could not be separated from the single-use anesthesia circuit (Becton-Dickinson #3063). Repeated attempts failed to allow separation or even rotation of the mask around the patient-end of the circuit. With a final forceful attempt the circuit broke at the level of the swivel adapter (Fig. 1) making any form of ventilation impossible. A replacement circuit was readily available and the patient suffered no harm. Using identical equipment, it was subsequently quite easy to reproduce a similar tight bond.

This incident suggests caution in the concomitant use of disposable equipment from different manufacturers and reemphasizes the need for a resuscitation bag in every operating room.

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Moyamoya Disease and Anesthesia

To the Editor—Moyamoya (puff of tobacco smoke) disease is a rare, chronic, occlusive cerebrovascular disease with an unusual network of vessels in the base of the brain.\footnote{1} The clinical manifestations are variable and include hemiplegia, convulsive seizures, and speech disturbances.\footnote{2} In the last few years, this disease has been the object of surgical treatment, including superficial temporal-middle cerebral artery anastomosis\footnote{3} and encephalomyosynangiosis.\footnote{3}

We have anesthetized four patients seven times. They were 6, 10, 32, and 52 years old, and had no abnormal neurologic findings preoperatively. Two patients were anesthetized with neuroleptanaesthesia with controlled ventilation. Frequent analysis of arterial blood gases revealed \( P_{\text{aco}} \) values ranging from 30 to 35 mmHg. These patients had delayed recovery of consciousness postoperatively, and also obvious neurologic deficits including hemiplegia, amnesia, dyscalculia, and speech disturbance in one case. These complications were severe and lasted for a month or more. On the other hand, the other patients, including the second anesthetics of three patients, were anesthetized with halothane and \( \text{N}_2\text{O} \) with spontaneous ventilation, except in one patient in whom ventilation was controlled. In these cases, an infrared \( \text{CO}_2 \) analyzer was used to monitor the end-tidal \( \text{CO}_2 \) that was maintained at 40–50 mmHg. Postoperatively, all of these patients showed prompt recovery of consciousness and no neurologic complications.

It seems unlikely that the surgical procedure itself caused the neurologic deficits, because in these operations, there was nothing we could identify that would cause a decrease in the cerebral blood flow. Furthermore, the direct arterial blood pressures were monitored in each patient and maintained an adequate range. Neuroleptanaesthesia itself might not be an exacerbating factor in these two cases because, in humans, this type of anesthesia has not been shown to have a significant influence on either the cerebral blood flow (CBF) or the cerebral metabolic rate for oxygen.\footnote{4}

Hypocapnia may be the main cause of the neurologic deficits. The degree of hypocapnia in our patients should produce about a 30% decrease in the CBF\footnote{5} and may have been a critical effect on moyamoya disease.
Although the possibility of the Robin Hood effect is possible in areas of partial vascular occlusion,\textsuperscript{5} in moyamoya disease, hypocapnia might steal blood away from the area supplied by the compromised vessel. On the other hand, hypercapnia, such as 50 mm Hg of end-tidal CO$_2$, does not seem to have a harmful effect on the moyamoya disease. On the basis of our cases, we recommend that end-tidal CO$_2$ should be maintained in the range of 40–50 mmHg throughout anesthesia in patients with moyamoya disease.

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Pulmonary Vascular Response is Dependent Upon Initial State of the Vasculature

To the Editor:—The conclusions of Schulte-Sasse et al.,\textsuperscript{1} concerning the importance of control values of pulmonary vascular resistance (PVR) with respect to the response to nitrous oxide, are interesting in light of recent findings by Cox.\textsuperscript{2} In in vitro studies of the canine pulmonary arteries, Cox found that active stress development increased from the main pulmonary artery to intralobar artery sites. The calculated muscle length at which the maximum active force development occurred was found to correspond to transmural pressures of a magnitude normally found in the systemic circulation. Consequently, at normal pulmonary artery pressures and pulmonary vascular smooth muscle (PVSM) fiber lengths, there would be little potential for the activation of the vascular smooth muscle. However, if pulmonary transmural pressures were abnormally high, the PVSM fiber length would move closer to the optimal length for active force development. These findings would also be consistent with the data of Lappas et al.\textsuperscript{3} In addition, unpublished observations by Cox in the dog with chronic filarial infestation showed an increased capacity for force development in the extralobar arteries.

Filarial infestation results in a proliferative pulmonary vascular response to pulmonary hypertension and may be analogous to pulmonary hypertensive states in humans with respect to abnormal force development in pulmonary vascular smooth muscle. If the findings of Cox are applicable to in vivo studies, however, one would expect background anesthesia to be a factor. While we found that the addition of nitrous oxide to different halothane levels did not change PVR in the dog, the capacity for reflex changes in PVR was modified by the accompanying concentration of halothane.\textsuperscript{4}

These various clinical and experimental findings suggest to us that the primary determinant of a significant pulmonary vascular response is the initial state of the pulmonary arterial smooth muscle.

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