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GENERAL SECTION

VEHICLE SMOKE AND LEAD POISONING

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Lead — element 82 in the periodic table has been known for about 5000 years. It is relatively more abundant than other heavy metals in the atmosphere. During the Roman Empire, its production was bout 80000 tons per year and has risen, very much more rapidly this century, to about three million tons annually. Because of this increase, Americans are said to have bout 500 times more lead in their skeletons than Peruvians of 1800 years age.[1]

Lead pollution has become more common in developed countries. The main sources of air-contamination are the use of lead in the industry, petrol and canned foods and some waters. Hence lead is both ingested and inhaled, being distributed via the red cells throughout the soft tissues and accumulated in the skeleton, though much is excreted. About 10% in adults[2] and upto 53% in children[3] is absorbed by the gastrointestinal tract. Over 94% in adults and 64% in children is accumulated in the bones, the toxic effects being given by that in the soft tissues, principally the brain[4]. Excretion is principally by the liver into the bile, part of which is lost in the faeces and part is reabsorbed into the general circulation and excreted by the kidneys.

The early signs and symptoms of lead toxicity can be vague and non-specific and there appears to be a continuum of illness from the non-specific central nervous system (CNS) symptomatology to the acutely dramatic catastrophe.[5] Chronic exposure may result weakness, headache, vomiting, body pain, depression, memory loss, reduced sexual potency and oedema of the brain.[6] Excessive lead exposure during pregnancy has been associated with premature delivery and give rise to neurological damage and growth retardation to infants.[7] In 1987, it was reported that mental development of infants over the first two years of life, whose cord blood levels had been 25 ug/dl, was slower than those whose levels had been lower.[8] However, after two years blood levels between the two groups had not been significantly different.

There is no evidence that lead is involved in any of the physioloigcal functions of the body and also has no known metabolic utility. Hence theoretically, the normal lead level in any body fluid should be zero. After an exposure to lead blood level rise rapidly and its relates to recent exposure only as its half life is about 18 days, whereas in the brain it is much longer.[9] This means that blood lead levels are a measure of recent exposure to lead only, but brain levels remain elevated considerably longer after even a short term exposure. The safety limits for blood lead level given by Bio-science laboratories still are 40 ug/dl for adults and 30 ug/dl for children.[10] Blood lead levels of 120 ug/dl in adults and 80 ug/dl in children can be critical while those about 100 and 60 ug/dl respec-

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tively are toxic[11]. In 1985 levels above 25 ug/dl were regarded as elevated in U.S.A.[12] Recently, it has been shown that blood lead levels as low as 4 ug/dl raise hearing thresholds, 7 ug/dl can cause irreversible neurotoxic effects, 8 ug/dl increases blood pressure, 10 ug/dl can cause shortened red cells life and 25 ug/dl irreversible chronic nephropathy and loss of Intelligence Quotient (I.Q.) in children.[11, 13] As there is no true safety limit, any lead has an effect.[14]

Individuals also very greatly in their response to lead. A blood level of 100 ug/dl may kill one person and have no effect on another.[14] There is also racial differences in the response to lead e.g., black Americans children are less affected than white.[13] Recently, a low molecular weight protein has been isolated from red blood cells which chelates with lead and prevents its toxic actions.[15]. The level of this protein was low in those with low lead levels or with high lead levels having clear symptoms of lead toxicity. High levels of the same protein occurred with high lead levels without toxicity.

There are many sources of lead pollution but a major source is the uncontrolled emission of exhaust fumes from vehicles running on leaded petrol. Seventy percent of the atmospheric lead originate from the combustion of that petrol. A drop of 36.7% in the mean blood lead level was reported in USA mainly due to the introduction of lead free petrol.[16] Lead compounds (Lead tetra alkyl) used in petrol as anti-knocking agents are oxidized in internal combustion and produce toxic fumes:[17] 25% of lead is retained in the car body and 75% goes to the atmosphere. From the atmosphere 25% of lead is removed by wind and 50% is gradually deposited on nearby fruit crops.[18] Peoples living and working along roads sides having heavy traffic are affected by these toxic compounds directly or indirectly.

Beside causing neurological abnormalities the elevated lead level displaces calcium from the hydroxyapatite crystal. Due to chemical analogy of Pb^{++} with Ca^{++} both follow similar Physiological and metabolic pathway.[19] During metabolic process calcium competes with lead and decreased intake of calcium in diet increases lead absorption, retention and toxicity and decreases its excretion. Because of this fact, milk had been given free to lead industry workers to prevent toxic effect of lead.[20] This may be the reason that children and low income groups take up lead more readily and the effects are more severe.[21] Lead poisoning can also be cured by treatment with chelating agents which strongly bind Pb^{++}. Thus calcium chelate in solution is fed to the victim of lead poisoning. Pb^{++} displaces Ca^{++} from the chelate and the resulting Pb^{++} chelate is rapidly excreted in the urine.

Lead poisoning also inhibits several of the enzymes involved in haem synthesis and eventually produce anemia. An important phase of haem synthesis is the conversion of delta-aminolevulinic acid to porphobilinogen which Pb^{++} inhibits. Conditions producing increased levels of any of the haem precursoers are called porphyrias are erythropoietic porphyrias, in which the major diagnostic abnormalities occur in red cells chemistry and hepatic porphyrias, in which haem precursor are found in urine or faeces. A research worker reported that because of this inhibition anemia may be caused in children at lead level of 37 ug/dl and in adults at 60 ug/dl.[22]

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