INTRODUCTION

Cardiovascular compromise is common in sick term and preterm infants **(Gill , 1993).** Impaired myocardial contractility and low cardiac output are common complications of such conditions as respiratory distress syndrome **(Von Bell ,1990).** This reduced cardiovascular reserve may present clinically with hypotension, which is associated with increased mortality and adverse neurological outcomes **(Goldstein , 1995).**

It has been suggested that this myocardial dysfunction, or stunning, is due to ischaemia and/or necrosis **(Clark , 2004).** Previous studies in neonates have used creatine kinase isoforms as biochemical markers of myocardial injuries. However, these markers, have been largely discarded because gestation, sex, mode of delivery, and birth weight all affect creatine kinase activity **(Bhayana , 1995).**

Troponin is an inhibitory protein complex forming part of the contractile apparatus of all striated muscle, including the heart. Specific forms of the three troponin subunits T,C, and I exists in different muscle types. Cardiac specific troponins I has become established as the best biochemical marker for myocardial necrosis **(Oyvind Heitland , 1998).** They start to increase two hours after myocardial infarction, and concentrations can remain raised for up to 5-7 days after a full thickness infarct.

Indeed the assays for cardiac troponin I are now so sensitive and specific, mainly because of the use of the latest third generation assays, that a concept of minimal myocardial damage has arisen in adult medicine.

These marginal increases in cardiac troponin I are associated with worse outcomes in adult patients after admission to hospital **(James , 2000).** Previous studies in children and neonates have used older first or second generation assays and have referred to adult reference ranges **(Narin , 1999).**

Previous reports suggested that cardiac troponin I concentration in the cord blood of neonates is unaffected by gestation, birth weight, and sex. Furthermore, increases in cardiac troponin I in the cord blood were found to independently predict the development of respiratory distress syndrome **(clark , 2002).**