The United Kingdom and a few other countries stand apart from the rest of the world of anaesthesia in having carbon dioxide cylinders mounted on anaesthetic apparatus, and with a flowmeter which admits carbon dioxide to the fresh gas supply, a practice dating from at least 1927. Logic is not always the deciding factor in such differences in practice, and this is an appropriate time to consider the arguments for and against the presence of cylinders of carbon dioxide. In July 1989, the Department of Health in the U.K. issued Safety Action Bulletin No. 49 which drew the attention of anaesthetists to another case of inadvertent administration of carbon dioxide, this time resulting in serious injury to the patient.

The sole purpose of exogenous carbon dioxide in anaesthesia is to increase the patient’s PaCO₂. This has many effects, including increasing cerebral blood flow, but the most relevant is to stimulate breathing. Historically, carbon dioxide entered the anaesthetic armamentarium for the purpose of accelerating induction with inhalation anaesthetics of high blood solubility, particularly diethyl ether. Surprisingly, 144 of the 1100 anaesthetists responding to the survey of Razis [1] still used carbon dioxide to speed induction with inhalation anaesthetics. However, as this indication has declined in response to the decreasing blood solubility of more recently developed anaesthetics, a new indication for carbon dioxide has taken its place.

During anaesthesia with paralysis and artificial ventilation, there is a tendency to overventilate, which is manifestly safer than underventilation. It is a common observation that arterial Pco₂ values during anaesthesia with unmonitored artificial ventilation are below normal, often below the apnoeic threshold of Pco₂, and sometimes as low as 2 kPa. This is of particular relevance now we know that inhalation anaesthetics abolish the peripheral chemoreceptor ventilatory drive and have a profound effect even at 0.1 MAC [2]. The use of high concentrations of oxygen at the end of anaesthesia also suppresses peripheral chemoreceptor drive. Thus a hypocapnic patient may be totally devoid of chemoreceptor drive at the end of a period of anaesthesia with artificial ventilation.

Under these adverse conditions of chemoreceptor drive, spontaneous breathing may be re-established by the simple expedient of allowing consciousness to be regained. In most patients, Pco₂ less than the apnoeic threshold does not inhibit breathing when consciousness has been regained [3], although it does prevent breathing during anaesthesia. Alternatively, the re-establishment of spontaneous breathing may be hastened usually by increasing the Pco₂ to a value greater than the apnoeic threshold, so restoring central chemoreceptor drive before consciousness is regained, and typically while the surgical wound is being closed. This can be achieved in three ways. First, the alveolar ventilation may be reduced. Second, carbon dioxide may be re-breathed by taking the soda-lime out of circuit. Third, exogenous carbon dioxide may be administered.

The time course of increase in Pco₂ by each of these expedients was examined by Ivanov and Nunn [4]. The first method is very slow and the time course of increasing Pco₂ by decreasing alveolar ventilation is not the mirror image of decreasing Pco₂ by increasing alveolar ventilation. The latter is a matter of washing out body stores of carbon dioxide (with a half-time of about 4 min), while the former depends on the metabolic production of carbon dioxide, and the half-time of the resultant increase in Pco₂ may be as long as 15 min, with an initial increase limited to a maximum of about 0.6 kPa min⁻¹. Clearly, this is rather a slow method of increasing Pco₂ at the end of an operation when the patient has been hypocapnic for a long time. However, the situation is changed
when end-tidal $P_{\text{CO}_2}$ is monitored. This permits $P_{\text{CO}_2}$ to be maintained close to normal throughout surgery and to be brought above the apnoeic threshold in anticipation of resumption of breathing. However, restriction of ventilation has other effects, particularly on uptake of a volatile anaesthetic and on arterial $P_{\text{O}_2}$, the latter being affected both by alveolar ventilation and by development of pulmonary collapse. However, ventilation and $P_{\text{CO}_2}$ may be varied independently by such means as the introduction of apparatus deadspace, or restriction of fresh gas flow in systems which permit variable rebreathing. These systems include circles without soda-lime and the Bain system. Monitoring of $P_{\text{CO}_2}$ is essential for best results with such arrangements.

The second method (bypassing the soda-lime) depends on the retention of a part of the metabolically produced carbon dioxide and is little faster than the first method [4]. The third and final method (addition of exogenous carbon dioxide) increases $P_{\text{CO}_2}$ to any required value in less than 1 min [4] and is manifestly the quickest and most controllable method of increasing $P_{\text{CO}_2}$.

There is no doubt that the presence of a cylinder of carbon dioxide on the anaesthetic apparatus exposes the patient to the risk of accidental administration of very high concentrations of carbon dioxide. In Razis’ survey, 200 respondents knew of an accident with carbon dioxide that occurred in the hospital at which they currently worked: 29% of these accidents resulted in death. However, it seemed possible that the same incident might have been reported by more than one respondent. The same author reviewed nine cases of carbon dioxide-related deaths and cases of cerebral damage reported by various authors in the period 1950–1977. The danger lies in the bobbin moving to the top of the carbon dioxide flowmeter, where it may be unnoticed.

There is widespread recognition of the hazard of inadvertent administration of carbon dioxide. Furthermore, the incidence of this hazard appears to be greater than that of some of the rare complications of anaesthesia which have generated much more publicity and a great deal of research activity. Razis found that, in spite of recognition of the hazard, more than 60.9% of U.K. anaesthetists included in his survey still used carbon dioxide daily and another 20.4% did so at least once a week [1]. Withdrawal of carbon dioxide would be objectionable to 77.1% of his respondents—a figure virtually identical to that elicited at a Symposium of the Association of Anaesthetists on “The place of carbon dioxide in anaesthesia”, held on December 14, 1989 under the Chairmanship of Dr R. Greenbaum.

There appear to be two solutions to this dilemma. The first is to remove the cylinder from the apparatus except when it is required for administration. However, this relies on human compliance and might engender misplaced confidence. The second solution is the imposition of a flow restrictor, limiting flow to (say) 500 ml min$^{-1}$ in the gas lead to the carbon dioxide flowmeter. This would have two advantages: first, it would restrict the total flow of carbon dioxide to a value which is unlikely to be dangerous; second, the range of the carbon dioxide flowmeter is such that the bobbin could not rise above the mid point of the scale, where its presence could hardly pass unnoticed. This simple modification would appear to resolve the dilemma. Safety Action Bulletin No. 49 recommends that the limiting flow rate of 500 ml min$^{-1}$ should be halfway up the flowmeter tube. This would require development of a new flowmeter tube and it would be unfortunate if introduction of flow restrictors were delayed by development of a 1-litre carbon dioxide flowmeter tube.

J. F. Nunn

REFERENCES
4. Ivanov SD, Nunn JF. Methods of elevation of $P_{\text{CO}_2}$ for restoration of spontaneous breathing after artificial ventilation of anaesthetized patients. British Journal of Anaesthesia 1969; 41: 28–37.