Treating hyperhidrosis

Humans have two functionally separate sets of eccrine sweat glands that respond to heat and emotion. One is unique to the species, populates the entire skin apart from the palms and soles, and is responsible for thermal regulation. Sweat glands of the palms and soles respond mostly to emotion, but those of the axilla respond to both heat and emotion. Sweating presents to doctors when patients perceive it as inappropriate and excessive.

Generalised hyperhidrosis may be a presenting feature of hyperthyroidism, chronic infection, or autonomic neuropathy and has been described with pheochromocytoma, the carcinoid syndrome, diabetes mellitus, acromegaly, hyperpituitarism, and gout. Local hyperhidrosis of the palms, soles, and axillae should be managed by topical treatment with a saturated solution of aluminium chloride hexahydrate in absolute alcohol, which is effective for most patients. This substance is irritating in the axilla, but the production of sweat can be controlled by carefully applying the lotion last thing at night and cautiously using mild topical steroids to suppress irritation.

A few patients do not respond to this treatment, and drugs, an operation, or both have been tried to relieve their symptoms. Systemic treatment with benzodiazepines to reduce anxiety is ineffective unless extreme drowsiness is produced. Anticholinergic drugs are a logical choice but the side effects of dry mouth, blurred vision, etc may limit their use. Axillary hyperhidrosis may be reduced by excising an ellipse of skin from the vault, but scarring and loss of full mobility may ensue. Sympathectomy is effective for palmar hyperhidrosis but carries a small risk of operative side effects—and the permanently non-sweating palm may become hyperkeratotic with both fissuring and scaling as distressing sequels.¹

Electrical treatments for hyperhidrosis were used in the 1930s,² and the publication of a description of a simple device for iontophoresis has led to a revival of interest in this subject.³ Two electrodes and a reliable source of low amperage direct current are required: one electrode is placed under the area to be treated in a shallow water bath or under a pad of cotton wool soaked in electrolyte; the other is attached similarly to the contralateral limb. Axillae may be treated by a moistened pad placed over an electrode in the axillary vault. Iontophoresis together with induction of the anticholinergic agents poldine methosulphate⁴ or glycopyrronium bromide⁵ into the skin produce freedom from sweating for four to six weeks after a few exposures. Iontophoresis with plain tap water alone is also effective but needs more exposures for the same effect.⁶ Tap water also eliminates the side effects from systemic absorption of the anticholinergic drugs. Iontophoresis works for both palmar and plantar hyperhidrosis and may reduce sweating enough for daily application of aluminium chloride hexahydrate to work. Unfortunately the technique is less successful in the axillae, which may need up to three times as many exposures as hands and feet. The mechanism of action of iontophoresis remains a mystery. Suggestions have included blockage of the sweat duct⁷ and a reversal of a so called "electric gradient" along it.⁸ These conclusions have been drawn, however, from experiments on skin from the back or the forearm, both of which are the wrong models for axillary, palmar, or plantar sweat glands.

Most dermatologists in Britain use the services of local

physiotherapy departments, where the right electrical equipment can be found. A battery run machine has been recommended for home use in the United States of America⁹; with the heavy workload in departments of physical medicine such a machine might be useful here.

NICK SIMPSON

Consultant Dermatologist,
Glasgow Royal Infirmary,
Glasgow G4 0SF

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Restraining infants in cars

In 1985, 29 children aged 4 or under were killed while travelling in cars in Britain. A further 328 were injured seriously, and 2471 sustained minor injuries.¹ Such figures represent incalculable pain and suffering to the children and families affected, and one way to improve them would be to ensure that more children in cars are restrained.

Restraint systems for infants have been available in Britain for many years. The earliest were harnesses that secured the child's carrycot across the rear seat. Recently the rearward facing infant carriers have become popular, allowing babies to face their mothers while strapped into reinforced seats that are secured on front passenger seats by adult seatbelts. These infant seats are inexpensive and require no new fitments.

The value of restraining children in cars is well established, but further research is badly needed on the injuries sustained specifically by restrained and unrestrained infants. A study by the Transport and Road Research Laboratory showed that of 150 child passengers in 1126 road traffic accidents in the Thames Valley 134 (89%) were unrestrained.² Of these unrestrained children 17 were killed or sustained serious injuries—five as a result of ejection from the car and two from sitting on their parents' laps. Yet none of the 16 restrained infants sustained serious injury. A further study of children of all ages showed that restraining children reduced the overall injury rate by two thirds,³ while a survey in the United States indicated that children under 4 were more than ten times more likely to be killed in a road traffic accident if unrestrained.⁴

Despite this evidence few people in Britain take the trouble to restrain their children in cars. Recent surveys agree that at best only about one third of children in cars are restrained.³ Infants and young children are more likely to be restrained than older children, but compliance is far from satisfactory. The failure to use restraint systems may be because of the cost of purchase or installation or simply the familiar attitude that "It won't happen to me (mine)."

To overcome these problems schemes have sprung up to lend safety seats to the mothers of newborn children. One of the earliest schemes was organised in Dunedin, New Zealand, in 1981: the parents of every child born in the town's hospital were offered the loan at minimal cost of an infant car seat.⁴ After the initial capital expenditure the scheme became self funding. It was so successful that within three years 300 others had been set up in New Zealand. Loan schemes now exist in many parts of the Western world. In Britain about 50 schemes are known to exist: about half a dozen have been organised by public bodies, and the rest are being run by commercial organisations. The Child Accident Prevention Trust maintains a watching brief over these schemes and is available to offer advice when required. The trust is also conducting a detailed study of eight of the schemes in association with the Transport and Road Research Laboratory.

The loan schemes ensure that mothers have the means to carry their babies home safely from the maternity hospital, setting a safe pattern. They offer the double benefit of both accident prevention through education and practical help and should be available to all families in Britain.

M A MASON

Consultant in Charge,
Accident Centre,
Medway Hospital,
Gillingham,
Kent ME7 9NY

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Replacement of the shoulder

Hip replacement is an everyday occurrence in Britain, and in the past decade knee replacement has become almost as common. But what hope is there of replacing the shoulder for patients with painful arthritis of that joint?

In 1982 Lettin *et al* showed that it was possible to replace the shoulder with a constrained ball and socket cemented into the humerus and scapula (the Stanmore prosthesis).¹ Patients gained early relief of pain, but restoration of active movement was inconsistent probably because the prosthesis did not allow the normal descent of the humeral head on abduction. In addition, many of the glenoid components eventually came loose causing increasing pain. Also in 1982 Neer *et al* described an unconstrained surface replacement.² Pain relief was as good as with the Lettin prosthesis, but restoration of active movement was better and the glenoid components did not loosen so often. In patients with large rotator cuff ruptures (more than 5 cm) pain relief was good, but restoration of active movement was poor. Comparison between the results of these two groups has to be crude because neither used a standard assessment. Standardisations suggested by the American Shoulder and Elbow Surgeons and the European Society for Surgery of the Shoulder and Elbow should allow more accurate comparison soon.

Other surveys have used the Neer prosthesis,^{3,6} and all have achieved poorer results with patients with large cuff ruptures. Barret *et al* also noted that a large cuff rupture was associated with glenoid components loosening more often.⁵ Poppen and Walker showed that the cuff's function is to stabilise the joint while the deltoid muscle moves the arm on the scapula.⁷ A large cuff defect causes this function to be lost, and the joint subluxes on attempted abduction.

The Stanmore prosthesis.

The Neer prosthesis.

Unfortunately it seems unlikely that surgeons will be able to repair the large cuff defects around the prostheses. Attempts have been made to repair the large cuff defects around the Neer prostheses but active movement has not been much improved.^{2,6} Indeed whatever method of repair is used the most important determinant of the outcome is the size of the cuff defect: the bigger the defect the poorer the result.⁸

Repair of the cuff is also unlikely to reduce the rate of loosening of the glenoid component because such loosening is probably caused by eccentric loading of the component by the subluxed humeral component on attempted abduction. It remains to be seen whether the recently developed reinforce-