lation, as mirrored by increase in venous admixture, did not occur. Housley et al., from a study of patients with pulmonary disease, provided C1 and A-aDO2 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 C1, decrease in lung volume, increase in A-aDO2 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 PaO2 other than A-aDO2 and Qs/Qt phenomena. It is a fact that artificial sighing has not yet been associated with harm to patients.

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References

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-
gressive increase in pulmonary shunting owing to microatelectasis in the absence of periodic deep breaths or sighs. The preponderance of evidence suggests that this particular train of events does not occur with sufficient frequency to constitute a real problem in the anesthetized patient. Although provision of intermittent deep breaths or sighs may seem beneficial on an intuitive basis, there is little objective evidence to support this practice either during anesthesia or during prolonged artificial ventilation under other circumstances.

Although artificial sighing is apparently innocuous, incorporation of this procedure of doubtful therapeutic value into medical practice does have disadvantages. The most obvious of these, as Dr. Gold points out, is the incorporation of elaborately programmed sighing mechanisms in many commercially available mechanical ventilators. Such a feature adds to the initial cost of the devices, creates maintenance problems, confuses relatively untrained personnel, and distracts the attention of the physician from other more important aspects of respiratory care.

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Kidney

PENTHRANE AND RENAL FAILURE Among 115 patients who had abdominal or thoracic surgery under methoxyflurane anesthesia, seven received tetracycline hydrochloride immediately before or after the operation. Five of the seven had increasing levels of blood urea nitrogen (BUN) and serum creatinine. Three patients died, and necropsy of the kidneys showed numerous calcium oxalate crystals. The remaining 108 patients either did not receive any antibiotic or received penicillin, streptomycin sulfate, or chloramphenicol. None of these patients had postoperative renal failure or a significant increase in BUN or creatinine level. Forty patients received tetracycline after spinal anesthesia without evidence of renal failure postoperatively. It is likely that methoxyflurane and tetracycline, when administered concurrently, can seriously impair renal function, which may lead to death.

(Kuzucu, E. Y.: Methoxyflurane, Tetracycline, and Renal Failure, J.A.M.A. 211: 1162 (Feb.) 1970.)