activity or because of the observed changes in cardiac output alone.

The methods employed leave further unanswered questions. Does the presence of an endotracheal tube which could stimulate autonomic activity alter the observed values of shunting or the responses to drugs? Since it is known that local anesthetics alter the lung's responsiveness to hypoxia,[1] instillation of lidocaine into the trachea could possibly have altered the results further.

BENJAMIN H. GORSKY, M.D.
Intern in Anesthesiology
University Hospitals of Cleveland
Cleveland, Ohio

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To the Editor:—Regarding our high control $\dot{Q}_s/\dot{Q}_t$, values of about 10 per cent during the postoperative period are not rare, even in patients without cardiopulmonary abnormalities. As Gorsky points out, the increased shunt could be from the operation or from general anesthesia. It could also be from the prolonged oxygen breathing before the control measurements were made. Our unpublished data from other postoperative patients show that $\dot{Q}_s/\dot{Q}_t$ may increase from 6.7 ± 1.2 (SE) to 10.9 ± 1.3 per cent ($P < 0.001$) after 19.7 ± 1.1 minutes of oxygen breathing after epidural analgesia. It is generally believed that the effect of $\dot{V}_A/\dot{Q}$ inequality on shunt is minimized by oxygen breathing and that total $\dot{Q}_s/\dot{Q}_t$ falls.[1-3] It has also been suggested that denitrogenation promotes alveolar collapse of poorly ventilated alveoli because of oxygen absorption and $\dot{Q}_s/\dot{Q}_t$ increases. This possibility has been suggested by Severinghaus[4] and reported by Déry *et al.*[5]

Dr. Gorsky believes that without the knowledge of the cause of the high control shunt, the effects of catecholamines remain unclear. In our study we believed that the achievement of a steady condition of atelectatic lung area (or volume) during the experiments was more important than the absolute value of $\dot{Q}_s/\dot{Q}_t$. Therefore, we began the experiment one and a half hours after operation and after a relatively long period of oxygen breathing. Regarding the effects of catecholamines, two points deserve emphasis. First, if the number of atelectatic alveoli increased during isoproterenol infusion, it would be difficult to conclude anything as to the changes in distribution of pulmonary blood flow. Fortunately, the bronchodilator action of isoproterenol makes further alveolar collapse unlikely. Second, alveolar P_{O_2} could still vary with drug-induced changes in the distribution of $\dot{V}_A/\dot{Q}$ even during oxygen breathing. We believe the effect of drug-induced variations in $\dot{V}_A/\dot{Q}$ on $P_{A_{O_2}}$ was not large, considering the small changes in $P_{a_{CO_2}}$ (table 2). This mechanism, however, cannot be ruled out.

We are unable to estimate the effect of the endotracheal tube on the activity of the pulmonary vessels. Like many other investigators, we found it necessary for the conduct of the study. It is difficult for us to believe that our small dose of lidocaine into the trachea exerted a large and prolonged effect on the vasoactivity of the pulmonary vessels. In Lloyd's study,[6] excised dog lobes were perfused with blood containing relatively high concentrations of procaine.

MANNOSUKE MUNEYUKI, M.D.
AKIRA INAMOTO, M.D.
Department of Anesthesiology
Faculty of Medicine
Kyoto University
Kyoto, Japan

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