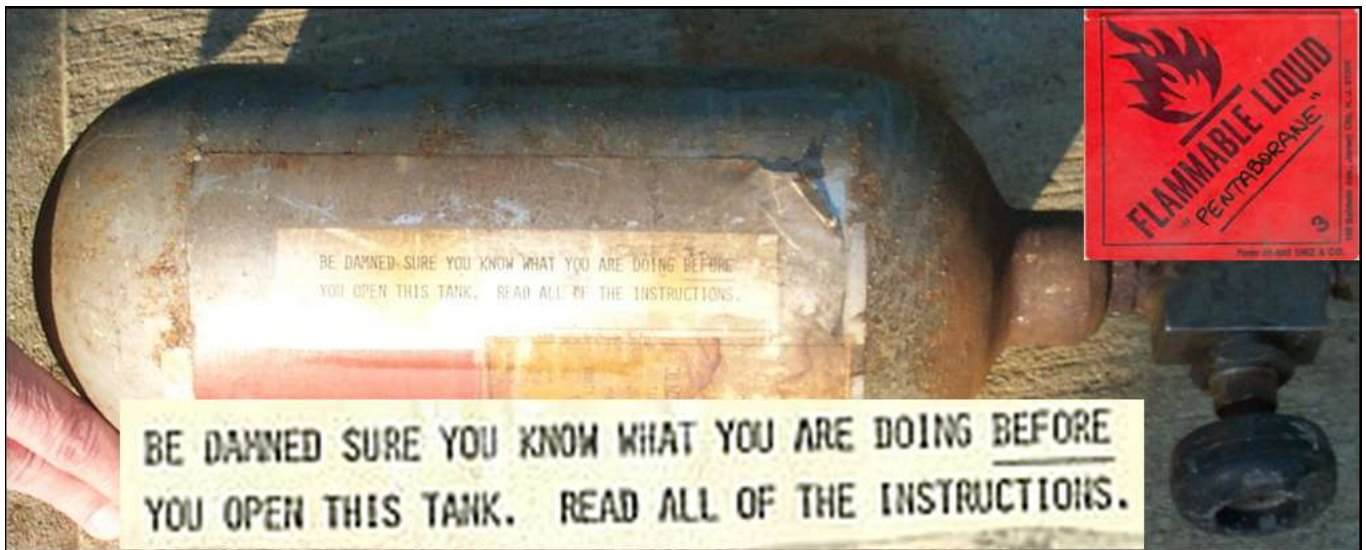




Metal Hydrides III: Boron Hydride — Diborane and Pentaborane



## ARDS, the Green Dragon, and Delayed-onset Neurotoxicity

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## Introduction

We've been working through the toxicity of metal hydride gases (**Figure 1**) and have come to the third and last installment. In first post we introduced the use of metal hydride gases in semiconductor manufacturing and discussed [arsine \(https://toxandhound.com/toxhound/ff-metal-hydrides/\)](https://toxandhound.com/toxhound/ff-metal-hydrides/) as well as the hydrides of silicon, germanium, antimony, tellurium, and selenium; explored metal biomethylation; reviewed poisoning by wallpaper; and discussed the toxicologic odor of garlic. The second post focused on [phosphine \(https://toxandhound.com/toxhound/ff-phosphine/\)](https://toxandhound.com/toxhound/ff-phosphine/), including self-combustion, fumigation and unintentional production during methamphetamine synthesis. We now will conclude with the boron hydrides and focus on diborane and pentaborane(9), which we will refer to as pentaborane, one of the most frightening and toxic agents historically encountered in industry and research. While pentaborane is a liquid at room temperature, it is volatile enough to quickly enter the atmosphere and produce toxicity by inhalation.

Element	Formula	Name
Antimony	$\text{SbH}_3$	Stibine
Arsenic	$\text{AsH}_3$	Arsine
Boron	$\text{B}_2\text{H}_6$	Diborane
Boron	$\text{B}_5\text{H}_9$	Pentaborane
Germanium	$\text{GeH}_4$	Germane
Germanium	$\text{Ge}_2\text{H}_6$	Digermane
Phosphorus	$\text{PH}_3$	Phosphine
Selenium	$\text{SeH}_2$	Hydrogen selenide
Silicon	$\text{SiH}_4$	Silane
Tellurium	$\text{TeH}_2$	Hydrogen telluride

Figure 1 – Metal hydrides

Eugene Ngai is with us again on this final installment and works internationally and extensively with diborane. Both of us are pleased Jeffrey Gold accepted our invitation to join us for this post on boron hydrides. Jeff is president and owner of Integrated Environmental Services, Inc. and also chief operating officer and founder of Nexus Circular. His career has focused on addressing chemical weapons, hazardous waste, plastic pollution, and the manufacturing of high-pressure chemical containment devices. Jeff undoubtedly has the world's greatest experience in dealing with and disposing of old pentaborane cylinders and brings first-hand knowledge of pentaborane's physical and toxic hazards.

## Brief History

The famous German chemist, Alfred Stock (**Figure 2**), began synthesizing and studying boron hydrides in the early 1900s.



Figure 2 – Alfred Stock. Wikipedia public domain.

Stock and chemists initially found 3 boron hydrides of particular interest. Diborane ( $\text{B}_2\text{H}_4$ ) was a gas and served as the building block to create higher boron hydrides. Pentaborane ( $\text{B}_5\text{H}_9$ ) was a liquid at room temperature, while decaborane ( $\text{B}_{10}\text{H}_{14}$ ) was a solid. Scientists found these substances to be strong reducing agents. Diborane would spontaneously combust/explode in air, and fires involving either diborane or

pentaborane, with typical green flames, were extremely difficult to extinguish. Odors were repulsive, and the boron hydrides were observed to be toxic by skin contact or inhalation. Who would want to deal with such substances? After WW II jet engine and rocket scientists were attracted to boron hydrides because of their greater heat of combustion than jet fuel – about 50% more on a gram for gram basis. There were hopes of pentaborane-powered jet bombers able to fly 2,000 MPH. Even the SR71 Blackbird was considered for being fueled by pentaborane. Because of its tendency to ignite and produce a super-hot green flame, pentaborane became known as the *green dragon* by those who worked with it. John Clark authored a wonderful book on the history of rocket propellants (**Figure 3**) that I have long recommended to those who enjoy chemistry, explosions and toxicology, as many propellants are well-known for their toxicity (boron compounds, hydrazines, chlorates, fluorine,  $\text{NF}_3$ , chromium salts, etc.).

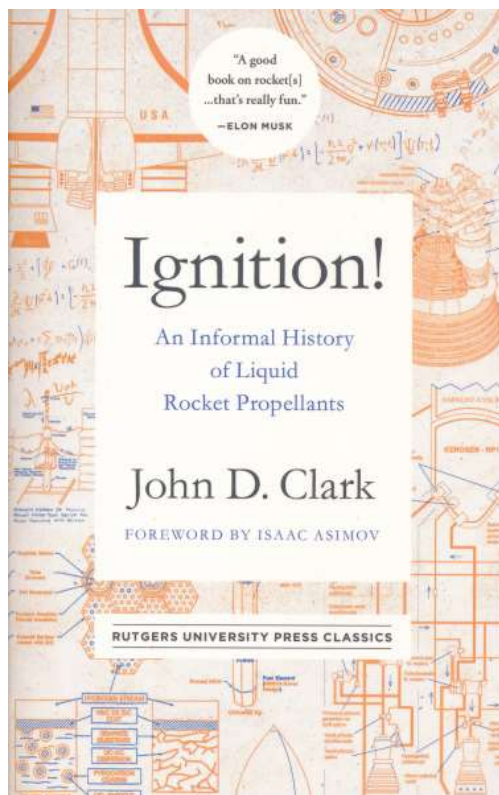


Figure 3

Post-WW II, government/military funding resulted in partnerships with private industry to construct various production facilities for boron hydrides and other boron compounds. These compounds initially made their way to jet and rocket engine test centers and military bases. Despite prolonged and extensive efforts, boron hydrides and other boron compounds (e.g., boron nitride, alkyl boron hydrides) were abandoned as fuels, in part because of density, freezing points, temperatures of combustion, and most importantly, deposition of boron oxides in turbines and rocket engines. However, triethylboron mixed with triethylaluminum (TEA-TEB) is used to ignite primary rocket/missile engines and rocket stages today, producing a green color with initial combustion (**Figure 4**).



Figure 4 – Ignition with triethylaluminum/triethylboron  
<https://www.nasa.gov/exploration/systems/sls/sls-acoustic-testing-2014.html>

Diborane is still produced and, as we'll see, mainly used in semiconductor manufacturing, with some use in rubber vulcanization, hydrocarbon polymerization and other processes.

When pentaborane production was abandoned as a rocket or jet fuel, industry and governmental/military entities found themselves in possession of more than 200,000 pounds of the hydride. Some was distributed to universities or other facilities to be used for research. Large numbers of cylinders were placed in storage or buried. Extensive efforts have since been made to recover all cylinders of pentaborane for secure storage and/or safe destruction because of both physical and profound toxicological hazards (**Figure 5**). Some cylinders remain unaccounted for, and cylinders continue to be discovered.

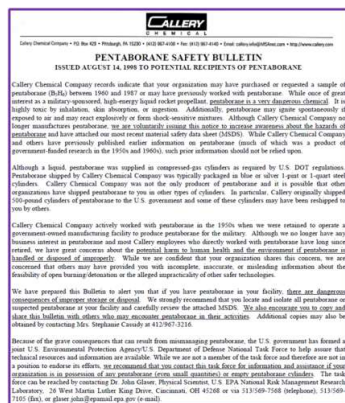


Figure 5 – Pentaborane safety bulletin  
<https://media.msanet.com/www/PDFs/MSANorthAmerica/SafetyNotices/Pentaborane081498.pdf>, 1998.

Jeff has spent a significant portion of his career tracking down, sometimes excavating, and safely destroying cylinders of pentaborane. He has authored a wonderful paper (**Figure 6**) describing the history of pentaborane production, accidents, and methods of destruction that I highly recommend for the interested reader.



Figure 6 – [Pentaborane Disposal](https://www.scribd.com/document/15062569/Pentaborane-Taming-the-Dragon-pdf)

(<https://www.scribd.com/document/15062569/Pentaborane-Taming-the-Dragon-pdf>)

## Diborane

Diborane is a thermodynamically unstable, pyrophoric, colorless gas with an odor described as repulsive, sickeningly sweet, rotten eggs, or musty (**Figure 7**). The odor threshold appears to be 2 to 4 ppm. Air Liquide is the sole U.S. manufacturer of diborane.

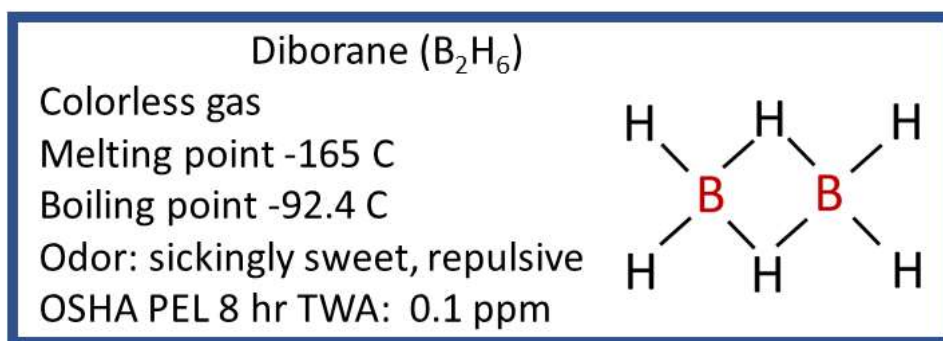


Figure 7

Diborane is supplied in cylinders for industrial use (**Figure 8**), most commonly diluted in an inert gas. While some is used for rubber vulcanization and other processes, the great majority of diborane is used in semiconductor manufacturing. Ion implanting (see the post on arsine) utilizes small amounts of diborane as a p-type dopant. The biggest use is for production of borophosphosilicate glass (made from oxygen, silane, diborane and phosphine), which protects the silicon substrate and conductive paths in semiconductor and other devices.



Figure 8 – 5% diborane in helium

Diborane can spontaneously combust in air or when in contact with other substances. Explosions and fires may be limited to cylinders and the immediate area (**Figure 9**).



Figure 9 – Melted valve on a cylinder of diborane after

However, a large explosion at a specialty gas producer in South Korea in April of this year speaks to the hazards of working with this hydride. Note the typical green color of combustion involving a boron compound (**Figure 10**).



Figure 10 – [Diborane explosion](https://www.youtube.com/watch?v=_ktaO5nYK4Q) ([https://www.youtube.com/watch?v=\\_ktaO5nYK4Q](https://www.youtube.com/watch?v=_ktaO5nYK4Q)).

Apart from an explosive/fire hazard, diborane is absorbed by inhalation and, if dissolved in a solvent, through skin contact. The major toxic effect is delayed-onset respiratory irritation with delayed-onset pulmonary edema confirmed in animal studies. A 4-hour exposure to 50 ppm diborane kills about 50% of rats from pulmonary edema and pulmonary hemorrhage, with toxicity comparable to that of phosgene.<sup>1</sup> Cordasco reported 26 cases of acute diborane inhalation over 5 years in workers, and 18 men developed respiratory symptoms, comprising chest tightness, dyspnea, cough, and wheezing lasting from 3 to 5 days in the majority of cases.<sup>2</sup> Ten percent developed nausea, anorexia and hypersalivation. Treatment is that of supportive care.

There is potential for formation of pentaborane and other boron hydrides within a diborane cylinder. This is discussed in a separate section, below.

## Diborane Differential Diagnosis

The main toxicologic differential diagnosis for causes of delayed onset respiratory symptoms, including pulmonary edema, such as that produced by diborane, has mainly centered on phosgene, nitrogen dioxide, and nickel carbonyl. Phosgene is used in chemical syntheses, but also can arise whenever fire or flame involves a chlorinated substance (e.g., when welding steel degreased with a chlorinated solvent). Phosgene is colorless and its odor is said to be that of freshly mowed hay (**Figure 11**).

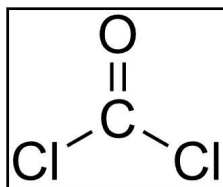


Figure 11 –  
Phosgene

Nitrogen dioxide is a red/brown gas that most commonly arises from nitric acid reacting with numerous other substances, including various metals. Below is a cloud of NO<sub>2</sub> rising from a spill of concentrated nitric acid onto steel plating near Phoenix (**Figure 12**). Of course, acute inhalations of extremely high concentrations of diborane, phosgene or NO<sub>2</sub> can produce rapid onset of symptoms and lung injury, although such exposures are exceptional.





Figure 12 – NO<sub>2</sub> formed from spill of concentrated HNO<sub>3</sub>

Nickel carbonyl is a highly volatile liquid formed during the Mond process of nickel smelting when carbon monoxide reacts with impure nickel (**Figure 13**). Inhalation can produce delayed onset pulmonary edema with a flu-like syndrome.<sup>3,4</sup> Medical toxicology fellows should remember that diethyldithiocarbamate is a chelating agent used to treat patients with nickel carbonyl poisoning.

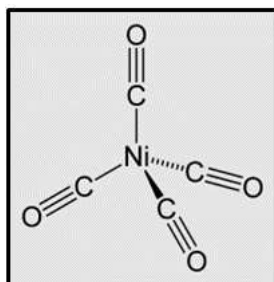


Figure 13 – Nickel carbonyl

## Pentaborane

Pentaborane is a colorless, volatile liquid with a vapor pressure that increases to 215 mm Hg at room temperature, making it many times more volatile than water. The median odor threshold in humans is 1 to 3 ppm in air. But like H<sub>2</sub>S, the ability to detect the odor decreases with exposure (**Figure 14**).

<b>Pentaborane (B<sub>5</sub>H<sub>9</sub>)</b>	
<b>Colorless liquid</b>	
<b>Melting point -47 C</b>	
<b>Boiling point 60 C</b>	
<b>Vapor pressure 171 mmHg @ 20 C</b>	
<b>Odor: garlic, acetylene-like, slightly sweet, sour milk, pungent</b>	
<b>OSHA PEL 8 hr TWA: 0.005 ppm</b>	

Figure 14

Pentaborane was produced and stored in cylinders of various sizes, ranging from those that could be easily held in hands to those containing hundreds of pounds of the hydride (**Figures 15 and 16**). Pentaborane cylinders were pressurized with nitrogen so that liquid pentaborane would be expelled when the valve was opened; nitrogen also prevented air from entering the cylinder. Pentaborane is pyrophoric with a lower flammability limit (LFL) of <0.5% in air, while the upper flammability limit (UFL) has not been measured. Pentaborane is absorbed from skin, but most commonly by inhalation.



Figure 15 – Pentaborane cylinders pressurized with nitrogen



Figure 16 – Pentaborane cylinders in storage. Photo from U.S. Army Corp of Engineers.

Numerous studies have examined pentaborane inhalation in animals. All mice inhaling 20 ppm for 1 to 2 hours died with convulsions, tremors, and restlessness. Similar findings were found in rats, demonstrating more severe toxicity than HCN at similar concentrations.<sup>1,5</sup>

Most human toxicity, apart from explosions, has resulted from pentaborane production or from efforts at destruction of pentaborane cylinders. Pentaborane toxicity mainly comprises a neurotoxic syndrome, and depending on the degree of acute exposure, onset of symptoms may vary from several minutes to greater than 40 hours. Early symptoms commonly include nausea, lethargy, personality changes, headaches and dizziness. More severe poisoning produces myoclonus, encephalopathy, and recurrent seizures. While chronic exposures during manufacturing procedures have resulted in some neurotoxicity, the greatest concern has been for acute poisoning from acute or subacute exposures.

Yarbrough reported on 3 workers, one with a very intense exposure, that serves to exemplify the potential for severe toxicity.<sup>6</sup> Twenty-one cylinders of pentaborane were unearthed at a construction site where they had been buried about 20 years earlier. Cylinders were placed inside a thick-walled steel destruction cylinder, and the cylinders were pierced, allowing contents to be purged into an alkaline solution for chemical destruction. During a malfunction contents remained in one cylinder, and one worker came in dermal contact with pentaborane. Generalized seizures began only 4 minutes later. En route to the hospital he experienced shallow respirations and remained comatose, and on arrival in the ED he had no BP or pulse. Sinus tachycardia, atrial fibrillation, and profound metabolic acidosis were noted. The EEG was flat 6 hours after exposure, and he eventually died.

A second employee was exposed to pentaborane vapor that entered an adjacent building. He was conscious and transported to a hospital. En route, he developed myoclonic jerks and complained of dyspnea. On arrival to the ED he experienced a cardiac arrest. He was shocked to sinus rhythm, but experienced frequent seizures and metabolic acidosis (pH 6.41). After sodium bicarbonate and resolution of seizures, acidosis did not recur, suggesting acidosis was not a direct effect of pentaborane. After 2 weeks of coma, he could withdraw from painful stimuli. Four months later he regained consciousness, but had difficulty speaking. At 6 months, he was spastic and displayed poor coordination. CT scan showed cortical atrophy, and he remained institutionalized.

A third employee was only briefly exposed to pentaborane by inhalation while working as an assistant to the first employee and briefly entering the building where the second employee was working. On the way to the hospital, he also developed myoclonus and then experienced generalized seizures. In the ED he was confused, agitated, hallucinating, and had another seizure. BP remained satisfactory, pH was 7.02 and lactate was 16 mEq/L. Yet another seizure ensued. No further seizures occurred, and acidosis resolved. The patient was discharged on day 11 without neurological deficits. Both the second and third employee developed rhabdomyolysis, typical of recurrent convulsions.

Mindrum described another typical case of delayed-onset neurotoxicity in a 27-year-old man who experienced onset of fatigue 6 hours after pentaborane exposure.<sup>7</sup> The next day he remained tired, but developed weakness, agitation, numbness, and blurred vision in the afternoon. That evening (the day after exposure) he appeared intoxicated and, two hours later, developed recurrent generalized seizures. Between convulsions he was confused. Seizures were treated with barbiturates and resolved the following day. He was discharged 4 days after exposure, experienced residual lethargy for a week, and then recovered.

The cardiovascular effects, metabolic acidosis, and rhabdomyolysis in patients described by Yarbrough can be partly or completely explained by hypoxia from recurrent seizures and coma with respiratory depression. The mechanism by which pentaborane produces CNS toxicity remains unknown, and no antidote is known. Treatment is entirely supportive.

Of note, methylene blue, a weak oxidizing agent, has been given antidotally to rabbits who received intraperitoneal injections of decaborane (a solid) dissolved in corn oil.<sup>8</sup> Animals treated with continuous IV methylene blue infusions survived longer. The clinical relevance remains unknown.

## Diborane to Pentaborane

Being thermodynamically unstable, pure diborane decomposes slowly to higher boranes and hydrogen at ambient temperature, with acceleration of the reaction as temperature and pressure increase. Thus, over time, a cylinder of pure diborane may come to contain pentaborane (liquid) and decaborane (solid) as well as lesser amounts of other higher boron hydrides.

Pure diborane that is shipped in the U.S. is limited to a cylinder fill amount so that if all the diborane decomposes, the operating pressure of the cylinder will not be exceeded. Pure diborane in cylinders, then, is limited to filling only 7% of the cylinder's volume. An important exception is that the US Department of Transportation may issue a special permit that allows a 28% fill if the cylinder is packed in an insulated metal drum filled with dry ice (**Figure 17**). The drum must be delivered to the customer within 10 days. If not, the supplier must immediately conduct a search to locate the drum and cylinder.



Figure 17 – insulated drum containing dry ice and diborane cylinder

Most cylinders of diborane provided to end users in semiconductor or chemical industries contain relatively low concentrations of diborane (e.g., 5%) in an inert or flammable gas, limiting the amount of decomposition to higher boranes. Sometimes mixed metal hydrides are provided in a cylinder, such as those containing both diborane and phosphine. Shipments of *pure* diborane are delivered to gas specialty companies who will mix diborane with other gases for end-user customers. It is important to remember, though, that removing valves and cleaning/testing diborane cylinders has potential for some exposure to higher boranes such as pentaborane.

## Delayed-onset CNS Toxicity Following Inhalation

Medical toxicology fellows should be familiar with a differential diagnosis for delayed-onset central neurotoxicity following acute or subacute inhalation exposures. Several additional substances capable of producing such toxic responses immediately come to mind and are briefly described, below (**Figure 18**). You may think of additional agents.

**Delayed-Onset CNS Toxicity After  
Acute or Subacute Inhalation**

Tetraethyllead  
Trimethyltin  
Dimethylmercury  
Ethylene oxide  
Methyl halides  
Nitriles

Figure 18 – Examples of agents capable of producing delayed-onset central nervous system toxicity after acute or subacute inhalation

### Tetraethyllead

Tetraethyllead (TEL) has mainly been used as an additive to fuels to prevent preignition (knock) and to lubricate valve seats (**Figure 19**).

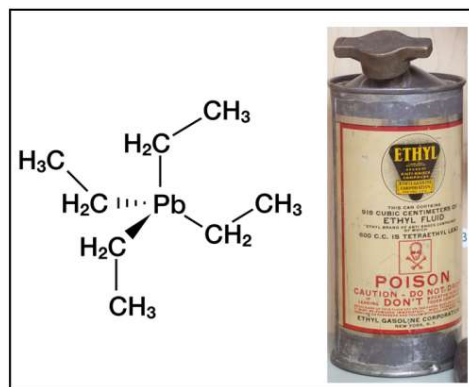


Figure 19 – [Tetraethyllead structure and early 20th century container](https://airandspace.si.edu/collection-media/NASM-NASM2011-00560)  
 (<https://airandspace.si.edu/collection-media/NASM-NASM2011-00560>). Structure: Wikipedia creative commons.Container: Smithsonian Air & Space Museum

Alkyl lead compounds are very lipophilic, and after crossing into the brain, undergo oxidation and concentration. Beattie described 4 men who cleaned a tank of aircraft fuel containing TEL.<sup>9</sup> A few days later they experienced agitation, insomnia, nausea, confusion, headache and delirium. Blood lead concentrations were elevated. Machle noted that onset of symptoms following inhalation of TEL were typically 3-12 hours post-exposure, and seizures were possible. Apart from acute exposures, chronic exposures during manufacturing (or recreational inhalation of leaded gasoline) are well-recognized for CNS toxicity. In 1925 Hamilton reported that over a 17-month period, 11 men died and 1 man was declared insane from TEL poisoning in a production facility.<sup>10</sup> In another 28 cases of TEL poisoning, the most frequent findings were insomnia, nausea, weakness, vertigo, headaches, and annoying dreams.

### Trimethyltin Chloride

Alkyltin compounds are used as biocides and in organic synthesis, especially in radical chain reactions. Alkyltin products are commonly found as chlorides in industry. Trimethyltin chloride melts at about 38°C with a significant vapor pressure, allowing for inhalation (**Figure 20**).

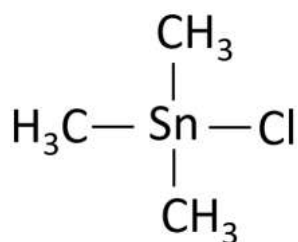


Figure 20 – Trimethyl tin chloride

Rey described 6 men who cleaned a container containing dimethyltin dichloride and trimethyltin chloride. Exposures varied, with the maximal being nine 10-minute exposures over 3 days.<sup>11</sup> After latent periods of 1-3 days, workers experienced onset of headache, tinnitus, deafness, forgetfulness, disorientation, aggressiveness, psychosis, syncope, coma and respiratory failure. Partial complex seizures were suspected in some cases. One patient died 12 days later in coma, ARDS, shock, and with hepatic dysfunction. Autopsy revealed cerebral edema with degeneration of the amygdala. A second patient developed fixed neurological deficits comprising extrapyramidal findings and hyperkinesias and remained wheelchair-bound 4 years after exposure, unable to feed himself. A 3rd man was left with severe memory deficits and remained aggressive. Three others recovered over 6 months.

Feldman and others reported on a 23-year-old chemistry student involved in a laboratory explosion involving alkyltin who experienced burns to the head, neck and chest without loss of consciousness.<sup>12</sup> Three days later he developed delirium and memory deficits. Blood trimethyltin

concentrations were elevated. Five months later he required treatment for partial complex seizures. Four years after the event he still displayed cognitive dysfunction and impaired memory.

## Dimethylmercury

Delayed onset CNS toxicity is well-described following ingestion of alkylmercury compounds, such as methylmercury chloride. However, dimethylmercury is a liquid at room temperature (**Figure 21**).

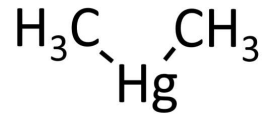


Figure 21 –  
Dimethylmercury

In 1998 Nierenberg and others reported on the death of a chemistry professor who presented with neurological complaints months after what was reported to be a single exposure to a drop(s) of dimethylmercury (**Figure 22**).<sup>13</sup> Laboratory and postmortem studies confirmed elevated mercury levels. Topical exposure was emphasized as it should have been, given the very low vapor pressure of this substance. But the authors noted that some exposure may have occurred through inhalation, though this would seem to be a minor route of acute/subacute exposure in most instances.

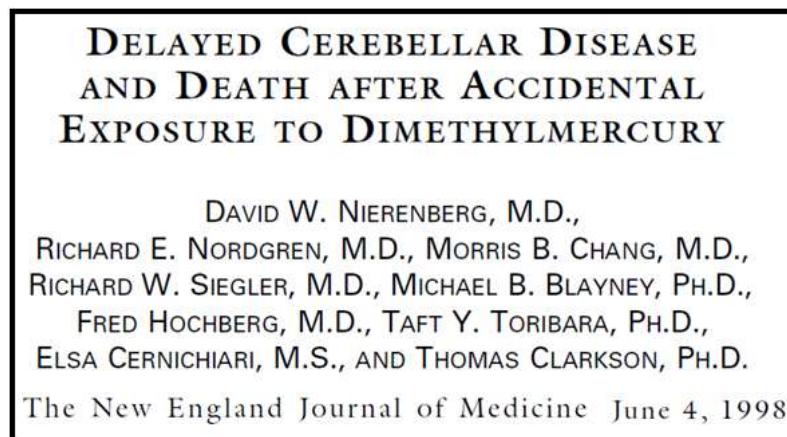


Figure 22

## Ethylene Oxide

Ethylene oxide is an alkylating agent that exists as a gas at room temperature (**Figure 23**). It is used extensively for gas sterilization and as a common intermediate in the chemical industry, such as for the synthesis of ethylene glycol.

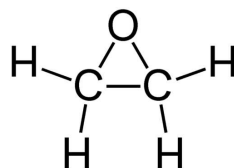


Figure 23 – Ethylene  
oxide

Chronic exposures are reported to produce peripheral neuropathy, nausea/vomiting and mucous membrane/skin irritation. Acute or subacute exposures have rarely been reported to produce dramatic but transient CNS toxicity. Gross described a man performing gas sterilization while using a sterilizer that was leaking ethylene oxide.<sup>14</sup> At the end of a work shift he developed headache, vomiting and lethargy followed by recurrent generalized seizures for two days. After one week he appeared to have recovered. Salinas and colleagues described a 43-year-old

woman who dropped an ampul of ethylene oxide and noted “fumes” around her.<sup>15</sup> Nausea developed within 2-3 minutes, and she left the area and became light-headed before passing out; workers found her in a generalized seizure sometime later. She awoke but was reported to seize two more times prior to what appeared to be a full recovery.

## Methyl Halides: Methyl Bromide, Methyl Chloride, Methyl Iodide

Like ethylene oxide, methyl halides are also alkylating agents. Methyl bromide gas has been used for fire extinguishing, as a refrigerant, and for fumigation (**Figure 24**). Methyl bromide is still used for soil fumigation.



Figure 24 – Methyl bromide (bromomethane). Cylinders:  
US Department of Agriculture

*Methyl bromide* inhalation can result in delayed and profound neurotoxicity. As examples, Hine reported 10 cases of methyl bromide toxicity, including a man who ate lunch in a work area into which methyl bromide had leaked.<sup>16</sup> That evening he experienced a headache, and 9 hours after exposure developed a generalized seizure followed by gasping respirations, tachycardia and death about 20 minutes later. Prain and Smith described 8 boys who hid in a small boat and discharged a fire extinguisher containing methyl bromide.<sup>17</sup> Two boys felt ill the next day at school, and both collapsed with generalized seizures. The other 6 boys were located on the boat and findings included seizures, confusion, and coma. Six boys died, some with pulmonary edema.

*Methyl chloride* has also been used for fumigation and as a refrigerant (**Figure 25**).

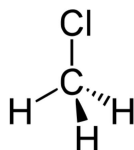


Figure 25 –  
Methyl chloride  
(chloromethane)

In the late 1920s, Kegel noted that there were 29 cases of methyl chloride poisoning in Chicago with 10 deaths over a 1-year period, mainly resulting from leaks in refrigerators in apartments.<sup>18</sup> Initial symptoms were typically drowsiness, confusion, stupor and vomiting which would then progress to convulsions and coma prior to death. Hiccups and tremors were common. Legge commented that there was a distinct delay in onset of symptoms after exposure to moderately low concentrations.

*Methyl iodide* is a colorless liquid used as a methylating agent in the chemical industry (**Figure 26**).

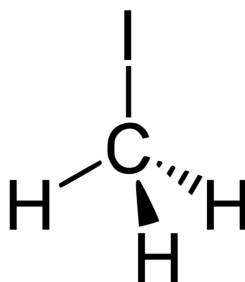


Figure 26 – Methyl  
iodide (iodomethane)

After acute to subacute exposures there is an onset of encephalopathy with ataxia, blurred vision, and slurred speech which can progress over hours to days to lethargy and semi-coma, sometimes with quadriparesis.<sup>19</sup> Impaired memory and parkinsonian findings may appear along with paranoia. Dysmetria, ataxia, dysarthria and nystagmus may last for weeks or more, but can resolve in some instances. Other victims appear to be permanently affected.

## Nitriles

Acetonitrile and acrylonitrile are liquids at room temperature (**Figure 27**). Nitriles are used as solvents and in chemical manufacturing as well as for production of synthetic textiles and fibers.

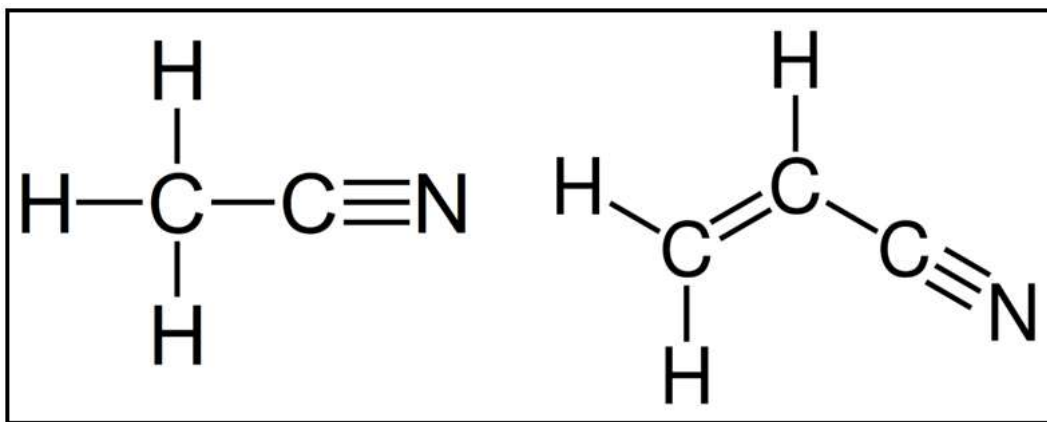


Figure 27 – Acetonitrile (left) and acrylonitrile (right)

Both compounds are gradually metabolized to HCN after absorption, producing delayed-onset signs of cyanide poisoning, which include seizures, coma, and other neurological findings. Muraki reported a man who washed the inside of a reactor kiln with acetonitrile for an hour while wearing a facemask and with fresh air being pumped into the reactor.<sup>20</sup> Fifteen hours later he suffered onset of vomiting, weakness, and diarrhea. More than 20 hours after exposure he experienced a convulsion and subsequent coma with profound metabolic acidosis.

Acrylonitrile has been used as a pesticide for lice and bed bugs in Germany, and occupants of fumigated dwellings have experienced delayed-onset coma, seizures, and death.<sup>21</sup>

## Conclusion

Diborane consumption in semiconductor manufacturing continues to expand, and respiratory complications of inhalation along with risk of injury from fire and explosion will continue to be a reality. Pentaborane toxicity will be a rare event, but the delayed-onset profound neurotoxicity, including potential for permanent impairment, must never be forgotten in the event old cylinders are discovered, which is very possible. Again, under some conditions, diborane cylinders will contain pentaborane, as well. If you happen to run across a pentaborane cylinder, don't touch it, call the authorities, and give Jeff a call for some advice.

This completes the series on metal hydride gases. I am considering topics for the next Fellow Friday post, so if anyone has any suggestions, please let me know in the comments.

## Postscript

Fellows, here are some questions you should be able to answer for the boards.

1. Tributyltin is used as molluscicide, including in marine paints. What is its toxicity? And what is the main toxicity of triphenyltin?
2. Diethyldithiocarbamate has been used to chelate nickel in patients with nickel carbonyl poisoning. This agent is a metabolite of what prescription drug?



3. In what over-the-counter beauty products was acetonitrile found before being removed following deaths of children after exploratory ingestions?
4. Finally, contrast the chemical structures and odors of phosgene, phosphine, arsine, and diborane.

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