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myosin. The myofibrils are scanty, poorly defined and aligned peripherally with limited capacity of protein synthesis. The myocardium is composed of higher proportion of non-elastic and non-contractile proteins (60% versus 30% in the adult myocardium) which renders it less compliant as compared to the adult heart. There is a greater extent of Type I collagen in the newborn's myocardium which is more rigid as compared to the adult Type III collagen. The neonatal myocytes contain higher proportion of water than contractile elements accounting for their decreased contractility.

The cell replication process varies in the foetus as compared to the adult. Foetal cardiomyocytes demonstrate the ability to undergo hyperplasia in addition to hypertrophy while the adult mature myocytes can divide only by hypertrophy, i.e. increase in size. The cell dimensions and surface area of the myocytes rapidly increase in the postnatal life.

The functional limitations of the neonatal heart are largely attributed to its structural immaturity. The poor compliance of the myocardium leads to its limited cardiac reserve. The heart is unable to respond to an increase in volume on the Frank-Starling curve to an extent the adult heart would. This results in the cardiac output being dependent upon heart rate more than stroke volume. The adult mechanism of increasing stroke volume to cause a proportionate rise in cardiac output is more resourceful. But infants and children, on the other hand, expend more energy in the process of increasing cardiac output by increasing their heart rate during stressful situations.

The changes in ventricular pressure are more easily transmitted to the opposite ventricle in the immature myocardium. Also, the LV and RV diastolic filling is severely impaired. Limited compliance and equal masses of both the ventricles contribute to their interdependence. The pressure and volume overload experienced by both the ventricles at birth lead to their hypertrophy. The adult ratio of LV to RV mass of 2:1 is attained several months after birth. Also, the biventricular interdependence makes the heart more susceptible to myocardial depression when faced with adverse events such as hypoxia, acidosis or anaesthetic agents. The stiffer nature of the myocardium restricts it potential to respond adequately to fluid overload. Thus, the neonates poorly withstand any increase in preload, afterload or depressed myocardial contractility.

Another important point pertaining to calcium ions is worthy of mention here. Calcium ions are essential for the excitation-contraction coupling resulting in myocardial contraction. These ions diffuse into the t-tubules and are released when required to enhance contractility. The earliest evidence of longitudinal sarcoplasmic reticulum is seen by the beginning of second trimester, but t-tubules are visible only after birth. Due to this underdeveloped sarcoplasmic reticulum in the neonatal heart, there is a limited storage capacity of calcium. Thus the neonatal myocardium is more dependent on sodium calcium channels for calcium influx from the extracellular space. This explains the increased sensitivity of neonatal heart to hypocalcemia and drugs like digitalis, calcium channel blockers, etc. and its dependence on extraneous calcium sources. Also, the reduced Ca-ATPase enzyme activity on the sarcoplasmic reticulum results in a decreased calcium release and reuptake.

Lastly, the source of energy for myocyte metabolism in the neonate is lactate while in adults long chain fatty acids are favored. This difference is due to deficiency of enzyme carnitine palmitoyl transferase-1 which transports long-chain fatty acids into the mitochondria. The relative dependence of the neonatal myocardium on anaerobic metabolism offers a somewhat protective role against hypoxia. The fundamental differences in the cardiovascular functioning of the neonate and adult are summarized in Table 3.2.

AUTONOMIC REGULATION OF CARDIAC FUNCTION

The cardiovascular functioning is regulated by the nervous system so as to ensure its optimum performance under different physiological circumstances. The autonomic system plays a crucial role in the control of cardiovascular homeostasis. During foetal period, this autonomic regulation of the heart is undeveloped

Table 3.2 Functional differences in the neonatal and adult cardiovascular parameters

	Neonate	Adult
Cardiac output	HR dependent	SV and HR dependent
Compliance	Less	Normal
Starling response	Limited	Normal
Preload reserve	Limited	Normal
Afterload compensation	Limited	Effective
Ventricular inter-dependence	High	Relatively low
Myocardial metabolism Chief substrate	Anaerobic Lactate	Aerobic Long chain fatty acids

4

Essentials of Respiratory System in Infants and Children

Vaishali P Chaskar and Jalpa Arvind Kate

INTRODUCTION

The most common critical incidents related to respiratory system are seen in paediatric age group. There are anatomical and physiological differences between neonates, infants and child. Again they differ from adult respiratory system. The knowledge about development in respiratory system helps to provide appropriate anaesthesia to paediatric group and to reduce complication rates. The chapter will provide appropriate informations and also anaesthetic considerations in detail.

Key Points

1. At birth, control of ventilation is immature. This maturity occurs at 3 weeks of age in term baby.
2. Neonates and preterm babies are more prone for post operative apnoea episodes. Risk of postoperative apnoea is less after 1 month of age in term and 60 weeks PCA in preterm babies.
3. In first few weeks, response to hypercapnia is blunted.
4. In hypoxia, neonate responds with hyperventilation followed by apnoea. But the apnoeic response to hypoxia is suggestive of respiratory muscle fatigue or upper airway obstruction.

Developmental Changes

Developmental changes in all systems should be so sufficient to withstand drastic changes at the time of birth. All systems adapt the changes from gestational age and makes foetus to survive in external environment too. Cardiorespiratory adaptation is one of the very crucial adaptations. After the birth, within few minutes, neuronal drive and respiratory muscles must replace all liquid filled in lungs by sufficient amount of air. Therefore, gas exchange will take place. This chapter will provide a clear view of developmental and relevant aspects of respiratory systems and will also discuss about anaesthetic considerations in detail.

DEVELOPMENT OF LUNGS

Development of lungs (Fig. 4.1) starts in prenatal period and changes are as follows:[1,2]

1. Embryonic phase: Groove in ventral foregut, endoderm surrounding mesenchymal tissue to form lung buds.
2. Pseudoglandular phase: [Till 17 weeks of gestation (WOG)]—rapid budding of bronchi and lung growth, preacinar branching is complete. During this phase, any disturbance to free expansion leads to hypoplasia as occurs with *diaphragmatic hernia*.
3. Canalicular phase: (Till 24 WOG), development of respiratory bronchiole, and capillaries surrounding it.
4. Terminal sac period: (After 24 WOG)—appearance of clusters of air spaces (saccules), with thick and irregular septa.
5. (At 26–28 WOG) Capillary proliferation around saccules. In premature infants, it may be seen earlier at 24 WOG, hence they can survive in neonatal intensive care.
6. At 28 WOG: Thinning of saccular walls, lengthening of saccules with additional generation.
7. At 32 WOG: Alveolar formation starts from saccules. Most of alveolar formation occurs in 12–18 months of postnatal life.

However, morphologic and physiologic development of lungs continues during the first decade of life.

1. **Lung volume** in early postnatal period, is disproportionately small compared to body size (Fig. 4.2).

7. FRC further reduces 10–15% more in anaesthesia, apnoea and paralysis. In awake patient, FRC is maintained by sustained tonic activity of the inspiratory muscles.
8. The closing capacity is more than the FRC in infants and children leads to early airway closure.
9. Pulmonary diffusion in childhood is mainly affected by changes in surface area of alveolar capillary membrane. Thickness of blood gas barrier for diffusion declines in early gestational ages. Therefore, alvoeloar arterial oxygen difference is higher in term neonates and even higher in preterm.
10. Venous admixture is higher in infants than in adult. It is at 10–20% of cardiac output. While in adult, it is 2–5% of cardiac output.
11. In the newborn, blood oxygen affinity is extremely high and P50 is low (18 to 19 mmHg), because 2,3-DPG is low and foetal haemoglobin (HbF) reacts poorly with 2,3-DPG. Oxygen delivery at the tissue level is low despite high red blood cell mass and haemoglobin level.
12. Inhalation of warm air with 50% humidity maintains normal ciliary activity, whereas breathing dry air for 3 hours results in a complete cessation of mucus movement.

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