attention to inspired flow rates, however, it seems that clinically compromising hypercapnia may develop. In our study several patients reduced the recommended flow rate because of headache and shaking. Despite this, most of the patients who had used cylinders before being allocated a concentrator found the concentrator a more acceptable means of oxygen delivery.

We conclude that the oxygen concentrator is the most economical means of providing domiciliary oxygen and appears to be acceptable to patients as a delivery system. Patients shown to benefit so far are those with cor pulmonale associated with chronic obstructive airways disease. The installations need to be supervised by chest physicians with a special interest in the treatment. After installation the oxygen flow rates should be adjusted in the home environment to achieve a $Pao_2$ of at least 8 kPa (60 mm Hg). This will be the recommended flow rate given to the patient. Machines need to be checked at minimum intervals of three months. For physicians concerned with only one or two machines it would be far better to arrange servicing through the manufacturer. At each visit the oxygen concentration of the effluent gas should be checked as well as the filters and mechanical components of the machine. The flow rate used by the patient should be compared with the recommended level. All machines should be purchased with a clock inside the mechanism so that the hours of daily use can be calculated. Ideally arterial gas tensions should be measured at each visit. The recent introduction of transcutaneous oxygen electrodes might simplify this process. A 24 hour replacement service for faulty concentrators works adequately and would save the cost of a spare cylinder. The dangers of smoking need to be constantly emphasised.

Large numbers of concentrators are now being sold. It must be remembered that few machines have been tested under the circumstances described in this study. Thus when the oxygen concentrator is introduced physicians must be careful to determine that the manufacturer is aware of the problems described and that equipment servicing schedules are designed to deal with them.

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# SHORT REPORTS

## Swimming pool wheezing

Swimming in indoor pools is promoted as an activity in which most people with asthma can participate without experiencing exercise induced wheeze.[1] Within the past few years, because of rapidly increasing energy costs, operators of about 200 public swimming pools have installed sophisticated heat reclamation systems that recirculate pool air and therefore concentrate chlorinous smells.

Chlorine gas is being largely phased out as a disinfectant of pool water for safety reasons, and the effects of other chlorine sources on bather comfort have recently been studied.[2] I carried out a survey using standard challenge swims in which bathers were asked to swim for 20 minutes sufficiently vigorously that they could just hold a conversation; they were allowed to rest holding the pool side when necessary. Standard temperatures (water 27·8°C and air 28·9°C) were usually maintained in the pools studied. During this study it became clear that some people with asthma (and some subjects with no history of wheezing) suffered attacks of bronchospasm when heat reclamation systems were in operation. Several small outbreaks and some individual cases were reported to me, and I investigated seven subjects. I describe here one typical case of swimming pool wheezing to illustrate the problems that may occur.

### Case report

A 57 year old man who was keen on physical exercise, especially jogging, swam regularly in two swimming pools in the same town. Both pools were under the same management, used the same water supply and the same disinfectant, and were maintained to the same standards. One pool had been built recently and used a heat reclamation system that recirculated a high proportion of the air in the pool hall and was controlled automatically. The older pool had a simple air extractor, which was under the control of the pool attendant.

The patient complained of coughing, sometimes severe, for 12 to 24 hours after swimming in the modern pool. He was apparently unaffected by swimming in the old pool. Swimming in the modern pool affected him more severely in winter than in summer. He related his coughing to "chlorine gas" present in the pool air but could not account for the seasonal variation or difference between the old and modern pools. There was no personal history of asthma exercise induced asthma, or atopy, although his sister had late onset asthma and his 28 year old daughter had had asthma since childhood.

Examination showed that he was in excellent health; height was 1·778 m (5′ 10″) and weight 68·9 kg (10 st 12 lbs). His vital capacity was 4·1 l, and forced expiratory volume in one second ($FEV_1$) 3·3 l.

Standard challenge swims in the new pool on two occasions reduced the $FEV_1$ to 2·2 l and 2·5 l respectively; bronchospasm was present on auscultation. Two challenge swims in the old pool reduced the $FEV_1$ from 3·3 to 3·1 l; 30 minutes' jogging did not affect it. Breathing air at water level in the modern pool for 20 minutes without exercising on two occasions reduced the $FEV_1$ to 2·5 l and 2·6 l respectively.

### Comment

Contrary to a widely held belief the chlorinous smells in swimming pools are caused not by chlorine gas but by nitrogen trichloride (an intense irritant) and, to a lesser extent, monochloramine and chloroform, which are produced when free chlorine (in solution as hypochlorous acid) reacts with organic contaminants introduced into the pool by bathers.[3] These contaminants are mainly urea and creatinine, which come from urine and sweat. Provided that organic contaminants are not present irritants are not produced and swimmers experience virtually no eye or respiratory problems.

The mechanism of production of bronchospasm in the patient described was probably similar to that in an outbreak in Manchester, in which an irritant stimulated hyperreactive bronchi.[4] Chlorine dioxide, which had been used to disinfect the water in Manchester, is no longer recommended because of severe recurring problems.[5]

The patient described here was troubled more when swimming in the winter because the automatically controlled heat reclamation system recirculated a high proportion of air in the winter to conserve heat and, conversely, expelled a high proportion of warm air in the summer because of solar heat gain. Nitrogen trichloride was therefore more concentrated in the winter and his bronchospasm more severe. In the older pool the simple air extraction system controlled by the bath attendant facilitated increased extraction of air when chlorinous smells became stronger.

In the modern pool complaints of respiratory and eye irritation were common from swimmers, spectators, and staff. The baths manager reported that these complaints had stopped almost entirely when a

fault occurred in the heat reclamation system and simple air extraction was used.

For intensively used public swimming pools there are no obvious alternatives to chlorine based disinfectants. A survey of bather comfort showed that problems with chlorinous smells are virtually non-existent when the organic contaminants are removed by the powerful oxidising action of ozone.[3] In this process swimming pool water is treated with ozone during filtration, the ozone is removed by a carbon filter before the water is returned to the pool, and a small free chlorine residue is maintained as a disinfectant in the pool. Unfortunately, the process is expensive to install in existing pools.


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# Dermatoses associated with brominated swimming pools

In recent years a small but increasing number of public swimming pools in the United Kingdom have been disinfected with a solid brominated compound rather than chlorine. This product has two proprietary names, Di-halo and Aquabrome, and its active constituent is 1-bromo-3-chloro-5,5-dimethyl-hydantoin. We present evidence that dermatoses have become more commonly associated with brominated pools than with chlorinated pools.

## Case reports

We saw 48 patients who developed dermatoses after swimming in pools. Two case reports are given below.

*Case 1*—The disinfectant used in a swimming pool was changed from chlorine gas to Di-halo. A month later a 32 year old male attendant began to develop widespread itchy red papules after being in the pool. Eczema subsequently erupted on his hands and patchily on his body; it improved away from work and relapsed rapidly on return. He could swim without symptoms in a chlorinated pool, but a rescue dive into the brominated pool resulted in an itchy red papular eruption within 20 minutes. He had no personal or family history of eczema, asthma, or hay fever. Clinically, he had a discoid (nummular) eczema of the trunk and limbs with a vesicular eczema of the palms and fingers. Patch tests with the International Contact Dermatitis Research Group standard series of allergens and with Di-halo (1% in water and 1% in petrolatum) gave negative results, as did prick tests with Di-halo (1% in water and 1% in petrolatum). No reactions occurred in eight other symptomatic patients patch tested with Di-halo at the same concentration.

*Case 2*—A 40 year old swimming instructor presented a similar picture. Her rash cleared when the pool changed to using a solid chlorine disinfectant (dichlorisocyanurate) but relapsed when she began to work in another pool treated with Di-halo.

### OTHER CASES

One of us (RJGR) has had eight independent reports, five from consultant dermatologists, of dermatoses associated with brominated pools. These dermatoses included pruritus, urticaria, patchy or discoid eczema, and more diffuse eczema.

We visited 19 brominated pools because of reports of rashes. At least 5% of users of a pool treated with Di-halo had recently experienced pruritus after swimming and had then developed rashes. A hot whirlpool bath treated with Di-halo was also used by some of these subjects. The most common dermatoses seen were discoid and asteatotic eczemas. Visits to pools indicated that high proportions of the staff were affected, which suggested that frequent exposure was relevant. Older age groups were affected much more commonly than children.

Postal surveys were made among the readers of the *Journal of the Institute of Baths and Recreation Management* and *Swimming Times*. As a result we received reports from 70 people who had suffered more than trivial rashes, 65 of which were associated with pools treated with Di-halo. Other symptoms particularly associated with such pools included soreness of the mouth, throat, vulva, female urethra, and breasts. Complaints of respiratory symptoms and eye irritation came from users of both brominated and chlorinated pools. Although the great majority of public swimming pools in the United Kingdom are chlorinated, in general only trivial rashes were associated with them. Of the 65 patients with rashes associated with pools treated with Di-halo, 58 developed the rash within 12 hours of swimming. None of these patients was affected by chlorinated pools unless the rash was severe and chronic.

## Comment

This report summarises the strong circumstantial evidence that swimming in pools disinfected with Di-halo or Aquabrome is associated with dermatoses, mainly eczematous in nature.

Bromine and chlorine ions are released into water from 1-bromo-3-chloro-5,5-dimethyl-hydantoin to leave 5,5-dimethyl-hydantoin (DMH). Accompanying reactions with pool contaminants such as urea and creatinine produce other chemicals including bromamines, chloramines, and complex organic bromine and chlorine compounds. The manufacturers' data on toxicity and results of our patch and prick tests indicate that allergy to the parent chemical or DMH is unlikely to be responsible; *Pseudomonas aeruginosa* infection is also unlikely. Brominated pools with the highest incidence of dermatoses tended to have the highest levels of total bromine residue (as measured with diethyl-p-phenylenediamine as an indicator) and hence the lowest bacterial counts. Dermatoses caused by *P aeruginosa* are clinically follicular and usually occur later than 12 hours after swimming. Several other factors that contribute to swimming pool rashes include wetting, wetting and drying cycles, previous skin disease, and dry skins, but these apply equally to brominated and chlorinated pools.

New products tend to receive an initial flurry of complaints that soon subsides, but complaints about Di-halo appear to be continuing. The evidence suggests that dermatoses occurring among users of pools treated with Di-halo may be forms of cumulative irritant contact dermatitis subject to acute exacerbations. The precise irritant or irritants have yet to be identified. It remains possible, though less likely, that sensitisation may be taking place.

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# Intranasal glucagon raises blood glucose concentrations in healthy volunteers

Polypeptide hormones are usually given parenterally, either by subcutaneous, intramuscular, or intravenous injection, since proteolytic digestion remains a limiting factor to administration by mouth.[1] The mucosa of the respiratory system is able to absorb some inhaled materials such as vasopressin[2] and luteinising hormone releasing hormone.[3] In addition, insulin is well absorbed through the nasal mucosa and lowers blood glucose concentrations in normal subjects as well as in patients with insulin dependent diabetes mellitus.[4,5] Hypoglycaemic episodes are a common emergency in the daily management of insulin dependent diabetes and glucagon, given subcutaneously or intramuscularly, is an effective remedy.