IJFANS INTERNATIONAL JOURNAL OF FOOD AND NUTRITIONAL SCIENCES
ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, Iss 10,Oct 2022

Cardiopulmonary and Cardiovascular Physiology in Children

Dr. Rinku Garg[1*], Dr. Jyoti Batra[2], Dr. Deepika Agarwal[3], Aarushi Batra[4]

[1*]Professor, Department of Physiology, Santosh Deemed to be University, Ghaziabad
[2]Professor, Department of Biochemistry, Santosh Deemed to be University, Ghaziabad
[3]Professor, Department of Community Medicine, Santosh Deemed to be University, Ghaziabad
[4]MBBS final year, LLRM Medical College Merrut, UP

Corresponding Author: [1*]Dr Rinku Garg

ABSTRACT

In comparison to adults, children have a higher risk of perioperative respiratory and cardiovascular problems due to their distinct respiratory and circulatory physiology. Due to their inability to control breathing and innate sensitivity to quick desaturation, airway obstruction, early respiratory fatigue, and lung atelectasis, anaesthesia can exacerbate respiratory decline in young children. Premature newborns (less than 60 weeks postconceptional age) are at risk for persistent apnoea and the resulting deterioration of respiratory function. In neonates, the transitional phase of circulation is susceptible to reverting to persistent foetal circulation. In comparison to older children and adults, the myocardium and autonomic regulation of the heart are young and distinct in neonates and babies, making them susceptible to life-threatening haemodynamic alterations during the perioperative period. In this review, we examine the variations in respiratory and cardiovascular physiology between neonates, babies, and younger children and older children and adults. We concentrate primarily on the transitional physiology of the respiratory and circulatory systems in neonates and infants, as well as the detrimental changes that can occur during anaesthesia or perioperative care.

Keywords: Cardiovascular physiology, children, foetal circulation, respiratory physiology

1. INTRODUCTION

Cardiovascular and respiratory systems undergo significant changes during foetal, neonatal, infant, and early childhood development. hence respiratory .The cardiovascular physiology of young children, particularly newborns and infants, is distinct from that of older children and adults. Young children are more susceptible to anesthesia-related critical episodes and potentially cardiac arrest due to these differences. [1,2]

As the majority of anesthesia-related deaths in children are due to unintended respiratory (airway-related) and cardiocirculatory events,[1] understanding the key developmental changes of these two important organ systems from birth makes anaesthesia safer for infants and toddlers. To apply appropriate anaesthetic principles and improve perioperative outcomes

IJFANS International Journal of Food And Nutritional Sciences
Official Publication of International Association of Food and Nutrition Scientists

Research paper UGC CARE Listed (Group -I) Journal Volume 11, Iss 10,Oct 2022

in paediatric patients, it is necessary to recognise the differences in cardiovascular and respiratory physiology between different groups of children and adults.

Initiation of Breathing

Prenatal lung tissue is compressed and loaded with fluids. The initial inhalation produces a high negative inspiratory pressure to commence lung function. Expansion by overcoming opposing forces, such as airway resistance, fluid inertia in the airway, and the surface force of airfluid interface in alveoli.[4] The resulting active pressure gradient moves lung fluids into interstitial tissue, where lymphatics and pulmonary circulation remove them gradually. Delayed clearance of lung fluids, which can occur in some children, can result in "transient tachypnea of infant" that can last between 24 and 72 hours. Surfactant, which is secreted near the end of pregnancy, reduces the air-liquid interfacial tension and "opening pressure" required for aerating the alveoli and preventing alveolar collapse. The generation of surfactant is adequate in neonates delivered after 35 weeks of gestation. [5-6] It may be necessary to administer exogenous surfactant to premature infants in order to aid normal lung breathing.

Regulation of Breathing

At birth, respiratory control is rudimentary and requires several weeks to months to develop.The ventilatory response of neonates to hypoxia and hypercarbia is compromised. After a brief initial increase, the respiratory rate of premature newborns drops and frequently leads in apnoea under hypoxia. [7] During the first week of life, term infants exhibit a similar pattern of reaction to chronic hypoxia, which is followed by a protracted increase in breathing. Infants have a respiratory pattern that is both irregular and periodic. Periodic breathing is distinct from clinical apnoea and is frequently observed in both preterm and term newborns. Apneas that threaten life are common, especially in premature newborns.[8] In newborns younger than 60 weeks post-conception, prolonged apnoea lasting longer than 20 seconds is prevalent during the first 12 hours following surgery (often within 2 hours). In many cases, the danger continues for more than twelve hours, necessitating vigilant monitoring of these high-risk newborns for at least twenty-four hours in a tertiary care setting. If surgery cannot be postponed in such newborns, intraoperative prophylactic intravenous caffeine (10 mg/kg) or theophylline (8 mg/kg) may be administered to lessen the risk of postoperative persistent apnea. In appropriate surgical situations, regional anaesthesia techniques without severe sedation should be preferred. It is rational to employ short-acting anaesthetics (such as sevoflurane or desflurane) and opioids (fentanyl or sufentanyl) when general anaesthesia is chosen. Certain newborns may require methylxanthines (theophylline or caffeine), continuous positive airway pressure (CPAP), or possibly mechanical ventilation. [9] Afferent laryngeal stimulation during laryngoscopy or excessive lung inflation (Herring–Breuer inflation reflex) also contribute to apnoea in infants.

Cardiovascular Physiology in Children

The cardiovascular architecture and physiology of infants evolve fast within a few days to several weeks after birth, reaching adult levels later in childhood. As neonates develop, the circulatory channel changes, the myocardium gradually matures, and the autonomic regulation of the heart evolves. These major changes distinguish the cardiovascular physiology of newborns and babies from that of older children and adults.[9-11] To create a

Research paper UGC CARE Listed (Group -I) Journal Volume 11, Iss 10,Oct 2022

safe anaesthetic approach that helps to reduce perioperative morbidity and mortality in children, it is vital to recognise these distinctions .

Fetus Circuitry and its Transition to Adult Type

The shift from foetal to neonatal to adult circulation is complicated due to the considerable changes in circulatory anatomy and physiology that occur.For the infant to become independent of the mother (placenta) for oxygen and other nutrients in the extrauterine environment, a smooth and effective transition is required. During the transitional phase, the delayed or difficult transfer of foetal circulation or return of the neonatal circulation to persistent foetal circulation has various anaesthetic implications.

Foetal Circulation

As the lungs stay deflated and filled with liquids during foetal life, pulmonary vascular resistance is particularly high. The single umbilical vein transfers oxygen from the mother's blood via the placenta to the developing foetus. Approximately half of this umbilical venous blood enters the inferior vena cava via the ductus venosus, bypassing the liver. This oxygenated blood stream, once entering the right atrium, is guided preferentially into the left atrium via the foramen ovale and subsequently into the left ventricle. [12] This blood is ejected by the left ventricle into the aorta and subsequently distributed to the brain, myocardium, and upper body. Deoxygenated blood drained from the upper body enters the right atrium via the superior vena cava and flows preferentially through the right ventricle through the tricuspid valve, into the right ventricle. 810% of total cardiac output flows via high vascular resistance pulmonary circulation. [13-15] A portion of the blood in the descending aorta nourishes the lower half of the body, while the remaining portion is reoxygenated in the placenta via two umbilical arteries.

Thus, the foetal circulation is parallel. The left ventricle serves the brain, the heart, and the upper half of the body with welloxygenated placental blood, whereas the right ventricle supplies the lower half of the body and the placenta with blood with a lower oxygen tension.

Transitional Circulation

The change from foetal (parallel) to postnatal (series) circulation begins during delivery, following lung inflate (first cry) and umbilical cord expulsion of the placental circulation (umbilical cord clamping). Inflation of the lungs decreases the high pulmonary vascular resistance, whereas the elimination of the low-resistance placental circulation increases the systemic vascular resistance. These modifications improve pulmonary blood flow and promote blood return to the left atrium.

Consequently, left atrial pressure rises. Ductus venosus restricts and reduces blood flow to the right atrium via inferior vena cava, resulting in a drop in right atrial pressure. Thus, the pressure gradient between the left and right atrium reverses, resulting in the functional closure of the foramen ovale over the next several breaths. [16-18]

Oxygen Carriage

At birth, neonatal blood contains more foetal than adult haemoglobin (HbF) (HBA).HbF has a greater oxygen affinity than HbA. Thus,Despite having increased haemoglobin levels, infants are susceptible to hypoxia due to decreased tissue oxygen delivery. During the neonatal period, however, the concentration of 2,3 diphosphoglycerate

increases and the oxygen dissociation curve moves to the right. These modifications reduce HbF's affinity for oxygen and enhance tissue oxygen delivery in babies.

About six months after birth, the production of foetal haemoglobin ceases totally and is completely replaced by adult haemoglobin.[18-20] Due to the low level of circulating haematopoetin throughout early childhood, HbA is synthesised more slowly than HbF is eliminated from circulation. Around 912 weeks of age, an increase in HbF loss, a decrease in HbA synthesis, and a relative rise in circulating blood volume result in a drop in total haemoglobin and physiological anaemia in term newborns.[21-22] The decrease in haemoglobin level is proportional to the gestational age of preterm newborns. They also reach their lowest haemoglobin level earlier than term newborns (between 4 and 8 weeks of age). However, as infants develop, the affinity of oxygen for haemoglobin decreases, resulting in increased oxygen unloading at the tissue level. In terms of tissue oxygen supply, a lower haemoglobin concentration in newborns and young children is equivalent to a higher concentration in adults. [23-25]

2. CONCLUSION

Young children's respiratory and cardiovascular physiology differs from that of older children and adults, making them more susceptible to perioperative respiratory and cardiocirculatory problems. Neonates and newborns, particularly premature neonates, exhibit immature respiratory control, ineffective inspiratory muscles, distinct airway and lung mechanics, and a greater basal metabolic need from oxygen Under anaesthesia, undiagnosed apnea or airway obstruction, respiratory exhaustion, or lung atelectasis may cause fast respiratory deterioration in these children. Hypoxia, hypercapnia, acidosis, or electrolyte abnormalities may cause neonates' transitional circulation to revert to persistent foetal circulation by increasing pulmonary vascular resistance. Young children have diminished cardiac reserves and output dependent on heart rate. During anaesthesia, they have a poor tolerance for depression of cardiac contractility and alterations in systemic vascular resistance or circulatory volume. In response to several painful and autonomic stimuli, predominant parasympathetic regulation of the heart frequently induces bradycardia and its detrimental effects in neonates and young babies. Despite having a higher haemoglobin level, newborns are susceptible to hypoxia due to a higher HbF content in their blood, which causes poorer oxygen delivery at the tissue level. Therefore, anesthesiologists must recognise these distinctive features of respiratory and cardiovascular physiology in young children, particularly in neonates and infants, and develop a safe and effective anaesthetic plan to prevent perioperative morbidity and mortality in children.

Acknowledgement
Conflicts of interest

3. REFERENCES

1. Ford S, Calvert J. Adaptation for life: A review of neonatal physiology. Anaesth Intensive Care Med 2009;9:84-9.
2. Roberts D, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. Cochrane Database Syst Rev 2003;3:CD004454.
3. Rigatto H, Kalapesi Z, Leahy FN, Durand M, MacCallum M, Cates D. Ventilatory

Research paper UGC CARE Listed (Group -I) Journal Volume 11, Iss 10,Oct 2022

response to 100% and 15% O2 during wakefulness and sleep in preterm infants. Early Hum Dev 1982;7:1-10.

4. Carroll JL, Agarwal A. Development of ventilatory control in infants. Paediatr Respir Rev 2010;11:199-207.
5. Mathew OP. Apnea of prematurity: Pathogenesis and management strategies. J Perinatol 2011;31:302-10.
6. Wheeler M, Cote CJ, Todres ID. The pediatric airway. In: Cote CJ, Lerman J, Todres ID, et al. editors. A Practice of Anaesthesia for Infants and Children. 4th ed. Philadelphia, PA: Saunders Elsevier; 2009. p. 237-73.
7. Numa AH, Newth CJ. Anatomic dead space in infants and children. J Appl Physiol 1996;80:1485-9.
8. Moss ML. The veloepiglottic sphincter and obligate. Nose breathing in the neonate. J Pediatr 1965;67:330-1.
9. Langston C, Kida K, Reed M, Thurlbeck WM. Human lung growth in late gestation and in the neonate. Am Rev Respir Dis 1984;129:607-13.
10. Stocks J, Godfrey S. Specific airway conductance in relation to postconceptional age during infancy. J Appl Physiol 1977;43:144-54.
11. Zauder HL. The Macintosh laryngoscope blade. Anesthesiology 2005;102:241-2.
12. Marciniak B. Growth and development. In: Cote CJ, Lerman J, Todres ID, et al. editors. A Practice of Anaesthesia for Infants and Children. 4th ed. Philadelphia, PA: Saunders Elsevier; 2009. p. 7-21.
13. Schramm C, Grunstein M. Pulmonary function tests in infants. In: Chernick V, Boat T, Wilmott R, Bush A, editors. Kendig's Disorders of the Respiratory Tract in Children. New York: Elsevier Saunders; 2006. p. 129-67.
14. Blackburn S. Placental, fetal and transitional circulation revisited. J Perinat Neonatal Nurs 2006;20:290-4.
15. Schmidt KG, Silverman NH, Rudolph AM. Assessment of flow events at the ductus venosus-inferior vena cava junction and at the foramen ovale in fetal sheep by use of multimodal ultrasound. Circulation 1996;93:826-33.
16. Rudolph AM. Distribution and regulation of blood flow in the fetal and neonatal lamb. Circ Res 1985;57:811-21.
17. Sharma A, Ford A, Calvert J. Adaptation for life: A review of neonatal physiology. Anaesth Intensive Care Med 2010;12:85-90.
18. Miller-Hance WC, Wilmot I, Andropoulos DB. Developmental physiology of the cardiovascular system. In: Gregory GA, Andropoulos DB, editors. Gregory's Pediatric Anesthesia. 5th ed. West Sussex, UK: Wiley Blackwell; 2012. p. 60-94.
19. Singh Y, Tissot C. Echocardiographic evaluation of transitional circulation for the neonatologists. Front Pediatr 2018;6:140.
20. Askin D. Newborn adaptation to extrauterine life. In: Simpson, Creehan, editors. AWHONN's Perinatal Nursing. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008. p. 527-45.
21. Bökenkamp R, DeRuiter MC, van Munsteren C, Gittenberger-de Groot AC. Insights into the pathogenesis and genetic background of patency of the ductus arteriosus. Neonatology 2010;98:617.

Research paper UGC CARE Listed (Group -I) Journal Volume 11, Iss 10,Oct 2022

22. Breall JA, Rudolph AM, Heymann MA. Role of thyroid hormone in postnatal circulatory and metabolic adjustments. J Clin Invest 1984;73:1418‑24.
23. Mahdavi V, Izumo S, NadalGinard B. Developmental and hormonal regulation of sarcomeric myosin heavy chain gene family. Circ Res 1987;60:804‑14. Rudolph AM. Fetal circulation and cardiovascular adjustments after birth. In: Rudolph AM, editor. Pediatrics. East Norwalk, CT: Appleton and Lange; 1987.
24. Anderson PA, Glick KL, Manring A, Crenshaw C Jr. Developmental changes in cardiac contractility in fetal and postnatal sheep: In vitro and in vivo. Am J Physiol 1984;247:H371‑9.
25. Klopfenstein HS, Rudolph AM. Postnatal changes in the circulation and responses to volume loading in sheep. Circ Res 1978;42:83945.