With this simple technique, we are able to maintain the patients' prebronchoscopy oxygenation and avoid further worsening of the pulmonary disorder.

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# Measurements of Occlusion Pressure during Anesthesia with Volatile Anesthetics in Humans

*To the Editor:*—Recently, Canet *et al.*,[1] reporting on changes in occlusion pressure and ventilation with different depths of anesthesia using either halothane or isoflurane, suggested that only one study previously analyzed occlusion pressure in humans, at increasing concentrations of a halogenated anesthetic agent and that studies of the occlusion pressure wave are scarce.

In an analysis of the occlusion pressure wave and inspiratory flow patterns of patients anesthetized with enflurane and nitrous oxide,[2] I found that ventilatory depression caused by an increase in inspired enflurane concentration was related solely to a reduction in central drive and not to the peripheral factors more favored by Canet *et al.* in their discussion. The same study also reported changes in the occlusion pressure and inspiratory flow pattern caused by opioid administration. There was no marked change in estimates of active elastance and impedance, again suggesting that the depression was central. Occlusion pressure also was used in a further study, investigating the findings of Wahba and Sadkova,[3] who found that atropine increased ventilation during anesthesia with enflurane. The effects of atropine administration were studied in patients anesthetized with enflurane and halothane, using occlusion pressure to distinguish central effects from peripheral, mechanical effects. The findings suggested a minor effect after premedication with meperidine, consistent with the possibility of bronchodilation: Atropine caused an increase in inspiratory flow, although occlusion pressure was unaltered.[4]

These observations support the suggestion of Canet *et al.* that further analysis of occlusion pressure waveform in spontaneously breathing anesthetized patients can be helpful to investigate the neural mechanisms of respiratory depression.

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