The study of poisoning from arsenic trioxide and copper acetoarsenite (Paris green) in industry is largely a study of skin lesions, and these run all the way from inflammation, with or without ulceration, to scleroderma, melanoderma, warts, and a slowly developing cancerous degeneration which usually has a fairly benign course and does not give rise to metastases. (See page 438.) Loss of hair and trophic changes in the nails are common. Some interesting cases have been reported due to the handling of paper dyed with arsenic green, as, for instance, one reported by Ayres (32) in the foreman of a paper works. This was a case of scleroderma, with arsenic in the urine. A similar case in the out-patient clinic at the Massachusetts General Hospital was traced to arsenic-containing paper which was pasted on skyrockets.

The picture of generalized, arsenical poisoning from solid arsenic, as seen in exceptional cases in industry, rarely is severe. Neuralgic pains and multiple neuritis with motor palsy, slight in degree, are characteristic of these cases. The palsy is likely to affect the long extensors of fingers and toes, as does lead, but it is distinguished from the latter by the severe neuralgic pains and by the tendency toward a multiple, symmetrical neuritis. A very rare form of industrial poisoning involving the optic nerve was reported by Moleen (838), who described a case of skin lesions and double optic atrophy in a man using arsenical insecticides.

Of 135 patients having arsenic poisoning by solid compounds of arsenic, discussed by Legge (682) 38 had gastric symptoms, 13 had tremors or muscular cramps, 6 had peripheral neuritis; of the remainder all had lesions of the skin, in 6 of whom there were keratoses or epithelioma. Legge speaks of 3 cases of epileptiform seizures, a most unusual manifestation of such poisoning.

There is, however, a general consensus that serious cases of injury from white arsenic or Paris green are extremely rare in industry. It is hard to explain the difference between industrial arsenical poisoning and nonindustrial, but there is a difference and it is noted in all the industrial countries. With hydrogen arsenide gas, the story is quite different. The lack of serious symptoms in industrial arsenic poisoning is not to be explained on the ground of slight exposure, for in the great smelting works of the West, the lead works in Colorado, and the

copper works in Montana the ores contain a high proportion of arsenic, which is volatilized in the furnaces and carried in the flues to be deposited in the bag-house and tunnels as white arsenic. Sometimes in Colorado smelting works as much as 60 per cent of the flue dust is arsenic trioxide. This dust must be shoveled into trucks and transported for a second sublimation, after which the product, almost pure, must be handled and packed; yet the men carrying on this work almost never show any but local effects on skin and mucous membranes. Years ago, in 1912, a case of generalized polyneuritis came to light in a Montenegrin working in a western smelter when the arsenic content of the ore was high. There used to be also occasionally among Paris-green packers cases with gastrointestinal symptoms and neuralgia and even one fatal case with fatty degeneration of the liver came to light.

Arsenic is given as the cause of 6 cases of poisoning and one death in England in 1924, all the subjects being employed in making or handling sheep dip. One man, 44 years of age, suffered from epithelioma of the left side of the face, after employment in contact with arsenic for twenty-six years. The others had ulceration of the septum, dark pigmentation of the skin around the neck and axillae, and irritation of the skin. The history of the fatal case is scanty, we are told only that there was swelling of the eyelids, intense vomiting, and that arsenic was found in the organs.

Among the unusual sources of arsenic poisoning are: in glass manufacture the emptying of sacks of white arsenic, adding it to the glass mixture; the stitching of window shades dyed with arsenic green; the mixing of arsenic with enamel for iron bathtubs, shot-blasting such enamel from defective ware (Australia); the handling of hides and bird skins that have been treated with white arsenic.

Of course it is impossible in these industries to protect the nostrils and throat entirely from arsenic without putting each worker into a protective pressure mask, which is not always practicable. However, the skin and eyes can be protected against the dust and every effort should be made to do this; otherwise there will be painful ulceration, especially of the skin of the scrotum, the lips, and the nostrils.

Lead and Calcium Arsenate. A great deal of study has been devoted of late to the toxic action of the arsenates, lead and calcium,

in medicolegal cases. The early symptoms are those of anoxemia; the later symptoms are largely those caused by the effort of the body to excrete the debris of red cells which clog the liver and kidneys (Stadelmann [1122]). Thus the acute symptoms will be followed in four to six hours by the passage of dark urine or bloody urine, and this is often the first symptom that alarms the victim. Later, in twenty-four to forty-eight hours, jaundice appears, and a blood count shows a loss of red cells which may fall below two million. As the disease progresses, jaundice deepens and the urine grows scantier until just before death there is complete suppression (Jones [574]). The British industrial physicians report these cases under the head of toxic jaundice. Arsenic can always be detected in the urine and may continue to be excreted for many weeks after the acute symptoms have disappeared (Wignall [1245]). The anemia is likely to persist even longer.

The source of this dangerous gas is always some process in industry which causes the formation of nascent hydrogen in the presence of arsenic; in other words, a process which reproduces the conditions of Marsh's test for the detection of arsenic, and there are many industrial processes in which these conditions may be present. In making Marsh's test, zinc dust and hydrochloric acid are used for the production of hydrogen. That is the way hydrogen is produced for filling balloons, and many cases of poisoning have occurred in such work because zinc is very commonly contaminated with arsenic; in fact it is difficult to get arsenic-free zinc. Lead-burners who use an oxyhydrogen flame may have to produce their own hydrogen, and then they run this same risk. Most of the iron, lead, and antimony used in industry is likely to be contaminated with arsenic, as is also commercial sulphuric acid made by the ordinary chamber process and the hydrochloric acid which is made from it. In the production of aniline dyes and coal-tar drugs, one of the most important processes is reduction by means of nascent hydrogen, which is produced from iron dust or zinc dust and hydrochloric acid, and here there is always a risk that arsine may be formed. Five severe cases of this form of poisoning occurred in a New Jersey plant making dye intermediates during the First World War. In one of these the body of the victim was bronzed to a mahogany color, according to the physician's report.

The most frequent cause of poisoning from arsine in this country is

the cleaning out of tanks and tank cars that have contained a heavy acid, either hydrochloric or sulphuric. Strong acid does not attack iron or steel tanks, but weak acid does. Therefore there is no trouble while the tank is full, but when it is emptied and flushed out with water, weak acid is left in the residue which is called "sludge," and when the laborer or lead-burner goes in to dip out the sludge or to make repairs, the arsine formed by the action of the weak acid on the arsenic-containing iron rises and poisons him. Three men in a plant in New Jersey were sent into an acid tank to make repairs after it had been supposedly well-flushed out. There was, however, about a bucketful of residue left at the bottom and this contained enough arsine to poison the men, two of whom died. In some cases it was the galvanized iron dipper that was responsible, and accordingly the great chemical works in Germany prohibit the use of zinc-covered vessels and make the men wear gas-masks while they work.

There is a general belief in industry that pickling metal, *i.e.*, treating it in tanks of sulphuric or hydrochloric acid, hot or cold, is unhealthful work, and it seems probable that small amounts of arsine formed from time to time in such tanks may be responsible for this. Muehlberger, Loevenhart, and O'Malley (*856*) describe a case of arsenical poisoning, with multiple neuritis, in a man employed in pickling iron and steel. Legge reported 7 cases in 1902, the acid containing 0.035 per cent of arsenic or 350 parts per million. In the Navy Yard at Puget Sound two men were severely poisoned by the arsine evolved from a pickling tank. One died after thirteen days' exposure; the other had nephritis and anemia. The man who died was in a coma for four days, with anuria, hemoglobin under 40 per cent, red cells about one million, brownish pigment in the skin, edema of the lungs, and albumin and blood in the urine. The acid in the tank was used over and over again so that the content of arsenic rose from 0.0005 per cent to 0.05 per cent.

The same sort of accident is possible in the charging and the forming departments in storage battery manufacture, where lead antimony grids are immersed in weak sulphuric acid. Epidemics of poisoning with arsine occurred during the First World War in two Italian submarines and one English, and in each the gas was traced to the storage batteries. It was found that the acid had eaten into the lead anti-

exposures of two, eight, and thirteen years respectively. The result in the man with only two years' exposure was complete healing with no deformity; in the one with eight years', recovery but loss of all teeth of the lower jaw and part of the left mandible with some facial deformity. The third case and the one slowest to develop was much more serious. In this man the upper jaw as well as the lower was involved, with loss of a large portion of the palate bone and the formation of fistulous tracts leading from the mouth to the nose and nasal accessory sinuses. At the time of writing, eighteen months after the onset, the condition was still active.

Heimann discusses methods of protection against the fumes of yellow (or white) phosphorus. The red allotrope does not seem to be poisonous. Since yellow phosphorus burns when exposed to the air, it is always handled under water and is transported from one receptacle to another in liquid molten form under water. At this stage small particles will separate from the flowing mass, rise to the surface and volatilize (or burn). Local exhausts must be provided wherever this may occur.

A still more recent industry which carries with it the danger of phosphorus poisoning is the electrothermal process for the production of elemental phosphorus to be used in the preparation of phosphate fertilizer. Fleming Miller, and Swayne (327), of the Tennessee Valley Authority, attempted to determine the threshold of physiologic tolerance when white phosphorus, or its suboxides, are administered in small doses over a long period. They found significant changes in the bones of animals given phosphorus by injection under the skin. The limit of tolerance seemed to lie betwen 0.05 mg. of phosphorus per kilogram of body weight and 0.8 mg. given daily.

The practical impossibility of providing complete protection against the action of phosphorus is seen in the factory inspectors' reports from English and German match factories when yellow phosphorus was used. Workers had to be forbidden to keep a glass of water on the work bench, to chew gum or eat an apple, or put a finger in the mouth, or pick the teeth; but in spite of the efforts of sanitary engineers, physicians, and dentists, cases persisted in appearing.

It is noteworthy that all three of the victims reported by Heimann had had a dental examination within one month of the onset of the disease and had been pronounced normal.

The match now made is the safety match, for which amorphous red phosphorus is used, and the "strike anywhere" match, made with the sesquisulphide of phosphorus; neither of these has the poisonous properties of white phosphorus. In a French match factory Nicolas and his colleagues (878) found that there was a great deal of skin disease due to the handling of phosphorus sesquisulphide. There was a rapidly developing erythema with formation of vesicles or pustules accompanied by severe subjective sensations and, sometimes, by conjunctivitis. Greasy skins were most susceptible. An eruption was produced in volunteers by the use of the paste and also the powder. A wash of potassium permanganate and sodium bicarbonate caused prompt recovery.

Cases of phosphorus poisoning, however, have been reported from other industries, chiefly in the production of phosphorus, in the chemical industry, in the manufacture of phosphor bronze, and in making lights for miners' lamps. In Great Britain most of the cases of phosphorus necrosis coming to the knowledge of the Factory Inspection Department have occurred in persons employed in the production of phosphorus. Copper ores sometimes carry phosphorus.

A rare source of phosphorus poisoning is the accidental formation of phosphoretted hydrogen (phosphine, PH_3), in the production of acetylene gas from carbide for use in autogenous welding. Several cases have been reported from Germany[1] from this source and it is of course possible that cases have occurred in this country but have not been recognized because it is a poison unfamiliar to American physicians. There is another source of poisoning from phosphoretted hydrogen which may or may not be of occupational character, that is the escape of PH_3 from ferrosilicon in the same way that the formation and escape of AsH_3 occurs. Either or both of these gases may be formed, but in the larger number of instances it is the latter that has been held responsible. Only analysis of the fumes would settle this question, although the absence of a hemolytic action would point to phosphorus.

[1] Thiele. Zentralbl. f. Gewerbehyg. 9: 94, 1921.