ABSTRACT
The methods used to assess the respiratory depressant effects of inhalation anaesthetics are illustrated by reference to observations made during halothane and enflurane anaesthesia in dogs. The timing of events of the respiratory cycle, determination of the functional residual capacity with the maximum pressure generated in the occluded airway, ventilation, and the response of these variables to increases in PaCO₂ need to be combined to give a complete profile of an agent’s effects.

INTRODUCTION
The methods used to study the respiratory effects produced by anaesthetic agents have become more refined with the passage of time. Early observers noted that breathing became more shallow as the depth of central nervous (CNS) depression increased and that it ceased before circulatory arrest occurred. From these observations the characteristics of the breathing pattern came to be used in assessing the depth of anaesthesia and Guedel’s classical description of the signs and stages of diethyl ether anaesthesia is characterized by extensive reference to them, but it soon became apparent that respiratory signs must always be related to a particular agent. For
example, under halothane anaesthesia respiration may become severely depressed before reaction to painful stimulation is abolished, whereas during ether anaesthesia it is well maintained long after reaction to stimulation disappears. These simple observations suggest that halothane is much more of a selective respiratory depressant than ether but how much greater cannot be deduced.

The introduction of the concept of MAC (minimal alveolar concentration), made direct comparison of the effects of different inhalation anaesthetics much easier and the arrival of simple methods for the determination of blood gases enabled the clinician to determine the influence of anaesthetics and analgesics on oxygenation of the blood and/or the removal of carbon dioxide from the body. Clearly, determination of the blood gases at equivalent levels of CNS depression allows assessment of the respiratory effects of the agents in question, but it gives no indication of how any observed respiratory depression arises.

Other methods of assessing the respiratory effects of drugs centre on the measurement of ventilatory responses to changes in arterial or end-tidal carbon dioxide levels during anaesthesia or after the administration of single doses of analgesics such as the opiates. Many variations of the basic technique have been described in the literature but they all involve measurements of pulmonary ventilation, either as end-tidal carbon dioxide tension is increased or during steady state conditions at different carbon dioxide tensions. The various techniques yield carbon dioxide response curves and there can be little doubt that the determination of these at equivalent levels of anaesthesia has proved to be a useful tool in the comparison of the respiratory effects of the inhalation anaesthetics. The response curve undoubtedly defines the way in which depression of the central chemoreceptor is translated into gaseous exchange, but again, it gives no indication of the mechanism involved.