Vanadium is considered both as essential and toxic element (Hopkins and Mohr, 1974). Eight concentrations of vanadium have been tested for its toxicity on Clarias batrachus. All the concentrations tested have shown toxic effect upon this fish. Vanadium in 29 ppm was found to be highly toxic in which all the fishes died within 24 hrs of exposure, whereas 5 ppm concentration of vanadium exhibited delayed toxicity where the 100% mortality was reached in twenty one days of treatment (Fig. 1). The LC\textsubscript{50} values for Clarias batrachus in relation to the duration of treatment were 23.33 ppm (48 hrs), 16.59 ppm (72 hrs) and 14.16 ppm (96 hrs), respectively
(Table 3). Knudtson (1979) had reported LC\textsubscript{50}s for *Cerassium auratus* (2.45 ppm) and *Lebistes reticulatus* (0.47 ppm) at 144 hrs using sodium metavanadate. The LC\textsubscript{50} value recorded for *Clarias batrachus* with the same vanadium source though at 96 hrs, appears to be higher than the observation of Knudtson (1979). It may be due to the specific variation in the test fish, and also in the difference of the environmental conditions for which this fish is adapted.

In the experimental fishes, the vanadium toxicity symptoms appeared in whitening of skin, sluggishness, gradual loss of sensitivity and equilibrium and ultimate death. Alternation in the levels of brain amines of vanadium treated rats have been reported by Witkowska and Brzezinski (1979) which they considered as early signs of toxic action on central nervous system (C.N.S.) function. The signs of vanadium toxicity in man include tremor and C.N.S. depression (Done 1979). The loss of sensitivity and equilibrium observed for *Clarias batrachus* in the present study might also be imputed to impairment of central nervous system function principally caused by the vanadium toxicity.

The proven toxicity of vanadium (sodium metavanadate) has been further enlarged to determine its effect on certain metabolic enzymes and their activities in different organs of the fish. Though, a much variable effect was observed in each stage of experimentation, the overall effect was significant in several determinations.
The three concentrations of vanadium, i.e., 20, 10 and 5 ppm were selected for further studies. In general, it has been observed that the glutamate-pyruvate transaminase activity (GPT) is decreased in liver, kidney, gill, muscle and spleen tissue of vanadium exposed fishes. However, in the liver and kidney, the decrease in glutamate-pyruvate transaminase activity is preceded by an increased level. In the brain tissue of experimental fishes, in contrast to liver, kidney, gill, muscle and spleen, an increased level of glutamate-pyruvate transaminase activity was recorded (Fig. 2 to 13). However, the glutamate-oxaloacetate transaminase (GOT) exhibited different response as compared to glutamate-pyruvate transaminase (GPT). In general, an increased level of GOT activity was recorded in the kidney, gill, muscle and spleen as well as brain tissue as compared to liver where a decrease in the level was noted (Fig. 2 to 13). The GOT is widely distributed in human tissue, heart, liver, kidney, skeletal muscle being the richest source but smaller amounts are found in pancreas and spleen (Wroblewski and La Due, 1956), whereas the liver is the richest source of GPT. These enzymes play a key role in the intermediary metabolism by transamination which provides a means for the synthesis and degradation of amino-acids in living cells. The three amino acids, glutamate, aspartate and alanine may, by such means, be converted into corresponding α-keto-acids, which are the components of
tricarboxylic acid cycle. Thus, they can be oxidized to provide a source of energy. Vanadium has been reported to inhibit the glycolysis by inhibiting several enzymes in glycolytic pathway (Simons 1979), including glyceraldehyde-3-phosphate dehydrogenase (De Raster and Mitchell 1973), phosphoglucomutase and phosphoglyceromutase (Climent et al. 1981, Carreras et al. 1982, Vives-Corrons 1981). In the present study, the increased activity of GOT in kidney, gill, muscle, spleen and brain along with an increased activity of GPT only in the brain, may be an attempt to obtain energy through amino-acid catabolism by vanadium treated fishes. Decreased activity of GPT in liver, kidney, gill, muscle and spleen may be due to some inhibitory action of vanadium.

An increased level of alkaline and acid phosphatase activity was recorded in the liver and kidney tissues of vanadium treated *Clarias batrachus*. However, increase in the alkaline phosphatase activity was preceded by its decreased level in both the organs. In gill, muscle, spleen and brain, the levels of both the alkaline and acid phosphatase activity have been found to be decreased. However, in muscle, the decrease in the alkaline and acid phosphatase activity was preceded by an increased level. Further, an increased level of acid phosphatase activity was found in gill and spleen tissue in the latter period (5th and 6th day) of 10 ppm exposed fishes (Fig. 2 to 13).
Vanadium compounds have been shown to inhibit the acid and alkaline phosphatases (Lopez et al. 1976, Choata and Bhurendhar and Rai 1986). Alkaline and acid phosphatases catalyze the hydrolysis of almost any phosphomonoester to give inorganic phosphate and the corresponding alcohol, phenol or sugar (Fernly 1971). Alkaline phosphatase is widely distributed in human tissue and in each case the enzyme activity is localized at the cell membrane where it appears to play a part in the transport mechanism involving phosphate (Engstrom 1964b). Acid phosphatase activity has been considered as a marker for lysosomes. The lysosomal enzymes are associated with the degradative processes and their higher rates of activity are often correlated with greater turnover of molecules (Allison 1968). Increased level of acid phosphatase activity in the liver and kidney may be correlated with increased catabolic activity which may be a metabolic readjustment to endure toxic effect of vanadium. However, the increased alkaline phosphatase activity in liver and kidney suggests an increased active uptake of ions in vanadium treated fishes.

Occurrence of variable effects of vanadium, in the activity levels of these enzymes, as an effect of treatment duration, exhibited relative influence of the amount of residual accumulation of vanadium which tended to increase with the longer exposure period ordinarily.
Disturbances caused in these metabolic activities are a positive indicators of the stressed condition of the system. These studies seem very much relevant in the context of men who is being exposed to ever increasing amounts of vanadium in his environment mainly due to the burning of fossil fuels.

This experimental protocol has also yielded data worthy of consideration from the point of view of chrono-toxicology, as the biological rhythms are known to exist at various levels of biological organization and function (Hulberg and Conner, 1961; Reinberg, 1974; Scheving, 1976; Pati et al., 1987; Mayersbach, 1976; Chemnitz, 1975; Mayhsach et al., 1977). Circadian rhythms (daily rhythm) in toxicity of various toxic agents have been documented for human beings in the clinics as well as for laboratory rodents (Nelson and Halberg, 1973; Muller, 1971). Keeping in view, we have examined the effects of vanadium at different time of the day to elucidate whether the effects of vanadium on *Clarias batrachus* are circadian stage dependent. The diurnal rhythm in alkaline phosphatase activity, although present in kidney, muscle, spleen and brain, is modified by the vanadium treatment only in spleen and brain and both, the rhythm and vanadium effect are dependant on each other. Similarly the diurnal rhythm in acid phosphatase activity appears in all the tissues studied, but rhythm in enzyme is modified only
in the gill tissue (Table 29A, 31A, 33A, 35A, 37A, 39A; Fig. 14 to 19).

The diurnal rhythm in glutamate-oxaloacetate transaminase (GOT) is present in all the tissues studied except brain, however, the rhythm in GOT is modified in kidney, gill, spleen and muscle and both the rhythm and vanadium effect are found to be dependent on each other. However, the glutamate-pyruvate transaminase showed diurnal rhythm only in liver, muscle and spleen; unaffected by vanadium treatment (Table 29B, 31B, 33B, 35B, 37B, 39B).

Therefore, the fluctuation in the activity levels of these metabolic enzymes at different time intervals of exposure would not directly be a product of vanadium accumulation in the tissues as the rhythm would also be a factor.