progressive decreases in Pa_{O_2} in the absence of periodic deep breaths suggests that this phenomenon, if it occurs at all, must be a rare event, perhaps associated with special circumstances or conditions not yet recognized. Therefore, at present there is little objective evidence for beneficial effects of periodic deep breaths or sighs during anesthesia.

Perhaps the views of Shakespeare on this subject are somewhat extreme:

> A plague of sighing . . . . .
> It blows a man up like a bladder.
> (King Henry IV, Part I. II, 4)

However, it should be possible to achieve optimum oxygenation during anesthesia by providing our patients with:

> . . . . . a sleep
> Full of sweet dreams, and health, and quiet breathing.
> (John Keats, Endymion (1818), Book 1)

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Intraoperative Fluid Therapy in Infants

TRANSLOCATION of body fluids and electrolytes associated with surgical operation in the adult has been well documented by Shires, Williams and Brown.[1] During major operations in man, simultaneous measurement of plasma volume with ^{131}I-labeled albumin, erythrocyte mass with ^{51}Cr-labeled erythrocytes, and extracellular fluid volume with ^{35}S sulfate revealed a marked reduction in functional extracellular fluid volume without external loss or change in plasma electrolyte concentrations. This was interpreted as a redistribution of isotonic fluid from the extracellular space to some other place, perhaps an edematous area in the surgical field. The magnitude of this shift of fluid (as much as 28 per cent of the extracellular fluid volume) was related to the degree of surgical trauma incident to the operation itself and to its duration, but independent of moderate blood loss. As a result of this contraction of the extracellular fluid volume, renal excretion of sodium in the postoperative period was reduced.

The reduction in sodium excretion has been managed in the past by withholding sodium during operation and the early postoperative period. Shires advocated an opposite point of view, reasoning that until this loss of extracellular fluid was replaced by infusing the same volume of isotonic fluid, renal function would be compromised. When loss of extracellular fluid volume was balanced by treatment with lactated Ringer's solution, renal excretion of sodium promptly occurred. This concept was extended to hemorrhagic shock when evidence was obtained that hemorrhagic shock in the dog also reduced extracellular fluid volume. Replacement with lactated Ringer's solution plus the volume of blood lost increased survival to 70 per cent, compared

with a 20 per cent survival with replacement by blood alone. Further work in dogs[3] showed that the lactate in lactated Ringer's solution did not result in a further increase in the high plasma lactate concentration associated with shock; on the contrary, plasma lactate in treated dogs promptly returned to normal, whereas in the untreated dogs (given whole blood alone) lactate remained high. Cardiac output, stroke volume, and peripheral resistance in dogs treated with lactated Ringer's solution were better than in untreated dogs.

In this issue of ANESTHESIOLOGY, Bennett, Daughety and Jenkins[4] have studied the reaction of the newborn infant to infusion of 5 per cent dextrose in lactated Ringer's solution given during operation and in the postoperative period. They suggest that the neonate requires increased amounts of multielectrolyte solutions for the same reasons that Shires found for adults. There was no evidence that those infants receiving increased amounts of sodium and water were overhydrated.

On the other hand, measurements of extracellular fluid volume were not made, and we are not sure of the amount and kind of fluid translocated. We do know that the normal newborn is quite different from the adult in body composition and fluid requirements. Total body water is 80 per cent of body weight in the infant, 60 per cent in the adult; extracellular fluid volume is 40 per cent of body weight in the infant, 20 per cent in the adult; daily water exchange in the infant is about 50 per cent of extracellular water, in the adult, 15 per cent. Although infant and adult can excrete water loads equally rapidly in dilute urine,[5] the infant can concentrate only to about 700 milliosmols/l while the adult can concentrate to about 1,400 milliosmols/l. Bennett *et al.* point out that the infant seems unable to retain sodium despite hyponatremia.

In the management of fluid therapy in cholera (admittedly, cholera provides a more severe stress on fluid and electrolytes than surgical trauma) it was found that cholera in children had a higher mortality than cholera in adults when both were treated with isotonic or hypertonic intravenous solutions. In the child, the need for more free water, potassium, alkali and dextrose was thought to be responsible. A "pediatric cholera replacement fluid," more hypotonic than that used for adults, was designed with this in mind and found to have superior results when compared with lactated Ringer's solution.[6] Therapy is not always the same for the adult and the infant.

In the infant undergoing operation and anesthesia, actual measurements of changes in extracellular fluid volume should be made. Fluid therapy can then be managed more confidently. The work of Bennett, Daughety and Jenkins is an excellent step in this direction.

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