for medical recognition of nursing autonomy and expertise in this field."

In view of the medicolegal and other implications of this article, I wish to state that I and many other dermatologists consider that short term use of a Eusol dressing to clear adherent slough from an ulcer is perfectly reasonable. We object to the way in which Eusol has been effectively banned by nursing diktat without appropriate consultation and for no very good reason.

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1 Leaper DJ. Eusol. *BMJ* 1992;304:930-1. (11 April.)
2 Burton JL. Wound healing. In: Champion RH, Burton JL, Ebling FGJ, eds. *Textbook of dermatology*. 5th ed. Oxford: Blackwell Scientific, 1991:824-32.
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5 United Kingdom Central Council for Nursing, Midwifery, and Health Visiting. *Exercising accountability: a framework to assist nurses, midwives and health visitors to consider ethical aspects of professional practice*. London: UKCC, 1989:16-7.

SIR,—Despite the many papers that have been against the continued use of Eusol opinion remains divided about Eusol's value as well as its toxicity. Farrow even commented that the issue may actually be "the surface manifestation of a deeper dispute between doctors and nurses about authority and autonomy."

David J Leaper points out that there have not been any reliable, randomised controlled trials that might resolve the issue.² Most of us have been brought up to believe that no treatment should be given unless it has been validated by randomised controlled trials, but in the case of Eusol I suspect that an ethics committee's approval would be almost impossible to obtain, particularly as nurses are represented on most committees.

I conducted a controlled experiment on myself. I applied full strength Eusol to a cotton wool ball to one forearm and saline to the contralateral arm. The cotton wool was taped down, non-occlusively, with Micropore tape. I did not abrade any skin surfaces. Within one hour I experienced discomfort and, later, pain on the Eusol side only. Twenty four hours later there was an area of erythema, which subsequently progressed to blistering and superficial ulceration (figure). No lesion appeared on the contralateral arm. The lesion on the Eusol side was still visible two weeks later, and some skin pigmentation persisted for a further two years.

My experiment leads me to believe that Eusol should not be applied to normal, let alone wounded, skin. It would therefore be difficult to persuade me to subject any of my patients to a randomised controlled trial. We succeeded in persuading our pharmacy department to stop dispensing Eusol, although a senior surgical colleague circumvented

the embargo by obtaining supplies from an adjoining district hospital.

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1 Farrow S. The place of Eusol in wound management. *Nursing Standard* 1991;5(22):25-7.
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Adrenaline in allergic emergencies

SIR,—In their paper on angio-oedema related to angiotensin converting enzyme inhibitors Thomas Hedner and colleagues suggest that with "symptoms . . . an immediate subcutaneous adrenaline administration is of vital importance and the patient should be observed and hospitalised."¹ This recommendation is repeated in the relevant paragraph on the "This week in the *BMJ*" page, which states that "admission and prompt subcutaneous adrenaline administration are vital."²

Current recommendations suggest that adrenaline should be given intramuscularly rather than subcutaneously, as in allergic emergencies absorption from the site of the subcutaneous injection may be too slow, especially if the patient is shocked due to severe anaphylaxis.^{2,3} After intramuscular injection absorption is rapid and usually adequate if reasonable circulation is present.

As soon as any allergic reaction is recognised, first line treatment should include managing the airway, restoring blood pressure by laying the patient flat, and giving 0.5-1.0 mg adrenaline intramuscularly.^{2,3} Intravenous adrenaline is rarely warranted in allergic emergencies and, indeed, may be hazardous.⁴ If required it should be given slowly and in high dilution.⁴ Second line treatment should include an antihistamine—for example, intravenous chlorpheniramine 10-20 mg—which should be given immediately after intramuscular adrenaline.³ Intravenous corticosteroids have little place in immediate management as the beneficial effects are delayed.³

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1 Hedner T, Samuelsson O, Lunde H, Lindholm L, Andren L, Wiholm B-E. Angio-oedema in relation to treatment with angiotensin converting enzyme inhibitors. *BMJ* 1992;304: 941-6. (11 April.)
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AUTHORS' REPLY,—Gregory Y H Lip and Malcolm J Metcalfe state that current recommendations suggest that adrenaline should be given intramuscularly rather than subcutaneously, referring to the *British National Formulary*.¹ In Sweden, though, the recommendation is to give adrenaline (1 mg/ml) 0.5-0.8 ml subcutaneously as soon as possible in cases of suspected anaphylactic reactions, urticaria, or angio-oedema.^{2,3} The subcutaneous administration of adrenaline was also considered to be preferable in a recent review on anaphylaxis in the *New England Journal of Medicine*.⁴

In severe cases when cutaneous circulation is impaired, other ways of giving adrenaline must be considered. The current recommendation in Sweden is to give diluted adrenaline (0.1 mg/ml) slowly intravenously in cases of anaphylactic shock. Circulatory shock was not seen in any of the

Swedish cases of angio-oedema in association with angiotensin converting enzyme inhibitors that we reported.

Regardless of the route of administration, we agree that adrenaline is the preferred drug for treating angio-oedema affecting the mouth, pharynx, or larynx.

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1 BMA and Royal Pharmaceutical Society of Great Britain. *British national formulary No 22*. London: BMA and Pharmaceutical Press, 1991.
2 Socialstyrelsens kungerelse om förebyggande och behandling av vissa överkänslighetsreaktioner. Uppsala: Socialstyrelsens Författningssamling (SOSFS), 1977:2. (Notification from the National Board of Health and Welfare regarding prevention and treatment of some hypersensitivity reactions.)
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Steroid ulcers

SIR,—M Guslandi and A Tittobello¹ accept the conclusion of Piper *et al* that patients taking a combination of corticosteroids and non-steroidal anti-inflammatory drugs double their chances of developing peptic ulcer.² This is an erroneous conclusion from that study. Piper *et al* reported that patients admitted to hospital with a peptic ulcer were more than twice as likely to be taking this combination of drugs than controls, an interesting observation that does not, however, prove that these drugs lead to the development of peptic ulcers. Alternative explanations are that this combination of drugs increases the risk of complications of peptic ulcer and that ulcer healing is delayed by this combination of drugs.

The distinctions are more than academic as a full understanding of mechanisms should aid the development of strategies for preventing drug associated complications of peptic ulcer disease. We provided direct evidence that non-steroidal anti-inflammatory drugs retard the healing of ulcers.³ In experimental studies with cryoulcers in rats the ulcers were larger, healed slower, and showed less reparative proliferative activity in the healing edge when non-steroidal drugs were given. When peptic ulcers from patients taking non-steroidal drugs were compared histologically with those from patients not taking such drugs the mitotic index of gastric crypts, a marker of the rate of regenerative repair, was considerably reduced at the ulcer edge in the patients taking the drugs.

We have studied the size and rate of repair of experimental gastric ulcers in rats receiving prednisolone and compared these with the effects of indomethacin or control vehicle (table). We have also investigated the effect of misoprostol, an analogue of prostaglandin E₁, on the healing rate of cryoinduced ulcers during administration of corticosteroid. Our results show that prednisolone, like indomethacin, results in larger gastric ulcers after cryoinjury than occur in the absence of these

Effect of various drugs on size of experimental gastric ulcers in rats (eight animals per group)

Drug treatment	Mean (SE) ulcer size (mm ²)	
	After 3 days	After 6 days
Control	3.1 (0.4)	1.6 (0.3)
Indomethacin 2 mg/kg	4.8 (0.3)*	3.1 (0.2)*
Prednisolone 10 mg/kg	5.7 (1.0)*	3.4 (0.3)*
Misoprostol 300 µg/kg	2.2 (0.3)	1.6 (0.2)
Prednisolone + misoprostol	2.5 (0.8)	2.0 (0.0)

*p<0.05 compared with control.

Arm 24 hours after application of Eusol