Correspondence

The Present Status of Sighing

To the Editor:—The editorial by Bergman,[1] "Concerning Sweet Dreams, Health and Quiet Breathing," in the April 1970 issue, seems to spread clouds over already muddy ground. Because Stone's and Sullivan's[2] article in the same issue was the basis for the editorial, it is important to note that they did find a significant increase in alveolar-to-arterial gradient ($A\text{-}aD_{O_2}$) in "shallowly" ventilated (9.8 ml/kg) patients, as did Bendixen *et al.*[3] in patients more shallowly ventilated (5–7 ml/kg). Minimal (but some) increase in shunting did occur, but from both the editorial and the Stone article the reader is left with the impression either that shallow ventilation is "harmless" and/or that sighing may be unnecessary. In Stone's study, one wonders about the rapid respiratory rate of 60/min which provides average inspiratory and expiratory times of 0.5 sec. It is difficult to conceive of "atelectasis" under such conditions, since what is tantamount to a "retard" or positive end-expiratory pressure was present[4]; therefore, there was no opportunity for the functional residual capacity (FRC) to fall.

Ultimately the FRC may hold the key to this puzzle, and when conditions such as "shallow ventilation" or "constant-volume ventilation" are such that FRC falls, compliance will decrease and both $A\text{-}aD_{O_2}$ and venous admixture (Q_S/Q_T) will increase. Likewise, when FRC rises $A\text{-}aD_{O_2}$ and Q_S/Q_T probably will decrease. Colgan and Whang[5] found no fall in FRC or significant change in compliance (C_L) or Q_S/Q_T during halothane anesthesia in patients given pentobarbital for premedication. In patients given premedication and thiopental for induction, Don[6] found a significant fall in FRC under otherwise similar circumstances. While both studies failed to demonstrate progressive deterioration in any variable, Don's investigation presented strong evidence that a deep breath raises FRC.

In 1959, Mead and Collier[7] found that C_L fell progressively along with lung volume in anesthetized dogs breathing spontaneously or ventilated by a pump in the tidal-volume range. These changes were reversed by a forced inflation, giving rise to the concept of a physiologic role for sighing in providing for reinflation or preventing atelectasis.[8] Bendixen claimed that at constant-volume ventilation (low tidal volumes) although Pa_{O_2} remained unchanged, total compliance and Pa_{O_2} fell progressively and were reversed by forced inflations.[3] Further studies by this group attempted to validate the reasons for sighing[9] and relate the degree of atelectasis, expressed by an increasing $A\text{-}aD_{O_2}$, to the size of the tidal volume.[10] Many ventilators, including the Bennett MA-1, the Emerson Postoperative, the Ohio 560 and the Drager Spiromat-661, have built-in, elaborately programmed sighing mechanisms.

In 1965, Nunn *et al.*,[11] reported a 10 per cent "shunt like" effect during anesthesia and IPPB with tidal volumes higher (700 ml) than in most of the earlier studies. This shunt did not increase in patients under the age of 43 years, but a progressive increase in venous admixture did occur beyond this age. They were unable to raise Pa_{O_2} by hyperinflation.

Subsequent investigations have cast doubt on progressive changes in C_L, FRC or venous admixture occurring during anesthesia and spontaneous breathing, although significant increases in $A\text{-}aD_{O_2}$ and Q_S/Q_T and low FRC may be present at all times.[5, 6] In one study, during IPPB at low, constant-volume ventilation no change in Pa_{O_2} was found, but following hyperinflation consistent increases in Pa_{O_2} occurred when 30 and 40 cm H_2O airway pressures were used.[12] With the publication of Panday's and Nunn's paper, "Failure to Demonstrate Progressive Falls of Arterial P_{O_2} during Anaesthesia," in 1968, the issue was polarized.[13] They concluded that little was to be gained by introducing sighs because progressive pulmonary atelectasis during spontaneous breathing and constant-volume venti-

lation, as mirrored by increase in venous admixture, did not occur. Housley *et al.*,[14] from a study of patients with pulmonary disease, provided C_L and $A\text{-}aD_{O_2}$ data showing little difference before or after sighs, again throwing doubt on the value of the sigh.

Many studies have attempted to relate the state of anesthesia, constant-volume ventilation, or some respiratory variable such as low tidal volume, decrease in lung volume or a combination of these, to decrease in C_L, decrease in lung volume, increase in $A\text{-}aD_{O_2}$ and venous admixture effect. Critical analysis of these studies indicates that many variables differ: tidal volumes, flow rates, frequencies of respiration, modes of respiration, durations of study, inhaled concentrations of oxygen, and non-homogeneity of subjects, including body configuration and freedom from pulmonary disease. Until the variables are controlled, and until the multicausal nature of these pulmonary phenomena are elucidated, cause-and-effect relationships cannot be established, and the final word on sighing has not yet been heard. While it seems popular presently to invalidate the sigh, there may be reasons for the lowered Pa_{O_2} other than $A\text{-}aD_{O_2}$ and Q_S/Q_T phenomena.[15, 16] It is a fact that artificial sighing has not yet been associated with harm to patients.

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To the Editor:—As Dr. Gold correctly points out, many factors have the potential of influencing oxygenation during anesthesia. The editorial, "Concerning Sweet Dreams, Health, and Quiet Breathing," did not imply that efficiency of pulmonary gas exchange during anesthesia is completely independent of ventilatory patterns, although to date such a relationship has not been consistently demonstrated during "routine" anesthesia. Certainly, further investigations designed to study factors influencing oxygenation during anesthesia should be performed. Subsequent developments will be awaited with interest. The conclusions stated in the editorial related to a specific phenomenon postulated to exert a significant influence on oxygenation during anesthesia: pro-