Postural hypotension in the elderly

Aging may cause an appreciable age related impairment in the homeostatic mechanism that corrects blood pressure as posture changes.¹ The impairment seems to be related to physiological insufficiency in the aged autonomic nervous system and to failure of vascular righting reflexes.²⁻⁸ When a patient with postural hypotension changes position hypotension causes secondary cerebral ischaemia: a sensation of giddiness (commonly referred to as a "giddy spell") may be followed by syncope. This happens most commonly when the patient stands quickly after lying or sitting. The problem unnerves elderly people and may lead to loss of confidence and a fear of falling, and it may cause serious bodily injury, a fractured femur,⁷ or even death^{8,9} if unconsciousness occurs with the patient wedged in the vertical or sitting position.

Postural hypotension of sudden onset may result from a symptomless cardiac infarction or occult arrhythmia.^{2,3} It is more likely in patients with diabetes complicated by autonomic neuropathy. The patient usually presents with a complaint of giddy turns and falls. The giddiness is described vaguely and with difficulty and may be misdiagnosed as vertigo: a useless prescription of prochlorperazine is often made. But patients do not have a sensation of rotation, and vertigo is a rare cause of falling in old age.^{6,8} The relation to postural change makes the diagnosis, and patients should have their blood pressure measured in the lying and sitting positions: a drop of 20 mm Hg or more in the systolic pressure on sitting is taken as diagnostic.

Some cases are caused by drugs. Postural hypotension may follow the overenthusiastic treatment of hypertension or appropriate long term treatment with diuretics for postural oedema or heart failure that may have resolved.⁵ Hyponatraemia is the mechanism. Many drugs (at least 90) cause hypotension,⁷ all may aggravate or precipitate postural hypotension.

Simple measures may ease the effects of postural hypotension in the elderly. Firstly, the mechanism of the symptoms should be explained to patients, and they should be taught a gradual drill of changing posture slowly with 10 second pauses to allow the failing reflexes to accommodate. Secondly, patients may have their symptoms relieved if they sleep with the head of the bed raised.^{2,10-12} Thirdly, vulnerable patients should always be accompanied when out of doors by someone who understands the problem. Fourthly, any drugs the patient is taking should be reviewed: diuretics causing hyponatraemia should be stopped and the benefit of hypotensive treatment reassessed. Only one drug is advised for treatment—fludrocortisone acetate 0.1 mg daily.^{10,13,14} It should be used only when the patient is under hospital surveillance.^{10,13,14} Patients with persistent symptoms may benefit from wearing elastic stockings or support hosiery, but these are unpopular unless there is someone to help put them on. With fairly simple measures the distressing effects of this common condition may often be ameliorated, and the quality of life improved.

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Steroids and growth

The most compelling evidence that steroids slow growth is the catch up in growth that occurs when Cushing's syndrome in a young child is treated.¹ Exogenous glucocorticoids have been known to be potent inhibitors of growth in children almost since they first became available in the 1950s.² Giving long term treatment with adrenocorticotrophin was advocated to achieve the same therapeutic effect yet avoid suppression, but this unpleasant regimen is no longer used.³

Later glucocorticoids given on alternate days were shown to have less effect on growth than daily glucocorticoids when prescribed for chronic disorders such as rheumatoid arthritis, the nephrotic syndrome, and asthma.^{4,6} A daily prednisolone dose of 0.6 mg/kg body weight suppressed growth in children with rheumatoid arthritis, whereas prednisolone 2 mg/kg given on alternate days maintained normal growth.⁶ Converting children from a daily to an alternate day regimen produced catch up growth in those whose growth had been suppressed. Other factors associated with diseases such as rheumatoid arthritis, renal insufficiency, and ulcerative colitis also slow growth, and controlling the activity of the primary disease with the minimum amount of glucocorticoid is not easy. Choosing the glucocorticoid dose required to prevent graft rejection in children after renal transplantation is equally difficult.⁷

What glucocorticoid dose avoids growth suppression in children? No single figure can be used with certainty for any one child. The type of glucocorticoid, the way it is given, and the length of treatment must all be considered. The normal cortisol secretion rate is 31.7 (SEM 6.9) $\mu\text{mol/m}^2/\text{day}$.⁸ The relative potencies of some commonly used glucocorticoid preparations compared with cortisol (defined in standard texts in relation to effects on carbohydrate metabolism) are: cortisone 0.8, prednisone and prednisolone 4, and dexamethasone 30. Their potency in suppressing pituitary-adrenal function and possibly growth may, however, be different—for example, dexamethasone is 80-100 times more potent than cortisol in suppressing adrenal function.⁹ Prednisolone 1-2 mg/kg given in the morning on alternate days is unlikely to inhibit growth when used as maintenance treatment. Replacement dosages are required for children with hypopituitarism and primary adrenal insufficiency and should be given as cortisol (hydrocortisone) 5-10 mg daily. Hydrocortisone pellets 2.5 mg (Corlan) designed for topical treatment of oral lesions may be used to ensure accurate dosage in the young child.

Aerosol steroids are now often used to treat children with

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2 Bannister R. Chronic autonomic failure with postural hypotension. *Lancet* 1979;ii:404-6.

asthma, and recent reports show no evidence of growth retardation in the long term with doses of beclomethasone of 400-600 µg/day.^{10,11} Endogenous cortisol secretion may be affected by these doses,^{12,13} and injudicious use of the stronger formulations of beclomethasone now on the market may slow growth. Glucocorticoids in large doses will inevitably suppress growth, but the recent development of knemometry, which means that rates of linear growth can be calculated at time intervals as short as daily or even less,¹⁴ might be used as a sensitive marker of growth suppression. Treatment with growth hormone will not overcome the effects of steroids because they act directly on epiphyseal cartilage metabolism. Catch up growth will occur when the glucocorticoid dose is reduced. Those who prescribe maintenance glucocorticoid treatment in children must ensure that monitoring of growth is an integral part of their management.

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Expanding possibilities

The principle of tissue expansion is plain for all to see. Pregnancy, obesity, lymphoedema, and various other conditions may stretch the skin and produce redundant tissue that may need surgical removal. It requires, however, an imaginative leap to turn a surgical problem into a surgical tool. In 1976 Radovan presented skin expansion as a new technique for the reconstructive surgeon (C Radovan, American Society of Plastic and Reconstructive Surgery Forum, Boston, September 1976). An inflatable silicone implant is placed surgically underneath normal skin adjacent to a defect or area that is to be excised, and over the next few weeks the implant is inflated through a remote subcutaneous or external valve by injecting normal saline. Once enough skin has been generated the implant is removed, the lesion excised, and the defect covered using the excess skin produced by the expander. In effect, a skin flap has been preformed at an ideal site.

The technique is now widely used in the United States and

is slowly becoming established in Britain. The concept is appealing because in many areas of the body the skin has specific characteristics that cannot be matched by grafts or distant flaps. The best example is the hair bearing scalp—extensive loss of hair leaves a defect that is difficult to replace permanently. Tissue expansion allows the use of adjacent matching scalp, leaving no donor defect. The technique is simple in principle and requires no specialist knowledge or equipment; it may therefore be used by many disciplines. (G H Sasaki, T J Krizek, Plastic Surgery Research Council, Durham, North Carolina, May 1983.)

Tissue expansion started as a clinical experiment, and only in the last few years have researchers looked at what happens to the expanded skin. It has now been shown that the epidermal thickness remains the same as in non-expanded skin but the dermis becomes progressively thinner.^{1,2} The dermal thinning is, however, compensated for by the thickness of the capsule surrounding the implant. Whether the dermis eventually returns to normal is not known. Hair follicles and other adnexal structures become separated in expanded skin, but their morphology remains normal and the separation is rarely clinically important. The blood supply of flaps raised in expanded skin is superior to that of identical flaps raised in normal skin.³ This may be caused by the vascularity of the capsule around the implant,⁴ or by an increase in the size and number of vessels that are clearly visible in expanded skin.

The effect on other structures is less clear. Expanded muscle undergoes some atrophy, but function is rarely lost.⁵ This opens up possibilities in tailoring innervated muscles to reconstruct something like facial palsy. The effects on nerves, tendons, and other structures are being investigated; the benefits of being able to fashion muscle, tendon, and nerve units would be enormous.

Tissue expansion is not, however, a panacea for all reconstructive problems. Time—from 3-25 weeks depending on site, age, the amount of skin required, and possible complications^{1,6}—is required to inflate the balloon. In general, patients are warned that 6-10 weeks will be required between inserting the expander and its final removal. During this time the patient has to attend the hospital at least twice a week to have the expander inflated by a percutaneous injection. This is expensive for patients who live a long way from the hospital or who have other social or economic commitments. There is also the problem of the appearance of the expander, and, however thorough the preoperative counselling, few patients seem prepared for the size of the temporary deformity that is produced. When the expander cannot be hidden under clothing or a hat—as in facial reconstruction—many patients prefer to remain indoors between hospital visits.

The biggest problem is the complication rate, which may be as high as 40%.⁶ The commonest complications are infection, haematoma, exposure of the expander or tubing, and failure of the implant, causing a leak and deflation. Most of these complications do not affect the final result and can be controlled conservatively while inflation continues. This complication rate will be reduced with experience and with advances in implant design.

Few areas of the body cannot be expanded, but we do not know how often the same area of skin can be expanded. We have done it three times with no noticeable skin changes. Multiple expansion offers possibilities of being able to resurface large areas of the body, when treating scarring from burns or giant hairy naevi. So far tissue expansion has been