A Recent Case of Mild Tetra-ethyl Lead Intoxication

By

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Story of Case

ON THE 27TH FEBRUARY, 1956 an employee of my Company presented at a routine medical examination for tetra-ethyl lead workers with the appearances of an anxiety state. He complained of sleeplessness, headache, a metallic taste which made cigarette smoking unpleasant, general weakness, a feeling of tension and complete loss of libido. On examination he was found to be pale, had a worried expression, and was in a state of extreme agitation, weeping and wandering restlessly about the room, clutching at his hair. His temperature was 97.4 and his pulse rate 92, with a blood pressure of 110/65 and weight of 152 lb. (a loss of 6 lb. in four weeks). He had a marked tremor of his outstretched hands, but otherwise his reflexes, muscle tone and power were normal, and there were no other central nervous signs.

The man concerned joined the Company in April, 1955, when aged 40 years, with no previous occupational record of exposure to lead. His only past history of illness, and an important one in this case, was in 1944, when he was invalided out of the army following psychiatric treatment for neurasthenia and amnesia.

Between April, 1955, and February, 1956, he was engaged on maintenance work in the tetra-ethyl lead plant at one of our factories, and during the period from 15th January to 27th February, 1956, he was engaged on work in a pumphouse which required entry into what was virtually a tetra-ethyl lead saturated atmosphere. The normal procedures for working in this type of hazard involve safety supervision and the wearing of full protective equipment, which consists of a works clothing strip, P.V.C. suit and hood, P.V.C. gloves, Wellington boots and a full face air-line respirator.

At his previous routine medical examination on 31st January, prior to that of 27th February, there was no clinical or analytical evidence to suggest an excessive lead absorption. The only exposure to which this man admitted was on 11th February, when his air-line became accidentally disconnected for a period of about two minutes. During this brief period he inhaled a few lungfuls of the contaminated air, but this alone could not account for this clinical picture, and it is assumed that previous or subsequent exposure must have occurred, probably of a repeated nature. He mentioned this incident on 11.2.56 to the Safety Attendant on duty, but its significance was not fully appreciated and it was not reported further.

Two days after the routine examination of 27th February, his symptoms had increased in intensity, the blood pressure then being 105/45, and the pulse rate 74. He was immediately sent home with the diagnosis of mild tetra-ethyl lead poisoning, which, as will be shown, was confirmed by lead analysis. His own doctor was advised, and conservative treatment, consisting of bed rest, sedation with barbiturates, high fluid intake, and normal solid diet, was instituted. A week later he was suffering from frightening dreams, periods of depression, and a sense of impending doom. His weight at this time had gone down to 145 lb., i.e., a total loss of 13 lb. since just before these events.

After this time his symptoms progressively diminished in intensity, and on 4th April, consisted of mild headaches and dizziness, occasional dreams, depression of libido and metallic taste. His anxiety state had much improved and he was sleeping better and feeling less fatigued. The blood pressure was 130/75, pulse rate 66, and weight 153 lb. There was no evidence of hand tremor and reflexes, muscle tone and power were normal.

On 7th May he started back at work, his only complaint at this time being that of moderate headaches. For obvious reasons he was restricted from working in the tetra-ethyl lead plant. On 10th May he was sent home again complaining of severe occipital headache which was aggravated by noise and physical exertion. Following a two-week spell at a seaside convalescent home, he was much improved, and started work again on 25th June. He again complained of headaches; however, and on 26th July he was seen by a consultant physician, who gave the opinion that the employee was now suffering from a simple anxiety state of psychoneurotic origin. His lead excretion had by this time returned virtually to within normal limits. By the beginning of August his headache was of mild intensity only, and was no longer a constant feature.

Analytical Findings

The clinical diagnosis of tetra-ethyl lead in-
The cation made in this case was supported by the analysis of lead excretion in the urine. Prior to 27th February, routine urinary lead figures were within normal limits.

**Urine (Figure 1)**

Urine lead figures, obtained at weekly intervals from samples provided by the patient during the period 27th February to 25th April, showed figures ranging from a maximum of 583 ug. Pb/1 urine down to 60 ug. Pb/1 urine. The higher figure-ranges indicate an abnormally high lead intake, the upper normal urinary excretion limit being of the order of 100 ug. Pb/1 urine.

**Blood (Figure 2)**

During the period 27th February to 25th April, 1956, blood samples taken at weekly intervals demonstrated figures ranging from 112 ug. Pb/100 gm. to 33 ug. Pb/100 gm. blood. Stipple cells, estimated by the methylene blue staining technique, Ware's stain, rose from nil, prior to his exposure, to 6,000/1,000,000 on 29th February, 3,000/1,000,000 on 4th April, 11,500/1,000,000 on 9th May, and 400/1,000,000 on 2nd July. The haemoglobin, which was assessed at weekly intervals, varied from 90 per cent to 106 per cent.

**Faeces (Figure 3)**

During the period 27th February to 25th April, 1956, faeces samples taken at weekly intervals demonstrated figures ranging from 280 ug. Pb/gm. Ash to 110 ug. Pb/gm. Ash.

Discussion of Case

The case described is typical of the picture presented in mild tetra-ethyl lead intoxication, and occurred as a result of a failure fully to appreciate the necessity for the maintenance of the highest levels of safety procedures when working with this material. There was little doubt on examination of the background of this case that liberties were taken with well-known safety measures which normally provide complete protection.

Tetra-ethyl lead is a slightly volatile organic lead compound consisting of one lead molecule to which are attached four ethyl radicals. It is a colourless, oily liquid, is insoluble in water, but is
soluble in fats and their solvents, this property accounting in large measure for its affinity for the nervous system and for particular toxic effects, and differentiating its toxic manifestations from those commonly associated with inorganic lead compounds.

The use of this material commercially is, as is known, as an anti-knock agent in the petrol of internal combustion engines. A small quantity of the material when blended with petrol (less than 2 c.c. to the gallon) raises the efficiency of the fuel so that it burns more evenly in the engine combustion chamber, eliminating the pinking that represents a mepol violent uncontrolled explosion of less efficient low octane, i.e. cruder, fuel.

Tetra-ethyl lead can be absorbed into the body through the lungs from inhalation of its vapour, or through the skin or by ingestion of the liquid, and is taken up from the blood by the fatty tissues of the body, in particular the C.N.S. The toxic quality of the material is primarily due to the lead content itself.

The classical symptoms associated with tetra-ethyl lead poisoning are those of central nervous system intoxication and consist of an acute anxiety state progressing to a state of mania typical of acute toxic psychosis. Early symptoms and signs manifest themselves in sleep disturbances, dreams, anorexia, nausea and vomiting, metallic taste, headache, mental irritability and anxiety neurosis. The patient may be pale, exhibit tremors and have a slow pulse. Later these symptoms and signs may increase in intensity. The temperature falls and reflexes become exaggerated. Weight loss, change of bowel habit and vomiting may also be noted. Abnormal physical movements develop, together with an intense anxiety and nervousness, nightmares and muscular weakness. In severe cases of poisoning, the condition progresses to a state of intermittent manic excitement with complete disorientation, hallucinations, and delusions with varying paranoid or schizophrenic tendencies. Death may be the final outcome due to extreme physical exhaustion and collapse or to a successful suicide attempt, but in the event of recovery this is always complete and none of the sequelae sometimes associated with other forms of lead poisoning have been noted in these cases. A matter of 5-6 months may elapse before total recovery is complete in the more seriously ill patients, and during this period, relapses are not uncommon.

Even in the milder case, hospitalisation is probably advisable because of the uncertainties of behaviour, but in such cases conservative treatment is indicated as was given to this one. A good diet and mild barbiturate sedation may be all that is needed. Time does the rest.

In the more serious cases, however, considerable care and attention must be given, and constant supervision of the patient is necessary. If the patient is difficult to control, he must be moved to a mental hospital. The great danger, with the manic patient, is of losing control of his fluid and electrolyte balance, and of his refusing nourishment to the point of starvation. Fluids must, if necessary, be given intravenously. Morphia does not help these cases; if anything, it makes them more confused and irrational. Nor can one rely on sedatives only, as toxic levels of administration are soon approached; thus the forcible control of cases has at times to be entertained.

The use of deleading agents has had only a very limited trial in these cases, and at the present time no report of any marked clinical benefits from these can be made.

**DISCUSSION**

DR. J. FERGUSSON (A. Holt and Co.) said most of the data did not concern him personally—he was concerned with the risk among men in enclosed spaces in ships. He asked if there were any sudden acute symptoms brought about by short term exposure to these gases and, if so, what was the best method of emergency treatment? Dr. Barry pointed out that less than 2 c.c. per gallon are put into gasolene and it is not until the gasolene is evaporated to perhaps about a third of its volume that any tetraethyl lead came off. In a confined space this may not be quite so true. A running petrol engine entails a risk of poisoning from gasolene fumes but not tetraethyl lead. If there were a spill of tetraethyl lead in the hold of a ship, access to that hold should be barred until spilled liquid had been completely cleared.

DR. T. G. FAULKNER HUDSON (Imperial Smelting Corporation) asked what was the incidence from tetraethyl lead poisoning in this country; what success had there been to find a substitute with anti-knock properties for this compound; and what routine investigations were made? In reply Dr. Barry said that there had been one case of tetraethyl lead poisoning in the manufacture of the compound in this country, namely the one under discussion. There had been a number of other incidents in the country among tank cleaners, i.e., people who clean out petrol storage tanks; and about ten cases had occurred since the war. On the question of substitutes, he said that no good substitute had yet been found. There were other anti-knock compounds, more efficient but much more dangerous than tetraethyl lead.