PREFACE
The incidence of pesticide poisoning in the third world has assumed alarming proportions following the intensive and indiscriminate use of a wide range of "highly vicious" agro-chemicals designed to boost the green revolution. Five lakh cases of pesticide poisoning are reported each year from the third world, of which nearly 10,000 are fatal. For instance, in U.S.A. 20,000 individuals who had imbibed a certain brand of alcoholic extract of "jamaica ginger" suffered a debilitating peripheral neuropathy commonly known as "Ginger jake paralysis". In 1964, many people suffered from toxic effects of endrin of which, one died due to consumption of contaminated rice. In February 1977, villagers in the remote hilly tracts of Karnataka, in India were bed ridden with a strange type of crippling disease called the "Handigodu Syndrome" in local parlance. Statistical surveys of mortality and morbidity resulting from the acute and chronic exposures, due to pesticides reveal that neonates are the victims of a high percentage of fatal poisonings. Uppal et al. (1983) and Rony Nethew et al. (1989) observed suppression of learning process by various insecticides. A report by an American environmental group National Resources Defense Council (NRDC), that preschoolers are more susceptible to pesticide residues. However scant information is available on the
toxic effects of pesticide methyl parathion on vertebrate during the critical stage of "CNS" development. Hence, keeping in view the heterogeneity of the brain tissue and the dearth in the literature on the altered physiology and biochemistry of the developing "CNS" as a function of pesticide exposure, the present study was proposed.

Developing albino rat pups, 2nd and 7th day-old were used for the study. Sublethal dose (1/3rd of LD50) of methyl parathion (o,o-dimethyl-o-nitrophenyl Phosphothionite, Bayer Leverkusen, W. Germany) was administered intraperitonially to experimental animals. Controls were injected with distilled water. Animals were decapitated 48 hrs after insecticide administration. Different regions of the brain like cerebral cortex, brain stem and spinal cord were dissected and separated at 0°C and used immediately for analysis.

Detailed analysis of several physiological and biochemical changes connected to protein metabolism as a result of methyl parathion exposure have been carried out.

Studies have been directed to resolve the regulatory calcium binding protein, calmodulin in view of its important role (it plays) in the synaptic chemistry. Changes occurring in the brain specific acidic proteins viz., S-100 (protein) was also followed. Methyl parathion caused a significant
decline in the concentration of S-100 protein and calmodulin.

Changes in the cyclic AMP dependent phosphodiesterase activity have been studied. Molecular heterogeneity of the above enzyme in the developing central nervous system was followed. Methyl parathion substantially decreased the qualitative nature of phosphodiesterase isozymic pattern.

Studies on alterations occurring in the cholinergic transmission have been conducted on methyl parathion treatment. Changes in the levels of excitatory neurotransmitter ACh was studied. The activity levels of AChE and choline acetyl transferase (ChAT) and its molecular heterogeneity was determined. Acetyl cholinesterase and choline acetylase activities showed significant decrease on methyl parathion exposure. The multiple forms of both the enzymes showed remarkable disruptions in the pattern as a function of methyl parathion exposure.

The inhibitory neurotransmitter GABA and the enzymes connected to GABA shunt pathway viz. GAD and GABA-T were studied in view of the involvement of GABA in the neuronal transmission. The activity level of GAD and GABA-T showed a significant increase on exposure to methyl parathion indicating augmented synthesis of GABA.
Changes in the levels of excitatory amino acid transmitters like glutamic acid and aspartic acid and inhibitory transmitter glycine were studied as indices of neuronal activity after pesticide exposure. The levels of these amino acid transmitters increased considerably on NPE.

Changes in the levels of catecholamines and in the activity levels of monoamine oxidase were studied as a function of pesticide exposure. Methyl parathion substantially altered the activity level of MAO.

Changes occurring in the levels of total phosphate and in the activity levels of Na\(^+\)-K\(^+\) ATPase, Ca\(^{2+}\) ATPase and Mg\(^{2+}\) ATPase were followed. Considerable alterations in the activity levels of ATPases were observed as a function of methyl parathion exposure.

The activity level of selected dehydrogenases like SDH, GDH and PDH were measured as an index of the metabolic activity on pesticide exposure. Molecular heterogeneity of the LDH in the developing brain was followed. Methyl parathion substantially affected the qualitative nature of brain LDH isozymic pattern.
Thus these changes found in the developing nervous system on methyl parathion exposure indicate that the protein synthetic machinery is affected due to the toxic impact of an organophosphorous insecticide. This in turn leads to biochemical lesions and disruptions in the functioning of the brain.