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In reply:—We thank Dr. Drummond for his interesting comments. However, we would like to add the following remarks.

Previous work has shown, in *in vitro* experiments, that high concentrations of halothane are responsible for a decrease in diaphragmatic function after both direct and indirect stimulation.¹ Therefore, the purpose of our study was to determine, *in vivo*, if it was possible to observe a decrease in diaphragmatic function in dogs anesthetized with halothane.² As we mentioned in our study, transdiaphragmatic pressure (P_{di}) is influenced by the length and the shape of the diaphragm. During airway occlusion there is a slight shortening of the diaphragm.

However, the purpose of our study was not to demonstrate that during airway occlusion P_{di} remains a truly isometric pressure, but to observe the changes in this widely used index during increasing concentrations of halothane (F_Ihal). We found that increasing levels of halothane are associated with a proportional decrease in P_{di}. Halothane inhalation is associated with reduced stability of the chest wall that can result in an indrawing of the thorax during occluded inspiration, and therefore in a decrease in P_{di}. However, there is no evidence that the instability of the chest wall increases with increasing levels of halothane anesthesia. Instability of the chest wall is already present with 0.5% halothane, and no further significant changes are observed with higher concentrations of halothane. With isoflurane, Mankikian *et al.*³ observed the same phenomenon: the instability of the chest wall appears at 0.5 MAC, but no further change is observed with higher concentrations. Therefore, we believe that the progressive decrease in P_{di} associated with increasing F_Ihal results from an effect of halothane on diaphragmatic function.

The other comment made by Dr. Drummond concerns the possibility that the decrease in P_{di} may result from hypercapnia associated with halothane anesthesia, rather than from the effect of halothane. In three dogs of our experiment that stopped breathing at 1.5 and 2% halothane, we repeated P_{di} measurements during phrenic nerve stimulation, while the animals were mechanically ventilated and had normal values of P_ACO₂. In these three

animals, P_{di} was still lower than the values obtained at the lower F_Ihal.

Moreover, since this experiment, it has been shown in as yet unpublished *in vivo* experiments (effect of halothane on diaphragm and hindlimb muscle in rats. B. Dureuil *et al.*) that clinical levels of halothane decrease diaphragmatic contractility in a dose-related fashion in mechanically ventilated rats in which P_ACO₂ has been maintained constant.

Further studies are now needed to determine the precise mechanisms of the halothane-induced diaphragmatic dysfunction and its importance in the ventilatory depression observed during halothane anesthesia.

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