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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 (Pdi) 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 Pdi remains a truly isometric pressure, but to observe the changes in this widely used index during increasing concentrations of halothane ($F_I$hal). We found that increasing levels of halothane are associated with a proportional decrease in Pdi. 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 Pdi. 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 Pdi associated with increasing $F_I$hal results from an effect of halothane on diaphragmatic function.

The other comment made by Dr. Drummond concerns the possibility that the decrease in Pdi 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 Pdi measurements during phrenic nerve stimulation, while the animals were mechanically ventilated and had normal values of PA_{CO_2}. In these three animals, Pdi was still lower than the values obtained at the lower $F_I$hal.

Moreover, since this experiment, it has been shown in as yet unpublished *in vivo* experiments (effect of halothane on diaphragm and hindlimb muscle in rats.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 Pa_{CO_2} 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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