

in cancer patients with morphine-associated constipation. Unless a very much cheaper formulation becomes available, naloxone is unlikely to replace alternative laxatives.

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1. Culpepper-Morgan JA, Inturissi C, Portnoy R, Kreek MJ. Oral naloxone treatment of narcotic induced constipation: dose response *NIDA Res Monogr* 1989; **95**: 399-400

** This letter has been shown to Dr Sykes, whose reply follows.—ED. L.

SIR,—The percentages given in my letter refer to the 24 h naloxone dose as a proportion of the 24 h opioid dose. Since individual naloxone doses were given 4-hourly, those associated with clinical laxative effects were 3.3% or more of the daily opioid dose (ie, the daily naloxone dose was 20% or more of the 24 h opioid dose). It seems that only 2 of the 12 patients reported by Robinson and her colleagues received naloxone doses at this level.

The laxative response to naloxone does not necessarily occur after a single dose—it is unclear whether the New Zealand patients received a solitary dose of naloxone or repeated doses and, if so, for how long and at what interval. In our experience a response, although it may be very rapid, may take four or more 4-hourly doses to appear. The study is continuing and thus far 8 out of 10 patients receiving the 20% level of naloxone or more have shown a laxative response.

The dose limit of 12 mg was derived by Culpepper-Morgan et al¹ from an analysis of 3 patients, and thus cannot be regarded as an exact figure. We have seen a patient who experienced some reversal of analgesia at individual naloxone doses of 10 mg. Further clarification of this point is needed.

Naloxone is clinically available only as a sterile preparation for injection, in which form it is expensive. Chemically pure naloxone unlicensed for medical use can, however, be bought commercially for less than £0.06/mg based on an initial purchase of 5 g from Sigma Chemical Company. This price indicates that far cheaper preparations could be produced.

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Falafel-burger anaphylaxis due to sesame seed allergy

SIR,—With the increasing demand for vegetarian food the sale of the "vegetable burger" is now widespread. Often such products contain potent allergens without a clear declaration. Their ingestion can lead to anaphylaxis in allergic individuals.^{1,2} We describe here an anaphylactic reaction to a falafel vegetableburger.

A 23-year-old woman presented to a casualty unit after eating a falafel-burger consisting of a wheat-flour bun filled with chickpea balls and served with red and white sauce. After taking the first bite she felt an immediate burning sensation in her mouth. Pharyngeal oedema soon developed, followed by dyspnoea, Quincke's oedema, widespread urticaria, nausea, diarrhoea, and chill. She responded to intravenous corticosteroids and antihistamines and was discharged home after an observation period of a few hours.

She had no history of allergy. A nephew had allergic rhinoconjunctivitis. Skin prick tests were negative with common inhalant and food allergens but strongly positive with the white sauce, containing sesame seed paste. Scratch tests with sesame seeds elicited a severe local wheal reaction with flare, pruritus, and pseudopods. Specific IgE to sesame seeds ('Phadebas RAST', Pharmacia) was class 3 positive (8.5 units/ml). The anaphylactic episode after ingestion of a falafel-burger with red and white sauce is therefore explained by a sesame seed allergy. Upon further questioning we learned that sensitivity was probably due to the use of a commercial spice mixture containing sesame seeds; rhinitis,

itching eyes, and burning skin developed every time she added the spice during cooking.

Sesame seeds must be regarded as very potent allergens. They can cause severe anaphylactic reaction in allergic individuals.^{3,4} In a large retrospective study of 173 consecutive cases of food allergy at our allergy unit from 1978 to 1982 there were 4 cases (2.3%) of sesame seed anaphylaxis.⁵

The allergens in sesame seeds may be inactivated or destroyed through heating. To our knowledge no anaphylactic episode due to heat-extracted sesame seed oil has been reported and the baked sesame seeds on usual burger buns do not cause anaphylactic reactions in sesame seed allergic individuals.

This episode highlights the potential danger of vegetarian food and underlines the importance of a proper allergological assessment if food allergy is to be recognised promptly so that the patient can eliminate the causative agent and use suitable medication in the event of a dietary indiscretion.

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Unexpected trend in chemosensitivity of *Plasmodium falciparum* in Brazzaville, Congo

SIR,—Resistance to amino-4-quinolines has spread rapidly in Central Africa, especially in the Congo. The first cases were reported in 1985 in European expatriates examined in France,¹ and widespread drug resistance was also seen in Congolese subjects in three regions of the Congo.² In surveys in Brazzaville in 1987, no strains were resistant in quinine (n=54) or mefloquine (53) in vitro.³ In 1989, quinine⁴ was still very effective in the treatment of malaria attacks. Surveillance of resistance to amino-4-quinolines in systematic surveys of semi-immune children showed that this was stable in two regions of the country.⁵

In February, 1990, another in-vitro study was done in Brazzaville and was comparable to that in 1987 both epidemiologically (strains taken from a comparable population) and technically (radioisotope microtests done according to the same protocol). In addition to chloroquine, quinine, and mefloquine, halofantrine was also tested. The results of the 1990 study (average median inhibitory concentration [IC₅₀]) showed a higher sensitivity to chloroquine than did those of the 1987 study, with a lower sensitivity to quinine and mefloquine (table). In addition, 3 of 67 strains were resistant to halofantrine, which correlated closely with resistance to mefloquine (r=0.362, n=53, p=0.0077). Since the emergence of chemoresistance, the changes in behaviour of the population towards antimalarials must partly be accounted for by the variations in sensitivity of *P. falciparum* strains. For quinine, the decrease in sensitivity was concomitant with the striking increase in consumption since 1985. Resistance to mefloquine was not seen immediately, in contrast with findings in Cameroon.⁶ It is difficult to account for the rapid trend that we observed. Indeed, for the amino-alcohols, drug pressure is unlikely to be implicated in the Congo since mefloquine was introduced only in 1990, and halofantrine was marketed in 1987, is not prescribed for chemoprophylaxis, and has a short half-life. However, halofantrine, which produces cross resistance,⁷ is mainly used in Brazzaville in districts where transmission can be intense and localised. For chloroquine, this in-vitro study confirms the trend of in-vivo survey in the same period,⁵ but the frequency of resistance in the semi-immune population with a dose schedule of 35 mg/kg was less than 10%.⁸ However, the decrease in consumption of amino-4-