

POST-OPERATIVE COMPLICATIONS
IN PAEDIATRIC SURGERY DUE TO ANAESTHESIA

Essay

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TABLE OF CONTENTS

	PAGE
INTRODUCTION	1
PAEDIATRIC ANATOMY AND PHYSIOLOGY	
IN RELATION TO ANAESTHESIA	2
I. Central Nervous System	2
II. Respiratory System	4
III. Cardiovascular System	9
IV. Physiology of Thermal Homeostasis	13
V. Metabolism	15
VI. Composition and Regulation of Body Fluids	17
PHARMACOLOGIC DIFFERENCES BETWEEN	
ADULTS AND CHILDREN	19
I. Premedicants and Parenteral Anaesthetics	21
II. Inhalation Anaesthetics	23
III. Muscle Relaxants	25
POST-OPERATIVE COMPLICATIONS RELATED	
TO ANAESTHESIA	30
I. Post-operative Respiratory Complications	32
II. Post-operative Circulatory Complications	53
III. Post-operative Fluid and Electrolyte Disturbances	60
IV. Post-operative Renal Complications	67
V. Post-operative Neuropsychiatric Complications	73
VI. Post-operative Gastrointestinal Complications	84

27

PREVENTION OF POST-OPERATIVE COMPLICATIONS	
"GENERAL APPROACH"	90
I. Preoperative Assessment	91
II. Preoperative Preparation of the Child for Hospitalization, Anaesthesia and Surgery	92
III. Anaesthetic Technique	96
IV. Intraoperative Support	99
V. Post-operative Care	102
SUMMARY	108
REFERENCES	110
ARABIC SUMMARY	

INTRODUCTION

INTRODUCTION

*"Whenever I approach a child
his presence inspires two feelings in me;
affection for what he is now,
and respect for what he may one day become."*

Louis Pasteur

This should be our concept, as anaesthesiologists, on dealing with the paediatric sector of the population. We need to conduct an anaesthetic which provides the surgeon with ideal operating conditions which results in a child in the best possible state in the postoperative period.

Complications noted after surgery and anaesthesia may be categorized epidemiologically into those related to the surgical procedure, to the patient's medical condition, and to the anaesthetic. It is an important goal to diagnose the complications early, the second goal is to treat them efficiently and correctly, but it is the third and final goal to prevent such complications altogether. Anaesthesia for paediatrics requires an understanding of anatomic, physiologic, and pharmacologic differences from adults and how these differences affect the anaesthesiologist's ability to provide adequate operative conditions and homeostasis intra- and post-operatively. The success of paediatric anaesthesia also depends on meticulous attention to detail and an intimate knowledge of the pathological processes involved. Errors and lapses do account for some, perhaps a majority of "anaesthetic deaths" and at least half of anaesthesia-related mortality and serious neurologic adverse outcomes are preventable. However, anaesthesia is not without risk. There is inherent toxicity in the drugs and techniques we use, which will likely always be so that not every anaesthetic death need necessarily be due to an error or lapse. To those who say that because anaesthesia is not therapeutic, nothing less than zero risk should be tolerated, we retort that although anaesthesia may not be therapeutic, it most certainly is beneficial. To every benefit there is a risk. The concept of risk versus benefit is applicable to anaesthesia.



ANATOMY AND PHYSIOLOGY

PAEDIATRIC ANATOMY AND PHYSIOLOGY IN RELATION TO ANAESTHESIA

Anaesthesia for neonates (0 to 30 days of age), infants (1 to 12 months of age), and children (1 to 12 years of age) requires an understanding of anatomic, physiologic, and pharmacologic differences from adults and how these differences affect the anaesthesiologist's ability to provide adequate operative conditions and homeostasis intra- and post-operatively (*Gregory, 1986*).

Some of the differences result from the fact that the infant at birth has to possess all the machinery for changing from one environment to a totally different one, some are the result of such a small creature requiring special protective mechanisms to meet a hostile world and yet others are merely the result of adult functions being as yet underdeveloped. Thus the child, especially the neonate, should not be regarded merely as an incomplete adult, but rather as a different organism (*Rees, 1980*).

I. Central Nervous System

A. Anatomy

The intact skull is less rigid in infants than in adults. As a result, an increase in the volume of the contents (blood, cerebrospinal fluid, and brain tissue) can be accommodated to some extent by expansion of the fontanelles and separation of the suture lines. Thus, palpation of the fontanelle can be used to assess the intracranial pressure in infants (*Steward, 1985*).

The spinal cord extends to the third lumbar vertebra at birth. When the child is one year old, the spinal cord takes its permanent position, ending at the first lumbar vertebra (*Snow, 1982*).

B. Physiology

The central nervous system in the newborn differs from that in the older child in three ways: Myelination of nerve fibers is incomplete, muscle tone and reflexes are different, and the cerebral cortex is less developed (its cellular elements increase during the first years of life).

The ability of neonates to appreciate pain is undefined. Babies react to a painful stimulus but apparently can not determine the site of the pain. The pain threshold for older children has been stated to be lower than that for adults. In view of our lack of knowledge of pain sensibility and the possible adverse neuroendocrine effects of continuing pain, we should provide analgesia for babies with the same care as for adult patients (*Steward, 1985*).

Motor function in the newborn is dependent on gestational rather than postnatal age. That is an infant born at 28 weeks gestation who is 3 months old will exhibit motor responses similar to those of a full-term newborn. Although potentially modifiable by cortical influence, most neonatal motor behaviour is subcortically controlled, permitting normal motor behaviour in newborn infants with severe cortical damage (*Crone and O'Rourke, 1986*).

Children are susceptible to convulsions. This may be due to infant's lack of myelin, greater water content of the brain, a higher metabolic rate, or an immature inhibitory response pattern (*Show, 1982*).

Autoregulation of the cerebral blood flow is impaired in sick newborn infants. Thus blood flow is pressure-dependent, and increased blood pressure is transmitted to the capillaries. In the preterm infant, the cerebral vessels are very fragile, especially in the region of the germinal matrix overlying the caudate nucleus. Rupture of these vessels leads to intracerebral haemorrhage, which often

ruptures into the ventricular system causing intraventricular haemorrhage (IVH). Predisposing factors to IVH include hypoxia, hypercarbia, hypernatraemia, fluctuations in arterial or venous pressures, and possibly the excessive administration of hypertonic fluids (e.g., sodium bicarbonate) (*Tarby and Volpe, 1982*).

II. Respiratory System

The respiratory system is of very special interest to the anaesthesiologist. It is the route of administration of inhaled anaesthetic agents, and its function may be significantly altered during and after anaesthesia. Changes occur continuously from infancy to about age 12 as the system grows to maturity.

A. Anatomy

The dimensions of the nasal airway at birth are relatively greater than in adults, nasal resistance to airflow being only 40-50% of the total airway resistance compared to over 60% in the adult (*Stocks and Godfrey, 1978*). On the other hand, the anatomy of the pharynx at birth is similar to the more primitive herbivores which are obligatory nose-breathers with a highly developed sense of smell. The relatively large tongue is in contact with the palate, and this also discourages mouth breathing. During the first few weeks of life infants are thus at risk from severe respiratory distress should blockage of the nasal airway occur (*Hatch, 1985*).

The infant epiglottis lies at an angle of 45° to the posterior pharyngeal wall, and is a soft, short structure which is often folded in a "V" shape. The larynx itself lies with the glottic aperture more anterior than in the adult. It is situated opposite the lower border of the fourth cervical vertebra, descending to lie in the adult position opposite the fifth or sixth cervical vertebrae by the fourth year of life. Because of these differences, endotracheal intubation in the infant is best performed

using a straight-bladed laryngoscope placed posterior to the epiglottis. Even then the larynx may be difficult to visualize, especially in the presence of micrognathia, and firm backward pressure over it by an assistant may be required (*Hatch, 1985*).

The narrowest part of the child's upper airway is the subglottic area until about the end of the first decade of life. Because the cricoid forms a complete ring of cartilage, damage caused by an endotracheal tube in this area will produce oedema and possible fibrosis which narrows the airway. The average cross-sectional area at the cricoid ring at birth is 14 mm^2 , and a 1 mm ring of oedema or fibrosis will reduce this area by as much as 65% (*Sumner, 1984*), whereas in an adult this degree of oedema would not cause significant obstruction.

The trachea is short (approximately 5 cm in the neonate); therefore precise placement and firm fixation of endotracheal tubes are essential (*Steward, 1985*).

In small children under the age of three years the angulation of the two main bronchi at the carina is equal on both sides (*Linton, 1984*), making the incidence of either lung intubation the same.

Because the ribs are almost horizontal, the ventilation is primarily diaphragmatic. The abdominal viscera are bulky and can readily hamper diaphragmatic excursion, especially if the stomach or bowel is distended (*Steward, 1985*).

B. Physiology

Respiratory function in the infant and child differs in a number of ways from that in the adult. Unlike the adult, the child is a developing organism: central control of breathing follows neurologic development, while structural and

biochemical maturation of the lungs continues from foetal life throughout childhood, ending in late adolescence (*Crone and O'Rourke, 1986*).

Periodic breathing (rapid ventilation alternating with periods of apnoea lasting 5-10 sec) occurs in many preterm and some term infants. It is thought to result from incoordination with the feed-back loops controlling ventilation. During episodes of periodic breathing, the arterial carbon dioxide tension ($P_a\text{CO}_2$) is above normal but the heart rate does not change significantly. Periodic breathing seems to have no serious physiological consequences and usually ceases by 6 weeks of age. Some preterm infants demonstrate far more serious and indeed life-threatening episodes of apnoea. These commonly exceed 20 sec and are accompanied by bradycardia. Apnoeic episodes may represent an example of failure of the response to hypoxia. Because many apnoeic episodes occur during rapid eye movement (REM) sleep, it is possible that ventilatory muscle fatigue may be an aetiological factor as well as an impaired chemoreceptor response to hypoxaemia. Preterm infants must be carefully monitored to detect apnoeic episodes. Treatment is by tactile stimulation or bag-and-mask resuscitation. The incidence of apnoeic episodes is decreased by therapy with aminophylline (central stimulation) or by instituting continuous positive airway pressure (CPAP) (increased reflex activity of lung and chest wall reflexes) (*Rigatto, 1982*).

Because lung compliance is directly related to elasticity, lung compliance peaks in late adolescence; it is relatively low in the very young and the very old. The chest wall compliance of the infant (especially the preterm) is very high, and does not contribute significantly to respiratory motion. The net effect of the infant's compliant chest wall and poorly compliant lung is a lower resting volume or functional residual capacity (FRC) (*Crone and O'Rourke, 1986*).

In the term infant total lung capacity (TLC) is approximately 160 ml, and the FRC is about half this volume. The tidal volume (V_T) is approximately 16 ml, and the dead space volume (V_D) is about 5 ml (0.3 of the V_T). Relative to body size, all these values are similar to adult values. However, any dead space in anaesthesia or ventilator circuits is much more significant when related to the small volumes of the infant (i.e., a 5 ml apparatus dead space would increase the total effective V_D by 100%) (Steward, 1985).

In contrast to the static lung volumes, alveolar ventilation (V_A) is proportionally much larger in the newborn ($100-150 \text{ ml.kg.}^{-1}\text{min.}^{-1}$) than in the adult ($60 \text{ ml.kg.}^{-1}\text{min.}^{-1}$). This high V_A in the infant results in a V_A : FRC of 5:1, compared to 1.5:1 in the adult. Consequently the FRC is a much less effective "buffer" in the infant, so that changes in the concentration of inspired gases (including anaesthetic gases) are more rapidly reflected in alveolar and arterial levels (Steward, 1985).

It is postulated that the elastic tissue helps keep the airway open, so that the greater the elastic stroma in the small airways, the lower the lung volume required before gravitational forces can close small non-cartilagenous airways (Crone and O'Rourke, 1986). Therefore, the closing volume (CV) is higher in infants and young children than in young adults and may exceed the FRC to encroach on the V_T during normal ventilation. Airway closure during normal ventilation may explain the lower normal values for P_aO_2 during infancy and childhood. A fall in FRC, which usually occurs during general anaesthesia and persists into the postoperative period, may be expected to further increase the significance of the high CV, and further increase the difference between alveolar and arterial oxygen tensions ($P_{(A-a)}O_2$). The younger the infant or child, the larger is this fall in FRC. Hence there is a requirement to increase the oxygen concentration of inspired gases. The peri-

operative fall in FRC may be less during operations with the patient prone and the abdomen hanging free, and it may be partially reversed by CPAP (*Steward, 1985*).

The total surface area of the air-tissue interface of the alveoli is small in the infant (2.8 m^2). When this area is related to the metabolic rate for O_2 ($\dot{V}_{\text{O}_2}$) it is apparent that the infant is dependent on a smaller air-tissue interface: $\dot{V}_{\text{O}_2}$ ratio than the adult and as a result has a reduced reserve capability for gas exchange (Table 1). This may assume great significance if congenital defects (e.g., diaphragmatic hernia) interfere with lung growth or the lungs become damaged (e.g., from meconium aspiration). Then the remaining healthy lung tissue may be inadequate to sustain life (*Steward, 1985*).

The oxygen requirement ($\dot{V}_{\text{O}_2}$) of neonates ($6\text{--}12 \text{ ml.kg}^{-1}$) is at least twice that of older patients (*Lister et al., 1974*). The requirement for oxygen increases if neonates have cardio-respiratory disease or are exposed to cold. The $\dot{V}_{\text{O}_2}$ of spontaneously breathing patients with chronic lung disease is three times greater than that during mechanical ventilation. Because of these difference in the respiratory system, the ventilation of patients under 6 months of age should be controlled during anaesthesia and surgery (*Gregory, 1986*).

The infant is a very metabolically active organism: Oxygen consumption at birth is $6 \text{ to } 8 \text{ ml.kg}^{-1}\text{min}^{-1}$ and falls to $5 \text{ to } 6 \text{ ml.kg}^{-1}\text{min}^{-1}$ over the first year of life. The decreased ventilation/perfusion ratio, the decreased P_{50} (O_2 tension at which haemoglobin gives up 50% of its O_2) of foetal haemoglobin and the physiologic anaemia characteristic of infants can make adequate oxygen delivery difficult. The infant compensates for this by having a very high minute ventilation per body weight and initially a high cardiac output up to $250 \text{ ml.kg}^{-1}\text{min}^{-1}$, which remains elevated until 4 to 5 months of age, at which point total haemoglobin levels reach childhood levels (*Oski, 1973*). When oxygen delivery to the tissues is