home places the absolute numbers of demented, incontinent, heavily dependent patients in nursing homes outweighed the total of those in continuing care wards.

Health economists, managers, and politicians need to grasp several points. Firstly, equating nursing home costs to those of continuing care in hospital is increasingly specious if it is assumed that nurse staffing levels are adequate for the patient cohort of today. In our experience nursing homes will often accept apparently uneconomic heavily dependent patients provided they have several "lighter" patients to offset this load. This, however, cannot be the case in hospital wards, which are caring for increasingly dependent patients. Secondly, we are troubled by dual standards. The quality of life of many dependent patients is often maintained by paramedical professions such as physiotherapists and occupational therapists, yet some patients in private nursing homes do not receive basic geriatric care. At the same time we find hospital patients enduring impersonal care on public dormitory wards in conditions that fall a great deal short of registration standards for private homes; this is permitted only because the health authorities are charged with supervising the private sector while providing the resources for public continuing care provision against the competing claims of other departments.

We hope that future planning for long term care of dependent elderly patients will recognise the potential benefits of improving the range of small units providing continuing care rather than relying on the chronic sick wards that currently provide the nucleus of long term geriatric care in hospital. The time is long overdue for some sense to be injected into the system of funding continuing care, by recognising disability as the index of payment rather than simply place of residence. The quality and quantity of care provided must be assessed, supervised, and helped by active geriatric departments to avoid recreating conditions that confronted Marjorie Warren and her peers on taking over chronic sick wards in the 1940s.

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Psoriasis

SIR,—In his otherwise comprehensive review article on psoriasis Dr R H Champion makes two rather sweeping and unqualified statements with which we feel we must take issue.

The comments that "no major breakthrough has been made as to our understanding or treatment of the disease" in the past five years and that immunological changes are "quite likely, too, to be secondary" are untrue. From our departments and others there is now an increasing body of evidence which testifies to the importance of an interaction between epidermal T lymphocytes and antigen presenting cells in the initiation and maintenance of psoriatic lesions.¹⁻³ Early guttate psoriatic lesions are characterised by activated T helper cells in the epidermis, and the close apposition of these cells to antigen presenting cells is a striking feature. In contrast, resolution is characterised and preceded by an epidermal influx and activation of T suppressor cells.

Studies on unaffected skin have shown T cell

infiltration of the dermis, similar to that of affected skin, with a predominance of T helper cells, of which about 40% are activated. Our studies of T cells in psoriatic epidermis during treatment with steroids or psoralen and ultraviolet A (PUVA)^{4,5} showed a pronounced fall in numbers of T cells before healing of the psoriatic plaque. Moreover, the rate of healing correlated in each individual with the rate of disappearance of T cells from lesional epidermis.

The clearance of psoriasis with cyclosporin,⁶ which has a selective action on T helper cells but no direct effect on epidermal cell kinetics,⁷ adds further credence to the notion that psoriasis is a disorder of abnormal keratinocyte proliferation induced by T lymphocytes.³ Indeed, many of the current therapeutic regimens for psoriasis may act on immunological processes rather than being directly cytostatic for epidermal cells. We believe that the emphasis in the treatment of psoriasis should now be shifted away from attempts to inhibit keratinocyte proliferation to agents which can influence the regulation of immune mechanisms.

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SIR,—We would like to take up one issue arising out of Dr R H Champion's review of psoriasis (28 June, p 1695). He mentioned the drug cyclosporin, among others, as being interesting, but we believe that this is an understatement. We would like to report our experience with this drug in the treatment of one very severe case of erythrodermic and pustular psoriasis.

A 30 year old woman with longstanding psoriasis and pronounced mutilating arthropathy had received most forms of conventional treatment as well as methotrexate and razoxane over the past six years. None of these treatments, either alone or in combination, made a significant impact on her psoriasis and she continued to require frequent hospital admissions. On her last admission flexural pustulation added a further dimension to her already difficult problem. More recently her haemoglobin dropped to below 100 g/l and she became hypoalbuminaemic, though her plasma urea and serum creatinine concentrations remained normal. She was started on oral cyclosporin in a dose of 150 mg twice daily (5 mg/kg/day), and her response was extremely impressive. Within three days her erythrodermic skin became considerably paler and by the end of the week she was virtually clear except for the flexures, where the response was slower. Her progress continued, and by the end of the second week the flexures were also clearing. During this period her

renal function, as measured by regular estimations of plasma urea, serum creatinine, and creatinine clearance, did not deteriorate. There was no proteinuria and her haemoglobin and serum albumin concentrations started rising.

We therefore feel that the potential value of cyclosporin in psoriasis is more than interesting. Clearly further studies to determine longer term effectiveness and side effects are required, but we have never previously encountered such a dramatic response to treatment in psoriasis and are encouraged to believe that cyclosporin is in a different category from the other drugs that Dr Champion listed. The experience of others in using cyclosporin to treat psoriasis appears to have been similar to our own.¹⁻³

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Does sodium restriction lower blood pressure?

SIR,—Drs D E Grobbee and A Hofman conclude (5 July, p 27) that sodium restriction seems to be of limited use in those who are most eligible for non-pharmacological treatment of high blood pressure—namely, young patients with mild hypertension. While probably true for white people, this conclusion may not hold for blacks.

There is growing evidence that the range of hypertension in blacks is different and may be more salt and volume dependent. They tend to have low renin hypertension¹ and to be more sensitive to thiazides and less sensitive to β blockade and angiotensin converting enzyme inhibition.² On being salt loaded their blood pressure rises more than that of whites³ and they have an increased age related prevalence of hypertension.⁴

Recent work in this department suggests a renal basis for this salt sensitivity and volume expansion. Whereas whites mobilise renal dopamine promptly when salt loaded,⁵ blacks have lost the sodium to dopamine coupling and do not mobilise renal dopamine on being given added dietary salt.⁶ Dopamine generated intrarenally contributes to natriuresis and renal vasodilation, a hypotensive response.⁷

Blacks probably lost (or never acquired) the renal dopamine response as an evolutionary adaptation to hot humid environments.⁸ Now that salt is superabundant this good adaptation has become a maladaptation and results in a high prevalence of hypertension in blacks with increasing age.

In our view salt restriction, to between 50 and 100 mmol (mEq) a day, should be a central plank of management in blacks with essential hypertension; in black populations living with a Westernised diet; and in whites of the "salt sensitive" subgroup. The prevalence of the dopamine fault in the latter group has yet to be established.

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