from darkness when the light is of a very low intensity. But, with the exception of the work by Yerkes on the dancing mouse, there is no data on the differential limen.

B. Methyl alcohol poisoning:

A large percentage of the earlier experimental investiga-

tions with alcohol was made for the purpose of determining the relative toxicity of various alcohols. Much of the more recent literature also touches upon this question, but as yet there seems to be no very definite agreement as to the exact position which methyl alcohol holds in the scale of toxicity of alcohols.

The experimental evidence bearing upon the physiological effects of methyl alcohol and its behavior in the animal organism is extremely limited. Consequently, the exact nature of the bio-chemical changes which take place in the organism upon the introduction of methyl alcohol in the body is not known.

The literature of the past ten years is composed mainly of clinical observations and reports of cases of methyl alcohol poisoning, with an occasional discussion of the underlying causes of the harmful effects of methyl alcohol upon human and animal organisms.

Although the literature contains very little conclusive evidence concerning the effects of methyl alcohol upon vision and, therefore, has no direct bearing upon my own study, and although I have been unable to get direct access to much of it, I shall review it briefly in order to show more clearly the present status of knowledge in the field, and because of the value it might have as an explanatory supplement to the results obtained in the present investigation. I shall first consider the studies which are concerned chiefly with the question of the relative toxicity of alcohols, from which it may be observed that there is no conclusive experimental evidence that

methyl alcohol is more toxic than ethyl alcohol.

Cros (28)*, in 1863, performed the first experiment with methyl alcohol from which he concluded that methyl alcohol is less poisonous than ethyl alcohol.

Rabuteau (69)* found methyl alcohol to be the least poisonous of all alcohols and established the law that the toxicity of alcohols increases with their boiling point.

Richardson (70)* found that the toxicity of alcohols increases with the number of atoms of carbon. Thus, methyl alcohol would be the least poisonous.

Houdaille (49)*, experimenting with fish in the laboratory of Richet, obtained results comparable to those of Richet (72)* and Picaud (66)*. They all agree that the toxicity of alcohols increases with their insolubility, which conforms to the law of Rabuteau.

Joffroy and Serveaux (52)* (53)*, using the method of intravenous injections, found that for dogs the mortal dose of methyl alcohol was 9 grams per kilogram of the animal's weight, while the mortal dose of ethyl alcohol was only 8.6 grams per kilogram. According to these authors the toxicity increases with the molecular weight, beginning with methyl alcohol which is the least poisonous.

Note: Those references marked with a star were not available and all the information concerning them which I was able to obtain was found in other articles. Neither from the articles read nor from cross references, however, was there any suggestion that any work has been done which closely resembles the present investigation, with the exception of the study already referred to, by Bills and Maupin.

Führer (4) found that for embryos of sea urchins ethyl alcohol was three times more toxic than methyl alcohol and three times less toxic than propyl alcohol.

Billard and Dieulafé (19)* (20)* have found the toxicity of alcohols directly proportional to viscosity and superficial tension.

By means of intra-venous injections in rabbits, Lesieur (57), like other investigators, placed methyl alcohol at the foot of the ladder of toxicity. He concluded that the toxicity of alcohols increases with their molecular weight, that is, with the number of molecules of carbon, with their boiling point, and with their insolubility.

Dujardin, Beaumetz and Audigé (32)*, performing experiments with dogs, fixed the extreme toxic dose of methyl alcohol at 7 grams per kilogram of body weight, and of ethyl alcohol at 7 grams per .75 kilogram. They concluded that the toxicity of alcohols, with the exception of methyl alcohol, increases with the number of molecules of carbon.

Another investigator, Lewin (58)*, found methyl alcohol to be more poisonous than ethyl alcohol.

In order to account for cases of poisoning by methyl alcohol, and to explain the results of occasional experimental investigations which show methyl alcohol to be an exception to the law of toxicity of alcohol, various authorities have put forward the impurity or strange substance hypothesis; that is, that the unusual toxicity

of methyl alcohol is due to impurities in the alcohol and not to any property of the alcohol itself. The following brief resumé of the literature which touches upon this phase of the problem indicates that there is a paucity of experimental fact but an abundance of speculation and theorizing about the nature of the foreign substances and their importance in determining the harmful action of wood alcohol upon the human organism and upon vision.

Daremberg (30)* stresses the great toxicity of superior alcohols and strange substances, such as "furfurol".

By placing fish in water mixed with either ethyl or methyl alcohol, Cololian (26) studied the phenomena produced by intoxication. He found that drunkenness from methyl alcohol passed away but that the fish died in a short time after it was returned to fresh water. His belief was that drunkenness from ethyl alcohol was so grave that if the fish did not die in the experimenting tank, it would live for a long time when returned to running water. According to Cololian, the mortal dose is not related to the weight of the fish, but is the same for both small and large fish. His final conclusion was that those alcohols which have high boiling point, greater molecular weight, and least solubility are the ones which poison gravely and mortally. He added, that theoretically methyl alcohol is less poisonous than ethyl alcohol, but that in practice it is very difficult to obtain chemically pure methyl alcohol. Instead, it contains traces of aceton which increases its toxicity.

Arends (12)*, in a review of the question of the toxicity of methyl alcohol, gave the belief that the reported toxic effects of methyl alcohol are due to impure alcohol or crude wood spirit used for denaturing ethyl alcohol.

In the Berlin Letter (18), which was written concerning the series of deaths that occurred in Berlin as a result of wood alcohol poisoning, Eisne and Zuntz were mentioned as authorities who believed that there is a justifiable doubt whether methyl alcohol as such played the principal role in the Berlin episode.

Kroeber (54)* thought that the cause of the Berlin catastrophe was the presence in the alcohol of the extremely poisonous methyl sulphate, which he believed to be formed during the process of the manufacture and purification of commercial wood alcohol.

From experiments on dogs, Ziemke (96)* found that when ordinary wood alcohol was administered, the dogs died in a short time, but when pure methyl alcohol was administered, they did not die until after the lapse of several months.

In the report on wood alcohol by the Factory Investigating Committee (36), Vandevelde is mentioned as having claimed to have proven that methyl alcohol free from all impurities is less toxic than ethyl alcohol, and that the toxicity of commercial wood alcohol is largely due to the presence of impurities.

Goldsmith and Kobert, cited by Robinson (74), agree that the ill health of factory workers is in part due to piridin bases

used in denaturing the alcohol.

Franceschi (37)* administered 32 grams of pure methyl alcohol to himself daily for nine months without any appreciable injury resulting. He concluded that the toxic elements in wood alcohol are fusel oil and other substances.

Hunt (50) argues that, although fusel oils, furfurol, etc. are more poisonous than ethyl alcohol, they are present in even the worst grades of liquor in such small quantities that in order to get a fatal dose of the impurities, many times the fatal dose of ethyl alcohol would have to be consumed. He believes it probable that these impurities do increase the toxicity of methyl alcohol, but that methyl alcohol is the chief toxic agent.

Among others who disagree with the impurity hypothesis are Tyrer and Gosling (79).*

The following statement is made in an editorial in the Journal of the American Medical Association (33), p. 1975: "Methyl alcohol is itself a poison and that fact must not be overshadowed by more problematic possibilities of incidental impurities. The 'impurity' hypothesis is often given in instances in which lack of knowledge or rather paucity of accurate information of all the factors has driven an observer to seek a new explanation for his facts."

Several investigators have given evidence to show that the relation between the toxicity of methyl and ethyl alcohol is not the same in chronic intoxication that it is in acute intoxication.

Although these investigations are not very numerous and there is almost no comparison of the influence of different methods of administering the alcohol, all agree that in chronic intoxication, methyl alcohol is more poisonous than ethyl alcohol.

Pohl (67), experimenting with dogs and rabbits suggests that the extreme toxicity of methyl alcohol is found in cases of chronic intoxication. He found that with small doses of methyl alcohol given at intervals of two days, the animals died within a few weeks, but that such difficulty was not encountered with ethyl alcohol. Ethyl alcohol could be administered for months without causing marked anatomical and functional disturbances.

Joffroy and Serveaux (52)*(53)* found that in chronic poisoning, methyl alcohol is unusually poisonous, not only more so than ethyl alcohol, but that animals could stand fairly large doses of furfurol for longer periods than they could methyl alcohol. They concluded that it is impossible to foretell the toxicity of an alcohol in chronic poisoning by experiments on acute poisoning.

Birch-Hirschfeld (22)* spoke of the difficulty of keeping animals alive for even short periods when small doses of methyl alcohol were given at short intervals.

Hunt (50) performed experiments on sub-acute poisoning where comparatively large but not immediately fatal doses of methyl alcohol were given to dogs and rabbits. Tests were made with commercial grain alcohol, and pure ethyl alcohol. In all cases where

the animal received methyl alcohol, death resulted; while those that received equal or larger doses of ethyl alcohol did not die. It was also noticed that when the dose was slightly larger, death was produced by one alcohol almost as rapidly as by another. Hunt therewith concluded that the great difference between the toxicity of methyl and ethyl alcohol might be overlooked entirely in experiments upon the effects of single large doses.

Nicloux and Placet (63), using rabbits and dogs, studied intoxication by methods of ingestion, intra-venous injection, and inhalation of vapor of alcohols and concluded that by a large dose in intravenous injection, methyl alcohol is less poisonous than ethyl alcohol, but by ingestion and with doses repeated every twenty-four hours, the toxicity of methyl alcohol is the greater.

From the results of extensive physiological experiments with guinea pigs, Langgaard (56)* concluded that small doses of methyl alcohol repeated daily are more poisonous than ethyl alcohol, but in single large doses ethyl alcohol is the more poisonous.

Cutler (29) says that chronic poisoning in man which follows exposure to methyl alcohol fumes is more insidious than the cases of acute poisoning which lead rapidly to death and to blindness.

It is a well established fact that ethyl alcohol is largely oxidized within the body to carbon and water which are harmless products. On the other hand in the investigations concerning the oxidation of methyl alcohol within the animal organism, the bio-chemical

changes and the exact nature of its physiological effects, the results are in no way conclusive. However, it is generally accepted that formic acid is the agency which produces degeneration of the optic nerve and, consequently, blindness. But there is no agreement upon the details of this transformation and degeneration. Although the evidence bearing upon these questions is scattered throughout the literature and it is not at all conclusive, they are more directly related to the present investigation, and I shall present both data and arguments for what they may be worth in this study and in future experimentation.

In his animal experiments, Pohl (67) found that narcosis with methyl alcohol intoxication lasts for several days, longer than with ethyl alcohol; that the methyl alcohol is slowly eliminated and partially transformed into formate. Formic acid is formed and reaches its maximum three or four days after ingestion. He believed that this is not due to an accumulation of formic acid, but that methyl alcohol or some unknown intermediate product of the formation remains in the body during this time. Other investigators have believed that this intermediate product is formaldehyde, but Pohl gives evidence against this theory. Since the physiological effect of formaldehyde is so different from methyl alcohol poisoning, he concluded that the formation of formaldehyde could not take place in the oxidation of methyl alcohol. Mayer (60)* has found the fatal dose of formic acid for rabbits, which is one sixth as large as the fatal dose of methyl al-

cohol. Thus formic acid is six times more poisonous than methyl alcohol. Aronsohn (13)* found formaldehyde to be thirty-three times more poisonous than methyl alcohol. Pohl found a close parallelism between the intensity of the intoxication and the excretion of formic acid. He thought it probable that all methyl alcohol administered is converted into formic acid and is then oxidized to carbon dioxide, but Bongers (23)* found that considerable quantities of methyl alcohol are excreted in the urine.

Joffroy and Serveaux's (52)* (53)* results show that the state of coma does not appear for some time, even when methyl alcohol is injected directly into the vein of the animal, and lasts for a longer time than with ethyl alcohol. With methyl alcohol intoxication the state of coma may last for two, three, or four days, but with ethyl alcohol it appears immediately and usually lasts about six hours, never longer than twenty-four hours.

From an examination of 80 patients poisoned with liquor substitutes, Yakovleva (90)* concluded that the cumulative action is a peculiarity of methyl alcohol poisoning. The symptoms developed only after repeating the dose, usually the next day.

Völtz and Dietrich (85) concluded from respiration experiments with dogs that after a dose of 2 cc. of methyl alcohol per body kilogram, complete elimination comes only after three or four days, and therefore, the consumption of larger amounts over several days must lead to an increase of poison and to death. Similarly they

found that within twenty hours after consumption of 2 cc. of ethyl alcohol per body kilogram, it was completely oxidized in the organism.

The great toxicity of methyl alcohol is explained in much the same way by Harnack (46)*. The alcohol is slowly oxidized in the animal and the human body to formic acid which can be detected in the urine. Formic acid is an aldehyde, and the toxicity of an aldehyde decreases with the number of carbon atoms which is in direct contrast to the toxicity of alcohols which increases with the number of carbon atoms.

Nicloux and Placet (63) noted the prolonged drunkenness from methyl alcohol. They found that methyl alcohol is eliminated more slowly than ethyl alcohol, and that it is eliminated more slowly in the dog than in the rabbit. With one dog they found that ethyl alcohol was eliminated in twenty-three hours, and that methyl alcohol was not eliminated for five days. In the rabbit from 92% to 96% of the methyl alcohol is oxidized or transformed, and in the dog from 70% to 75% of methyl alcohol and almost 95% of ethyl alcohol is oxidized. Nicloux and Placet believed that methyl alcohol in reality is no more poisonous than ethyl alcohol, but that the harmful results are due to the phenomenon of accumulation. They concluded that incomplete combustion, slow elimination, and possible accumulation are important factors in the harmful action of methyl alcohol, but are not sufficient to explain all the cases of poisoning reported in medical literature, and that either the impurities which accompany a cheap grade of methyl alcohol play a large part or else man is more sensitive than lower

animals to the poisonous nature of methyl alcohol. In determining the amount of alcohol in the body at the time of death, Nicloux and Placet found a greater proportion in the brain. They also noted a dilation of the pupil of both the rabbit and the dog.

The experimental evidence, bearing upon visual disturbances and nerve degeneration, is very meager, but from the few experimental studies which have been made, together with the ophthalmascopic findings, we have a fairly complete picture of the final results, but no very definite notion of the way in which these changes have come about.

Joffroy and Serveaux (52)* (53)* noticed convulsive movements of the eyes, often pronounced nystagmus, and usually dilation of the pupil.

Hunt (50) said that the organs most affected by methyl alcohol are the most highly differentiated nerve structures as is shown by the deep and prolonged coma, and by blindness in man and monkeys. In his investigations of sub-acute poisoning, his primary purpose was not to study the influence upon vision, but incidentally in his experiment upon dogs he noticed a purulent or fibrino-purulent conjunctivitis, and in at least one case the animal seemed blind. He concluded that the long continued effects of methyl alcohol in acute poisoning and the ease with which a chronic condition is produced by small repeated doses depends upon the slowness with which the alcohol and its oxidation products are eliminated, that this is the probable cause of blindness which so frequently follows methyl alcohol

poisoning in man, and that the highly differentiated nerve structures such as those of the retina are especially likely to suffer when exposed to the action of a poison for a long time.

H. Woods (90) considers formaldehyde a by-product of methyl alcohol. He says that small and repeated doses may not cause intoxication but still may lead to serious results on the nervous system, and that the cause of blindness is atrophy of the optic nerve with probable destruction of the ganglion retinal cells. He finds that some persons are more susceptible than others to the influence of methyl alcohol.

De Schweinitz (76)* gave ethyl alcohol to a monkey for several days in succession. There were no indications of visual disturbance, nor were there any degenerative or inflammatory changes found when the animal was killed and a microscopic study was made of the eyes and optic nerve.

Having noticed the close resemblance between methyl alcohol blindness and the diminution of vision or blindness resulting from profuse or repeated hemorrhage of the gastro-intestinal tract, Holden (48) made experimental investigations to find data bearing upon this resemblance. He bled dogs and rabbits. One or two days later there were signs of oedema of the nerve - fibers and ganglion cell layers of the retina and some of the ganglion cells showed evidence of beginning degeneration. Two weeks later advanced degenerative changes were noticed, however, the retinal neurones suffer more than the basal

and cortical neurones of the visual tract. After methyl alcohol was given to several dogs, autopsy showed great congestion and numerous hemorrhages in the menginges of the cord and brain, slight oedema of the nerve fiber and ganglion cell layers, and degeneration of many ganglion cells. Holden thereupon concluded that amblyopias due to methyl alcohol come into the category of those due to nutritive disturbances. The animals, according to Holden, did not prove to be so susceptible to the toxic effects as might have been expected from what is known concerning the effects of methyl alcohol on man.

Norris (64), in a discussion of the lesions in wood alcohol poisoning, says that methyl alcohol attacks the most highly differentiated nerve elements causing prolonged coma and blindness, and it has a marked selective affinity for the most highly differentiated nerve elements of man which are therefore more rapidly and severely damaged than those of lower animals. He adds that drunkenness in man from methyl alcohol is almost uniformly fatal.

Other evidence to indicate man's peculiar sensitivity to methyl alcohol is found in the results of animal experiments by Harnack (46)*. He concluded that methyl alcohol is more severe on the human body than in animals. The Factory Investigating Committee (36) in their report on wood alcohol made the statement that methyl alcohol is less poisonous to lower plants and infusoria than ethyl alcohol, but for higher animals and especially for man, it is a severe toxic agent.

Birch-Hirschfeld (22)* made an experimental study of the effects of methyl alcohol upon the retina and optic nerve of dogs and monkeys. He used pure methyl alcohol diluted with several times its volume of water, given in doses of three to six or seven cubic centimeters every one or two days. When near the point of death the animals were killed and their eyes and optic nerves were studied microscopically. Of the three monkeys, two had marked degenerative changes and one was totally blind. Similar histological changes were found in the retinas of the three dogs although it had been impossible to detect disturbances of vision during life. Birch-Hirschfeld concluded that methyl alcohol was a nerve poison, causing pathologic changes in ganglion cells of the retina and cornea and a degenerative change in the optic nerve. He also says that blindness ensues after acute intoxication and even after very small doses. It is said (Moulton and Moulton (60)) that one teaspoonful of wood alcohol has caused blindness and one ounce has caused death.

Norris (64) criticizes the method of examination of sections of the optic nerve. He considers such a method unsatisfactory because the changes in the nerve are slight when compared with the normal nerve. Ziegler (94) believes that animal experiments are not entirely dependable since post-mortem degeneration is too rapid and interferes with accurate study of these tissues. There seem to have been no histological examinations of human eyes and brains subjected to methyl alcohol.

Tyson and Schoenberg (80), by means of inhalation, introduced

alcohol into the system of guinea-pigs, rabbits, dogs and a monkey. Some were kept in a closed space, others had free ventilation, and for others fresh air was pumped into the container. Periods of exposure varied from two to three and six hours at a time. In all cases one prolonged exposure to inhalation of the vapor without free ventilation produced loss of consciousness, loss of pupillary reflex, slight contraction of the pupils, marked ocular tension, coma and death. A similar exposure with active ventilation produced no such marked effect. The injurious effect upon the eyes and general health was proportionate to the amount of methyl alcohol used, the humidity and temperature of the air and length of exposure, as well as the species of the animal. The susceptibility increases with the development of the animal species. Among the animals upon which investigation was made, the monkey proved to have the greatest susceptibility to its deleterious effect.

It was found that the electro-conductivity of the blood serum was increased after inhalation of methyl alcohol fumes. Tyson and Schoenberg say that this increase is produced by a change in the corpuscles, either from an increase in Hydrogen-ion content or from an increase in organic salts. An increase in Hydrogen-ion content produces an increase in the acidity of the blood, greater viscosity, and greater specific gravity. This they consider an important factor in the causation of symptoms produced by methyl alcohol poisoning. The aqueous humor is normally neutral but after the inhalation

of methyl alcohol vapor its reaction is acid. These investigators came to the final conclusion that methyl alcohol is a true haemotatoxic which has a decided effect upon all the tissues producing degeneration, and under extreme conditions, amblyopia and death.

Grignolo (44) by feeding methyl alcohol to dogs obtained results similar to those of Tyson and Schoenberg. He found that the morphological changes which result are wrinkling of the ganglion layer and atrophy of the optic nerve. He believed that the epithelial cells of the ciliary body are particularly affected by methyl alcohol.

Most of the investigations which have been described have been concerned with the bio-chemical changes in the organism upon the administration of alcohol. In some of the studies, because the animal's behavior seemed to indicate visual defect or because degenerative changes were observed in the retina and optic nerve, it has been assumed that the animal was blind. However, in none, with the single exception of the experiment by Bills and Maupin, has there been an objective study of the effects of methyl alcohol upon vision.

Bills and Maupin (21) studied the effects of methyl alcohol fumes upon brightness discrimination in white rats. The rats were trained to discriminate between light and darkness, the threshold of brightness sensitivity was found, the rats were placed in alcohol fumes, and then they were retested in the discrimination box. Most of the animals remained in the fumes constantly, and in no case was there any loss of discriminating ability. That the fumes do exert a

harmful physiological effect there can be no doubt, for all of the animals became sick and badly bloated and some of them died.

The clinical picture of methyl alcohol poisoning as obtained from case reports adds considerable to the knowledge gained from experimental investigations. The first case of wood alcohol blindness in man was recorded by Mengin in 1879. Since 1900 the cases have become more numerous and a large percentage of the literature upon blindness from methyl alcohol is in the form of case reports by physicians. The following brief summary of a few of these cases shows that more deaths and more cases of blindness have been caused by ingestion of wood alcohol than by inhalation of the fumes, and more have been due to inhalation than to absorption through the skin. However, we cannot assume on the basis of this information that methyl alcohol is more poisonous when taken into the body by one method than by another, because the general political, social and industrial conditions make wood alcohol more accessible in one form than in another.

In 1903-04 Buller and Wood (25) had collected histories of wood alcohol poisoning and found 275 instances of death or blindness due to drinking or inhaling of some form of 'deodorized' wood alcohol. The Report on Wood Alcohol by the Factory Investigating Committee (36) states that data obtained from the literature indicate that drinking of liquor containing methyl alcohol is responsible for most of the deaths and blindness attributable to methyl alcohol.

Fridenberg (38), Robinson (74), Hiram Woods (89)*, and

Buller and Wood (25) cite cases of blindness through absorption of methyl alcohol through the skin. The Report on Wood Alcohol (36), which gives a summary of methyl alcohol poisoning, says that poisoning through absorption is much like that caused by ingestion only it is slower.

Cases of blindness through inhalation of the fumes are less numerous than those caused by drinking wood alcohol, but there are several authentic records of such blindness. In summarizing the cases, Ziegler (94) finds 11 recorded by Buller and Wood, 2 by Gruening, 3 by Tyson, 1 by de Schweinitz, and 2 by himself. Tyson and Schoenberg (80) find 100 cases of amblyopia and death resulting from inhalation of wood alcohol fumes reported in the literature prior to 1912.

The Factory Investigating Committee (36) stresses the importance of the factor of poor ventilation as a cause of wood alcohol poisoning through inhalation of the fumes. Casey Wood (88) says that poisoning by inhalation of methyl alcohol fumes generally occurs from inhaling the fumes and rebreathed air in a confined space. Hiram Woods (90) agrees that in all cases with which he is familiar, inhalation blindness has occurred in persons working in closed places where rebreathed air necessarily becomes charged with wood alcohol fumes.

Bankerville (16) collected 60 cases of chronic poisoning due to drinking of small quantities or to absorption of wood alcohol for a long period of time. Vision was often reduced to half, but none of the patients were blind and some had been exposed to the fumes

for from two to thirty years. Gruening (45) tells of having seen cases of blindness due to inhalation of methyl alcohol fumes, but finds that in all those cases, sight was recovered.

In general the reports of clinical observations of wood alcohol poisoning agree upon the symptoms and diagnostic distinctions, although there are slight differences which may be due to individual susceptibility to the poison, the amount of alcohol administered and the method of exposure. The ophthalmascopic descriptions of wood alcohol blindness agree fairly well with the histological examinations of the eyes and optic nerves of animals poisoned with methyl alcohol.

Fridenberg (38), as a result of ophthalmascopic studies of a series of cases, has given the following description of the nerve-head as it appears in wood alcohol poisoning: In the later atrophic stages, a deep excavation is noticed, extending almost to the scleral ring and at times including it so as to simulate a glaucomatous cupping. The excavation shows distinctly mottled markings of the lamina cribrosa. The color is pure white and at times silvery and glistening. Other ophthalmologists have reported that the nerve-head is white or a bluish tint. In the acute stages, Fridenberg noticed that the discs may appear intensely red and somewhat blurred. The condition of neuritis passes and the disc appears swollen, prominent, soggy and white, the veins large and tortuous and the arteries narrow. According to Fridenberg, no other toxic agent produces intense edema of the optic nerve followed by rapid and complete degeneration. He says,

(p. 514) "Nerve degeneration is so marked and complete as to cause not only a saucer-like excavation, but a glaucomatous cupping." He believes that atrophy in wood alcohol amaurosis is undoubtedly a post-neuritic atrophy, and there is always evidence of a previous neuro-retinitis about the central vessels and in the retina immediately surrounding the nerve-head. His theory is that the poison is not alcohol but a decomposition product of oxidation in the form of an aldehyd as formalin or formaldehyde circulating in the blood as a result of incomplete combustion of the methyl alcohol ingested, inhaled, or otherwise introduced into the system. This decomposition product causes intense edema with some neuritis followed by atrophy of the optic nerve which is the cause of wood alcohol amaurosis.

Nearly all reports make note of the dilation of the pupils. Some also remark upon the sluggish action of the pupils, that they may or may not react to light and convergence. Otherwise there usually is no change in the external appearance of the eyes, however, the Report on Wood Alcohol (36) described the inflammation and burning of the eyes. Jones, in a discussion of Rightor's (73) report, says that in some cases there is an inflammation of the conjunctiva, and that blindness may be prior to or at the same time as this inflammation.

Yakovleva (92)* says that amblyopia sets in rapidly in a few days or weeks with great impairment of visual acuity, that there is frequent appearance of absolute central scotoma and a concentric narrowing of the field. Robinson (74) tells of the contraction of the

color fields and in a perfect case of the appearance of scotoma. Rightor (73) says that no other disease which causes blindness will produce the same reduction of vision in both eyes simultaneously. Cutler (29) says that partial or complete blindness may follow even a small dose of wood alcohol, and in chronic cases there is a chronic conjunctivitis and pharyngitis with moderately congested or atrophic nerves. Vision is often reduced to half the normal. Of the 60 cases examined by Franceschi (37)* there was complete blindness in 50% of the cases and pronounced amblyopia in all.

Hiram Woods (90) says the cause of blindness is atrophy of the optic nerve with probable destruction of the ganglion retinal cells. There is either a total loss of sight or central scotoma, and the ultimate result is seldom better than the poorest vision at any given time, but in the meantime there may be many variations in vision. He says that color losses indicate that the central cells are partially though as yet not entirely destroyed and that fibers of the optic nerve are undergoing atrophy. Yakovleva (92)* tells of a secondary aggravation of the impairment of vision followed by a temporary improvement. Cutler (29) says that the persistence of the symptoms and progress of the atrophy of the optic nerve, after a brief period of improvement, to total blindness is a characteristic of wood alcohol poisoning.

Franceschi (37)* has found that the visual disturbances developed about the third day in the majority of cases and continued a progressive course for from two to four weeks after which there was

usually a slight temporary improvement. Ziegler (94) says that blindness may be early, sudden and complete, and that there may be a marked recovery, sometimes permanent, but more often there is a gradual failure and ultimate blindness ensues. Moulton and Moulton (62) have observed that blindness usually occurs on the second or third day after the symptoms of general intoxication have disappeared. At first optic neuritis is observed and in less than a week atrophy appears, and the field is contracted with scotoma. He says that 95% remain permanently blind or practically so.

Hurst (51) describes a case which he calls "flame blindness." When vision had improved a great deal, the patient could not locate a 100 C. P. light. The color vision was normal but the field was contracted. Later he could distinguish light by holding it at the periphery of the visual field.

Mann, in discussing Rightor's (73) paper, says there is first a retro bulbar neuritis, that is, an optic inflammation starting back in the optic tract and extends down so that optic atrophy cannot be diagnosed with an ophthalmascope until later. Birch-Hirschfeld (22)*, Holden (48), and de Schweinitz (76)* hold the view that the ganglion cells of the retina are injured first, while others believe that the optic nerve fibers are attacked first.

The question of tolerance or of an acquired immunity to methyl alcohol has been mentioned by a very few authors. Cutler (29) says that this is an interesting question and has not been suffi-

ciently studied. He also says that it is customary to think of formic acid, which is formed in the system and slowly excreted, as a cumulative poison, but it might be possible that in some cases the transformation may be altered by rapid oxidation or the tissues may be more resistant. Cutler tells of a physician who believes that immunity is created by continued use of wood alcohol. In a letter to Cutler, this physician says that in his city is a formaldehyde plant in which the purest wood alcohol is used and to his knowledge there have been no cases of wood alcohol poisoning. The natives drink it as a regular thing; and one man is mentioned in particular who for years has taken a quart of denatured alcohol once a month.

Hunt (50) says that a large percentage of the human race have acquired a certain degree of tolerance for ethyl alcohol but not for methyl alcohol, that this is almost always true of those who die of methyl alcohol poisoning.

To Ziegler (94) the fact that painters can work in a tainted atmosphere for long periods without succumbing to the poison demonstrates that the system can acquire a certain tolerance for toxic substances.

In view of the evidence herein presented we may conclude that methyl alcohol is more poisonous for animals of a highly developed nervous system than for lower animals. This extreme toxicity is due to the slow combustion and elimination which results in an accumulation of highly poisonous oxidation products, that is formic acid and possibly

formaldehyde. These poisons are particularly effective upon the highly differentiated nervous centers, producing atrophy of the optic nerve and degeneration of the ganglion cells of the retina, which result in complete or partial blindness. Not all individuals are equally susceptible to the poison, and there is a possibility that acquired immunity may account for this difference.

The literature upon vision of animals is rather limited and very little of it is concerned with brightness vision. The Bills-Maupin paper is the only one found in the literature which gives an experimental determination of the effect of methyl alcohol on vision. It is this work which my own investigations were intended to supplement.

III. EXPERIMENTAL WORK

A. Apparatus.

The apparatus used in the present investigation consisted of a Yerkes discrimination box and brightness apparatus as shown in Fig. I, and an alcohol box, Figs. II and III. The brightness apparatus was set up in a dark room in which the only illumination, aside from the stimulus lights, was a 25-watt mazda lamp, heavily shaded with four or five thickness of paper toweling, and hung directly over the discrimination box. This light served to illuminate all parts of the discrimination box equally. A heavy cardboard was placed over the discrimination box above the stimulus plates of the light box so that they were not directly illuminated by the light above.

The brightness apparatus was 179 cm. long, 39.5 cm. high, and 40 cm. wide, and was divided into two compartments by means of a lengthwise partition placed vertically through the center. Each compartment was covered with a door or lid which opened outward and closed tightly so that very little if any light shone through. In each compartment a 25-watt mazda lamp was attached to a carrier that moved on ball bearings along a track on the floor. Since mazda lamps burn with the same intensity only for approximately fifty hours, the lights in the light box were changed for new ones whenever they had been used fifty hours. At the end of the light box nearest the discrimination box in each compartment was a heavy glass through which

the light passed and which served to shut out the heat of the lamps from the alleys of the discrimination box, thus eliminating a possible thermal cue for discrimination.

Between the light box and the discrimination box was the movable stimulus adapter, a board 68 cm. by 17 cm. in which were three round stimulus plates 6.5 cm. in diameter. The stimulus adapter slid back and forth in such a way that two of these holes coincided with the lights of the light box. At first the middle hole was covered with heavy black paper and the other two with white paper, so that, regardless of the position of the stimulus adapter, one side of the discrimination box was light and the other dark, and the light stimulus could be shifted from one side to the other by moving the stimulus adapter. In later experiments the black paper was removed from the center hole and all three were covered with the same sort of white paper, so that the stimulus on each side was light. The stimulus adapter was now kept stationary, and the intensity of the stimulus was changed between trials by raising the lid of the light box and moving the lamps either closer to or farther from the stimulus plates.

The lamps of the light box were set up in series on the regular lighting circuit, the wires entering the light box through very small holes at the end farther from the discrimination box. A voltmeter was placed in circuit with the lights, and the voltage was recorded daily.

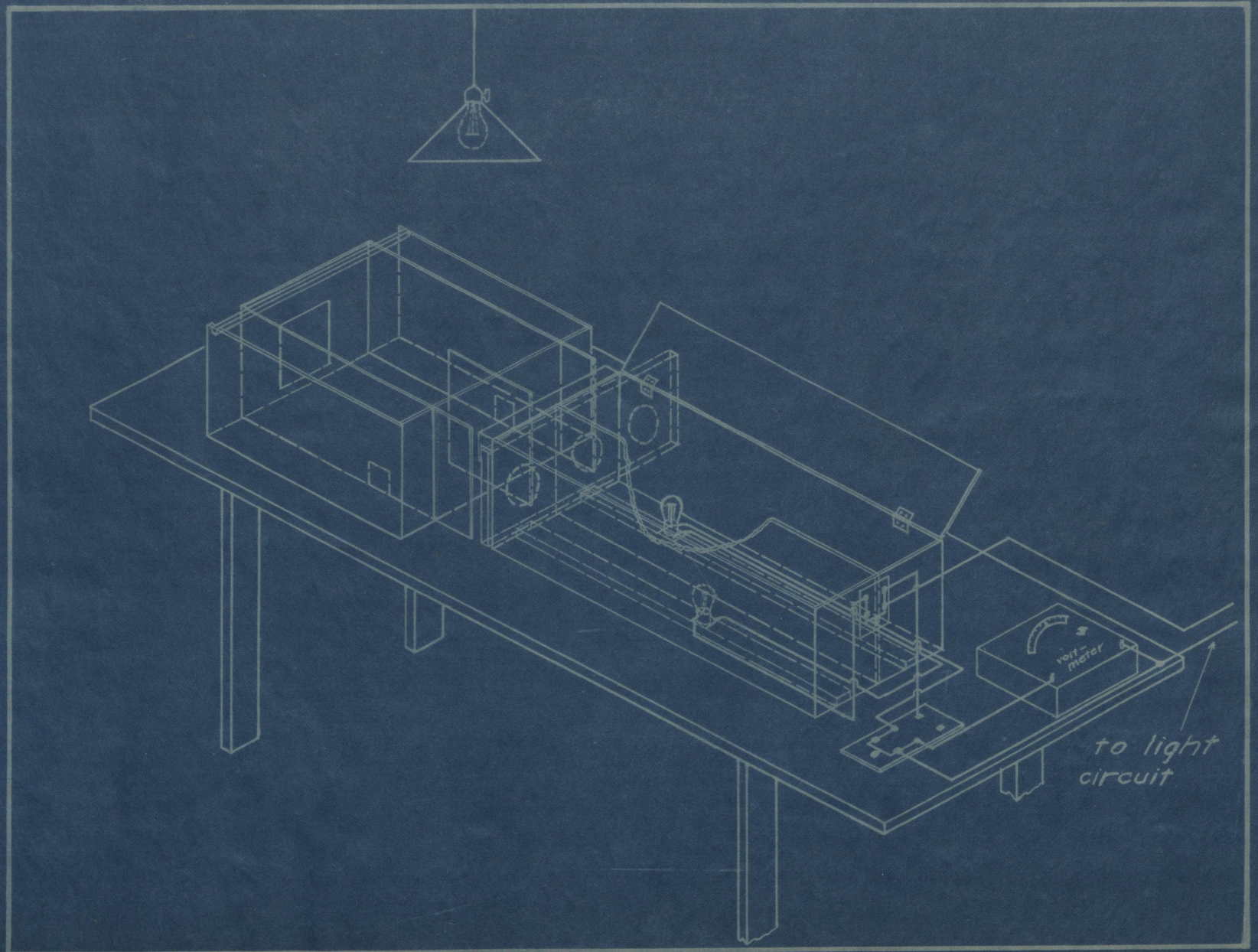

Fig. I

Fig.II.Alcohol Box

C,The cage in which the rats lived and through which the fumes passed;V,ventilator or exit for the fumes;A,box in which the fumes were generated.

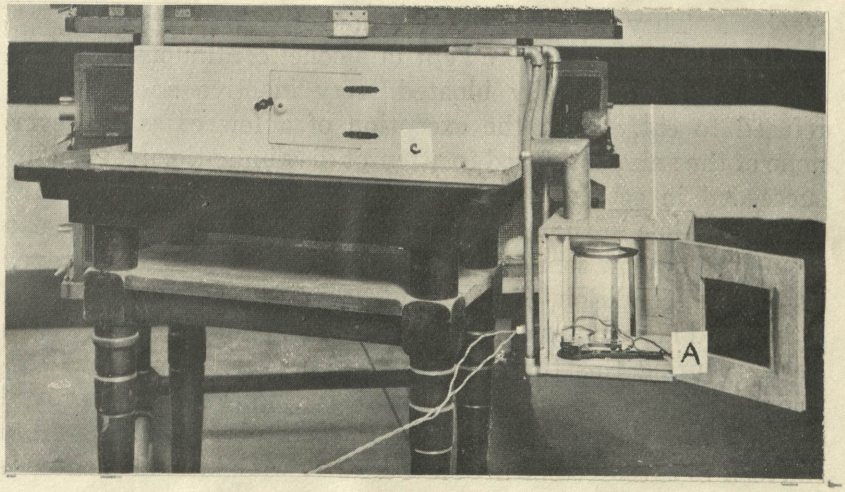

Fig.III.Alcohol Box

Box,A,contains:the thermometer,the electric light as a source of heat, and the alcohol upon a tripod.

The alcohol box (Figs. II and III) consisted, first, of a cage, C, 77 cm. by 21 cm. by 20 cm. in which the rats lived and through which the fumes passed and, second, a box, A, where the fumes were generated, and which contained a 40-watt mazda lamp as a source of heat and a tripod that held the pan of methyl alcohol. The thermostat that was placed in circuit with the lamp failed to regulate the heat as desired and was removed since it was found that the temperature was fairly constant without it. V is the ventilator, or exit, for the fumes from cage, C.

B. Method.

During the entire experiment fourteen rats were used. Six of these, I to VI were approximately full grown when training was begun, and were used only in the preliminary part of the work which was carried on during the latter half of the school year, 1923-24, and the following summer. All the others were slightly more than 60 days old at the time their training was started. Rats, No. VII to XI, were started in the discrimination box at the beginning of the fall semester, 1924, and Rats, No. XII, XIII, and XIV, one month later.

The rats were fed by the experimenter for a few days or a week in the living cages until they became accustomed to being handled. Then before actual experimentation was begun, they were fed for a few days on the table at the entrance to the problem box, the number of days being determined by the timidity of the animal. For the follow-

ing two or three days, each rat was given from four to six runs through the discrimination box, an equal number to right and to left, and without lights, for the purpose of setting up the association between going through the small opening at the end of the alley and returning to the table for food.

Throughout the entire experiment, in both the preliminary and the main tests, which will be described later, the following series of presentations were used, r and l indicating the side on which the positive stimulus was presented.

1st day - r l l r r r l r l l

2nd day - l r r l l r l l r r

3rd day - r l r l l l r r r l

4th day - l r r l l l r l r r

On the fifth day the first series was again used, etc. After each correct choice, the rat returned to the position at the entrance of the discrimination box and was given a bite of food which he ate while the experimenter moved the stimulus adapter or shifted the lights in the light box as the case might be. Punishment was omitted during the entire experiment. All right and wrong choices were recorded by the letters R and W. The daily number of correct responses for each rat with the distance of the lights from the stimulus plate or the distance between the two lights is given in the appendix.

Aside from the factors given above which are common to both the preliminary and the later tests, two separate and distinct methods

were followed. In the preliminary work, that is, until May 19, 1924, in which training was conducted to find the lower limen of brightness sensitivity, the procedure was as follows:

Both lights of the light box were placed 10 cm. from the stimulus plates, but since the middle opening of the stimulus adapter was covered with heavy black cardboard, only one light was shown at a time and the rat must choose between total darkness and a light of high intensity. The position of the positive stimulus was changed from right to left by moving the stimulus adapter. T raining was continued with the lights at the greatest possible intensity until the rat had reached the criterion of 80% correct choices for three successive days, when control tests were given to determine the presence of factors. other than brightness which might serve as cues for discrimination. In these tests, the end stop was left out of the discrimination box and inserted only after the rat had made the wrong choice but before he had reached the food, and the stimulus adapter was moved back and forth before each trial regardless of the order of the presentation of the light.

If the discriminating reactions did not break down upon the introduction of these controls, the next day the lights were moved farther from the stimulus plates, thus decreasing the intensity of the positive stimulus, and training was continued. The intensity of the lights were decreased in this manner a little each day until they were as far from the stimulus plates as the length of the light box would

permit, at which time the rats were still discriminating with an accuracy of approximately 90%. In order to decrease further the intensity of the light so that the lower limen for brightness sensitivity could be determined, a slight change in apparatus would have been necessary, that is, a resistance coil to reduce the current, and an ammeter were required. It seemed probable that the light which was above the apparatus, though very dim and heavily shaded, gave enough illumination that the rats could discriminate between the light and dark sides of the discrimination box by means of the black cardboard which covered one opening and the white paper which covered the other opening of the stimulus adapter. In other words there was a question involved here of discrimination between black and white which might have been possible without lights in the light box. Accordingly, a change was made in the method.

The purpose was now to determine the differential limen rather than the lower limen for brightness sensitivity. The cardboard was removed from the stimulus adapter, and the middle opening was covered with white paper exactly like the others. The stimulus adapter was kept stationary during the remainder of the experiment. One light, the positive stimulus, was placed as close to the stimulus adapter as possible, that is, 10 cm., and the other one was moved to the opposite end of the light box, 169 cm. from the stimulus plate. In order to reverse the order of these lights to fit the series of presentations given earlier in this paper, the lids to the light box were raised between trials

and the lights were moved by hand. With the facilities at hand it seemed impossible to arrange any better devise for shifting the lights, however, this method presented no serious difficulties.

Voltmeter readings were taken at the beginning of experimentation each day, and it was found that the average voltage was 113.9. Thus, knowing the strength of the current, the kind and size of the lamps used, and their distance from the stimulus plates, the intensity of the light could be determined and the experiment could be accurately repeated.

When the rat had mastered the discrimination at this setting, the lights were moved closer together by moving the more distant light closer to the stimulus plate, thus increasing the intensity of the negative stimulus and keeping the positive stimulus constant throughout. At least two days, or twenty trials were given at each setting before the distance between the two lights was again decreased. When the lights were so nearly together that the rat was unable to retain his standard of 80% correct choices, the lights were again moved far enough apart that discrimination was easily possible and this same process was repeated. The differential limen for brightness sensitivity was always determined at least twice in this manner before methyl alcohol was administered, and if for any reason it was believed that greater discrimination was possible, or if the rat's reactions were extremely erratic, the limen was found a third or even a fourth time. Then before placing the rat in the alcohol cage, the distance between

the lights was again increased and training was continued for a few more days, or until automaticity of response was obtained.

During these tests to determine the differential limen three controls were introduced:

1. Controls to break up position habits of running to the right or to the left. A series of presentations was given which forced the rat to go in the opposite direction in a majority of the ten trials. These tests were given from time to time while the two stimulus lights were at opposite ends of the light box and training was being conducted to set up the habit of going to the brighter light.

Position habits of alternating from right to left, alternating after success, etc., were eventually broken up if the regular series of presentations were continued.

2. Controls to render auditory, cutaneous, and olfactory factors constant. The end stop was left out of the discrimination box and inserted only after the rat had made the wrong choice but before he had reached the exit, and after each trial while the rat was eating, the lids of the light box were raised and the lights were slid back and forth. Thus the possibility of discrimination by means of the noise of shifting the lights, by differences in the odors or in the currents of air in the two sides of the discrimination box was decreased.

These controls were first given when there was the

greatest possible difference between the two lights of the light box and training had been continued until the rat had reached the criterion of 80% correct choices for three successive days. From this time, or shortly afterward, all the tests were of this type, that is, the end stop was left out and the noise of moving the lights was made at each trial.

3. Controls to eliminate discrimination by means of brightness in order to determine the influence of other factors. Both lights were placed 10 cm. from the stimulus plates making them equal in intensity while all other conditions were the same as in a normal presentation. Such tests were made with Rats II, III, V, and IX when the lights were very close together and the rat was reacting with an accuracy of 80% or 90%.

Data pertaining to brightness discrimination were obtained from all of the rats. It was found extremely difficult to give the fumes in sufficient intensity and duration to bring out the possible effects on vision and at the same time to keep the animal alive and active enough for experimentation. Of the nine rats that were exposed to the fumes, two died before the effects upon vision could be determined, but the others were kept until the end of experimentation and results as complete as time would allow were obtained for each one.

When the first rat was placed in the alcohol fumes, a 75-watt lamp was used in the alcohol box as a source of heat for the evaporation of the alcohol. However, at the end of the first day the

rat was so weak and inactive that it was deemed necessary to provide some method for reducing the rate of evaporation and the concentration of the fumes. Therefore, the 75-watt lamp was exchanged for a 40-watt lamp. This rat was left in the alcohol cage day and night with the light on all the time in order to retain a constant temperature, but the alcohol was removed at night. At the end of three days of such exposure, the rat was in a state of coma,and three days later he was dead. Since it was found that with such long periods of exposure, it would be impossible to keep the rat alive to make further tests upon brightness discrimination, the plan used with the majority of the rats was as follows:

1st exposure - The rat was placed in the fumes three hours per day for six days. During the remainder of the day the rat was kept in the living cage with the other rats. Each day, whether or not the rat had been in the fumes, the usual ten trials in the discrimination box were given. At the end of the six days exposure to the fumes, the tests were continued until the differential limen had been found.

2nd exposure - Exactly like the first exposure.

3rd exposure - The rat was put in the alcohol cage three hours per day for an indefinite number of days during which time tests were given each day until the limen was found again.

4th exposure - A continuation of the third exposure.

If at the end of the third exposure to the fumes no change was found

in the differential limen, the length of time in the fumes was increased to six hours per day for one or two days, when the rat usually presented a state of coma indicating that further exposure to the fumes would result in death. The rat was not returned to the alcohol cage but was kept in the living cage and as soon as his physical condition would permit, tests in the discrimination box were made each day until the limen was found.

Each day that a rat was in the alcohol fumes readings were taken from the thermometer in the alcohol box at the time the rat was put in the alcohol cage before the light in the alcohol box was turned on, and again when the rat was removed from the fumes while the light was still on. These readings gave a record of the temperature at which the alcohol evaporated. Record was also kept of the amount of alcohol which evaporated each day while the rat was in the alcohol fumes. It would be very desirable to know the amount of alcohol that was taken into the animal's system in order that the data obtained could be related to the poison experiments. However, without this information the results are not valueless because the experiment can be repeated at any time in exactly the same manner.

The methyl alcohol which was used was obtained from the chemistry department of the University and was 95% pure methyl alcohol and 5% water. Fresh alcohol was placed in the alcohol box each day. That which remained in the pan at the end of the day's exposure to the fumes was always discarded.

C. Results.

1. Brightness discrimination.

Brightness discrimination was possible in all of the rats used in this investigation. However, as in all learning problems, individual differences are found in the amount of training necessary to set up the discrimination habit. In section VII of this thesis are given all the data upon brightness discrimination, that is, the daily record of the number of correct choices made by each rat, both in the preliminary and in the later tests.

Table I summarizes the results of the preliminary tests in which Rats I to VI were trained to discriminate between light at the greatest possible intensity and total darkness. Column A shows the number of trials required for each rat to reach the standard of 80% correct choices for three successive days. In Column B are given the number of additional trials necessary to reach this standard again after the introduction of controls to rule out possibility of discrimination on the basis of sound, etc.

Table I

Showing number of trials necessary to set up discrimination between light and darkness.

Rat	Trials	
	A	B
I	250	20
II	300	0
III	240	90
IV	210	80
V	300	0
VI	160	70
Av.	243.6	43.6

The number of trials necessary for learning the discrimination ranged from 160 to 300 with an average of 243.6. Two rats, II and V required 300 trials for learning, however, upon the introduction of controls these two rats retained their high percentage of correct responses, whereas the other four which had had fewer trials failed to discriminate and additional training was necessary. This seems to indicate that factors other than intensity of the light were serving as secondary cues for discrimination. At least, the control tests produced a disturbing element, the exact nature of which could not be ascertained since these tests were rather complex, involving more than one change in the experimental conditions. It is probable, however, that the sound of shifting the stimulus adapter is the more important

of these cues.

Although a continuation of these tests to determine the lower threshold of brightness sensitivity called for a change in the apparatus, which, for reasons given in the description of the method, did not seem worth while, training was continued for some time after the rats had learned to discriminate. The lights were moved away from the stimulus plate a short distance each day, thus decreasing the intensity of the positive stimulus while the other side of the discrimination box remained dark. When the lights were at the opposite end of the light box, decreasing the intensity of the stimulus as much as possible by this method, the rats were still discriminating with an average of approximately 90% correct choices. At this point, a slight change was made in the method, and training was begun again.

Now the rat must discriminate between two lights of different intensities. This difference in intensity was represented by the distance between the two 25-watt lamps used as stimuli which distance, when training was begun, amounted to the length of the light box, or approximately 160 cm.

In Table II are given the number of trials required to set up the discrimination with untrained rats. Column A shows the number of trials necessary to reach the criterion of 80% correct choices for three successive days, and Column B, the number of trials to regain this standard after the control test was given, which in this case consisted of leaving the end stop out of the discrimination box and of

shifting the lights after each trial.

Table II

Showing the number of trials necessary to set up discrimination between two lights differing widely in intensity.

Rat	Trials	
	A	B
VII	430	0
VIII	290	0
IX	270	0
X	280	0
XI	330	60
XII	380	40
XIII	290	10
XIV	340	0
Av.	326.25	13.75

That there is a high degree of transfer from discrimination between light and darkness to discrimination between lights of widely different intensity is indicated by a comparison of the number of trials necessary to set up the discrimination between lights of different intensity for those animals that had not been trained before, with the number of trials necessary for those that had been previously trained to discriminate between light and darkness. An average of 326.25 trials were required for the untrained rats and an average of 41.66 trials for those previously trained.

That the rats were discriminating on the basis of any cues other than brightness is highly improbable. During the latter part of the tests to find the differential limen of brightness sensitivity, care was taken to make all factors constant except the intensity of the negative stimulus, which was increased day by day moving the more distant light closer to the stimulus plate. The food which was given in small bits after each trial was always placed upon the table in the same place, that is, at the entrance to the discrimination box, the end stop was left out of the discrimination box all the time after the discrimination was learned, and was inserted only after a wrong choice was made so that the animal could not obtain food except by going to the correct side. The rat was always picked up by the experimenter and put through the entrance to the discrimination box in as nearly the same manner as possible. After each trial, while the rat was eating, the experimenter went to the right side of the light box, raised the lids and shifted the lights back and forth in order to produce approximately the same sound each time.

Hunter (2a) has shown the difficulty with which a complex position habit is formed. Since the series of presentations used in this investigation were rather complex, there is no evidence from previous investigations or from close inspection of results found in this study to indicate that the rats were discriminating by means of kinaesthesis. The only position habits which could be detected were simple alternation from right to left or a habit of going to the right

or to the left. When the lights were so close together that discrimination was impossible, the rats resorted to one or the other of these forms of reaction.

During experimentation, when the lights were very close together and the rats were reacting with an accuracy of 80% or 90%, a test of ten trials was given with the lights equal in intensity while all other conditions were the same as in previous tests. Such tests were made with Rats II, III, V and IX, and in no case did a rat make more than 6 correct choices.

A failure to retain a high percentage of correct responses over a long period of time does not indicate a final inability to discriminate, but is merely characteristic of rat behavior. Aside from this irregularity of response, brightness discrimination is improved by training, and until the lights are almost equal in intensity there is a possibility that additional training will decrease the differential limen.

The differential limen was determined at least twice for each rat before exposure to the alcohol fumes. If there was any indication that the rats could discriminate between two lights more nearly equal in intensity than the determined limen seemed to indicate, training was continued and another determination of the limen was made before alcohol was administered. When the rat's reactions were quite irregular, that is, if the rat made 8 or 9 correct responses one day and only 6 the next when no changes had been made in the position of the

lights, continued training/most always overcame the irregularity. For illustration we may take a part of the results for Rat IX. When the lights were 75 cm. apart, 6 correct responses were made one day, 9 the next and only 7 the next. Training was continued with the lights in the same position and for the next three days, 9, 10, and 8 correct responses were made. The negative stimulus was moved first 70 cm., then 65, 60, and 55 cm. from the positive stimulus and the rat made 10, 10, 9, and 9 correct choices. Then with the lights 50 cm. apart we find a repetition of the behavior when the lights were 70 cm. apart. The lights were kept at this distance for six days during which time the rat made 7, 10, 6, 9, 8, and 10 correct responses. As the lights were moved closer together by steps of 5 cm. per day on no day were there less than 90% correct responses until the lights were 20 cm. apart when the rat made only 7 correct choices one day and 6 the next. Although it is possible that additional training might have decreased the number of errors and finer discrimination could have been made, it was assumed that the limen was reached, the lights were moved farther apart and a new determination was made to verify this assumption. Table III gives the two lowest determinations of the limen for each rat before exposure to methyl alcohol fumes. In each case the differential limen is expressed by the distance in centimeters between the two lights as they were placed when the rat last discriminated with an accuracy of at least 80%.

Table III

Differential limen before exposure to methyl alcohol fumes.

Rat	First Determination	Second Determination	Av.
II	20 cm.	Died	20 cm.
III	15 cm.	Died	15 cm.
IV	25 cm.	45 cm.	35 cm.
V	10 cm.	10 cm.	10 cm.
VII	20 cm.	10 cm.	15 cm.
VIII	40 cm.	40 cm.	40 cm.
IX	15 cm.	15 cm.	15 cm.
X	25 cm.	35 cm.	30 cm.
XI	15 cm.	10 cm.	12.5 cm.
XII	35 cm.	20 cm.	27.5 cm.
XIII	30 cm.	25 cm.	27.5 cm.
XIV	25 cm.	25 cm.	25 cm.
Av.	22.9 cm.	23.5 cm.	

2. The effects of methyl alcohol fumes.

a. On the lower limen of brightness sensitivity.

Since the tests upon discrimination between light and darkness were not continued until the lower threshold of brightness sensitivity was determined, there are no data upon the effects of alcohol fumes on the lower limen.

b. On the differential limen.

The temperature of the alcohol box was read each day before the light was turned on and again when the rats were removed from the alcohol cage three or six hours later, as the case might be. The average temperature at the time the light was turned on each day was 25.27° C. and ranged from 21° to 29° C. The average temperature at which the alcohol evaporated, taken at the end of the three hour period of exposure to the fumes, was 33.99° C. and ranged from 30° to 39° C. At this temperature the alcohol was found to evaporate at the rate of 31 grams per hour. During the three hour period of evaporation, there was no day in which less than 83.5 grams of alcohol were evaporated, and the largest amount evaporated in this same length of time was 106.9 grams.

The effect of the alcohol fumes upon the differential limen is shown by Table IV. In all cases after exposure to the fumes there was very little if any change in the differential limen of brightness sensitivity. The decrease in the limen which occurred in a few cases is without doubt due to the continued training. The original determination was not the true limen. At no time after an exposure to the fumes was the limen increased more than 5 centimeters and such a small difference in either direction could well be accounted for by chance factors, the erratic nature of the rat, or the amount of training. There is no justification for assuming that the alcohol fumes have in any way had an effect upon brightness discrimination.

Table IV

Showing differential limen before and after exposure to methyl alcohol fumes.

Rat	Before exposure to fumes — Limen 1st	Before exposure to fumes — Limen 2nd	First exposure H	First exposure L	D	Second exposure H	Second exposure L	D	Third exposure H	Third exposure L	D	Fourth exposure H	Fourth exposure L
VIII	40 cm.	40 cm.	27	20 cm.	114	18	10 cm.	12	27	15 cm.	0	12	15 cm.
IX	15 cm.	15 cm.	18	20 cm.	15	18	15 cm.	13	42	5 cm.	0	15	15 cm.
X	25 cm.	35 cm.	18	15 cm.	28	18	15 cm.	12	33	20 cm.			
XI	15 cm.	10 cm.	18	15 cm.	19	18	15 cm.	10	63	15 cm.	0	12	15 cm.
XII	35 cm.	20 cm.	18	15 cm.									
XIII	30 cm.	25 cm.	18	20 cm.									
XIV	25 cm.	25 cm.	18	25 cm.	17	18	25 cm.						

L - Limen given in distance between the two lights.
H - Number of hours in the fumes.
D - Number of days between the two exposures

Due to lack of time, complete results were not obtained for Rats X, XII, XIII, and XIV.

Since the rats were not tested at the limen immediately after exposure to the fumes, but with the lights far enough apart that discrimination was easily possible, and then gradually approached the limen as in the original training series, we may study the results of those first tests after exposure to observe any immediate effects which the fumes might have had on brightness discrimination. Table V gives the number of correct responses out of each ten trials for the first three days after exposure to the fumes.

Table V

Showing number of correct responses in first three tests immediately following exposure to methyl alcohol fumes.

	First exposure			Second exposure			Third exposure			Fourth exposure		
	Correct responses			Correct responses			Correct responses			Correct responses		
Rat	1st day	2nd day	3rd day	1st day	2nd day	3rd day	1st day	2nd day	3rd day	1st day	2nd day	3rd day
IV	5*											
V	9	10	9									
VIII	5*	3*	5*	10	10	9	10	9	10	0*	10	8
IX	7	8	7	10	8	10	9	10	9	0*	6*	6*
X	10	9	9	9	9	7	10	10	8			
XI	10	10	9	10	10	10	10	7	8	9	7	10
XII	8	9	10									
XIII	9	8	8	9	9	8						
XIV	9	9	8	5	8	7	8	9	7			

*In these tests the rat was sick apparently from effects of the fumes.

Rats IV and V died before further tests could be made, and experimentation was discontinued before Rats X, XII, XIII and XIV were given all 4 exposures to the fumes.

In a few cases, less than 80% of the choices were correct. However, in the majority of those cases, the rat was sick, very weak and inactive, did not eat well, and made all reactions slowly. Very often it was impossible to complete all ten trials of the test. These low scores might have meant a visual defect, but it is more probable that they are due to the physiological condition of the animal, inactivity, lack of hunger incentive, and to the change in environment.

The record of Rat V, which is not given in Table IV since the limen was not determined, is nevertheless interesting and well worth mentioning. This rat was placed in the alcohol cage three hours each day for 23 days, during which time training was continued. On the 23rd day the lights of the light box were 30 centimeters apart. The rat moved rather slowly, but made 9 correct choices. That afternoon the rat was returned to the alcohol fumes for the usual three hour period, at the end of which time he was weak and unsteady and died before morning. The limen for this rat was 10 cm., and although training had not been continued long enough to find whether or not this limen had changed, at the time the rat died there was no indication of inability to discriminate, and even if there was a change in the limen it could not be a very significant change.

At no time in these tests was there any significant evidence

to indicate visual defect unless it might be in the results for Rat VIII. This rat was in the fumes 27 hours, six hours per day for four days and three hours on the fifth day. When removed from the fumes the last day, the rat was nearly dead. For several days following, it was very inactive and discrimination was poor. A great deal of training was necessary to reach a discrimination equal to discrimination before exposure to the fumes. However, a close study of the responses does not indicate inability to discriminate so much as it shows an erratic nature. Part of the time the rat discriminated with a high percentage of accuracy. One day 9 or 10 correct responses were made and only 5 or 6 the next day. Eventually, with continued training, a limen was reached which was lower than that found before the rat was in the fumes.

C. On bodily conditions.

The only results in this investigation upon the effects of methyl alcohol fumes upon the physical condition of the rat were obtained by observation. There are no objective, quantitative data, but the general behavior of the animals is indicative of a harmful physiological effect.

Often when the rats were removed from the cage, they exhibited a state of intoxication. Many times with continued exposure and in every case after a six hour period in the fumes, the animal was in a state of coma near the point of death. As soon as the animal recovered sufficiently to move through the discrimination box as usual,

the responses were approximately 90% correct. The bloated condition observed by Bills and Maupin did not occur in any of these rats.

When removed from the fumes all rats did not behave in the same way which indicates the factor of individual differences in the susceptibility to the poison. Some showed signs of intoxication while others seemed entirely normal.

After an exposure of three hours there was often slight evidence of intoxication. The rat was unsteady, seemed drowsy and weak, and staggered when he walked. A three hour period of exposure might cause unsteady movements and drowsiness one day, and on the next day after a similar exposure, there would be no indications of intoxication. Intoxication was as likely to result on one day as another, showing no evidence of either tolerance or accumulation of the poison.

With such short periods of exposure, no ill effects remained the following day until exposure had been continued for several days. In most cases the exposure was not continued over a long enough period to cause any serious physical disturbances, and consequently the majority of the rats remained in good condition, ate well, were active and their reactions in the problem box were highly automatic throughout the entire experiment. However, at the end of five days in the fumes, Rat XIV was quite weak and inactive and unable to complete the usual ten trials in the discrimination box.

Rat V was exposed to the fumes three hours per day for 23 days. For the first few days the rat seemed rather unsteady and gave

indications of slight intoxication at the time he was removed from the fumes. On later days there were only slight indications of intoxication. Each morning after exposure to the fumes the rat apparently had recovered. He ate well and was as active as usual. However, after 20 days of exposure the reactions became slower. On the 23rd day the rat was returned to the alcohol cage and when removed at the end of three hours, he was weak and unsteady and died during the night.

With longer periods of exposure, six hours or more each day, severe intoxication was noticed in every case, and usually the rat reacted rather slowly in the discrimination box the following day. If such an exposure was repeated one or two days longer, there were very decided ill effects upon the behavior the following day. The rat seemed weak and moved slowly through the problem box, or went to one corner and sat motionless. Usually, however, the rat was in a state of coma, was limp and motionless, and death resulted if the rat was exposed to the fumes again without allowing time for recovery.

IV. THEORETICAL DISCUSSION

The negative results concerning the effect of methyl alcohol upon brightness discrimination in the white rat may be due to:

1. The method of administering the alcohol.

a. There are fewer recorded cases of death and blindness from inhalation of methyl alcohol fumes than from ingestion of the alcohol.

No comparison of the methods of ingestion and inhalation can be made here, but there have been cases recorded of blindness from inhalation of methyl alcohol indicating that in this form the alcohol is toxic and will cause blindness.

b. Gruening (38) has observed that blindness from inhalation of methyl alcohol is not permanent, but that vision is eventually recovered.

There is no significant evidence in this investigation nor in that of Bills and Maupin to indicate either temporary or permanent visual defect. There is a slight indication that rat VIII was unable to discriminate after exposure to the fumes and later was able to do so, but it is more probable that the long period in the fumes resulted in erratic activity.

2. The acquisition of immunity to the poison.

A theory has been posited that through long exposure to methyl alcohol, immunity or adaptation takes place. At the end of ex-

perimentation, when the animals had had three previous exposures to the alcohol fumes without any effects upon brightness discrimination the period of exposure was increased to 6 hours a day. At the end of such an exposure to the fumes the rats were usually very inactive, almost unable to move. With two days of such exposure, experimentation was impossible. It is still possible that with a longer period of investigation, immunity might have been acquired, but there is no indication that the rats used in this study became adapted to the fumes.

3. The masking of effects by training.

Training was continued so long before the rats were placed in the alcohol cage that in most cases additional training could not have increased the discrimination to any great extent. Therefore, it is highly improbable that the effect of the fumes was counterbalanced by training.

4. Less highly differentiated structure of the eye and optic nerve in the rat.

The more logical explanation seems to be that the eye and optic nerve in the rat are less highly differentiated than in higher organisms and, therefore, less susceptible to the toxic action of the methyl alcohol.

Authorities agree that the vapor of methyl alcohol is more poisonous when the ventilation is poor and the fumes are mixed

with rebreathed air. Such were the conditions used in this investigation, and in spite of the unfavorable conditions there were no effects upon brightness discrimination.

V. SUMMARY AND CONCLUSIONS

1. The white rat is able to discriminate between lights very nearly equal in intensity. In no case was the differential limen greater than 25 cm. and more often it was 15 cm. For one rat one determination of the differential limen was found to be only 5 cm.

2. Brightness discrimination is improved by training.

3. Under the conditions of the present investigation, methyl alcohol fumes did not affect the brightness discrimination of the white rat. On the contrary training decreased the limen in spite of the exposure to the fumes.

4. Methyl alcohol fumes are poisonous to white rats to the extent that with very long periods of exposure, or intense heat, or long continued short periods of exposure will cause sickness and death.

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VII. APPENDIX

In order to continue or repeat this investigation the complete results would be a valuable aid. I have, therefore, listed the distance between the two lights and the daily number of correct responses for each rat throughout the entire experiment.

Results of Preliminary Tests

Rat I.

D.	N.
10 cm.	3 - 3 - 4 - 4 - 5 - 4 - 7 - 4 - 7 - 5 - 4 - 5 - 7 - 5 - 6 - 3 - 7 - 6 - 6 - 8 - 6 - 6 - 6 - 6 - 6 - 3 - 9 -10 - 9 -7**- 8 - 9 - 9 - 10**
40 cm.	9 - 9
60 cm.	9 - 6 - 9
80 cm.	6 - 9 - 9
90 cm.	7 - 9 - 6 - 8 - 7 - 9 - 9 - 9 - 10
95 cm.	10
100 cm.	9 - 9
105 cm.	10
110 cm.	9 - 10
120 cm.	10
130 cm.	10
150 cm.	9 - 10
165 cm.	8 - 9 - 10 - 8

D - Distance of positive stimulus from stimulus plate
N - Number of correct responses out of 10 trials
** - Control test. End stop left out and stimulus adapter shifted after each trial

Rat II

D	N
10 cm.	3 - 6 - 0 - 2 - 6 - 5 - 4 - 5 - 7 - 6 - 6 - 5 - 5 - 5 - 7 - 8 - 6 - 6 - 6 - 3 - 6 - 7 - 9 - 6 - 7 - 8 - 8 - 6 - 7 - 7 - 9 -10 - 8 -10 - 9**
40 cm.	10
60 cm.	9 - 10
80 cm.	9 - 8 - 6 - 9 - 7 - 8 - 7 - 8 - 10
90 cm.	8 - 9
100 cm.	9 - 10
120 cm.	10
140 cm.	9 - 6 - 8

Rat III

D	N
10 cm.	3 - 4 - 5 - 3 - 2 - 2 - 2 - 5 - 5 - 4 - 7 - 5 - 8 - 7 - 5 - 6 - 7 - 8 - 7 - 8 - 6 - 9 - 7 - 7 - 7 - 8 - 8 - 9 - 2**-8 - 7 - 7 -10 - 9 - 9 - 7**-8 - 9 -10 - 9**
40 cm.	10
60 cm.	9 - 10
80 cm.	9 - 10
90 cm.	9 - 9
100 cm.	8 - 7 - 8
120 cm.	10
140 cm.	9 - 9
150 cm.	9 - 9

Rat IV

D	N
10 cm.	3 - 6 - 7 - 3 - 6 - 6 - 5 - 5 - 5 - 7 - 7 - 6 - 6 - 7 - 4 - 4 - 6 - 8 - 8 - 5 - 6 - 8 - 8 - 8 - 6**-7 - 7 - 8 - 8 - 8 - 7 - 7 -10 - 9 -10 - 8**
40 cm.	9 - 10
60 cm.	10
80 cm.	10
90 cm.	10 - 9 - 10

Rat V

D	N
10 cm.	4 - 7 - 8 - 6 - 6 - 7 - 6 - 6 - 3 - 5 - 6 - 7 - 7 - 8 - 7 - 5 - 5 - 8 - 9 - 5 - 8 - 7 - 5 - 7 - 7 - 7 - 7 - 7 -10 - 6 - 10-9 - 8 - 10**
40 cm.	8 - 8 - 10
60 cm.	8 - 8 - 8 - 9 - 10 - 10

Rat VI

D	N
10 cm.	2 - 4 - 7 - 3 - 5 - 4 - 5 - 5 - 5 - 7 - 7 - 7 - 5 - 7 - 5 - 7 - 9 - 8 - 9 - 7** 6 - 9 - 7 - 8 - 8 - 7 -10 -10 -10 - 9**
40 cm.	9 - 10
60 cm.	9 - 10
80 cm.	7 - 9 - 10
90 cm.	7 - 8 - 9
100 cm.	8 - 10 - 10

Results of Later Tests

Rat I

D	N
159 cm.	7 - 10 - 7 - 7 - 7 - 7 - 10 - 7 - 8 - 8 - 9 - 10 - 9**
130 cm.	10
110 cm.	9 - 6 - 8 - 9
90 cm.	7 - 8 - 8 - 5 - 9 - 7 - 7 - 8 - 6 - 9 - 7 - 10 - 7 - 8 - 10- 10
80 cm.	9 - 8
70 cm.	8 - 7 - 8 - 8 - 9
60 cm.	6 - 8 - 9 - 10
50 cm.	9 - 6 - 8 - 3
55 cm.	8 - 8 - 10

Vacation of 8 weeks followed, after which, training with this rat was not resumed.

D - Distance between the two stimulus lights.
N - Number of correct responses out of each 10 trials.
** - Control test in which end stop was left out and the lights were shifted after each trial.
o - distance between the two lights - control test eliminating discrimination on basis of brightness in order to see if discrimination was possible by all other cues.
* - Control test forcing the rat to go to one side in a majority of the trials in order to break up position habits to the right or to the left.
() - Days on which alcohol was administered. Exposure to the fumes came in the afternoon, following the training for that day.

Rat II

D	N
159 cm.	8 - 9 - 2 - 8 - 9 - 10 - 8 - 7 - 9 - 8 - 10 - 8 - 9**
130 cm.	9
110 cm.	9 - 8 - 8 - 9
90 cm.	9 - 9
80 cm.	9 - 8
70 cm.	9 - 9
60 cm.	10
50 cm.	8 - 9 - 9
40 cm.	10
30 cm.	4 - 6 - 8 - 10 - 8
25 cm.	7 - 8 -10 - 5 - 9 - 9 - 9
20 cm.	8 - 10 - 8
0	4
20 cm.	5
30 cm.	10 - 7 - 9 - 7 - 7
35 cm.	7 - 7
40 cm.	7

Rat II died during summer vacation.

Rat III

D	N
159 cm.	8 - 9 - 8 - 10 - 8 - 7 - 10 - 10 - 9 - 9 - 10 - 8 - 10- 8 - 10**
130 cm.	9
110 cm.	9 - 7 - 10
90 cm.	10 - 9
80 cm.	6 - 9 - 8
70 cm.	9 - 9
60 cm.	10
50 cm.	9 - 10
40 cm.	7 - 9 - 9 - 10
30 cm.	7 - 9 - 8 - 9
25 cm.	8 - 10
20 cm.	9 - 8
15 cm.	8 - 9
10 cm.	7 - 6
0	6
10 cm.	6
30 cm.	7 - 10 - 10 - 10 - 7**
35 cm.	8 - 7

Rat III died during summer vacation.

Rat IV

D	N
159 cm.	1 - 8 - 8 - 10 - 9 - 10 - 8 - 9 - 8 - 9 - 9**
130 cm.	9
110 cm.	9 - 10
90 cm.	8 - 9
80 cm.	10 - 9
70 cm.	9 - 9
60 cm.	10 - 10
50 cm.	9 - 7 - 9 - 5 - 8 - 7 - 8 - 8 - 6 - 8 - 9 - 9
40 cm.	9 - 10
30 cm.	8 - 8
25 cm.	7 - 6 - 6
30 cm.	10 - 7 - 10 - 10 - 8 - 9**
25 cm.	10 - 8 - 9**

Vacation of 8 weeks

D	N
60 cm.	5 - 8 - 8 - 10 - 6** - 8 - 8 - 8 - 8 - 8**
50 cm.	6 - 7 - 6
55 cm.	7 - 7
60 cm.	8 - 9 - 8 - 9**
55 cm.	7 - 8 - 7 - 8 - 9 - 10 - 8

Each of the following tests was a control test in that end stop was left out and lights were shifted after each trial.

D	N
50 cm.	9
45 cm.	8
40 cm.	7 - 9 - 6
45 cm.	6 - 9 - 9 - 8 - 8
40 cm.	8 - 7
45 cm.	9 (in fumes 3 days without training)
	2 days later
45 cm.	5
	Died from effects of alcohol.

Rat V

D	N
159 cm.	7 - 8 - 8 - 10 - 8 - 10 - 10 - 10 - 10 - 8 - 9**
130 cm.	10
110 cm.	10 - 5 - 9
90 cm.	7 - 9 - 8
80 cm.	7 - 7 - 9 - 9
70 cm.	10
60 cm.	7 - 9 - 9
50 cm.	7 - 7 - 7 - 8 - 6 - 10 - 9 - 8
40 cm.	7 - 8 - 7 - 9 - 8 - 8
30 cm.	9 - 8 - 9
25 cm.	7 - 8 - 5
30 cm.	9 - 8 - 9 - 9** - 9

Vacation of 8 weeks

D	N
70 cm.	5 - 6 - 7 - 10 - 7 - 10 - 6 - 9 - 9 - 9 - 9**
60 cm.	6 - 8 - 8 - 8 - 9*
55 cm.	8 - 9**
50 cm.	9 - 9**
45 cm.	8 - 4**
50 cm.	9 - 9**

Each of the following tests was a control test.
End stop left out and lights shifted at each trial.

45 cm.	8 - 7 - 9 - 7 - 10 - 7 - 7
50 cm.	8 - 8 - 10

Rat V (continued)

D	N	D	N
45 cm.	9 - 10 - 9	30 cm.	9
40 cm.	9 - 9 - 9	25 cm.	10 - 8 - 9
35 cm.	8 - 7	20 cm.	9 - 8 - 9
40 cm.	9 - 10 - 9	0 cm.	2
35 cm.	10 - 8 - 8 - 9	55 cm.	8 - 10
30 cm.	9 - 7	50 cm.	8
35 cm.	9	45 cm.	9
30 cm.	6	40 cm.	10
35 cm.	9	35 cm.	8
30 cm.	9 - 8 - 9	30 cm.	8
25 cm.	9 - 9 - 7	25 cm.	7 - 7 - 9 - 9 - 8
30 cm.	8	20 cm.	8
25 cm.	7	15 cm.	7 - 7 - 7
50 cm.	9	25 cm.	8
45 cm.	9	20 cm.	9
40 cm.	9	15 cm.	9
35 cm.	7	10 cm.	8
55 cm.	8	5 cm.	6 - 5
50 cm.	8	25 cm.	8
45 cm.	10	20 cm.	9
40 cm.	9	15 cm.	10
35 cm.	10	10 cm.	8

Rat V (continued)

D	N
5 cm.	5
10 cm.	6
20 cm.	5
30 cm.	6
50 cm.	(9)
70 cm.	(9 - 10)
65 cm.	(10 - 9)
60 cm.	(9 - 9)
55 cm.	(10 - 9)
50 cm.	(10 - 7 - 10 - 9 - 10)
45 cm.	(10 - 10)
40 cm.	(9 - 9)
35 cm.	(9 - 9)
30 cm.	(6 - 8 - 9)

Died from the effects of fumes.

Rat VI

D	N
159 cm.	8 - 6 - 7 - 9 - 9 - 6 - 6 - 8 - 8 - 9 - 10**
130 cm.	8 - 7 - 10
110 cm.	6 - 8 - 8
90 cm.	9 - 6 - 8* - 8 - 10
80 cm.	10
70 cm.	10
60 cm.	9 - 10
50 cm.	10
40 cm.	9 - 9
30 cm.	6 - 8 - 6 - 5 - 6* - 6 - 7* - 8 - 4 - 6 - 5
40 cm.	10 - 10 - 10
35 cm.	7-9-7 - 10 - 9 - 9 - 10**

Vacation of 8 weeks

D	N
70 cm.	5 - 8 - 8 - 5 - 5 - 9 - 7 - 8 - 6 - 8

Rat died of middle ear disease.

Rat VII

D	N
159 cm.	4 - 1 - 3 - 4 - 2 - 4 - 2 - 5 - 5 - 3 - 3 - 5 - 1 - 5 - 7 - 4 - 3 - 7 - 6 - 4 - 5 - 6 - 4 - 5 - 5 - 5 - 4*- 4*- 4*- 4*- 4*- 5*- 3*- 3*- 4 - 4*-6* - 8 - 6 - 7 - 7 - 10- 7 - 9 - 10- 8 - 9**

All tests following were given as control tests, that is, the end stop was left out and the lights were shifted after each trial.

130 cm.	9
110 cm.	8 - 5 - 7 - 8 - 9 - 9
90 cm.	8 - 8 - 8 - 10
80 cm.	7
110 cm.	10
90 cm.	9
80 cm.	7
110 cm.	9
90 cm.	7
130 cm.	6 - 8 - 8 - 9 - 7 - 9 - 8 - 8 - 5 - 9 - 8 - 9
120 cm.	8
110 cm.	8
100 cm.	8
90 cm.	10
85 cm.	7 - 9 - 6 - 7 - 5
110 cm.	9
100 cm.	6 - 7 - 6
130 cm.	9
120 cm.	9

Rat VII (continued

D	N
110 cm.	6 - 7 - 7 - 9 - 7 - 5
130 cm.	4 - 8 - 7 - 7 - 8 - 7 - 7 - 7
159 cm.	8 - 8 - 9
130 cm.	8 - 9
120 cm.	10 - 9
110 cm.	9 - 10
100 cm.	6 - 10 - 9 - 9
90 cm.	7 - 8 - 8 - 10
85 cm.	8 - 9
80 cm.	10 - 8
75 cm.	8 - 10
70 cm.	9 - 9
65 cm.	9 - 9
60 cm.	10 - 10
55 cm.	8 - 10
50 cm.	10 - 10
45 cm.	10 - 8
40 cm.	7 - 10 - 10 - 9
35 cm.	7 - 10 - 10 - 9
30 cm.	9 - 9
25 cm.	10 - 10
20 cm.	9 - 7 - 9 - 9 - 8
15 cm.	6 - 6

D	N
60 cm.	7 - 10 - 10 - 9
55 cm.	10 - 9
50 cm.	8 - 10
45 cm.	10 - 8
40 cm.	10 - 10
35 cm.	10 - 10
30 cm.	10 - 9
25 cm.	10 - 10
20 cm.	10 - 8
15 cm.	8 - 9
10 cm.	6 - 8 - 5 - 8 - 9 - 8
5 cm.	6 - 5

Died of intestinal disease.

Rat VIII

D	N
159 cm.	3 - 4 - 4 - 3 - 4 - 3 - 4 - 6 - 2 - 2 - 5 - 5 - 2* - 1*- 4*- 8 - 1 - 3 - 6 - 6 - 7 - 6 - 6 - 9 - 6 - 5 - 10- 7 - 7 - 8 - 9 - 10- 10**

All following tests are given as control tests, that is, the end stop was left out and the lights were shifted after each trial.

130 cm.	10 - 7 - 8 - 10 - 10
110 cm.	10
90 cm.	8 - 10
80 cm.	9
70 cm.	9
60 cm.	4
70 cm.	9
60 cm.	10 - 7 - 8 - 9 - 9
55 cm.	9
50 cm.	8 - 6
55 cm.	9
50 cm.	9 - 9 - 9
45 cm.	10
40 cm.	10
35 cm.	7
50 cm.	8
45 cm.	9
40 cm.	7
60 cm.	8

Rat VIII (continued)

D	N
55 cm.	8
50 cm.	8
45 cm.	8
40 cm.	9
35 cm.	7

Rat was placed in methyl alcohol fumes for 3 1/2 days, 6 hours per day, during which time training was discontinued.

D	N	D	N
70 cm.	5 - 3 - 5	90 cm.	7 - 9 - 7 - 8 - 9 - 10
110 cm.	8 - 8 - 8	85 cm.	10 - 10
100 cm.	10	80 cm.	10 - 9
90 cm.	8	75 cm.	9 - 9
80 cm.	7 - 7 - 8 - 10 - 10	70 cm.	9 - 10
75 cm.	9	65 cm.	10 - 8
70 cm.	10	60 cm.	9 - 10
65 cm.	9	55 cm.	8 - 7 - 10 - 9 - 10
60 cm.	8	50 cm.	8 - 9
55 cm.	10	45 cm.	8 - 10
50 cm.	6 - 7 - 4	40 cm.	9 - 5 - 8 - 10 - 6
90 cm.	9	70 cm.	8 - 10
80 cm.	7 - 10 - 7 - 7 - 7	65 cm.	9 - 9
110 cm.	9	60 cm.	8 - 9
100 cm.	9	55 cm.	7 - 8 - 7 - 8 - 9 - 9

Rat VIII (continued)

D	N
50 cm.	8 - 6 - 8 - 7 - 10 - 6 - 9 - 7
80 cm.	9 - 10
65 cm.	10 - 9
60 cm.	8 - 8
55 cm.	8 - 9
50 cm.	9 - 8
45 cm.	10 - 10
40 cm.	9 - 8
35 cm.	8 - 8
30 cm.	8 - 6 - 8 - 7 - 10 - 9 - 8
25 cm.	7 - 10 - 8 - 9
20 cm.	8 - 9
15 cm.	8 - 7 - 7 - 7
50 cm.	8 - (10)
45 cm.	(10)
40 cm.	(10)
35 cm.	(9)
30 cm.	(10)
25 cm.	(8) - 9
20 cm.	6 - 8 - 9 - 9
15 cm.	9
10 cm.	8 - 9
5 cm.	8 - 6 - 4

Rat VIII (continued)

D	N
40 cm.	10 - (10 - 10)
35 cm.	(9)
30 cm.	(10)
25 cm.	(10)
20 cm.	(9)
15 cm.	(9)
10 cm.	(6 - 6)
40 cm.	(9 - 8) - in state of coma, unable to run - 10. Last day rat was exposed to fumes 6 hours.
35 cm.	8 - 10
30 cm.	10 - 9
25 cm.	9
20 cm.	10
15 cm.	10
10 cm.	7 - 6

Rat IX

D	N
159 cm.	4 - 3 - 2 - 5 - 6 - 3 - 7 - 5 - 6 - 3 - 5 - 2* - 3* - 3*- 7*- 4 - 6 - 7 - 5 - 6 - 9 - 7 - 9 - 6 - 7 - 6 - 6 - 9 - 10- 10- 9 - 9**
	In following tests controls were used, that is, the end stop was left out, and noise was kept constant.
130 cm.	8 - 8 - 9
110 cm.	8
90 cm.	6 - 8 - 10 - 8
80 cm.	9
70 cm.	5 - 5
80 cm.	9
70 cm.	9 - 6 - 4
80 cm.	7 - 5
90 cm.	7 - 6 - 6 - 9 - 8 - 8 - 8 - 8
80 cm.	7
110 cm.	6 - 8 - 9 - 8
90 cm.	8
80 cm.	6
130 cm.	8
110 cm.	10
90 cm.	8
85 cm.	9
80 cm.	9
75 cm.	10

Rat IX (continued)

D	N	D	N
70 cm.	10	45 cm.	10 - 10
65 cm.	7	40 cm.	8 - 10
75 cm.	9 - 7	35 cm.	9 - 9
110 cm.	10	30 cm.	10 - 9
90 cm.	8 - 9	25 cm.	10 - 9
85 cm.	10	20 cm.	9 - 8
80 cm.	8	15 cm.	10 - 8
75 cm.	6 - 9 - 7 - 9 - 10 - 8	10 cm.	6 - 4
70 cm.	10	50 cm.	10 - 9
65 cm.	10	45 cm.	9 - 9
60 cm.	9	40 cm.	8 - 10
55 cm.	9	35 cm.	9 - 9
50 cm.	7 - 10 - 6 - 9 - 8 - 10	30 cm.	9 - 8
45 cm.	9	25 cm.	7 - 8 - 9 - 8
40 cm.	10	20 cm.	9 - 8
35 cm.	9	15 cm.	8 - 6 - 10 - 8 - 8
30 cm.	9	10 cm.	6 - 5 - 3
25 cm.	9	30 cm.	(10)
20 cm.	7 - 6	40 cm.	(7)
65 cm.	8 - 10	50 cm.	(8 - 7)
60 cm.	9	70 cm.	(10 - 9)
55 cm.	10 - 10	65 cm.	10
50 cm.	10 - 10	60 cm.	10

Rat IX (continued)

D	N
55 cm.	10
50 cm.	9
45 cm.	10
40 cm.	10
35 cm.	10
30 cm.	9
25 cm.	10
20 cm.	8 - 9
15 cm.	7 - 6 - 7
50 cm.	9 - (8 - 10)
45 cm.	(8 - 10)
40 cm.	(10)
35 dm.	(9)
30 cm.	6 - 8 - 9 - 8
25 cm.	9 - 10
20 cm.	8 - 10
15 cm.	10
10 cm.	8 - 4 - 6
50 cm.	9 - (9 - 9)
45 cm.	(10)
40 cm.	(9)

Training stopped

D	N
35 cm.	(9)
30 cm.	(10)
25 cm.	(8 - 8)
20 cm.	(9)
15 cm.	(9)
10 cm.	(9)
5 cm.	(8 - 8)
0	(5)
40 cm.	(9 - 8) - sick from fumes, does not run - 6 - 6

6 hrs. exposure each of last 2 days

50 cm.	10
45 cm.	10
40 cm.	9
35 cm.	10
30 cm.	10
25 cm.	8 - 9
20 cm.	8 - 9
15 cm.	9
10 cm.	7 - 6
15 cm.	(9 - 8) - 9

6 hrs. exposure each day

Rat X

D	N
159 cm.	2 - 2 - 4 - 1 - 3 - 3 - 5 - 4 - 3 - 4 - 5 - 2* - 1* - 3* - 5*- 3 - 3 - 4 - 7 - 7 - 5 - 6 - 6 - 5 - 5 - 6 - 6 - 4* - 9 -10 -10 -10**
	End stop left out and noise made constant throughout the rest of the tests.
130 cm.	9 - 9
110 cm.	9
90 cm.	7 - 9 - 8 - 9 - 6 - 10 - 9
80 cm.	6 - 7 - 9 - 8 - 8
70 cm.	8
60 cm.	8
55 cm.	7
60 cm.	8 - 8 - 10
55 cm.	8 - 5
60 cm.	6 - 7
80 cm.	8
70 cm.	9
60 cm.	8
55 cm.	9
60 cm.	9
55 cm.	8
50 cm.	5
70 cm.	9
60 cm.	7

Rat X (continued)

D	N
90 cm.	9
80 cm.	8
70 cm.	8 - 7
90 cm.	7 - 6 - 9 - 7 - 9 - 6 - 7
130 cm.	7 - 7 - 7 - 8 - 8 - 9 - 7 - 7 - 8 - 6 - 8 - 6 - 7 - 8 - 10 - 9
120 cm.	8
110 cm.	8
100 cm.	9
90 cm.	8
85 cm.	9
80 cm.	8
75 cm.	8
70 cm.	9
65 cm.	10
60 cm.	8
55 cm.	10
50 cm.	9 - 9
45 cm.	8 - 9
40 cm.	9 - 9
35 cm.	7 - 8 - 9 - 9
30 cm.	8 - 9
25 cm.	7 - 9 - 9 - 9

Rat X (continued)

D	N
20 cm.	7 - 8 - 8 - 4
70 cm.	8 - 10
65 cm.	9 - 10
60 cm.	8 - 10
55 cm.	7 - 5 - 5 - 8 - 9 - 9
50 cm.	8 - 5 - 9 - 8 - 9
45 cm.	8 - 7 - 6 - 9 - 10 - 8
40 cm.	8 - 6 - 9 - 9 - 9
35 cm.	8 - 10
30 cm.	8 - 7 - 6 - 8 - 8 - 5
80 cm.	9 - 8 - (10 - 10 - 9)
75 cm.	(9)
70 cm.	(8 - 10)
65 cm.	10
60 cm.	10
55 cm.	10
50 cm.	9
45 cm.	9
40 cm.	8 - 9
35 cm.	10 - 9
30 cm.	10 - 9
25 cm.	9 - 9
20 cm.	8 - 8

Rat X (continued)

D	N
15 cm.	8 - 7 - 9 - 9 - 7 - 5 - 8 - 9 - 10
10 cm.	7 - 8 - 6 - 6
50 cm.	10 - (10 - 9)
45 cm.	(9)
40 cm.	(7 - 9 - 10)
35 cm.	9
30 cm.	10
25 cm.	10
20 cm.	10
15 cm.	7 - 8 - 9 - 8
10 cm.	7 - 8 - 7 - 3
40 cm.	(10 - 10)
35 cm.	(10)
30 cm.	(8 - 9)
25 cm.	(7 - 6 - 8 - 9)
20 cm.	(6 - 9)

Experimentation was discontinued.

Rat XI

D	N
159 cm.	5 - 3 - 2 - 4 - 6 - 5 - 5 - 5 - 7 - 5 - 3 - 4 - 4 - 8 - 8 - 7 - 8 - 8 - 5 - 7 - 7 - 8 - 7 - 7 - 8 - 8 - 7 - 4*- 6*- 6*- 7 - 3*- 10- 8**
	End stop left out, and noise kept constant during following tests.
130 cm.	5 - 8
159 cm.	9 - 8 - 7 - 8 - 9 - 10 - 8
130 cm.	7 - 9 - 7 - 9 - 9 - 10
110 cm.	9 - 10
90 cm.	8 - 7 - 8 - 8 - 7 - 10 - 9 - 8
80 cm.	10
70 cm.	7
110 cm.	10
90 cm.	10
80 cm.	9
75 cm.	7
110 cm.	9
90 cm.	10
80 cm.	8
75 cm.	7 - 10 - 9 - 8
70 cm.	7 - 9 - 7 - 9 - 8 - 10
65 cm.	10
60 cm.	8

Rat XI (continued)

D	N
55 cm.	8
50 cm.	10
45 cm.	7 - 9 - 8 - 9
40 cm.	10
35 cm.	7 - 8
30 cm.	10
25 cm.	7 - 8 - 8 - 7 - 9 - 8 - 7
50 cm.	8 - 9
45 cm.	7 - 9 - 6 - 8 - 10 - 9
40 cm.	9 - 10
35 cm.	9 - 8
30 cm.	9 - 9
25 cm.	9 - 8
20 cm.	9 - 9
15 cm.	8 - 5 - 9 - 7 - 8 - 9 - 9
10 cm.	7 - 6 - 8 - 4
40 cm.	10 - 10
35 cm.	10 - 8
30 cm.	10 - 8
25 cm.	9 - 8
20 cm.	8 - 8
15 cm.	9 - 5 - 9 - 9 - 8

D	N
10 cm.	8 - 8
5 cm.	8 - 7 - 6 - 8 - 7 - 4 - 3
30 cm.	9 - (10)
60 cm.	(10 - 10)
55 cm.	(9)
50 cm.	(8 - 8)
45 cm.	8 - 7 - 8 - 10 - 9
40 cm.	9
35 cm.	7 - 10 - 10 - 8
30 cm.	10
25 cm.	8 - 10
20 cm.	10
15 cm.	7 - 7 - 5
50 cm.	10 - (10 - 10)
45 cm.	(10)
40 cm.	(10)
35 cm.	(10)
30 cm.	(10)
25 cm.	9
20 cm.	8 - 8
15 cm.	8 - 8
10 cm.	6 - 8 - 7 - 6

Rat XI (continued)

D	N
50 cm.	10 - (9 - 10)
45 cm.	(7 - 8 - 7 - 10 - 9 - 9)
40 cm.	(8 - 8)
35 cm.	(9)
30 cm.	(9)
25 cm.	(8 - 10)
20 cm.	(10)
15 cm.	(10)
10 cm.	(7 - 9 - 9 - 7 - 6)
40 cm.	(8 - 9) - 7 - 10 - 8 - 9

6 hrs. exposure last day

35 cm.	10
30 cm.	8 - 9
25 cm.	8
20 cm.	9
15 cm.	8

Experimentation discontinued

Rat XII

D	N
159 cm.	6 - 2 - 5 - 4 - 5 - 5 - 1* - 6 - 8 - 4*- 7 - 5 - 5 - 4 - 6 - 5 - 5 - 5 - 4 - 6 - 1 - 3 - 4*- 6 - 2 - 5 - 6 - 6 - 5 - 7 - 4 - 7 - 8 - 8 - 7 - 5 - 5 - 5 - 8 - 8 - 8 - 7** 8 - 5 - 7 - 8 - 8 - 8 - 9**
	The end stop was left out and noise kept constant during remainder of the tests
130 cm.	9
120 cm.	6 - 9 - 9 - 10
110 cm.	7 - 8 - 9 - 9
100 cm.	9
90 cm.	9
80 cm.	9
70 cm.	10
65 cm.	7 - 6
110 cm.	7 - 7 - 7
130 cm.	9 - 8 - 9
120 cm.	10 - 7 - 8 - 8 - 7 - 9 - 10 - 9
110 cm.	7 - 9 - 9 - 10
100 cm.	9 - 10
90 cm.	10 - 10
85 cm.	10 - 8
80 cm.	9 - 10
75 cm.	10 - 9
70 cm.	10 - 9
65 cm.	6 - 8 - 10 - 10

Rat XII (continued)

D	N
60 cm.	10 - 9
55 cm.	9 - 9
50 cm.	10 - 9
45 cm.	9 - 9
40 cm.	6 - 9 - 10 - 9
35 cm.	8 - 10 - 8
30 cm.	9 - 7 - 8 - 7 - 6
60 cm.	9 - 9
55 cm.	10 - 10
50 cm.	10 - 10
45 cm.	9 - 5 - 9 - 9 - 10
40 cm.	7 - 7 - 9 - 10 - 10
35 cm.	9 - 10
30 cm.	9 - 7 - 9 - 10 - 9
25 cm.	6 - 9 - 9 - 10
20 cm.	9 - 9
15 cm.	7 - 6
60 cm.	8 - (9 - 8 - 9)
55 cm.	(10)
50 cm.	(8 - 7) - 5 - 9 - 9 - 7 - 7 - 8 - 8 - 7
70 cm.	7 - 9 - 10 - 7 - 10 - 8 - 9
65 cm.	8 - 10
60 cm.	9

Rat XII (continued)

D	N
55 cm.	9
50 cm.	7 - 9 - 9 - 9
45 cm.	8 - 8
40 cm.	10
35 cm.	8 - 8
30 cm.	10
25 cm.	10
20 cm.	10
15 cm.	7 - 9

Rat XIII

D	N
159 cm.	6 - 4 - 4 - 6 - 5 - 4 - 6 - 7 - 4 - 4 - 6 - 5 - 9 - 5 - 6 - 6 - 6 - 6 - 6 - 8 - 6 - 7 - 5 - 7 - 6 - 8 - 6 - 5 - 5 - 9 - 9 -10 -6**-8**- 8 - 9 - 6 - 9 - 9 - 9 - 10**
	End stop left out and noise kept constant during rest of the tests.
130 cm.	8 - 8
120 cm.	8 - 9
110 cm.	7 - 8 - 10 - 9
100 cm.	8 - 10
90 cm.	8 - 9
80 cm.	6 - 8 - 6
130 cm.	10
120 cm.	8
110 cm.	10
100 cm.	9
90 cm.	9
80 cm.	9
70 cm.	9
65 cm.	10
60 cm.	9
55 cm.	9
50 cm.	9
45 cm.	7 - 8 - 9 - 10
40 cm.	9 - 9

Rat XIII (continued)

D	N
35 cm.	8 - 8
30 cm.	8 - 8
25 cm.	6 - 9 - 7 - 8 - 7
70 cm.	6 - 8 - 7 - 8 - 7 - 6
90 cm.	8 - 9
85 cm.	9 - 10
80 cm.	10 - 8
75 cm.	7 - 10 - 8 - 7 - 9 - 8 - 7
90 cm.	9 - 8 - 9
80 cm.	7
90 cm.	7 - 9 - 7 - 8 - 10 - 8 - 8
85 cm.	10 - 10
80 cm.	8 - 8
75 cm.	10 - 10
70 cm.	10 - 9
65 cm.	10 - 9
60 cm.	9 - 10
55 cm.	9 - 9
50 cm.	9 - 10
45 cm.	9 - 9
40 cm.	7 - 9 - 10 - 9
35 cm.	9 - 10
30 cm.	9 - 8

Rat XIII (continued)

D	N
25 cm.	10 - 9
20 cm.	7 - 7 - 7
60 cm.	8 - 6 - 7 - 7
70 cm.	8 - 8 - 7 - 8 - (9 - 9)
65 cm.	(8 - 8)
60 cm.	(8 - 8)
55 cm.	9
50 cm.	8 - 8
45 cm.	9
40 cm.	8 - 9
35 cm.	9
30 cm.	9
25 cm.	10
20 cm.	8 - 8
15 cm.	7 - 8 - 7 - 8 - 8 - 5
50 cm.	8 - 6 - 10 - 6
60 cm.	8 - 8 - (10 - 9)
55 cm.	(9)
50 cm.	(8 - 5 - 9) - 10

Training stopped.

Rat XIV

D	N
159 cm.	3 - 2 - 3 - 6 - 6 - 4 - 2 - 4 - 5 - 3 - 5 - 5 - 4 - 4 - 6 - 6 - 7 - 4 - 4 - 3 - 5 - 3*- 5*- 4*- 8 - 2 - 7 - 1*- 5*- 5*- 4*- 3 - 7 - 7 - 9 - 10- 9 - 9**
	End stop left out and noise kept constant during remainder of the test.
130 cm.	10
110 cm.	8 - 9
90 cm.	9
80 cm.	10
70 cm.	10
65 cm.	9
60 cm.	10
55 cm.	10
50 cm.	10
45 cm.	9
40 cm.	6
70 cm.	10
65 cm.	9
60 cm.	8
55 cm.	8
50 cm.	8
45 cm.	8
40 cm.	8
35 cm.	10

Rat XIV (continued)

D	N
30 cm.	10
25 cm.	7 - 7
70 cm.	8 - 9
65 cm.	8
60 cm.	8
50 cm.	8
45 cm.	8 - 7 - 9 - 8 - 9
40 cm.	10 - 7 - 7 - 9 - 10 - 8
35 cm.	8 - 5 - 7
70 cm.	8 - 9
65 cm.	8 - 8
60 cm.	8 - 8
55 cm.	7 - 8 - 10 - 10
50 cm.	9 - 9
45 cm.	9 - 8
40 cm.	9 - 10
35 cm.	8 - 8
30 cm.	6 - 8 - 9 - 8
25 cm.	9 - 7 - 10 - 9 - 5 - 9 - 9 - 9
20 cm.	9 - 5 - 7 - 3
40 cm.	8 - 9 - 7
50 cm.	10 - 7 - 8 - 8 - 8
70 cm.	7 - 8 - 8 - 8
65 cm.	10 - (10)

Rat XIV (continued)

D	N
60 cm.	(9)
55 cm.	(9)
50 cm.	(8 - 8)
45 cm.	(9)
40 cm.	8 - 9
35 cm.	9
30 cm.	8 - 9
25 cm.	7 - 8 - 9 - 7 - 8 - 8 - 8
20 cm.	6 - 8 - 6 - 5
60 cm.	8 - (9 - 5 - 8 - 7 - 7)
70 cm.	Unable to run for 2 days 5 - 7
80 cm.	9
75 cm.	9
70 cm.	10
65 cm.	10
60 cm.	10
55 cm.	8 - (8)
50 cm.	7 - 9 - 8 - 9
45 cm.	10
40 cm.	10
35 cm.	8 - 8
30 cm.	9
25 cm.	8 - 9

Rat XIV (continued)

D	N
20 cm.	5 - 8 - 6 - 9 - 7
50 cm.	(10)
45 cm.	(8 - 9)
40 cm.	(7 - 9)

Training stopped.

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