Correspondence

Adrenal Venous Catecholamines during Methoxyflurane Anesthesia

To the Editor:—Re the paper by Dr. Li in the November–December issue: Decreased adrenal venous catecholamine concentrations during methoxyflurane anesthesia may be due to "a depressive effect on the sympathoadrenal system," to an increase in adrenal blood flow with no change in catecholamine release, or to both. Without information on adrenal blood flow, the conclusion cannot be made that the sympathoadrenal system has been depressed by methoxyflurane.

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To the Editor:—Adrenal blood flow was unaltered during methoxyflurane anesthesia as compared with control values when the blood pressure was not reduced more than 35 per cent from control level. Adrenal blood flow in our experiments remained in the range of 4–4.5 ml per 10 minutes before, during and after methoxyflurane anesthesia. These findings were not presented in the paper because when the adrenal blood flow is constant, the catecholamine concentration value serves as a more accurate measurement and assessment of secretory activity of the adrenal medulla. The study was made under these conditions.

Moreover, although it is easy to measure the blood flow from the whole adrenal gland, one cannot readily or accurately distinguish distribution of blood between cortex and medulla. The former presumably constitutes 85–95 per cent (R. J. Kramer and L. A. Sapirstein, Endocrinology 81: 403, Aug. 1967). Unless one can determine accurately the exact portion from the medulla, measurement of the total adrenal blood flow tends to introduce and magnify errors in the calculated results. In addition, during the recovery period the adrenal venous catecholamine concentration invariably rose to the control, pre-methoxyflurane level (sometimes with an overshoot) without a change in adrenal venous blood flow.

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Surgery

LARYNGEAL STENOSIS A 16-year-old girl, deeply unconscious following ingestion of phenobarbital, was ventilated for 96 hours through a Woodbridge cuffed endotracheal tube. The tube was changed daily. Following extubation, there was increasing hoarseness and respiratory distress. Despite treatment with steroids and antibiotics, tracheotomy became necessary three weeks after extubation. Examination revealed ankylosis of both cricoarytenoid joints and subglottic stenosis causing complete airway obstruction. The author, an otolaryngologist, stresses that a properly performed tracheotomy prevents, with "sufficient certainty," damage to the tracheal wall in all age groups. (*Minnigerode, B.: Ankylosis of Both Cricoarytenoid Joints and Laryngeal Stenosis Following Endotracheal Intubation of Several Days' Duration, Der Anaesthesist 17: 230 (July) 1968.*)

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