CORRESPONDENCE

Although the possibility of the Robin Hood effect is possible in areas of partial vascular occlusion, in moyamoya disease, hypocapnia might steal blood away from the area supplied by the compromised vessel. On the other hand, hypercapnia, such as 50 mmHg of end-tidal CO₂, does not seem to have a harmful effect on the moyamoya disease. On the basis of our cases, we recommend that end-tidal CO₂ should be maintained in the range of 40–50 mmHg throughout anesthesia in patients with moyamoya disease.

KOJI SUMIKAWA, M.D.
Assistant Professor of Anesthesiology
HIROTOSHI NAGAI, M.D.
Instructor in Anesthesiology
Department of Anesthesiology,
Shiga University of Medical Science
Otsu, Shiga 520-21, Japan

REFERENCES


(Accepted for publication August 30, 1982.)

Anesthesiology
59:205–206, 1983

Pulmonary Vascular Response is Dependent Upon Initial State of the Vasculature

To the Editor:—The conclusions of Schulte-Sasse et al., 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. 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. In addition, unpublished observations by Cox in the dog with chronic filarial infestation showed an increased capacity for force development in the extralobar arteries.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.

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.

ROGER J. BAGSHAW, M.D.
Associate Professor of Anesthesiology
ROBERT H. COX, PH.D.
Associate Professor of Physiology
Department of Anesthesia
University of Pennsylvania
3400 Spruce Street
Philadelphia, Pennsylvania 19104
REFERENCES


(Accepted for publication July 26, 1982.)

Risk of Air Embolism during Catheter Placement

To the Editor:—In a recent clinical report, Westheimer1 reported a technique for right atrial catheter placement with the use of a wire guide. The technique described seems quite simple and successful. Once the J-wire is inside the catheter, and the catheter is advanced to the correct position in the right atrium, the catheter will be open to room air. This poses the danger of air embolism from the open catheter if the patient suddenly takes a deep breath. The proximal end of the catheter will be difficult to occlude due to the presence of J wire. What precautions are taken to prevent air entry during catheter advancement?

CHARUL MUNSHI, M.D.
WILLIAM C. ROUMAN, M.D.

Medical College of Wisconsin
Milwaukee, Wisconsin 53226

REFERENCE


(Accepted for publication July 26, 1982.)

In reply.—Dr. Munshi has questioned whether the patient is at risk of air embolism while using a wire guide for intravascular ECG monitoring during right atrial catheter placement. Air embolism has not occurred with our technique for the following reasons: 1) prior to catheter insertion, the patient is anesthetized, his ventilation is controlled, and he is placed in Trendelenburg’s position; and 2) the presence of the wire guide in the catheter effectively seals it and prevents leakage of blood out of the catheter or entrainment of air into it.

D. N. WESTHEIMER, M.D.
Anesthesiology
6565 Fannin
Houston, Texas 77030

(Accepted for publication July 26, 1982.)