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NEONATAL AND PEDIATRIC MECHANICAL VENTILATORY SUPPORT

Essay

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in Anaesthesia and Intensive Care

by

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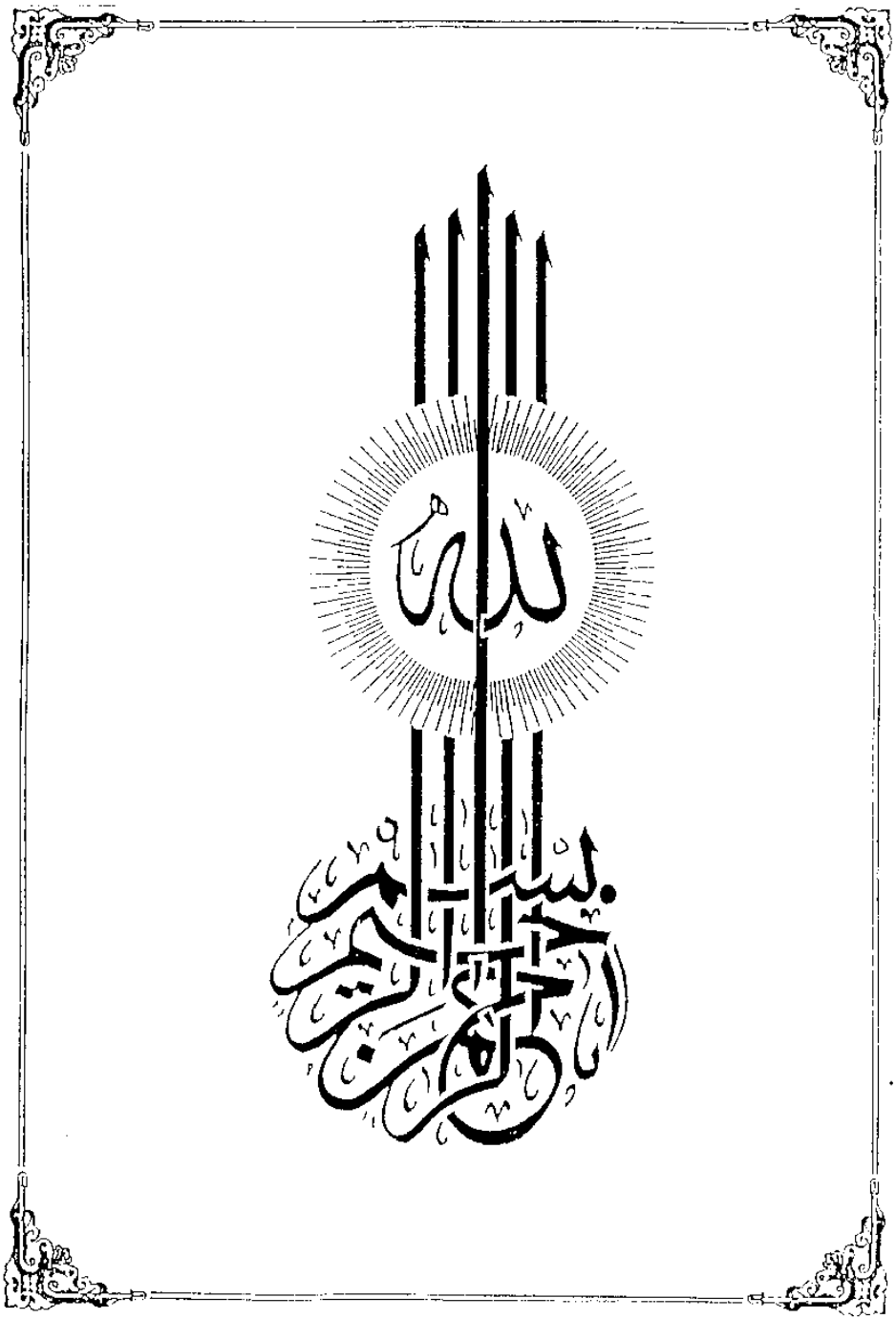
بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

سورة البقرة – الآية ٣٢




To my parents,
my sister,
and my fiance

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INTRODUCTION

Introduction

The modern era of infant and paediatric mechanical ventilation may be dated from the 1953 report of Donald and Lord, who described their experience with a patient-cycled, servo controlled respirator in treating several newborn infants with respiratory distress.

In the three decades following Donald and Lord's pioneering efforts, the field of neonatal and paediatric ventilation made dramatic advances. Because of the epidemic poliomyelitis in the 1950's, experience was gained with the tank-type negative pressure ventilators of the Drinker design ***(Kirby, 1981)***.

In 1959, Smyth and co-workers reported the successful use of intermittent positive pressure ventilation in the treatment of tetanus neonatorum.

Modern paediatric respiratory care required a major institutional commitment in resource allocation for the state of the art management of the mechanically ventilated patient ***(Fuhrman and Zimmerman, 1992)***.

Safe and effective assisted ventilation of the newborn infant and paediatric patient requires an understanding of the disease state and the likely course of the disease being treated. A knowledge of certain concepts of pulmonary physiology, pathophysiology, and flow mechanics should be understood before establishment of mechanical ventilation. Successful management depends upon awareness of the advantages and disadvantages of various modes of mechanical ventilation ***(Harris, 1988)***.

The outcome of cases of respiratory failure depends primarily on early detection of cases, determination of the causative condition

as well as tiding the neonate and paediatric patient over the critical period of distress *(Goldsmith and Karotkin, 1988)*.

As a result of improved devices, new techniques and better support systems, weigh-specific survival rates for infants dependent on ventilation have dramatically improved *(Goldsmith and Karotkin, 1988)*.

Death and damage can result from ignorant; improper, insensitive and often unnecessary use of available technology. Mostly, if not all, poor outcomes arise because we have triggered the complex, dangerous warheads of mechanical ventilation. Moreover, some authors concluded that poor outcomes are largely our doing, they usually result from what we do, not what we do not do *(Goldsmith and Karotkin, 1988)*.

So a team approach between the physician, the nurse, the respiratory therapist, and the family is required, where the role and responsibilities of each of the team members is clearly defined and respected *(Fuhrman and Zimmerman, 1992)*.

Based on the above mentioned facts, the idea is to focus on the recent advances and mechanical ventilatory strategies in various diseases of newborn and paediatric patients. Recent modes of ventilation of the newborn will be mentioned in contrast to the old techniques of conventional mechanical ventilation, together with their advantages and disadvantages, to which every team member must become familiar so that they can be applied safely and effectively.

PHYSIOLOGY AND DEVELOPMENT OF THE RESPIRATORY SYSTEM

Physiology and Development of the Respiratory System

Lung Development and Maturation :

Respiratory failure is one of the most common problems of the newborn period, especially in the premature infant. The reasons for this are several, but most are related to the incomplete structural and biochemical development of the respiratory system ***(Hodson and Belesky, 1975).***

Between 17 and 24 weeks of gestation, the development of the lung is in a canalicular stage characterized by elongation of the subdivisions of airways and appearance of cuboidal cells in the epithelium. The mesoderm becomes thinner, and toward the end of this stage, a proliferation of capillaries comes close to the air spaces for the first time. The terminal sac period begins at 24 weeks' and lasts until 40 weeks' gestation. This stage of development is characterized by approximation of the blood capillaries to the respiratory epithelium and differentiation of the epithelial cells into their mature forms. At 24 weeks, it is already possible to identify type I and type II cells lining the air spaces. This is the earliest stage at which gas exchange can occur in the lung, but the thickness of the tissue between the airspaces and capillaries is two to three times that of adult. By 30 - 32 weeks, the terminal bronchioli which eventually give origin to several elongated saccules. These continue to subdivide until term and give origin to several elongated saccules. These continue to subdivide until term and give origin to alveolar sacs. Between 24 and 32 weeks' gestation, the lung gradually increases its functional surface and capacity for gas exchange. The production of surfactant also begins at 23 - 24 weeks' gestation, first appearing at the same time oesinophilic bodies are seen in type II

cells. However, it is only after 28 - 30 weeks that surfactant appears in sufficient quantities to be detected in the amniotic fluid ***(Gluck and Kwlovich, 1973)***. At this time, the risk for developing respiratory distress syndrome decreases rapidly ***(Farrell and Avery, 1975)***.

Both the structural and biochemical development of the lung can be accelerated by corticosteroids. Steroid levels in the foetus can be increased by administration to the mother or by conditions that produce foetal stress, e.g., placental insufficiency, prolonged rupture of membranes, or active labour. Other substances, e.g., aminophylline and thyroid hormone, also accelerate foetal lung maturation ***(Liggins and Howie, 1972)***.

Physiology :

Pulmonary Foetal Circulation :

The medial smooth muscle of the precapillary pulmonary arterioles is quite thick and extremely sensitive to low arterial oxygen tension that produces vasoconstriction of these vessels. Maintenance of an elevated resistance is also achieved by tonic arteriolar vasoconstriction regulated by sympathetic nerves as well as by tortuosity and kinking of these vessels in the collapsed lung. Therefore, only a very small fraction of the right ventricular output passes through the lung because of high pulmonary vascular resistance, the presence of patent ductus arteriosus and low-resistance placental component of the systemic circulation ***(Rudloph and Heyman, 1974)***.

Since about two thirds of the right ventricular output of the foetus goes from the pulmonary artery to the aorta through the ductus arteriosus and since the ductus arteriosus is widely opened, pulmonary and systemic arterial pressures are approximately equal

at that time, and the pulmonary vascular resistance in the foetal lung (fluid-filled lung) is of the same orders of the systemic resistance *(Rudloph and Heyman, 1974)*.

Changes in Foetal Circulation and Respiration at Birth :

Aeration of Lungs at Birth :

Replacement of lung liquid by air is accomplished within few minutes of birth probably under the influence of the sympathetic activation with labour *(Largercrantz, 1987)*. Some liquid is squeezed out under the high vaginal pressure during the 2nd stage of delivery while the majority is absorbed into the pulmonary lymphatics and capillaries *(Srang, 1977)*.

High pressures are initially required to draw air into the lung to overcome the high flow resistance and inertia of the liquid in the airways, the elastic recoil of the lungs and the surface tension at the air-liquid interface. The pressures required to overcome surface tension are inversely related to the radius of curvature so they are higher in the small bronchioles and alveoli than in the trachea. Also, these pressures are found to be affected by presence or absence of surfactant *(Avery and Mead, 1959)*.

Circulatory Changes at Birth :

Aeration of the lungs would be futile if it were not matched by an increase in pulmonary blood flow. During foetal life, only 10% of the cardiac output passes through the lungs before birth. Clamping the umbilical cord leads to increase in peripheral resistance, finally the infant gasps and lung expand *(Ganong, 1983)*.

Once the lungs are expanded, the pulmonary vascular resistance falls dramatically. Based on experiments, approximately 1/3 of the pulmonary vasodilatation is due to mechanical effects of