

Impairment Of Cardiovascular Functions By Mercury Intoxication

Dear Editor:

In etiology of cardiovascular diseases, mercury is considered to be one of the most deleterious heavy metals when compared to lead, arsenic, cadmium and nickel. Mercury (Hg), a cardiovascular toxicant and an environmental pollutant can cause hypertension, arteriosclerosis, tachycardia, cardiac arrhythmia, coronary ischemia, damage to vascular endothelial cells and contractile proteins. Hg decreases oxyhemoglobin level, inhibits cytochrome P450 / heme synthesis, increases the risk of acute myocardial infarction and also generates reactive oxygen species (ROS). Regulation of arterial blood pressure through arterial baroreceptors can be affected by generation of free- radicals. An imbalance in the antioxidant protective mechanisms leading to oxidative stress in the cells is being identified as a major effect of mercury exposure. Mechanisms of impairment of neural control of arterial blood pressure regulation after heavy metal intoxication are ambiguous. The aim of our study was to investigate the toxic effects of inorganic (HgCl₂) and organic Hg (MeHg) after acute and chronic exposure on the cardiovascular system. Anesthetized Wistar albino rats were administered, a single dose of MeHg (2.5mg/kg, i.v.) for acute exposure. Dose (0.5mg/kg/day) for one month given through gavage was used for chronic exposure. HgCl₂ was given in a dose of 5mg/kg, i.v., and 3.75uM/lit. drinking water for one month for acute and chronic exposure respectively. Changes in systolic, diastolic, mean arterial pressure, heart rate, left ventricular pressure (LVP), left ventricular end diastolic pressure (LVEDP), LVdP/dt, LVdP/dtmax, cardiac output and baroreflex sensitivity were recorded, analyzed and compared with the control group. Results from the present study suggest that mercury intoxication either acute or chronic may alter the regulation of neural control of blood pressure mediated through arterial baroreceptors. The haemodynamic parameters were impaired and baroreflex sensitivity was markedly attenuated as compared to the control animals suggesting that cardiovascular functional changes by mercury exposure may be mediated through in -vivo generation of reactive oxygen species.

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