The bitter experience of Itai-Itai disease in Japan and alkyl-mercury poisoning in Iraq have shown that the heavy metals in the environment can have disastrous results for the health of animals and human beings. Industries are increasing day by day thereby increasing the concentrations of metals in the environment and little is known about their risks.

Vanadium is considered both as essential and toxic element (Hopkins and Mohr 1974). It is found in varying concentrations in air, water, soil, plants and animals, as
well as in fossil fuel resources such as coal, oil shale, crude oil, mineral ores and basic rocks (Faulkner-Hudson 1964, Lee and Lehnden 1973). Waste effluents produced from the processings of shales, coal, oil and other sources of vanadium could be expected to create health hazard by increasing the amount of vanadium in the environment. High energy consumptive activities in terms of fossil fuel usage such as steel making industries and thermal power plants are known to be adding vanadium to their immediate environment (Parker et al. 1978, Seth and Pandey 1983, Patel and Pandey 1985).

Vanadium has been recognized as a health hazard since the turn of the century as a result of occupational exposure (National Academy of Sciences 1974). Since then most attention to occupational vanadium exposure has focussed primarily on respiratory effects with a variety of problems, e.g. bronchitis, dyspnea, conjunctivitis, tremor of hands and green tongue.

Various studies have been performed on the vanadium toxicity in mammals, e.g. rat, mice, rabbit, sheep, etc. However, lesser experiments have been performed on aquatic toxicology, especially on fishes. In the present study, the fish, Clarias batrachus has been selected as the experimental organism to assess the toxic effects of vanadium in the form of sodium-metavenadate. These studies have been carried out in the following respects:
1. Determination of toxicity dose at LC$_{50}$.

2. Effects of vanadium concentrations and duration of exposure on the enzymes alkaline phosphatase (alkaline phosphomonoesterase), orthophosphoric-monoester phosphohydrolase (alkaline optimum) EC. No. 3.1.3.1 and acid phosphatase (acid phosphomonoesterase), orthophosphoric-monoester phosphohydrolase (acid optimum) EC. No. 3.1.3.2, glutamate-oxaloacetate transaminase (aspartate amino transferase), L-Aspartate : 2-oxoglutarate aminotransferase EC. No. 2.6.1.1, glutamate-pyruvate transaminase (alanine amino transferase), L-Alanine : 2-oxoglutarate aminotransferase, in liver, kidney, gill, muscle, spleen and brain tissue of Clarias batrachus.

3. Chronotoxicological considerations.