III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

Pure acetylene, C_2H_2 (molecular weight 26.04), is a colorless and odorless gas. The characteristic garliclike odor of technical grade acetylene is attributable to contamination by impurities. Impurities which may be present include: divinyl sulfide, ammonia, oxygen, nitrogen, phosphine, arsine, methane, carbon dioxide, carbon monoxide, hydrogen sulfide, vinyl acetylene, divinyl acetylene, diacetylene, propadiene, hexadiene, butadienyl acetylene, and methyl acetylene. [1] Pertinent physical properties of acetylene are listed in Table XII-1. [2-4]

The oldest and most widely used method for producing acetylene is the reaction of water with calcium carbide. The other major method of acetylene production is by thermal cracking of hydrocarbons. [1]

Acetylene produced on a large scale for commercial purposes was first reported by Willson and Suckert [5] in 1895. In their process, calcium carbide was produced from lime and coke in an electric furnace. The use of acetylene produced from calcium carbide spread quickly and, before the end of the 19th century, it was manufactured in 10 countries. [6]

Calcium carbide served as the sole raw material source for acetylene manufactured in the US until 1951, when production of acetylene from hydrocarbon sources began. [7] Initially, hydrocarbon-produced acetylene was recovered as a byproduct of ethylene production. Ethylene recovered from refinery gases or manufactured by cracking other hydrocarbons contained 0.2-2.0% acetylene by weight.

The three general methods for commercial production of acetylene from

hydrocarbons are the thermal (Wulff) process, [8] the partial oxidation processes, [7] and the electric arc process. [7] The Wulff process uses a feed gas (methane, ethane, propane, or any C2 to C15 hydrocarbon stream) which is passed through a heated furnace. Partial oxidation processes depend on the partial oxidation of a hydrocarbon from which acetylene is isolated and produced. In the US, methane is used as a feed gas, yielding a gas mixture containing 8-10% acetylene. [7] The electric arc process uses an electric arc to produce the heat necessary for hydrocarbon conversion to acetylene. [7]

In 1972, Japanese researchers introduced the use of a high-temperature gas (plasma) jet to produce acetylene from higher aliphatic hydrocarbons. [9] In this process, propane and isobutane were found to be the most suitable starting materials. The advantage of this system is that the cost of plant construction is reduced because a large quantity of acetylene can be produced from a small unit. Since oxygen is not present in the plasma jet, acetylene is easily separated and refined. [10]

In 1973, a new process for producing acetylene from coal was reported by the US Bureau of Mines' Office of Coal Research. [11] This process may allow acetylene to be economically competitive with ethylene as a starting material for vinyl chloride production.

The production method used determines the nature and amount of the impurities which will be present. Acetylene manufactured from hydrocarbon feedstock is inherently free of phosphine, arsine, and hydrogen sulfide, but all of these impurities may be present in acetylene mixtures produced from calcium carbide. [12] Production of crude acetylene from calcium carbide in acetylene generators results in a number of other impurities.

Calcium sulfide, calcium phosphide, and calcium cyanamide, which are present in some industrial grades of calcium carbide, produce hydrogen sulfide, phosphine, and ammonia, respectively. These substances are present in varying but small proportions in crude acetylene gas. Both arsine and silane reaction products may be produced from trace amounts of other contaminants present in the calcium carbide. [13]

In 1958, worldwide production of acetylene amounted to 2.1 million tons, of which 83% was generated from calcium carbide. [14] In the US, 0.4 million tons of acetylene were produced, 60% of which was generated from calcium carbide. In 1966, US acetylene production amounted to 0.5 million tons, 23% of which was generated from calcium carbide. In 1974, seven companies in the US were responsible for the production of 0.2 million tons of acetylene from hydrocarbons and from ethylene byproduct processes. [15] No information is available on the percentage produced by the carbide process in 1975.

The first major commercial use of acetylene was as an illuminant and heat source. [1] Concomitant with the advancement of oxyacetylene technology in the first decade of the 20th century, the use of acetylene was expanded to the welding and cutting industries. [6] This development was enhanced by the discovery that hazards due to the explosibility of compressed acetylene could be overcome by dissolving acetylene in acetone disseminated on a porous mass inside a steel container. [16]

The current uses of acetylene may be categorized as nonchemical or chemical. Nonchemical uses of acetylene are as a fuel gas, eg, in the welding and cutting industries, and other miscellaneous uses, eg, as a temporary or emergency illuminant. Chemically, acetylene is used as a

starting material for a number of products such as vinyl chloride, vinyl acetylene, acrylonitrile, vinyl acetate, tetrachloroethane, acrylic acid, methyl and ethyl acrylates, propargyl alcohol, vinyl ethers, acetylene black, 2-butyne-1,4-diol, 3-methyl-1-butyn-3-ol, 3-methyl-1-pentyn-3-ol, bicycloheptadiene, and ethylidene fluoride. [7] In addition, there are miscellaneous chemical uses of acetylene, eg, as a ripening agent for fruit. The use of acetylene as a raw material for chemical synthesis during the years 1935-1966 is summarized in Table XII-2. [7] In 1973, 0.2 million tons of acetylene produced from hydrocarbons were used in the US, most of it in the synthesis of other chemicals: vinyl chloride monomer, 33%; acrylates, 26%; acetylenic chemicals, 20%; vinyl acetate, 17%; and chlorinated solvents, 4%. [15]

The industrial importance of acetylene has been eclipsed by the introduction of less expensive raw materials, such as ethylene and propylene. [15] Acetylene has been competing with butadiene as a starting material in the manufacture of neoprene, which, prior to 1968, was produced entirely from acetylene. [7]

An increase in the use of Reppe chemicals (chemicals synthesized from acetylene at pressures greater than atmospheric), such as propargyl alcohol, may increase the demand for acetylene, thus checking to some extent its present production decline. [7] In addition, use of acetylene for the production of tetrahydrofuran is increasing. [15] In 1966, approximately 110 million pounds of acetylene were used for nonchemical purposes, principally in welding and metal-cutting. Other gases such as liquid petroleum gas and physical agents such as electric-arc procedures have also been used in welding and cutting operations. [7]

A commodity specification for acetylene was developed by the Compressed Gas Association in 1972. [12] It attempted to set specification requirements for all grades of acetylene which were available commercially and for which an end use had been established through industrial experience. This information is presented in Table XII-3. [12]

A number of occupations that involve potential exposure to acetylene are listed in Table XII-4. [17] Since acetylene is used in a wide variety of industrial operations, NIOSH estimates that approximately 1,700,000 workers are potentially exposed to acetylene in the US.

Historical Reports

Miller [18] reported that Edmund Davy first produced acetylene in 1836 by the reaction of potassium carbide with water. The first report of exposure to acetylene described its use at high concentrations as a general anesthetic. [19] These reports involved exposure to partially purified acetylene produced by the action of water on crude calcium carbide. This acetylene probably contained impurities such as phosphine, arsine, hydrogen sulfide, ammonia, and carbon monoxide. Biologic or adverse effects attributed to acetylene were probably caused in part by one or more of these impurities. [20,21]

Von Oettingen [20] reported that, in 1883, Lewin produced anesthesia by administering acetylene in air at a concentration of 1% (10,000 ppm). Experimental results reported by Lewin and others before 1900 were probably obtained using unpurified acetylene of unknown composition generated from carbide, and thus the exact anesthetic concentration cannot be related to

later work. [21]

The first extensive laboratory and clinical investigations on the use of acetylene as an inhalation anesthetic reportedly were conducted in the early 1920's by Wieland and Gauss, as reported by Reisch. [22] Following their work, acetylene was used as an inhalation anesthetic in central Europe, and the majority of published reports, both experimental and clinical, appeared in the German literature. At that time, purified acetylene for anesthetic use was being marketed under the name of Narcylene. [23] By 1924, Goldman and Goldman [24] reported that Gauss had successfully used acetylene as an anesthetic in over 2,000 human operative procedures lasting from 3 to 180 minutes. Acetylene never became popular as an anesthetic because of its disagreeable odor. [25] Another drawback noted by Adriani [26] was its undesirable effect on the circulation.

No reports of acute or chronic toxic effects were found in early clinical or laboratory investigations of acetylene as an anesthetic.

Effects on Humans

Acetylene is toxic in the sense that it will produce varying degrees of temporary and reversible narcosis when administered with oxygen in concentrations of 100,000 ppm or greater. [27,28] Acetylene has been described as a safe anesthetic exhibiting in humans the advantages of an almost total lack of immediate aftereffects and no evidence of any residual or permanent toxicity. [24,29,30] In any event, the results of the anesthetic studies found in the literature cannot be directly related to most workplace environments because of the high acetylene and varying

oxygen concentrations used in these anesthetic studies.

Only one report has been found on industrial exposure to acetylene prior to 1940. In 1934, Eichler [31] reported the case of a worker exhibiting a variety of symptoms including bronchitis and a stomach ulcer after having been exposed to acetylene in an industrial environment. The employee had worked for an unspecified period of time in a welding plant where acetylene was produced from calcium carbide. No mention was made of any attempt to measure concentrations of acetylene or acetylene impurities present in the workplace. The opinion of the examining physician was that the worker's symptoms could have originated from chronic phosphine poisoning. In recent years, a number of case studies have appeared on the incidence of various health effects associated with the manufacture and use of acetylene. [32-38] In 1958, Harger and Spolyar [32] reported the case of a 16-year-old boy described as an inexperienced operator of a carbide-acetylene generator who was found dead, lying prone, with his face very near or partly over an open carbide hopper. The cause of death established at autopsy was acute pulmonary edema of undetermined cause. The authors concluded that the responsible toxic agent was probably phosphine. The presence of an 8-ppm average phosphine concentration was established by subsequent analyses performed on the air near the operator's breathing level during the hopper-filling period. Phosphine concentrations in the hopper ranged from 75 to 95 ppm. The raw acetylene produced in this plant was found to contain less than 3 ppm of arsine (no estimations of breathing zone concentrations were reported), and the "highest concentration of hydrogen sulfide breathed" was estimated at less than 10 ppm, and this for "only a short period." It is of interest

that, about 2 weeks before his death, the youth had begun to complain of periods of dizziness while filling the generator hopper with calcium carbide, and that, on two such occasions, he had reported losing consciousness. Ten days before his death, he had blacked out while driving home from work.

In 1960, Jones [33] reported a somewhat similar fatal incident in Australia. A carbide-acetylene generator operator was found either unconscious or already dead, lying across the top of a calcium carbide feed hopper, his head inside the filler hole. He was pronounced dead on arrival at a nearby hospital. The only significant autopsy findings reported were the presence of 8% carboxyhemoglobin in the blood (he was not a known smoker) and intense congestion of the lungs with widespread edema and areas of recent hemorrhage. A verdict of "death due to the effects of accidental poisoning by an irritant gas incurred in the course of his employment" was returned at the subsequent inquest. The author concluded from the circumstantial evidence that the victim might have been exposed to an atmosphere containing up to 80% acetylene with corresponding oxygen depletion, with resulting loss of consciousness. The presence of phosphine and hydrogen sulfide in the raw acetylene, and consequently in the air of the carbide hopper, was postulated but not established by analysis. It was suggested that carboxyhemoglobinemia was produced from carbon monoxide "trapped" in the calcium carbide and released on its reaction with water, but that the carbon monoxide did not contribute to the death of the victim.

In 1963, there appeared an isolated report [34] of urticaria and dyspnea associated with the use of an oxyacetylene torch in preheat welding. These signs occurred in one subject within 15 minutes of starting

a welding operation. The dyspnea disappeared within 1 hour after cessation of use of the torch, and the urticaria subsided within 6 hours. An experimental reexposure was performed 1.5 weeks later in the presence of several physicians. The precise working circumstances of the original incident were recreated and, 10 minutes after the test started, the subject had to discontinue welding because of marked dyspnea. At the same time, 10 extensive erythematous wheals appeared scattered over his chest, abdomen, arms, and back. These areas were pruritic; also, there was evidence of dermographism which had not been present before the test. Kaplan and Zeligman [34] concluded that the urticaria and "asthma" resulted from inhalation of "gases or particulate matters which originated from the contents of the acetylene tank, the combustion products of the commercial acetylene gas, and/or the combustion products of preheat welding with the railroad rod."

In the 1969 edition of their textbook, Deichmann and Gerarde [35] reported the case of a worker who inhaled acetylene gas from a leaking torch and was hospitalized 18 hours later because of severe dyspnea and chest pain. There was clinical and radiologic evidence of extensive pulmonary edema, bronchopneumonia, and bilateral pleural effusion. These authors believed that an unidentified impurity in the acetylene was probably responsible for the pulmonary irritation. They observed that acetylene previously had been used as an anesthetic with no reports of pulmonary changes of the type described in this case.

In 1970, Ross [36] reported loss of consciousness in a worker using an oxyacetylene flame in a confined space. Ross did not attribute the loss of consciousness to the effects of acetylene, but concluded that the

probable causal factors were oxygen depletion of the atmosphere linked with a high carbon dioxide concentration and the heat of the job. All these were aggravated by the small, confined working space. Here, the worker was using the torch inside a very small reheater boiler.

In 1971, de Hamel [37] reported what he described as "the first case of acute acetylene anesthesia which I have ever come across." An oxyacetylene torch was used under water to cut some steel reinforcing rods sticking out of a riverbed. The operator sat on the riverbed with his head just above the water. Bubbles of gas from the underwater torch-cutting process rose to the surface close to the worker's face. After an unstated period of time, the worker complained of feeling very weak, "like having an anesthetic." He turned off his torch and rested for a while. Shortly after resuming his work, he collapsed and was incapable of movement. After a third attempt, he collapsed and could not move for 15 minutes. He recovered completely by the following day. The author did not estimate exposure levels or give any patient history. It is unlikely that this worker was exposed to any appreciable concentration of uncombusted acetylene in his breathing zone; therefore, it is doubtful that this was a case of acute acetylene anesthesia.

In 1973, Ross [38] reported two additional cases of loss of consciousness, one fatal; both were associated with the use of oxyacetylene torches in a confined space. In this incident, the oxyacetylene torch was being used to "metallize" aluminum onto steel inside a tank where ventilation was inadequate, and it was concluded that the accident was caused by "an adverse breathing atmosphere" in the storage vessel (ie, the workspace). Once again, acetylene probably did not make a significant

contribution to the adverse breathing atmosphere because very little free acetylene would have been present with the torch lit. The cause of loss of consciousness was probably oxygen deficiency.

In none of the above studies is the causative or even contributory role of acetylene clear. Furthermore, in all of these studies, proper attention to good work practices might have prevented these health hazards.

No reports on humans have been found in the literature in which any histopathologic effects attributable to acetylene have been established. In 1926, Brandt [29] reported that an acetylene-oxygen mixture administered as an anesthetic at an initial concentration of 70% (700,000 ppm) did not appear to have any effect on either the liver or the kidney. Mueller [39] studied the effects of Narcylene (acetylene) on blood components. He reported that it produced no untoward hematologic effects when administered to 12 patients in anesthetic concentrations.

Acetylene has not been shown to cause any abnormal effects on heart function. In studies where acetylene was used at high concentrations in anesthesia, there have been some reports of an increase in blood pressure. Brandt [29] found no effects on heart function when acetylene was administered at a concentration of 200,000-700,000 ppm in oxygen, although blood pressure was seen to increase by 20, 40, or even 50 mmHg in some of the subjects studied. Franken and Schurmeyer [40] found in 1928 that acetylene-oxygen mixtures (750,000-800,000 ppm) produced an increase in blood pressure during anesthesia. The authors concluded that this effect was attributable to stimulation of the vasomotor center by acetylene. The full significance of this finding is not clear.

No reports have been found in the literature on the effects of concentrations of acetylene lower than 200,000 ppm in oxygen or air on the cardiovascular system in humans.

Goldman and Goldman [24] reported that Gauss used 80% (800,000 ppm) acetylene-oxygen mixtures in over 2,000 anesthetic procedures lasting from 3 minutes to 3 hours and that no signs of asphyxia were noted. Brandt [29] stated that acetylene, even when administered in high concentrations (up to 900,000 ppm), was not capable of paralyzing the respiratory center. Thus, the author concluded that an overdose in anesthetic procedures was "impossible." No mention was made of increased respiration rates in either study. Franken [41] found that acetylene administered in high concentrations (700,000-800,000 ppm) as an anesthetic to seven human subjects "stimulated" respiration. [41] However, all but two of the subjects had been premedicated with a morphine-containing preparation which affects respiration. No studies on the mechanism by which acetylene stimulates respiration have been found in the literature. However, Rehn and Killian [42] reported in 1932 that the sensitivity of the respiratory center to carbon dioxide was not altered by acetylene. No studies have been found in the literature on the effects of acetylene-oxygen or of acetylene-air mixtures on the respiratory system at concentrations below 200,000 ppm.

The reported effects of acetylene on metabolism are limited to a single study in 1930 by Von Ammon and Schroeder [43] who found that the alkali reserve in humans was lowered during acetylene anesthesia in a wide variety of operative procedures. However, the value of this study is diminished by the fact that all patients were given morphine and atropine

after the baseline reading had been established. It is impossible to assign the observed changes to the premedication, the anesthetic agent, or the operative trauma. No published reports have been found concerning the effects of acetylene-oxygen or of acetylene-air mixtures on human metabolic parameters at concentrations below 200,000 ppm.

In literature published prior to 1930, narcosis has been described as the main central nervous system effect of acetylene when administered with oxygen at concentrations of 100,000 ppm or greater. [28] A summary of the anesthetic effects of different acetylene-oxygen mixtures is presented in Table XII-5. [24,28-30,40,41] No reports have been found on the effects of acetylene-oxygen or of acetylene-air mixtures on the central nervous system at concentrations below 100,000 ppm.

In summary, on the basis of the foregoing review of available reports, both of acetylene-related industrial accident histories and of clinical investigations of acetylene anesthesia, there is no evidence of toxic effects of acetylene on humans.

Epidemiologic Studies

A search of the literature on acetylene has yielded neither reports of epidemiologic studies nor data on group exposure or effects that could be analyzed epidemiologically.

Animal Toxicity

Acetylene is toxic to animals only in the sense that it will produce varying degrees of narcosis when administered with oxygen in concentrations

exceeding 200,000 ppm. Furthermore, acetylene has been described as a safe anesthetic exhibiting an almost total absence of aftereffects and no evidence of any residual or permanent toxicity. [21,44-46]

The only report on the toxic effects of acetylene in air was published by Flury [27] in 1928. The author stated that toxicity to warm-blooded animals was as follows:

500,000 ppm after 5-10 minutes	-	fatal
250,000 ppm after 30-60 minutes	-	toxic
100,000 ppm for 30-60 minutes	-	tolerated

It should be noted in interpreting this report that the author did not specify species, method of administration, duration of exposure, or experimental conditions. Furthermore, acetylene concentrations of greater than 100,000 ppm in air would have reduced the oxygen content of the mixtures to below 18%. Thus, the observed toxic effects may not have been produced by the acetylene but rather by the oxygen-deficient atmosphere.

Other studies found involved the use of acetylene-oxygen mixtures containing more than 100,000 ppm of acetylene. In 1933, Franken and Miklos [47] looked for possible organ damage from the administration of acetylene at anesthetic concentrations to rats, mice, guinea pigs, rabbits, and dogs. The authors found no evidence of cellular injury to the parenchymatous cells of the heart, lungs, liver, kidneys, or spleen. Animals were exposed to acetylene in oxygen at concentrations of 250,000, 500,000, or 800,000 ppm for 1-2 hours daily. The longest total exposure times for some of the animals were: rats, 93 hours at 250,000 ppm or 18 hours at 800,000 ppm;

guinea pigs, 18 hours at 500,000 ppm or 10 hours at 800,000 ppm; rabbits, 10 hours at 800,000 ppm; and dogs, 12 hours at 800,000 ppm. Acetylene caused slight capillary hyperemia in some animals exposed at 250,000-ppm concentrations. This effect was observed until at least the second day after the last exposure to the gas but was not evident in animals killed later (up to 5 days after the last exposure).

Acetylene administered to cats and rabbits at concentrations greater than 200,000 ppm produced a limited and temporary rise in blood pressure. [41,48-50] The full significance of this finding is not clear. No reports were found in the literature of any studies pertaining to the effect of acetylene or acetylene mixtures on the cardiovascular system at concentrations below 100,000 ppm. Hildebrandt et al, [48] in 1926, determined that the rise in blood pressure observed in cats during surgical administration of acetylene at a concentration of 800,000 ppm in oxygen was caused by a shift in blood flow to the periphery. The authors concluded that this shift was caused by splanchnic vessel constriction initiated by acetylene. Franken et al [49] observed a rise in the blood pressure of cats administered acetylene-oxygen anesthetic mixtures containing up to 800,000 ppm of acetylene. The magnitude of this rise was found to vary among individual animals. No increase was observed if the anesthetic was administered slowly and carefully. Bollert et al, [50] in 1927, conducted 30 experiments on cats to study the effects of acetylene on the circulation. Using acetylene concentrations of 200,000-800,000 ppm in oxygen, they found that systolic blood pressure climbed to a maximum and then slowly dropped to normal. During initial anesthetic administration, the blood pressure increased by 30-40 mmHg with some animals exhibiting an

increase of 80-100 mmHg. The authors concluded that this increase was caused by a stimulation of the vasomotor center with a concurrent displacement of blood from the splanchnic to the peripheral vessels. An observed slowing of the pulse rate was judged not to be related to the blood pressure increase but to a stimulation of the vagal center by acetylene. An increase in the amplitude of the pulse was also noted to be a consequence of this direct action on the heart. In 1930, Franken [41] reported that both blood pressure and systolic output increased slightly when acetylene-oxygen anesthetic mixtures were administered to cats and rabbits.

The effects of acetylene on the respiratory system reported in the literature have not been consistent; both stimulation and depression of respiratory function have been observed. No reports pertaining to the effects of acetylene or acetylene mixtures on the respiratory system at concentrations of less than 100,000 ppm have been found. Riggs, [51] in 1925, reported that a 900,000-ppm acetylene concentration produced respiratory failure in rats after 2 hours of administration. The author, however, mentioned neither the oxygen content of the anesthetic mixture nor the numbers of animals tested. Schoen and Sliwka, [52] in 1923, reported an increase in the respiration of rabbits after the termination of anesthetic procedures in which up to 600,000 ppm of acetylene in oxygen had been used. Heymans and Bouckaert, [45] in 1925, found that both the volume and the frequency of respiration were increased in dogs during the administration of an anesthetic mixture of acetylene at a concentration of 850,000 ppm in oxygen. The authors concluded that acetylene caused an increase in respiratory frequency and volume even during deep anesthesia,

and that it slightly stimulated the elimination of carbon dioxide. In 1930, Franken [41] found that respiration was decreased in cats and rabbits which were administered acetylene-oxygen anesthetic mixtures.

There have been some reports in the literature of the effects of acetylene-oxygen anesthetic mixtures on various metabolic parameters. However, there has been no consistency in the reported data, with the exception of a slight decrease in both the carbon dioxide content and the alkali reserve of the blood. [52-54] The full significance of these findings in terms of occupational exposure is not clear because high concentrations of acetylene in oxygen were used. No studies were found in the literature on the effects of acetylene or acetylene mixtures in concentrations below 100,000 ppm on animal metabolic parameters. In 1923, Schoen and Sliwka [52] used acetylene-oxygen mixtures in anesthetic studies with rabbits. The authors observed decreases both in carbon dioxide tension in the arterial blood and in carbon dioxide-combining power of the blood and a lowering of the blood alkali reserve, but they observed no effect on the oxygen content of the arterial blood. Derra and Fuss, [53,54] in 1932, studied the effects of acetylene anesthesia on carbohydrate and acid-base regulation in the blood of dogs which had been administered acetylene at a concentration of 800,000 ppm in oxygen for 1 hour. The authors observed the following metabolic changes: a decrease in both the alkali reserve and carbonic acid levels; an increase during, and a fall after, anesthesia in both the oxygen-binding capacity of the blood and the arterial-oxygen deficit (ie, the degree of oxygen unsaturation of the blood); and a slight increase in blood sugar levels during anesthesia. The decrease in blood carbonic acid levels was reported by the authors to be a

consequence of the fall in alkali reserve.

The only reported effect of acetylene on the central nervous system of animals was the narcosis induced by acetylene when administered with oxygen in concentrations greater than 200,000 ppm. [21,44,45]

No studies were found in the literature on the effects of acetylene, or acetylene mixtures, on the central nervous systems of animals at concentrations below 100,000 ppm. In 1923, Jordan [21] administered acetylene in oxygen at concentrations of 750,000-900,000 ppm by mask to produce anesthesia in dogs. The author observed a rapid recovery (less than 5 minutes) in the dogs, with no aftereffects. Prolonged exposure to the anesthetic mixture did not produce toxic effects in animals of any age; 4 hours under anesthesia was the same as 4 minutes with respect to ease of recovery.

These animal studies are summarized in Table XII-6.

Correlation of Exposure and Effect

In both humans and animals, the principal effect attributable to acetylene is a clinically safe and reversible anesthetic effect which is observed when acetylene is administered with oxygen in concentrations of 100,000-800,000 ppm in humans or 200,000-900,000 ppm in animals. The relevance of these anesthetic studies to occupational exposure to acetylene is not clear because the acetylene used was administered (1) in high concentrations in oxygen, and (2) only for short periods of time. No experimental studies, either chronic or acute, were found in the literature pertaining to the biologic effects of acetylene or acetylene mixtures at

concentrations below 100,000 ppm in either humans or animals.

Partially purified acetylene was used successfully, although not widely, as an inhalation anesthetic gas for about two decades and was abandoned, according to Sollmann, [25] only because of the disagreeable odor caused by its impurities. Concentrations as high as 40% (400,000 ppm) have been recommended for anesthesia in humans. [20,25] Most studies on the use of acetylene as an anesthetic ended in the 1930's. None of these studies can be definitively related to present-day occupational exposure to acetylene. The low toxicity observed in these acute studies reporting high concentrations of only partially purified acetylene would seem to indicate a low toxicity at concentrations near the proposed environmental limit of 2,500 ppm. However, no reports on chronic inhalation studies of acetylene have been found in the literature.

Other biologic effects reported in association with the manufacture or use of acetylene, such as pulmonary edema, [32,33] bronchospasm, [34] urticaria, [34] and acute anesthesia, [37] are probably due to the presence of toxic impurities in acetylene, to materials produced during its use, or to physical agents. In all such instances, proper attention to good work practices might have prevented these health hazards.

Carcinogenicity, Mutagenicity, and Teratogenicity

No reports have been found in the literature pertaining to the mutagenicity, carcinogenicity, or teratogenicity of acetylene.

Moreover, there are no reports of studies in either humans or animals aimed at elucidating and testing acetylene metabolites for carcinogenic, mutagenic, and teratogenic activities.