During the first day of life, the average blood pressure (BP; systolic pressure/diastolic pressure) is 62.6/38.9 mm Hg for term infants but rises in the first week and month of life. The pooled normative data from >70 000 children (2526 aged <1 month) from the Report of the Second Task Force on Blood Pressure Control in Children in 1987 showed that the average BP in female subjects from >8 days of life to <1 month is 81.7/50.7 mm Hg and in males, it is 82.0/50.3 mm Hg. This finding represents an almost 30% rise in systolic blood pressure (SBP) from the first day of life compared with the last 3 weeks of the first month of life. Thus, the Pediatric Advanced Life Support (PALS) definition of hypotension (SBP <60 mm Hg for infants) represents a drop in SBP of 27% from the expected SBP in infants aged >1 week, which is consistent with the “normal” practice of anesthesiologists allowing a 20% to 25% drop in BP during general anesthesia. Ex-premature infants have normative BP data that are generally higher than that of term infants. In a study of 45 very low birth weight infants born at <34 weeks’ gestation, there was a gradual, progressive rise in BP over the first 10 weeks of life. At the completion of the study, when the ex-premature infants had a mean postmenstrual age of 41 weeks, BP was substantially higher than that expected of term infants at 1 week of age. The reason for this difference in cardiovascular development is unclear, but the authors posited that the stress of extrauterine life led to a higher systemic vascular resistance in ex-premature infants.

One of the conundrums confronting anesthesiologists is whether it is more important to target the normal range for BP, as per the patient’s age, or whether to use the patient’s baseline BP as a benchmark from which to calculate a 20% to 25% “allowable” drop in BP if, indeed, there is an allowable, safe BP drop for young infants. Single preoperative values measured in infants awaiting surgery may not represent the typical awake, calm BP in the infant. The accuracy of noninvasive or oscillometric BP measurements compared with indwelling arterial catheter measurements in very young infants cannot easily be quantified. Although there may be inaccuracies between oscillometric noninvasive BP measurements compared with invasive arterial BP measurements, most studies in preterm and term infants show either good agreement or an overestimation of BP during hypotension.

Cerebral autoregulation may be disturbed in ill preterm and term infants suffering from cerebral hypoxia-ischemia. If pressure-flow autoregulation is absent, then increased cerebral perfusion pressure (CPP [the difference between mean BP and mean intracranial pressure or jugular venous pressure, whichever is higher]) is expected to result in increased cerebral blood flow (CBF) and vice versa. In general, as CBF increases, cerebral oxygenation also rises in patients lacking autoregulation. This phenomenon is the basis for examining the correlation or coherence between regional cerebral oxygen saturation ($r$ScO$_2$) levels and mean BP, with $r$ScO$_2$ being a surrogate for CBF and mean BP being a surrogate for CPP. In general, a high correlation or coherence between $r$ScO$_2$ and mean BP is taken to indicate absence of autoregulation. It must be stated that $r$ScO$_2$ levels are measured by using near-infrared spectroscopy, which does not measure the levels of oxygen in the deeper subcortical structures of the brain. The $r$ScO$_2$ is also dependent on arterial oxygen-hemoglobin saturation, hematocrit, cerebral oxygen diffusivity, and cerebral metabolic rate for oxygen; therefore, these other variables should be static to estimate cerebral autoregulation by using this method. Another method for assessing CBF is transcranial Doppler ultrasonography, which actually measures blood flow velocity in a major artery, rather than CBF. Both of these methods have been validated in humans and animal models. The arterial PaO$_2$ and PaCO$_2$ are also known to influence CBF.

The lower limit of cerebral autoregulation of 29 mm Hg has been described in nonanesthetized preterm infants <30 weeks’ gestational age. There are no available data on which to determine whether the cerebral autoregulation curve for ex-premature infants mimics that of term infants, although it is plausible that there is similarity to the curve described in term infants. There is evidence that autoregulatory reserve is less in older infants than in children and adults. Vavilala et al described, by...
using Doppler flow velocity, that in infants older than 6 months, the lower limit of autoregulation was a mean BP of 59 ± 17 mm Hg, which was the same for older children. The mean BP in the infants was 70 mm Hg compared with 80 mm Hg for older children, suggesting that infants had less reserve. A study of children aged <2 years undergoing sevoflurane anesthesia found that in infants <6 months of age, the lower limit of autoregulation occurred at 38 mm Hg or a 20% decrease from baseline awake mean BP.18 There is also evidence that the physical state of the neonate matters, with at least 1 study finding that sick premature infants had pressure-passive cerebral circulation 20% of the time that they were monitored.19 The most conservative approach, in light of the paucity of data regarding the cerebral autoregulatory range in neonatal infants undergoing surgical procedures, would be to assume a very narrow autoregulatory range around baseline BP. However, it should be noted that BP readings below the cerebral autoregulatory curve do not automatically mean that the brain is not being adequately perfused. These values represent a zone of pressure-passive circulation that is considered to place the brain at risk for inadequate perfusion and thus potentially susceptible to damage from fluctuation in BP.

Hypocapnia does not alter the lower limit of cerebral autoregulation in mature animals but does lead to lower CBF per unit change in CPP.20 At lower BP (i.e., below the lower level of cerebral autoregulation), there is an attenuation of the slope of the CBF/CPP graph suggesting that the cerebral vascular response is attenuated with hypotension. CBF in preterm infants is highly dependent on Po2 as determined by transcutaneous Po2 measurements.21 The cerebral vascular response to Po2 is less in the first day of life and increases with gestational age. It is also attenuated, but not eliminated, in hypotensive infants. This reactivity is robust even in preterm infants; it is estimated to be ~4% per mm Hg Po2.22 Intraoperatively, it is also preserved in children (18 months–7 years of age) anesthetized with sevoflurane.23 Hypocapnia during anesthesia may, therefore, result in cerebrovascular vasocstriction and reduction of CBF.

In preterm infants, even mild hypocapnia (Po2 <35 mm Hg) is associated with cerebral palsy and cystic periventricular leukomalacia, and seizures are associated with rapid correction of hypocapnia, leading some neonatologists to advocate stepwise correction in low Po2.24 In term infants with congenital diaphragmatic hernia, it has been shown that there is a positive correlation between IQ on follow-up and postoperative Po2.25 There is also a positive association between the neurocognitive outcome of term infants with hypoxic-ischemic encephalopathy and hypocapnia.26 That said, association does not prove causality and, to date, there is no clinical evidence that hypocapnia in anesthetized term infants causes later learning delay or neurologic problems.

REFERENCES


