postoperative scores, and the units are not comparable across categories. All of the patients who had a left-sided procedure showed some degree of improvement in verbal abilities, while both the patients who had a right-sided operation in whom visual perception was tested showed improvement in this function.

Comment

These preliminary results suggest that extracranial to intracranial bypass may produce an improvement in some patients with long-standing moderate completed ischaemic stroke. As some of our patients had a longstanding total paralysis of arm before surgery, these deficits may have been regarded as too severe to fall within the criteria for entry into the Canadian co-operative study.⁵

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How quickly can haemoglobin A₁ increase?

The proportion of glycosylated haemoglobin (HbA₁) correlates with blood and urine glucose measurements, especially those made one to two months before.¹ HbA₁ is particularly valuable in assessing diabetic control, since its concentration is independent of short-term fluctuations in blood glucose. There is evidence that HbA₁ decreases one to two months after improved regulation of blood glucose,² though the time it takes to increase after development of hyperglycaemia is uncertain. A rise in HbA₁ was observed four weeks after development of hyperglycaemia in mice³ and six weeks after relaxation of control in postpartum diabetics.⁴ A significant increase in HbA₁ was also observed in normal people about one month after a glucose load.⁵ We decided to find how quickly a period of hyperglycaemia can lead to increased HbA₁ concentrations.

Patients, methods, and results

Twenty-nine insulin-dependent diabetics were studied. In each case HbA₁ was estimated at intervals of two to eight weeks over eight months by macrocolumn chromatography. Diabetic control was assessed with regular urine tests and morning blood glucose estimations. Of the 131 HbA₁ estimations performed, only those in which HbA₁ was increased are reported here.

Comparing each observation with the immediately preceding one we found an increase greater than 1% in 15; of these, six had increased more than 2% and three more than 3%. Five showed an increase in HbA₁ by two weeks (see figure). HbA₁ increased from a range for well-controlled diabetes⁴ in three patients (at intervals of two, four, and six weeks respectively). Before 11 of the 15 estimations hyperglycaemia had worsened: before six of these 11 patients developed persistent 2% glycosuria (four with symptoms), while five of the estimations were from postpartum diabetics whose control had been deliberately relaxed after delivery.

Comment

Most patients showed little change in their HbA₁ concentrations over two months. Some, however, showed large increases within even two weeks, and in two patients HbA₁ increased from a range for well-

Percentage increases in haemoglobin A₁ in 15 subjects according to interval between observations.

controlled diabetes by four weeks. Thus a single HbA₁ estimation may relate to a shorter period of hyperglycaemia than has been suggested.¹ Such rapid changes in HbA₁ limit its value in assessing diabetic control retrospectively, and conclusions about diabetic control drawn from single HbA₁ estimations should be treated with caution.

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Upper airways obstruction after Dettol ingestion

The following case report describes a hitherto unreported effect of Dettol ingestion which may be important in the management of Dettol poisoning.

Case report

A 22-month-old girl was admitted to hospital 40 minutes after drinking a maximum of 125 ml Dettol. She complained of a sore mouth but had not vomited. Her birth weight had been 1960 g at 33 weeks' gestation. She had been asphyxiated at birth and developed the idiopathic respiratory distress syndrome, from which she had recovered quickly. There was no record of tracheal intubation or controlled ventilation during this period. She had not been prone to upper respiratory tract infections subsequently and had had no hoarseness or noisy breathing. She had had no respiratory symptoms or contact with infectious illnesses immediately before ingesting Dettol.

On arrival in hospital she was conscious and alert. Her temperature was 36.5°C, pulse 112/min, and respiration 28/min. She had mild inspiratory stridor and erythema of the chin and anterior chest wall. Her buccal mucosa was inflamed. Vomiting was induced with syrup of ipecachuana. Thereafter she became progressively more stridulous with subcostal and intercostal retractions. She did not appear to have inhaled vomitus, and there were no signs suggestive of aspiration on a chest radiograph. Within two hours of vomiting she had pronounced stridor with severe retractions and was in imminent danger of complete upper airway obstruction. A nasotracheal tube was passed under general anaesthesia, which immediately relieved her

symptoms. Haematological values, plasma calcium, electrolyte, and urea concentrations were all normal before intubation. Bacteriological and virological studies of secretions from the nose and nasopharynx gave negative results.

Extubation was attempted after two days, but she had to be reintubated one hour later because of persistent severe stridor. She was extubated again on day five. Direct laryngoscopy showed hyperaemia and ulceration of the vocal cords, which moved normally. The subglottic area was normal. She could not maintain an unobstructed airway and was intubated for a third time.

At 10 days the vocal cords were inflamed and ulcerated. The buccal mucosa had healed but her chest wall was still strikingly erythematous. Tracheostomy was performed as she remained stridulous after further extubation. The tracheostomy tube was removed seven days later, by which time her vocal cords looked normal. Thereafter she made a full and uneventful recovery.

Comment

Dettol is a widely used household disinfectant, described on the label as non-poisonous. Serious poisoning is rare, though deaths have been reported in adults.^{1,2} In one study of 687 cases of Dettol ingestion² there were three deaths. The toxic effects of Dettol and its active constituents are well documented,^{1,3} and its respiratory complications include wheeze and respiratory depression. Stridor has not been reported, though in one patient tracheal corrosion was found at necropsy.¹

Ulceration of the vocal cords in our patient was probably due to Dettol, perhaps following the induction of vomiting. Gastric emptying is generally advised as part of the management for potentially serious Dettol ingestion. If gastric lavage is carried out, or vomiting occurs or is induced, it would be a wise precaution to have facilities and staff available for tracheal intubation or tracheostomy.

I thank Dr Hamish Simpson for granting me permission to report this case.

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Contact allergy to methoxsalen

Photochemotherapy (PUVA) is effective in treating psoriasis. Since a major report¹ in 1974 it has been extensively used. Earlier, furcoumarins (psoralens) had been advocated to repigment vitiligo.² In treatment with PUVA psoralen preparations are given either by mouth or topically as a solution, ointment,³ or emulsion and are then exposed to long-wave ultraviolet light (UVA; 320-400 nm). For localised forms of psoriasis, particularly psoriasis of the palms and soles, topical application of psoralen is preferable. Although topical preparations have been used for some time, contact allergy to psoralen has not been reported. I here report two cases of allergic contact sensitivity to methoxsalen (8-methoxypsoralen).

Case reports

Case 1—A 50-year-old housewife who had had psoriasis for about 18 months, mainly on her palms, was treated with PUVA as her response to conventional local treatment was poor. Methoxsalen (0.15% Meladinine emulsion; Basotherm) was applied, and 30 minutes later the areas were exposed to a UVA light source. The procedure was repeated thrice weekly. Initially a slight improvement occurred, but after 16 treatments she developed an erythematous rash on her chin and complained of increased palmar irritation. This rash was thought to be a contact eczema due to Meladinine emulsion. Patch tests with the International Contact Dermatology Research Group (ICDRG) series were negative, but an allergic-type reaction occurred to 0.15% Meladinine emulsion. Subsequently she was patch tested with different constituents of the Meladinine emulsion. The table shows the results.

Results of patch testing two patients with ICDRG series and constituents of Meladinine emulsion at 48 and 96 hours

Substance tested	Form of substance	Case 1		Case 2	
		48 h	96 h	48 h	96 h
ICDRG series		—	—	—	—
Propylene glycol	10% aq sol	—	—	—	—
Benzalkonium chloride	1:1000 aq sol	—	—	—	—
Lanette (emulsifying wax)	as is	—	—	—	—
Spermaceti	as is	—	—	—	—
Span 40 (sorbitan derivative)	5% aq sol	—	—	—	—
Tween 40 (sorbitan derivative)	5% aq sol	—	—	—	—
Cetiol V (Basotherm)	as is	—	—	—	—
"	10% in petroleum	—	—	—	—
Meladinine emulsion	as is	++	++	++	++
Methoxsalen	10% in petroleum	++	++	++	++
"	1% in petroleum	++	++	++	++

ICDRG = International Contact Dermatology Research Group.

Case 2—A 56-year-old shop assistant with psoriasis of the palms of five months' duration had been treated with PUVA, as in case 1. There was steady improvement in the first six weeks. She then started to complain of increased irritation of her palms and spreading of rash to the fingers. Examination at that stage showed psoriasis of the palms and pompholyx eczematous changes of the fingers. She was patch tested with the ICDRG series and with 0.15% Meladinine emulsion and its constituents. The table shows the results.

Comment

By using psoralen topically the systemic absorption is reduced as well as the total exposure to UVA. As the topical preparations contain different ingredients allergic reaction to these substances may occur. Although psoralen preparations such as trioxsalen⁴ and methoxsalen³ have been used before, contact allergy to them has not been reported. Both our patients showed a contact allergy to methoxsalen.

The possible development of an allergic reaction to psoralen may be an important consideration in patients receiving PUVA treatment with topical psoralen preparations. Additionally, such patients may also be sensitised to oral psoralen, since ingestion of a contact allergen by a sensitised person can cause dermatitis.⁵ Thus in patients whose response to topical PUVA is poor or in whom the psoriasis suddenly worsens during treatment with PUVA allergic sensitisation to topical psoralen should be considered.

I thank Drs J S Pegum and H Baker for their advice and permission to report their cases.

The individual constituents of Meladinine emulsion were kindly supplied by Drs Renovanz and Schulz (Basotherm).

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Corrections

Blood-pressure screening and supervision in general practice

The above paper by Professor J H Barber and others was printed in the *BMJ* of 31 March (p 843). We regret that the name of Dr G A Walker was inadvertently omitted from the draft list of authors we received. His address and name should have appeared in the list as follows: Bridge of Allan, Stirlingshire, G A WALKER, MB, MRCPGLAS, general practitioner.

Oral metronidazole in *Clostridium difficile* colitis

In the above paper by Dr N L Pashby and his colleagues (16 June, p 1605) the dosage of oral clindamycin was given wrongly in both case reports. It should have read 150 mg six-hourly (not 250 mg).