CORRESPONDENCE

Anesthesiology
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An Easy, Safe, and Clean Procedure to Open Propofol Ampules

To the Editor:—In our hospital we use syringes that come sealed in a throw-away plastic container. I cut off the wide-mouth end of the throw-away cap of a 3-ml syringe with a strong and inexpensive pair of scissors. Another short cut is made along the length of the cap (fig. 1).

To open a propofol or any other ampule, wrap the upper part with an alcohol swab before inserting it in the plastic cap to snap the glass at the neck of the ampule. I discard the alcohol swab with the short glass tube of the ampule. The plastic cap can be reused.

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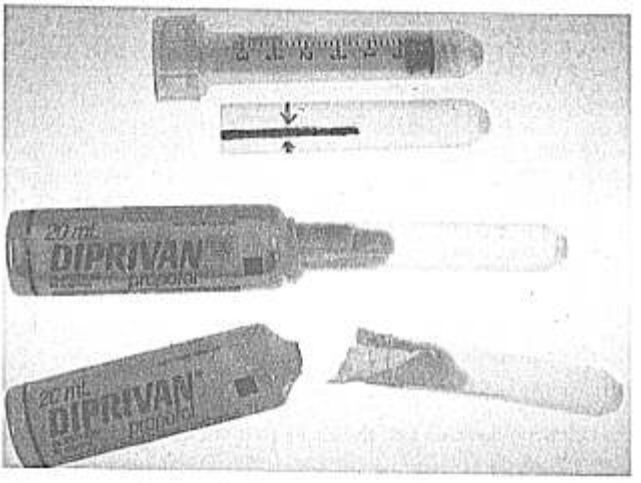

Fig. 1. Cut-off end of a disposable syringe plastic container, used to snap the neck of a propofol ampule.

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Atmospheric Pollution with Topical Anesthetics

To the Editor:—Chlorofluorocarbons (CFCs) are man-made molecules polluting our atmosphere and are the focus of much political and environmental attention.[1] CFCs now rank second only to carbon dioxide as a cause of the increasing global greenhouse effect.[2] Most of the world's governments have agreed to a timed reduction in CFC use, in accordance with the Montreal Protocol of 1987.*

I recently discovered the widespread use of CFCs in our hospital as a topical anesthetic and skin refrigerant. We were using Fluro-Ethyl® (Gebauer Company), which contains 75% dichlorotetrafluoroethane (DTE), also known as CFC-12 or Freon, to numb skin and check levels of spinal and epidural anesthetics. We now have eliminated it from our institutional formulary and recommend others consider their need for this and similar drugs.

The problem with CFCs is their relative stability. They build up in the troposphere, diffuse around the globe, and after several years migrate into the stratosphere. CFC molecules undergo photolysis in the stratosphere; this process releases highly reactive chlorine atoms, which destroy the ozone present there. Stratospheric ozone at natural levels absorbs nearly all solar radiation with wave lengths between 240 and 320 nm before the radiation strikes the earth's surface. Solar radiation, if unblocked by ozone, would kill many unicellular organisms, damage the surface cells of many plants and animals, and augment greenhouse warming.

DTE has an atmospheric lifetime of 170 yr before breaking apart, which means DTE will continue to accumulate in the stratosphere for several years after we stop its release. DTE is more environmentally toxic than our halogenated inhalational anesthetics, for instance, which have atmospheric lifetimes of only 2–6 yr.[3] I believe we can find nonpolluting alternatives to CFCs and DTE in anesthesiology and that to do so is important to our environment.

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* Montreal Protocol on Substances that Deplete the Ozone Layer. United Nations Environment Programme, 1987.

References

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2. Gribbin J: Hothouse Earth. New York, Groven Weidenfield, 1991
3. Brown AC, Canosa-Mas CE, Parr AD, Pierce JMT, Wayne RP: Tropospheric lifetimes of halogenated anaesthetics. Nature 341:635–637, 1989

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In Reply:—Johnstone's letter concerns our Fluro-Ethyl® spray, (a nonflammable prescription topical anesthetic or skin refrigerant) and chlorofluorocarbons (CFCs) implicated in stratospheric ozone depletion.

Fluro-Ethyl contains 75% (by volume) dichlorotetrafluoroethane (114), a CFC also known as Freon® 114 or Dymel® 114, and 25% ethyl chloride. Johnstone is incorrect in stating that dichlorotetrafluoroethane is Freon 12. The chemical name for Freon 12 is dichlorodifluoromethane.

Freon 114 has been identified as a stratospheric ozone-depleting chemical. An accelerated phase-out is in progress for this chemical and for the other class I chemicals listed in the Clean Air Act Amendments of 1990, Title VI-Stratospheric Ozone Protection. The original phase-out, once set to be January 1, 2000, has been accelerated by Executive Order of President Bush. By the end of 1995, there will be no production of most class I chemicals; dichlorotetrafluoroethane is included in this phase-out. It also should be noted that this phase-out is far more strict than the United Nations-sponsored Montreal Protocol.

Johnstone states that other products, such as ethyl chloride, are available to anesthetize skin. However, ethyl chloride is flammable; Fluro-Ethyl® is not. Ethyl chloride, therefore, is sometimes not permitted in hospitals if not stored according to the recommendations of the Joint Commission on Accreditation of Hospitals.

It also should be noted that the current U. S. production of 114 is 50% of the 1986 volume, 360 million pounds. Fluro-Ethyl® uses 0.004% of this amount.

Gebauer is complying with the phase-out schedule. Regardless of the fact that we have no replacement at present for Fluro-Ethyl®, there will be no production of 114 in 1995; therefore, there will be no product.

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Risk of Ischemia in Patients Receiving Desflurane *Versus* Sufentanil: Sample Size and Clinical Significance

To the Editor:—Anticipating the arrival of desflurane into our clinical armamentarium, I eagerly read Helman *et al.*'s randomized trial of sufentanil *versus* desflurane in patients with documented coronary artery disease.[1] Helman *et al.* performed what appears to be a methodologically exacting study demonstrating that desflurane, like the other potent inhalational agents, can be used, with appropriate adjuncts, without unacceptable hemodynamic consequences in this patient population.

What is unclear to me as a clinical reader is their discussion directed at differences between the sufentanil and desflurane groups relative to adverse cardiac outcome. First we are informed that "our sample size was insufficient to detect a significant difference, and that even a "10-fold" increase in sample size would have been unable to detect a difference. But then the authors conclude that on the basis of a "relative risk" calculation, we should not rule out that desflurane could be associated with more prebypass ischemia than sufentanil in these patients.

What does "not significant" mean to the clinical reader? To me, it suggests that under the rules established by the investigators at the outset of the study, and subsequently validated by referees, they were unable to demonstrate a difference in the tested treatments. Clearly, Helman *et al.* were plagued with the common problem of a sample size insufficient to deal with a small "effect size" (*i.e.*, the strength of the treatment on the measured outcome). Another example of the Beelzebub of the clinical trial is, namely, does absence of evidence constitute evidence of absence?

What really disturbs me about Helman *et al.*'s paper is where it leaves us. First, the authors conclude that there was no significant