Table 1 Nitric oxide (NO) and nitrogen dioxide (NO_2) concentrations and their gradients between inlet and outlet of Soda sorb in six test gas mixtures. Extraction ratio was calculated using the following formula: (gradient)/(inlet) × 100

Gas mixture	Inlet (ppm)		Outlet (ppm)		Gradient (ppm)/ (Extraction ratio (%))			
	NO	NO_2	NO	NO_2	ΔNO		ΔNO_2	
1	40.7	0	39.3	0	1.4	(3.4)	0	—
2	35.4	4.1	31.2	0	4.2	(11.9)	4.1	(100)
3	30.3	10.7	19.0	0.1	11.3	(37.3)	10.6	(99.1)
4	20.6	20.2	3.7	0	16.9	(82.0)	20.2	(100)
5	10.3	30.6	0.3	0	10.0	(97.1)	30.6	(100)
6	0	40.8	0	0.1	0	—	40.7	(99.8)

Table 2 Mean concentrations and extraction percentages for nitric oxide (NO) and nitrogen dioxide (NO_2) during passage through the three different varieties of soda lime

	Bedfont monitor						Micromedical monitor					
	Inlet (ppm)		Outlet (ppm)		Extraction (%)		Inlet (ppm)		Outlet (ppm)		Extraction (%)	
Soda lime	NO	NO_2	NO	NO_2	NO	NO_2	NO	NO_2	NO	NO_2	NO	NO_2
Green to brown	46.6	3.4	2.4	0.4	94.8	88.2	46.6	3.0	0.0	0.0	100.0	100.0
Pink to white	41.6	3.1	36.3	0.5	12.7	83.9	41.1	3.0	37.7	0.0	8.3	100.0
White to violet	39.9	3.1	34.6	0.6	13.3	80.6	42.7	3.1	39.2	0.0	8.2	100.0

Table 3 Chemical composition and indicators of the three different varieties of soda lime

Soda lime	Calcium hydroxide	Sodium hydroxide	Water	Indicator
Green to brown	> 75.5%	< 3.5%	< 21.0%	< 0.2% (Potassium permanganate)
Pink to white	> 75.5%	< 3.5%	< 21.0%	< 0.02% (Cleyton yellow)
White to violet	> 75.5%	< 3.5%	< 21.0%	< 0.02% (Ethyl violet)

reaction described by Wilson and Wilson in 1959, and again cited by Kain [3]:

$$2NO + N_2O_4 + 4NaOH \rightarrow 4NaNO_2 + 2H_2O$$

In summary, soda lime does appear to remove nitrogen dioxide from a flowing gas stream during administration of inhaled nitric oxide. The three varieties we have studied also however remove nitric oxide to a varying degree. A perfect nitrogen dioxide scavenger for use during inhaled nitric oxide therapy remains to be discovered.

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Myotonic dystrophy and target-controlled propofol infusions

Sir,—Myotonic dystrophy is a rare autosomal dominant condition which presents the anaesthetist with many problems. These have been reviewed by Russell and Hirsch [1] and include increased sensitivity to drugs used in anaesthesia, such as induction agents, neuromuscular blocking agents and opioids. Propofol has been used successfully for induction and maintenance of anaesthesia [2, 3]. We report our experience using a target-controlled propofol infusion system and the problems we encountered.

A 23-yr-old, 63-kg Caucasian male was admitted for operative fixation of a fractured mandible. He was of good health apart from myotonic dystrophy which manifested itself as muscle fatigue. He had not previously been anaesthetized. No premedication was given. After securing i.v. access and preoxygenation, anaesthesia was induced using a target-controlled propofol infusion as described by White and Kenny [4]. The target concentration was set at 2 μg ml^{-1} and increased in steps of 2 μg ml^{-1}. Alfentanil 1 mg was administered when verbal contact with the patient was lost. When the target concentration reached 12 μg ml^{-1} and the patient's jaw was relaxed, laryngoscopy was performed. Nasotracheal intubation was straightforward and the patient's lungs were ventilated with 50% nitrous oxide in oxygen. No neuromuscular blocker was used at any time. Anaesthesia was maintained with a target concentration of 6 μg ml^{-1}, which was reduced to 5 μg ml^{-1} after 30 min and 4 μg ml^{-1} at 60 min. Total operative time was 75 min.

Towards the end of the procedure the target concentration was set to, 0 μg ml^{-1} so that the reduction in calculated propofol concentration could be followed. The patient did not commence spontaneous respiration until the calculated propofol concentration decreased to less than 2 μg ml^{-1}. Respiration was slow and shallow initially but improved with time. There were no signs of awakening or response to voice even when the predicted propofol concentration was less than 1 μg ml^{-1}. He was observed for a further 30 min with no change. He was then transferred to the intensive care unit where he was observed while still breathing spontaneously through the tracheal tube, attached to a T-piece. Arterial oxygen saturation and end-tidal carbon dioxide remained satisfactory throughout. Approximately 1 h after the end of surgery, he began to show signs of awakening and extubated his own trachea. His subsequent postoperative course was uneventful and the following morning he was discharged to the general ward.

Recommended settings for target-controlled propofol infusion systems are 3–6 μg ml^{-1} for induction and 2–6 μg ml^{-1} for maintenance of anaesthesia, depending on age, patient fitness, degree of surgical stimulation and use of nitrous oxide [4]. In the absence of other guidelines, we decided on values appropriate for a young, reasonably healthy male with a moderate degree of surgical stimulation. Although this anaesthetic technique was successful in our patient, the prolonged recovery time would seem to indicate that target concentrations at the lower end of the range should be used in patients with myotonic dystrophy.

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Sir,—The uneventful use of a propofol infusion in a patient with myotonic dystrophy has been described previously [1], albeit without the use of a computer-controlled infusion. Prolonged recovery after an induction dose of the drug has also been reported [2, 3], and the occurrence of prolonged recovery after an infusion is therefore not surprising. In one of the references quoted [4], the injection of propofol appeared to induce an episode of myotonia, so the assertion that it was "used successfully" might be viewed with some reservations.

The phenomenon of delayed recovery has been reported with all commonly used anaesthetic agents and is presumably a result of the disease process rather than of any particular drug. It is a wise precaution to ensure the ready availability of an intensive care bed before embarking on general anaesthesia in these cases.

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Fires and explosions

Sir,—I wish to express my appreciation for the highly educational and entertaining account entitled "A short history of fires and explosions caused by anaesthetic agents" by Dr A. G. McDonald [1]. This is a useful collection of historic anecdotes, particularly now that flammable agents are not commonly used. I wish to comment on the case cited from the paper by Carl Walter [2] in which Dr McDonald neglected to describe an important factor which was included by Dr Walter that oxygen was in the cyclopropane tank and hung back on the machine. Such a circumstance is simply an explosion waiting to happen.

The cited case is very similar to the description of one which was recounted to me by Ernesto Frias, senior anaesthetist, as having occurred shortly before I visited Santiago, Chile in 1964. Again there were two operating tables in the room with two patients and two surgical teams. The resulting explosion caused several deaths and multiple injuries as well as destroying the operating theatre itself. The circumstances were set up by an operating room orderly who transfilled oxygen into a cyclopropane tank and even put it back on the anaesthesia machine.

A similar situation occurred at the Kupat Cholim Hospital in Israel shortly before my visit there in 1951. According to Hans Winc, chief anaesthetist, in that instance a colour blind orderly, illiterate in English, transfilled oxygen into a red-coded, essentially full cyclopropane tank. A nurse anaesthetist had noticed that the cyclopropane was not as effective as usual, but ascribed it to biological variation and response. Later when she was cleaning the anaesthetic machine apparently a static spark ignited a leaking slow flow from that tank with a resulting detonation back into the explosive mixture in the tank. The nurse died as a result of shrapnel from the exploding tank.

Both of these instances were caused by transfilling oxygen from a larger supply into small tanks and by less knowledgeable individuals who did not fully appreciate the dangers involved in mixing gases.

Cyclopropane had been used at the University of Wisconsin for more than 15 yr after its introduction without any incident relating to ignition and explosion. As its general use spread to other parts of the world however, there were some dramatic episodes which dampened the enthusiasm for this otherwise valuable and versatile agent.

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Sir,—Professor Morris is correct in saying that there was also oxygen in the cyclopropane cylinder. Limitation of space prevented the inclusion of much detail in the review. Dr Walter, writing in 1964, described the incident as follows:

"Upon opening the valve on one of the cylinders supposedly containing only cyclopropane, the cylinder exploded... This explosion dislodged the second cyclopropane cylinder from its machine and the cylinder valve broke off releasing cyclopropane which ignited and burned as a torch... The valves of the remainder of the cylinders were broken, but only one cylinder of oxygen was dislodged from the machine... Three ether containers became involved... with a resulting intense fire... Subsequent investigation revealed that the cylinder had been partially filled with oxygen by error and subsequently charged with cyclopropane. The cylinder valve... regulators, hoses, etc, were not suitable for oxygen. It is also likely that the cyclopropane apparatus was not kept as clean of grease, dust, etc, as is necessary for oxygen service. In addition, it is possible that the accidental mixture of cyclopropane and oxygen was in the explosive range. Evidence indicates that the cylinder valve was opened more rapidly than usual. This was possibly a factor in ignition either by producing heat from adiabatic compression or friction or possibly by creating a static spark."

No reference is given for this incident, nor is the year in which it occurred stated. The article does not state to what extent the cylinder may have been filled with oxygen before the error was realized or whether an attempt was made to rid the cylinder of