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Correspondence

Reducing the Soda-lime Hazard

To the Editor:—In reference to the article "Overdistention of the rebreathing bag, a hazardous test for circle-system integrity," by Debban and Bedford, (ANESTHESIOLOGY 42: 365–366, 1975), we have made several attempts to reproduce their observation with our Ohio model machine, with the prepacked disposable tandem CO_2 absorber canisters, using inflation pressures of 50 to 60 cm H_2O, without result. This may be one advantage of using the prepacked disposable tandem canisters versus repacking canisters. However, the soda-lime hazard might also be diminished by venting through the pop-off valve which is usually located on the expiratory side of the anesthesia circuit.

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Ventilatory Responses in COPD

To the Editor:—Pietak *et al.*[1] ably demonstrated the behavior of patients with chronic obstructive pulmonary disease (COPD) during halothane anesthesia with spontaneous ventilation. By correlating the marked carbon dioxide retention with an index of airflow obstruction, the forced expiratory volume in one second ($FEV_{1.0}$), they implied the role of mechanical factors in the alveolar hypoventilation. Although they speculated about a reduced central nervous system response to carbon dioxide, they did not address themselves to its role in either awake or anesthetized patients.

Numerous investigators have evaluated abnormalities of ventilatory control in COPD, but few have been able to relate the two basic factors responsible for a reduced responsiveness to carbon dioxide: decreased respiratory center sensitivity and mechanical inefficiency of the ventilatory apparatus. Lourenco and Miranda[2] found that mechanical factors play the fundamental role in CO_2 accumulation. In patients with normocapnia but evidence of airflow obstruction (mean airway resistance 3 cm H_2O/l/sec; $FEV_{1.0}$ 52 per cent of vital capacity) they observed a decrease in the slope of the curve of the ventilatory response to CO_2 but a marked increase in the integrated electrical activity of the diaphragm, indicating drive from the respiratory center. A subsequent study[3] further supports the mechanical origin of the reduced ventilatory response. On the CO_2–ventilation diagram, ventilation was plotted as "proportional ventilation" (ventilation/maximum voluntary ventilation), and most of the difference in slope between normals and COPD patients was eliminated. This suggests that the increment in ventilation per unit increase in CO_2 is a constant fraction of one's ventilatory capacity.

In patients with hypercapnia, however, the decreased respiratory center drive assumes equal importance. Lourenco's patients with CO_2 retention[2] (mean Pa_{CO_2} 61 torr) showed profound reductions in the slopes of their CO_2 response curves. Although airflow obstruction was more severe (mean airway resistance 7 cm H_2O/l/sec; $FEV_{1.0}$ 39 per cent of vital capacity), the electromyographically determined diaphragmatic response to hypercapnia was reduced tenfold compared with normal patients, indicating decreased central nervous system sensitivity. The influence of hypercapnia on the ventilatory response is emphasized by other investigators[4] who found that in patients with hypoxemia (Pa_{O_2} less than 70 torr) only those with CO_2 retention or his-

tories of CO_2 retention had significant attenuation of the ventilatory responses to CO_2 compared with controls. Pulmonary function data from Pietak's patients[1] with COPD indicate that neither hypoxemia nor hypercapnia was present. Thus, one would be reasonably accurate in ascribing the basic alterations in their ventilatory responses to CO_2, awake or anesthetized, to abnormal respiratory mechanics. The role of a greatly altered central nervous system response to CO_2 in these patients seems unlikely.

Many of the adverse effects of anesthesia on respiration result from the pattern of ventilation common with most inhalational anesthetics, namely, reduced tidal volume and increased respiratory rate. Although the magnitude of these changes is roughly similar in COPD patients,[1] the physiologic impact is greater. The diminished tidal volumes render ventilation less efficient in both groups of patients, but the increased respiratory rate helps to compensate somewhat in normals. However, in patients with airway obstruction it exacts a serious price in the form of reduced dynamic compliance, non-uniform gas distribution, and increased resistance to breathing. Thus, the wasted ventilation as well as the work of breathing increases significantly.

The authors[1] state that controlled or assisted ventilation is mandatory in patients with COPD. One might further recommend the pattern of ventilation. Slow deep breathing is effective in improving gas exchange in COPD patients, compared with their usual rapid, shallow ambient breathing patterns.[5] A similar approach to controlled ventilation, with paralysis if necessary, seems appropriate in patients with significant airflow obstruction. This, of course, does not preclude close monitoring of Pa_{CO_2}, as would be appropriate with any ventilatory mode in these patients.

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Gas Embolism during Pneumoencephalography

To the Editor:—We read with interest the case report "Air embolism through a ventriculoatrial shunt during pneumoencephalography" by Youngberg, Kaplan and Miller (ANESTHESIOLOGY 42:487, 1975), as we recently encountered a similar case.

A 16-year-old boy, who four years previously had had a pinealoma removed, developed headache and anorexia. Increase in cerebrospinal fluid (CSF) pressure was suspected, even though a ventriculo-peritoneal shunt had been placed at the time of operation. In preparation for ventriculography the patient was sedated with Innovar and a ventriculostomy was performed using local anesthesia. The ventricular catheter was attached to a pressure transducer and CSF pressure was continuously recorded. Oxygen was used as the contrast medium. Following an exchange of 50 ml CSF with oxygen, nitrous oxide inhalation was administered in 50 per cent concentration as part of an experimental study