Introduction
INTRODUCTION

Metals are unique environmental and industrial pollutants as they are neither created nor destroyed by humans but are only transported and transformed into various forms. Often these activities result in the exposure of persons to trace elements not ordinarily in contact with them. Sometimes chemical forms are created that are not usually present in nature. Industries are increasing day by day, thereby increasing the concentrations of metals in the environment and little is known about their risks. Mercury poisoning in Iraq and Itai-Itai disease in Japan have shown that the metals in environment and industrial exposure can have disastrous result to the health of animals and human beings.

Vanadium is considered both as an 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 (Judson 1964, Lee and Lehmden 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 1973, 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 Science 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., either by injection or respiratory route. However, very little experimental observations are available on the vanadium toxicity by oral administration, especially in rabbits. In the present study, rabbits have been selected as an experimental animal to assess the toxic effects of vanadium in the form of sodium metavanadate at different concentrations. These studies have been carried out in the following respects:
1) Effect of vanadium concentrations and duration of exposure on the haematological parameters such as red blood cell number, white blood cell number, haemoglobin concentration, packed cell volume and absolute values such as mean cell volume, mean cell haemoglobin and mean cell haemoglobin content in rabbits.

2) Effects of vanadium concentrations and duration of exposure on the biochemical parameters such as enzymes: alkaline phosphatase (alkaline phosphomonoesterase) orthophosphoric-monoester phosphohydrolase (alkaline optimum) E.C. No. 3.1.3.1.; acid phosphatase (acid phosphomonoesterase). Orthophosphoric monoester phosphohydrolase (acid optimum) E.C. No. 3.1.3.2.; glutamate-oxaloacetate transaminase (aspartate amino transferase), L-aspartate : 2-oxo-glutarate aminotransferase E.C. No. 2.6.1.1.; glutamate-pyruvate transaminase (alanine amino transferase), L-Alanine: 2-oxo-glutarate amino transferase E.C. No. 2.6.1.2.; Succinate dehydrogenase (succinate: dehydrogenase E.C. No. 1.3.99.1; Glutamate dehydrogenase (L.Glutamate: NAD (deaminating) oxidoreductase) L.C. No. 1.4.1.2.; Lactate dehydrogenase (L-Lactate: NAD oxidoreductase) E.C. No. 1.1.1.27; histaminase and plasma cholesterol.

3) Effects of vanadium concentrations on immunoglobulin-G levels in plasma of rabbits.

4) Morphological changes in organs of rabbits, their survival and body weight following vanadium exposure.