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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