CONTINUING EDUCATION

Pediatric Review and Perioperative Considerations

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Perioperative management of pediatric patients demands knowledge of the relevant ways in which pediatric physiology and physiological regulation differs from the adult. This article will outline some of these most pertinent differences, and also present current recommendations for perioperative management.

Keywords: *pediatrics, anesthesia, pharmacology.*

OBJECTIVES—1. DISCUSS THE management of the pediatric patient in the perioperative setting; and 2. review the pharmacokinetics and pharmacodynamics of medications for the pediatric patient.

Pediatric patients vastly differ from adults anatomically, physiologically, cognitively, and socially. Familiarity with these differences assists providers to formulate patient-, needs-, and age-specific approaches to pediatric care and facilitates early recognition of abnormalities. Knowledge of pediatric attributes is essential to perioperative care, management, and outcomes of this patient population. The purpose of this article is to equip providers with fundamental pediatric knowledge to guide and improve their practice.

Cardiovascular Physiology

The cardiovascular system undergoes many changes after birth and continues to mature through adolescence. Changes include closure of the patent foramen ovale and patent ductus arteriosus (PDA). Complete closure of the PDA does not occur until 2 to 3 weeks of age. During those few weeks, neonates can return to fetal circulation because of hypoxia or vascular resistance.[1] At any stage of development, cardiac output (CO) is dependent on heart rate (HR) and stroke volume (SV) with SV dependent on preload, contractility, and systemic vascular resistance (SVR). These factors have different effects on CO as the cardiovascular system matures. In the neonate and infant, the ventricles are less compliant and respond poorly to excess preload. As a result, SV is relatively fixed, and CO is more sensitive to changes in HR. Conditions leading to decreased HR, such as parasympathetic stimulation, hypoxia, or excessive anesthetic depth lead to significant reductions in CO. Volume depletion is seen clinically as hypotension without tachycardia.[2]

Respiratory Physiology

Many differences exist between pediatric and adult patients when considering airway and respiratory form and function. Initially, life outside the uterus is not possible until 24 to 26 weeks of gestation, in large part because of the immaturity of the lungs.[1] Neonatal and infant lungs contain alveoli that are fewer in number and smaller in size, which results in reduced lung compliance. This results in a tendency toward chest wall collapse during inspiration

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and low residual volumes at expiration.[2] A resultant decrease in functional residual capacity contributes to a rapid desaturation of arterial oxygenation during apneic episodes and predisposes these patients to atelectasis.[2] Smaller diameter airways result in greater resistance to airflow, which further hinders respiratory function.[1,3] An increase in oxygen requirements results in a doubled alveolar ventilation compared with adults. The chest wall, however, being primarily composed of a cartilaginous rib cage, is quite compliant. Whereas carbon dioxide (CO_2) production is also increased, the increased respiratory output functions to keep arterial CO_2 levels fairly comparable to adult levels. This increase in alveolar ventilation is primarily because of an increased respiratory rate, as tidal volume on a per-kilogram basis is also similar to that of children and adults.[4] As opposed to adults, the hypoxic and hypercapnic ventilatory mechanisms are at best, poorly functional, and at worst, may in fact function to depress respiration.[2] As for the airway itself, four main differences exist: (1) the larynx is more cephalad; (2) the epiglottis is larger, stiffer, and angled more anteriorly; (3) the angle of the vocal cords is less perpendicular than adults; and (4) the infant larynx is shaped in a funnel fashion, so that the airway at the level of the cricoid cartilage is the narrowest part of the airway as opposed to the glottis window, as in adults.[1] Finally, nasal obstruction can potentially be devastating as infants have been described as obligate nose breathers.[1]

Rather than consisting solely of the rib cage, lung tissue, and airways, the respiratory system also involves pulmonary gas exchange into and out of the bloodstream. As expected, differences are also found in this component of the respiratory system. In terms of a ventilation-perfusion relationship, the neonatal inhalation of gases occurs at a periodic rate that is 20% to 50% of the HR.[5] The arterial PO_2 of the newborn is less than that of adults, resulting in decreased oxygen stores within the bloodstream. Premature infants are at increased risk of developing hypoxemia because of immature respiratory centers, lung collapse, and chest wall compliance.[5] High oxygen affinity of fetal hemoglobin that is well suited for intrauterine life presents drawbacks to the infant in the postpartum period. The lower PO_2 at the tissue level decreases oxygen's driving potential for diffusion, so the rate at which oxygen can be delivered is decreased. This results in a rapid decrease in the infant's arterial hemoglobin oxygen affinity. Hemoglobin F decreases from 75% of the total hemoglobin concentration to 2% within the first year of life.[5]

Renal Physiology

The renal system is not fully developed in the pediatric population and continues to mature with age. Some of the differences in renal physiologic mechanisms are related to underdevelopment of renal structures and decreased perfusion pressures.[1]

In the weeks after birth, a steadily rising arterial blood pressure accentuates a rising renal blood flow. Renal blood flow in the healthy human adult reaches 25% of CO. The greatest factor in the large increase in renal blood flow in the maturing infant and child is attributed to a large decrease in renal vascular resistance.[5]

One important developmental feature involves the glomerular filtration rate (GFR). Several factors serve to increase GFR in the postnatal period: a reduced resistance in the renal vascular bed, an increase in blood pressure, and an increase in filtration pressure.[5,6] Newborns are less able to concentrate urine, which increases the risk for dehydration, bleeding, and third space loss. These developmental differences and ongoing maturation will impact fluid volume, electrolyte regulation, and ultimately pharmacology. Complete maturity of the renal system is typically present by 2 years of age.[6]

Thermoregulation of the Pediatric Patient

Shivering is the primary involuntary means by which adults regulate heat production. Nonshivering thermogenesis (NST) is the most important thermoregulatory mechanism of heat production in infants because they cannot shiver. Research points to NST as the primary source of heat production during the first 3 months of life.[1] Shivering has been seen in infants after birth with severe hypothermia, which suggests that although the mechanisms for shivering are intact, they are perhaps both suppressed by nonshivering mechanisms and the threshold may be altered.