

Editorial Views

Pulmonary Function in the Normal Infant

MANY PROBLEMS in pediatric anesthesia are entirely technical in nature, arising both from the small size of some patients and from the large range of sizes that make up the entire population of infants and children. Some problems are not technical but involve a fundamental knowledge of anesthesia as an applied science. These are every day clinical problems, such as: "how fast should a newborn breathe under anesthesia?; will spontaneous respirations usually be sufficient?; and if controlled respirations are needed, what should the rates and pressures be?" Even if the answers to these and similar questions were known, which is often not the case, it soon becomes obvious that it is a hopeless task to memorize the necessary details that apply to each size of patient. It is from this point on that the modern anesthesiologist leans upon a knowledge of respiratory physiology as an organized way of thinking about problems in clinical ventilation. Not only are there fewer general principles than details to master, but a perspective of wide scope is provided.

It is said that the infant and small child are different from the adult, that they are not simply small adults. Yet, measurements obtained from many pulmonary function tests are essentially the same in the normal infant and in the adult. For example, the ventilation equivalent for O_2 , V_T/V_{O_2} , in resting adults with widely differing basal metabolic rates,¹ and in normal adults under widely differing work loads,² is between 22 to 25 ml. of ventilation/ml. O_2 uptake. In the one day old infant, it is about 23 ml./ml. O_2 .^{3,4} This holds for the range of uptake from 10-30 ml. O_2 /minute in the premature and full-term infant to 200-2,500 ml. O_2 /minute in the resting

or working adult. The respiratory quotient (R.Q.) is about 0.8 in both age groups, indicating that the pulmonary load for CO_2 removal, relative to O_2 uptake, is also similar. Other pulmonary function ratios that are the same in infants and adults are the functional residual capacity (F.R.C.)/kg., the tidal volume/F.R.C., and the dead space/F.R.C.

However, there are some important differences between the infant and the adult. Whether these differences affect anesthetic management remains to be seen. The infant has about twice the O_2 uptake, 6.0 to 6.7 ml. O_2 /minute/kg.,^{5,6} as does the adult, and therefore he must ventilate almost twice as much. The ventilation pattern is different from the adult: the higher respiratory frequency of the infant is very familiar to the anesthesiologist. Since the T.V./F.R.C. is the same, this high frequency is a mechanism to increase minute volume (as is tachycardia for increasing cardiac output under a stress). Although the high frequency results in additional work of respiration, because of the dead space, it is also a way of making pulmonary work easier for the infant. Mead⁷ suggests that the frequency of respiration is correlated with the minimal average force of inspiratory muscle and is not correlated with the minimal work of respiration as found by others.⁸⁻¹¹ Anyone who has lifted a heavy load with a block and tackle knows how much easier it is to use the block and tackle even though the added friction increases the total work, just as the additional ventilation of dead space increases the total work of respiration. However, rates which are unusually rapid for infants, do not add to the total ventilation. Prod'hom *et al.*⁶ have shown that infants with

a frequency over 60/minute have a decreased tidal volume/kg. compared to infants with a lower frequency and that both groups end up with the same alveolar minute volume, $\dot{V}_A/\text{kg.}$, \dot{V}_A/\dot{V}_{O_2} , and arterial P_{CO_2} .

The classical concept of control of ventilation is the regulation by medullary chemoreceptors sensitive to arterial CO_2 tension, P_{aCO_2} . Stahlman¹² suggests that in the first few weeks of life, regulation is by way of tissue chemoreceptors sensitive to tissue stores of CO_2 . In this period, the infant normally has a low P_{aCO_2} of 25 to 30 mm. of mercury.^{4, 13} This is not due to high sensitivity of the medulla to CO_2 because the infant shows a poor response to inhaled 4 per cent CO_2 .¹² During the recovery after CO_2 inhalations, and after exercise, the P_{aCO_2} is still lower, yet both ventilation and CO_2 output increase above normal. This is more compatible with a tissue chemoreceptor sensitive to tissue CO_2 than to a medullary chemoreceptor sensitive to P_{aCO_2} . An open ductus arteriosus functioning as a left to right shunt, together with a significant right to left shunt of about 20 to 25 per cent of the cardiac output (based upon A-a O_2 gradients during inhalation of 100 per cent O_2 ^{6, 14}), does suggest that P_{aCO_2} might be a poor mechanism for ventilatory control in the neonate. More direct evidence is needed before the mechanism of control of ventilation in infants is clearly understood.

Two reports in this issue of the JOURNAL present information on ventilation in the infant and small child during clinical anesthesia. The data of Wilson and Harrison on spontaneous respirations with halothane show tidal volume and frequency, with and without surgical stimulation. It can be calculated from their data that without surgical stimulation, ventilation was considerably lower than that predicted by the Radford nomogram, uncorrected. This implies that during periods of non-stimulating surgery, unassisted ventilation in children may be inadequate with halothane. It would be of value to have data on P_{aO_2} and P_{aCO_2} measurements under these same conditions. Calculations of the data of Freeman, St. Pierre and Bachman on infants spontaneously breathing during cyclopropane anesthesia (atropine medication only!) agree well with the predicted values of the Radford nomogram, uncorrected. Here, arterial measurements would be of value to confirm the quality of end-tidal sampling. These authors recommend controlled respirations for infants

using inflating pressure of 30 cm. of water at a frequency of 40/minute with an occasional sigh added to prevent atelectasis.

Advances made in the elucidation of respiratory physiology of infants and children are the basis for improvements in pediatric anesthesia. Quantitative studies, such as those reported here, are very much needed.

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