ASPHYXIA NEONATORUM

THESIS

Submitted For Partial Fullfilment of a Master Degree
of Pediatrics

BY

Dr. DOHA EL MASSRY

SUPERVISOR

Prof. Dr. M. ESSAWY
Professor of Pediatrics
Faculty of Medicine
Ain Shams University

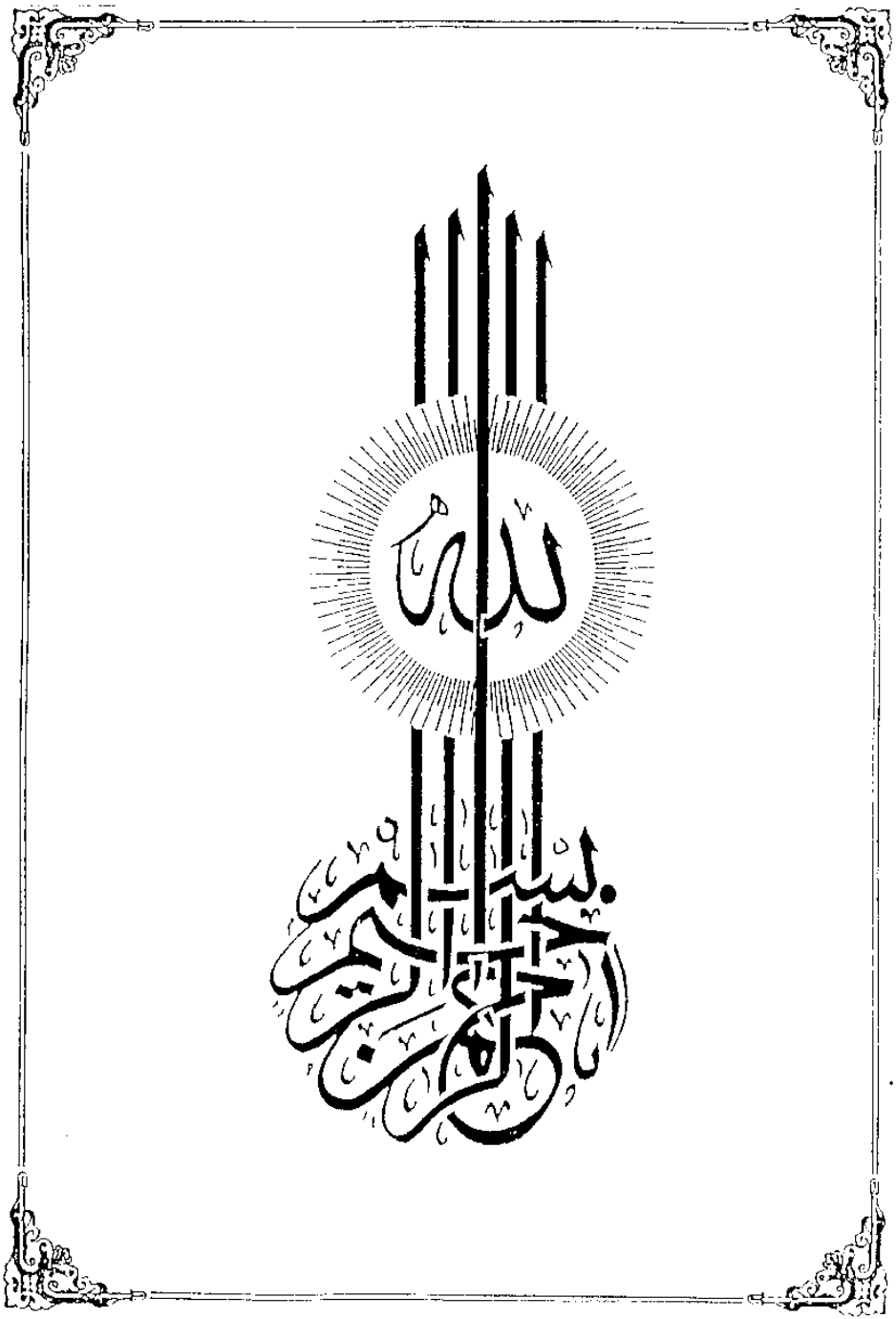
Dr. FARIDA FARIED
Asisstant Professor of Pediatric
Ain Shams University

1986

ACKNOWLEDGEMENT

I would like to express my profound gratitude and sincere appreciation to ***Professor MAHMOUD ESSAWY,*** *Professor of Pediatrics, Ain Shams University for his constructive supervision and encouragement.*

I would like also to express my cordial thanks and deep gratitude to ***Professor FARIDA AHMED FARID,*** *Professor of Pediatrics, Ain Shams University for her suggestions, her great care and her guidance given through-out the work.*



ABBREVIATIONS

<	Less than
>	More than
↑	Increase
↓	Decrease
→	Normal
A.T.P.	Adenosine triphosphate
CK.BB	Brain type isoenzyme of creatine phosphokinase
C P K	Creatine phosphokinase
C.S.F.	Cerebro spinal fluid
C.S.	Cesarean section
ECG	Electrocardiogram
EEG	Electroencephalogram
hr	hour
HR	Heart rate
IM.	Intramuscular
I.V.	Intravenous
IPPB	Intermittent positive pressure breathing
Iu/L	International unit per liter
$PaCO_2$	Partial arterial carbon dioxide pressure
PaO_2	Partial arterial oxygen pressure

The aim of the work is to throwlight upon, the aetiology, pathophysiology, diagnosis, management, complication, prognosis and prevention of the asphyxia neonatorum.

CONTENTS

....

INTRODUCTION AND AIM OF THE WORK

Asphyxia neonatorum means failure of the newborn infant to establish spontaneous respiration immediately following complete delivery. This leads to a succession of serious consequences affecting various systems of the body, and unless adequate respiration can be quickly initiated, the newborn will die, or survive with permanent damage in the brain (Hutchison, 1975).

Asphyxia is the single most important perinatal cause of neurological morbidity in term infant (Volpe, 1981).

There are number of situations in which it is important to recognize the problem and act promptly among them in asphyxia. The first consequences of failure to breath is anoxia, which means; abscence or deficiency of oxygen, as reduction of oxygen (O_2) in body tissue below physiologic level, rapidly followed by the excess accumulation of carbon dioxide (CO_2) in the blood and by a fall in the blood pH (Hutchison, 1975).

It is important to understand the aetiological factors and physiological effects of asphyxia neonatorum which enable us to foresee an emergency and so reach a better state of management.

REVIEW

Aetiology of Asphyxia Neonatorum:

There are numerous causes of asphyxia neonatorum, the causes have in common anoxia of fetus or newborn before, during and after delivery.

Prenatal Asphyxia (Intrauterine Asphyxia):

In the great majority of cases the causes of asphyxia neonatorum have been operating before the birth of the baby (antepartum or fetal asphyxia). The probability of fetal anoxia can often, however, be deduced from the state of the mother before or during labor, so causes of fetal anoxia might be either in the fetus itself or in the mother (Hutchison, 1975).

Hutchison (1975), classify the prenatal anoxia into:

I- **Anoxic anoxia:** It means reduction of oxygen (O_2) in the fetus tissue due to interference with the O_2 supply, this may occur from maternal anoxia which arise from a decrease in oxygen tension in the mother's blood as in;

1- **Cardio pulmonary disease:**

As in congestive cardiac failure, pneumonia, bronchitis, and emphysema.

2- **Mismanaged anaesthesia:**

Mismanaged inhaled anaesthesia, often complicated by inhalation of vomit, during labor. There is also a great danger to the fetus if the mother's blood pressure is allowed to fall abruptly during spinal anaesthesia (Donald, 1957).

3- **Placental insufficiency:**

The oxygen supply from the mother to the fetus is entirely dependant upon the efficiency of placenta:

a- **Maternal toxemia and maternal hypertension:** lead to reduced placental blood flow and this may exist for some time before the onset of labor (Morris et al., 1955).

b- **Post maturity:**

Walker (1954), has found evidence of a diminished transfer of oxygen due to placental insufficiency which is more common in postmaturity.

c- **Premature separation of placenta:**

It leads to cut of fetal life line as in case of placenta praevia,and abruptio placenta (accidental hemorrhage).

d- **Prolonged and complicated delivery:** (Over strong uterine contraction):

The fetus is in danger during prolonged labor because the oxygen supply is reduced during every uterine contraction.

II- Anaemic anoxia: It exists when the fetal blood has a low percent of haemoglobin concentration, red cell count below the normal values, that the blood can not carry sufficient oxygen to meet the needs of the tissues. This may arise in Rnesus incompatibility or from fetal hemorrhage (Craig et al., 1969).

III- Stagnant anoxia: It means conditions due to interference with the flow of blood and it's transport of oxygen to the fetus as in compression, prolapse or knotting of the umbilical cord.

IV- Histotoxic anoxia: Arises when the fetal tissues, especially the nervous system are so poisoned by drugs or damaged by hemorrhage that the cells can no longer utilize the oxygen delivered to them. Every drug which has so far been devised for the relief of pain during labor is capable of depressing the fetal respiratory centre if unskilfully used. The danger of certain drugs as: pethedine, trichloraethylene, morphine, hyoscine and related drugs are particularly great during premature labor. (Myers and Myers, 1979).

Natal and Postnatal Asphyxia:

It is important to know that the causes of fetal anoxia often produce the very circumstances in which further anoxia becomes inevitable during or immediately after birth (Hutchison, 1975).

Some of the most important natal and postnatal causes are:

I- Obstruction of the respiratory passages:

When anoxia occurs the fetus makes vigorous gasping movements in utero so that his lungs may be deeply filled with irritating meconium. (Hutchison, 1975).

Obstruction may occur by mucous or meconium inhaled during or after labor by the anoxic fetus. There may be massive aspiration of meconium that can not be removed by suctioning (James, 1983).

Inhalation of infected liquor by the infant may cause pneumonia before birth (Brown and Valman, 1979). The intrauterine pneumonia with organization of exudate may cause obstruction, the infant are usually severely depressed at birth (James, 1983).

II- **Prematurity:**

Fetal anoxia occurs in abruptio placenta is a common cause of premature labor. The premature birth leads to asphyxia due to:

a- **Respiratory centre of the premature** newborn is immature and functionally inadequate. It may indeed, have been further depressed by fetal anoxia, drugs administered to the mother or by raised intracranial pressure due to hemorrhage or cerebral edema.

b- **Soft unduly pliable thoracic cage:** The premature newborn often suffers a soft thoracic cage surrounded by a weak musculature so that he is unable to produce the negative intrathoracic pressure required to expand his immature lungs.

(Fanarrof and Martin, 1983)

III- **Delivery by Cesarean Section (C.S):**

Cesarean section still has a significantly greater risk compared to vaginal delivery. The incidence rate of respiratory disease still was twice as high as after vaginal delivery (Hjalmarson et al., 1982). Neonatal respiratory disease after C.S. has been related to an increased lung water content due to an insufficient clearance of fluid from the potential airways owing to lack of thoracic compression during the terminal phase of vaginal delivery (Adams et al., 1971).

Milner et al. (1978), showed that, somtimes the reasons for performing the C.S. may present problems for the neonate, but with all section deliveries the posibility of aspiration of amniotic fluid or retension of lung fluid may cause respiratory difficulty. Aspiration may occur as the result of tactile stimulation initiating gasping, lung fluid may be retained due to lack of thoracic compression at delivery.

However, (Kitterman et al., 1979) showed that secretion of liquid from lungs of fetal lambs decreased already before spontaneous, vaginal delivery and even before the onset of labor, this may be mediated by catecholamines due to stress of labour.

Bland et al. (1979), observed that in rabbits, delivered by C.S., the lungs contained more water than after vaginal delivery only if the animals had been killed before labor. These studies if valid in humans-may indicate that the observed, unfavourable effect of elective C.S. was due not to the operation and the lack of thoracic compression but to the intervention before the onset of labor.

IV- **Breech presentation:**

Asphyxia occurs with breech delivery due to:

1- **Rapid delivery of the head** with risk of damage to skull membranes by sudden compression and release. Also intracranial hemorrhage may occur from the rupture of tentorium cerebelli or falx cerebri due to rapid moulding of the head.

2- **After coming head and inhalation:**

The placenta separates in the second (2nd) stage of labor, as the active uterus contracts and the fetal head is in the pelvis, with liability of inhalation of blood and mucous. (Garry et al., 1974).

Not all breech presentation will be delivered vaginally, so that problems related to the C.S. may be encounted (Brauer, 1975).

Breech delivery is more dangerous to the baby than the C.S. (Donald, 1979).

Rates of asphyxia in breech delivery by C.S. have remained the same and continue to be much higher than asphyxia rates in vertex deliveries, which emphasizes the fact that the

risk inherent to the maneuvers of extracting a breech by C.S., is similar to that associated with the delivery of a breech via the vaginal route (Collea, 1980). The breech delivery is associated with higher incidence of long term brain damage (Fianu and Joelsson, 1979; alexopoulos, 1973).

V- Congenital anomalies which can lead to asphyxia in newborn:

a- Choanal atresia:

It is a bony or membranous obstruction between the nose and pharynx. It is suspected if respiratory difficulty is present and persists after pharyngeal suctioning. Clinically although the airway from the mouth to the larynx is patent this anomaly may result in complete respiratory obstruction and asphyxia, since infants are obligatory nose breathers. Choanal atresia usually presents as respiratory distress or apnea after the onset of breathing at 5-10 minutes of age (James, 1983).

Diagnosis of choanal atresia can be done by inserting a catheter first into one nostril and then into the other, then ventromedially along the floor of the nose (James, 1983). Also by lacking of movement of a wisp of cotton wool held at the nostril on each side (Brown and Valman, 1979).