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UNSUSPECTED INDUSTRIAL POISONINGS DISCOVERED IN SMALL INDUSTRIES*

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The problem of poisoning occurring in large industry is not a new one but is daily becoming more complex with the demands of our heavy chemical industrial life. However, large industries are so well equipped with medical personnel and ancillary services that a real case of occupational disability and death is very rare.

In the small industries, however, which make up more than 80% of employees, the problem of unsuspected poisoning is not rare, contrary to current medical opinion. Also, such cases are not infrequently fatal. To a large extent this is due to the fact that neither the employer nor the employee in the small industry realize the potential dangers of the materials with which they work. Also, it is unusual to find that a doctor or nurse has been asked to come in to look after the health of the worker even on an occasional or part-time basis.

Moreover, small industries have another additional factor in that the process is not too well understood by the manager-owner.

Those who are familiar with this field know that there is an urgent need here for medical supervision and advice. There is also a very real opportunity for the general practitioner and internist who with some intensive study can qualify themselves to be competent consultants to industry as a part-time service. They will not only find it remunerative, but it will give them something of the thrill of the detective who is tracking down a murderer or a potential murderer. Furthermore, when the part-time medical consultant to industry follows a definite plan of attack on the problem, he should be able to prevent disability and death as effectively as his full-time brother physician in the large industries.

Medical students are prone to think that they will have only a millionaire or at least a white-collar clientele, but all practicing physicians know that the majority of their patients work in some industry. It therefore behooves them to ask the simple but often forgotten question, what is your occupation?

The following cases of serious but unsuspected poisoning occurring in groups of workers in small plants illustrate the earnestness with which the employer and the physician must face these problems.

Whenever we consider the possibility of poisoning from toxic dusts, fumes, vapors, or gases in industry, we remember that the commonest portal of entry is by inhalation; the next is by skin absorption; rarest from ingestion by mouth.

One should again remember the point of attack on a specific organ or body system which differs with different toxic materials. In some cases the brain or

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central nervous system or even the peripheral nervous system will be involved, while in others it will be the blood-forming organs, the liver, the kidney, or the muscles themselves. Toxic involvement of the lung proper is rare, except by irritation in certain instances, contrasting the effect on the lung and lymphatic structures following the inhalation of certain non-toxic but fibrogenic dusts such as silica.

When one recalls that the circulation time from the lung to the general system is a matter of a few seconds and that the area exposed in the pulmonary alveoli is upwards of 60 to 70 square meters, he can realize how rapidly toxic materials are absorbed by inhalation thus exposing the entire body, system by system.

The doctor's first contact with patient and his family will be the presenting symptoms. Consider first, for example, the headache of carbon monoxide absorption or mild intoxication with characteristic pressure over the temples associated with dizziness and nausea. This is suggestive of the possibility of carbon monoxide poisoning in its preliminary and warning stage. Carbon monoxide exposure, moreover, occurs in the home, in the automobile, as well as at the plant, wherever there is incomplete combustion of carbon.

In any of these places the physician should not be satisfied to treat symptoms only after the disease or poisoning is full-blown. The moment he hears of a new process or suspects a new hazard, he should be eager to become familiar with all the details, both engineering and medical. Only then will he be able to help prevent the occurrence of disability, temporary or permanent. I am thus detailing the currently known facts of my most recent case to emphasize the importance of the physician's responsibility to participate in the control of his patient's working environment.

Within the week I was asked to see a young man supposed to be a malingerer who had developed just this type of headache within the first few hours after he was assigned to work on a new type of welding process. As you realize, welding of steel and its alloys is now an integral part of many modern manufacturing processes. Temperature of the arc, particularly when the hard alloy metals are being welded, often runs from 4000 to 4500 degrees Fahrenheit. Since the war it has been found that welding can be done with many fewer imperfections in the welded area if the flame is shielded by some type of an inert gas to prevent oxygen in the surrounding air from causing imperfections in the weld due to oxide bubbles. At first rare gases such as argon and helium, the so-called Heliarc process, were used as the shielding gas. This appeared harmless except occasionally when in an enclosed space the presence of ozone irritated the lung proper.

Recently it has been found that carbon dioxide is just as effective and much less expensive than the rarer gases. Therefore, you can see that carbon dioxide will soon be in great demand as a shielding gas in the welding industry. However, at the temperature of 4500 degrees, we know from tests which I have observed myself that the carbon dioxide may often break down to some extent and give off some carbon monoxide at the exact point of welding. Though this will probably be rapidly dissipated in the average large shop with ordinary moving air currents, if the work is done in an enclosed space such as a tank or down in a pit, there may be appreciable exposure to carbon monoxide. As far as my studies of this particular young man's

headache have gone, I would say he is probably not malingering but has been made uncomfortable due to a small amount of carbon monoxide or an oxygen lack with excess carbon dioxide down in the pit where he worked. In the few days he had worked he had not been seriously poisoned and there has been no residual.

Another man working in the same pit for five months but making no complaint has admitted now to having so much nausea that he had no appetite and has lost twenty pounds in the last four months. We owe a debt to the complainer who brought the danger to our attention.

When the industrial engineer and I went down into the pit where the second man was at work with the carbon dioxide shielded arc, building up the imperfections in the large steel casting with a two foot rim extending over his head, we found by holding the CO tester at nose level, fourteen inches from the arc, that the CO concentration was 50 to 100 ppm. just below the maximum safe threshold. At seven inches the concentration was 75 to 100 ppm. At five inches the concentration was 600 ppm. and at three inches, 1000 ppm. The readings at seven to fourteen inches showing 50 to 100 ppm. might be safe on a large and open floor because of dilution by air currents, but when down in a pit, in this case eight feet deep and sixteen feet square, with part of the casting making a hood over the patient's head, such diluting air currents would not be present to carry away all the CO.

Furthermore there was a large heating torch on each side of the casting which in itself would consume considerable oxygen and the CO_2 shielding equipment delivered CO_2 at the rate of 30 cu. ft./hour. While there is only a minimal amount of CO, there is additional CO_2 and reduced oxygen. This could account for an honest complaint of nausea, headache, and general "lousy feeling". This must be prevented by forced ventilation.

We do know that in circumstances of danger much more intense than this case that when an individual is exposed to the inhalation of CO_2, such as in "black damp" of an abandoned mine shaft or in the pit of a nearly empty silo he may be rapidly suffocated.

In another instance of unrecognized carbon monoxide exposure, a small shaft was being drilled through a hard-rock hill in Northern Wisconsin. It is known that after blasting, carbon monoxide is formed. In this case a young and inexperienced worker was sent in to set up drilling machinery each time that a blast had been set off. Towards the end of the first week he became confused and dizzy and developed a terrific headache. His fellow workers, three or four of them, loaded him into a car and took him home to his rooming house. They then drove away. The landlady thought that perhaps he had been brought home drunk and was not perturbed by the fact that he did not come out of his room for over 48 hours. At the end of this time she called the fire department who found that the boy was unconscious. Though they revived him quite rapidly with oxygen, he had lain unconscious so long that the carbon monoxide had interfered with oxygen, to the basal areas in the brain, resulting in what is called "central pain". This cerebral area already has a low blood supply and is very sensitive to oxygen lack of carbon monoxide poisoning. It was many months before this man was able to be relieved of his terrible headache that often required an intravenous anesthesia.

Marked weakness and collapse may confuse a physician who feels that such a case may either be a severe anemia due to an internal hemorrhage or perhaps a case of heart failure on a coronary artery basis. A few years ago I was called to see a patient who had suddenly developed a bloody urine associated with marked weakness and anemia. On the invitation of the employer I inspected a small plant employing only 4 or 5 people in a room in a second floor building in a very small country town. They did metal casting and darkening of the casting using aluminum. They placed these aluminum castings in a solution of equal parts of hydrochloric acid and water into which several handfuls of white arsenic were dumped. To indicate how disgraceful the conditions were, which would never be tolerated in a large industry, the white arsenic was kept in open paper sacks on the shelf, like flour in a bakery.

On one Saturday the castings were changed without warning to 100% zinc. The full operation took only a minute, but this was done seven or eight times over a period of two hours with no immediate symptoms. About six hours after this procedure the patient, a high school boy, began to feel nausea, weakness, and noted bloody urine. The next day I was called to investigate the case and had the operation demonstrated to me two or three times over a period of half a minute each time. I sniffed the gas which was evolved and noted the garlic-like odor. Three or four hours later I became very weak and tired out but did not pay any attention to it until twelve hours later when I noted that my own urine was also burgundy red in color. At this time I was informed that the foreman who had shown me the operation had also the same group of symptoms with the same burgundy red urine.

Those of you who recall your high school chemistry remember when zinc is dropped into hydrochloric acid containing arsenic, that free arsenious hydride, or arsine, is developed. This is twenty times as toxic as cyanide. The action of the arsine consists in laking the red cells so that there is a rapid anemia and an associated jaundice as the liver works overtime in attempting to dispose of the broken down hemoglobin.

All three patients are now alive because of the prompt use of the Bristish Anti-Lewisite, or BAL, which was developed to counteract the deadly effects of Lewisite or arsine war gas.

So much for a poison to which three patients were suddenly exposed without warning in a very small country town where there was not supposed to be any industry present, certainly no more than that of cheese making.

Now imagine another dramatic picture of a young man coughing up frothy sputum and barely able to gasp out the cause of his industrial poisoning. About an hour or two before his death, one resident was alert enough to ask him the question, what is your occupation? Five days before this, he stated, he had been asked by his foreman to use an oxyacetylene torch to clean out a cadmium tank in a small plating company. Cadmium had been used in this tank and they wished to transfer back to zinc plating. The cadmium, however, had collected in a rather large lump at one of the electrical poles. After working an hour at this with an ordinary torch, the patient complained of a slight cough and stopped the burning of cadmium. He went home never to return.

Two other maintenance men laughed at the first worker and completed the operation in another hour and a quarter. That evening they both had rather severe dyspnea but recovered after a few days.

Three days later the patient's wife called the plant and reported that he had "pneumonia" and had been taken first to one hospital and then to another because of his critical state. Five days after the original exposure the patient died.

The use of cadmium in the ordinary plating operation at ordinary temperatures is harmless, but when it is fumed by burning, the very small fume particles are inhaled deep into the lung where they have the same effect on the pulmonary alveolar membrane as inhaled phosgene. The result is a diffuse burn of the entire lining of the lung with an outpouring of fluids which cannot be told from the acute heart failure syndrome with pulmonary edema. Positive pressure applied early might have helped, but no intelligent treatment was given because no one knew the sequence of events and the occupational exposure. I myself was first informed of this case long after he had expired, and I was asked to review the whole matter and to recommend proper precautions in the plant in regard to all their industrial operations.

Now let's take another symptom complex which may confront you in the middle of the night as it did me one sultry midnight last summer. The wife of a young and vigorous man called his physician saying that he was suddenly very weak, that he had twitching of the fingers, was nauseated, was dizzy, had a headache with convulsions and had fainted. The doctor ordered him taken to the County Hospital. The resident then called me for suggestions. Inquiry as to the occupation showed that four hours previous to his collapse he had sprayed "parathion" in an enclosed greenhouse in order to kill spiders infesting roses. The patient worked one hour wearing a mask and rubber gloves, but no cap and no rubber coat as it was extremely hot. That was 5:30 to 6:30 p.m. At 9:00 p.m. he came back to open the greenhouse windows. This took him only fifteen minutes, but for so short a time he put on no mask, no gloves, and wore a tee shirt. It should be remembered that parathion, which is the civilian equivalent for the so-called war "nerve-gas", is absorbed not only by inhalation but through the skin, especially a moist hot skin. Parathion has the faculty of very efficiently inhibiting cholesterinase enzyme which normally inactivates the acetyl choline which forms at the end of nerve fibers. With the accumulation of the acetyl choline, the individual has a muscarine-like poisoning. If untreated he often will go on to collapse, unconsciousness, convulsions, and death. The only treatment is atropine given intravenously in heavy doses. Upon my advice the resident gave him a thirtieth of a grain of atropine intravenously every hour from 12:30 until 5:30 a.m. Then he was on the verge of mild atropine poisoning with dilated pupils and excitation so that the atropine was stopped. The patient then became conscious and gradually got over the parathion poisoning. Blood studied for cholesterinase showed a marked drop characteristic of parathion poisoning.

Incidentally, it took two to three more months for the cholesterinase level to approach normal. This point has to be borne in mind in chronic exposure such as occurs in spraying farms with this substance from an airplane. In the Mid-west and West there are many small companies now employing three or four pilots to spray

this type of material over fields as an insecticide. Furthermore, this material can now be bought over the counter by the small do-it-yourself gardener who I am sure has no comprehension of the danger involved.

Another interesting case is that of a young man also working on a very hot night stripped to the waist as a janitor in a small chemical plant. His job was to collect waste papers and smash them down into barrels so that they could be disposed of. On this particular night he had run out of barrels on his own floor so he went upstairs and got three or four barrels there. To pack them tight, he climbed into the barrel and jumped up and down. He noticed that there was some dust in the barrel but he paid no attention to it. He worked with these extra barrels for about an hour before he stopped work near midnight. He went across the street to a tavern and there his friends noticed that his nails and lips were blue, and he himself felt very weak and nauseated. He remembered collapsing but nothing else until he found himself in the hospital. When I saw him there at 3:00 a.m., his mucous membranes were still very blue and cyanotic and he was in a state of collapse. His meta-hemoglobin anemia responded well to treatment with oxygen inhalation. Investigation of the dirty barrels which the man had packed for the last hour of his work indicated that they had contained para nitra aniline. This substance is absorbed through the skin and produces marked metahemoglobin anemia with weakness and cyanosis.

A tentative diagnosis of leukemia had brought another patient to the hospital because of jaundice and marked hemorrhagic diathesis. Finally, a few hours before the patient died of purpura and bleeding from all body orifices, I was asked to see him. When I took his history, really a death-bed statement, I found that some six or eight weeks before he had bought a second-hand cabin cruiser which badly needed repainting. He built a small shelter about it and proceeded to remove the paint using gallons of the paint remover. We know that many paint removers contain at least 50% benzol. Two or three weeks after he began this part-time work he developed marked weakness and jaundice. Examination showed a definite anemia with a depression of white cells as well as the red cells. Though there are now many relatively safe substitutes for benzol in paint removers, benzol is still so much more effective and cheap, it is to be suspected until proved otherwise that benzol is the culprit when we see such a case history.

Now let us imagine that we had been called to see a young man ill with abdominal distress and difficult vision associated with headache. This is the history that he gave me when I was called to see him five days after he went to the hospital. Four days previously his boss, a small linoleum floor laying contractor, employing three or four men, had sent this man out to pull up, patch and relay linoleum in a small basement apartment. While the man had always used gasoline previously to take off the cement underneath the linoleum which he would then relay, this particular morning of October 21, the boss insisted that the patient use a new, much more effective solvent. The patient worked eight hours on his hands and knees in the basement using ¾ of a gallon of this material to take the cement from the linoleum. The next day he got up with a headache and was dizzy and nauseated. After two more days, he called his doctor who put him into the hospital on October 25 because he could not see while trying to read a newspaper, and he was also nearly anuric. When

I first saw him he had already received ample fluids, much too ample, because these patients usually die of fluid retention and pulmonary edema. I advised concentrated glucose with a reduction of fluids, but by November 1, the patient began to develop pulmonary edema and three days later, on November 4, he was dead. Post-mortem showed that the cause of death was:

1. Acute toxic nephrosis
2. Acute myocardosis
3. Acute hepatitis.

The ¾ of a gallon of the clear fluid and excellent solvent which the patient had used as a linoleum cement remover, the nature of which neither he nor his boss knew, turned out to be carbon tetrachloride.

In my city we have many grain elevators for developing those famous refreshing drinks. In the grain there are many weevils. One of the most effective medications used to deweevilize the grain is a fluid called Weevilcide, a gallon of which is put into the elevators with each five feet of grain. The worker involved in this history did not like the odor of the Weevilcide and was given a respirator. Unfortunately, this was a dust respirator which did not keep out any of the vapor. He was a maintenance man in a crew of three. At times he had to clean out grain elevators that became nearly empty and the solvent settled to the lower part where it was very strong. Once after working two or three consecutive days he became nauseated and was sent to a doctor who discovered albumin in his urine for which the physician prescribed two weeks rest. However, when the patient noticed that urination was becoming scanty and nausea was increasing, he was sent to me for the first time for study. Liver tests showed no abnormalities and with no excess resort to intravenous fluids, the urine gradually became normal in amount and clear of albumin. Investigation finally revealed that the Weevilcide contained a large percentage of carbon tetrachloride.

A third experience with carbon tetrachloride shows the careless use of this material by a cleaner of machinery and motors. The patient in question had worked three or four days, along with three or four men, cleaning the grease off some second-hand machinery in a small boiler repair shop. The patient used most of the material himself including four or five gallons of carbon tetrachloride on each of the four days, throwing it on with brushes and rags. The fourth day he was so weak that he had to be sent home and two days later he was hospitalized in a private hospital. As anuria tendency developed, he was then admitted to the County Hospital where he was dialyzed on the artifical kidney because the urine output had dropped to 125 cc. for each of two days. The NPN had risen to 380. Ten days later the patient began to receive oral fluids and responded well, though he then developed a temporary hemorrhagic diathesis. The patient also had every evidence of involvement of the renal tubules with a nephrosis. This lasted for nearly a year before he was able to return to work.

All of these three cases, then, developed lower nephron nephrosis and in all instances the material was being used with no idea that it was toxic. In reality carbon tetrachloride is one of the most dangerous poisons known to both modern industry and the home. However, any child can buy a bottle over the counter at a drug store.

Turning to other apparently milder symptoms when patients have a simple complaint of diarrhea, we usually think of so-called intestinal influenza. During the last war I was asked to study a group of five or six men in a small operation where bichloride of mercury was developed in a war product. These men had so-called intestinal influenza according to their foreman, but also they had a very foul breath due to a severe gingivitis. Investigation showed that mercury was being heated in a silica retort with free chlorine to form bichloride of mercury in the manufacture of mercury dry batteries. All went well until the demands of the Armed Forces required increased production. This meant increased heat and turning up of the Bunsen Burners under the silica retorts. As a result there was frequent cracking of the retorts and spread of the mercury vapor and chlorine into the small room. Respirators were furnished but the men rarely took time to use them as they replaced the retorts. The diagnosis probably would have remained intestinal influenza had not one of the members been a temporary worker whose wife demanded vigorously that he be investigated for his very foul breath. After a month's work at the above operation he finally quit work because of marked soreness of the gums and swollen neck glands. One of his fellow workers had severe diarrhea so that he passed clear blood in the stool.

Physical examination of four of these men showed a very fetid breath with swollen inflammed gums. Though none of the four cases showed any evidence of renal involvement, one showed rather typical "erethism" or nervous involvement such as was described in *Alice in Wonderland* in the person of the Mad Hatter. All had increased mercury in the urine. Unfortunately these cases were studied before BAL was available for treatment except for the Armed Forces. Removal from exposure and treatment of the gingivitis with penicillin removed the wife's presenting complaint of bad breath. Time had to cure the mercury poisoning, though we called it expectant treatment.

Sometimes it has been my experience to know nothing of these cases until I have been asked to review the post-mortem findings. The following incident is such an example. The manufacture of an "oil-free" or self-lubricating bearing seemed to be the answer to prayer for a certain small machine shop in Milwaukee which needed a new operation to expand its activities in order to keep the men busy. Four or five men were detailed to the new operation. This involved boring holes in bronze bearings, then closing up the holes with a waxy filling containing a secret formula. This acted very efficiently as a permanent self-lubricating agent for the bearing. After the material had been placed in a number of holes in the side of the bearing, the bearing was then placed on a lathe and the surface ground down with a file. The black dust evolved was so concentrated that within a minute or two after the man started the grinding operation his face became completely black. Now a dirty face is not necessarily an indication of danger, but it does indicate a concentrated exposure to dust. It has to be proved in each case whether the dust is poisonous.

Neither the owner of the small shop nor the operator knew the true contents of the secret formula so no effort was made to prevent the inhalation of this dust. For several weeks prior to stopping work one of the employees had a recurrence of an old ulcer complaint. He was admitted to a hospital for treatment about six months after he had started grinding the oil-free bearing. Upon admission he developed a

constant pain in the back of the head and neck with marked restlessness so that it was felt that he probably had a brain tumor. However, just before the operation, laboratory studies showed that he had marked basophilic stippling and secondary anemia. Re-examination of the gums then showed that he had a marked lead line. The urine showed lead also. These new developments suggested that the man might have lead encephalopathy for which he was given calcium gluconate intravenously. However, the decompression was done to relieve pressure within the skull, four days after the patient came into the hospital. He died within a few hours.

After the post-mortem was done I was called in for the first time, to investigate the case. I then found that the secret material combined in the wax and carbon mixture which produced the self-lubricating effect on the bearings was 90% lead oxide. This is simply another example of this commonest and most insidious poison in industry. As some of you may know, I have been working with lead since I first had the chance to study six cases here at the Ford Hospital over thirty years ago.

Following this disastrous episode, the employer and his insurance company took vigorous steps to reduce the possibility of inhaling any of the lead dust at the grinding or buffing operation. Air analysis subsequently showed an entirely safe level of lead in the air at this operation so that when I re-examined the three or four other patients involved, I found no evidence of lead in blood, urine, and of course no lead line of the gums. However, not long afterwards a poorly educated man was put on the operation at night. The importance of using all precautions to reduce dust at this operation was not taken seriously by him. After two months of working nights with no supervision, he began to be absent from work because of indigestion. The employer who by this time was rather sensitive to gastrointestinal complaints at this particular operation sent him down to me for study. I found that he had a 12 plus lead line and many stippled cells as well as a 4 plus porphyrin in the urine. Fortunately by this time Calcium EDTA was available and we deleaded him thoroughly with the usual marked increase of lead in the urine, in his case rising from .2 to 13.3 mgs. of lead in 24 hours immediately after he started the first course of Calcium EDTA given intravenously. Oral medication with Calcium EDTA is practically useless, in my opinion.

Two or three years ago in the midst of a lecture at the Medical School I was called out to take an emergency telephone message. An excited voice stated that I must see him at once because his physician had told him that he had lead poisoning. When I saw him that afternoon at the hospital, I found that the diagnosis was correct. The patient had a marked lead line, many stippled cells, 86 per 50 fields, and lead in the urine. An interesting fact was that this man was the superintendent and part-owner of a small brass foundry employing seven other men. Besides the superintendent, five others of these men had marked lead absorption. The brass foundry had been in operation only six weeks, pouring brass without any respirators or any ventilation. The brass contained 10 to 12% lead. Of eight employees working, six had heavy lead absorption and three of these required hospitalization including the superintendent I have just described. At my request the plant was closed until proper engineering study could be done and ventilation control had been checked by the Industrial Hygiene Unit of the State Health Department of Wisconsin. After two or three

weeks, the plant resumed work with proper ventilation and respirator control. Then all men were able to return to work pouring brass.

One summer night a confused colored man wandered into our County Hospital complaining bitterly of headache. He was so confused that no history could be obtained. Spinal fluid was negative and the patient was treated expectantly. After about a week, laboratory tests showed a secondary anemia with 200 stippled cells per 50 fields and the laboratory technician suggested that the physicians study the gums for a lead line, and that perhaps they had better call me. We found that he had a marked 12 plus lead line. After much careful questioning and investigation an astute resident found that the patient's occupation was that of an operator of a waste paper press in a small junk shop employing only three men. This shop received, however, the trash paper from several large neighboring companies. The paper was heavily contaminated with lead arsenate and other lead compounds. No respirator or suction had been provided for the individual as he and his employer had never thought of the possibility of lead exposure. As he put the lead contaminated trash paper into the press, he would get in to jump up and down to pack the paper in. Of course, this caused evolution of much lead dust. At the first admission the patient was given only expectant treatment and up-building therapy with iron. He was also advised to stay away from this type of lead exposure.

However, a few months later it was found that he had returned to the same work. All his symptoms recurred so that he was then started on the new treatment with Calcium EDTA. This caused a rise of lead in the urinary excretion per liter from .6 to 6.0 mgs. per 24 hours. Since apparently it was hopeless to control the working conditions in this small plant, the patient was advised to find work elsewhere.

Not long afterwards at the same County Hospital, a Puerto Rican who could talk very little English, appeared complaining of headache and inability to feed himself because of marked weakness of both wrists. Our investigation showed that he had been employed for five years as a grinder on brass castings with only 2 or 3 other workers in a very small brass foundry. Brass contains 5 to 30% lead.

This patient had been operated on previously, not only for appendicitis but also twice for alleged recurrent intestinal obstructioin which was quite possibly recurrent lead colic.

On physical examination it was found that he had a lead line and that there were many stippled cells, increased lead in the urine together with bilateral wrist drop. Following several courses of Calcium EDTA, the abdominal symptoms have disappeared, anemia has cleared and the bilateral wrist drop has been practically cured so that he is now able to return to his original work, though he will now wear an approved lead respirator.

From these three cases alone it is apparent that contrary to the general impression, lead poisoning is still frequent. Lead rarely kills and even the small industry can use it safely. Carbon tetrachloride, on the other hand, should not be permitted in large or small industries.

These two hazards have been recognized for many years but new processes and new chemical compounds daily challenge the engineer and physician team.

In summary, it should be obvious that every physician should still ask the question of his patient originally propounded by Hippocrates, "How are your bowels?" In our more modern industrial age, he must also ask one more question, "What is your trade?" just as Ramazzini, the Father of Occupational Medicine did 250 years ago when he used to descend into the pits to study first-hand the operations of seventeeth century industry.

Surely when we are called out to see a new medical emergency in the middle of the night, we should always make sure of the occupation. The acute pulmonary edema may not be a simple heart failure as it seems to be on first glance, but really may be due to the inhalation of ozone or cadmium fume. Headache may be due to carbon monoxide or lead. Convulsions or collapse may not be epilepsy but may be parathion or para nitra aniline poisoning. Marked weakness and anemia may be due to benzol or lead and not pernicious anemia or leukemia. The bloody urine may not be due to a renal stone but may be from benzol or arsine poisoning. An anuria tendency with high blood pressure, nausea, jaundice, loss of vision and headache may not be a simple chronic nephritis on a post-infectious basis but may really be a carbon tetrachloride intoxication.

Bloody stools, too, may not be intestinal influenza or even ulcerative colitis but may be due to an acute mercury intoxication. Finally, the chronic abdominal distress accentuated by occasional violent abdominal colic and rarely complicated by wrist or foot drop may not be merely an acute appendicitis or intestinal obstruction, but really heavy lead absorption and lead intoxication.

In the face of this expereience of mine and that of many others in occupational medicine, the following conclusions seem inescapable:

1. It is imperative to insist on a good occupational history as a part of every differential diagnosis. We should be particularly careful not to make a careless diagnosis of malingering.
2. This occupational history should be confirmed by first-hand observation of the patient's work station right out in the plant by the physician-consultant working hand in hand with the engineer. Thus if we know with what we are working, they can handle any poison safely.
3. We should know the basic mechanism of the action of a poison, both as to its portal of entry and to its point of attack on a particular organ, whether it be on the central or peripheral nervous system, liver, kidney, bone marrow, or heart.
4. Prevention is the best treatment because specific therapies are few, though in appropriate instances described above, these may be oxygen, atropine, BAL, and most recently Calcium EDTA, or calcium ethylene disodium tetra-acetate.

5. The physician can easily become familiar with common sense good plant-housekeeping as well as the general principles of proper ventilation together with protective equipment such as adequate respirators.
6. If placement and periodic re-examination studies are planned and carried through by the physician, they will make him an irreplaceable adjunct to small industry.
7. Finally, all physicians should realize that occupational medicine is preventive, internal medicine applied to industry.

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