common varieties of smokeless tobacco used by our audited patients are known colloquially as *zarda* and *paan masalla*.

In our opinion, awareness that non-smoking tobacco use results in an increased cardiovascular risk and that its use is widespread among some non-Caucasian ethnic groups is not commonly appreciated by anaesthetists. We therefore wish to raise the profile of this issue.

Conflict of Interest

None declared.

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1 Boffetta P, Straif K. Use of smokeless tobacco and risk of myocardial infarction and stroke: systematic review with meta-analysis. *Br Med J* 2009; **339**: b3060

2 Health Survey for England 2004. *The Health of Minority Ethnic Groups—Headline Tables*. London: NHS Health and Social Care Information Centre, 2005

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Fluoroderma after exposure to sevoflurane

Editor—Sevoflurane is currently used as an inhaled anaesthetic agent with few adverse drug reactions. Skin reaction after general anaesthesia is uncommon. We report a case of fluoroderma after exposure to sevoflurane.

A 56-yr-old man developed ulcerated, erythematous, painful nodules on his neck, face, arms, and hands 8 h after surgery for right retinal detachment (Fig. 1). Perioperatively, he received propofol, remifentanil, paracetamol, tramadol, and ketoprofen, and anaesthesia was maintained with sevoflurane 1.1% for 2 h. He had a history of exposure to sevoflurane anaesthesia 3 yr previously (1 h with alveolar concentration at 1.3%). Skin biopsy revealed epidermal hyperplasia with infiltrates of dermal neutrophils without leucocytoclasic vasculitis (Fig. 1c). Tissue culture was negative for microorganisms. Protein electrophoresis was normal.

Sevoflurane [$CH_2F-O-CH(CF_3)_2$] is metabolized (5–8%) into fluoride ions by oxidative defluorination by the cytochrome P450 system, resulting in high serum concentrations of inorganic fluoride with prolonged anaesthesia at higher concentrations.[1–3] The patient's serum fluoride level was 182 μmol litre^{-1} (normal level, <50 μmol litre^{-1}). A diagnosis of fluoroderma was made. Review of the

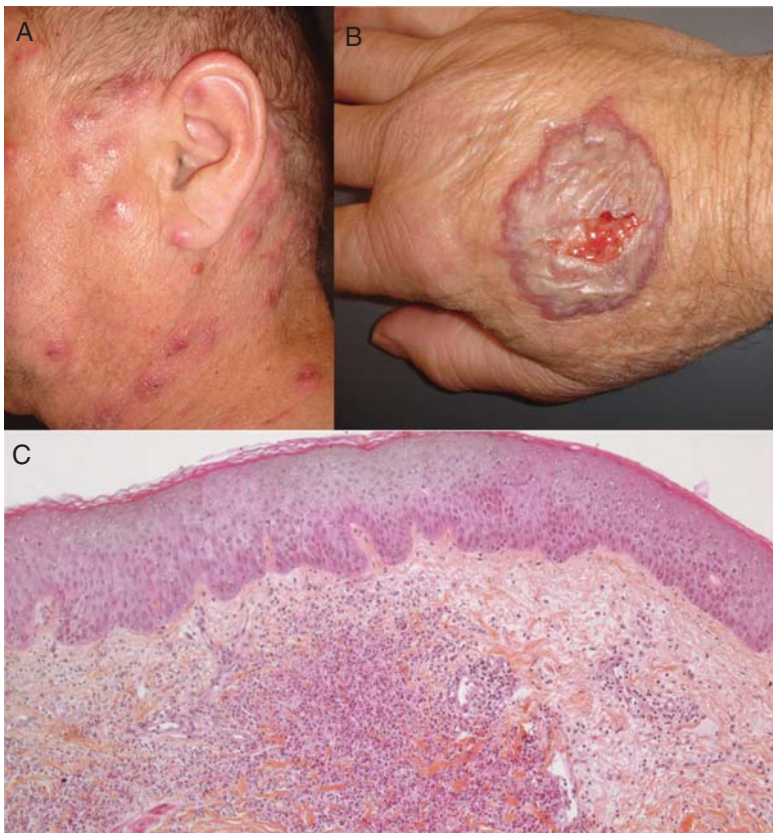

Fig 1 (A) Erythematous nodules on the face. (B) Ulcerated erythematous nodules on the dorsal aspect of the hands. (C) Skin biopsy: epidermal hyperplasia with infiltrates of dermal neutrophils, without leucocytoclasic vasculitis.

patient's medications and additional history did not reveal other sources of fluorine exposure. Epicutaneous tests made 6 months after the episode were negative. Because colchicine is thought to be antineutrophilic, treatment with colchicine was started. The half-life of excretion of inorganic fluoride from sevoflurane biodegradation is ~2–4 days.[3] Treatment with colchicine and topical corticosteroids was followed by resolution of the skin lesions within 7 days during hospitalization. There was no recurrence after discontinuation of treatment with colchicines during the following year.

A plasma inorganic fluoride concentration of 50 μmol litre^{-1} was previously associated with nephrotoxicity with methoxyflurane and was defined as the upper normal range.[4] Fluoride is excreted primarily via the urine. Accumulation of fluoride is rare, but the cytochrome P450 system could not be explored in this patient. No specific source of fluoride was found, but other sources including natural fluoride in foodstuffs and water (fluoridated water, fluoride supplements, fluoride dentifrices, and professionally applied fluoride gel) could not be easily detected.[5 6] The main source of fluoride for humans is the intake of groundwater contaminated by geological sources.[7]

Fluoroderma (a halogenoderma) is a rare cutaneous hypersensitivity reaction to fluorine exposure. Patients typically present with exudative plaques, fungating nodules, necrotic ulcers, and acneiform eruptions. Histological analysis reveals papillomatosis and accumulation of neutrophils in the dermis (without vasculitis). Treatment includes avoidance of the source of fluorine, wound care, and the administration of topical or systemic corticosteroids. To increase renal excretion of fluorine, diuretics and sodium chloride can be used. Although our patient appeared to have a good response to colchicine treatment, we are unable to rule out spontaneous resolution due to the excretion of inorganic fluoride. Fluorine intoxication from ingestion or dental care has been described,[8] but we are unaware of other reports of fluoroderma caused by sevoflurane. Most reported cases of halogenoderma have occurred after exposure to bromide and iodide.[9 10] In some studies, lymphocyte transformation tests were positive to iodinated proteins, suggesting a mechanism of hypersensitivity. The main hypothesis was that biotransformation of sevoflurane has exacerbated a chronic exposure to fluoride.

Millions of general anaesthesia are given each year around the world with sevoflurane. Physicians should be aware that fluoroderma is a rare, dose-related but life-threatening potential complication of sevoflurane. Colchicine seems to be an adequate treatment.

Conflict of interest

None declared.

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1 Holaday DA, Smith FR. Clinical characteristics and biotransformation of sevoflurane in healthy human volunteers. *Anesthesiology* 1981; **54**: 100–6
2 Kobayashi Y, Ochiai R, Takeda J, Sekiguchi H, Fukushima K. Serum and urinary inorganic fluoride concentrations after prolonged inhalation of sevoflurane in humans. *Anesth Analg* 1992; **74**: 753–7
3 Kharasch ED, Karol MD, Lanni C, Sawchuk R. Clinical sevoflurane metabolism and disposition. I. Sevoflurane and metabolite pharmacokinetics. *Anesthesiology* 1995; **82**: 1369–78
4 Cousins MJ, Mazze RI. Methoxyflurane nephrotoxicity. A study of dose response in man. *J Am Med Assoc* 1973; **225**: 1611–6
5 Hagmann WK. The many roles for fluorine in medicinal chemistry. *J Med Chem* 2008; **51**: 4359–69
6 Urbansky ET. Fate of fluorosilicate drinking water additives. *Chem Rev* 2002; **102**: 2837–54
7 Barbier O, Arreola-Mendoza L, Del Razo LM. Molecular mechanisms of fluoride toxicity. *Chem Biol Interact* 2010; **188**: 319–33
8 McIvor ME. Acute fluoride toxicity. Pathophysiology and management. *Drug Saf* 1990; **5**: 79–85
9 Roujeau JC. Neutrophilic drug eruptions. *Clin Dermatol* 2000; **18**: 331–7
10 Jih DM, Khanna V, Somach SC. Bromoderma after excessive ingestion of Ruby Red Squirt. *N Engl J Med* 2003; **348**: 1932–4

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Emergency airway management in a patient with a Montgomery T-tube *in situ*

Editor —We report a case of successful airway management during cardiorespiratory arrest in a patient with a Montgomery T-tube *in situ* and make suggestions for the management of similar cases. The patient was a 38-yr-old male with background medical history of quadriparesis after a cervical spinal cord injury who had multiple intensive care admissions for pneumonia. At the age of 35, he developed subglottic tracheal stenosis and had a long-term Montgomery T-tube inserted.

On this admission, the patient was treated for bronchopneumonia on a medical ward. He subsequently developed type 2 respiratory failure and rapidly progressed to respiratory arrest. The extratracheal lumen of the Montgomery T-tube was occluded with the stopper attached, and ventilation with a bag-valve face mask was attempted but proved difficult. The patient developed pulseless electrical activity and standard advanced life support management