RENAL FUNCTION IN NEONATES WITH RESPIRATORY DISTRESS SYNDROME

THESIS

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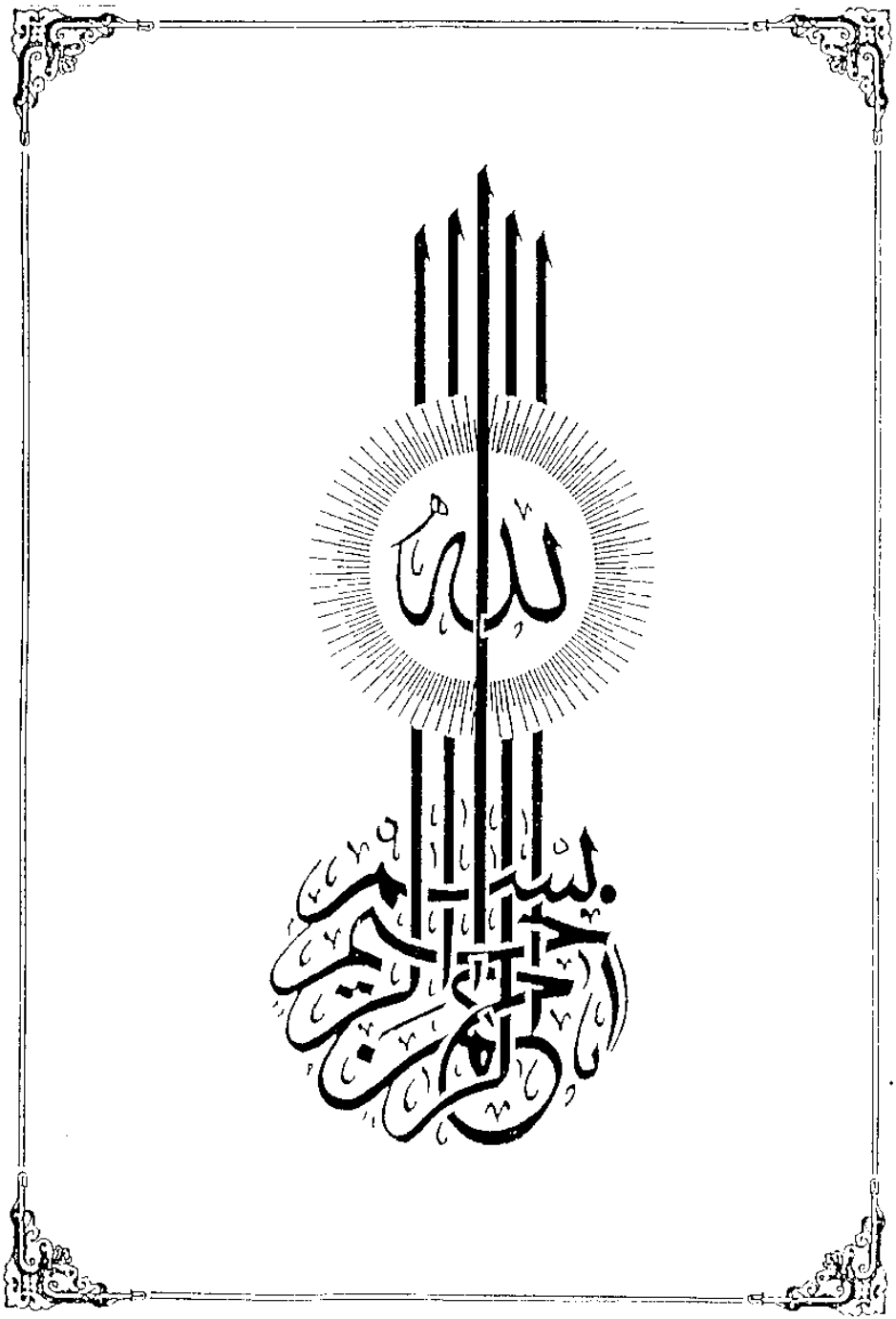

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Adel Khairy

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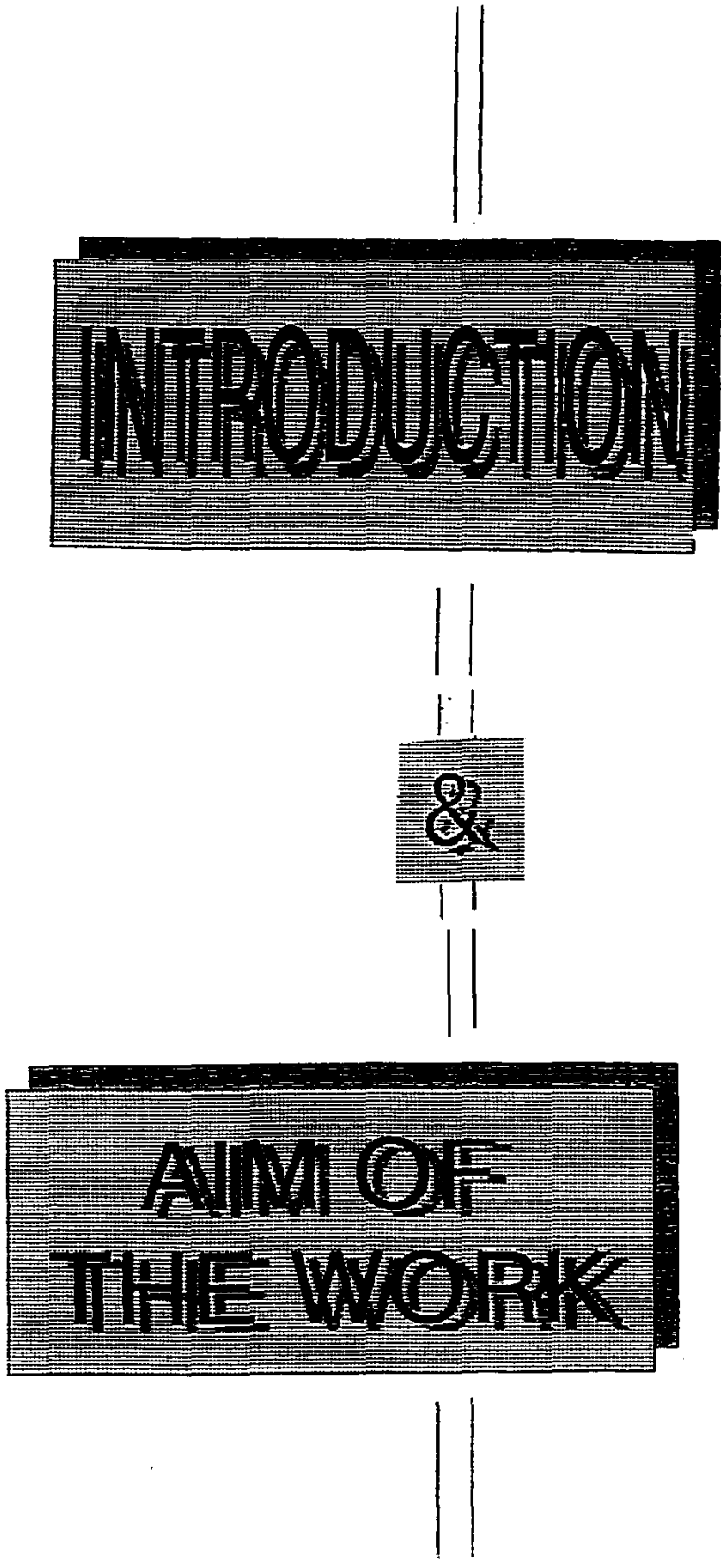
RDS	→ *Respiratory distress Syndrome*
FRC	→ *functional residual capcity*
PDA	→ *patent ductus ar-teriosus*
IVH	→ *intraventricular haemorrhage*
FSI- test	→ *foam stability index test*
L/S Ratio	→ *lecithin/ sphingomyelin Ratio*
SPC	→ *saturated phosphatidyl choline*
Torr	→ *unit of pressure (one torr = 1 m m Hg)*
CPAP	→ *contineous postive airway pressure.*
JMA	→ *Juxta medullary Apperatus*
GFR	→ *glomerular filteration Rate*
LBW	→ *low birth weight*
PTH	→ *parathyroid hormone*
ADH	→ *Anti diuretic Hormone*
F_ENa	→ *Fractional urinary Na excretion*
RFI	→ *Renal failure index*
α1,.M	→ *alpha- one- micro globulin*
PPD	→ *purified protein derivative.*
FPF	→ *Fibroblast pneumocyte factor.*



Introduction & Aim of the work

One of the common & serious disease in The neonatal period is respiratory distress syndrome which has serious sequalae on the morbidity & mortality in the neonatal period [Gortner et al 1992].

Inspite of advances in the management of the respiratory distress syndrome through surfactant replacement therapy yet., more than 30% mortality occur in the developed world due to R. D. S indicating that it is not only the surfactant deficiency which is responsible for the sequalae of the disease., Multipte organs failure is also existing in such diseases. [Chan et al 1992].

One of the vulnearable organs is the Kidney., Disorders in the regulation of the fluid & electrolytes balance in infants with respiratory distress syndrome has been reported [Brem 1992].

Hypoxic stress & Negative haemodynamic effects which may impair the kidney function are common during this diseases [Zanardo et al 1990].

The aim of this work is to study the Kidney function in sick neonates with respiratory distress syndrome which may have implications on the mangement of such neonates.

REVIEW OF LITERATURE

Chapter (1)
Respiratory Distress Syndrome

Definition:

*Respiratory distress syndrome is a disorder mostly of the premature infants that is clinically manifested as respiratory distress. It is widely believed that lack of sufficient pulmonary surfactant is the cause of R.D.S. in the immediate neonatal period. It remains the major pulmonary problem of the newborn despite the many advances in the therapy (**Vidyasagar 1985**).*

Incidence:

*The major problems with defining the incidence of this disease are difficulties of making a precise diagnosis and a lack of accurate statistics. even in competent hands an accurate diagnosis can only be achieved in 80-90% of cases. (**Farrell and avery: 1975**).*

*It is inversely proportional to the gestational age, it occurs in about 60% of infants with less than 28 weeks of gestational age, in 15% -20% of those between 32-36 weeks in about 5%-beyond 37 weeks and rarely at term. (**Behrman and Kliegman; 1992**).*

***Farrell and Wood; 1976** concluded that RDS accounted for approximately 20% of the neonatal mortality with a seasonal trend of 20% more deaths in mid Summer than in midwinter despite a steady birth rate throughout the year.*

Babies mainly die from RDS in the first few days, with 92% of deaths occurring by the fourth day. There are considerable racial variations with premature negro babies having a 40% lower incidence and mortality than caucasion babies of the same birth weight.

*Since the introduction of neonatal intensive care the mortality from RDS has dropped from around 70% to 10-20%. (**Hallman etal.; 1980**).*

Aetiology:

Pulmonary immaturity consisting of surfactant deficiency, incomplete structural development of the lung and a highly compliant chest wall, are the major contributors to RDS.

*Pulmonary over perfusion secondary to left to right shunting through the ductus arteriosus may also play a significant role, especially in less mature infants. (**Stark and Frantz; 1986**).*

Predisposing Factors For RDS:

1- Prematurity, *2- male sex,(male: female is 17:1)*

3- Acidosis, *4- Hypoxic or hypercarbic episodes,*

5- Hypothermia, *6- Hypoglycemia,*

7- Caesarean Section *8- Second twin,*

9- Intracranial hemorrhage

10- Babies of diabetic mothers

11- Patent ductus arteriosus

12- Persistent fetal circulation,

13- Left ventricular failure,

14- Pneumothorax or interstitial emphysema,

15- Endotracheal tube present with no applied pressure

16- Hypotension.

All the above are factors which contribute to RDS (Morley; 1986). Prematurity is the major factor predisposing to RDS.

The more immature the baby the greater are its chances of developing RDS. The incidence is inversely proportional to gestational age (Usher et al; 1971).

Prematurity is strongly associated with RDS because the disease is affected by immaturity of not only all aspects of lung structure and circulation, but also, by immaturity of the respiratory muscles, central respiratory control and particularly the surfactant system (Morley; 1986).

Boys are 25% more likely to develop RDS than girls and suffer more severely from the disease (Farrell and Avery; 1975). The reason for this is not fully understood but it is probably due to the female fetus maturing earlier than the male (Kotas and Avery; 1971; Dhanireddy et al; 1983), the stimulating effect of oestrogens on surfactant synthesis (Khosla et al.; 1983) and delay in the male lung beta receptor maturation (Padbury et al.; 1981).

Fedrick and Bulter; 1970. Showed that the incidence of RDS in babies born before 32 weeks gestation was 57.6% after caesarean section and 22.2% after vaginal delivery.

Usher et al., 1971 showed that caesarean section delivery without labour pain significantly increased the risk of R.D.S between 33-36 week gestation. Labour pain inhibit lung fluid secretion and encourages its absorption (Walters and Olver; 1978). So babies born without its influence have more lung fluid and a lower thoracic gas volume over the following hours (Milner and Vyas.; 1982). This effect seems to be caused by the adrenergic hormone surge which only occurs with labour stress (Faxelius et al.; 1983). Adrenergic agents not only reduce lung fluid secretion but also increase surfactant synthesis and secretion (Enhorning et al.; 1977, Corbet et al; 1977). The fluid in the alveoli interferes with their expansion which in turn, reduces their ability to secrete surfactant (Lawson et al., 1977).

During labour, there is a release of surfactant into the airways this may explain why the pharyngeal L/S ratio is higher in babies born by caesarean section after normal labour pain than after elective caesarean section (Challen et al; 1979).

Jones et al.;1975 found the incidence of RDS in premature babies with an apgar score of five or less, was twice as high as those with a score above five. There is no single reason for this difference but asphyxia and acidosis predispose to: