Introduction

Cardiovascular compromise is common in sick term and preterm infants *(Gill and Weindling, 1993)*. Impaire l myocardial contractility and low cardiac output are common compli :ation of such conditions such as respiratory distress syndrome and pe inatal asphexia *(Clark et al., 2004)*. This reduced cardiovascular rese ve may present clinically with hypotension which is associated with increased mortality and adverse neurological outcome *(Goldstein e al., 1995)*. It has been suggested that myocardial dysfunction is lue to ischaemia and/or necrosis *(Finley et al., 1979)*. Previous stud es in neonates have used creatine kinase isoforms as biochemical ma kers of specific hypoxic injury *(Primak et al., 1985)*. However these narkers have been largely discarded because gestatation, sex, birth weig t and mode of delivery all affect creatine Kinase activity *(Bhayana, 199:)*.

Troponin is an inhibitory protein con plex forming part of the contractile apparatus of all striated muscle, i cluding the heart specific forms of the three troponin subunits T, C and I exist in different muscle types. Cardiec specific troponin T and I have become established as the best biochemical markers for myocardial necrosis *(Hetland and Dickstein, 1998)*.

Clark et al., (2000) has previously repo ted that cardiac troponin T concentration in the cord blood of neonates s unaffected by gestation, birth weight and sex. Furthermore increases i cardiac troponin T in the cord blood were found to independently p edict the development of respiratory distress syndrome.

In the study of *Clark et al., (2006)* h igher level, of troponin T were found in respiratory distress syndrom infants and the elevation was early and sustained, suggesting signific ant myocardial damage of antenatal/ intrapartum origin giving rise to m asurable dysfunction.