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Editorial Views

Concerning Sweet Dreams, Health, and Quiet Breathing

In 1963, a phenomenon which appeared to exert a marked influence on the magnitude of physiologic shunt during anesthesia was reported by Bendixen and his associates.[1] These workers proposed that a monotonous ventilatory pattern lacking deep breaths or "sighs" caused progressive pumonary microatelectasis undetectable by physical or radiographic examination. Objective manifestations of the atelectasis were progressive decreases in Pa_{O_2} and respiratory compliance. Pa_{O_2} and compliance reverted toward normal with hyperinflation of the lung in the form of periodic deep breaths, provided during the course of anesthesia, which were presumed to expand atelectatic lung. Confirmatory evidence for the existence of this sequence of events was the observation that periodic deep breaths are a feature of the normal respiratory pattern of the conscious healthy individual.[2] On the basis of these observations, it was suggested that respiratory pattern had a considerable effect on oxygenation during anesthesia, and that the anesthetized patient should be provided periodically with deep breaths, or artificial "sighs." Provision of periodic deep breaths apparently has become standard practice, not only for the anesthetized patient but also for other individuals who require artificial ventilation in a wide variety of circumstances.

Subsequent work has failed to substantiate the suggestions of Bendixen and his associates. Generally, other investigators have not found progressive decreases in Pa_{O_2} during spontaneous ventilation or during artificial ventilation at constant volume or constant pressure without periodic hyperinflations. Effects of pulmonary hyperinflation on Pa_{O_2} have been small and inconsistent, even in the presence of sizeable AaD_{O_2}'s.[3-5] Studies in the dog by Stone and Sullivan, reported in this issue of ANESTHESIOLOGY,[6] again demonstrate that long periods of ventilation lacking periodic deep breaths need not be associated with progressive increases in physiologic shunting. These results are somewhat surprising since the dog has a reputation as a species particularly prone to pulmonary collapse in the circumstances under which the studies were done. The small increase in AaD_{O_2} which occurred during the experiments was consistent with known effects of changes in cardiac output on magnitude of AaD_{O_2}.[7]

Although Stone and Sullivan state that "few studies of oxygenation under anesthesia have emphasized the actual values of pH_a and Pa_{CO_2}," previous investigators indeed have speculated that inadequate ventilation might have contributed to the progressive increase in AaD_{O_2} reported by Bendixen *et al.* In their initial publication Bendixen and his associates clearly showed a relationship between shallow breathing, hypercarbia, and progressive fall in Pa_{O_2}.[1] Subsequent studies of Lumley *et al.*[4] and Panday and Nunn,[5] however, have shown that periods of shallow ventilation, frequently at hypercarbic levels, are not necessarily associated with progressive falls in Pa_{O_2}. In the present study by Stone and Sullivan, ventilation, although shallow, was adequate to maintain Pa_{CO_2} at normal levels.

Failure of recent investigations to demonstrate

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