Sir,—One of the main criticisms raised by Dr Henville was that the device would not meet the Canadian specification. Our device was made a considerable time in advance of that publication. However, as the requirements of “Standard” organizations of the different countries vary from country to country, it is very probable that the device in its present form would not comply to one or more standards. We feel, however, that the unit could be modified to meet any standard of any country.

For the sake of brevity we simply mentioned that the first principle of Rosen and Hillard, namely that the device be gas operated, was outdated in our opinion. In modern medicine, many devices which are powered by electricity are used, for example, ventilator monitor alarms and dialysis machines, and we considered that an oxygen failure device powered by electricity was admissible. This gives us one very great advantage, in that we are able to detect a very small reduction in the oxygen supply pressure. All the safety devices which are operated by gas pressure alone give a warning of an impending failure when the supply pressure has already decreased by 60%, when failure is well established and the time available for rectification is very limited. An auditory alarm powered by electricity can be made as loud as required and is continuous until the fault has been rectified. In devices powered by the failing oxygen supply, the auditory alarm is weak and as the remaining oxygen expires so does the alarm. We considered that devices which are triggered and maintained by other gases, such as nitrous oxide were not worth comparison.

Finally, some devices already in use divert the anaesthetic gas supply from the patient in the event of failure and allow him to breathe theatre air. However, they have the disadvantage of using the anaesthetic gases to trigger the alarm and therefore those gases are vented to the theatre atmosphere. Our device contains all gases within its system, protecting the theatre staff and allowing the patient to be connected to the theatre environment.

K. A. FLOWER
Salford

PHARMACOKINETICS OF HIGH-DOSE FENTANYL

Sir,—We read with interest the article by Bovill and Sebel (1980) as our work with much smaller doses of fentanyl confirms their findings.

Four patients undergoing elective cardiac surgery received fentanyl \(5 \mu g \text{kg}^{-1}\) following induction of anaesthesia and normal plasma fentanyl concentration (PFC) decay curves were noted. Shortly before commencing cardiopulmonary bypass, which produced a mean haemodilution of 28%, each received a second dose of fentanyl \(5 \mu g \text{kg}^{-1}\). In three patients PFC was not detectable (less than \(0.7 \text{ng ml}^{-1}\)) by the assay method used (Michiels, Hendriks and Heykants, 1977), 1–5 min after bypass. In the fourth patient, PFC decreased from \(20 \text{ng ml}^{-1}\) immediately before bypass to \(1 \text{ng ml}^{-1}\) after 5 min bypass. Thus although the proportional change in PFC can only be calculated for the last patient, it was always greatly in excess of a decrease caused by haemodilution.

Our plan of study differed from that of Bovill and Sebel in that a second dose of fentanyl was given and that bypass was initiated during the initial part of the decay curve, while fentanyl was being rapidly redistributed. In view of the great affinity of fentanyl for lung tissue (Hess, Herz and Friedel, 1971), PFC was measured in pulmonary artery blood samples from two patients as soon as bypass was established, and simultaneously with peripheral arterial samples. In both patients peripheral arterial PFC was too small to measure, but the pulmonary artery samples contained \(1.4 \text{ng ml}^{-1}\) and \(1.6 \text{ng ml}^{-1}\)—decreases of 85 and 53% from pre-bypass PFC. Thus, when fentanyl is given shortly before bypass, apparent disappearance from the circulation may be a result of sequestration within the lung.

We also observed a reappearance of fentanyl in the circulation during rewarming after hypothermic bypass to \(28^\circ\text{C}\). In the patient who was noted to have PFC of \(1 \text{ng ml}^{-1}\) following bypass, the value was \(2.2 \text{ng ml}^{-1}\) after 30 min rewarming. Another patient who had no measurable plasma fentanyl after bypass had a concentration of \(1.3 \text{ng ml}^{-1}\) after 20 min rewarming. This may represent fentanyl previously sequestered in skeletal muscle during hypothermia (Hess, Herz and Friedel, 1971).

D. P. CARTWRIGHT
J. C. CHAPMAN
J. R. DAVIES
A. M. SCOGGINS
Bristol

REFERENCES


PANCURONIUM AND NODAL RHYTHM

Sir,—Pancuronium has largely replaced tubocurarine because of its relative freedom from side-effects (McDowell and Clarke, 1969; Levin and Dillon, 1971). However, the drug may cause hypertension in some individuals (Fraley, Lemoncelli and Coleman, 1978). Cardiac arrhythmias, however, have been virtually unknown with pancuronium.

Recently, one of us (O. S.) has witnessed three cases of nodal rhythm directly related to the injection of pancuronium, and inquiries at another hospital in the region have revealed two more cases.

All of the patients were premedicated with morphine and either atropine or hyoscine. Anaesthesia was induced with thiopentone and maintained with nitrous oxide in oxygen supplemented by either pethidine or droperidol–fentanyl. Before tracheal intubation, pancuronium (Pavulon, Organon) was given in a dose of \(0.1 \text{mg kg}^{-1}\). In all five patients, 1–2 min later the ECG showed a change from sinus rhythm to nodal rhythm which reverted spontaneously after 20–90 s. (While the primary diagnosis of nodal rhythm was made from the oscilloscope, we managed in one case to record the arrhythmia on paper as well for accurate diagnosis.) In two of the patients, the nodal rhythm reappeared as a maintenance dose of pancuronium was given later. During the period of nodal rhythm, the arterial pressure was stable.

We have been unable to find any mention of this side-effect and would like to know, therefore, if any readers have seen a similar effect.

OLLE SAEMUND
ESKIL DALENIUS
Falun, Sweden

REFERENCES

