CASE REPORTS

ACCIDENTAL POISONING BY VINYL CHLORIDE: REPORT OF TWO CASES*

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VINYL CHLORIDE (VCl), also called chloroethylene or chloroethene, has the formula $CH_2{=}CH.Cl$ with the characteristic double bond of the ethylene group. Though first prepared in 1833, it has only lately been found of much technical use. It is the chloride of the hypothetical vinyl alcohol and is gaseous at ordinary room temperature.

Table I will clarify its chemical formula and relations to some other well-known volatile aliphatic compounds widely used as anæsthetics.

TABLE I.

	Alcohol	*Ether*	*Chloride*
Methyl	$CH_3.OH$	$(CH_3)_2.O$	$CH.Cl_3$ (chloroform) $CH_3.Cl$ (methylchloride
Ethyl	$CH_3.CH_2.OH$	$(C_2H_5)_2.O$	$CH_3.CH_2.Cl$
Vinyl	$CH_2{=}CH.OH$	$(CH_2{=}CH)_2.O$	$CH_2{=}CH.Cl$

VCl is technically produced by the action of hydrochloric acid upon acetylene; mercuric chloride is used as a catalyst. It is a colourless gas of not unpleasant odour. It vapourizes at a temperature of −14° C.; the freezing point is −159.7° C. It is a combustible and explosive substance and has to be handled with the necessary precautions. The density is 2.15 times heavier than air.

VCl is used as a refrigerant and in organic synthesis. It is the substance from which vinyl polymers, now widely used in the production of plastic materials, are made.

In the plant in which the two cases of fatal accidental poisoning occurred, VCl is not produced but brought in from another place in a compressed liquid form, then polymerization with the aid of catalysts is carried out. The polymer, polyvinyl resin, is a whitish powdery substance. The polymerization process is not followed through to completion. There is always some residual VCl left which is evacuated before the tank is opened and the polymer is collected. Chemical analysis of this residual gas, using a sample from the plant in question, showed it to consist of about 90% vinyl chloride, 8.5% carbon dioxide, small amounts of vinyl acetate, trichloroethylene and nitrogen and traces of argon.

Explosiometer tests are completed before a workman is allowed to enter the tank for cleaning, and during the cleaning process the air is continuously sucked out and replaced by fresh air.

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VCl has anæsthetic properties and produces narcosis in experimental animals. A number of reports of experiments with dogs, rabbits, guinea pigs, rats and mice are available.[2-7] VCl acts very much like ethyl chloride, but appears to be somewhat less toxic. The induction of narcosis is rapid and when low concentrations are used recovery is fast. It appears that chlorine is split off only with difficulty in the animal so that physiologically VCl is relatively free from many of the untoward effects of ethyl chloride.[3] It seems to stimulate respiration in the third stage of anæsthesia and also to have some tendency towards convulsant effects in very deep anæsthesia.[4] Elimination occurs very quickly.[5] VCl appears to sensitize the myocardium of the dog to adrenaline less frequently than ethyl chloride.[7] According to Schaumann (quoted from Oster *et al.*[6]), the narcotic limiting concentration is 7 to 10%; 12% is dangerous and its use in man is unwarranted. In the dog, marked changes in the cardiac rhythm during surgical anæsthesia were observed and electrocardiographic examinations revealed abnormalities varying from sinus arrhythmias and transitory left axis deviation to very serious conditions including atrioventricular block and multifocal extrasystoles.

No case of human death due to VCl has been reported to date. There is a report of a case[8] where a man had his hands accidentally sprayed with VCl. He developed erythema and some second-degree burns which healed without complication.

Two men exposed to 2.5% VCl in air for approximately three minutes reported that the gas had a fairly pleasant odour. They began to feel dizzy and slightly disoriented and complained of a burning sensation in the soles of their feet. They immediately recovered upon leaving the chamber, except for a slight headache lasting 30 minutes.[1]

The "Guide to the Diagnosis of Occupational Diseases" compiled by the Federal and Ontario Health Departments[10] states: "VCl is considered to be one of the least dangerous chlorinated hydrocarbons, no case of industrial poisoning having been reported. The maximum allowable concentration is about 500 p.p.m. Repeated daily exposures should be limited to concentrations below 500-1000 p.p.m."

CASE 1.—This was a young man, aged 21, who was employed to clean out the tanks after completion of the polymerization process. He had done this work for more than a year.

The polymerization tanks are eight feet high and five feet wide. An elliptic manhole just wide enough for one man to enter is in the upper part, about three feet above a platform. The "Factory, Shop and Office Building Act" of the Department of Labour of the Ontario Government (last issue 1958) requires very strict precautions for work of this type (No. 74, 5a-e). Locations in which dangerous fumes are liable to be present have to be properly aired and tested before anybody is allowed to enter. The workman has to have a suitable breathing apparatus and a

belt to which a rope is securely attached that is to be held by a second person outside. When it appears necessary, a suitable reviving apparatus has to be on hand.

The subsequent investigations showed that in this particular case explosiometer tests were made and the tank was considered safe to enter. However, the air hose appeared somewhat defective, the rescue apparatus was not immediately available, the man was not on a rope and nobody was in constant attendance outside.

About 10 minutes after the foreman had talked to the man, he found him lying on the bottom of the tank, apparently dead. After some delay in getting a rescue sling from the foreman's office, another man entered the tank to attach the noose to the victim's feet, who was then hauled out feet first. This man stated that he did not detect any odour of VCl in the tank. Artificial respiration, first by mouth-to-mouth breathing, was tried, but to no avail. Again it was stated that there was no odour of VCl on the dead man.

A post-mortem examination was begun three hours after death. The body was that of a young man of athletic build, length 177 cm. and estimated weight 60-65 kg. Cyanosis of fingernails and toenails was observed. The essential findings at autopsy were: Increase in size and weight of heart (445 g.), which was in systole. Coronary arteries were patent, but with more evidence of atherosclerosis than usually seen at this age. Lungs, trachea and bronchi were not remarkable on gross examination; kidneys congested, bluish red; brain not remarkable. No abnormal odour of the organs, especially of the lungs, was noted. The blood did not clot.

Microscopical examination did not yield any evidence of myocardial infarction or scarring. There was congestion of liver, spleen and kidneys. Some early degenerative changes of the tubular epithelium of the kidneys were observed; no fat was demonstrated in frozen sections of the kidneys. The lungs were slightly congested with small patchy areas of œdema. Some heart failure cells were found which gave a positive iron reaction.

Material was sent to the Attorney General's Laboratory in Toronto. As expected, nothing was found at toxicological examination. VCl is a very volatile gas and as its boiling point is −14° C. it would very quickly evaporate, especially with the attempts of artificial respiration and the exposure of the organs to the open air.

I stated at the inquest that this man died of asphyxia but that I could not determine the cause of it. The heart was definitely enlarged and the presence of heart failure cells in the lungs makes one think of some degree of heart insufficiency during life. The family of the deceased, however, stated that he had never had any heart trouble. One can speculate that he fainted and inhaled residual gas at the bottom of the tank, but there is no proof of it. In view of the experiments with dogs it seems also possible that a functional disturbance of an already diseased heart developed which caused the death, but again this is only guesswork.

The coroner's jury decided that the death was accidental, possibly due to inhalation of VCl, and recommended better precautionary measures. I feel that positive proof of poisoning by VCl is lacking, but it cannot be entirely excluded, especially in view of the similar autopsy findings in the second case in which there is no doubt that death was caused by inhalation of VCl.

In regard to the second case, it has been mentioned that some VCl remains in the tanks after completion of the polymerization process. This gas is pumped through a pipeline into a tank outside the main building for storage and re-use. The tank is about 75 yards from the main building and there is a rectangular pit in front of it, which is about seven feet deep. There is a valve in the pipe about 18 inches above the bottom of the pit through which condensed water has to be let out periodically, about three or four times during a 24-hour period.

CASE 2.—A man went out alone during the night shift to do this, as he had done before on several occasions. No light was provided above the pit. Since he did not return, somebody went out to look for him about 20 minutes later. He found him lying in the pit and climbed in to get him out, but was himself overcome by gas. He quickly shut the open valve. He felt giddy and saw circles in front of his eyes. He dragged himself through the snow on his hands and knees for about 15 yards and shouted for help. He recovered rather quickly and was able to help in the removal of the body of the victim. Another man wearing a gas mask had gone down and lifted the body out. Artificial respiration was tried but without success.

An autopsy was performed about eight hours after death. In view of the negative toxicological findings in the first case, we did not think it necessary to perform the autopsy during the night.

The body was that of a 39-year-old man; length 167 cm. and estimated weight 70-75 kg. Finger nails were deeply cyanotic, and superficial excoriations and cuts of the skin of the face were present. Both bulbar conjunctivæ showed wedge-shaped brown discoloration corresponding to the palpebral slits, and conjunctivæ and corneæ appeared dried out.

The heart was not remarkable, weighing 390 g. The blood was dark red and did not clot during four hours' observation. The lungs (right lung 525 g., left lung 470 g.) had some increase in fluid content. Trachea and larger bronchi had slight swelling and reddening of the mucosa, a small amount of secretion being present in the lumina. Liver was not remarkable. Kidneys were of ordinary size and weight, blue-red and firm, with evidence of congestion. Brain was not remarkable.

On microscopical examination, the lungs showed evidence of acute hyperæmia. There was some desquamation of alveolar epithelium. The pigment present gave a negative iron reaction. Intense hyperæmia of the submucosa of the bronchi and trachea was seen. Liver and spleen were less hyperæmic. Some destruction of the lining epithelium of the convoluted tubule of the kidneys, due to post-mortem autolysis, was present, along with severe acute hyperæmia.

Again material was sent to the Attorney General's Laboratory; no VCl was found.

It appears then that this man died from the inhala- of a very high concentration of VCl. As VCl is times heavier than air and as the valve was wide he must have inhaled the gas in a very con- rated form. The lesions of the eyes can be ex- ined by the local effect of the gas.

The autopsy findings in both cases are similar, but in no way diagnostic. Similar findings are described in cases of poisoning by methyl chloride, methyl bromide, ethyl chloride and trichloroethylene and in acute death due to chloroform and ether. In most of these cases the blood failed to clot and internal organs were congested.[9]

Mastromatteo[12] and associates lately made animal experiments with VCl obtained from the industrial plant in question. The experimental findings will be published elsewhere; with the author's permission I will summarize the experiments briefly.

Batches of mice, rats and guinea pigs were exposed to concentrations of VCl in air, increasing from 10 to 40%. Lower concentrations produced narcosis from which the animals recovered rather quickly. All mice and rats were killed by a concentration of 30% VCl. Some guinea pigs survived even a 40% concentration. Autopsy and microscopical findings were not diagnostic. Hyperæmia of the organs and failure of the blood to clot were observed.

SUMMARY AND CONCLUSIONS

Two cases of accidental fatal poisoning by vinyl chloride are reported. The second case appears proved by the circumstances; there is some doubt that VCl was responsible for the first death. The main pathological findings were cyanosis, local burns of conjunctivæ and corneæ, congestion of internal organs, especially lungs and kidneys, and failure of the blood to clot. Whereas it may be possible to make vinyl-chloride level estimations in blood and organs of experimental animals, this cannot be done in medico-legal practice because of the highly volatile nature of the gas. Diagnosis will depend mainly upon the surrounding circumstances.

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ACUTE RENAL TUBULAR NECROSIS REPORT OF CASE WITH HYPERPYREXIA TREATED BY REDUCTION OF BODY TEMPERATURE*

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THERE is evidence that the severe oliguria and azotæmia after an episode of hæmorrhagic shock or some other cause of severe renal ischæmia are the result of focal tubular necrosis involving both proximal and distal parts of the nephron.[1] Consequently the term "lower nephron nephrosis", previously applied to this disorder, has been replaced by "acute tubular necrosis". A complete description of the condition and its treatment can be found in the recent monograph by Merrill.[2]

The patient whose illness is to be reported had a complicated and stormy course and presented some unusual problems in therapy. The authors are not aware of any previous report in which reduction of 10° F. in temperature was induced as a therapeutic measure in acute tubular necrosis with or without hyperpyrexia.

R.C., a 39-year-old white housewife, was admitted to the Victoria General Hospital on August 12, 1959, with the diagnosis of menorrhagia, rectocœle and cystocœle. The latter had recently been associated with frequency and burning on urination, but there was no past history of kidney disease. The patient was normally developed and nourished, and, apart from the pelvic abnormalities, physical examination was within normal limits. Blood pressure was 110/68 mm. Hg. Urinalysis was negative and the hæmoglobin value was 14.3 g. %.

On August 13, a vaginal hysterectomy was carried out as well as a posterior colporrhaphy and Mayo repair. Three and one-half hours after this procedure, her blood pressure dropped to 70 mm. systolic, which suggested concealed hæmorrhage. The hypotension failed to respond to several litres of blood and 2 mg. noradrenaline diluted in 500 c.c. of 5% glucose in saline. A laparotomy was performed seven hours after the onset of severe hypotension and approximately 1500 c.c. of blood was removed from the peritoneal cavity. One litre of this intraperitoneal blood was filtered and autotransfused, after which she received a further 500 c.c. of bank blood. After securing haemostasis, 5 g. of sulfathiazole powder was placed in the peritoneal cavity and the abdomen was closed.

Fig. 1 charts the patient's urine output over the next two weeks. The low output should suggest strongly the presence of acute renal tubular necrosis when it persists after correction of deficiencies in water, salt and blood volumes. The pattern seen is atypical, however, in that the urine volume gradually rose to 750 c.c. daily on the fourth postoperative day and then, after an attempted intravenous pyelogram with Diodrast which the kidneys were unable to concentrate, the urine volume dropped to approximately 500 c.c. daily for five more days.

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