INTRODUCTION
Coxsackie viruses were identified during the campaign of controlling the height of growing epidemics of poliomyelitis of nineteen forties. With the subsequent identification of the virus in pure form by Melnick and his group (1949) it was gradually recognised that the Coxsackie group B viruses produce a mild illness in man. This illness runs a short course with complete recovery without any sequelae. Since the report of Montgomery et al. (1955), the Coxsackie group B viruses were regarded to be the responsible agent in causing significant number of deaths occurring during the neonatal period (Benirschke and Pendleton, 1958; Kibrick and Benirschke, 1958; Sussman et al., 1959; Jack and Townley, 1961). In such cases transplacental passage of virus (Benirschke and Pendleton, 1958; Kibrick and Benirschke, 1958) or the direct contact of the infected individual in first few days of life was implicated (Sussman et al., 1959; Moosy and Gear, 1960). Encephalitis and myocarditis have also been reported and virus was recovered from heart, brain, spinal cord, faeces and blood of the infants (Benirschke and Pendleton, 1958; Delany and Fukunaga, 1958; Kibrick and Benirschke, 1958; Sussman et al., 1959; Jack and Townley, 1961; McLean et al., 1961).
Fatal myocarditis due to Coxsackie group B viruses were usually seen in newborn or infants below 2 years of age. Fletcher and Brennan (1957), and Weinstein (1957) brought out evidences that Coxsackie group of viruses could also produce heart diseases in adults. Smith (1968) regarded Coxsackie group B as a leading cause of heart disease of viral origin. Coxsackie virus group B infection can be demonstrated up to 39% of cases of otherwise unexplained acute myocarditis and pericarditis (Grist and Bell, 1969). Reports from Western Australia (Smith, 1968); U.K. (Grist and Bell, 1968); Finland (Helin et al., 1968); U.S.A. (Sainani et al., 1968); and India (Agarwal et al., 1970; Madhavan et al., 1974; Chandrasekhar et al., 1975) further indicated causal relationship between the Coxsackie group B virus and myocarditis. Burch et al. (1967) first brought out more direct evidences and could identify, employing immunofluorescent techniques, Coxsackie virus antigen in routine autopsy specimens of heart in 17 cases. This brought into sharp focus the relationship between Coxsackie group B virus and myocarditis.

Coxsackie viruses are found to be pathogenic for immature mice (Kaplan and Melnick, 1951).
Gifford and Dalldorf, 1952) and induce in them distinctive lesions that permit identification of subgroup A and B. The group A virus causes extensive hyaline degeneration of skeletal muscle. The group B type causes encephalitis, remarkable lesion in brown fat and focal muscle degeneration.

Histological study was initiated by Pappenheimer et al. (1950) when they have to work with virus suspension of brain material, as such it was difficult to isolate them. Subsequently, Kaplan and Melnick (1951), Gifford and Dalldorf (1952) extended this work. Grodums and Dempster (1959 a, b) first brought out the importance of age factor with Coxsackie group B3 virus infection in cases of myocarditis. They observed severe myocardial lesion in mice of 12-23 day old. Subsequently, Grodums and Dempster (1962), thus, observed that there was difference in the intensity of myocardial dependence on types of B virus. They further stated that Coxsackie B3 caused most severe cardiac lesions in the weanling mice, whilst the B1, B2 and B4 types were mildly cardiotropic in the suckling mice but had a reduced effect on the weanlings. Rabin et al (1964) observed myocarditis in 12-20 days old mice with Coxsackie B3 virus. They also observed progressive scarring and
calcification with B₃ virus in heart. The observations of Miranda et al (1973 b) with B₅ virus showed focal scarring in certain cases. Thus, the myocardial lesion with scarring was observed by them when they have used Coxsackie B₃ or B₅ virus (Rabin et al., 1964; Miranda et al., 1973 b).

Soike (1967) studied the effect of virus on pregnant mice following intraperitoneal inoculation on their litters. This worker, however, observed evidences of affection on the litters and ascribed these to affection of various tissues from isolation of virus. He brought out an indirect evidence of a latent or an active Coxsackie virus infection in the reproductive organs from his rebreeding progeny study. Lansdown and Cold (1974), Lansdown and Ellaby (1974), Lansdown (1975 a, b) extended the above study and found histological and biochemical evidences of the liver damage. They felt this change secondary to pancreatic lesion. Lansdown (1975 b) observed that casein supplement reduces or abolishes the virus affection on the progeny. These were very interesting observations in connection with B₃ virus.

Miranda et al (1973) observed diminution of body weight of virus infected mice. This low body weight
persisted as long as six months although no B5 virus could be recovered after 9 days following intracerebral inoculation. On the other hand, heart weight/body weight ratio was found to be increased. They ascribed this to accumulation of blood in heart due to dilatation.

From the above study it appeared lack of coordinated study on the various parameters with any single standard type of virus have not been carried out. Coxsackie B1 was, therefore, chosen because of mild degree of infection it produces as reported by Grodums and Dempster (1962) and has not been studied extensively. Since it was very difficult to relate the different observations by these authors only from mere presence or passage of virus in different organs, it was felt that there could have some biochemical alterations produced by the virus infection at the cellular level. The latter could have caused a profound alteration of cell metabolism in a way which could be present even when virus could not be isolated from the particular tissue. The present work was, therefore, aimed to assess the various parameters of Coxsackie B1 virus infection in neonatal mice, pregnant mice and also in 5-day old mice. Later, this was extended
to evaluate whether such virus infection could have made some imprint at the cellular level, even when virus could not be isolated by recovery study.