INTRODUCTION

Not more than 35-40 years ago it was postulated that oxygen is toxic due to its tendency to create oxygen radicals *(Saugstad, 1992)*. It is speculated that numerous diseases in both adult and neonatal medicine are associated with free radicals and their damaging effects *(Halliwell, 1991 and Halliwell, 1994)*.

The premature baby is more susceptible to tissue injury than the term one. So that babies born preterm are more prone to free radical injury than later life. Free radicals could cause a series of well known complication in the neonatal period such as nectotizing enterocolitis, persistent patent ductus arteriosus and possibly also intraventricular hemorrhage (*Saugstad, 1990*).

There for these conditions do not represent different diseases, but rather constitute different facets of one single disease, neonatal oxygen radical disease (*Saugstad, 1992*). The theoretical basis for free radical involvement in these disorders is that oxygen centered radicals and related reactive oxygen metabolites are formed too rapidly to be detoxified by the antioxidant defense mechanisms in specific tissues (Kelly, 1993). And as premature infant has poorly developed antioxidant systems and therefore may be of increased risk of radical damage (*Pittkanen et al., 1990 and Frank, 1992*).