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Forty Years in the Poisonous Trades

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I AM afraid that the title of my paper may give the impression that I mean to indulge in the vice of old age and waste my time on tales of the far-distant past. I will not do that but I cannot resist the temptation to give you of the later generation something of a picture of what industrial medicine was 40 years ago. For only if you look at it can you appreciate the remarkable record your country has made in that really brief period, a record starting in ignorance almost complete, and willfully so, to a position of what we may almost call world leadership, the countries which for so long led us being now unable to keep pace because of war destruction and poverty.

When I entered this field in 1910, after two years study of all the available English and German material, industrial medicine did not exist in the U.S.A. and industrial surgery was rather discredited. I think you could have counted the published articles on industrial poisoning on the fingers of one hand. There were of course men in industry, foremen especially, who had a lot of practical experience, and a few doctors really studied their cases, but the prevailing attitude was ostrich-like. American factories were so much better than European, American workmen so much better housed and fed than European, that the industrial diseases described by foreign authorities simply did not exist over here. I remember the shocked surprise I felt when I visited the model town of Essen, after I had seen South Bethlehem.

The most difficult obstacle I found was the universally held doctrine that the only way to prevent industrial poisoning was to keep the skin clean, to scrub the hands and especially the fingernails. I have been reading over some of the papers I wrote before the First World War and in all of them I plead with physicians and employers to give up the clean fingernail doctrine and face the fact that a lead worker eats only three times a day and even then he does not wash his hands in his soup or coffee, but he breathes 16 times a minute and if there is lead in the air, he will get it no matter how often he scrubs his nails. But it was uphill work, for I was trying to upset a cherished myth. I remember the head surgeon of a great Colorado smelting company saying in a public meeting: "It is not the lead a man absorbs during his work that poisons him but what he carries home on his skin." I remember the physician in charge of a Utah smelter telling me he always instructed the men—blast furnace tenders, flue and baghouse cleaners—to scrub their nails carefully.

At that time we believed that the lead which was inhaled reached the stomach by being mixed with the saliva and swallowed and the only reason why men working in fumes and dust were intoxicated so much more rapidly and severely than those handling solid lead (a fact noted by Tanquerel des Planches almost a century ago) was that the quantity entering the body was greater. K. B. Lehmann, then one of our greatest authorities, thought he had proved this by a few questionable human experiments. It was not until J. C. Aub made his studies in the twenties that we understood the difference between absorption

through the gastro-intestinal tract, with its many natural defenses, and that through the almost completely defenseless respiratory tract. These studies really revolutionized the attitude of physicians and of practical men towards the protection of lead workers. For many years I believed that the body cleanliness story was a thing of the past and then not long ago I picked up a publication of one of our governmental industrial hygiene outfits, and read: "In the prevention of benzol poisoning the most important element is bodily cleanliness." Sometimes it seems as if a lie crushed to earth will rise again. Imagine a man who has to breathe benzol fumes protecting himself by bathing.

The earliest method of protection against severe exposure to an industrial poison was to shorten the time of exposure and the first such measure of which we know was introduced in 1556 in the Austrian mercury mines of Idria. The working day was shortened by law to six hours. In Spain, in the great quicksilver mines of Almaden as late as 1921 30 hours constituted a month's work and in England in 1899 strippers of the white lead in white lead works were forbidden to do that work on more than two successive days. Practically the only method of prevention I found before the First World War in lead smelting and in the production of white lead and in roasting oxides was to rush the dangerous jobs and then discharge the men. It was always a matter for the foremen to decide. The management knew nothing about it except that there was an enormous labor turnover. But in those days that was a matter for pride. Foremen would boast of having to bring in from 30 to 50% new men every pay day. In that way, they thought, not many would get severely leaded. Of course there were always plenty of newly arrived immigrants to fill the ranks.

Even now the shortening of exposure may be adopted as the only practical way of avoiding serious trouble. There are emergencies, such as accidental spillage, or breakage, or unusual repair work, which have to be dealt with at once. It may be that the only possible way to manage is to let no individual work in the poisoned air for more than a few minutes.



Dr. Alice Hamilton, Cummings Lecturer for the Ninth Annual Meeting of the American Industrial Hygiene Association, is the world's foremost woman industrial hygienist. Whenever workers have been subjected to poisonous materials in their trades, Dr. Hamilton has brought the facts to light by visiting the plants and talking with the affected workers. She has continually fought for control of industrial health hazards through legislation, by convincing management to take action, and through education. Her investigations have included the lead industries, viscose rayon production, potteries, steel mills, mining.

The first woman on the faculty of Harvard Medical School, the only woman to serve on the Health Committee of the League of Nations and to receive the American Public Health Association's Lasker Award for combatting causes of disease and death, Dr. Hamilton is author of several books on industrial toxicology. She is an active member of the Committee on Permissible Limits of Dusts, Gases, and Vapors of the American Standards Association. Her work over the past forty years has done much to demonstrate the necessity of vigorous, scientific control of poisonous materials incident to the occupation.

The Cummings Memorial Lecture is given annually by an outstanding personality in the field of industrial hygiene in commemoration of Donald E. Cummings, Past-President of the American Industrial Hygiene Association, killed in a plane crash en route to control an occupational disease situation jeopardizing production of a critical war material.

Two subjects that in the early days bothered us a great deal were the special danger to young people from industrial poisons and the special danger to women.

As to the first, the great susceptibility of youth is admitted and the Federal Children's Bureau has for some years examined the industries employing minors and secured *gentlemen's agreements* to rule out minors from possibly hazardous jobs. When it comes to the over-susceptibility of women, a theory which has prompted some quite foolish legislation, careful observation of large groups in the poisonous trades has not given proof of it. We still hold that plumbism is more harmful in a woman because it is a poison to the germ cell and although that is true too of the male germ cell, still in the woman there is the additional injury to the fetus by the lead in the mother's blood while she is carrying the child. This is probably true also of mercury. As to benzol we still believe that it is more harmful to menstruating girls than to young men, though we are not so strongly convinced now as we used to be. Certainly there is no ground for forbidding women to work in jobs that expose them to carbon monoxide or to silicosis. Statistics seem to point the other way.

Lead

It is in the industrial control of lead poisoning that we see, I think, the greatest advance over the situation that obtained in this country up to the First World War. Let me give briefly a few pictures of the chief lead industries as they were then, reminding you that whatever statistical information we had was based on personal surveys including interview with physicians, labor leaders, social workers, druggists, and on hospital records, not on routine medical examination of the individuals in lead work.

The production of white lead and of the oxides was the most notorious of the lead trades and in every center it was possible to pick up reports of palsies, encephalopathies, deaths from severe intoxication. That industry has been revolutionized, the dusty processes controlled, the workers kept under medical supervision. In the old days, Philadelphia and Chicago had so many cases of plumbism from white lead

and red lead plants that Cook County Hospital and old Blockley listed them under the name of the plant, not under lead poisoning—that was taken for granted.

Pottery glazing and decorating was another notorious source for we used white lead unchanged in the glaze and the colors and made no effort to control the dust. It was then that I came across my first cases in women, dippers' helpers and lithotransfer makers, and convinced myself that women are more susceptible than men to cerebral plumbism. Then I went to England and Germany and saw what could be done with good exhausts and with wetness everywhere. I saw how the English changed soluble white lead in the glaze by fritting it to insoluble disilicate. We had in 1922 a rate of 22 to 23% of plumbism among potters, the English had a rate of 0.9%. Now we frit our glazes and we practice good housekeeping in our potteries, so Ohio, New Jersey and West Virginia are no longer happy hunting grounds for the student of plumbism. An equally great reform has occurred in the making of sanitary ware. That used to seem to me the worst of the lead trades. The enamellers scattered a powder containing up to 20% soluble lead over red hot iron sinks and bathtubs, the men and all surfaces were dust-covered. In one Pennsylvania plant employing 130 enamellers, doctors and hospital records showed that in two years there had been 72 cases of severe plumbism, with five of palsy, two of encephalopathy and three deaths. Now we use a leadless enamel on clay bathtubs and sinks.

The rubber industry was once a source of severe forms of plumbism, because lead oxides and the basic carbonate were important compounds and men in the compounding rooms and on the mills were exposed to soluble lead in the form of very fine dust. Now lead has almost vanished from the rubber industry.

The old notorious lead trades, painting, printing, plumbing, which gave rise to the expressions "painters' or printers' colic and palsy," are no longer important in this respect. The newer coatings, enamels, lacquers, plastics, are lead-free, titanium dioxide displaces white lead to a great extent in paint, and interior decoration is done with leadless paint. Printing plants

are far better equipped and largely mechanized, plumbers hardly come in contact with lead except for rare jobs.

Yet lead still holds an important place in the list of industrial poisons for though it vanishes from an old trade, it may suddenly appear in a new one. Painting automobiles with many coats of lead paint gave way to lead-free lacquers, but when streamlining came in, the lead problem made a dramatic reappearance for the surfaces of the car must be absolutely smooth and this means that seams must be covered with lead solder and then sandpapered and smoothed. The scrapping of steel structures by the use of intense heat is an increasing source of danger. Recently Cotter described some 300 cases of "lead intoxication by inhalation" in men scrapping the elevated railroads in New York by using blow torches on the steel which had had many coats of red lead paint. Even more dangerous because it is often carried on in enclosed places and with extreme degrees of heat is welding, which is more and more replacing riveting. Tetraethyl lead is an ever-present danger, though scrupulously safeguarded now. And the trade which in all industrial countries yields the greatest number of cases of plumbism, the making of storage batteries, is not only still with us but increasing in importance. It is a very difficult problem for the industrial hygienist. I have never seen a plant which was entirely safe, though the English system of keeping everything dripping wet has much in its favor. Belknap of Milwaukee puts his mixers, pasters and finishers into positive pressure masks, but I believe that even this drastic system is not entirely foolproof. We are told that a new kind of lead-free storage battery, developed by the French, is being introduced in this country but since it uses cadmium, it is rather like leaping from the frying pan into the fire.

There are questions still unsettled with regard to lead poisoning in spite of the enormous amount of study which has been devoted to the subject: the possible relation between lead poisoning and peptic ulcer, between lead poisoning and multiple sclerosis, arteriosclerotic kidney, generalized arteriosclerosis. Here the English are doing the most significant work. for they

need not depend on animal experiments, they can follow human lead workers over a long period of years, men and women who worked in the lead trades as much as 50 years ago before sanitary control was perfected, and who at the end show the effects of what the English believe to be slow, prolonged poisoning. The English law accepts as occupational in character chronic nephritis with or without general arteriosclerosis in an old lead worker.

Unfortunately for us, during the years when lead poisoning of a severe character was prevalent in this country, no real interest was taken in its study by physicians. It was not till hygienic reform was well under way and severe poisoning controlled that we began intensive research.

Mercury

THE old familiar poison, mercury, has held its place in industrial toxicology since Roman days when the quicksilver mines of Spain were exploited, but the industry most notorious for its disastrous effects has always been the making of felt hats. We do not know when mercury nitrate was first used to help in felting fur but we know that when the Huguenots fled from France after the Revocation of the Edict of Nantes in 1685, they carried with them the secret of mercurial carrot, as we call it, the French still call it *le secret*. *Hatters' shakes* and *mad as a hatter* are old terms for the mercurial tremors and psychoses of chronic poisoning as seen in this trade in every country.

Studies of mercurialism in the hatters' trade in this country began with one published by J. Addison Freeman of Orange, New Jersey, in 1860. He described some 100 hatters of Orange, who were victims of typical mercurial poisoning, but 15 years passed before the next record appeared, quite as shocking as the first. Nothing was done about it, little attention was paid, none by the medical world. Then in 1912, 34 years later, the public was startled by a report made by a lay organization, the National Civic Federation which had made a survey of the mercury-using trades in New Jersey and New York. Of course the cases of poisoning the survey brought to light were only the severe, conspicuous ones. Of these 94 were found,

among them men incapacitated by palsies, four with complete paralysis of a limb, many cases of polyneuritis, two with insanity.

Now the situation in the felt hat industry is completely changed. This historic trade, full of traditions and established habits, has at long last abandoned the use of mercurial carrot and substituted mixtures containing sulphuric and nitric acids, and hydrogen peroxide. The great reform was accomplished in a way typical of our country, not by law but by agreement between manufacturers and state and Federal Public Health Services, just as was the control of radium in industrial use and of tetraethyl lead. It is the only way a quick and effective reform can be brought about in several different states simultaneously. Thus an old and notorious source of industrial sickness has been abolished, we hope completely.

Foreign literature is full of cases of mercurialism traced to the use of mercury vacuum pumps in making electric light bulbs. Nowadays we use these very seldom, never in electric bulb manufacture. Yet mercury is still an important industrial element, especially in the production of dry batteries, in making thermometers and barometers, in extracting gold and silver from the richer ores and in the electrical industry. High frequency induction furnaces, the constant potential department, the manufacture of tungsten and molybdenum rods and wires, all call for the use of quicksilver. And now a new source of poisoning, sometimes acute and severe in form, has appeared, namely the use of organic compounds as fungicides. The dimethyl compound of mercury seems to be the one most often used here, but a Canadian report mentions diethyl mercury. The symptoms as described by English, German, and Canadian writers differ from those caused by the inorganic compounds. There is no mention of gingivitis or salivation, the central nervous system is chiefly involved, peripheral nerves, cerebellum, cerebral cortex, and the onset is usually rapid. The most striking cases come from Germany. Koelsch writes of 22 fatal cases he was called on to investigate during the war. These were in a chemical plant where mercury ethyl acetone and mercury ethyl

bromide were produced for use as fungicides. He writes that the illness was characterized by profound cerebral involvement. One is reminded of the similar difference between the action of inorganic lead and of organic, tetraethyl lead.

Carbon Monoxide

FORTUNATELY for the industrial worker carbon monoxide poisoning has always

sation, and many have involved persons of the upper classes who had the best of medical care. As to whether chronic CO poisoning is a clinical entity there are still varying views. It is admitted by all that CO is not a cumulative poison for it is rapidly eliminated but there is evidence that repeated attacks of anoxia bring about an accumulation of effects. The experiments of Lewey and his colleagues during the war point to a cumulative effect on the brain by repeated short exposures to an atmosphere devoid of oxygen. The brains of animals which have recovered promptly and without any visible injury from such exposure have shown cell damage, first of reversible nature but later of permanent, if the anoxia was repeated. Lewey also exposed dogs to the standard concentration of 100 p.p.m. for five and a half hours a day, six days a week, for 11 weeks. The dogs seemed to continue in perfect health but as early as the second week electrocardiograph changes appeared and if they were killed, autopsy revealed degeneration of heart muscle fibres, hemorrhages and necroses. Some of the dogs showed disturbances of gait and of reflexes and histological changes were found in the central cortex and in the basal ganglia.

Phosphorus

ANOTHER old poison is phosphorus. It was deeply entrenched in the match industry for all the years between 1833 when Lorinser, an Austrian, described 22 cases of phossy jaw which had developed among match workers in the 12 years following the invention of strike-anywhere matches, and the year 1908 when European countries signed the Berne Convention by which they agreed to give up the manufacture and the importation of white phosphorus matches. In this country a very early case of typical phossy jaw was treated in the Massachusetts General Hospital in 1851, some 15 years after the manufacture of white phosphorus matches began in Springfield. This was a very severe case, ending after six months in death from toxemia. There was, however, no mention of phossy jaw in American medical literature from 1851 to 1910, when John Andrew published the report of an investigation he had made for the Federal Department of Commerce of

American match factories. We had always been assured that our factories were so superior to the British and European that phossy jaw was unknown in our country, but Andrews brought to light over 150 cases, some of them quite as severe as any in foreign countries. **AS** everyone knows, that report led to the passage of the Esch law, which placed a tax on white phosphorus matches and led to the substitution of the harmless sesquisulphide. The Diamond Match Company held the patent for this French discovery but generously threw open to the whole country the use of the process. So one notorious dangerous trade was rendered safe and we all thought that phossy jaw was a thing of the past. But eternal vigilance is the price of industrial safety.

A sudden and quite unexpected reappearance of severe phosphorus necrosis took place in 1923 when a new kind of fireworks was manufactured in three plants on the Atlantic seaboard and white phosphorus was one ingredient. The management was both ignorant and indifferent before the attention of physicians and state authorities was called to the situation. Fourteen workers, all women but one, had developed intensive necrosis of the jaw and two had died of septicemia. The period of exposure in these cases was unusually short for the exposure was very great.

That source in its turn was abolished, and years passed before we heard any more about phosphorus poisoning in industry. However, it has emerged again. In 1946 Heimann of the New York State Department of Labor found three cases of phosphorus necrosis of the jaw in men working in the production of red phosphorus, converting the yellow into the red allotrope. In spite of routine medical and dental examinations including dental x-rays, phosphorus necrosis of the jaw appeared after exposures of two years, eight years and 13 years. The man who had been exposed only two years recovered without deformity, the one who had worked eight years lost **all** his teeth and **part** of the left mandible, with some facial deformity; the case which was slowest in developing was much the most serious. This man lost a large portion of the palate bone and there were fistulous tracts leading from mouth to nose

and nasal sinuses. These were still active after 18 months. Heimann traces the trouble to the bubbles which form and burst from the water over the yellow phosphorus, for this must always be handled under water since it burns in the air.

Finally there is a still more recent source of trouble from this very troublesome element, although so far no human injury has been reported. This is the electro-thermal production of elemental phosphorus to be used in the preparation of phosphate fertilizer in the T.V.A. Fleming, Miller and Swayne of the T.V.A. have attempted to find the maximum allowable concentration of phosphorus by subcutaneous injection of animals, but such results are of little value in solving the problem of workers' protection.

Heimann's cases seem to show the practical impossibility of providing foolproof protection. These men had all had a dental examination within one month of the development of the necrosis. In the days when white phosphorus was still used for making matches the English took elaborate precautions in their factories. Workers were forbidden to keep a glass of water on the work bench, to chew gum or tobacco while at work or to eat an apple or put a finger in the mouth or pick their teeth. Strict cleanliness of benches and provision of fume removal were required of the management, yet phossy jaw continued to appear and apparently it always will so long as that subtle and relentless poison is used.

A few metallic poisons which were formerly little used or used in ways not endangering health have suddenly emerged with prominence and have begun to show ominous characteristics. The most important are aluminum and beryllium.

Aluminum

RECENTLY Koelsch of Munich, for many years the foremost authority in Germany in this field, has sent me abstracts of German reports on the effect of aluminum dust, reports which were not accessible to us during the war and which are of interest not only in connection with aluminum workers but also with men working in mines which are dusted with aluminum for the prevention of silicosis. Koelsch sent me abstracts of four papers which de-

scribe what the authors consider a definite characteristic picture of aluminum dust poisoning in men working in an atmosphere of finely divided particles. The symptoms consist of short breath, lessened vital capacity, stabbing pains in the chest, slight cough and expectoration. The special feature all note is the youth of the victims (older men are less susceptible) the shortness of exposure, the rapid course of the disease, (the latter two in sharp contrast with silicosis) and the fact that the process may progress after exposure ceases. Two cases of pneumothorax are described, attributed to great increase of intra-alveolar pressure, the aluminum dust impeding expiration. Two autopsies are described, the findings in which agree strikingly: extensive interstitial pneumonia with induration and shrinking. The interstitial tissue had undergone hyaline-collagenous degeneration. One author speaks of a silvery, metallic sheen on the cut surface of the lung. According to Koelsch there was an abnormal dust exposure in these factories, caused by war-time conditions, the lack of proper exhausts, the obstructed windows, necessitated by the black-out.

Beryllium

THE newest of the industrial poisons of practical and rapidly increasing importance is beryllium. For the description of its sources, uses and action I am indebted to Dr. Henri Hardy for I have had no experience at all in this field. It appears that descriptions of pulmonary lesions following exposure of workmen to dust or fumes of beryllium compounds were published in Germany, Italy and Russia during the thirties. The most extensive discussion was that of Gelman of Moscow who wrote the section on beryllium for the I.L.O. Encyclopedia. Observing not only what he called "disseminated injuries to the lung" but irritation of the mucous membranes and the skin, he wrote that it was not easy to separate the action of beryllium fluorides from that of other fluorides. However, in Germany the picture in the lungs described by Gelman was found in workers exposed to beryllium compounds free from fluorine. The picture was one of disseminated sclerotic changes with x-ray findings resembling military tuberculosis. Clinically there was

cough, reduction of chest expansion, mild dyspnea, with disproportionately severe physical findings of pulmonary involvement.

The first report of an occupational disease in the beryllium industry in this country came from the Cleveland Clinic in 1943. These first American cases were reported by Van Ordstrand, Hughes and Carmody in men producing beryllium oxide from beryl ore. The authors called the affection chemical pneumonia. The following year saw a similar report from Pennsylvania, this time from an industry employing a fluorescent powder which contained beryllium carbonate and manganese silicate. Then the Cleveland group came back with an extensive report of an illness which they boldly called beryllium poisoning, and of which they described 38 cases with five deaths. Briefly summarized, the symptoms were those of chemical pneumonitis, cough, dyspnea, substernal pains, anorexia, loss of weight, increasing fatigue. The onset was insidious, lasting some three weeks, after which a characteristic, diffuse bilateral haziness appeared in x-ray pictures of the lungs. Removal from exposure and rest in bed with oxygen, as indicated by cyanosis, was followed by recovery, but if then the man returned to work there was a relapse and the fatal cases were in part due to pulmonary edema following too early exertion. The autopsy findings were those of bilateral, acute, organizing, atypical bronchopneumonia. All the Cleveland cases were in men exposed to beryllium oxide in processing beryl ore.

In 1946 Hardy and Tabershaw published their report of 17 cases of what they called "delayed chemical pneumonitis" in 14 women and three men working in the fluorescent lamp industry and using zinc manganese beryllium silicate. Here we have a different clinical picture, one of delayed onset, the symptoms appearing from six months to three years after the victim had left the workshop, then dramatic dyspnea, great loss of weight and a poor prognosis. Six of the 17 died and the other 11 were disabled from 18 months to over four years with only one known case of complete recovery. The pathologic lesions in the lung were those of an atypical granulomatous process replacing normal tissue, and similar lesions were found in liver, spleen and lymph nodes.

A very strange feature of the Massachusetts experience is the discovery of three cases, two of them fatal, in individuals who were associated with, but never went into a plant making fluorescent lamps. These, in history and autopsy findings, seem to belong to the same group as the 17.

Since the publication of this report Dr. Hardy has had case records sent to her from seven different states, including Massachusetts. By April 1947, there were 40 cases of delayed chemical pneumonitis known to the Massachusetts Division of Occupational Hygiene in workers in the fluorescent lamp industry. Since some 1500 workers have been exposed, this would mean a case incidence of 2.5% but obviously that figure is far too low, based as it is on the more or less accidental discovery of fairly advanced cases.

The reported cases of beryllium poisoning therefore have come from plants producing and casting alloys, making and using fluorescent powders. No dependable reports have as yet been made of the amount of beryllium in the air of working places where the poisoning occurred. Chemical tests seem quite inadequate to reveal small amounts but a spectographic analysis, developed in the Kettering Laboratory in Cincinnati and in the Massachusetts Division of Industrial Hygiene, promises to give results.

The latest contribution in this field. January 1948, comes from Martland and his colleagues. They discuss four fatal cases, one acute, the others chronic, and include one which dates back to 1934 and has only now been recognized as a case of chronic beryllium poisoning in a worker in the fluorescent lamp industry. A most interesting addition to our knowledge is the proof of the presence of beryllium in the organs of two and in the skeleton of one of these two. This was shown by the spectographic method in the Kettering Laboratory.

The importance of this new poison is growing rapidly, for it is an excellent ingredient for alloys, which are light and resistant to strain, it is a good refractory for firebricks and is used not only for fluorescent lamps but for many other electric appliances. To anyone who, like me, can look back to the early days of industrial medicine, this story of beryllium poisoning

is an amazing thing. Here, in the space of some **six** or eight years a form of industrial poisoning which was highly dubious, involving only a small number of cases, not included under compensation laws except in full-coverage states, observed by a small number of physicians, was promptly made the subject of thorough study with full publicity. A conference was held in Boston at which the late Leroy Gardner of Saranac agreed to undertake animal studies with beryllium and last year a symposium was held in Saranac where the data were assembled from all over the country and discussed. When I look back on the days when we were urged not to mention such a thing as TNT poisoning lest we drive all workers out of the plants, I can hardly believe; it is the same country.

Cadmium

CADMIUM is another industrial poison whose use has lately increased, but our knowledge of its action has been fairly extensive for some time. However, the increasing use for alloys and for electric plating and recently for the production of storage batteries has added to the data formerly acquired. According to Fairhall the danger of cadmium poisoning in the electroplating process is slight. It is in the application of heat to the plated surface with the production of the oxide that the danger comes. The occurrence of cadmium poisoning in smelters of ore containing this element was noted by the German, Stockhausen, as early as 1656 and thoroughly discussed by Tracinski of Upper Silesia in 1888. By 1946 Fairhall was able to summarize the histories of 59 reported cases of acute cadmium poisoning with a mortality of 15%. The action of the cadmium oxide is chiefly on the lungs, and resembles that of nitrous fumes, with constriction of the chest, increasing dyspnea, cough, nausea and abdominal pain, bronchopneumonitis, cyanosis and pulmonary edema.

As for chronic cadmium poisoning there has been a wide spread scepticism but recently some French reports have come from plants producing storage batteries by the new method. A supposedly **slow** action of cadmium appeared after five to 14 years' exposure and began as obstinate pain in the lower back and legs which increased till

walking became impossible. By x-ray, certain bones, chiefly scapula, ilium and femur, showed lines of pseudofracture. Anemia of the iron-deficiency type was the only other definite finding except of a yellow discoloration of the teeth. Hardy and Skinner reported five cases of what they suspected to be chronic cadmium poisoning in men exposed to concentrations of **0.6 to 6.8 mg.** per 10 cu.m. The illness was subacute, with anorexia, nausea, vomiting, epigastric pain, fatigue, *dopeyness*, tooth decay, low hemoglobin values. The most recent work is that of Princi of the University of Colorado, who is studying workers in a cadmium smelter where the electrolytic method is used and the men are exposed to dust of the oxide. The concentration of cadmium oxide is sometimes as high as 31.3 mg. per 10 cu.m. He confirmed the French experience as to a yellow line on the teeth and also low hemoglobin values and low red cell count.

The experimental work of Prodan in 1932 is still the most valuable in that field, since it was an attempt to duplicate industrial exposure. Both the oxide and the sulphide were used and both are toxic, the latter less so because of its relative insolubility. The attack is chiefly on the respiratory tract causing a rapidly developing pulmonary edema or pneumonia, if the course is less rapid. A fibrotic thickening of the alveolar walls occurs, which is permanent. In animals these lasting lesions followed even brief exposure to the fumes. Liver and kidneys show fatty degeneration, both organs retaining much of the ingested cadmium.

Although not in the field of industrial toxicology one must not omit mention of the large number of cases of acute poisoning resulting from cadmium-plated eating utensils. According to Fairhall up to 1941 only 21 such accidents were reported, but since then no less than 268.

A curious fact noted by Church and Yrinci is worthy of mention. This is the apparent antagonistic action of selenium on cadmium as shown experimentally and confirmed in practice. In one cadmium smelter the workers were free from any sign of poisoning although the concentration of cadmium in the air was up to 2000 or 3000 times the maximum allowable con-

centration. The air of this plant has small traces of selenium.

Chrome

THE local action of chromic acid droplets and chromate dust is very familiar to workmen and has been for over a century. Chrome ulcers, chrome holes, perforated nasal septa are certainly troublesome but never really disabling and we still feel certain that such lesions do not undergo cancerous change, for they have been under observation for over a century. But lately reports have been coming from Germany of cases of pulmonary carcinoma in men exposed to chromate dust, not many cases, it is true, but numerous enough to make such authorities as Koelsch and Teleky believe that there is a causative relation. So far no such reports have been published in this country, but of course we must expect the same experience, that is observed in other countries invariably turns up over here. That the cases so far reported all occurred in men exposed to chromate dust, not to droplets of chromic acid, is probably due to the greater precautions taken against the very irritating action of the acid.

Benzene

TURNING now to the organic compounds, no industrial poison except lead and carbon monoxide has had the amount of scientific study devoted to it that has coal tar benzene, because it is so valuable a solvent and at the same time so dangerous to use. The history of the coal tar solvents, benzene, methyl benzene which is toluene, and dimethyl benzene or xylene, is fairly recent in the United States. Before 1914 we did not produce these solvents from coke. The valuable by-products of coke furnaces simply escaped into the air and we bought what benzene was needed from Germany. Then came the war and the blockade, the demand from the allies for explosives which require benzene, which was used in the rubber industry and in the hastily started aniline dye industry. So coke by-products plants were built and suddenly there was a wide spread use of coal tar solvents and their derivatives. It was only to be expected that cases of industrial poisoning began to appear, for medical men knew

nothing of the effects to be looked for and sanitary engineers had no idea of the danger points to be avoided. I remember a worried du Pont chemist telling me he could not understand why a worker should be fatally poisoned when there was no more than 5% of benzol in the air where he worked.

The study made by the National Safety Council under C-E. A. Winslow started us on the right road and soon American laboratory studies and observations in the field widened our understanding and led to a marked fall in the incidence of poisoning. But the greatest advance was made when latex came in as a substitute for the various solutions of rubber. Here, as in the case of white phosphorus matches and of carbon disulphide vulcanization and of mercury-free felt hat carrot, a safe substance takes the place of a poison. Latex is the gum of the rubber tree and instead of coagulating it and then dissolving it for dipped, spread and cemented rubber goods, it is shipped as a liquid in tankers and used as a liquid.

Unfortunately benzene has many other uses for which latex is not a substitute and it requires unremitting vigilance on the part of the industrial physician if he would avoid trouble. The more recent studies, especially those made by the Boston and New York groups, of workers exposed to benzene have shown that the earliest sign of intoxication is not always, as we used to believe, a drop in the white cell count or even a low polymorphonuclear count, but it may be a drop in the red cell count with the appearance of large erythrocytes, a macrocytic anemia. Another important addition to our understanding of the action of benzene we owe to the Boston and New York studies. No longer do we believe that the attack of benzene is on one element only of the bone marrow, rather we insist that any abnormality in the blood picture, if it appears in a benzene worker, must be treated as a warning of danger. That is the only safe course to follow.

Still another important finding is the fact that marrow aplasia is not an invariable finding in severe benzol poisoning, indeed hyperplasia is the characteristic finding in many cases. These observers suggest that the reason why we have believed women to be much more susceptible to benzene than men may be that cases with hyperplastic

marrow were rejected, and it is true that one can find in the older literature instances of such a decision. My experience with compensation boards bears this out. Nevertheless I still believe that benzene is especially dangerous to menstruating women.

The entrance of the methyl radical removes from toluene and xylene this special action of benzene on the bone marrow and several extensive investigations made during the war serve to confirm the comparatively harmless nature of these two solvents.

As to the benzene derivatives which have found a place in industry, their name is legion and our knowledge of their action in many cases is founded only on animal experiments. Even more numerous and more complicated are the derivatives of the petroleum series, the chlorinated hydrocarbons, so valuable because they are non-inflammable, a group of which the chlornaphthalenes are the newest members; the glycols, which are bivalent alcohols; the cyclohexanes and hexanols; dioxan, which is diethylene dioxide. These are all of increasing importance and call for continued observation in practical use.

Methanol

FORTUNATELY methyl alcohol is no longer a cause for apprehension. Our history in regard to that poison is unique and not very creditable. First, under the influence of the wood alcohol producers and the total abstinence advocates, we forbid the use of revenue-free grain alcohol, so that the use of wood alcohol was strongly entrenched in American industry at the time when all other industrial nations used denatured grain alcohol. Then when Prohibition came in and reports of blindness and death from bootleg liquor shocked the public, we went to the other extreme, forbidding even the addition of wood alcohol to industrial grain alcohol as one of the denaturants. Wood alcohol still carries its bad reputation and when it is used, elaborate precautions are taken, precautions one would like to see applied to more dangerous liquids.

Carbon Disulphide

CARBON disulphide is an industrial poison which was for many years curiously

neglected by the American medical profession. I came across it first in 1914 when I was studying the rubber industry. The German, the Italian and the French literature were full of discussions of this poison to the central nervous system, for carbon disulphide was an essential element in the rubber industry in those countries. England did not use it as much and we Americans still less. But we did at that time use it for the vulcanization of thin rubber goods and I discovered cases enough in Akron. Not through physicians, who seemed never to have heard of it, always only through foremen. Yet the cases of sudden maniacal attacks and then of more slowly increasing motor paralysis were dramatic enough to attract attention.

Fortunately this vulcanizing agent was given up by the rubber people soon after the First World War, but then it came back in greater force when viscose rayon appeared and it spread far more widely than it ever had in the rubber trade. Yet as late as 1938 this industrial poison was practically ignored by American doctors. I found that the insane asylums to which the victims were sent, recorded the cases with extreme caution as due to "some exogenous poison" or "etiology unknown" or "possibly occupational in origin." In those rare instances when compensation was sought, it was apparently always possible to secure the services of a professor of neurology or of a Psychiatrist to testify that there was no such thing as carbon disulphide intoxication. The only explanation for this ostrich-like attitude is that the plants were largely situated in states with very poor factory inspection departments and still poorer compensation systems.

Carbon disulphide is still essential in the production of viscose rayon but now the plants which used to ignore the danger are admirably safeguarded and routine medical examination serves to detect the trouble fit an early stage and remove the victim from further exposure. Trouble there will always be so long as carbon disulphide is used, for there is no way of detecting in advance which individuals will prove to be oversusceptible. One good feature of the poison is that both the central and the peripheral nerve injury are reversible. The tragically severe cases which we found 10

years ago were usually in men who were **allowed to go** back to their former jobs and of course that would never happen now.

Synthetic Rubber

ONE of the newer industries is the making of synthetic rubber which was of very great importance during the war and is now firmly established. Fortunately it was developed so recently that it had no bad practices inherited from the past and from its inception the industry has been much under the eyes of competent physicians and sanitary engineers so that the risks and the way to control the risks have been studied thoroughly. Of course there is no telling when our extremely capable and inventive industrial chemists will introduce new compounds, indeed already the list of organic accelerators and anti-oxidants is formidable, but the solvents are, mostly familiar to us, the coal tar solvents and the chlorinated hydrocarbons.

According to Mallette of the Firestone Company there are five principal types of artificial rubber manufactured in the United States. The toxic materials which are new are **not** many and **few** of them present a serious danger. The following have been tested on animals and on men: acrylonitrile, which is an organic cyanide, vinyl cyanide, and has the well-known cyan action; butadiene, a mild narcotic with an irritating effect on the eyes and the air passages, sometimes affecting the lungs, but with no cumulative action; monomeric styrene which is more productive of discomfort to human beings than either butadiene or toluene. The irritating effect on eyes and nose is supposed to give ample warning of danger, a rule which sad experience with other little known poisons has proved to be unreliable. In animals, heavy exposure to monomeric styrene causes immediate death from a primary action on the central nervous system, delayed death from pneumonia. Such a finding means that a job of repairing or cleaning apparatus may be fraught with great danger. The fourth one is chlorobutadiene, known as chloroprene. It has been found to be decidedly toxic to animals, causing depression of the nervous system, a low blood pressure and severe degenerative changes in the organs.

Then there is a long list of old familiar

poisons used as ingredients or as solvents for synthetic rubber, besides those already mentioned.

Prevention

IT IS now an accepted duty of the industrial physician to examine ~~for~~ **employment all new** men and assign them to various kinds of work according to their physical condition, but **such examination will never weed out the over-susceptible. Only a close watch over the newly employed during the early months will do that for, as is true of the infectious diseases, the victim of lead or mercury or nitrous fumes may be a physically perfect specimen whose idiosyncrasy cannot be detected in advance. Frequent re-examination during the first months is the only way the physician can detect and remove from exposure such a worker.**

This brings us to the question of alternative workers in dangerous jobs. When I was in England studying the storage battery industry, I found that they laid great **stress** on this method of preventing plumbism. We have never used it here to ~~any extent. The~~ workmen object to it, understandably. Of course we **do remove a man from** a toxic job but we do not send him back to it after a spell at other work. Moreover we have been told recently by the du Pont physicians that they have given up removing from the job men who show, on cystoscopic examination, a condition leading to what we still call aniline tumor of the bladder. This is because they found that the **process keeps on** after exposure ceases, unless properly treated. They leave the man at work under treatment.

Although no measures of personal hygiene will **save a worker** from occupational disease if the conditions of work are bad, yet there are certain rules of hygiene that are decidedly useful. Both human observation and animal experiments have shown that the presence of milk in the stomach inhibits to a decided degree the absorption of **lead**. British and German employers have for half a century had the habit of **providing milk for lead workers** the first thing in the morning or in the middle of the morning and afternoon. Studies of carbon tetrachloride therapy for hookworm infestation have shown that a **negative calcium balance** from **lack of calcium in the**

food encourages a toxic reaction, also that it is possible to defend the liver from damage by a diet rich in sulphur-containing proteids. German and Russian writers hold that the obese are more susceptible to benzene than the thin and that fatty food favors poisoning. This was confirmed by the experiments of Li and Freeman who showed that a diet low in proteins and high in fat increased the action of benzene on rats and on dogs. The effect of alcoholism is notoriously bad on poisoning from lead, mercury and TNT, and pharmacologists found that to be true of carbon tetrachloride **also**.

Foulger, Director of the Haskell Laboratory of the du Pont de Nemours Company, emphasizes the importance of proper food for workers in the poisonous trades. He has found that such workers need an optimal content of vitamin in their daily food, particularly of vitamin C and the B group. It may be simpler and more effective to administer these vitamins than to try to control the eating habits of the men and women in these jobs. Foulger quotes Viscount Chetwynd, manager of a TNT filling factory in the First World War, as saying that the women in the factory had far more gastric disturbances than the men. On the theory that this was caused by the less substantial meals eaten by the women, the management made a deduction from their pay and fed them well. As a result, the complaints fell off from 11.3% in September to 0.7-2.0% the following January. Foulger says that the night shift needs special attention in this respect.

May I close with a few warnings:

1) The establishment of so-called maximum allowable concentrations by the A.S.A. for industrial poisons has given us valuable guidance, but do not forget that these are based on the quantity inhaled by a man breathing at a normal rate for eight hours. Over-time and heavy, hot work, making the man breathe more deeply and rapidly, make necessary a lower standard.

2) Statements as to the toxicity of an element must be taken with caution for there may be a wide difference in the different compounds. The basic carbonate of lead is **much** more soluble in the body juices than the straight carbonate; the sulphate is fairly soluble, the sulphide very slightly so. We look upon zinc compounds

as only slightly toxic, but wartime experience here and in England showed that zinc chloride in the form of fumes can be deadly.

3) Be cautious in applying the results of animal experiments to human beings. We were misled for years by accepting the picture of benzene poisoning as seen in animals where leucopenia is the typical sign, appears early and is a safe warning of intoxication, anemia is slight or absent. So for years we depended on the white cell count only and missed many cases. In carbon tetrachloride poisoning in animals, it is the attack on the liver that is conspicuous, not the kidneys, in industrial poisoning the reverse is true. Animal experiments are of course very valuable but they can never do away with the need of close observation of human beings. They **would** never have helped us to detect early mercury poisoning, with its increasing timidity and depression, or early carbon disulphide poisoning, with its irritability and gloom.

4) In making your diagnosis do not follow too scrupulously the typical picture as given in the test-books, those books, with which you will be confronted when you go before the Compensation Board. It is only in industrial cases with their unfortunate medico-legal side that such a strict demarcation line exists. The literature on blood dyscrasias is full of atypical cases but we have only lately been able to discard the typical picture in benzene poisoning.

5) Be cautious in accepting the statement of the employer as to the compounds to which your patient was exposed. Often he does not know what a trade name means—there are more than a dozen strange names for carbon tetrachloride—or he may think he is still using tetrachlorethylene as a solvent when the purveyor has without warning switched over to tetrachlorethane. Or the straight-run gasoline he formerly used has been changed to "cracked gasoline" which contains benzene.

6) Finally we must take with caution the statement that experiments with a certain new poison show that sufficient warning of danger will be given in time, by an irritating action on the eye, nose and throat. That may be true when employment is high and compensation laws adequate. It cannot be depended on when jobs are scarce or in states with poor labor laws.